third edition

DEVELOPMENTAL PSYCHOPATHOLOGY

VOLUME 1

THEORY AND METHOD

EDITED BY **Dante Cicchetti**

DEVELOPMENTAL PSYCHOPATHOLOGY

DEVELOPMENTAL PSYCHOPATHOLOGY

THIRD EDITION

Volume One: Theory and Method

Editor

DANTE CICCHETTI

WILEY

This book is printed on acid-free paper.

Copyright © 2016 by John Wiley & Sons, Inc. All rights reserved.

Published by John Wiley & Sons, Inc., Hoboken, New Jersey.

Published simultaneously in Canada.

No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, scanning, or otherwise, except as permitted under Section 107 or 108 of the 1976 United States Copyright Act, without either the prior written permission of the Publisher, or authorization through payment of the appropriate per-copy fee to the Copyright Clearance Center, Inc., 222 Rosewood Drive, Danvers, MA 01923, (978) 750-8400, fax (978) 646-8600, or on the web at www.copyright.com. Requests to the Publisher for permission should be addressed to the Permissions Department, John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, (201) 748-6011, fax (201) 748-6008.

Limit of Liability/Disclaimer of Warranty: While the publisher and author have used their best efforts in preparing this book, they make no representations or warranties with respect to the accuracy or completeness of the contents of this book and specifically disclaim any implied warranties of merchantability or fitness for a particular purpose. No warranty may be created or extended by sales representatives or written sales materials. The advice and strategies contained herein may not be suitable for your situation. You should consult with a professional where appropriate. Neither the publisher nor author shall be liable for any loss of profit or any other commercial damages, including but not limited to special, incidental, consequential, or other damages.

This publication is designed to provide accurate and authoritative information in regard to the subject matter covered. It is sold with the understanding that the publisher is not engaged in rendering professional services. If legal, accounting, medical, psychological or any other expert assistance is required, the services of a competent professional person should be sought.

Designations used by companies to distinguish their products are often claimed as trademarks. In all instances where John Wiley & Sons, Inc. is aware of a claim, the product names appear in initial capital or all capital letters. Readers, however, should contact the appropriate companies for more complete information regarding trademarks and registration.

For general information on our other products and services please contact our Customer Care Department within the United States at (800) 762-2974, outside the United States at (317) 572-3993 or fax (317) 572-4002.

Wiley publishes in a variety of print and electronic formats and by print-on-demand. Some material included with standard print versions of this book may not be included in e-books or in print-on-demand. If this book refers to media such as a CD or DVD that is not included in the version you purchased, you may download this material at http://booksupport.wiley.com. For more information about Wiley products, visit www.wiley.com.

Library of Congress Cataloging-in-Publication Data:

Developmental psychopathology / editor, Dante Cicchetti. – Third edition.
pages cm
Includes index.
ISBN 978-1-118-12087-3 (volume 1 : cloth : alk. paper) – ISBN 978-1-118-12091-0 (volume 2 : alk. paper) – ISBN 978-1-118-12092-7 (volume 3 : alk. paper) – ISBN 978-1-118-12093-4 (volume 4 : alk. paper) 1. Mental illness–Etiology. 2. Developmental psychology. 3. Mental illness–Risk factors. 4. Adjustment (Psychology) I. Cicchetti, Dante.
RC454.4.D483 2016
616.89–dc23

2015018216

Printed in the United States of America

10 9 8 7 6 5 4 3 2 1

These volumes are dedicated to Marianne Gerschel in recognition
of her great vision and staunch support of the field
of developmental psychopathology.

Contents

Preface to Developmental Psychopathology, Third Edition xi
Dante Cicchetti

Contributors xiii

1 **ASSESSMENT OF PSYCHOPATHOLOGY IN YOUNG CHILDREN** 1
Margaret J. Briggs-Gowan, Leandra Godoy, Amy Heberle, and Alice S. Carter

2 **DEVELOPMENTAL ISSUES IN ASSESSMENT, TAXONOMY, AND DIAGNOSIS OF PSYCHOPATHOLOGY: LIFE SPAN AND MULTICULTURAL PERSPECTIVES** 46
Thomas M. Achenbach and Leslie A. Rescorla

3 **DEVELOPMENTAL EPIDEMIOLOGY** 94
E. Jane Costello and Adrian Angold

4 **USING NATURAL EXPERIMENTS TO TEST ENVIRONMENTAL MEDIATION HYPOTHESES** 129
Michael L. Rutter and Anita Thapar

5 **DEVELOPMENTAL MODELS AND MECHANISMS FOR UNDERSTANDING THE EFFECTS OF EARLY EXPERIENCES ON PSYCHOLOGICAL DEVELOPMENT** 156
Thomas G. O'Connor

6 **EMOTIONAL SECURITY THEORY AND DEVELOPMENTAL PSYCHOPATHOLOGY** 199
Patrick T. Davies, Meredith J. Martin, and Melissa L. Sturge-Apple

7 **EMOTION AND THE DEVELOPMENT OF PSYCHOPATHOLOGY** 265
Pamela M. Cole

8 **ATTACHMENT AND DEVELOPMENTAL PSYCHOPATHOLOGY** 325
R. M. Pasco Fearon, Ashley M. Groh, Marian J. Bakermans-Kranenburg, Marinus H. van IJzendoorn, and Glenn I. Roisman

9 **AUTONOMY AND AUTONOMY DISTURBANCES IN SELF-DEVELOPMENT AND PSYCHOPATHOLOGY: RESEARCH ON MOTIVATION, ATTACHMENT, AND CLINICAL PROCESS** 385
Richard M. Ryan, Edward L. Deci, and Maarten Vansteenkiste

10 **ROOTS OF TYPICAL CONSCIOUSNESS: IMPLICATIONS FOR DEVELOPMENTAL PSYCHOPATHOLOGY** 439
Philippe Rochat

11 **I-SELF AND ME-SELF PROCESSES AFFECTING DEVELOPMENTAL PSYCHOPATHOLOGY AND MENTAL HEALTH** 470
Susan Harter

12 **PEER RELATIONS AND DEVELOPMENTAL PSYCHOPATHOLOGY** 527
Mitchell J. Prinstein and Matteo Giletta

13 **FAMILY SYSTEMS FROM A DEVELOPMENTAL PSYCHOPATHOLOGY PERSPECTIVE** 580
Patricia K. Kerig

14 **ADOLESCENT/YOUNG ADULT ROMANTIC RELATIONSHIPS AND PSYCHOPATHOLOGY** 631
Joanne Davila, Deborah M. Capaldi, and Annette M. La Greca

15 **WHAT CAN DYNAMIC SYSTEMS MODELS OF DEVELOPMENT OFFER TO THE STUDY OF DEVELOPMENTAL PSYCHOPATHOLOGY?** 665
Michael F. Mascolo, Paul Van Geert, Henderien Steenbeek, and Kurt W. Fischer

16 **A SURVEY OF DYNAMIC SYSTEMS METHODS FOR DEVELOPMENTAL PSYCHOPATHOLOGY** 717
Isabela Granic, Tom Hollenstein, and Anna Lichtwarck-Aschoff

17 **MISSING DATA** 760
Todd D. Little, Kyle M. Lang, Wei Wu, and Mijke Rhemtulla

18 **PERSON-ORIENTED APPROACHES** 797
G. Anne Bogat, Alexander von Eye, and Lars R. Bergman

19 **PERSON-SPECIFIC APPROACHES TO THE MODELING OF INTRAINDIVIDUAL VARIATION IN DEVELOPMENTAL PSYCHOPATHOLOGY** 846
Michael J. Rovine and Peter C. M. Molenaar

20 **CONFIGURAL FREQUENCY ANALYSIS FOR RESEARCH ON DEVELOPMENTAL PROCESSES** 866
Alexander von Eye and Eun-Young Mun

21 **MODERATION AND MEDIATION IN INTERINDIVIDUAL LONGITUDINAL ANALYSIS** 922
Jennifer L. Krull, JeeWon Cheong, Matthew S. Fritz, and David P. MacKinnon

22 **LATENT GROWTH MODELING AND DEVELOPMENTAL PSYCHOPATHOLOGY** 986
Jungmeen Kim-Spoon and Kevin J. Grimm

23 **INTEGRATIVE DATA ANALYSIS FOR RESEARCH IN DEVELOPMENTAL PSYCHOPATHOLOGY** 1042
Eun-Young Mun, Yang Jiao, and Minge Xie

Author Index 1089

Subject Index 1125

Preface to Developmental Psychopathology, Third Edition

A decade has passed since the second edition of *Developmental Psychopathology* was published. The two prior editions (Cicchetti & Cohen, 1995, 2006) have been very influential in the growth of the field of developmental psychopathology. The volumes have been highly cited in the literature and have served as an important resource for developmental scientists and prevention and intervention researchers alike. In the present third edition, we have expanded from the three volumes contained in the second edition to four volumes. The increased number of volumes in this current edition reflects the continued knowledge gains that have occurred in the field over the past decade.

A not insignificant contributor to this growth can be found in the very principles of the discipline (Cicchetti, 1984, 1990, 1993; Cicchetti & Sroufe, 2000; Cicchetti & Toth, 1991, 2009; Rutter & Sroufe, 2000; Sroufe & Rutter, 1984). Theorists, researchers, and prevention scientists in the field of developmental psychopathology adhere to a life span framework to elucidate the numerous processes and mechanisms that can contribute to the development of mental disorders in high-risk individuals as well as those operative in individuals who already have manifested psychological disturbances or who have averted such disorders despite their high-risk status (Cicchetti, 1993; Masten, 2014; Rutter, 1986, 1987, 2012). Not only is knowledge of normal genetic, neurobiological, physiological, hormonal, psychological, and social processes very helpful for understanding, preventing, and treating psychopathology, but also deviations from and distortions of normal development that are seen in pathological processes indicate in innovative ways how normal development may be better investigated and understood. Similarly, information obtained from investigations of experiments of nature, high-risk conditions, and psychopathology can augment the comprehension of normal development (Cicchetti, 1984, 1990, 1993; Rutter, 1986; Rutter & Garmezy, 1983; Sroufe, 1990; Weiss, 1969).

Another factor that has expedited growth within the field of developmental psychopathology has been its ability to incorporate knowledge from diverse disciplines and to encourage interdisciplinary and translational research (Cicchetti & Gunnar, 2009; Cicchetti & Toth, 2006). In keeping with its integrative focus, contributions to developmental psychopathology have come from many disciplines of the biological and social sciences. A wide array of content areas, scientific disciplines, and methodologies has been germane. Risk and protective factors and processes have been identified and validated at multiple levels of analysis and in multiple domains.

The increased emphasis on a multilevel, dynamic systems approach to psychopathology and resilience, the increased attention paid to gene–environment interplay in the development of psychopathology and resilience, and the application of a multiple levels of analysis developmental perspective to mental illnesses that have traditionally been examined nondevelopmentally (e.g., bipolar disorder, schizophrenia, and the personality disorders) not only have contributed to a deeper understanding of the dysfunctions but also have educated the public about the causes and consequences of mental disorder (see Cicchetti & Cannon, 1999; Cicchetti & Crick, 2009a, 2009b; Miklowitz & Cicchetti, 2006, 2010; Tackett & Sharp, 2014).

Advances in genomics, GxE interactions, and epigenetics; growth in our understanding of neurobiology, neural plasticity, and resilience; and progress in the development of methodological and technological tools, including brain imaging, neural circuitry, hormone assays, immunology, social and environmental influences on brain development, and statistical analysis of developmental change, pave the way for interdisciplinary and for multiple levels of analysis research programs that will significantly increase the knowledge base of the development and course of maladaptation, psychopathology, and resilience. Moreover, randomized control prevention and intervention trials are being conducted based on theoretical models and efforts to elucidate the mechanisms and processes contributing to developmental change at both the biological and psychological levels (Belsky & van IJzendoorn, 2015; Cicchetti & Gunnar, 2008).

Despite the significant advances that have occurred in the field of developmental psychopathology, much important work lies ahead. Undoubtedly these future developments will build on the venerable contributions of the past; however, as work in the field becomes increasingly interdisciplinary, multilevel, and technologically sophisticated, it is essential that even more emphasis be directed toward the process of development (Harter, 2006; Sroufe, 2007, 2013). It is not only genes and environments but also the cumulative developmental history of the individual that influences how future development will unfold (Sroufe, 2007, 2013).

Developmental psychopathologists have incorporated concepts and methods derived from other disciplinary endeavors that are too often isolated from each other, thereby generating advances in knowledge that might have been missed in the absence of cross-disciplinary dialogue. The continuation and elaboration of the mutually enriching interchanges that have occurred within and across disciplines interested in normal and abnormal development not only will enhance the science of developmental psychopathology but also will increase the benefits to be derived for individuals with high-risk conditions or mental disorders, families, and society as a whole.

Dante Cicchetti, Ph.D.
Minneapolis, MN
January 2015

REFERENCES

Belsky, J., & van IJzendoorn, M. (2015). What works for whom? Genetic moderation of intervention efficacy. [Special Section]. *Development and Psychopathology*, *27*, 1–6.

Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development*, *55*(1), 1–7.

Cicchetti, D. (1990). A historical perspective on the discipline of developmental psychopathology. In J. Rolf, A. Masten, D. Cicchetti, K. Nuechterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 2–28). New York, NY: Cambridge University Press.

Cicchetti, D. (1993). Developmental psychopathology: Reactions, reflections, projections. *Developmental Review*, *13*, 471–502.

Cicchetti, D., & Cannon, T. (1999). Neurodevelopmental processes in the ontogenesis and epigenesis of psychopathology. *Development and Psychopathology*, *11*, 375–393.

Cicchetti, D., & Cohen, D. (Eds.). (1995). *Developmental psychopathology* (Vols. *1–2*). New York, NY: Wiley.

Cicchetti, D., & Cohen, D. (Eds.). (2006). *Developmental psychopathology* (2nd ed., Vols. *1–3*). New York, NY: Wiley.

Cicchetti, D., & Crick, N. R. (Eds.) (2009a). Precursors of and diverse pathways to personality disorder in children and adolescents. [Special Issue, Part 1]. *Development and Psychopathology*, *21*(3), 683–1030.

Cicchetti, D., & Crick, N. R. (Eds.). (2009b). Precursors of and diverse pathways to personality disorder in children and adolescents. [Special Issue, Part 2]. *Development and Psychopathology*, *21*(4), 1031–1381.

Cicchetti, D., & Gunnar, M. R. (2008). Integrating biological processes into the design and evaluation of preventive interventions. *Development and Psychopathology*, *20*, 737–743.

Cicchetti, D., & Gunnar, M. R. (Eds.). (2009). Meeting the challenge of translational research in child psychology: Minnesota symposia on child psychology (Vol. 35). New York, NY: Wiley.

Cicchetti, D., & Sroufe, L. A. (2000). The past as prologue to the future: The times they've been a changin'. *Development and Psychopathology*, *12*, 255–264.

Cicchetti, D., & Toth, S. L. (1991). The making of a developmental psychopathologist. In J. Cantor, C. Spiker, & L. Lipsitt (Eds.), *Child behavior and development: Training for diversity* (pp. 34–72). Norwood, NJ: Ablex.

Cicchetti, D., & Toth, S. L. (Eds.). (2006). Translational research in developmental psychopathology. [Special Issue]. *Development and Psychopathology*, *18*(3), 619–933.

Cicchetti, D., & Toth, S. L. (2009). The past achievements and future promises of developmental psychopathology: The coming of age of a discipline. *Journal of Child Psychology and Psychiatry*, *50*, 16–25.

Harter, S. (2006). Self-processes and developmental psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology* (2nd ed., 370–418). New York, NY: Wiley.

Masten, A. S. (2014). *Ordinary magic: Resilience in development*. New York, NY: Guilford Publications, Inc.

Miklowitz, D. J., & Cicchetti, D. (2006). Toward a life span developmental psychopathology perspective on bipolar disorder. *Development and Psychopathology*, *18*, 935–938.

Miklowitz, D. J., & Cicchetti, D. (Eds.). (2010). *Bipolar disorder: A developmental psychopathology approach*. New York, NY: Guilford.

Rutter, M. (1986). Child psychiatry: The interface between clinical and developmental research. *Psychological Medicine*, *16*, 151–169.

Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry*, *57*, 316–331.

Rutter, M. (2012). Resilience as a dynamic concept. *Development and Psychopathology*, *24*, 335–344.

Rutter, M., & Garmezy, N. (1983). Developmental psychopathology. In E. M. Hetherington (Ed.), *Handbook of child psychology* (pp. 774–911). New York, NY: Wiley.

Rutter, M., & Sroufe, L. A. (2000). Developmental psychopathology: Concepts and challenges. *Development and Psychopathology*, *12*, 265–296.

Sroufe, L. A. (1990). Considering normal and abnormal together: The essence of developmental psychopathology. *Development and Psychopathology*, *2*, 335–347.

Sroufe, L. A. (2007). The place of development in developmental psychopathology. In A. Masten (Ed.), *Multilevel dynamics in developmental psychopathology pathways to the future: The Minnesota symposia on child psychology* (pp. 285–299). Mahwah, NJ: Erlbaum.

Sroufe, L. A. (2013). The promise of developmental psychopathology. *Development and Psychopathology*, *25*, 1215–1224.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, *55*, 17–29.

Tackett, J. L., & Sharp, C. (2014). A developmental psychopathology perspective on personality disorder. [Special Issue]. *Journal of Personality Disorders*, *28*, 1–179.

Weiss, P. (1969). *Principles of development*. New York, NY: Hafner.

Contributors

Thomas M. Achenbach, PhD
University of Vermont
Burlington, VT

Adrian Angold, MRCPsych
Duke University Medical Center
Durham, North Carolina

Marian J. Bakermans-Kranenburg, PhD
Leiden University
Leiden, Netherlands

Lars R. Bergman, PhD
Stockholm University
Stockholm, Sweden

G. Anne Bogat, PhD
Michigan State University
East Lansing, Michigan

Margaret J. Briggs-Gowan, PhD
University of Connecticut
Farmington, Connecticut

Deborah M. Capaldi, PhD
Oregon Social Learning Center
Eugene, Oregon

Alice S. Carter, PhD
University of Massachusetts
Boston, Massachusetts

JeeWon Cheong, PhD
University of Florida
Gainesville, Florida

Dante Cicchetti, PhD
Institute of Child Development
University of Minnesota
Minneapolis, Minnesota

Pamela M. Cole, PhD
Pennsylvania State University
University Park, Pennsylvania

E. Jane Costello, PhD
Duke University Medical Center
Durham, North Carolina

Patrick T. Davies, PhD
University of Rochester
Rochester, New York

Joanne Davila, PhD
Stony Brook University
Stony Brook, New York

Edward L. Deci, PhD
University of Rochester
Rochester, New York

R. M. Pasco Fearon, PhD, DClinPsy
University College London
London, United Kingdom

Kurt W. Fischer, PhD
Harvard University
Cambridge, Massachusetts

Matthew S. Fritz, PhD
University of Nebraska
Lincoln, Nebraska

Matteo Giletta, PhD
Tilburg University
Tilburg, Netherlands

Leandra Godoy, PhD
Children's National Medical Center
Washington, District of Columbia

Isabela Granic, PhD
Radboud University Nijmegen
Nijmegen, Netherlands

Kevin J. Grimm, PhD
Arizona State University
Tempe, Arizona

Ashley M. Groh, PhD
University of Missouri
Columbia, Missouri

Susan Harter, PhD
University of Denver
Denver, Colorado

Amy Heberle, MS
University of Massachusetts
Boston, Massachusetts

Tom Hollenstein, PhD
Queen's University
Kingston, Canada

Yang Jiao, MS
Rutgers, the State University of New Jersey
Piscataway, New Jersey

Patricia K. Kerig, PhD
University of Utah
Salt Lake City, Utah

Jungmeen Kim-Spoon, PhD
Virginia Tech
Blacksburg, Virginia

Jennifer L. Krull, PhD
University of California
Los Angeles, California

Annette M. La Greca, PhD, ABPP
University of Miami
Coral Gables, Florida

Kyle M. Lang, PhD
Texas Tech University
Lubbock, Texas

Anna Lichtwarck-Aschoff, PhD
Radboud University Nijmegen
Nijmegen, Netherlands

Todd D. Little, PhD
Texas Tech University
Lubbock, Texas

David P. MacKinnon, PhD
Arizona State University
Tempe, Arizona

Meredith J. Martin, PhD
University of Rochester
Rochester, New York

Michael F. Mascolo, PhD
Merrimack College
North Andover, Massachusetts

Peter C. M. Molenaar, PhD
Pennsylvania State University
University Park, Pennsylvania

Eun-Young Mun, PhD
Rutgers, the State University of New Jersey
Piscataway, New Jersey

Thomas G. O'Connor, PhD
University of Rochester Medical Center
Rochester, New York

Mitchell J. Prinstein, PhD, ABPP
University of North Carolina
Chapel Hill, North Carolina

Leslie A. Rescorla, PhD
Bryn Mawr College
Bryn Mawr, Pennsylvania

Mijke Rhemtulla, PhD
University of Amsterdam
Amsterdam, Netherlands

Philippe Rochat, PhD
Emory University
Atlanta, Georgia

Glenn I. Roisman, PhD
University of Minnesota
Minneapolis, Minnesota

Michael J. Rovine, PhD
Pennsylvania State University
University Park, Pennsyvania

Michael L. Rutter, MD, FRS, FRC Psych., FBA, FAC Med Sci
King's College London
London, United Kingdom

Richard M. Ryan, PhD
Australian Catholic University
Strathfield, Australia

Henderien Steenbeek, PhD
University of Groningen
Groningen, Netherlands

Melissa L. Sturge-Apple, PhD
University of Rochester
Rochester, New York

Anita Thapar, MD, PhD
Cardiff University
Cardiff, United Kingdom

Paul van Geert, PhD
University of Groningen
Groningen, Netherlands

Marinus H. van IJzendoorn, PhD
Leiden University
Leiden, Netherlands

Maarten Vansteenkiste, PhD
University of Ghent
Ghent, Belgium

Alexander von Eye, PhD
Michigan State University
East Lansing, Michigan

Wei Wu, PhD
University of Kansas
Lawrence, Kansas

Minge Xie, PhD
Rutgers, the State University of New Jersey
Piscataway, New Jersey

CHAPTER 1

Assessment of Psychopathology in Young Children

MARGARET J. BRIGGS-GOWAN, LEANDRA GODOY, AMY HEBERLE, and ALICE S. CARTER

INTRODUCTION 1
EARLY PROBLEMS MATTER 2
Progress in Psychiatric Diagnosis in Young Children 3
IMPORTANT CONSIDERATIONS IN YOUNG CHILD ASSESSMENT 4
Reliance on Caregivers for Information 4
Sensitivity to Contextual Influences, Including Caregiving Contexts 6
DOMAINS OF DEVELOPMENT 9
SELECTING AN ASSESSMENT APPROACH AND TOOL 11
Types of Tools 11
Understanding Psychometric Properties 12
Reliability 12
Validity 13
Validity of Classification 14
Normatization 15
Cultural Validity and Cultural Norms 16
Knowing What Problems Are Really Being Assessed 16
Response Formats 17
Summary 17
ASSESSMENT TOOLS 17
Screening Methods 18
Screening Methods Characteristics of Screening Tools 19
Selected Screening Tools 20
Comprehensive Dimensional Tools for Assessing Social-Emotional/Behavioral Problems 23
Selected Dimensional Checklists 24
Variation in Emphasis of the Domains That Are Assessed 29
Diagnostic Approaches 29
Selected Diagnostic Interviews 30
Psychometric Properties of Diagnostic Interviews 32
Observational Assessment 35
Assessing Impairment 35
CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH 37
REFERENCES 38

INTRODUCTION

The past 20 years have witnessed a sea change for *young children*'s mental health. It is now recognized that early childhood (0–5 years) is a crucial period for the development of self-regulation, a critical set of competencies that have implications for adaptive functioning in school and through the life span. Early childhood is also recognized as a time when psychopathology may begin to emerge and disrupt young children's developmental progress. In addition, enormous progress has been made in demonstrating that, when psychiatric disorders are defined in a manner that is developmentally meaningful, even very young children suffer from psychiatric disorders that are valid, impairing, and clinically very similar to those experienced by older children (Egger & Angold, 2006; Egger et al., 2006). Indeed, recent research has indicated that psychiatric disorders are just as prevalent in early childhood as they are in school-age children (Egger & Angold, 2006). Moreover, when young children manifest psychopathology that is impairing, it is often persistent and predicts later difficulties once they become of school age. Equally important, there is increasing awareness that these problems can interfere with learning within early childhood and may set in motion a developmental cascade that likely predicts challenges to lifespan functioning in multiple domains. Research focused on specific disorders has driven discovery of neurobiologic substrates, which has further validated the relevance and reality of early life psychopathology (Luby, Belden, Pautsch, Si, & Spitznagel, 2009; Luby, Si, Belden, Tandon, & Spitznagel, 2009; Stalets & Luby, 2006). These advances in our understanding of young child psychopathology are paralleled by, and one might argue largely driven by, an explosion in reliable, valid, developmentally sensitive measures for assessing a full range of self-regulation, social-emotional development in young

[1]Color versions of Figure 1.1 are available at http://onlinelibrary.wiley.com/book/10.1002/9781119125556

children. Specifically, over the past 15-plus years, a number of instruments have been developed to assess parent and other caregiver appraisals of social-emotional functioning utilizing both questionnaire and interview methods. There have also been advances in observational tools to assess clinically significant emotional and behavior problems. With greater acceptance and building on advances in measurement, we are poised to evaluate the benefits of a broad range of prevention and intervention efforts and see increasing discovery of biological and environmental influences on young children's mental health.

As the field presses forward to address the mental health needs of young children both efficiently and effectively, success will be optimized by a well-informed approach to assessment that (1) acknowledges *contextual factors,* including the caregiving environments at home and in other settings, such as child care and early education environments, caregiver influences on social-emotional functioning and assessment, recent changes in family structure or contextual stressors, and sociocultural factors; (2) is framed within the context of a child's *functioning in other developmental domains,* such as language, cognition, adaptive functioning, health, and sensory; (3) is tailored to the *goals and purposes of the assessment and evaluation setting* (e.g., pediatric clinic, day care center, mental health clinic, or private practice); (4) utilizes *reliable, valid, developmentally sensitive tools*; and (5) employs an approach to *interpretation* that views the whole child in relation to contextual and developmental factors and evaluates his or her capacities and participation in developmentally appropriate activities and settings (i.e., impairment).

A primary goal of this chapter is to help clinicians and researchers determine the most suitable measures to use from a wide array of parent and other caregiver report, observational, and direct assessment measures that are now available. Rather than trying to offer an exhaustive list of all existing measures of social-emotional functioning and psychopathology appropriate for young children, we highlight some of the most widely employed and promising tools and approaches, including those that reflect advances in *screening*, comprehensive *dimensional* parent- and other caregiver-report instruments, and diagnostic approaches to young child evaluation. These assessment tools can be categorized as follows: (1) parent and other caregiver report instruments that focus on general problem behaviors; (2) parent and other caregiver report instruments that focus on specific problem areas or disorders (e.g., anxiety, disruptive behavior); (3) parent and other caregiver report instruments designed to assess both problem behaviors and competencies; (4) comprehensive diagnostic interviews for parents of young children; and (5) observational tools and methods. Within the first three categories, measures can be further divided according to whether they are brief tools appropriate for *screening* or longer *checklist* tools or *diagnostic interviews* that provide more detailed information. Finally, we will close the chapter with a discussion of ongoing challenges, future directions, and opportunities in research on and clinical applications with assessment of young child psychopathology. Although many researchers and clinicians continue to express discomfort about pathologizing, or labeling, young children, our focus is on assessment tools that enhance the recognition and detection of early emerging psychopathology to address mental health needs in an effort to minimize adverse developmental cascades. Moreover, we argue optimistically that by labeling systematic behavioral patterns observed within young children (rather than labeling individual children) we create the potential to develop and disseminate guidance regarding appropriate contextual supports and specific behavioral interventions that are tailored to the needs of children with different behavioral profiles; these early prevention and targeted interventions can be designed to support family beliefs, values, and goals and children's developmental progress while minimizing child and family distress.

EARLY PROBLEMS MATTER

There is now a consensus among child clinicians that children as young as 2 years of age can suffer from significant social-emotional and behavior problems, or psychopathology. Prevalence estimates of clinically significant problems in nonreferred samples have ranged considerably, from as low as 7% to as high as 26%, depending on whether problems are defined in terms of meeting criteria for psychiatric diagnosis or by exceeding a clinical cutoff on a checklist measure (Briggs-Gowan, Carter, Skuban, & Horwitz, 2001; Egger & Angold, 2006; Gleason et al., 2011; Karabekiroglu et al., 2013; Keenan et al., 1997; Lavigne, Lebailly, Hopkins, Gouze, & Binns, 2009; Wichstrom et al., 2012). Rates also tend to be higher among young children exposed to poverty and other psychosocial risk factors (McCue Horwitz et al., 2012; Qi & Kaiser, 2003; Weitzman, Edmonds, Davagnino, & Briggs-Gowan, 2014). Early social-emotional and behavioral problems are linked with impairment in child and family functioning as well as increased parenting stress and worry (Briggs-Gowan & Carter, 2008a; Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006; Briggs-Gowan et al., 2001; Egger & Angold, 2006; Fuchs, Klein, Otto, & von Klitzing, 2013; Keenan et al., 2007; Lavigne et al., 1996; Luby, Belden, Pautsch, et al., 2009). Moreover, social-emotional/behavior

problems in young children have been associated with concomitant delays in child social-emotional competence (Briggs-Gowan & Carter, 2008a; Briggs-Gowan et al., 2001) and shown to predict poorer social competence in elementary school (Briggs-Gowan, Carter, & Ford, 2011). Intervening in preschool to address both social emotional problems and competencies is associated with greater improvements in both areas, including on experimental tasks of emotion knowledge and social problem-solving strategies (Ştefan & Miclea, 2013). Furthermore, contrasting with the historical belief that young children's difficult behavior is just a phase (Keenan & Wakschlag, 2000), there is consistent evidence that for some children these early emergent social-emotional and behavioral problems are persistent and predict poorer functioning at later ages (Briggs-Gowan & Carter, 2008b; Briggs-Gowan et al., 2006; Kim-Cohen et al., 2005; Lavigne et al., 1998; Mathiesen & Sanson, 2000; O'Neill, Schneiderman, Rajendran, Marks, & Halperin, 2014; Shaw, Lacourse, & Nagin, 2005; Speltz, McClellan, DeKlyen, & Jones, 1999; Spence, Najman, Bor, O'Callaghan, & Williams, 2002; Stalets & Luby, 2006; Wakschlag, Briggs-Gowan, et al., 2008). Notably, persistence has been documented for a wide range of problems, including anxiety, depression, attention-deficit hyperactivity, and disruptive behaviors. Thus, consistent and convincing evidence indicates that young children can and do suffer from a wide spectrum of social-emotional and behavioral problems that are often persistent—the presence of impairment further underscores the importance of early identification and prevention in this developmental period.

Progress in Psychiatric Diagnosis in Young Children

Empirical and conceptual work in the areas of posttraumatic stress disorder (PTSD), depression, and disruptive behavior disorders illustrates the role that developmental factors can play in how psychopathology manifests and how several groups have endeavored to establish the relevance of these psychopathologies in young children.

PTSD

Considerable work by Michael Scheeringa and colleagues documents that children as young as 9 months of age can and do suffer from PTSD (Scheeringa, 2007, 2008; Scheeringa, Myers, Putnam, & Zeanah, 2012; Scheeringa, Peebles, Cook, & Zeanah, 2001). This work highlights the importance of considering the developmental capacities of young children when determining the appropriateness of criteria employed for older children and illustrates the utility of adopting a multi-informant, multimethod approach to assessment (Hunsley & Mash, 2007). Scheeringa et al.'s work in this area has driven important recognition of developmental factors that affect how PTSD presents in young children. For example, many avoidance and numbing symptoms are either developmentally implausible (e.g., sense of a foreshortened future) or internal in quality (e.g., avoidance of internal thoughts, feelings, or reminders of the event), making them very difficult to identify in young children who have limited verbal skills (Scheeringa, 2008). Scheeringa also noted that these types of symptoms may manifest differently in young children. For example, "markedly diminished interest in significant activities" is often observed as constriction of play, and "feeling of detachment or estrangement from others" is often observed as social withdrawal.

Scheeringa and colleagues further documented the central role that parental reactions play in the emergence, promotion, and maintenance of symptoms in young children (Scheeringa & Zeanah, 2001), highlighting the importance of assessing young children's possible PTSD symptoms in the context of parent– or caregiver–child relationships.

Depression

Luby and colleagues' work has established the presence of early manifestations of clinically significant signs and symptoms of depressive disorders in young children (Luby, Belden, Pausch, et al., 2009; Luby & Navsaria, 2010; Luby, Si, et al., 2009; Stalets & Luby, 2006). Their research addresses the complicated developmental question of whether young children's emotional repertoire is itself sufficiently differentiated to encompass true depressive or elated affect and how to differentiate atypical from normative developmental manifestations. They have further noted that greater variability in young children's mood states calls into question the relevance of duration criteria employed by diagnostic systems developed for older children (Gaffrey, Belden, & Luby, 2011). Luby and colleagues utilized a multimethod, multi-informant approach that included (1) parent reports on both dimensional ratings scales and in an age-appropriate *diagnostic interview*; (2) comprehensive observation of the young child's affective range via a laboratory based temperament assessment, thematic play, and parent–child interaction across structured and unstructured conditions; (3) a developmentally sensitive direct interview for preschoolers; (4) cognitive assessment of the child; and (5) neurocognitive assessment of the child. By employing these techniques, they were able to identify a group of young preschool-age children who met modified depression criteria developed for young children.

Further, Luby and colleagues' empirical data have provided crucial insights into how depression manifests in young children (Luby, Belden, Sullivan, et al., 2009;

Luby, Belden, Pausch, et al., 2009; Luby, Si, et al., 2009). For example, their work has shown that withdrawal and vegetative symptoms are less consistently evident in young children with Major Depressive Disorder relative to older children. They have also shown that sadness, irritability, and thoughts of death, while evident in young children with depression, also are often present in children with anxiety disorders and attention-deficit/hyperactivity disorder (ADHD). In contrast, anhedonia, feelings of guilt, and psychomotor agitation appear to be fairly characteristic of young children with depression. Changes in activity, appetite, and sleep also have been noted. Anhedonia appears to be a very specific marker of depression in young children: it is commonly present in young children with depression and rarely seen in other psychiatric groups or typically developing children (Luby et al., 2002). A subtype of preschool depression characterized by anhedonia also appears to be particularly severe (Luby, Belden, Pausch, et al., 2009). The work of Luby and her colleagues is notable for inclusion of biological correlates, such as cortisol reactivity and addressing family genetic risk to document the *validity* of the revised young child criteria (Luby, Belden, Pausch, et al., 2009; Luby, Si, et al., 2009; Stalets & Luby, 2006).

Disruptive Behavior Disorders

The study of disruptive behavior disorders highlights the complexity of distinguishing normative from clinically problematic behavior during early childhood when some "problem" behaviors are normative (Wakschlag et al., 2007; Wakschlag, Tolan, & Leventhal, 2010). Historically, concern was raised about whether disruptive behavior problems could be reliably assessed during this period (Campbell, 1990) and whether it may be premature to diagnose children whose challenging behaviors might be transient and diminish over time. At the same time, disruptive behavior is the most common reason for referral of young children to mental health clinics (Keenan & Wakschlag, 2000; Thomas & Guskin, 2001). Wakschlag and her colleagues have pressed for the refinement of diagnostic approaches in a manner that describes early manifestations of disruptive behavior in a developmentally appropriate and meaningful fashion and distinguishes between clinically significant disruptive behaviors and expectable variation in children's capacity to regulate emotions and behavior as they consolidate their self-regulatory skills (Gray & Wakschlag, in press; Wakschlag et al., 2007). This work and the work of others has demonstrated that disruptive behavior disorders can be reliably and validly diagnosed and are associated with marked impairment in preschool children (Egger & Angold, 2006; Egger et al., 2006; Keenan & Wakschlag, 2000, 2002; Keenan et al., 2007; Wakschlag et al., 2007). Through a longitudinal study, this group further demonstrated persistence over time in early disruptive behavior disorders. In one study, 55% of those with disorders at baseline continued to meet criteria for disorder one year later (Wakschlag, Briggs-Gowan, et al., 2008). These rates are strikingly similar to levels of persistence observed in school-age children (Briggs-Gowan et al., 2003).

Summary

As our understanding of specific early emerging disorders evolves, it is quite likely that additional diagnosis-specific instruments will be developed, allowing clinicians and researchers to elicit information about behaviors that assist in making differential diagnoses (i.e., are specific to diagnostic conditions). Clearly, multimethod, multi-informant assessment is integral to understanding young children's development. Moreover, observations of the parent–child or caregiver–child interactions across several contexts must be considered a critical component of any young child assessment of social-emotional problems or psychopathology.

IMPORTANT CONSIDERATIONS IN YOUNG CHILD ASSESSMENT

Though challenges exist when conducting any mental health assessment, several are unique to or require greater consideration when assessing young children, including the following: (1) young children's *limited communication skills*, which results in greater reliance on caregivers for information about the child's functioning; (2) young children's *sensitivity to contextual influences* on their functioning and the resulting importance of assessing context when working with this population; and (3) the influence of *sociocultural factors*, which affect the meaning of the child's behaviors to caregivers and other family members, the way that caregivers report on the child's behavior, caregivers' level of worry about the child overall and in relation to specific behaviors, caregivers' decisions to seek or not seek services, and the assessor's interpretation of findings. Understanding these challenges can enhance the evaluation process.

Reliance on Caregivers for Information

The fact that infants and young children have no or limited verbal abilities and metacognitive capacities makes it

difficult or impossible to directly collect information from them about their thoughts and feelings about presenting complaints, historical experiences and behaviors, and contextual events. Thus, in contrast to older children, youth, and adults, caregivers play a central role in providing information about the young child's behaviors across multiple settings and contexts, including crucial insight into when behaviors may have emerged and changed over time and contextual factors that may have exacerbated or attenuated symptoms. Collecting information from caregivers brings unique considerations to the assessment process when evaluating questionnaire and interview assessment tools.

First, it is important to establish the best informant in the child's life to best speak for him or her about a given topic. This is usually determined by asking who has a parental or caregiving role for the child in relation to the area or domain of inquiry, that is, the person who assumes responsibility for meeting his or her physical and emotional needs. In this chapter, the term *parent* refers to biological, adoptive, or foster parents or guardians or extended family members who care for the child on a regular basis. The term *caregiver* encompasses parents as well as individuals who routinely spend enough time with the child to be knowledgeable about multiple aspects of the child's social-emotional functioning and day-to-day behavior. Thus, caregiver may refer to extended family members and child-care or day-care providers who have cared for the child for at least one month and who care for the child on a regular basis. (Note, however, that some measures may list different criteria for classifying someone as a suitable informant on the child's functioning.) Typically, the person who brings a child to his or her evaluation appointment and who consents to the evaluation is a parental figure and should be included in the evaluation process; however, it is also important to assess others who may function as caregivers—particularly when a child experiences care in multiple contexts. These additional individuals should also be included as informants. Moreover, it is important to invite relevant caregivers to participate in any child evaluation, as young children's behavior is more variable across contexts (Clark, Tluczek, & Gallagher, 2004; Tronick, 1989).

An evaluation of the child's larger family context facilitates the process of identifying caregivers. It also provides important cues regarding factors such as recent stressors or strained dyadic and triadic family relationships that are currently influencing the child's functioning or that may have had a role in shaping the child's functioning over time (Carlson, 1990; Hayden et al., 1998). Information about the larger family context also enhances the assessor's understanding of a given caregiver's perspective on the child (e.g., how long the caregiver has known the child, who else is present in typical interactions between the caregiver and the child, who supports or influences the caregiver's beliefs about the child), potentially illuminating sources of bias and avenues for intervention. For example, if in the process of assessing the family context it comes to light that a young mother has received criticism about her child's behavior and her own parenting from her own mother, who cares for the child on a regular basis, this information would point to the need for further assessment of the mother–grandmother relationship. This evaluation could focus on the potential utility of intervention to improve communication between and address inconsistencies in the disciplinary styles, beliefs and expectations of the two caregivers. It is important to note that in using the word *family* we refer to any person, whether biologically related to the child or not, who is considered by the child or other adults close to the child to be a family member. The assessor should consider the total makeup of the family group (e.g., how many members), who is considered a family member, how family members define relationships with one another, where family members live in relation to one another, and what roles family members hold from the perspective of the target child (Carlson, 1990). In addition to evaluating the current family context, a history of the family makeup should be obtained, including ruptures in family relationships, new additions to the family, and changes in the way family members are distributed across households.

In the process of assessing the family and caregiving context and history, assessors must be alert to their own biases about what constitutes a family and how families function. Accurate assessment of family functioning will be facilitated by knowledge of cultural norms regarding family relationships in the groups to which a child belongs. In contrast to the popular notion of the two-parent household and caregiving system, many children are cared for in part or whole by relatives such as aunts, uncles, and grandparents (Dressler, 1985; Wilson & Tolson, 1990). In fact, as of 2012, 10% of all American children reside with a grandparent (with or without a parent also in the home); this number is even greater for Black (14%), Latino (12%), and Asian (14%) children (Ellis & Simmons, 2014). Twenty-six percent of children under the age of 5 whose mothers are employed have a grandparent or other relative as their primary child-care provider (Laughlin, 2013). Many more children may have close but informal caregiving relationships with relatives other than their parents.

Thus, many children have nonparental caregivers who should be included in the evaluation process to elicit an accurate assessment of the child's functioning. The utility of including multiple caregivers' reports in the evaluation process is further discussed in the next section.

Sensitivity to Contextual Influences, Including Caregiving Contexts

Young children's behavior is highly sensitive to contextual and relational influences (Clark et al., 2004; Dirks, De Los Reyes, Briggs-Gowan, Cella, & Wakschlag, 2012; Gray et al., 2012; Tronick, 1989), and it is therefore critical to include an assessment of relevant contextual factors in any evaluation involving a young child. When working with young children, social-emotional and cognitive functioning and impairment should always be assessed within the context of caregiving relationships and settings, with assessors incorporating what they are able to observe and measure about the various aspects of caregiver functioning and setting characteristics into their interpretation of findings from assessment instruments designed to measure an individual's functioning. In many cases, the family history will reveal recent or chronic stressors to which the child may be responding. Where major events have occurred in the child's history—whether these are identified as stressors by the reporter or not—it may be useful to explicitly inquire about the child's functioning before and after the event as well as changes since the event. As an example, moving houses is a stressful—and common—transition for young children (Stoneman, Brody, Churchill, & Winn, 1999). In some cases, the family history may reveal a recent change in the child's caregiving relationships, as in the case of a child who has recently transitioned to the care of a new foster parent, or in the case of a child whose parents have recently moved the family into their own apartment after having lived with the child's grandparents for the majority of her life. In cases such as these, repeat assessment after one or two months may be warranted to assess the child's functioning after a period of adjustment to the new caregiving environment.

Understanding the makeup of a particular child's family is an essential assessment task because young children's development, behavior, and functioning are embedded within their caregiving relationships. While it is also true that older children are influenced by their caregiving contexts, young children may be especially sensitive to contextual influences due to their dependence on others for their basic needs, often limited exposure to outside institutions such as schools, limited relationships with peers, and their unlikelihood of having developed independent emotion-regulation or coping strategies that they can utilize in the face of environmental stressors (Compas, 1987).

Caregivers provide structure to the physical, emotional, and behavioral aspects of the child's environment; for example, infants learn to regulate their own affect through exchanges with their caregivers and practice with sensitive, engaged caregivers to repair dysynchronous interactions, to reengage after a rupture, to recover behaviorally and physiologically after exposure to a stressor, and to convey information through affective expression (Bridgett, Burt, Laake, & Oddi, 2013; Haley & Stansbury, 2003; Kogan & Carter, 1996; Martinez-Torteya et al., 2014; Tronick, 1989). As another example, in the behavioral domain, children whose caregivers provide encouragement for independent efforts to achieve goals or learn new tasks may develop a sense of competence and intrinsic motivation that facilitates effortful engagement in challenging tasks in settings outside the home (Ryan & Deci, 2000). Relatedly, consistent with Vygotsky's (1978) theory of proximal development, children whose parents engage in scaffolding behavior (including teaching, praise, encouragement, and positive affective engagement) to enable them to perform within their zone of proximal development—the space between what they have already mastered developmentally and what they are capable of doing with parental support—show superior academic and social self-regulation abilities compared with children whose parents' style of instruction is less contingent on their current level of achievement (Clarke, Kelleher, Clancy, & Cannon, 2012; Neitzel & Stright, 2003).

Caregivers' structuring of the physical environment also may be influential; as an example, low-income children on average have less access to cognitively stimulating books and games (Evans, 2004), which may both impact their actual academic attainment and deflate their scores on assessments due to a lack of familiarity with the types of tasks commonly used in intellectual and academic assessment of young children. A striking percentage of low-income children are in child-care settings that provide inadequate (24%) or minimally adequate (36%) support for their development; in turn, these children show higher levels of internalizing and externalizing behavior problems and demonstrate lower levels of positive behavior compared with their peers in higher quality child-care settings (Votruba-Drzal, Coley, & Chase-Lansdale, 2004). Within all of the areas in which caregivers provide structure for their children, the impact of a given factor is dependent on the match or mismatch between the two; children's temperamental and developmental characteristics may strongly

influence how they respond to a situation. Similarly, caregivers' individual characteristics, including expectations regarding child development and availability of personal psychological resources with which to meet the children's needs for whom they care, will influence whether a set of behaviors is viewed as problematic (Seifer, 2000).

Characteristics of the setting (home, day care, school, and any other place the child spends substantial amounts of time), including both the overall quality of the setting and the match between the child and the setting, may also impact the child's functioning. As noted already, young children's behavior is highly sensitive to contextual and relational influences (Clark et al., 2004; Tronick, 1989), and it may therefore be more variable across settings than older children's behavior. It is common for parents and teachers, for example, to provide very different perspectives, with often very low correlations between their reports (Achenbach, McConaughy, & Howell, 1987). Though this may be due to differences in caregiver behavior or attitudes, it may also, in many cases, be due to differences in the setting, including the physical structure of the setting, the way time and activity is structured in the setting, adult-to-child ratio, number of children, behavior of other children in the setting, level of sensory stimulation, and adequacy of sleep support. Inconsistency in rules between home and school or day care (e.g., requirements for where eating and sleeping take place) may also be a source of differences between caregivers' reports. Given children's sensitivity to contextual influences such as these, the evaluation process would ideally include information gathering not only from multiple caregivers but also across multiple settings and within multiple types of interactions (e.g., unstructured play, challenging goal-directed tasks, shared reading). However, as this goal is often impractical, a compromise is to ensure that data are collected from multiple informants who are familiar with the child's behavior in different settings and in different types of interactions. When gathering information as part of an evaluation, reports from multiple caregivers are likely to yield a more complete picture of the child's functioning than reports from the child's parents alone (Dirks et al., 2012).

It is especially important to gather information from multiple caregivers in cases where a child regularly spends time in different contexts, with different primary caregivers in each (e.g., a child who is in full-time day care with a neighbor, is cared for by his mother at home during the week, and spends weekends in the care of his father). As discussed, where the reports of different caregivers diverge—for example, where a child's father reports problematic levels of disruptive behavior and her teacher reports no issues with disruptive behavior—the discrepancy should typically be viewed not as evidence of error on the part of one reporter but rather as evidence that contextual, cultural, interpersonal, or other influences may be contributing to the perceived problem. The review of assessment data should then include an analysis of the contexts in which a child is reported to have difficulties, with an eye toward identifying clues for intervention strategies to recommend for the child (e.g., creating consistency between home and day care with respect to the child's nap schedule). In the case in which the child's difficulties occur across multiple caregiving relationships or contexts, they may be more severe than the case in which the difficulties are limited to one relationship or one caregiving context.

Lastly, and briefly, it is also important to consider aspects of the child's context outside of his or her caregiving environment, such as neighborhood and community relationships and functioning. As an example, acts of violence occur considerably more frequently in high-poverty areas compared with more advantaged areas (Briggs-Gowan, Ford, Fraleigh, McCarthy, & Carter, 2010; Friday, 1995; Sampson, Raudenbush, & Earls, 1997), and some studies have reported extremely high rates of community violence exposure among children in high-risk urban areas, with estimates ranging from 42 to 78% (Schechter & Willheim, 2009). Based on these figures, the possibility that a child's behavioral, academic, or emotional difficulties are trauma related should become particularly salient when a child is known to live in a high-poverty/high-risk urban neighborhood; assessors will be even better served by more specific and nuanced knowledge of neighborhood characteristics in the communities in which they work. Even without identifying specific trauma exposure, living in underresourced neighborhoods is associated with elevated disruptive behavior in toddlers (Heberle, Thomas, Wagmiller, Briggs-Gowan, & Carter, 2014).

Sociocultural Factors

In addition to identifying the child's caregiving relationships, mapping out the larger web of family relationships in which the child is developing, and considering the ways the child's presentation may be influenced by the caregiving context and the setting in which the child is cared for, assessors must also consider the ways sociocultural factors may affect caregiver reports, children's performance on assessment instruments, the meaning of a child's behaviors to the caregiver and other family members, whether family members are worried or concerned about the child, family members' service seeking behavior and openness to referrals, and the assessor's own interpretation of the child

TABLE 1.1 Illustrative Examples of Sociocultural Factors that Impact Assessment Findings[1]

	Example of norm	Explanation of relevance in the assessment context
Communication style	Expectations for appropriate eye contact vary across cultural groups; for example, Asian American children may avoid eye contact with authority figures as a symbol of respect and deference (Thaler, Allen, & Scott, 2014).	Avoidance of eye contact may be interpreted as symptomatic if an assessor from a group in which sustained eye contact is normative (e.g., European Americans) is unaware of the variability in cultural norms for these behaviors.
Caregiving norms	Middle-class Puerto Rican mothers have been found to engage primarily in mother-led feeding practices (in which the mother feeds her infant with a spoon or bottle, in contrast to the infant using a spoon or bottle on his or her own) with their 12-month-old infants and to expect that their infants will not yet be able to self-feed at this age (Schulze, Harwood, & Schoelmerich, 2001).	Instruments designed to measure adaptive behavior through the lens of middle-class Euro American culture, in which early attainment of self-feeding skills is emphasized, may falsely identify delays in children whose caregivers have simply not yet organized their interactions with their children to require the development of these skills.
Priorities for child behavior	Endorsement of conformity as a child-rearing goal among mothers is negatively correlated with income, educational attainment, and occupational prestige; thus, overall, as socioeconomic status increases, mothers are less likely to see conformity to externally imposed norms as a model for their children's ideal behavior (Luster, Rhoades, & Haas, 1989).	An assessor primed to assess a given caregiver's attitude regarding conformity versus self-direction is likely to have greater success communicating with the caregiver about his or her child's strengths and weaknesses than an assessor who assumes that the caregiver shares his or her own values regarding conformity versus self-direction.
Beliefs about mental health services	Latinos, Asian Americans, and African Americans have been found to report high levels of perceived stigma regarding mental health problems in their communities (Alvidrez, 1999; Brown, Marshall, Bower, Woodham, & Waheed, 2014).	An assessor who is familiar with how perceived stigma might function as a barrier to service engagement is likely to be more effective than one who is not at addressing caregivers' concerns about confidentiality and encouraging the family's continued engagement in services (if needed).

[1] Endorsement of cultural norms may vary depending on ethnicity, race, socioeconomic status, immigration status, level of acculturation (for immigrant families), geographic location, and other aspects of identity, both separately and in intersection with one another.

and family's presentation (Table 1.1). Early childhood social-emotional and behavioral problems are identified when a set of behaviors appears with heightened or reduced frequency and intensity or when a child shows developmental deviance through the presence of unusual behaviors or the quality of typical behaviors. While diagnostic guidelines sometimes specify the frequency with which a behavior must occur to be consistent with a clinical diagnosis, it is often the case that caregivers are asked to determine whether a behavior occurs frequently or not, a determination that will necessarily be influenced by the caregivers' expectations for child behavior. Such expectations will vary across cultures and communities. In addition, the determination of whether a behavior (e.g., arguing with adults) has occurred at all is culturally mediated. Thus, the identification of social-emotional and behavioral problems is far from culturally neutral. In addition to problems related to the frequency, intensity, or quality of behavior, children may also show delays in the acquisition of social-emotional competencies (Briggs-Gowan & Carter, 1998). The identification of delays in competencies, like the identification of problem behaviors, is dependent on the caregivers' expectations for the child as well as the child's access to opportunities to develop and demonstrate competencies.

For these reasons, sociocultural considerations must inform the entire assessment process, from instrument selection through interpretation of findings. In selecting instruments for assessment, assessors should consider the following (note that this is not an exhaustive list): (1) Are the items on the instrument culturally relevant for this parent–child dyad? (2) Does the sample on which this instrument was normed represent this parent–child dyad? (3) Is the language in which this instrument is written the optimal language in which to assess this parent–child dyad? (4) If an instrument is designed to use familiar tasks as part of the assessment process (e.g., asking a child to complete visual puzzles), are these tasks actually familiar for this parent–child dyad?

Unfortunately, in many cases socioculturally and linguistically appropriate instruments may not be available. The use of culturally inappropriate instruments may have striking effects and is therefore no small problem; one example comes from a study of Australian Aboriginal children, in which all 124 children who participated in the study screened below the cutoff for detecting developmental disabilities or academic delays on a normed and validated screening tool (the Brigance screen) (D'Aprano, Carapetis, & Andrews, 2011). Such results highlight the damage that may occur when culturally invalid instruments are used

with young children—though, unfortunately, the failure of the instrument to work as intended will likely be far less obvious in the context of one-on-one assessment with a child (as opposed to a large research study or a large-scale universal screening effort). In work with young children and their families, several strategies may be used to enhance the overall cultural appropriateness of an assessment, even if culturally appropriate standardized instruments are not available. For example, it may be helpful to review items with a caregiver, discussing the caregiver's understanding of the meaning of both symptoms that have been endorsed and those that have not. Caregivers can be asked to give examples of a time when they observed a behavior that they have endorsed on a symptom checklist. (Note that this strategy is likely to yield valuable information from parents belonging to a cultural group on which an instrument was normed as well as from parents for whom an instrument may not be culturally valid.) In addition, it is important to ascertain whether the behaviors assessed by a given instrument—whether observed, assessed in an interview, or reported on a questionnaire—are culturally relevant for the target child. For example, if a child is from a cultural group in which nonverbal expression is valued, the examiner should be careful to look for signs that a child is responding nonverbally to a prompt meant to elicit a verbal response in the dominant culture but designed to fit a different cultural group (McLaughlin, Gesi Blanchard, & Osanai, 1995). (A nonverbal response in this case may indicate not a deficit in the skills needed to respond verbally but rather an incongruence between the forms of communication expected by the test designer and the forms of communication familiar to the child.) In addition to attempting to compensate for inaccuracies in a given standardized instrument (or set of instruments) that are not culturally valid for a given client, assessors should also consider whether there is important information about the child's development that is missed entirely by standard measures. For example, standard psychological assessment batteries typically do not include measures of ethnic and racial identity development, although these are essential aspects of the overall development of racial and ethnic minority children (Yasui & Dishion, 2007). Thus, overall, in cases in which the set of available instruments contains no culturally valid tools, assessors must be resourceful in identifying means for gathering supplemental information and extremely cautious about interpreting any standardized results obtained by calibrating a child's functioning against a normative group that does not represent their experience.

In addition to instrument selection, assessors must also consider cultural factors throughout the process of interviewing parents and children and interpreting the overall findings of the assessment (Kleinman, Eisenberg, & Good, 1978). We suggest that assessors consider the following questions as a starting point for framing a culturally informed evaluation: (1) What does the caregiver see as the problem? (2) What are the caregiver's expectations with regard to treatment? (3) How does the problem affect the child, caregivers, and other family members? (4) What does the caregiver see as optimal behavior for this child? (5) What are the caregiver's beliefs about child development? What does the caregiver see as the key tasks of development at this stage in the child's life? (6) How have community members responded to the child and caregiver? (7) What is the predominant attitude regarding help seeking in the caregiver's community? If known, how do community members see the organization with which the assessor is affiliated? Does the caregiver share these beliefs?

Culturally competent assessment requires that these and related questions be addressed for each family individually; however, familiarity with the cultural groups with which a family identifies is likely to enhance the assessment process by informing the questions asked by the assessor, the way questions are asked, and the way the caregiver's responses to questions are understood. To this end, consultation with colleagues who share an identity status with the caregiver may be invaluable.

In many cases, assessors may have only limited access to information about a child's caregiving context, family relationships, and cultural affiliations. For example, screening instruments may collect only minimal demographic information about a family and are often administered in contexts in which an assessor has little or no direct contact with caregivers. In these cases, the selection of instruments that are appropriate for the target population is particularly important, as is additional, culturally competent assessment of any problems identified by a screening instrument. Care should be taken to ensure that screening results are delivered in a culturally competent manner, with consideration given to the linguistic needs of the family receiving feedback as well as to beliefs known to be widely held in the community that may impact caregivers' understanding of screening results and their actions following the delivery of these results.

DOMAINS OF DEVELOPMENT

One of the myths about early emerging psychopathology that has been debunked in the past two decades of research on young children's mental health is that early emerging

psychopathology is undifferentiated. Indeed, whether validating the presence of differentiation across broad band domains of psychopathology, such as distinctions between disruptive or externalizing problems (which typically include problems in aggression, hyperactivity, inattention, and defiance), mood or internalizing problems (which typically include problems in social withdrawal, depression, and anxiety), and regulatory problems (which typically include sleep, eating, negative emotionality, and sensory symptoms) (Achenbach, 1966; Achenbach, Edelbrock, & Howell, 1987; Carter, Briggs-Gowan, Jones, & Little, 2003) or validating distinctions between different types of symptoms within a dimension of psychopathology, such as anxiety (Mian, Carter, Pine, Wakschlag, & Briggs-Gowan, in press; Mian, Godoy, Briggs-Gowan, & Carter, 2012; Spence, Rapee, McDonald, & Ingram, 2001) or disruptive behavior (Wakschlag et al., 2014; Wakschlag, Henry, et al., 2012; Wakschlag, Briggs-Gowan, et al., 2008; Wakschlag, Hill, et al., 2008), both parent reports and observations support differentiation of psychopathological symptoms in the toddler/preschool period. Based on this relatively new recognition of differentiation in the early emergence of psychopathology (and as reviewed later in this chapter), screening and assessment tools that focus on differentiated aspects of psychopathology have been developed.

Whether used in clinical or research settings, tools that assess social-emotional behaviors and psychopathology need to be interpreted in relation to knowledge about children's relational, family, and cultural contexts and in relation to the children's developmental context, or developmental functioning. We consider the child's developmental context quite broadly, as multiple developmental domains transact with normative social-emotional development and psychopathological trajectories. Risks for or the occurrence of psychopathology intersect with delays, deficits, or atypical functioning across many other developmental domains.

Indeed, intersections in developmental risk across psychopathology and other developmental domains is almost always observed; children with intellectual and developmental disabilities (Einfeld et al., 2006; Emerson, 2003), language delays and specific language disorders (Henrichs et al., 2013; Ross & Weinberg, 2006), learning disabilities (Morgan, Farkas, Tufis, & Sperling, 2008; Yu, Buka, McCormick, Fitzmaurice, & Indurkhya, 2006), or sensory processing disorders (Ben-Sasson, Carter, & Briggs-Gowan, 2009; Ben-Sasson, Soto, Heberle, Carter, & Briggs-Gowan, 2014) show increased rates of psychopathology. Importantly, the nature of these intersections varies from one condition to another, so knowledge of neurocognitive and linguistic profiles or identified intellectual and developmental disabilities can inform which aspects of psychopathology are assessed in greater depth. Thus, if a child has a known neurodevelopmental condition, such as autism or fragile X syndrome, a literature review should help to identify common areas of difficulty in the social-emotional domain (e.g., anxiety) that should be given additional attention in the assessment process. Conversely, if a social-emotional problem such as ADHD is under consideration, problems in executive functioning should be assessed as these two often co-occur.

Direct assessment of multiple developmental domains is often essential to understanding the *whole child*. Further, given that many young children will not be in environments in which informants will have the knowledge to report on the child's functioning across all of the relevant developmental domains, we strongly encourage including direct, norm-referenced assessment of cognitive and linguistic functioning prior to interpreting the results of social-emotional assessments, particularly since some assessments of social-emotional and behavioral functioning assume normative competencies in other developmental domains. single-informant screeners are not adequate for this purpose. For example, within assessments of attention, questions may include ratings of the child's ability to follow multistep instructions, which may be better explained by a receptive language deficit than inattention. Similarly, a young child with an intellectual disability may not be engaging in pretend play, but this may reflect a general cognitive delay or deficit rather than a specific delay or deficit in the social-emotional domain. In general, we do not expect gains in social-emotional development to exceed those observed in language or cognitive domains. Therefore, these two developmental lines are particularly crucial for contextualizing interpretation of findings in the social-emotional domain.

In clinical settings, assessment of other developmental domains may aid in motivating parents to participate in preventive and targeted interventions designed to reduce psychopathology. In early childhood parents are often more concerned about and more attuned to delays in language development than to atypical emotional, social, or behavioral development (Godoy, Carter, Silver, Dickstein, & Seifer, 2014). Presenting to parents the ways symptoms may be interfering with language (or other) cognitive learning can aid in building a shared view of the child that will motivate parents to participate in interventions focused on reducing symptoms.

Explaining the intersections of developmental lines in relation to establishing risk for psychopathology may also aid in freeing parents from the unfair burden of responsibility or feeling blamed for causing their child's social-emotional or behavioral problems. For example, explaining the ways challenges in processes such as inhibitory control, executive functioning, language processing, and emotional reactivity can intersect with and heighten risk for psychopathology—particularly given that the explanation can emphasize temperamental and neurocognitive vulnerabilities or adverse environmental exposures—can often facilitate greater empathy for a child who has been engaging in problem behaviors that cause burden to the family system. Therefore, knowledge of multiple developmental domains and their intersections, many of which are well documented in the literature, is extremely important in assessment domain and measurement selection. Moreover, knowledge of children's functioning in multiple developmental domains is often essential to appropriate interpretation of findings in research and clinical settings, and can be used to promote a stronger alliance between clinicians and parents when giving feedback and offering recommendations about intervention to parents whose children evidence elevated symptoms and disorders in clinical settings.

SELECTING AN ASSESSMENT APPROACH AND TOOL

When selecting tools to assess social-emotional problems in young children, a number of factors should be considered: (1) making sure the tool will best suit the needs and goals and purposes of the assessment and will fit within any setting constraints; (2) reviewing psychometric properties, such as *reliability* and validity; (3) evaluating the appropriateness of the tool; and (4) reviewing the *appropriateness of the tool for* children of a particular age or cultural background.

Types of Tools

When selecting a tool, it is important to determine what *type* of tool will provide the level of information required given the goals of the assessment. We discuss three types of tools for collecting information from parents and other caregivers: brief screeners, more comprehensive in-depth checklists, and psychiatric interviews (Table 1.2). These different types of tools vary tremendously in their format, the level of information that they provide, and the purposes for which they are most appropriate. Screeners typically yield scores that indicate whether a child *may* have

TABLE 1.2 General Characteristics of Different Types of Parent and Other Caregiver Report Tools

Type	General format	Type of information obtained
Screeners	5–10 minutes to complete Completed by parents or other caregivers Minimal training to administer or interpret Many assess a wide array of problems, such as behavior problems and emotional problems Some focus on a specific problem area, such as behavior problems only	Screeners usually provide one or two scores that indicate whether a child falls into a risk category suggesting that the child may have problems without providing detail into the specific nature of the problem. Results often trigger a more in-depth assessment to determine specific areas that are problematic, to identify treatment needs, and to inform planning.
Comprehensive checklists	15–30 minutes to complete Completed by parents or other caregivers Minimal training required to administer Interpretation typically requires training in psychological assessment Provide 2+ broad domain level scores (e.g., externalizing, internalizing) May provide narrowband scores about specific areas (e.g., anxiety)	Comprehensive checklists provide in-depth information about a child's functioning. Most cover multiple areas, but some focus on specific types of difficulties, such as executive functioning problems. Scoring typically provides a profile of functioning, showing strengths and weaknesses in different areas. These results can be used to establish eligibility for services, generating treatment plans, and documenting change over time.
Diagnostic interviews	1–2 hours to complete at a minimum Parent or other caregiver is interviewed by a trained individual All interviewers must be trained in administration Some may be administered by nonclinicians; others require clinical interviewers Interviews probe for information about whether symptoms are present, duration and onset of symptoms, and impairment	The detailed information obtained from these interviews allows determination of whether a child meets psychiatric criteria for a range of disorders. Scoring also can yield symptom counts within disorders, such as how many depressive symptoms were reported.

social-emotional problems but do not provide in-depth information about specific areas that are problematic. Longer, comprehensive checklists provide more in-depth assessment of problems within a specific area or areas. Many of these checklists provide detailed profiles that illustrate a child's strengths and weaknesses relative to children of a similar age in multiple areas, such as depression, anxiety, impulsivity, aggression, and social competencies. Such profiles and score details can be especially helpful for treatment planning, and documenting change over time or with treatment. However, checklist tools are *not* appropriate for establishing whether a child meets criteria for a psychiatric disorder. Instead, psychiatric interviews collect very detailed information necessary to establish whether a child meets diagnostic criteria for a range of psychiatric disorders (e.g., symptom presence, onset, duration, and impairment), and can be important for treatment planning. Observational methods also can supplement more traditional informant report methods. In the next section of this chapter, we will review each type of measure in detail and provide a review of selected measures available for assessing social-emotional functioning in young children.

Understanding Psychometric Properties

The first step in reviewing an instrument should be to examine its psychometric properties. Unfortunately, in both clinical and research settings, practical considerations such as a tool's ease of use; the time required for training administration, scoring, and interpretation; costs associated with use; the level of professional training required to administer, score, and interpret the instrument; and the influence of common regional practices often outweigh consideration of psychometric properties. The following section outlines psychometric issues most relevant to parent–caregiver reports in young child assessment: reliability, validity, sensitivity/specificity, positive and negative *predictive value*, and *normatization*. Other factors such as cultural considerations, developmental appropriateness, and response formats that may influence instrument adoption also are discussed.

Reliability

Reliability refers broadly to the consistency or stability of a measure. For measures that have scale scores, the consistency of a measure is typically evaluated by examining two or three aspects of reliability: internal consistency, test–retest reliability, and interrater reliability. Table 1.3 provides an overview of these types of reliability and the types of tools to which they generally apply. Rules of thumb for interpreting whether a tool has good enough reliability for different types of measures and related statistics are presented in Table 1.4.

Internal consistency is the extent to which individual items on the scale hang together and reflect the same construct. Usually, this is assessed with Cronbach's alpha (Cronbach, 1951). Scales with Cronbach's alpha of .70 or greater are usually considered to have adequate internal consistency, whereas those with alphas between .60 and .69 are considered marginal and those falling below .60 are considered unacceptable (Cicchetti & Sparrow, 1981; Nunnally, 1978). However, some clinically informative young child measures may not have acceptable internal consistency. This can occur if a measure includes behaviors that rarely occur in the population (e.g., atypical behaviors related to autism spectrum disorder; ASD) or sets of behaviors that are clinically concerning but not likely to co-occur (Achenbach, Edelbrock & Howell, 1987; Briggs-Gowan & Carter, 1998). For example, the ITSEA Atypical Behaviors Index and Maladaptive Index each concern a set of behaviors that are rare but clinically important (e.g., rocking, spinning, PICA, head banging). These indices are expected to have low internal consistency in a normative population (Carter & Briggs-Gowan, 2006),

TABLE 1.3 Types of Reliability

Type	Definition	Measures to which this is most relevant	Statistic used
Internal consistency	How well do the items in a scale hang together?	Measures with scale scores, such as screeners or checklists	Cronbach's alpha (α)
Test–retest reliability	How consistently does the tool provide the same results over time?	All measures	Correlation coefficients (r) or intraclass correlation coefficient (ICC) (Bartko, 1976)
Interrater reliability	How consistently does the tool provide the same results when different individuals rate it?	All measures, especially those where judgment is involved, such as semistructured interviews or observational systems	Intraclass correlations, kappa

TABLE 1.4 Reliability Statistics and Rules of Thumb for Interpreting Reliability

Measure type		Score types	Statistic	Criteria[1]
Dimensional	Dimensional scores Test–retest (about the same person at different times) Interrater (about the same sample of information about the same person by different raters)	Symptom counts Coding composite scores Scale scores	Intraclass correlation (ICC)	<.40 Poor .40 to .59 Fair .60 to .74 Good .75 to 1.0 Excellent
	Dimensional scores Internal consistency (how well the items hang together)	Items that comprise scale scores Codes the comprise composite scores	Cronbach's alpha	<.60 inadequate .60 to .69 poor/marginal .70 Fair .80 Good .90+ Excellent
Categorical	Categorical variables Interrater reliability Test–retest reliability	Disorder vs. not Below cut score vs. above cut score Ordinal coding scale	Kappa statistic (*K*)	<.40 Poor .40 to .59 Fair .60 to .74 Good .75 to 1.0 Excellent

[1]Cicchetti, 1994; Landis & Koch, 1977.

despite measuring behaviors that may be red flags for a need for further evaluation and be clinically and diagnostically important in and of themselves. Thus, while internal consistency is one important indicator of reliability, low internal consistency may be tolerated if a score is based on rare behaviors that when present may be clinically informative. Therefore, understanding the items that go into a scale is important to interpreting internal consistency (Cicchetti & Sparrow, 1981).

Test–retest reliability reflects the stability of a measure when it is completed twice over a relatively short time frame. High test-retest reliability suggests that respondents tend to understand items in a similar way over time and that the underlying construct that is measured does not change substantially over a brief period of time. When test–retest reliability is assessed in older children and adults a two- to four-week period is often used. However, a shorter time period is desirable for infants, toddlers, and preschoolers because the rapid pace of development in this period can reduce test–retest reliability. Contextual variation also could contribute to lower reliability between two *assessments*. For example, peer aggression scores may be quite different if a parent answers questions during a time period when the child has not had much interaction with peers (e.g., during summer break) versus when the child routinely attends a child care program. Thus, contextual and developmental variation may contribute to lower reliability over time in young children.

For interview assessments or observational measures, *interrater reliability,* or the consistency of the measure across raters, should also be evaluated. Interrater reliability indicates whether different people who have the same information about a child tend to rate that information in the same way. In the case of a checklist measure, this might refer to agreement between parents and teachers or between mother and father. In the case of a psychiatric interview, this would mean that after reviewing the parent response, two different people rate the symptom as similarly present or absent. For psychiatric interviews the unit of reliability analysis may be symptom counts or diagnosis. In general, the more structured the assessment format and the lower the inference level required to make ratings, the greater the probability that adequate test–retest and interrater reliability can be obtained in interviews. The availability of detailed administration manuals or training further enhances interrater reliability. A problem often encountered when using clinical interviews is that even the best-trained individuals may drift over time, gradually changing how they administer or rate an interview. This can be minimized by having periodic reviews by an expert who independently reviews and rates the same interview and provides feedback to the interviewer.

Validity

Validity refers to a measure's ability to measure the problem, skill, or trait that it is intended to measure. Using a measure that does not have adequate validity for measuring a particular problem may lead to mistaken conclusions about the nature of a child's problems and affect clinical decisions and care. For example, use of a measure with poor validity for assessing anxiety may mean that clinically impairing anxiety does not receive adequate clinical attention, potentially affecting not only anxiety outcomes but also other areas of functioning affected by anxiety, such as participation in age-expected social activities in

which social skills are honed. More accurate, valid characterization of a child's strengths and weaknesses can drive more targeted, tailored treatment planning and optimize outcomes. For research purposes, validity is essential to ensure that constructs of interest are well characterized and optimize capacity to truly test the hypotheses of interest. Several types of validity that are commonly studied in the course of developing social-emotional/behavioral assessment tools are briefly introduced in this section.

Content validity or *face validity* refers to whether the individual items, questions, and scales actually measure the underlying construct they are intended to measure. One aspect of this that is particularly important for young children is whether the items are developmentally appropriate for the target age range. This is often established during a measure's development phase by an independent expert review process. *Construct validity* refers to how well a measure assesses the specific construct it is intended to measure. This can be assessed by comparing scores on one measure with scores on another validated measure of the same problem. Another way to evaluate construct validity is to examine how well a tool classifies children with a particular kind of clinically significant problem or disorder from both other children with other types of clinically significant problems or disorders and from those without clinically significant problems and disorders. This type of validity is evaluated in terms of the measure's sensitivity and specificity, which is discussed in detail later in this section.

Factor structure provides another indicator of validity, by empirically testing whether the structure of the data fits together statistically in a manner that fits with expectations, based on theory or prior evidence in the area. An evaluation of factor structure can test, for example, whether different types of anxious behaviors cluster together into multiple discrete clusters (e.g., with separation anxiety items on one factor and phobic/fearful items on another) that load onto a higher order structure, as has been demonstrated in young children (Mian et al., 2012; Spence et al., 2001). *Convergent validity* refers to whether a measure is positively associated with other measures of related, but different, constructs. For example, given that anxiety and depression symptoms are often correlated, one would expect that a measure of early anxiety would correlate positively with a measure of early depression.

Discriminant validity or *between groups validity* refers to whether a measure differentiates children who are identified as having a particular problem it is intended to measure from children who do not have that problem. For example, one might examine whether children who have been diagnosed with anxiety disorders have higher scores on a measure of anxiety than healthy children who do not have anxiety disorders.

Predictive validity refers to a measure's ability to predict the same problem over time. For example, evidence that higher anxiety scores on a measure predict higher anxiety later in childhood would support predictive validity. Predictive validity often is not formally evaluated when a tool is being developed, but it can be an important form of validity for young child measures. Demonstrating that a tool predicts later functioning is essential to showing that it captures a clinically meaningful aspect of early functioning that is not simply a transient perturbation in functioning that wanes with development.

Validity Within a Longitudinal Developmental Context

Developmental processes also may affect construct validity. One of the steps in psychometric development is to test and establish the factor structure of a measure, or how items load on scales (e.g., aggression), how scales correlate with one another (e.g., aggression, hyperactivity, oppositional behaviors), and how multiple scales load onto higher order factors (e.g., an externalizing factor that includes aggression, hyperactivity, and oppositionality). However, when following children over time or using a measure that is the same across a broad range of ages, it is also important to evaluate a measure's factor structure over time or between more narrow age groups (Carter et al., 2003). This is rarely reported and it can be difficult or impossible to find information about the consistency of a measure's structure over time. Without knowledge that a measure demonstrates a consistent factor structure across development it is impossible to interpret whether longitudinal patterns represent heterotypic or homotypic stability or instead change.

Validity of Classification

Another important consideration is how well a measure classifies a child has *having a problem* or *not having a problem*. Although a test or measure may be a reliable and valid measure of an underlying construct or behavior, there is still an issue of how useful it might be for a given diagnostic or classification purpose (e.g., meets criteria for oppositional defiant disorder). This is important for tools that yield categorical scores that indicate whether a child's problems are above or below a clinical threshold indicative of problems. In psychological testing, this most commonly applies to screening tools or longer checklist measures that are used to identify children who may have problems and to trigger an action to be taken, such as further evaluation

or referral. The problem of misclassification and its impact on the person being classified is of particular concern.

Sensitivity, specificity, positive predictive value, and negative predictive value are statistics that pertain to classification. *Sensitivity* refers to the proportion of children who fall within a given problem group (e.g., those known to have a disorder according to another criterion) who are successfully classified by the tool in question. If a measure classifies 85 of 100 children with established anxiety disorders as having high anxiety, it has 85% sensitivity. As sensitivity goes up, false negatives go down. *Specificity* refers to the proportion of children who do *not* have a particular problem who are correctly classified by the tool in question. Therefore, a measure that classifies 90 of 100 children who do not have an anxiety disorder as not high in anxiety has 90% specificity. As specificity goes up, false positives go down.

Positive predictive value (PPV) is the proportion of children flagged as positive by a screening tool who truly have that problem according to another criterion. Thus, if 50 of 100 children who are positive according to the tool indeed have problems, PPV is 50%. PPV is correlated with prevalence rates; that is, as prevalence goes up or down, so does PPV, assuming sensitivity and specificity are constant. *Negative predictive value* (NPV) refers to the percentage of children who screen negative that are truly negative according to another criterion.

A general guideline is that assessment tools used for early identification should have minimum sensitivity and specificity of 70% and false positive rates of no greater than 30% of all screened (Cicchetti, Volkmar, Klin, & Showalter, 1995; Disabilities, 2001). Though no minimum PPV exists (to our knowledge) for social-emotional assessment tools, tools should successfully identify a subpopulation in which the prevalence of the problem is more common than in the original population (Milgrom, Mendelsohn, & Gemmill, 2011). Low PPV is usually tolerated when failure to detect a problem is associated with a very poor outcome, such as death. For example, a minimum PPV of 10% has been proposed for ovarian cancer screening because the likelihood of death when this form of cancer is not detected is high (Hensley & Spriggs, 2004; Milgrom et al., 2011) As these examples illustrate, different criteria may be appropriate depending on the purpose of the assessment and the risk of harm associated with failure to detect a problem. Generally, sensitivity should be high (and thus false negatives low) for assessment tools designed to aid in specific diagnoses or treatment recommendations (e.g., structured clinical interviews) because of the potential costs to the child and family of misclassifying a child who *actually has* a specific disorder as *not having* the disorder. However, when the goal is to screen large samples to identify at-risk individuals regardless of their diagnostic status, it can be challenging to find a balance whereby a sufficient proportion of children with problems are detected, while maintaining an acceptable rate of false positives. When scoring thresholds or cut scores are too high, screeners tend to have high specificity and a low rate of false positives, but low sensitivity, meaning that children are more likely to go unidentified. In contrast, when thresholds are too low, sensitivity will be high, but specificity will be low, leading to misclassification and potential flooding of the service system with false positives. False positives also bring some practical costs to the setting conducting the screening and potential emotional cost to families by raising concern or worry unnecessarily.

Finally, one should not assume that a given test will have stable sensitivity and specificity across developmental periods or over time. This is in part because the prevalence of the condition being assessed often varies across periods. A test that has excellent sensitivity, specificity, and positive predictive value with one age group could function poorly in a different age group, particularly if the prevalence of the problem changes. Thus, it is important to review a tool's classification statistics for your target developmental period.

Normatization

The purpose of norms is to provide a means of comparing individuals with their age-based peer group. In many ways, the concept of norms is central to a developmental perspective for assessment. Although traditionally a norm is considered to represent the average test performance of a standardization sample, norms also represent the average, median, or modal performance of a particular developmental level or stage. This is essential at all ages but is especially important in early childhood because children's social-emotional functioning will change with development within this domain and related domains. For example, as illustrated by our work with the ITSEA (Carter et al., 2003) there were age-related changes in some social-emotional problems, from 12 to 35 months, including increases in general anxiety and decreases in separation distress. We and others have also identified robust age differences in children's social-emotional competencies/skills (Carter et al., 2003). For example, on the ITSEA we observed linear increases in 6-month blocks from 12–17 months to 18–23 months to 24–29 months, followed by a social competence leveling from 24–29 and 30–35 months

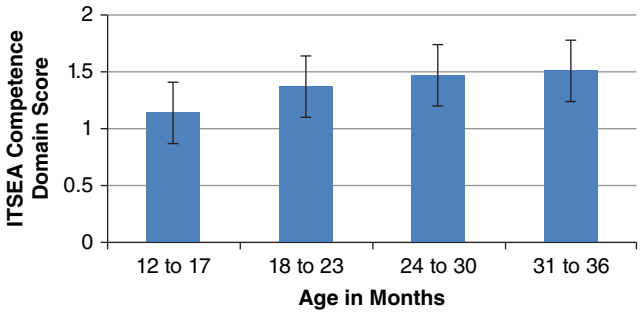


Figure 1.1 Illustration of age effects in social competence from 12 to 36 months of age. See footnote 1.

(Figure 1.1). We also identified sex differences, with greater competence in girls. Thus, a child's competence will be most accurately assessed in relation to children who are the same sex as the child and of similar age within the developmental period from 12 to 36 months.

In selecting a tool and interpreting its results, it is important to be familiar with the sample and conditions in which the normative data were collected—the more similar the normative sample is to your target population, the more relevant the norms (Carter, Marakovitz, & Sparrow, 2006). In a majority of the tests available, the most typical way to operationalize development has been based on chronological age. However, another consideration when comparing a particular child's scores with norms is the child's developmental functioning in other areas. Generally, social-emotional development does not exceed development in other areas. Thus, as noted already, if a child is substantially delayed in multiple areas, such as cognitive and language development, scores may need to be adjusted for developmental level. If a 4-year-old is functioning overall at a mental age of 36 months, then it may make sense to apply 36-month-old norms to determine whether social-emotional functioning is consistent with expectations for mental age. In an ideal world, special norm group data would be available for children with intellectual and developmental disabilities and with genetic and other conditions known to be associated with delays and deficits in social-emotional development and psychopathology.

Normative data also have to be considered within the historical and societal contexts in which they are established. Historical events such as natural disasters, war, or famine may impact the developmental progression of a cohort of children. Norms developed during these periods of relative variation may not be appropriate representations for children growing up in different periods, who are not exposed to these types of experiences. Thus, periodic restandardization of measures is very important.

Cultural Validity and Cultural Norms

Despite a dramatic increase in the diversity of the United States and around the world, issues of cultural validity and the establishment of cultural norms continues to be a challenge to the assessment of social-emotional and behavior problems and disorders in young children. Importantly, translation, even when forward- and back-translation methods are employed, is often inadequate to address issues of cultural appropriateness and validity (cf. van Widenfelt, Treffers, de Beurs, Siebelink, & Koudijs, 2005). Ideally, measures would be developed simultaneously in multiple languages for use in different parts of the world, and examinations of psychometric properties and item and construct equivalence would be established during development. However, there is still extremely limited information about the psychometric properties of measures that have been translated or about the samples or subsamples in which translations have been employed. For example, the ITSEA item "Runs away in public spaces," which is extremely rare in the U.S. context, was rated as normative in Finland until the addition of "for example, in train stations" was added to the item to increase the equivalence of item across these two countries. This is a good example of the limits of translation, as different caregiving patterns and expectations across cultures may lead to situations in which the same words do not have the same meaning in relation to young child psychopathology. Moreover, few measures that have been translated have gathered appropriate normative group data that would permit easy comparisons of the range of relevant psychometric properties across cultural groups. Even when normative data are available for a child and family's home country, these norms may not be appropriate in a new cultural context. For example, for families who have been living in a new country for some time, immigration stress and acculturation may also impact the appropriateness of the use of home country normative data among families. This is clearly an area that will require greater attention in the future.

Knowing What Problems Are Really Being Assessed

A name does not always adequately represent the content of a measure or the scales within it. Therefore, it very important to review the items that make up all of the scales within a measure to truly understand what is being captured and where. In such a review, you may find that a scale labeled "anxiety" includes items such as "cries easily" that could very well reflect other types of problems. Or, an "anxiety" scale might be composed largely or entirely

of items that describe fearful, shy, and socially anxious behaviors but may fail to assess separation anxiety. The term "withdrawal" is often part of a scale's name—item review is important to determine whether this refers behaviors that appear to reflect withdrawal related to (1) shy, slow-to-warm-up behaviors; (2) depressed, anhedonic behaviors; (3) behaviors that could be related to ASD problems such as lack of eye contact and unawareness of social context; or (4) some or all of these types of difficulties.

For clinical and research applications, another important consideration when goals involve following children over time is whether the tool (or tools) used are measuring the same construct over time. One might use the same tool over time or different tools because a single tool does not cover the whole intended age period. Whichever approach is used, we again recommend reviewing the items that make up the scale at different ages. The items within a scale may change in subtle or dramatic ways even within the same tool.

Response Formats

A final consideration when reviewing a checklist or interview is the type of response format that is used. Behaviors vary in terms of whether they are ever present, how frequently they occur when present, and how intensely they are displayed. Similarly, response formats vary in whether a problem is judged as present versus absent, ratings of frequency, or a judgment of how severely the behavior manifests (Achenbach & Rescorla, 2000; Carter & Briggs-Gowan, 2006). Many measures have a response option that crosses ratings of frequency with ratings of how typical the behavior is of the child. As most social-emotional and behavioral problems and competencies vary in frequency or intensity, a response format that captures variation in frequency and intensity makes some sense (Achenbach & Rescorla, 2000). For example, the response format for the ITSEA is 0 for "Not true/rarely," 1 for "somewhat true/sometimes," and 2 for "Very true/often." A contrasting approach, employed in the Behavior Checklist (BCL; Richman & Graham, 1971) is to ask the parent to rate the degree to which a particular behavior is perceived to be a problem. On the BCL parents are asked to rate a behavioral category as 0 for "No difficulties," 1 for "Moderate difficulties," and 2 for "Definitive difficulties." Although very different, each of these response scales is sufficient for identifying children at elevated risk for psychopathology when items are aggregated into meaningful clusters or scales (Achenbach & Rescorla, 2000; Campbell, 1995). For determining clinical diagnostic status, however, neither format is sufficient. Rather, information about onset, duration, and intensity of behaviors and impairment are required to determine diagnostic status. Given the complexity of the information that must be gathered to determine diagnostic status, it is usually preferable to gather this type of information in an interview format.

A final consideration is that some response formats can be overly complex, especially for informants with more limited educational backgrounds. Thus, a scale with a response format that changes from one question to the next or utilizes a wide response scale, but with far fewer anchors, can be confusing or off-putting to parents and introduce error variance into measurement. Items with double (or even single) negatives can also complicate a respondent's ability to use a particular response format (e.g., "Often/Very True" that this behavior is rarely present). Even a simple "Yes/No" format may introduce error, as the threshold for difficulty may not simply be the presence or absence of a behavior but the routine use of the behavior in appropriate contexts (e.g., eye contact). Finally, it is usually easier to rate one attribute or behavior at a time (e.g., child often has angry tantrums) rather than being forced to indicate whether the child is more like one pole of a continuum or another (e.g., whether being angry is more characteristic than being calm).

Summary

Many factors should be carefully reviewed and considered when embarking on a new type of assessment, including the psychometric properties and developmental appropriateness of the tool, how well its norm groups match with the target population (or child). Spending the time to understand the breadth and depth of the information the tool provides and the individual problems that go into different scales or domain scores is essential to a strong assessment foundation.

ASSESSMENT TOOLS

Several types of tools can be used to systematically gather information about social-emotional problems or competencies from parents and caregivers, including brief screening checklists, more comprehensive checklists designed to capture variation both in broad domains and within subareas within those domains, diagnostic interviews, and observational tools designed to capture clinically meaningful information that can supplement parent or teacher reports.

Screening Methods

Screening tools, designed as brief measures for detecting children who may be experiencing problems, are most appropriate when the goal is to quickly and efficiently identify such children from a larger population. Large-scale screening efforts can help to reduce the high rates of unmet mental health needs. Among school-age children, approximately 80% of children needing mental health services are not receiving them. Unmet mental health needs are even greater among young children, children of color, and uninsured children (Kataoka, Zhang, & Wells, 2002). The considerable unmet need for mental health services underscores the importance of enhancing early detection efforts through routine mental health screening.

Universal screening can help detect children at risk for developing mental health problems. Typically, screening yields information about which range a child's score falls within (e.g., normal, borderline, or abnormal). Screening should not be used for diagnosis but rather as a jumping-off point for discussions about mental health and further assessment. Once identified, at-risk children benefit from follow-up that can include more in-depth assessment (e.g., longer measure, clinical interview, clinical observations) to evaluate the extent and nature of the difficulties initially detected. This practice, referred to as multigated or multistage screening, can serve as a cost-effective and minimally burdensome means for screening large groups of children in a manner that permits characterization of risk status (stage 1) and a more detailed profile of problems and competencies when risk is elevated (stage 2). The decision to proceed to later stages of screening can be based on factors such as score profile, parent's indication of concern, and level of impairment. However, even if multigated screening is not being implemented, screening ideally involves attention to the child's behaviors, as well as contextual risk factors in the family and community, including exposure to violence, parental psychopathology, and neglectful parenting.

In addition to identifying the existence of concerns, screening can help to facilitate appropriate follow-up, such as referral for treatment, by identifying potential domains of concern (e.g., speech-language versus gross motor or anxiety versus disruptive behaviors) and severity of concerns. Agencies and providers employing multistage screening may wait until more comprehensive assessment is completed to make referrals, but in many cases second-stage screening does not occur and providers choose to use the results of screening to initiate a referral or begin addressing identified concerns. For example, within pediatric primary care settings, providers may refer a child to the local early intervention program following elevated developmental screening. However, in most cases, the places to which a provider is referring will do a more comprehensive assessment before beginning treatment. While screening tools can help in the treatment planning process and in the documentation of treatment gains, given the brevity of screeners and the limited information provided, it is not ideal to use screeners in this manner. Screening is ideally used at multiple points in time to ensure the continued monitoring of concerns among a large group of children.

Screening can occur universally or it can be targeted. In universal screening, all children in a particular setting or situation (e.g., all children seeing their physician for a well-child visit) would complete screening. Ideally, when universal screening is employed in pediatric practices, every parent (or adolescent child) completes a standardized screening tool at each well-child visit from infancy through adolescence, and identified risks or concerns are closely monitored and addressed as appropriate. In targeted screening, only those children identified via other means (e.g., developmental surveillance in which parents report their concerns about the child's behavior) would be screened.

Universal screening is ideal because it does not rely solely on provider judgment or parent expressions of concern, both of which can be flawed means of identifying issues particularly in early childhood. A review study of screening in pediatric primary care settings determined that sensitivity rates for pediatric providers ranged from 14 to 54%, and that specificity rates ranged from 69 to 100% (Sheldrick, Merchant, & Perrin, 2011). Furthermore, parents of children with mental health problems often do not report concerns or worries about their children's behavior (Godoy et al., 2014; Sheldrick, Neger, & Perrin, 2012). In particular, parents of younger children exhibiting social-emotional/behavioral problems report lower levels of worry or concern than parents of older children and often do not raise concerns with the pediatrician (Ellingson, Briggs-Gowan, Carter, & Horwitz, 2004; Godoy et al., 2014; Horwitz, Gary, Briggs-Gowan, & Carter, 2003; Sheldrick, Neger, et al., 2012). Thus, universal screening can help to identify at-risk children more accurately than when screening is not implemented. Moreover, universal screening can help to reduce disparities in health care because all children receive a similar assessment regardless of family characteristics and provider discretion.

Universal screening is growing increasingly popular, particularly within the pediatric primary care setting.

Pediatric primary care settings are well-suited to the task of detecting social-emotional and behavior problems ("Policy statement—The future of pediatrics: Mental health competencies for pediatric primary care," 2009) as pediatricians can act as the gatekeepers of mental health for a wide range of children. Pediatricians, who often have longitudinal, trusting relationships with families, regularly discuss social-emotional behavioral issues, and they are often the first professional with whom a parent discusses concerns. Thus, they are the people who can first identify problems and facilitate appropriate prevention and intervention services. Families with children who have social-emotional and behavioral problems tend to use pediatric services more than other families (Gadomski, Wissow, Slade, & Jenkins, 2010) and therefore have more opportunities for contact with pediatricians. Pediatric primary care may be particularly helpful in the identification and prevention and intervention of early childhood mental health problems given the more frequent visit schedule among children under 3 years. Additionally, prior to entering more formal child-care or school settings, pediatricians may be the only professional with whom some families have contact who can help to identify problems and facilitate referrals. Indeed, parents who discuss their children's social-emotional/behavioral problems with their pediatrician are more likely to obtain mental health services for their children than those who do not discuss these issues (Briggs-Gowan, Horwitz, Schwab-Stone, Leventhal, & Leaf, 2000).

Universal mental health screening is increasingly important given the growing list of topics pediatricians are expected to cover and shorter office visits. These factors make it more challenging for pediatricians to detect concerns on their own as they have less time to discuss behaviors and make clinical observations and as parents have less time to raise concerns. Additionally, implementing universal mental health screening, particularly in non–mental health settings, can help to destigmatize mental health and increase the likelihood that parents will raise and discuss concerns and seek treatment as needed.

Yet universal screening efforts can be hampered by logistical, systemic, and other barriers. Logistical barriers include issues like difficulty integrating screening into an already busy practice workflow. An additional barrier is pediatricians' self-reported lack of confidence and training in addressing mental health issues (Horwitz et al., 2007). Systemic issues include lack of adequate mental health resources, which is even more of an issue with early childhood mental health.

Despite these challenges, universal mental health screening in primary care has been shown to be feasible and effective in improving rates of referral (King et al., 2010; Schonwald, Huntington, Chan, Risko, & Bridgemohan, 2009). Not surprisingly, universal screening is gaining popularity within other early childhood settings, such as child care centers and women, infant, and children's offices. Given the many contexts in which children may be screened, it is important that states and local agencies work together to ensure that screening efforts are coordinated (e.g., discussing screening across settings, sharing results across agencies through a database or other means).

Screening Methods Characteristics of Screening Tools

Screeners should have several characteristics, especially if they are to be implemented widely. They should be brief and easy to administer, and most parents should be able to complete them independently. They should also be easy and quick to score and interpret. Ensuring these characteristics of a screener can ensure that a maximum number of screens are completed and that staff are not overly burdened. Advances in technology have led to moving screeners to an electronic format, which can further facilitate completion and scoring.

Screening tools vary in the scope of their assessment. Some screening tools are designed to detect a narrowly defined problem. For example, the Modified Checklist for Autism in Toddlers, Revised with Follow-Up (M-CHAT-R/F; Robins et al., 2014) is designed to detect ASD and would not be appropriate when the goal is to detect children with social-emotional or behavioral problems in areas outside of ASD. In contrast, more global screeners, such as the Ages and Stages Questionnaire: Social Emotional (ASQ-SE; Squires, Bricker, & Twombly, 2002) and the Brief Infant Toddler Social Emotional Assessment (BITSEA; Briggs-Gowan & Carter, 2006), are designed to identify children who may be evidencing problems in the broad domain of social-emotional/behavioral problems. Some screening tools, such as the BITSEA and the Strengths and Difficulties Questionnaire (SDQ; Goodman & Goodman, 2009), go a step beyond problems to also detect social-emotional competencies. Selecting a screening instrument that meets the goals of the screening process (narrow versus broad, problems versus competencies) is essential to ensure a successful screening initiative.

In recent years the number of screening tools available for the infant-toddler period has grown. However, many screeners now available focus on a specific area of social-emotional adjustment (e.g., behavior problems) and

do not provide comprehensive coverage of both problems and competencies. For example, the 35-item Eyberg Child Behavior Inventory (ECBI; Eyberg & Pincus, 1999), and the 40-item Toddler Behavior Screening Inventory (TBSI; Mouton-Simien, McCain, & Kelley, 1997) measure behavior problems in children 2 to 16 years of age and 1 to 3 years of age respectively whereas the Preschool Anxiety Scale (PAS; Edwards, Rapee, Kennedy, & Spence, 2010; Spence et al., 2001) measures anxiety symptoms in children 3 to 5 years of age. While these scales all demonstrate acceptable psychometric properties, they may be limited in their ability to screen for a wide range of problems and address social-emotional competencies. Additionally, screening tools that were designed for older children are often scaled down for use with younger children without adequate assurance regarding the developmental appropriateness of items nor the psychometric properties within a younger population.

Selected Screening Tools

Table 1.5 displays several commonly used, brief, parent-report social-emotional/behavioral screening questionnaires. We selected tools that cover an array of problems (and, in several cases, competencies) and that may be appropriate in settings in which wide-scale screening is occurring. These questionnaires are also described briefly. All the questionnaires selected can be completed by parents (and in some cases child care providers) on their own or with the assistance of a staff member. They all usually require 5–10 minutes to complete and are easy to score. Qualified individuals who have undergone appropriate training can score the instruments. Regarding interpretation, all the measures require knowledge of standardized assessment and supervised training in working with relevant clients.

The Ages & Stages Questionnaire–Social-Emotional Version (ASQ-SE; Squires et al., 2002), a screening tool that can be completed by parents of children 3 to 66 months of age, addresses seven behavioral areas: self-regulation, compliance, communication, adaptive functioning, autonomy, affect, and interaction with people. There are eight versions of the questionnaire that correspond with different child ages (6, 12, 18, 24, 30, 36, 48, and 60 months) and eight corresponding scoring sheets. The number of items varies depending on the questionnaire ranging from 22 to 36. For each question item, respondents are asked to indicate whether the behavior is something that the child does "Most of the Time," "Sometimes," or "Rarely or Never." In addition, respondents can indicate whether the behavior described by each item is something that is a "Concern." Several additional questions at the end of the questionnaire ask about parents' concerns about child social-emotional/behavioral functioning (e.g., "Do you have any concerns about your child's eating and sleeping behaviors or about her toilet training?"). An overall summary score can be compared with age-specific cutoff scores to identify children at risk for social or emotional difficulties. The ASQ: SE has strong internal consistency (.82), test–retest reliability (.94), sensitivity (.82), and specificity (.92) in detecting children with developmental delay or social-emotional diagnoses (Squires et al., 2002).

The BASC-2 Behavioral Emotional Screen System (BASC-2 BESS; Kamphaus & Reynolds, 2007) measures behavioral and emotional strengths and weaknesses from preschool through high school. In the early childhood age group (ages 2 to 5 years), tools are available for parents and teachers to complete. The tool asks about internalizing problems, externalizing problems, and adaptive skills, though questions in these domains are combined into a total score. Validity indexes can be used to identify overly negative or inconsistent responses. The BASC-2 BESS has demonstrated acceptable test–retest reliability (0.79–0.85), interrater reliability (0.76–0.90), validity with other measures of child behavior such as the Child Behavior Checklist (0.71–0.76), sensitivity (0.73–0.82), and specificity (0.97) in detecting children with social-emotional/behavioral problems (Kamphaus & Reynolds, 2007).

The Brief-Infant-Toddler Social-Emotional Assessment (BITSEA; Briggs-Gowan & Carter, 2006) is a forty-two-item measure for identifying social-emotional/behavioral problems and delays as well as child competencies in 1- to 3-year-old children. The BITSEA generates a Problem and a Competence score, each of which can be compared with age- and gender-specific cut points. The BITSEA has good psychometric properties, including test–retest reliability and interrater reliability. The BITSEA cut points have demonstrated high sensitivity in detecting children with social-emotional/behavioral problems according to other gold standards (diagnostic interview; Child Behavior Checklist) while maintaining acceptable specificity (Briggs-Gowan et al., 2013). In addition, the BITSEA is highly sensitive to autism spectrum disorders (Carter & Briggs-Gowan, 2005; Gardner et al., 2013). Finally, research suggests that BITSEA has acceptable sensitivity and specificity and predicts a majority (PPV of 67.9%) of children who meet the criteria for a psychiatric disorder, according to the Preschool Age Psychiatric Assessment (PAPA) interview (Briggs-Gowan & Carter, 2008b).

TABLE 1.5 Brief Checklist/Questionnaire Measures That Address Social-Emotional and Behavioral Problems Broadly

	ASQ-SE Ages and Stages Questionnaire Social-Emotional	BESS Behavioral Emotional Screen System	BITSEA Brief Infant-Toddler Social and Emotional Assessment	ECSA Early Childhood Screening Assessment	SDQ Strengths and Difficulties Questionnaire	SWYC Survey of Wellbeing of Young Children
	Squires, Bricker, & Twombly (2002)	Kamphaus & Reynolds (2007)	Briggs-Gowan & Carter (2006) Briggs-Gowan et al. (2013)	Gleason, Dickstein, & Zeanah (2010)	Goodman, 1997	Perrin & Sheldrick (2013)
Age	3 to 66 months	36 months+ (18 years)	12 to 36 months	18 to 60 months	24 months+ (18 years)	1 to 66 months
Respondent (for early childhood forms)	Parent	Parent Caregiver/teacher	Parent Caregiver/teacher	Parent Caregiver/teacher	Parent Caregiver/teacher	Parent
Available languages[1]	Spanish	Spanish	Spanish, Chinese, Dutch, French, German, Hebrew, Italian, Russian, Thai	Spanish, Romanian	Spanish and over 40 languages	Spanish, Burmese, Nepali
Reading level	5th to 6th	4th to 6th	4th to 6th	5th	Low (though not specified)	Low (though not specified)
Number of items	Approximately 30 items (varies by age)	25 to 30	42	36 Teacher 40 Parent	25 (plus up to eight items about impact of problems on the child and family)	~40 (varies by age)
Time frame covered	Not specified	Not specified	Past month	Not specified	Past 6 months	2 weeks for PHQ-2 Other areas not specified
Item response options	3-point rating scale: Rarely or Never; Sometimes; Most of the Time	4-point rating scale: Never; Sometimes; Often; Almost Always	3-point rating scale: Not True or Rarely; Somewhat True or Sometimes; Very True or Often	3-point rating scale: Rarely/Not True; Sometimes/Sort-Of; and Almost Always/Very True	3-point rating scale: Not True; Somewhat True; Certainly True	Variable
Normatization age bands	6- to 12-month age bands	12 months+	6-month age bands	None	24 months+	2 to 12 months
Normed for boys and girls separately	N	Y	Y	N	N	N
Standardized scores (e.g., *T* scores)	N	Y	Y	N	N	N
Scoring	One score produced, which is compared with clinical cut point that varies by child age	One score produced, which is compared with clinical cut point	Produces a Problem score and a Competence score, each of which can be compared with age- and sex-specific cut points	One score produced for the child-related items and one score for the parent-related items; each is compared to a clinical cut point	Produces five subscale scores (Emotional Symptoms; Conduct Problems; Hyperactivity; Peer Problems; Prosocial Behavior) plus a Total Difficulties score, which represents combination of 4 problem domains	Produces separate scores for each part of the SWYC (developmental milestones, family risk, BPSC, PPSC, POSI)

(*continued*)

TABLE 1.5 (*Continued*)

	ASQ-SE Ages and Stages Questionnaire Social-Emotional	BESS Behavioral Emotional Screen System	BITSEA Brief Infant-Toddler Social and Emotional Assessment	ECSA Early Childhood Screening Assessment	SDQ Strengths and Difficulties Questionnaire	SWYC Survey of Wellbeing of Young Children
COVERAGE:						
Problem domains covered	Problems are combined into one total score, though it covers a range of areas including self-regulation, compliance, communication, autonomy, and interaction with others	Problems are combined into one total score, though it covers internalizing and externalizing problems	Problems are combined into one total problem score, though it covers internalizing, externalizing, dysregulation, and maladaptive behaviors	Problems are combined into one total score, though it covers a range of areas	Emotional Symptoms; Conduct Problems; Hyperactivity; Peer Problems	BPSC includes 3 domains (Irritability, Inflexibility, Difficulty with Routines); PPSC covers several domains (internalizing, externalizing, attention problems, parenting challenges) though problems are combined into one overall score
Competencies/skills covered	Competencies are assessed but responses are integrated into overall score. There is not a separate competency scale.	Competencies are assessed but responses are integrated into overall score. There is not a separate competency scale.	Includes separate Competence domain with separate cut point	No	Includes separate Prosocial Behavior Scale domain with separate cut point	No
Parent worry or concern addressed?	Parent can indicate concern about each item; plus open-ended questions about concerns; not used in scoring	No	Two questions gauge parent worry about child behavior and language functioning; not used in scoring	Parent can indicate concern about each item plus there is one question asking about overall concern regarding child's emotional or behavioral development; not used in scoring	Not explicitly, though Impact Supplement asks whether caregiver thinks child has difficulties in emotions, behavior, concentration, or ability to get along with others	Two questions regarding parent concerns about child's behavior and child's learning or development; not used in scoring though providers are encouraged to follow up on concerns raised
Additional notes		Ability for parents to complete forms by listening to audio recordings; includes Validity Index to identify responses that may be overly negative or inconsistent		Parent report form includes two questions about parent depression (PHQ-2) and two questions about parent stress	Impact supplement can be used to assess distress and impairment associated with symptoms	Charts showing normative curves (percentiles) available for BPSC and developmental milestones available, though PPSC and POSI have one cut point

[1]Many measures are available in other languages but are often not normed in those languages.

The Early Childhood Screening Assessment (ECSA; Gleason, Zeanah, & Dickstein, 2010) is a brief screening tool designed to identify emotional and behavioral problems in children 18 to 60 months of age. There are two versions of the questionnaire—a parent report form, which consists of 40 items, and a caregiver/teacher report form, which consists of 36 items. The 36 child-specific items are similar across forms. The parent report form also includes the U.S. Preventive Health Task Force recommended Patient Health Questionnaire—2 questions to assess for parent depression (though the time frame "over the past two weeks" is not included) and two questions that ask about parent stress. Items are scored on a 3-point Likert scale (Rarely/Not True, Sometimes/Sort-Of, and Almost Always/Very True), plus parents can circle the "+" next to each item if they are "concerned about an item and want help with it." Parents can also indicate at the end of the questionnaire whether they are worried about their child's overall behavior. Scores on the child questions and the adult questions are separately compared with clinical cut points to determine risk status. Though research on the ECSA is not as abundant as that on other screening tools, preliminary research suggests that the ECSA has adequate sensitivity (86%) and specificity (83%) as well as good internal consistency (0.91) and test–retest reliability (0.81) (Gleason et al., 2010).

The SDQ (Goodman & Goodman, 2009) is a 25-item behavioral screening tool composed of five items in each area: (1) Emotional Symptoms; (2) Conduct Problems; (3) Hyperactivity; (4) Peer Problems; And (5) Prosocial Behavior (strengths). A separate one- to eight-item scale assesses problem presence, level of severity, and impairment (impact supplement). For providers using the SDQ over time when interventions are put in place, a follow-up form is also available, which asks two questions about the use of interventions (whether the intervention reduced problems or helped in other ways). The SDQ can be used with children 24 months to 18 years of age. There are three age groups for which separate forms are available: 2 to 4 years; 5 to 10 years; and 11 to 17 years. Parent report and teacher report forms are available. Scoring can be completed in less than 5 minutes either by hand or online. Results yield scores for the five scales, plus a Total Difficulties score (sum of the four problem domains, excludes prosocial behavior scale), and an Impact score. However, the SDQ authors have reported that in low-risk or general population samples, it may be better to examine two problem scales, internalizing (Emotional Symptoms and Peer Problems) and externalizing (Conduct Problems and Hyperactivity) rather than the four subscales (Goodman, Lamping, & Ploubidis, 2010). The SDQ has been widely used and investigated cross-culturally with normative data obtained in various countries including the United States using a large sample. While the psychometric properties among of the SDQ among school-age children are strong (Sensitivity = 85%; Specificity = 80%; Goodman, Ford, Corbin, & Meltzer, 2004), its use among preschoolers is less common and rigorous studies among this age group are currently lacking.

The Survey of Wellbeing of Young Children (SWYC; Sheldrick, Henson, et al., 2012) is a comprehensive screening instrument for children under 5 years of age designed to be used easily in busy practice settings, particularly within primary care. There are 12 age-specific versions of the SWYC (2, 4, 6, 9, 12, 15, 18, 24, 30, 36, 48, and 60 months) that vary in question content. All versions of the SWYC include sections on developmental milestones (10 questions covering language, motor, social, and cognitive), family context (nine questions covering family risk factors such as parent depression and substance use), parent concerns (two questions), and child behavior. Regarding child behavior questions, parents of children up to 18 months children complete the Baby Pediatric Symptom Checklist (BPSC; 12 items) and parents of children 18 to 66 months complete the Preschool Pediatric Symptoms Checklist (PPSC; 18 items). The BPSC and the PPSC represent downward extensions of the Pediatric Symptoms Checklist, a behavioral screening tool that can be used with children 6 to 16 years of age. The SWYC also includes a seven-item autism-specific screener, the Parent's Observations of Social Interactions (POSI), which parents of children 16 to 30 months of age complete. Sections of the SWYC that have been examined (BPSC, PPSC, POSI, milestones) demonstrate acceptable psychometric properties, including reliability (test–retest, internal consistency) and validity (Sheldrick, Henson, et al., 2012; Sheldrick et al., 2013; Sheldrick & Perrin, 2013; Smith, Sheldrick, & Perrin, 2013).

Comprehensive Dimensional Tools for Assessing Social-Emotional/Behavioral Problems

When the goal of the assessment is to gather detailed information about the specific areas in which a child is manifesting problems, longer, more detailed assessments are generally most appropriate. Comprehensive checklists can provide a detailed and differentiated understanding of the areas in which a child is reported by the parent to be manifesting a problem and those in which the child's behavior falls in the typical range for his age group. These

types of tools ensure that parents are queried about a broad array of social-emotional problems including behaviors that parents may not be aware can be problems for young children. Comprehensive checklists usually gather information at the level of broad domains and subscales within each domain. The most common domains covered are the internalizing and externalizing domains first identified by Achenbach (Achenbach, 1966). Internalizing problems include fears, anxieties, and depressive and mood problems. Externalizing problems generally include aggressive, disruptive, and non-compliant behaviors, as well as problems of hyperactivity and inattention. Some tools, such as the ITSEA, also assess functioning in other domains, such as dysregulation and social competence (Table 1.7). Similarly, some tools assess functioning in a single domain; for example, the Multidimensional Assessment Profile of Disruptive Behavior (Wakschlag et al., 2014) focuses solely on ensuring a comprehensive assessment of disruptive behaviors, such as noncompliance, temper loss and low concern for others. In addition to providing domain-level scores that indicate a child's overall level of problems within a given broad area, these types of tools also provide subscale scores that indicate a child's functioning in more specific, narrowband areas of functioning. Clinically, having a profile that can pinpoint specific areas of difficulty can be more informative for guiding conversations with parents and treatment planning than broad domain scores. For example, on the ITSEA (Carter & Briggs-Gowan, 2006) and the CBCL/1.5-5 (Achenbach & Rescorla, 2000), the externalizing domain score is calculated by summing across a number of hyperactivity/impulsivity items as well as items describing aggressive and noncompliant behaviors. Given the frequent co-occurrence of disruptive and hyperactivity/impulsive behaviors clinically, a high score may reflect the presence of a wide array of problems throughout the domain. However, some children may display primarily hyperactive/impulsive problems without any elevation in aggression or noncompliance and others may behave in primarily disruptive ways with elevations in all areas. Thus, an elevated domain score provides an imprecise reflection of a child's problem behaviors. In contrast, subscale scores provide more specific information, enabling the clinical formulation to focus on the presenting problems.

Clinically, comprehensive checklists can be especially helpful when the goal is to profile a child's strengths and weaknesses to guide treatment planning. They also are best suited when the goal is to track treatment outcomes, because they provide a detailed assessment of functioning within the specific subareas that were identified as problems when the child began treatment and targeted by the treatment plan. In contrast, using a tool that captures behavior only at the level of the broad domain runs the risk of underestimating improvement. This is because so many behaviors influence domain-level scores. Taking the externalizing domain as an example, if a child entered treatment with problems with hyperactivity/impulsivity and aggression and made significant gains in the area of aggression but not hyperactivity/impulsivity, the externalizing domain score could remain elevated above a clinical cutoff even though the child's aggressive behavior had dropped to normative levels.

Scientifically, increasing evidence suggests that narrowband subscales that capture discrete sets of behavior are more effective than scales that are comprised of heterogeneous sets of behavior for linking behaviors with neurobiological risk factors. For example, efforts to identify genetic contributions to psychopathology have been strengthened by the use of narrowband measures of phenotypic expression (Krueger, 1999; Krueger et al., 2002; Tackett et al., 2013; Todd et al., 2005). As an illustration in young children, one of our studies recently revealed that maternal reports of preschoolers' difficulty learning from punishment (or "Punishment Insensitivity" on the Multidimensional Assessment Profile of Disruptive Behavior) was associated with poorer performance on a computerized task that assessed learning from punishment (Briggs-Gowan et al., 2013). However, mothers' reports of other types of disruptive behavior were not associated with task performance. This narrowband approach also is consistent with the National Institute of Mental Health's Research Domain Criteria framework, which calls for research that employs dimensional measures to characterize aspects of functioning that can be more precisely linked with underlying biological processes, such as genetic vulnerability and neural pathways (Casey, Oliveri, & Insel, 2014; Insel et al., 2010). For these reasons, when the goals of an assessment are to dig more deeply in an effort to drive scientific discovery, comprehensive tools that provide both broad and narrow measures of functioning are often most desirable and a close inspection of the items that contribute to subscales and domains is essential to ensure that the tool captures the constructs of interest.

Selected Dimensional Checklists

Several norm-referenced tools for assessing social-emotional and behavioral problems common in this developmental period are summarized in Table 1.6 and 1.7 and described briefly in this section. These measures are fairly comparable in their general format (as shown

TABLE 1.6 Comprehensive Checklist/Questionnaire Measures That Address Social-Emotional and Behavioral Problems Broadly

	ITSEA	CBCL	BASC-2	PKBS-2	Conners-EC Behavior Scales[1]
	Infant-Toddler Social and Emotional Assessment (Carter & Briggs-Gowan, 2006; Carter et al., 2003)	Child Behavior Checklist ages 1.5-5 (Achenbach & Rescorla, 2000)	Behavior Assessment System for Children, Second Edition (Preschool Forms) (Reynolds & Kamphaus, 2004)	Preschool and Kindergarten Behavior Scales: Second Edition (Merrell, 2002)	Conners Early Childhood (Conners, 2009)
Age (months)	12 to 36 months	18 to 60 months	24 to 72 months	36 to 83 months	24 to 83 months
Norming age bands	6-month age bands	One norm group spanning 42 months	Separate norms for 2–3-year-olds and 4–5-year-olds	One norm group spanning 48 months	6 month age bands
Gender-based norms	Y	Y	Y	N	Y
Description	Checklist	Checklist	Checklist	Checklist	Checklist
Respondent	Parent/teacher	Parent/teacher	Parent/teacher	Parent/teacher	Parent/teacher
Reading level	4th to 6th	5th	4th	Not stated, but low	5th
Number of items	169	99 in parent	134 in parent 100 in teacher	76 in parent and teacher	110 in parent and 112 in teacher
Administration time	30 minutes	15 to 20 minutes	10 to 20 minutes	10 to 15 minutes	25 minutes
Time frame	Past month	Past two months	Last several months	Past 3 months	Past month
Response options	3-point rating scale: Not true or rarely, somewhat true or sometimes, very true or often	3-point rating scale: Not true, somewhat or sometimes true, very true or often true	4-point rating scale: Never occurs, sometimes occurs, often occurs, almost always occurs	4-point scale: Never, rarely, sometimes, often	4 point rating scale: Not true at all (never, seldom), Just a little true (Occasionally), Pretty much true (Often, Quite a bit), Very much true (Very often, Very frequently)
Standardized scores	Y	Y	Y	Y	Y
Clinical cut points	Y	Y	Y	Y	Y
Available in Spanish?	Yes	Yes	Yes	Yes	Yes

[1]The Conners-EC also includes Developmental Milestones Scales (an additional 75 parent and 70 teacher/child-care items) for assessing Adaptive Skills, Communication, Motor Skills, Play, and Pre-Academic/Cognitive development.

in Table 1.6). They are written at a fourth- to sixth-grade reading level, are composed of 100–200 items, and require 15–30 minutes to complete. As normed instruments, these tools provide a means of comparing a given child with population-based means to determine whether the child's difficulties fall in a range that is considered clinically concerning. Despite these commonalities, these measures vary with respect to key factors including (1) whether they were explicitly developed for young children or are instead downward extensions of tools originally designed for older children; (2) the number and content of the domains they cover; and (3) the extent to which they provide insight into specific types of problems.

These tools have very detailed manuals with extensive information about the psychometric properties of each measure. Therefore, we do not attempt to summarize them here. Overall, each of these tools *generally* has acceptable reliability and validity; however, because these are very comprehensive measures with many possible scores, it is possible that an individual scale or even a domain *may not have inadequate* reliability or validity in a particular population or age or sex group. Therefore, a careful review of a tool's reliability and validity within the population to be assessed is critical.

Comprehensive, Multidomain Checklists

The BASC-2 Preschool (Reynolds & Kamphaus, 2004) measures behavior problems and adaptive skills for children ages 2 to 5 years of age. The BASC-2 Preschool version is a parent or teacher report measure that is composed of 100–134 items depending on the informant. The reliability and validity of this measure have been

TABLE 1.7 Breadth and Depth of Coverage by Comprehensive Checklist Measures

	ITSEA	CBCL	BASC-2	PKBS-2	Conners-EC
BREADTH OF COVERAGE:					
Cover a broad array of problems?	Y	Y	Y	Y	Y
Are there scores for multiple problem domains?	Y Internalizing, Externalizing, and Dysregulation Domains	Y Internalizing and Externalizing Domains	Y Internalizing and Externalizing Problems and Behavioral Symptoms Index	Y Internalizing and Externalizing Domains	N
Are there domain scores for social competencies and skills?	Y Competence Domain	N	N Adaptive Skills includes both social and nonsocial content	Y Social Skills Domain	Limited Social Functioning/ Atypical
Are there scores for autism spectrum problems?	Y Atypical and Social Relatedness Indices, Empathy, Prosocial Peer, Imitation/Play	Y DSM-Pervasive Developmental Problems	N Some relevant behaviors in Atypicality scale	N	N Some relevant behaviors in Social Functioning/ Atypical Behaviors
Are there scores for atypical or maladaptive behaviors?	Y Social Relatedness, Atypical, Maladaptive Indices	Y DSM-Pervasive Developmental Problems	Y Atypicality	N	N Social Functioning/ Atypical Behaviors
Are there scores for sensory or regulatory problems?	Y Sleep, Eating, Sensory Sensitivities, Negative Emotionality	Y Sleep Problems, Emotionally Reactive	N	N	Y Sleep Problems
DEPTH OF COVERAGE WITHIN SPECIFIC AREAS:					
Scale scores specifically about anxiety?	Y General Anxiety, Separation Distress, and Inhibition to Novelty Subscales	Y DSM-Oriented Anxiety Problems	Y Anxiety Scale	Y Anxiety/Somatic Problems	Y Anxiety
Scale scores specifically about depression or withdrawal?	Y Depression-Withdrawal	Y Withdrawn or DSM-Oriented Affective Problems	Y Depression, Withdrawal	Y Social Withdrawal	Y Mood and Affect
Combined attention and hyperactivity and impulsivity scores	N	Y DSM-Oriented Attention Deficit/Hyperactivity	N	Y Attention Problems/Overactive (note that this scale includes disruptive behaviors)	Y Inattention/ Hyperactivity
Scale score specifically about attention?	Y Attention Skills	Y However, Attention Problems includes hyperactivity "Can't sit still"	Y Attention Problems	N	N

(*continued*)

TABLE 1.7 (*Continued*)

	ITSEA	CBCL	BASC-2	PKBS-2	Conners-EC
DEPTH OF COVERAGE WITHIN SPECIFIC AREAS: (*continued*)					
Scale score specifically about hyperactivity and impulsivity?	Y Activity/Impulsivity	N	Y Hyperactivity	N	N
Scale score specifically about disruptive behaviors	Y Aggression/Defiance, Peer Aggression	Y Aggressive Behavior or DSM-Oppositional Defiant	Y Aggression	Y Self-centered/ Explosive, Antisocial/ Aggressive	Y Defiant/Aggressive
Scale scores specifically about social skills and competencies	Y Empathy, Compliance, Prosocial Peer, Mastery Motivation, Imitation/Play	N	Y Social Skills, Adaptability	Y Social Cooperation, Social Interaction, Social Independence	Y Play subscale
Are there any questions to gauge parental worry, concern, or impairment?	Y Two questions gauge worry. They are not used in scoring	Y Open-ended question about presenting complaint	N	N	**Y** Parent rates how much the child's problems affect learning, peer relationships, and home life.

evaluated. Norms are provided for 2- to 3-year-olds and 4- to 5-year-olds, with additional breakdowns for boys and girls, as shown in Table 1.7. The BASC-2 Preschool version provides four broad scores: Internalizing Problems, Externalizing Problems, Behavior Symptoms Index, and Adaptive Skills. It is important to understand the subscales that go into scoring each of these domains because some subscales are used in multiple domain scores. More specifically, the Externalizing score is based on the Aggression and Hyperactive scales. The Internalizing Problems score is based on Anxiety, Depression, and Somatic problems. The Behavior Symptoms Index is comprised of Hyperactivity, Aggression, Depression, Withdrawal, Attention Problems, and Atypicality. Thus, the Behavior Symptoms Index includes all Externalizing subscales, only one of three Internalizing scales, and three scales not included on either the Internalizing or Externalizing Problem scores. Therefore, it is important to be very careful to interpret patterns cautiously. For example, if the Externalizing and Behavior Symptoms scores are both elevated, it will be relevant to inspect the scales within each to determine whether both of these elevations are driven primarily by Hyperactivity and Aggression problems or whether other scales such as Attention, Withdrawal, Anxiety, or Atypicality are also elevated and contributing to the high Behavior Problem Index score. Caution will need to be taken when using the BASC-2 in research applications to ensure that this overlap in scale content does not lead to misinterpretation of findings.

The Child Behavior Checklist for 1.5-5 (CBCL/1.5-5) (Achenbach & Rescorla, 2000) is a checklist measure that is a downward extension of the CBCL, originally developed for older children. The reliability and validity of the younger child version has been evaluated in children from 18 months through 5 years. The CBCL/1.5-5 addresses social-emotional/behavioral problems in three domains (Internalizing, Externalizing, and Total Problems), with more specific subscales within the Internalizing and Externalizing domains. The CBCL can also be scored to obtain *DSM*-oriented scales, which are designed to parallel symptoms that fall within specific diagnostic areas. It is worth comment that items that go into scoring the DSM-oriented scales also go into scoring the Internalizing and Externalizing Domain scores and subscale scores. Therefore, to avoid double counting individual problem behaviors, it is best to use one or the other set of scores but not both. Social-emotional competencies are not rated in

the CBCL/1.5-5. This measure also has been used internationally, including in Denmark (Kristensen, Henriksen, & Bilenberg, 2010), China (Liu, Cheng, & Leung, 2011), Taiwan (Wu et al., 2012), and Columbia (Flink et al., 2013).

The Conners Early Childhood (Conners-EC) (Conners, 2009) is a checklist measure that assesses both social-emotional and behavioral problems and developmental milestones in young children ages 2 to 6 years. Earlier forms of the Conners were developed for older children and focused on ADHD and other types of behavior problems (Conners, Sitarenios, Parker, & Epstein, 1998). The current version is extended to include anxiety, mood/affective problems, and atypical behaviors. The Conners-EC Behavioral form includes 115 parent-report and 116 teacher report items. It was normed within six-month age bands for boys and girls separately, which should enhance the developmental appropriateness and accuracy of its norms. This measure provides scores in six areas as follow: (1) Inattention/Hyperactivity; (2) Defiant/Aggressive; (3) Social Functioning/Atypical; (4) Anxiety; (5) Mood/Affective Problems; and (6) Physical Symptoms. The Conners-EC does not, however, offer broad domain scores for Internalizing or Externalizing Problems or for Social Skills/Competencies. However, the separate Developmental Milestones scales portion of the Conners-EC (eighty parent items, seventy-four teacher items) includes assessment of Play, as well as Adaptive behavior, Communication, and Motor Skills.

The Infant-Toddler Social and Emotional Assessment (ITSEA) (Briggs-Gowan & Carter, 1998; Carter & Briggs-Gowan, 2006; Carter et al., 2003) is a 169-item questionnaire about social-emotional and behavioral problems and competencies in 1- to 3-year-old children. From its inception, the ITSEA was developed for use with very young children. Items were developed based on literature review, clinical and developmental theory, and input from a large panel of experts from the fields of infant mental health, developmental psychology, and developmental psychopathology. It was developed as a comprehensive tool that would be appropriate for profiling children's strengths and weaknesses in the social-emotional domain. There are four broad domains, Internalizing, Externalizing, Dysregulation, and Competence, with multiple subscales within each domain. In addition, the ITSEA includes three indices, Social Relatedness, Atypical Behaviors, and Maladaptive. These indices consist of low base rate, but clinically significant behaviors. Reliability and validity have been established in four independent samples and in a national norming sample in the United States (Briggs-Gowan & Carter, 1998, 2008a; Carter & Briggs-Gowan, 2006; Carter et al., 2003; Carter, Little, Briggs-Gowan, & Kogan, 1999). In international samples, a number of researchers have established the ITSEA's reliability and validity in other languages and cultures, including China (Jianduan et al., 2009), the Netherlands (Visser et al., 2010), and France (Bracha et al., 2004). Additional studies have shown that the ITSEA is sensitive to treatment change (Barlow et al., 2013; Lowell, Carter, Godoy, Paulicin, & Briggs-Gowan, 2011; Spittle et al., 2010).

The Preschool and Kindergarten Behavior Scales (PKBS-2) (Merrell, 2002) is a checklist measure of problem behaviors and social skills in 3- to 6-year-olds. This 99-item measure includes identical items for different informants. The exact same form is used for parents and teachers, but different norms are applied depending on the informant. The PKBS provides a somewhat less detailed set of scores about a child relative to other measures in this section. It offers Internalizing, Externalizing, and Social Skills scores. Standard scoring provides broad scores for the Internalizing, Externalizing, and Social Skills domains, as well as specific scores for three social skills subscales, Social Cooperation, Social Interaction, and Social Independence. Although they are not described in the scoring forms for the PKBS, the manual describes scoring for several subscales within the internalizing and externalizing areas, including Anxiety/Somatic Problems, Social Withdrawal, Attention Problems/Overactive, Self-Centered/Explosive, and Antisocial/Aggressive. A full understanding of the content of these scales is important however, as some scales include items that are not clearly described by the name of the scale. For example, the Attention Problems/Overactive scale includes some items that appear to be more disruptive in nature. Given the high levels of comorbidity within specific externalizing behaviors, it is likely that these items loaded best on this scale. However, interpretation should be informed by the content of the scales.

Dimensional Checklists That Focus on Specific Areas

In addition to previously described the comprehensive measures, a number of tools focus more narrowly on specific aspects of social-emotional/behavioral problems and competencies. The Preschool Anxiety Scale (Edwards et al., 2010; Spence et al., 2001) assesses anxieties in the areas of social anxiety, generalized anxiety, separation, and specific fears. The PAS has demonstrated fair to good internal consistency and factor structure (Broeren & Muris, 2008; Edwards et al., 2010; Spence et al., 2001). However, some scales have had poor internal consistency in some studies. For example, Separation Anxiety had

poor internal consistency in Chinese and Dutch samples (Broeren & Muris, 2008; Wang, 2014). As discussed earlier, this could reflect problems with the translation, cultural variation in how items are understood, or differences in our separation anxiety manifests in those cultures. The PAS has not to our knowledge been formally normed. Another tool, the Multidimensional Profile of Disruptive Behavior (MAP-DB) (Wakschlag et al., 2014), is a new 78-item parent report measure of disruptive behavior in the areas of Aggression, Temper Loss, Low Concern for Others (disregard, punishment insensitivity), and Noncompliance. The MAP-DB has demonstrated acceptable internal consistency (Cronbach's alphas of .92 to .97) and test–retest reliability (ICCs of .76 to .85) (Nichols et al., in press; Wakschlag et al., 2014). Evidence of validity has begun to emerge (Briggs-Gowan et al., 2013; Nichols et al., in press; Wakschlag, Choi, et al., 2012). A final example is the Social Skills Improvement System (SSIS) (Gresham & Elliott, 2008), a checklist measure for children ages 3 to 18 that emphasizes the assessment of social skills but also includes some scales for assessing problems in other areas (e.g., externalizing).

Variation in Emphasis of the Domains That Are Assessed

A review of the domains and subscales assessed by these different measures, summarized in Table 1.7, reveals variation in the areas assessed. There are three primary points to note.

First, most but not all of these measures provide domain-level scores for both internalizing and externalizing problems (Table 1.7). The Conners-EC does not provide summary scores at the broad domain level. While this may be appropriate in clinical settings, it may be a disadvantage for some research applications where it is sometimes advantageous to examine patterns at the broad domain level instead of (or in addition to) at the level of multiple subscale scores.

Second, only some of these measures assess social skills/competencies and there is considerable variability within these in the types of competencies that are assessed. The BASC-2, ITSEA, and PKBS all assess social competencies and include subscales that assess specific aspects of social-emotional competence. For example, the PKBS assesses skills in the areas of social cooperation, social interaction, and social independence, the BASC captures social skills and adaptability, and the ITSEA provides scores for empathy, compliance, prosocial peer play, mastery motivation, and imitation/play. In contrast, the CBCL/1.5-5 does not assess any social competencies.

Third, there is considerable variability across these measures in their coverage of behaviors that may be associated with autism spectrum disorder. For example, the ITSEA includes indices designed to characterize these types of problems and has also demonstrated an empirical profile that is associated with autism (Carter & Briggs-Gowan, 2006). This profile is characterized by high scores on the Atypical Index and Depression/Withdrawal and low scores on the Social Relatedness Index and the Imitation/Play and Empathy. Similarly, the CBCL/1.5-5 has identified a Pervasive Developmental Delay subscale for assessing problems in this area. Other measures, such as the Conners-EC, have identified profiles that are associated with pervasive developmental delays. As a profile of specific weaknesses may be expected with children who are on the autism spectrum, a careful review of the manual is warranted to ensure appropriate interpretation of the profile of findings, is important when ASD is a consideration in assessment.

Finally, these measures differ in how deeply and specifically particular types of problems are assessed. ADHD problems provide a good example. Two measures have separate subscales for attention problems and hyperactivity/impulsivity problems, whereas the remaining measures combine these behaviors into a single score. For anxiety, most of the measures have a single subscale for assessing anxiety or combine anxiety and somatic problems; the latter approach can be problematic if working with medically ill children. The ITSEA is unique in distinguishing different types of anxiety, with scales for separation distress and general anxiety.

A final point to consider is variation in how child age and sex were addressed in the norming process. For example, the Connors-EC and ITSEA derived norms for six-month age bands for boys and girls separately and thus provide very narrow comparison groups against which children are compared. In contrast, the CBCL and PKBS employ the same norms across the entire age group for which the tool is assessed and did not derive norms separately for boys and girls. The BASC-2 used an intermediate approach, with age bands that span 24-month periods for boys and girls separately.

Diagnostic Approaches

Diagnostic interviews are designed to gather the detailed information that is needed to establish whether all of the criteria for making a particular diagnosis are met. The criteria required for a diagnosis varies across disorders, but often includes a specific number of symptoms, a period of duration, age of onset, and impairment. Although mostly

used in research settings, structured and semistructured interviews with parents or caregivers offer a more in-depth examination of child problems than can be obtained from questionnaires and can be particularly useful for helping to determine the child's diagnostic status (Egger & Angold, 2004). For example, in the PAPA interview, described in detail in the following section, interviewers can obtain specific examples of child behavior and inquire about the onset, offset, frequency, duration, intensity, quality, and contexts in which behaviors occur through follow-up questions and probes. Thus, interviews can provide a means of obtaining a more comprehensive understanding of the child's behavior and gathering information necessary to assign diagnoses.

The decision to use diagnostic interviews in the assessment process brings added challenges, including the staff and family time for the interview itself and staff time for review and scoring to establish diagnostic status. Interviews also typically require a formal training in administration and ongoing monitoring of the fidelity of administration and any ratings that are made in the scoring process to minimize drift over time. Such procedures are crucial to ensure that children are being consistently and systematically assessed in the same way. In practice, however, these standards can be difficult to uphold, especially when funding is limited or staff change over time, necessitating a system for training new staff. These issues are pronounced in early childhood because all of the interviews that are available are semistructured. Semistructured interviews provide interviewers with a clear framework for the interview, but interviewers must use their judgment about what constitutes a symptom, when to probe further to establish symptom presence, and what phrasing to use to describe a symptom (Angold et al., 2012). While the interviews vary in the background training required, from lay interviewer to clinician, the fact that interviewer judgment is employed means that training and monitoring are always essential.

The reliability of an interview provides information about how consistently the interview elicits the same information. Test–retest reliability indicates how well the interview provides the same results when administered twice at a short enough interval (typically 1–4 weeks) that true change in the child is not likely to have occurred. As it is administered by different interviewers at the two time points, test–retest reliability can be affected by differences in how deeply each interviewer probes or differences in how they perceive the information obtained even if they probe exactly the same way. Test–retest reliability can be influenced by true change in the child over time and development. Changes in how the parent perceives the child's behavior, fluctuations in the parent's attention level or mood can also affect reliability. The experience of being interviewed may influence a parent's understanding of the child's behavior. For example, they may come to realize that some behaviors that they thought were normal may be symptomatic or they may watch their child's behavior and realize it occurs more or less frequently than they thought it had at the first interview. Parents often also learn that if they deny the presence of symptoms the interview will go faster, resulting in lower scores at the second interview. Indeed, Egger and her colleagues note that attenuation (reduction) of symptoms and disorders is a common problem in test–retest studies of diagnostic interviews (Egger et al., 2006). Interrater reliability is a second type of reliability that's important for interviews. It refers to how consistently different people rate the same interview. Interrater reliability is typically assessed by having raters watch videotapes of the same interview and code it independently. It also can be assessed by having one rater administer and code the interview and the second rater code from an audio or videotape. A constraint of inter-rater reliability it does not establish how well the interview was administered, only whether the information obtained yields comparable diagnostic and symptom data when rated independently. Together, test–retest and interrater provide complementary information about reliability. Internal consistency can also be assessed for symptom scores obtained from interviews, but this is often not reported.

Selected Diagnostic Interviews

In this section, we will review and discuss four interviews that have been used with young children (Table 1.8). First we will review two interviews that were explicitly developed for early childhood: the Preschool Age Psychiatric Assessment (PAPA) (Egger & Angold, 2004; Egger et al., 2006) and the Diagnostic Infant and Preschool Assessment (DIPA) (Scheeringa, 2004; Scheeringa & Haslett, 2010). Then we will review two interviews originally developed for older children—the Anxiety Disorders Interview Schedule (ADIS) (Silverman, Saavedra, & Pina, 2001) and the Schedule for Affective Disorders and Schizophrenia – Present and Lifetime (K-SADS-PL) (Kaufman et al., 1997; Kaufman, Birmaher, Brent, Rao, & Ryan, 1996)—which have been used in studies of preschool-age children.

PAPA

The PAPA (Egger et al., 1999) was developed for parents of 2- to 5-year-olds. It is designed to provide comprehensive and developmentally sensitive coverage of the

TABLE 1.8 Diagnostic Interviews Used with Young Children

	PAPA (Egger et al., 2006)	DIPA (Scheeringa & Haslett, 2010)	K-SADS-PL Preschool (Kaufman, Birmaher, Brent, Rao, & et al., 1997)	ADIS (Silverman et al., 2001)
Age (months)	2 to 5 years	<7 years	1 to 6 years	3 to 5 years
Interviewer type	Lay or clinician	Lay or clinician	Clinician recommended	Clinician
Interview format	Semistructured	Structured and Semistructured	Semistructured	Semistructured
Training required	Yes	Yes	Yes	Yes
Respondent	Parent	Parent	Parent	Parent
Time frame covered	Past 3 months (and lifetime as appropriate)	Past 4 weeks	Past 3 months (and lifetime as appropriate)	Not specified
DSM version	*DSM-IV-TR*/RDC?	*DSM-IV*	*DSM-IV*	*DSM-IV*
Scoring	Electronic database	Hand scoring	Diagnostic consensus or by expert review	Hand scoring
Developmental modifications	Developed for young children	Developed for young children	Developed for older children. Studies vary in their developmental modification.	Developed for older children. Developmentally inappropriate questions are excluded.
Diagnostic areas covered	Common childhood disorders	Common childhood disorders	In preschoolers, reliability and validity primarily available for anxiety, ODD, CD, and ADHD	Anxiety disorders, ADHD, ODD, sleep terror disorder, selective mutism
Impairment	Impairment in multiple domains is evaluated in an incapacity module at the end of the interview	Impairment in multiple domains is evaluated at the end of each diagnostic section	Assessed in the process of assessing symptoms	Impairment in multiple domains is evaluated at the end of each diagnostic section
Test–retest reliability data	Reported (Egger et al., 2006)	Reported (Scheeringa & Haslett, 2010)	Reported for ODD/CD (Keenan et al., 2007)	Not reported for young children
Interrater reliability data	Not reported	Not reported	Reported for ODD/CD (Keenan et al., 2007) and for anxiety, ADHD, ODD, Mood (Birmaher et al., 2009)	Reported (Edwards et al., 2010; Rapee et al., 2005; Shamir-Essakow et al., 2005)
Validity data	Not explicitly studied, but various studies provide support for validity	Construct validity (Scheeringa & Haslett, 2010)	Reported for ODD/CD (Keenan et al., 2007) and for anxiety, ADHD, ODD, mood (Birmaher et al., 2009)	Not explicitly studied

symptom, frequency, duration, and onset criteria necessary to generate diagnoses for the *DSM-IV*, ICD-10, and DC: 0-3 diagnostic classification systems. In developing the PAPA, the authors conducted thorough reviews of the infant mental health literature and solicited detailed input from many infant mental health experts (Egger & Angold, 2001). The PAPA assesses a broad set of internalizing disorders, externalizing disorders, and regulatory disorders (addressing, e.g., sleep, feeding, and emotion regulation) as well as PTSD and reactive attachment disorder. At the end of the interview, an "incapacity" section is used to determine the level of impairment that is associated with symptoms of each diagnostic area. Impairment can be endorsed in several areas, including in parent–child and sibling relationships, in school or early care and education settings, and with peers. The PAPA may be administered by clinicians and laypersons, provided they have received formal PAPA training (Egger & Angold, 2004). Notably, the PAPA includes many adaptations that enhance its developmental appropriateness for young children. For example, it includes an extensive set of questions about temper tantrums, including probes to establish the types of behaviors that are manifested during tantrums (e.g., stamping feet, throwing things, holding breath), the

contexts in which they occur (home, school, elsewhere), how long they last, and whether the parent can identify triggers for tantrums (e.g., frustration, fatigue, transitions) or reports that they are out of the blue. A unique feature of the PAPA is that in determining whether symptom criteria are met, the diagnostic scoring algorithm incorporates information about the frequency of individual symptoms in a normative population. Thus, the algorithms incorporate empirical data to help distinguish normative from non-normative behaviors. Given that parents and professionals often have difficulty knowing when a particular behavior is problematic, this is a potentially valuable feature of the PAPA.

DIPA

Like the PAPA, the Diagnostic Infant and Preschool Assessment (DIPA) (Scheeringa, 2004; Scheeringa & Haslett, 2010) also covers a full range of psychiatric disorders that are common in childhood. It was designed for parents of children from infancy through 6 years of age. It is intended to provide a relatively quick and practical method for assessing psychiatric disorders. The DIPA is both structured and semistructured. It is structured in the sense that the initial stem questions about a given symptom are read verbatim, but semistructured in that the interviewer must probe for examples to establish whether symptom criteria are met. The interview is laid out in such a way that the interviewer can determine whether diagnostic criteria are met without requiring time-consuming data entry or computerized scoring. The DIPA also has incorporated a number of helpful developmental adaptations. For example, to aid the parent and interviewer in establishing whether a child's sleep patterns are symptomatic, the sleep disorders section incorporates empirical information for children of different ages (12 to 24 months versus 24 months and older) concerning how long children in this age group typically take to fall asleep and how often they typically waken during the night.

Two interviews that were originally developed for older children, the ADIS (Silverman et al., 2001) and the K-SADS-PL (Kaufman et al., 1996, 1997) have been used in studies of preschool-age children (Birmaher et al., 2009; Keenan et al., 2007; Kennedy, Rapee, & Edwards, 2009; Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2005; Shamir-Essakow, Ungerer, & Rapee, 2005). Because they were developed for older children, in their original forms, these interviews include some *DSM* symptom criteria and descriptors that are developmentally inappropriate for young children, such as sexually inappropriate touching, vandalism, and stealing (Wakschlag et al., 2010). Most studies that have used the ADIS and K-SADS with young children have simply excluded these developmentally inappropriate symptoms from the interview entirely (Birmaher et al., 2009; Shamir-Essakow et al., 2005). However, this approach may miss the opportunity to assess these symptoms in a developmentally meaningful and valid way. For example, Wakschlag and Keenan have shown that many disruptive behavior symptom criteria of the K-SADS can be operationalized in a developmentally appropriate way for preschool-age children. When they used the K-SADS disruptive behavior modules with preschool children, they excluded inappropriate items (e.g., forced sexual contact) and modified definitional examples of symptom criteria where developmentally relevant (Keenan et al., 2007). For example, an exemplar for "often bullies" could include threatening aggression to get a toy (Gray & Wakschlag, in press). As interviews vary in how they have handled these issues of developmental relevance, it is important to review the context of an interview carefully when deciding whether it suits your goals and when interpreting results.

Psychometric Properties of Diagnostic Interviews

A review of the literature reveals unevenness across interviews in the types of reliability that have been emphasized and reported. For example, test–retest reliability has been systematically assessed for the PAPA and DIPA, has been reported for only Disruptive Behavior Disorders in the K-SADS, and has not been reported for the ADIS. In contrast, interrater reliability has been reported in multiple studies that have used the ADIS with preschoolers.

Test–Retest Reliability

Our literature review identified studies that have reported test–retest reliability statistics in young children for the PAPA, DIPA, and the K-SADS Disruptive Behavior Disorders (K-DBDs) but not for the ADIS. As summarized in Table 1.9, this review suggests that test–retest reliability is generally better for symptom scores than for presence versus absence of disorder. The poorer reliability for disorders relative to symptoms may be explained by the fact that diagnostic status is less forgiving of modest change. For children whose difficulties fall right near the threshold for disorder, a minor change in number of symptoms can dramatically change diagnostic classification (from present to absent or vice versa) but have minimal impact on symptom scores. A detailed review of each interview is provided in the following sections.

TABLE 1.9 Test–Retest Reliability of Diagnostic Interviews Used with Young Children

	PAPA (Egger et al., 2006)		DIPA (Scheeringa & Haslett, 2010)		K-SADS (Keenan et al., 2007)	
	Disorders (Kappa) Normative Sample/ Screen Highs	Symptom Scores (ICCS)	Disorders (Kappa) With/ Without Impairment	Symptom Scores (ICCS)	Disorders (Kappa)	Symptom Scores (ICCS)
INTERNALIZING	**Poor to Excellent**	**Good**	**Fair to Good**	**Fair to Excellent**		
SAD	.60/.52	.63/.68	.50/.44	.78	-	-
GAD	.39/.34	.61/.61	Too rare	.41	-	-
Specific phobias	.36/.49	.57/.51	-	-	-	-
Social phobia	.54/.63	.73/.75	-	-	-	-
PTSD	.73/.79	.56/.71	.56/.67	.87	-	-
MDD	.72/.61	.71/.69	Too rare	.40	-	-
EXTERNALIZING	**Fair to Good**	**Good to Excellent**	**Fair to Good**	**Good to Excellent**	**Good to Excellent**	**Excellent**
ADHD	.74/.67	.8	-	-	-	-
ADHD–inattentive	-	-	.58/.78	.73	-	-
ADHD–hyperactive	-	-	.42/.47	.65	-	-
ODD	.57/.44	.67	.38/.47	.78	.81	.98
CD	.60/.54	.66	Not studied	Not studied	.73	.81

Note: SAD=Separation Anxiety Disorder; GAD=Generalized Anxiety Disorder; PTSD=Post-Traumatic Stress Disorder; MDD=Major Depressive Disorder; ADHD=Attention-Deficit/Hyperactivity Disorder; ODD=Oppositional Defiant Disorder; CD=Conduct Disorder

PAPA. The reliability of the PAPA was examined in a sample of 2- to 5-year-old children who were recruited from a normative, population-based sample (Egger et al., 2006). To ensure the presence of sufficient psychopathology to evaluate the PAPA's reliability, children with high scores on the CBCL/1.5-5 were oversampled. Reliability is reported both for these screen-high children, as an estimate of reliability in clinical populations, and in a subsample that was more similar to the original normative sample as an estimate of reliability in nonreferred populations. The PAPA generally demonstrated good test–retest reliability for disorders (Table 1.9). Of 18 kappas reported, nine fell in the "Good" range (.60 to .74) and six were "Fair." Three of eight kappas for anxiety disorders were "Poor." In discussing this low reliability, Egger suggested that this could reflect poor reliability of *DSM* anxiety disorders in young children (Egger et al., 2006). All symptom count variables were reported to have good to excellent reliability. As noted by Egger and her colleagues (2006), the test–retest reliability of the PAPA is comparable to what has been reported for diagnostic interviews of older children and adolescents.

DIPA. The test–retest reliability of the DIPA was evaluated in a sample of 50 outpatient children under the age of 7 (Scheeringa & Haslett, 2010). Review of the DIPA revealed generally fair to good reliability for separation anxiety, PTSD, ADHD, and Oppositional Defiant Disorder (Table 1.9). However, reliability could not be adequately assessed for other disorders because some disorders occurred too rarely to estimate reliability; a rule of thumb is that reliability should be estimated only if there are at least five children with the disorder in the sample. Specific and social phobias were not assessed due to time constraints and concern about burden on families and interviewers in this clinical sample. At the symptom level, similar to the PAPA, reliability for assessing ODD and ADHD symptoms was good to excellent. Reliability was excellent for Separation Anxiety and Post-traumatic Stress symptoms but fair for symptoms of Major Depression and Generalized Anxiety.

K-SADS. The test–retest reliability of the K-DBDs was evaluated in a study of 223 clinically referred and nonreferred children, ages 3 to 5 years (Keenan et al., 2007). The focus of this study was specifically disruptive behavior disorders. Test–retest reliability statistics for ODD and conduct disorder (CD) and symptoms were good to excellent (Table 1.9). To our knowledge, test–retest reliability has not been reported for other disorders assessed by the K-SADS in preschoolers.

Interrater Reliability

Much less information is available about the interrater reliability of these diagnostic interviews. A number of studies that have used the ADIS with preschool samples have

reported its interrater reliability in assessing presence or absence of anxiety disorders (Edwards et al., 2010; Rapee et al., 2005; Shamir-Essakow et al., 2005). Across these three studies, interrater reliability was consistently good to excellent for separation anxiety (K = .69 to .89), specific phobias (K = .69 to .83), and social phobia (K = .84 to .89). However, interrater reliability of generalized anxiety disorder has been only fair in two studies (K = .54 to .56) and excellent in one (K = .77). For the K-SADS-DBDS, Keenan and her colleagues (2007) reported excellent interrater reliability for ODD and CD symptoms (both ICCs = .98) and disorders (K = .96 and .98, respectively). Finally, although specific details were not provided for individual disorders, Birmaher and colleagues reported that the K-SADS demonstrated excellent interrater reliability for all disorders (all kappas >.80) in a study of preschool-age children (Birmaher et al., 2009).

Validity

Validity of diagnostic interviews in young children has rarely been tested explicitly. As a consequence, their validity in properly classifying children as having or not having psychiatric disorders is essentially unknown. This is at least partially a reflection of an inherent challenge in defining an appropriate gold standard for evaluating the validity of psychiatric diagnoses. While clinician-assigned diagnoses might be considered for this purpose, they can have poor reliability (Fisher et al., 1993). Thus, poor validity could arise from unreliability of the clinician diagnoses rather than a limitation of the interview. One approach to validate these measures is to demonstrate expected associations with checklist measures. This approach validates that the interview captures elevations in symptoms—it cannot validate that the diagnosis itself is valid because checklists cannot determine the presence or absence of disorder explicitly because they do not evaluate diagnostic criteria. Another approach is to demonstrate concordance between the interview and overall clinical status, that is that children referred for treatment are more likely to meet criteria for disorder than healthy controls.

The DIPA is unique in that Scheeringa and colleagues explicitly evaluated the validity of the continuous symptom scores in relation to the CBCL 1.5-5 (Scheeringa & Haslett, 2010). Results of these analyses indicated good validity for the externalizing symptom scores and for separation anxiety, with correlations from .44 to .59, but generally low validity for internalizing problems, such as generalized anxiety and depression, with most correlations below .25. However, this might be as much a limitation of the CBCL as it is of the DIPA. For the K-SADS-DBDS, ODD and CD diagnoses demonstrated good reliability relative to measures of impairment and referral status (Keenan et al., 2007). To our knowledge, the validity of the PAPA, ADIS, and other diagnostic areas of the K-SADS have not been studied explicitly.

Nonetheless, as researchers employ these interviews in their studies, we have opportunities to gain insights into validity. Various studies that have used these interviews suggest their value for characterizing preschool psychopathology. For example, the PAPA, ADIS, and K-SADS all have been used in multiple preschool studies for a variety of purposes, including as a means of validating another tool (Edwards et al., 2010; Wakschlag et al., 2008) and of documenting treatment outcomes (Comer et al., 2012; Rapee et al., 2005) and for characterizing psychopathology in young children (Briggs-Gowan et al., 2010; Bufferd, Dougherty, Carlson, & Klein, 2011; Shamir-Essakow et al., 2005; Sterba, Egger, & Angold, 2007). For example, one recent study demonstrated greater impairment in young children with anxiety or depressive disorders identified by the PAPA relative to healthy children without disorders (Otto, Andreas, von Klitzing, Fuchs, & Klein, 2014). Another demonstrated the predictive validity of irritability, assessed with the PAPA, for predicting later depression, oppositional defiant disorder, and functional impairment (Dougherty et al., 2013). Longitudinal prediction from PAPA diagnosis to school-age peer impairment also has been reported (Danzig et al., 2013). Yet another demonstrated significant functional impairment in children who meet criteria for disorders on the PAPA (Bufferd et al., 2011). Treatment studies that have used the ADIS to assess anxiety disorders with preschool-age children have been similarly fruitful, with evidence that the ADIS is sensitive to treatment effects on anxiety disorders (Kennedy et al., 2009; Rapee et al., 2005). While these studies do not explicitly demonstrate the validity of these interviews, they do suggest their value for studying emergent symptoms and disorders in this developmental period.

It is worth commenting that a consistent emerging pattern is that generalized anxiety disorder is less reliably assessed, with lower test–retest and interrater reliability in most studies. This may reflect a need for further research to establish the validity of this type of anxiety in young children. This type of anxiety also may be difficult to assess in young children because their more limited ability to verbalize their internal states may mean that parents and other caregivers are less consistently aware of worrying and apprehension in this age group.

Observational Assessment

Observation is routinely used to assess the quality of individual, dyadic, triadic, and family functioning in young child assessments. These methods include two components: the procedures that are used to create the context for the observational episodes, and the coding system designed to quantify the elements of interest. Interactional episodes vary along a spectrum from naturalistic observation to structured interactions. Coding systems vary from micro-analytic coding of discrete behaviors across very brief time segments of interaction to global coding that requires appraisal of multiple components of the interaction across the entire duration of the observation.

A number of methods for assessing child temperament originated from developmental psychology laboratories. These include behavioral inhibition paradigms in which children are observed in situations in which they are exposed to novel objects and social situations (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984; Reznick et al., 1986). The more extensive Laboratory Temperament Assessment Battery (Gagne, Van Hulle, Aksan, Essex, & Goldsmith, 2011) is a series of 3–5-minute naturalistic episodes that include the introduction of specific presses designed to capture variation in emotional expression, activity level, approach and avoidance, and other regulatory behaviors. Other measures focus on dyadic or triadic interactions. For example, the Parent-Child Early Relational Assessment (PCERA) is designed to assess individual child and parent affective and behavioral patterns and dyadic behaviors in both unstructured free play episodes and in more structured interactions (e.g., reading a book, doing a mildly difficult task) Clark, Tlucek, & Gallagher, 2004. Observations of triadic interactions, in which aspects of caregivers, cooperation, competition, and conflict are rated in relation to a particular child, have become more common in recent years (McHale & Coates, 2014; McHale & Fivaz-Depeursinge, 1999). The Disruptive Behavior Diagnostic System (DB-DOS) (Wakschlag et al., 2007, 2008) is an example of an observational paradigm and coding system that were developed to characterize children's functioning on a clinical continuum from normal to atypical. The interactions include parent–child and examiner–child interactions. Building on methods used in the area of ASD (Lord et al., 1989, 2000), each interactional context is designed to press for the full range of behaviors relevant to preschool disruptive behavior. For example, episodes include a don't touch shelf, a (presumably boring) sorting task, and a frustrating puzzle. As the presses build across the contexts, they can elicit valuable insight into how a child responds when stressed or frustrated. For example, providing qualitative and quantitative information about the intensity, pervasiveness, and ease of escalation of negative affect as well as whether it is displayed with both the parent and the examiner. We are have recently developed and reported the preliminary reliability and validity of an extension of this method to anxiety, the Anxiety Dimensional Observation System (Anx-DOS) (Briggs-Gowan, Mian, Carter, & Wakschlag, 2011; Mian et al., in press).

Observational methods such as these offer an important means of capturing independent information about a child's functioning. In practice, however, they are typically costly and burdensome to implement. To be valid, the observational paradigm must be administered in a standard fashion. Thus staff must be well trained, and there should be ongoing quality control checks to ensure that they continue to administer the protocol reliably over time. Similarly, observational coders must be trained to apply the coding system reliably, and their reliability must be maintained over time. The process of training a coder to the point of acceptable reliability can require many hours of coding and feedback with an expert coder. Once a coder is reliable, ongoing monitoring of reliability is essential to prevent drift over time. Consequently, standardized, coded observational approaches are primarily used in research settings. For coding of discrete behaviors, new technological advances are developing in which computers and computer software can assume some of the burden of coding; for example, improvements in face recognition and machine learning enabling coding of discrete emotions such as pain (Lucey et al., 2011).

Finally, along with observation, several researchers have developed methods for directly eliciting information about symptomatology from children. A variety of measures have been developed focused on the free responses of children to story stems, puppet interactions (Measelle, Ablow, Cowan, & Cowan, 1998; Warren, Emde, & Sroufe, 2000; Warren, Oppenheim, & Emde, 1996), and simple picture-based questionnaire responses (Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1993, 1995; Martini, Strayhorn, & Puig-Antich, 1990). However, in relation to other diagnoses, the questions of whether and how to incorporate observational measures, or such material elicited directly from the child, have not been settled.

Assessing Impairment

As in older children and adults, social-emotional and behavioral problems in young children can be associated with impairment in functioning (Briggs-Gowan &

Carter, 2008a; Briggs-Gowan et al., 2001, 2006; Egger & Angold, 2006; Keenan et al., 2007; Lavigne et al., 1996; Luby, Belden, Pausch, et al., 2009). Symptoms of psychopathology can interfere with a child's participation in age-appropriate activities, developmental progress or relationships with others. When evaluating impairment in a young child, it is important to consider how the child's developmental capacities in domains such as cognition, motor, or language affect functional impairment. Delays in other areas, such as language development, may impede progress in other domains. Consider for example, the challenges faced by a child with mixed language delays and gross motor delays when attempting to keep up with peers. It is important that challenges in functioning associated with the mixed language and gross motor delays not be attributed to the symptoms of psychopathology that the child may also be evidencing. Difficulties being understood by and understanding peers may impede opportunities to learn age-appropriate prosocial skills acquired through give and take with peers. Difficulties running and climbing may interfere with his efforts to join gross motor peer play, contribute to isolation, and interfere with the development of peer relationships. Thus, assessing impairment associated with psychopathology requires consideration of developmental trajectories in multiple domains and their impact on children's performance of daily routines and activities.

In older children and adults, impairment is typically assessed by evaluating the individual's interpersonal, academic, occupational, and adaptive functioning (Association, 2000; Organization, 2001). In contrast, working with young children necessitates shifting the emphasis from the child only to encompass both the child and the broader family system.

Very young children's functioning is dependent on parental scaffolding and support. Impairment may manifest in the extent to which parents make accommodations that ultimately impact their child's participation in age-appropriate activities or developmental progress. For example, although a parent may not be able to offer examples of *child* impairment, *the parent* may have quit his or her job and restructured the household to accommodate their child's special needs or social-emotional problems. The parent may also avoid situations that would trigger their child's problem behavior. For example, a parent with a very active child may refrain from taking her even to family-friendly restaurants or a parent with a socially anxious child may avoid even child-friendly activities in the community. Such minimization of the demands placed on the child may help to obscure the child's impairment (Clark et al., 2004) while at the same time limiting the child's ability to engage in and learn from age-appropriate activities and likely further compounding the problem. When conceptualized within a broader family framework, young child impairment may manifest as parental distress or low efficacy about the parent–child relationship or evidence that the child's behavior interferes with the parent's ability to maintain family routines (e.g., have family meals together, visit relatives), household activities (e.g., making phone calls), or employment (e.g., restrictions on work due to the challenges of obtaining appropriate care for the child) (Clark et al., 2004).

Thus, when evaluating functional impairment, it is often useful to consider how a child's social-emotional/behavioral problems may limit the child's participation in age-appropriate activities such as learning activities (e.g., listening to books, drawing, building); opportunities to acquire and practice social skills with peers and nonfamilial adults; and exposure to challenging situations key to practicing and acquiring self-regulation skills. In parallel with this, it may be very illuminating to query how the child's problems may strain the parent–child relationship, cause distress for child, parent, or other family members, interfere with family routines (meals, bedtime, getting ready for the day), impact siblings, limit parental employment, or be perceived as affecting parental well-being (e.g. lack of sleep, worry, fatigue) or straining family relationships (between parents or in the broader family context).

Many available assessments now include some gauge of impairment in child or family functioning, as noted in the aforementioned summary of assessments. In addition to these, a number of measures offer a means of gathering information about the impact of the child's behavior on family functioning, such as the Parenting Stress Index (Abidin, 1990) and the Family Life Impairment Scale (Briggs-Gowan, Horowitz, & Carter, 1998; Briggs-Gowan et al., 2006; Carter et al., 2010). Measures of adaptive behavior, such as various forms of the Vineland Adaptive Behavior Scales II (Sparrow, Cicchetti, & Balla, 2005) and the Adaptive Behavior Assessment System (ABAS-II) (Harrison & Oakland, 2003) are norm-referenced tools that provide comprehensive assessment of a child's adaptive functioning. Adaptive functioning that is significantly lower than cognitive functioning often reflects the child's inability to participate in age-expected activities or attain developmentally expected competencies. Thus, adaptive functioning may represent one important index of impairment.

CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH

There has been tremendous progress in developing new instruments that enhance assessment of young children's social-emotional and behavior problems and disorders. Specifically, an array of instruments now exist: parent-reported questionnaires are available for screening and more comprehensive assessments of social-emotional and behavior problem and competence dimensions; diagnostic interviews are available for parents of children 2 years and older; and observational assessments of temperament and specific symptoms (e.g., disruptive behaviors) have been shown to deepen understanding of children's development in relation to their peers. Yet, despite these efforts and successes, many challenges remain. First, integrating data across informants and methods continues to be a significant challenge in both clinical and research settings, particularly given the frequent occurrence of low correlations between informants and methods (i.e., questionnaires, interviews, and observations). Given that young children do not have the metacognitive capacities to report on their own symptoms until approximately 4 years of age (and then only when using developmentally appropriate interviews such as the Berkeley Puppet Interview (Measelle et al., 1998), greater variability in caregiver expectations for child behavior among very young children, meaningful variability in children's behavior in different contexts (Dirks et al., 2012), and lack of consistency in documenting impairment among young children, recommendations or guidelines for systematic and valid approaches for integrating data would be a major contribution to the field. Recommendations should be based on empirically supported methods for integrating of multimethod, multi-informant assessment data within the domain of social-emotional and behavior problems and disorders that often yield competing views of the child. Moreover, such methods should also address children's developmental functioning and contextual factors such as caregiver–child relationships, family functioning, culture, and broader contextual factors that may shape the behaviors and emotions that parents and other informants notice and the meanings that they attribute to these behaviors and emotions.

Second, with an increased push for screening children for social-emotional and behavioral problems early and often, it becomes imperative to know how repeated administration of the same instrument may influence psychometric properties over time. One could easily hypothesize attenuation effects due to repeated screening as parents pay less attention to items, or one could hypothesize increased rates of reporting as parents become familiar with items and may begin to pay more careful attention to the child behaviors associated with screening items. Repeated screening also offers an opportunity for developing new methods for determining screening thresholds. Specifically, it is plausible that a child whose screening score is at or is one point above the single time-point threshold score on a screening instrument should carry more weight in relation to follow-up assessment or referral if similarly elevated scores have been obtained on two earlier occasions and less weight if this is the first occurrence of a score that just exceeds the cut score that is routinely employed for this age group. Underscoring the potential value of new methods for integrating longitudinal screening information, emerging research has shown that repeated assessments of irritability enhance prediction of impairment in preschoolers (Wakschlag et al., in press).

Third, with increased global diversity, there is a striking need to improve the availability of social-emotional and behavioral measures that have culturally valid content and offer representative normative data that are appropriate to the diversity that currently exists within many countries. This work will likely require international collaborative efforts using mixed methods approaches (i.e., integrated qualitative and quantitative studies) to aid instrument developers to serve the diverse children and families whose children may be at risk for or exhibiting elevated social-emotional and behavioral symptoms or disorders. Excellent translations of instruments are insufficient to meet this challenge. We need information about the psychometric properties of widely used measures in the many cultural groups in which we are currently employing translated tools. Indeed, there may be opportunities to capitalize on ongoing large-scale longitudinal epidemiologic studies in various countries to establish cross-national norms and validation. Moreover, given the diversity within individuals who share a common language, in clinical settings it is always essential to follow up with parents to learn more about the meanings that they are attributing to screening and comprehensive assessment questions and the ways they see their children's behaviors transacting with family values, goals, and the child's broader contextual caregiving environment.

Fourth, the National Institute of Mental Health's new framework for advancing mental health research, the Research Domain Criteria (RDoC), emphasizes the importance of dimensional assessments that characterize problems along a normal to abnormal spectrum and chart the developmental trajectories of functioning over the

course of childhood (Casey et al., 2014; Cuthbert & Insel, 2013; Insel et al., 2010). A core goal of the RDoC initiative is to employ methods that strengthen efforts to link phenotypic manifestations with underlying biological processes (e.g., neural pathways, psychophysiology, genetic risk). Measures that capture discrete, narrowly specified aspects of mood or behavior (e.g., irritability, fear responses) will advance these goals. Such narrowband approaches have proven to be effective in genetics research (Krueger, 1999; Krueger et al., 2002; Tackett et al., 2013; Todd et al., 2005). In our recent work with preschoolers, narrowband dimensional assessments have proven to be effective for identifying linkages with neurocognitive indicators of risk—with evidence that mother-reported difficulty learning from punishment predicted task-based indicators of impairment in learning from punishment (Briggs-Gowan et al., 2013) and observed fear reactions in the Anx-DOS correlated with attention bias to threat, an attentional pattern commonly associated with anxiety (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van, 2007; Briggs-Gowan et al., in press). However, methods for integrating dimensional assessments of social-emotional and behavioral functioning with other methods of characterizing neurobiologic risk will be essential to fully capitalize on these innovative technologies.

Fifth, there is increasing knowledge about the impact of adverse environments and stress on young children (Cicchetti & Toth, 1995; Curtis & Cicchetti, 2011; Finkelhor, Turner, Shattuck, & Hamby, 2013; Finkelhor, Vanderminden, Turner, Shattuck, & Hamby, 2014; Shonkoff & Phillips, 2000; Shonkoff et al., 2012). There is particularly strong evidence that maltreatment and other forms of violence within families are associated with a wide array of social-emotional and behavioral problems in young children. However, we do not know why there is so much heterogeneity in the outcomes associated with apparently similar adverse experiences, ranging from no elevations in social-emotional problems to a heterogeneous set of frank, persistent, impairing, and often comorbid psychiatric disorders. As we become more sophisticated about enriching environments for children with specific social-emotional and behavioral deficits, delays and problems in the forms of early parent–child and child interventions, we need to ensure that the measures we are using are sufficiently sensitive to both maturational and intervention effects.

Given the increased attention to and understanding of the importance of early childhood in laying the foundation for lifelong mental health, it is a very exciting time to enhance the use of evidence-based psychometrically sound, validated tools. Such tools have been and will continue to be essential for early identification and comprehensive assessment. These also have enabled the scientific community to demonstrate the early problems matter, are persistent and predict functioning later in childhood and drive policy change. While there is still a great deal of work to be done, the extensive attention to measurement development in this developmental period over the past 20 years has enabled a sea of change in understanding of and attention to very young children's mental health needs.

REFERENCES

Abidin, R. R. (1990). *Parenting Stress Index Short Form-Test manual*. Charlottesville, VA: Pediatric Psychology Press.

Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs*, *80*(7), 1–37.

Achenbach, T. M., Edelbrock, C., & Howell, C. T. (1987). Empirically based assessment of the behavioral/emotional problems of 2- and 3-year-old children. *Journal of Abnormal Child Psychology*, *15*, 629–650.

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*(2), 213–232.

Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA preschool forms and profiles*. Burlington, VT: University of Vermont.

Alvidrez, J. (1999). Ethnic Variations in Mental Health Attitudes and Service Use Among Low-Income African American, Latina, and European American Young Women. *Community Ment Health J*, *35*(6), 515–530. doi: 10.1023/A:1018759201290

Angold, A., Erkanli, A., Copeland, W., Goodman, R., Fisher, P. W., & Costello, E. J. (2012). Psychiatric diagnostic interviews for children and adolescents: A comparative study. *Journal of the American Academy of Child & Adolescent Psychiatry*, *51*(5), 506–517. doi: 10.1016/j.jaac.2012.02.020

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: Text revision DSM-IV-TR* (Fourth ed.). Washington, DC: American Psychiatric Association.

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van, I. M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. *Psychological Bulletin*, *133*(1), 1–24. doi: 10.1037/0033-2909.133.1.1

Barlow, A., Mullany, B., Neault, N., Compton, S., Carter, A., Hastings, R., …, Walkup, J. T. (2013). Effect of a paraprofessional home-visiting intervention on American Indian teen mothers' and infants' behavioral risks: A randomized controlled trial. *American Journal of Psychiatry*, *170*(1), 83–93. doi: 10.1176/appi.ajp.2012.12010121

Bartko, J. J. (1976). On the various intraclass correlation reliability coefficients. *Psychological Bulletin*, *83*, 762–765.

Ben-Sasson, A., Carter, A. S., & Briggs-Gowan, M. J. (2009). Sensory over-responsivity in elementary school: Prevalence and social-emotional correlates. *Journal of Abnormal Child Psychology*. doi: 10.1007/s10802-008-9295-8

Ben-Sasson, A., Soto, T. W., Heberle, A. E., Carter, A. S., & Briggs-Gowan, M. J. (2014). Early and concurrent features of ADHD and sensory over-responsivity symptom clusters. *Journal of Attention Disorders*. doi: 10.1177/1087054714543495

Birmaher, B., Ehmann, M., Axelson, D. A., Goldstein, B. I., Monk, K., Kalas, C., … Brent, D. A. (2009). Schedule for affective disorders and schizophrenia for school-age children (K-SADS-PL) for the assessment of preschool children—A preliminary psychometric study. *Journal of Psychiatric Research*, *43*(7), 680–686. doi: 10.1016/j.jpsychires.2008.10.003

Bracha, Z., Perez-Diaz, F., Gerardin, P., Perriot, Y., de la Rocque, F., Flament, M., … Carter, A. S. (2004). A French adaptation of the Infant–Toddler Social and Emotional Assessment. *Infant Mental Health Journal*, *25*(2), 117–129. doi: 10.1002/imhj.10090

Bridgett, D. J., Burt, N. M., Laake, L. M., & Oddi, K. B. (2013). Maternal self-regulation, relationship adjustment, and home chaos: Contributions to infant negative emotionality. *Infant Behavior and Development*, *36*(4), 534–547. doi: 10.1016/j.infbeh.2013.04.004

Briggs-Gowan, M., & Carter, A. S. (2006). *Brief Infant Toddler Social Emotional Assessment (BITSEA)*. San Antonio, TX: Pearson.

Briggs-Gowan, M., Horowitz, S., & Carter, A. (1998). *The Family Life Impairment Scale*. New Haven, CT: Yale University Press.

Briggs-Gowan, M. J., & Carter, A. S. (1998). Preliminary acceptability and psychometrics of the Infant-Toddler Social and Emotional Assessment (ITSEA): A new adult-report questionnaire. *Infant Mental Health Journal*, *19*(4), 422–445.

Briggs-Gowan, M. J., & Carter, A. S. (2008a). Applying the Infant-Toddler Social & Emotional Assessment (ITSEA) and Brief-ITSEA in early intervention. *Infant Mental Health Journal*, *28*, 564–583.

Briggs-Gowan, M. J., & Carter, A. S. (2008b). Social-emotional screening status in early childhood predicts elementary school outcomes. *Pediatrics*, *121*(5), 957–962. doi: 121/5/957 [pii]10.1542/peds.2007-1948

Briggs-Gowan, M. J., Carter, A. S., Bosson-Heenan, J., Guyer, A. E., & Horwitz, S. M. (2006). Are infant-toddler social-emotional and behavioral problems transient? *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(7), 849–858. doi: 10.1097/01.chi.0000220849.48650.59

Briggs-Gowan, M. J., Carter, A. S., Clark, R., Augustyn, M., McCarthy, K. J., & Ford, J. D. (2010). Exposure to potentially traumatic events in early childhood: differential links to emergent psychopathology. *Journal of Child Psychology and Psychiatry*, *51*(10), 1132–1140. doi: 10.1111/j.1469-7610.2010.02256.x

Briggs-Gowan, M. J., Carter, A. S., & Ford, J. D. (2011). Parsing the effects violence exposure in early childhood: Modeling developmental pathways. *Journal of Pediatric Psychology*, *37*(1), 11–22. doi: 10.1093/jpepsy/jsr063

Briggs-Gowan, M. J., Carter, A. S., McCarthy, K., Augustyn, M., Caronna, E., & Clark, R. (2013). Clinical validity of a brief measure of early childhood social-emotional/behavioral problems. *Journal of Pediatric Psychology*, *38*(5), 577–587. doi: 10.1093/jpepsy/jst014

Briggs-Gowan, M. J., Carter, A. S., Skuban, E. M., & Horwitz, S. M. (2001). Prevalence of social-emotional and behavioral problems in a community sample of 1- and 2-year-old children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *40*(7), 811–819.

Briggs-Gowan, M. J., Ford, J. D., Fraleigh, L., McCarthy, K., & Carter, A. S. (2010). Prevalence of exposure to potentially traumatic events in a healthy birth cohort of very young children in the northeastern United States. *Journal of Traumatic Stress*, *23*(6), 725–733. doi: 10.1002/jts.20593

Briggs-Gowan, M. J., Horwitz, S. M., Schwab-Stone, M. E., Leventhal, J. M., & Leaf, P. J. (2000). Mental health in pediatric settings: Distribution of disorders and factors related to service use. *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*(7), 841–849.

Briggs-Gowan, M. J., Mian, N., Carter, A. S., & Wakschlag, L. S. (2011). *The Anxiety Dimensional Observation Schedule (Anx-DOS), Version 1.0*. Chicago, IL: Northwestern University Press.

Briggs-Gowan, M. J., Nichols, S. R., Voss, J., Zobel, E., Carter, A. S., McCarthy, K. J., … Wakschlag, L. S. (2013). Punishment insensitivity and impaired reinforcement learning in preschoolers. *Journal of Child Psychology and Psychiatry*. doi: 10.1111/jcpp.12132

Briggs-Gowan, M. J., Owens, P. L., Schwab-Stone, M. E., Leventhal, J. M., Leaf, P. J., & Horwitz, S. M. (2003). Persistence of psychiatric disorders in pediatric settings. *Journal of the American Academy of Child & Adolescent Psychiatry*, *42*(11), 1360–1369. doi: 10.1097/01.CHI.0000084834.67701.8a

Briggs-Gowan, M. J., Pollak, S. D., Grasso, D., Voss, J., Zobel, E., Wakschlag, L. S., & Pine, D. S. (in press). Attention bias and anxiety in young children exposed to family violence *Journal of Child Psychology and Psychiatry*.

Broeren, S., & Muris, P. (2008). Psychometric evaluation of two new parent-rating scales for measuring anxiety symptoms in young Dutch children. *Journal of Anxiety Disorders*, *22*, 949–958.

Brown, G., Marshall, M., Bower, P., Woodham, A., & Waheed, W. (2014). Barriers to recruiting ethnic minorities to mental health research: a systematic review. *International Journal of Methods in Psychiatric Research*, *23*(1), 36–48.

Bufferd, S. J., Dougherty, L. R., Carlson, G. A., & Klein, D. N. (2011). Parent-reported mental health in preschoolers: Findings using a diagnostic interview. *Comprehensive Psychiatry*, *52*(4), 359–369. doi: 10.1016/j.comppsych.2010.08.006

Campbell, S. B. (1995). Behavior problems in preschool children: A review of recent research. *Journal of Child Psychology & Psychiatry*, *36*, 113–149.

Carlson, C. I. (1990). Assessing the family context. In C. R. R. R. W. Kamphaus (Ed.), *Handbook of psychological and educational assessment of children: Personality, behavior, and context* (pp. 546–575). Guilford Press. New York, NY.

Carter, A. S., & Briggs-Gowan, M. J. (2005). *ITSEA/BITSEA Infant Toddler and Brief Infant Toddler Social Emotional Assessment examiner's manual*. San Antonio, TX: Harcourt Assessment, Inc.

Carter, A. S., & Briggs-Gowan, M. J. (2006). *ITSEA Infant-Toddler Social and Emotional Assessment examiner's manual*. San Antonio, TX: Harcourt Assessment, Inc.

Carter, A. S., Briggs-Gowan, M. J., Jones, S. M., & Little, T. D. (2003). The Infant-Toddler Social and Emotional Assessment (ITSEA): Factor structure, reliability, and validity. *Journal of Abnormal Child Psychology*, *31*(5), 495–514.

Carter, A. S., Little, C., Briggs-Gowan, M. J., & Kogan, N. (1999). The infant-toddler social and emotional assessment (ITSEA): Comparing parent ratings to laboratory observations of task mastery, emotion regulation, coping behaviors and attachment status. *Infant Mental Health Journal*, *20*(4), 375–392.

Carter, A. S., Marakovitz, S., & Sparrow, S. S. (2006). Comprehensive psychological assessment: A developmental psychopathology approach for clinical and applied research. In D. Cicchetti. & D. J. Cohen (Ed.), *Developmental Psychopathology* (2nd ed., Volume I: Theory and Method, pp. 181–210). New York: John Wiley.

Carter, A. S., Wagmiller, R. J., Gray, S. A., McCarthy, K. J., Horwitz, S. M., & Briggs-Gowan, M. J. (2010). Prevalence of DSM-IV disorder in a representative, healthy birth cohort at school entry: Sociodemographic risks and social adaptation. *Journal of the American Academy of Child & Adolescent Psychiatry*, *49*(7), 686–698. doi: 10.1016/j.jaac.2010.03.018

Casey, B. J., Oliveri, M. E., & Insel, T. (2014). A neurodevelopmental perspective on the research domain criteria (RDoC) framework. *Biological Psychiatry*, *76*(5), 350–353. doi: 10.1016/j.biopsych.2014.01.006

Cicchetti, D., & Toth, S. L. (1995). A developmental psychopathology perspective on child abuse and neglect. *Journal of the American Academy of Child & Adolescent Psychiatry*, *34*(5), 541–565. doi: 10.1097/00004583-199505000-00008

Cicchetti, D. V. (1994). Guidelines, criteria, and rules of thumb for evaluating normed and standardized assessment instruments in psychology. *Psychological Assessment*, *6*, 284–290. doi: 10.1037/1040-3590.6.4.284

Cicchetti, D. V., & Sparrow, S. S. (1981). Developing criteria for establishing interrater reliability of specific items: Applications to assessment of adaptive behavior. *American Journal of Mental Deficiency*, *86*, 127–137.

Cicchetti, D. V., Volkmar, F., Klin, A., & Showalter, D. (1995). Diagnosing autism using ICD-10 criteria: A comparison of neural networks and standard multivariate procedures. *Child Neuropsychology*, *1*(1), 26–37.

Clark, R., Tluczek, A., & Gallagher, K. (2004). Assessment of Parent-Child Early Relational Disturbances. In R. DelCarmen-Wiggins & A. S. Carter Carter (Ed.), *Handbook of infant and young child mental health assessment*. Oxford, UK: Oxford University Press.

Clarke, M. C., Kelleher, I., Clancy, M., & Cannon, M. (2012). Predicting risk and the emergence of schizophrenia. *Psychiatric Clinics of North America*, *35*(3), 585–612. doi: 10.1016/j.psc.2012.06.003

Comer, J. S., Puliafico, A. C., Aschenbrand, S. G., McKnight, K., Robin, J. A., Goldfine, M. E., & Albano, A. M. (2012). A pilot feasibility evaluation of the CALM Program for anxiety disorders in early childhood. *Journal of Anxiety Disorders*, *26*(1), 40–49. doi: 10.1016/j.janxdis.2011.08.011

Compas, B. E. (1987). Coping with stress during childhood and adolescence. *Psychological Bulletin*, *101*(3), 393–403.

Conners, C. K. (2009). *Conners Early Childhood (Conners EC) manual*. North Tonawanda, NY: Multi-Health Systems Inc.

Conners, C. K., Sitarenios, G., Parker, J. D., & Epstein, J. N. (1998). Revision and restandardization of the Conners Teacher Rating Scale (CTRS-R): Factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psychology*, *26*(4), 279–291.

Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests. *Psychmetrika*, *16*, 297–334.

Curtis, W. J., & Cicchetti, D. (2011). Affective facial expression processing in young children who have experienced maltreatment during the first year of life: An event-related potential study. *Development and Psychopathology*, *23*(2), 373–395. doi: 10.1017/S0954579411000125

Cuthbert, B. N., & Insel, T. R. (2013). Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Med*, *11*, 126. doi: 10.1186/1741-7015-11-126

D'Aprano, A. L., Carapetis, J. R., & Andrews, R. (2011). Trial of a developmental screening tool in remote Australian Aboriginal communities: a cautionary tale. *Journal of Paediatrics and Child Health*, *47*(1–2), 12–17. doi: 10.1111/j.1440-1754.2010.01883.x

Danzig, A. P., Bufferd, S. J., Dougherty, L. R., Carlson, G. A., Olino, T. M., & Klein, D. N. (2013). Longitudinal associations between preschool psychopathology and school-age peer functioning. *Child Psychiatry and Human Development*, *44*(5), 621–632. doi: 10.1007/s10578-012-0356-4

Dirks, M. A., De Los Reyes, A., Briggs-Gowan, M., Cella, D., & Wakschlag, L. S. (2012). Annual research review: Embracing not erasing contextual variability in children's behavior–Theory and utility in the selection and use of methods and informants in developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *53*(5), 558–574. doi: 10.1111/j.1469-7610.2012.02537.x

Disabilities, C. o. C. W. (2001). Developmental surveillance and screening of infants and young children. *Pediatrics*, *108*(1), 192–196. doi: 10.1542/peds.108.1.192

Dougherty, L. R., Smith, V. C., Bufferd, S. J., Stringaris, A., Leibenluft, E., Carlson, G. A., & Klein, D. N. (2013). Preschool irritability: longitudinal associations with psychiatric disorders at age 6 and parental psychopathology. *Journal of the American Academy of Child & Adolescent Psychiatry*, *52*(12), 1304–1313. doi: 10.1016/j.jaac.2013.09.007

Dressler, W. W. (1985). Extended family relationships, social support, and mental health in a southern Black community. *Journal of Health and Social Behavior*, *26*(1), 39–48.

Edwards, S. L., Rapee, R. M., Kennedy, S. J., & Spence, S. H. (2010). The assessment of anxiety symptoms in preschool-aged children: The revised Preschool Anxiety Scale. *Journal of Clinical Child & Adolescent Psychology*, *39*(3), 400–409. doi: 10.1080/15374411003691701

Egger, H. L., & Angold, A. (2004). The Preschool Age Psychiatric Assessment (PAPA): A structured parent interview. In R. DelCarmen-Wiggins & A. Carter (Eds.), *Handbook of infant, toddler, and preschool mental assessment* (pp. 223–243). New York, NY: Oxford University Press.

Egger, H. L., & Angold, A. (2006). Common emotional and behavioral disorders in preschool children: Presentation, nosology, and epidemiology. *Journal of Child Psychology and Psychiatry*, *47*(3–4), 313–337. doi: 10.1111/j.1469-7610.2006.01618.x

Egger, H. L., Erkanli, A., Keeler, G., Potts, E., Walter, B. K., & Angold, A. (2006). Test–retest reliability of the Preschool Age Psychiatric Assessment (PAPA). *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(5), 538–549. doi: 10.1097/01.chi.0000205705.71194.b8

Einfeld, S. L., Piccinin, A. M., Mackinnon, A., Hofer, S. M., Taffe, J., Gray, K. M., … Tonge, B. J. (2006). Psychopathology in young people with intellectual disability. *JAMA*, *296*(16), 1981–1989. doi: 10.1001/jama.296.16.1981

Ellingson, K. D., Briggs-Gowan, M. J., Carter, A. S., & Horwitz, S. M. (2004). Parent identification of early emerging child behavior problems: Predictors of sharing parental concern with health providers. *Archives of Pediatric and Adolescent Medicine*, *158*(8), 766–772. doi: 10.1001/archpedi.158.8.766 158/8/766 [pii]

Ellis, R., & Simmons, T. (2014). *Co-resident grandparents and their grandchildren: 2012*. In *Current Population Reports*. U.S. Census Bureau. Washington, DC.

Emerson, E. (2003). Prevalence of psychiatric disorders in children and adolescents with and without intellectual disability. *Journal of Intellectual Disability Research*, *47*, 51–58.

Evans, G. W. (2004). The environment of childhood poverty. *American Psychologist*, *59*(2), 77–92. doi: 10.1037/0003-066X.59.2.77

Eyberg, S., & Pincus, D. (1999). *Eyberg Child Behavior Inventory (ECBI) & Sutter-Eyberg Student Behavior Inventory-Revised*. Lutz, FL: Psychological Assessment Services.

Finkelhor, D., Turner, H. A., Shattuck, A., & Hamby, S. L. (2013). Violence, crime, and abuse exposure in a national sample of children and youth: An update. *JAMA Pediatrics*, *167*(7), 614–621. doi: 10.1001/jamapediatrics.2013.42

Finkelhor, D., Vanderminden, J., Turner, H., Shattuck, A., & Hamby, S. (2014). Youth exposure to violence prevention programs in a national sample. *Child Abuse & Neglect*. doi: 10.1016/j.chiabu.2014.01.010

Fisher, P. W., Shaffer, D., Piacentini, J. C., Lapkin, J., Kafantaris, V., Leonard, H., & Herzog, D. B. (1993). Sensitivity of the Diagnostic Interview Schedule for Children, 2nd ed. (DISC-2.1) for specific diagnoses of children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, *32*(3), 666–673. doi: 10.1097/00004583-199305000-00026

Flink, I. J., Restrepo, M. H., Blanco, D. P., Ortegon, M. M., Enriquez, C. L., Beirens, T. M., & Raat, H. (2013). Mental health of internally

displaced preschool children: A cross-sectional study conducted in Bogota, Colombia. *Social Psychiatry and Psychiatric Epidemiology*, *48*(6), 917–926. doi: 10.1007/s00127-012-0611-9

Friday, J. C. (1995). The psychological impact of violence in underserved communities. *Journal of Health Care for the Poor and Underserved*, *6*(4), 403–409.

Fuchs, S., Klein, A. M., Otto, Y., & von Klitzing, K. (2013). Prevalence of emotional and behavioral symptoms and their impact on daily life activities in a community sample of 3 to 5-year-old children. *Child Psychiatry and Human Development*, *44*(4), 493–503. doi: 10.1007/s10578-012-0343-9

Gadomski, A., Wissow, L. S., Slade, E., & Jenkins, P. (2010). Training clinicians in mental health communication skills: Impact on primary care utilization. *Academic Pediatrics*, *10*(5), 346–352. doi: 10.1016/j.acap.2010.06.012

Gaffrey, M. S., Belden, A. C., & Luby, J. L. (2011). The 2-week duration criterion and severity and course of early childhood depression: Implications for nosology. *Journal of Affective Disorders*, *133*(3), 537–545. doi: 10.1016/j.jad.2011.04.056

Gagne, J. R., Van Hulle, C. A., Aksan, N., Essex, M. J., & Goldsmith, H. H. (2011). Deriving childhood temperament measures from emotion-eliciting behavioral episodes: Scale construction and initial validation. *Psychological Assessment*, *23*(2), 337–353. doi: 10.1037/a0021746

Gardner, L. M., Murphy, L., Campbell, J. M., Tylavsky, F., Palmer, F. B., & Graff, J. C. (2013). Screening accuracy for risk of autism spectrum disorder using the Brief Infant-Toddler Social and Emotional Assessment (BITSEA). *Research in Autism Spectrum Disorders*, *7*(5), 591–600. doi: 10.1016/j.rasd.2013.01.004

Gleason, M. M., Zamfirescu, A., Egger, H. L., Nelson, C. A. III, Fox, N. A., & Zeanah, C. H. (2011). Epidemiology of psychiatric disorders in very young children in a Romanian pediatric setting. *European Child & Adolescent Psychiatry*, *20*(10), 527–535. doi: 10.1007/s00787-011-0214-0

Gleason, M. M., Zeanah, C. H., & Dickstein, S. (2010). Recognizing young children in need of mental health assessment: Development and preliminary validity of the early childhood screening assessment. *Infant Mental Health Journal*, *31*(3), 335–357. doi: 10.1002/imhj.20259

Godoy, L., Carter, A. S., Silver, R. B., Dickstein, S., & Seifer, R. (2014). Infants and toddlers left behind: Mental health screening and consultation in primary care. *Journal of Developmental & Behavioral Pediatrics*, *35*(5), 334–343. doi: 10.1097/DBP.0000000000000006000004703-201406000-00004 [pii]

Goodman, A., & Goodman, R. (2009). Strengths and difficulties questionnaire as a dimensional measure of child mental health. *Journal of the American Academy of Child & Adolescent Psychiatry*, *48*(4), 400–403. doi: 10.1097/CHI.0b013e3181985068S0890-8567(09)60047-2 [pii]

Goodman, A., Lamping, D. L., & Ploubidis, G. B. (2010). When to use broader internalising and externalising subscales instead of the hypothesised five subscales on the Strengths and Difficulties Questionnaire (SDQ): Data from British parents, teachers and children. *Journal of Abnormal Child Psychology*, *38*(8), 1179–1191. doi: 10.1007/s10802-010-9434-x

Goodman, R., Ford, T., Corbin, T., & Meltzer, H. (2004). Using the Strengths and Difficulties Questionnaire (SDQ) multi-informant algorithm to screen looked-after children for psychiatric disorders. *European Child & Adolescent Psychiatry*, *13 Suppl 2*, II25–31. doi: 10.1007/s00787-004-2005-3

Gray, S., & Wakschlag, L. (in press). The contribution of developmentally sensitive measurement to assessment of disruptive behavior in young children. In R. DelCarmen-Wiggins & A. S. Carter (Ed.), *Handbook of infant, toddler and preschool mental health assessment* (2nd ed.). Oxford University Press.

Gray, S. A., Carter, A. S., Briggs-Gowan, M. J., Hill, C., Danis, B., Keenan, K., & Wakschlag, L. S. (2012). Preschool children's observed disruptive behavior: Variations across sex, interactional context, and disruptive psychopathology. *Journal of Clinical Child and Adolescent Psychology*, *41*(4), 499–507.

Gresham, F., & Elliott, S. N. (2008). *Social Skills Improvement System (SSIS) Rating Scales*. Pearson. San Antonio, TX.

Haley, D. W., & Stansbury, K. (2003). Infant stress and parent responsiveness: Regulation of physiology and behavior during still-face and reunion. *Child Development*, *74*(5), 1534–1546.

Harrison, P. L., & Oakland, T. (2003). *Adaptive Behavior Assessment System, second edition (ABAS-II)*. San Antonio, TX: Psychological Corporation.

Hayden, L. C., Schiller, M., Dickstein, S., Seifer, R., Sameroff, A. J., Miller, I., ... Rasmussen, S. (1998). Levels of family assessment: I. Family, marital, and parent–child interaction. *Journal of Family Psychology*, *12*(1), 7–22.

Heberle, A. E., Thomas, Y. M., Wagmiller, R. L., Briggs-Gowan, M. J., & Carter, A. S. (2014). The impact of neighborhood, family, and individual risk factors on toddlers' disruptive behavior. *Child Development*, *85*(5), 2046–2061. doi: 10.1111/cdev.12251

Henrichs, J., Rescorla, L., Donkersloot, C., Schenk, J. J., Raat, H., Jaddoe, V. W., ... Tiemeier, H. (2013). Early vocabulary delay and behavioral/emotional problems in early childhood: The generation R study. *Journal of Speech Language and Hearing Research*, *56*(2), 553–566. doi: 10.1044/1092-4388(2012/11-0169)

Hensley, M. L., & Spriggs, D. R. (2004). Cancer screening: How good is good enough? *Journal of Clinical Oncology*, *22*(20), 4037–4039. doi: 10.1200/JCO.2004.06.129

Horwitz, S. M., Gary, L. C., Briggs-Gowan, M. J., & Carter, A. S. (2003). Do needs drive services use in young children? *Pediatrics*, *112*(6 Pt 1), 1373–1378.

Horwitz, S. M., Kelleher, K. J., Stein, R. E., Storfer-Isser, A., Youngstrom, E. A., Park, E. R., ... Hoagwood, K. E. (2007). Barriers to the identification and management of psychosocial issues in children and maternal depression. *Pediatrics*, *119*(1), e208–218. doi: 10.1542/peds.2005-1997

Hunsley, J., & Mash, E.J. (2007). Evidence-based assessment. *Annual Review of Clinical Psychology*, *3*, 29–51.

Ialongo, N., Edelsohn, G., Werthamer-Larsson, L., Crockett, L., & Kellam, S. (1993). Are self-reported depressive symptoms in first-grade children developmentally transient phenomena? A further look. *Development and Psychopathology: Special issue: Advances and Challenges in the Study of the Sequelae of Child Maltreatment*, *5*, 433–457.

Ialongo, N., Edelsohn, G., Werthamer-Larsson, L., Crockett, L., & Kellam, S. (1995). The significance of self-reported anxious symptoms in first grade children: Prediction to anxious symptoms and adaptive functioning in fifth grade. *Journal of Child Psychology and Psychiatry*, *36*(3), 427–437.

Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., ... Wang, P. (2010). Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, *167*(7), 748–751. doi: 10.1176/appi.ajp.2010.09091379

Jianduan, Z., Huishan, W., Shuhua, S., Xiaonan, H., Guoyan, L., Guangli, L., & Junxin, S. (2009). Reliability and validity of standardized Chinese version of Urban Infant-Toddler Social and Emotional Assessment. *Early Human Development*, *85*(5), 331–336. doi: 10.1016/j.earlhumdev.2008.12.012

Kagan, J., Reznick, J.S., Clarke, C., Snidman, N., & Garcia-Coll, C. (1984). Behavioral inhibition to the unfamiliar. *Child Development*, *55*(6), 2212–2225.

Kamphaus, R. W., & Reynolds, C. R. (2007). *BASC-2 Behavioral and Emotional Screening System (BASC-2 BESS)*. San Antonio, TX: Pearson.

Karabekiroglu, K., Uslu, R., Kapci-Seyitoglu, E. G., Ozbaran, B., Oztop, D. B., Ozel-Ozcan, O., . . . Cengel-Kultur, E. S. (2013). A nationwide study of social-emotional problems in young children in Turkey. *Infant Behavior and Development*, *36*(1), 162–170. doi: 10.1016/j.infbeh.2012.11.007

Kataoka, S. H., Zhang, L., & Wells, K. B. (2002). Unmet need for mental health care among U.S. children: Variation by ethnicity and insurance status. *American Journal of Psychiatry*, *159*(9), 1548–1555.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., . . . Ryan, N. (1997). Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(7), 980–988. doi: 10.1097/00004583-199707000-00021

Kaufman, J., Birmaher, B., Brent, D., Rao, U., & Ryan, N. (1996). The schedule for affective disorders and schizophrenia for school-age children. Pittsburgh: University of Pittsburgh Medical Center.

Keenan, K., Shaw, D. S., Walsh, B., Delliquadri, E., & Giovannelli, J. (1997). DSM-III-R disorders in preschool children from low-income families. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(5), 620–627.

Keenan, K., & Wakschlag, L. S. (2000). More than the terrible twos: The nature and severity of behavior problems in clinic-referred preschool children. *Journal of Abnormal Child Psychology*, *28*(1), 33–46.

Keenan, K., & Wakschlag, L. S. (2002). Can a valid diagnosis of disruptive behavior disorder be made in preschool children? *American Journal of Psychiatry*, *159*(3), 351–358.

Keenan, K., Wakschlag, L. S., Danis, B., Hill, C., Humphries, M., Duax, J., & Donald, R. (2007). Further evidence of the reliability and validity of DSM-IV ODD and CD in preschool children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *46*(4), 457–468. doi: 10.1097/CHI.0b013e31803062d3 00004583-200704000-00007 [pii]

Kennedy, S. J., Rapee, R. M., & Edwards, S. L. (2009). A selective intervention program for inhibited preschool-aged children of parents with an anxiety disorder: Effects on current anxiety disorders and temperament. *Journal of the American Academy of Child & Adolescent Psychiatry*, *48*(6), 602–609.

Kim-Cohen, J., Arseneault, L., Caspi, A., Tomas, M. P., Taylor, A., & Moffitt, T. E. (2005). Validity of DSM-IV conduct disorder in 41/2-5-year-old children: A longitudinal epidemiological study. *American Journal of Psychiatry*, *162*(6), 1108–1117. doi: 10.1176/appi.ajp.162.6.1108

King, T. M., Tandon, S. D., Macias, M. M., Healy, J. A., Duncan, P. M., Swigonski, N. L., . . . Lipkin, P. H. (2010). Implementing developmental screening and referrals: Lessons learned from a national project. *Pediatrics*, *125*(2), 350–360. doi: 10.1542/peds.2009-0388

Kleinman, A., Eisenberg, L., & Good, B. (1978). Culture, illness, and care: Clinical lessons from anthropologic and cross-cultural research. *Annals of Internal Medicine*, *88*(2), 251–258.

Kogan, N., & Carter, A. S. (1996). Mother-infant reengagement following the still-face: The role of maternal emotional availability an infant affect regulation. *Infant Behavior and Development*, *19*(3), 359–370.

Kristensen, S., Henriksen, T. B., & Bilenberg, N. (2010). The Child Behavior Checklist for Ages 1.5-5 (CBCL/1(1/2)-5): Assessment and analysis of parent- and caregiver-reported problems in a population-based sample of Danish preschool children. *Nordic Journal of Psychiatry*, *64*(3), 203–209. doi: 10.3109/08039480903456595

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry*, *56*(10), 921–926.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*, *111*(3), 411–424.

Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics*, *33*(1), 159–174.

Laughlin, L. (2013). Who's minding the kids? Child care arrangements: Spring 2011. In *Current Population Reports* (pp. 70–135). Washington, DC: U.S. Census Bureau.

Lavigne, J. V., Arend, R., Rosenbaum, D., Binns, H. J., Christoffel, K. K., & Gibbons, R. D. (1998). Psychiatric disorders with onset in the preschool years: II. Correlates and predictors of stable case status. *Journal of the American Academy of Child & Adolescent Psychiatry*, *37*(12), 1255–1261.

Lavigne, J. V., Gibbons, R. D., Christoffel, K. K., Arend, R., Rosenbaum, D., Binns, H., . . . Isaacs, C. (1996). Prevalence rates and correlates of psychiatric disorders among preschool children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*(2), 204–214.

Lavigne, J. V., Lebailly, S. A., Hopkins, J., Gouze, K. R., & Binns, H. J. (2009). The prevalence of ADHD, ODD, depression, and anxiety in a community sample of 4-year-olds. *Journal of Clinical Child and Adolescent Psychology*, *38*(3), 315–328. doi: 10.1080/15374410902851382

Liu, J., Cheng, H., & Leung, P. W. (2011). The application of the preschool Child Behavior Checklist and the caregiver-teacher report form to Mainland Chinese children: Syndrome structure, gender differences, country effects, and inter-informant agreement. *Journal of Abnormal Child Psychology*, *39*(2), 251–264. doi: 10.1007/s10802-010-9452-8

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., . . . Rutter, M. (2000). The Autism Diagnostic Observation Schedule—Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism & Developmental Disorders*, *30*(3), 205–223.

Lord, C., Rutter, M., Goode, S., Heemsbergen, J., Jordan, H., Mawhood, L., & Schopler, E. (1989). Autism diagnostic observation schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders*, *19*(2), 185–212.

Lowell, D. I., Carter, A. S., Godoy, L., Paulicin, B., & Briggs-Gowan, M. J. (2011). A randomized controlled trial of Child FIRST: A comprehensive home-based intervention translating research into early childhood practice. *Child Development*, *82*(1), 193–208. doi: 10.1111/j.1467-8624.2010.01550.x

Luby, J., Belden, A., Sullivan, J., Hayen, R., McCadney, A., & Spitznagel, E. (2009). Shame and guilt in preschool depression: Evidence for elevations in self-conscious emotions in depression as early as age 3. *Journal of Child Psychology and Psychiatry*, *50*(9), 1156–1166. doi: 10.1111/j.1469-7610.2009.02077.x

Luby, J. L., Belden, A. C., Pautsch, J., Si, X., & Spitznagel, E. (2009). The clinical significance of preschool depression: Impairment in functioning and clinical markers of the disorder. *Journal of Affective Disorders*, *112*(1–3), 111–119. doi: 10.1016/j.jad.2008.03.026

Luby, J. L., Heffelfinger, A. K., Mrakotsky, C., Hessler, M. J., Brown, K. M., & Hildebrand, T. (2002). Preschool major depressive disorder: preliminary validation for developmentally modified DSM-IV criteria. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*(8), 928–937.

Luby, J. L., & Navsaria, N. (2010). Pediatric bipolar disorder: Evidence for prodromal states and early markers. *Journal of Child Psychology and Psychiatry*, *51*(4), 459–471. doi: 10.1111/j.1469-7610.2010.02210.x

Luby, J. L., Si, X., Belden, A. C., Tandon, M., & Spitznagel, E. (2009). Preschool depression: Homotypic continuity and course over 24 months. *Archives of General Psychiatry*, *66*(8), 897–905. doi: 10.1001/archgenpsychiatry.2009.97

Lucey, P., Cohn, J. F., Matthews, I., Lucey, S., Sridharan, S., Howlett, J., & Prkachin, K. M. (2011). Automatically detecting pain in video through facial action units. *IEEE Transaction Systems Man Cybernetics B Cybernetics*, *41*(3), 664–674. doi: 10.1109/TSMCB.2010.2082525

Luster, T., Rhoades, K., & Haas, B. (1989). The relation between parental values and parenting behavior: A test of the Kohn hypothesis. *Journal of Marriage and the Family*, 139–147.

Martinez-Torteya, C., Dayton, C. J., Beeghly, M., Seng, J. S., McGinnis, E., Broderick, A., . . . Muzik, M. (2014). Maternal parenting predicts infant biobehavioral regulation among women with a history of childhood maltreatment. *Development and Psychopathology*, *26*(2), 379–392. doi: 10.1017/S0954579414000017

Martini, D. R., Strayhorn, J. M., & Puig-Antich, J. (1990). A symptom self-report measure for preschool children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *29*(4), 594–600. doi: 10.1097/00004583-199007000-00013

Mathiesen, K. S., & Sanson, A. (2000). Dimensions of early childhood behavior problems: Stability and predictors of change from 18 to 30 months. *Journal of Abnormal Child Psychology*, *28*(1), 15–31.

McCue Horwitz, S., Hurlburt, M. S., Heneghan, A., Zhang, J., Rolls-Reutz, J., Fisher, E., . . . Stein, R. E. (2012). Mental health problems in young children investigated by U.S. child welfare agencies. *Journal of the American Academy of Child & Adolescent Psychiatry*, *51*(6), 572–581. doi: 10.1016/j.jaac.2012.03.006

McHale, J. P., & Coates, E. E. (2014). Observed coparenting and triadic dynamics in African American fragile families at 3 months' postpartum. *Infant Mental Health Journal*, *35*(5), 435–451.

McHale, J. P., & Fivaz-Depeursinge, E. (1999). Understanding triadic and family group interactions during infancy and toddlerhood. *Clinical Child and Family Psychology Review*, *2*(2), 107–127.

McLaughlin, B., Gesi Blanchard, A., & Osanai, Y. (1995). Assessing language development in bilingual preschool children, NCBE Program Information Guide Series No. 22. National Clearinghouse for Bilingual Education. Washington, D.C.

Measelle, J. R., Ablow, J. C., Cowan, P. A., & Cowan, C. P. (1998). Assessing young children's views of their academic, social, and emotional lives: An evaluation of the self-perception scales of the Berkeley Puppet Interview. *Child Development*, *69*(6), 1556–1576.

Merrell, K. W. (2002). *Preschool and Kindergarten Behavior Scales (PKBS-2), second edition*. Austin, TX: Pro-Ed, Inc.

Mian, N., Carter, A. S., Pine, D. S., Wakschlag, L., & Briggs-Gowan, M. J. (in press). Development of a novel observational measure for anxiety in young children: The anxiety dimensional observation scale. *Journal of Child Psychology and Psychiatry*. doi: 10.1111/jcpp.12407

Mian, N. D., Godoy, L., Briggs-Gowan, M. J., & Carter, A. S. (2012). Patterns of anxiety symptoms in toddlers and preschool-age children: Evidence of early differentiation. *Journal of Anxiety Disorders*, *26*(1), 102–110. doi: 10.1016/j.janxdis.2011.09.006

Milgrom, J., Mendelsohn, J., & Gemmill, A. W. (2011). Does postnatal depression screening work? Throwing out the bathwater, keeping the baby. *Journal of Affective Disorders*, *132*(3), 301–310. doi: 10.1016/j.jad.2010.09.031

Morgan, P. L., Farkas, G., Tufis, P. A., & Sperling, R. A. (2008). Are reading and behavior problems risk factors for each other? *Journal of Learning Disabilities*, *41*(5), 417–436.

Mouton-Simien, P., McCain, A. P., & Kelley, M. L. (1997). The development of the Toddler Behavior Screening Inventory. *Journal of Abnormal Child Psychology*, *25*(1), 59–64.

Neitzel, C., & Stright, A. D. (2003). Mothers' scaffolding of children's problem solving: Establishing a foundation of academic self-regulatory competence. *Journal of Family Psychology*, *17*(1), 147–159.

Nichols, S., Briggs-Gowan, M. J., Estabrook, R., Burns, J., Kestler, J., Berman, G., . . . Wakschlag, L. S. (in press). Punishment insensitivity in early childhood: A developmental, dimensional approach. *Journal of Abnormal Child Psychology*.

Nunnally, J. C. (1978). *Psychometric theory*. New York, NY: McGraw-Hill Publishing Co.

O'Neill, S., Schneiderman, R. L., Rajendran, K., Marks, D. J., & Halperin, J. M. (2014). Reliable ratings or reading tea leaves: Can parent, teacher, and clinician behavioral ratings of preschoolers predict ADHD at age six? *Journal of Abnormal Child Psychology*, *42*(4), 623–634. doi: 10.1007/s10802-013-9802-4

Otto, Y., Andreas, A., von Klitzing, K., Fuchs, S., & Klein, A. M. (2014). [Sadness, worries and fears: depression and anxiety disorders in preschool age—Results of relevance, symptoms and impairment]. *Prax Kinderpsychol Kinderpsychiatr*, *63*(3), 154–176.

Policy statement—The future of pediatrics: Mental health competencies for pediatric primary care. (2009). *Pediatrics*, *124*(1), 410–421. doi: 10.1542/peds.2009-1061

Qi, C. H., & Kaiser, A. P. (2003). Behavior problems in preschool children from low-income families: Review of the literature. *Topics in Early Childhood Special Education*, *23*(4), 188–216.

Rapee, R. M., Kennedy, S., Ingram, M., Edwards, S., & Sweeney, L. (2005). Prevention and early intervention of anxiety disorders in inhibited preschool children. *Journal of Consulting and Clinical Psychology*, *73*(3), 488–497. doi: 10.1037/0022-006X.73.3.488

Reynolds, C. R., & Kamphaus, R. W. (2004). *BASC-2 Behavior Assessment System for Children: Second edition manual (BASC-2)*. Minneapolis, MN: NCS Pearson.

Reznick, J. S., Kagan, J., Snidman, N., Gersten, M., Baak, K., & Rosenberg, A. (1986). Inhibited and uninhibited children: A follow-up study. *Child Development*, *57*(3), 660–680.

Richman, N., & Graham, P. J. (1971). A behavioural screening questionnaire for use with three-year-old children. Preliminary findings. *Journal of Child Psychology and Psychiatry*, *12*, 5–33.

Robins, D. L., Casagrande, K., Barton, M., Chen, C. M., Dumont-Mathieu, T., & Fein, D. (2014). Validation of the modified checklist for Autism in toddlers, revised with follow-up (M-CHAT-R/F). *Pediatrics*, *133*(1), 37–45. doi: 10.1542/peds.2013-1813

Ross, G., & Weinberg, S. (2006). Is there a relationship between language delays and behavior and socialization problems in toddlers? *Journal of Early Childhood and Infant Psychology*, *2*, 2101–2116.

Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist*, *55*(1), 68–78.

Sampson, R. J., Raudenbush, S. W., & Earls, F. (1997). Neighborhoods and violent crime: A multilevel study of collective efficacy. *Science*, *277*(5328), 918–924.

Schechter, D. S., & Willheim, E. (2009). The effects of violent experiences on infants and young children. In C. Zeanah (Ed.), *Handbook of infant mental health* (third edition, pp. 197–213). New York, NY: Guilford Press.

Scheeringa, M. S. (2004). *Diagnostic Infant and Preschool Assessment (DIPA) manual*. Institute of Infant and Early Childhood Mental Health: Section of Child and Adolescent Psychiatry, Department of Psychiatry and Neurology. Tulane University, New Orleans, LA.

Scheeringa, M. S. (2007). A research agenda for posttraumatic stress disorder in infants, toddlers, and preschool children. In W. E. Narrow, M. B. First, P. J. Sirovatka, & D. A. Regier (Eds.), *Age and gender considerations in psychiatric diagnosis* (pp. 151–162). Arlington, VA: American Psychiatric Association.

Scheeringa, M. S. (2008). Developmental considerations for diagnosing PTSD and acute stress disorder in preschool and school-age children. *American Journal of Psychiatry*, *165*(10), 1237–1239. doi: 10.1176/appi.ajp.2008.08070974

Scheeringa, M. S., & Haslett, N. (2010). The reliability and criterion validity of the Diagnostic Infant and Preschool Assessment: A new diagnostic instrument for young children. *Child Psychiatry and Human Development*, *41*(3), 299–312. doi: 10.1007/s10578-009-0169-2

Scheeringa, M. S., Myers, L., Putnam, F. W., & Zeanah, C. H. (2012). Diagnosing PTSD in early childhood: An empirical assessment of four approaches. *Journal of Traumatic Stress*, *25*(4), 359–367. doi: 10.1002/jts.21723

Scheeringa, M. S., Peebles, C. D., Cook, C. A., & Zeanah, C. H. (2001). Toward establishing procedural, criterion, and discriminant validity for PTSD in early childhood. *Journal of the American Academy of Child & Adolescent Psychiatry*, *40*(1), 52–60.

Scheeringa, M. S., & Zeanah, C. H. (2001). A relational perspective on PTSD in early childhood. *Journal of Traumatic Stress*, *14*(4), 799–815.

Schonwald, A., Huntington, N., Chan, E., Risko, W., & Bridgemohan, C. (2009). Routine developmental screening implemented in urban primary care settings: More evidence of feasibility and effectiveness. *Pediatrics*, *123*(2), 660–668. doi: 10.1542/peds.2007-2798

Seifer, R. (2000). Temperament and goodness of fit: Implications for developmental psychopathology. In A. J. Sameroff, M. Lewis, & S. M. Miller (eds). *Handbook of developmental psychopathology* (2nd edition, pp. 257–276). New York: Plenum Press

Shamir-Essakow, G., Ungerer, J. A., & Rapee, R. M. (2005). Attachment, behavioral inhibition, and anxiety in preschool children. *Journal of Abnormal Child Psychology*, *33*(2), 131–143.

Shaw, D. S., Lacourse, E., & Nagin, D. (2005). Developmental trajectories of conduct problems and hyperactivity from ages 2 to 10. *Journal of Psychology and Psychiatry*, *46*(9), 931–942.

Sheldrick, R. C., Henson, B. S., Merchant, S., Neger, E. N., Murphy, J. M., & Perrin, E. C. (2012). The Preschool Pediatric Symptom Checklist (PPSC): Development and initial validation of a new social/emotional screening instrument. *Academic Pediatrics*, *12*(5), 456–467. doi: 10.1016/j.acap.2012.06.008S1876-2859(12)00178-7

Sheldrick, R. C., Henson, B. S., Neger, E. N., Merchant, S., Murphy, J. M., & Perrin, E. C. (2013). The baby pediatric symptom checklist: Development and initial validation of a new social/emotional screening instrument for very young children. *Academic Pediatrics*, *13*(1), 72–80. doi: 10.1016/j.acap.2012.08.003S1876-2859(12)00202-1

Sheldrick, R. C., Merchant, S., & Perrin, E. C. (2011). Identification of developmental-behavioral problems in primary care: A systematic review. *Pediatrics*, *128*(2), 356–363. doi: 10.1542/peds.2010-3261

Sheldrick, R. C., Neger, E. N., & Perrin, E. C. (2012). Concerns about development, behavior, and learning among parents seeking pediatric care. *Journal of Developmental and Behavioral Pediatrics*, *33*(2), 156–160. doi: 10.1097/DBP.0b013e3182420f4a

Sheldrick, R. C., & Perrin, E. C. (2013). Evidence-based milestones for surveillance of cognitive, language, and motor development. *Academic Pediatrics*, *13*(6), 577–586. doi: 10.1016/j.acap.2013.07.001S1876-2859(13)00202-7

Shonkoff, J., & Phillips, D. (Eds.). (2000). *From neurons to neighborhoods: The science of early childhood development*. Washington, DC: National Academy Press.

Shonkoff, J. P., Garner, A. S., & The Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood, Adoption, and Dependent, Care, and Section on Developmental and Behavioral Pediatrics. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*(1), e232–246. doi: 10.1542/peds.2011-2663

Silverman, W. K., Saavedra, L. M., & Pina, A. A. (2001). Test–retest reliability of anxiety symptoms and diagnoses with the Anxiety Disorders Interview Schedule for *DSM-IV*: Child and parent versions. *Journal of the American Academy of Child & Adolescent Psychiatry*, *40*(8), 937–944. doi: 10.1097/00004583-200108000-00016

Smith, N. J., Sheldrick, R. C., & Perrin, E. C. (2013). An abbreviated screening instrument for autism spectrum disorders. *Infant Mental Health Journal*, *34*(2), 149–155. doi: 10.1002/imhj.21356

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland Adaptive Behavior Scales: Second edition (Vineland-II), Survey Interview Form/Caregiver Rating Form*. Livonia, MN: Pearson Assessment.

Speltz, M. L., McClellan, J., DeKlyen, M., & Jones, K. (1999). Preschool boys with oppositional defiant disorder: Clinical presentation and diagnostic change. *Journal of the American Academy of Child & Adolescent Psychiatry*, *38*(7), 838–845. doi: 10.1097/00004583-199907000-00013

Spence, S. H., Najman, J. M., Bor, W., O'Callaghan, M. J., & Williams, G. M. (2002). Maternal anxiety and depression, poverty and marital relationship factors during early childhood as predictors of anxiety and depressive symptoms in adolescence. *Journal of Child Psychology and Psychiatry*, *43*(4), 457–469.

Spence, S. H., Rapee, R., McDonald, C., & Ingram, M. (2001). The structure of anxiety symptoms among preschoolers. *Behavioral Research and Therapy*, *39*(11), 1293–1316.

Spittle, A. J., Anderson, P. J., Lee, K. J., Ferretti, C., Eeles, A., Orton, J., . . . Doyle, L. W. (2010). Preventive care at home for very preterm infants improves infant and caregiver outcomes at 2 years. *Pediatrics*, *126*(1), 171–178.

Squires, J., Bricker, D., & Twombly, E. (2002). *Ages and Stages Questionnaire: Social-Emotional: A parent-completed, child-monitoring system for social-emotional behaviors*. Baltimore, MD: Paul H. Brookes Publishing Co.

Stalets, M. M., & Luby, J. L. (2006). Preschool depression. *Child and Adolescent Psychiatry Clinics of North America*, *15*(4), 899–917, viii–ix. doi: 10.1016/j.chc.2006.05.011

Ştefan, C. A., & Miclea, M. (2013). Effects of a multifocused prevention program on preschool children's competencies and behavior problems. *Psychology in the Schools*, *50*(4), 382–402. doi: doi:10.1002/pits.21683

Sterba, S., Egger, H. L., & Angold, A. (2007). Diagnostic specificity and nonspecificity in the dimensions of preschool psychopathology. *Journal of Child Psychology and Psychiatry*, *48*(10), 1005–1013. doi: 10.1111/j.1469-7610.2007.01770.x

Stoneman, Z., Brody, G. H., Churchill, S. L., & Winn, L. L. (1999). Effects of residential instability on Head Start children and their relationships with older siblings: Influences of child emotionality and conflict between family caregivers. *Child Development*, *70*(5), 1246–1262.

Tackett, J. L., Lahey, B. B., van Hulle, C., Waldman, I., Krueger, R. F., & Rathouz, P. J. (2013). Common genetic influences on negative emotionality and a general psychopathology factor in childhood and adolescence. *Journal of Abnormal Psychology*, *122*(4), 1142–1153. doi: 10.1037/a0034151

Thaler, N. S., Allen, D. N., Scott, J. G. (2014). Neuropsychological assessment of Asian American children and adolescents. 407–425.

Thomas, J. M., & Guskin, K. A. (2001). Disruptive behavior in young children: What does it mean? *Journal of the American Academy of*

Child & Adolescent Psychiatry, *40*(1), 44–51. doi: 10.1097/00004583-200101000-00015

Todd, R. D., Huang, H., Smalley, S. L., Nelson, S. F., Willcutt, E. G., Pennington, B. F., … Neuman, R. J. (2005). Collaborative analysis of DRD4 and DAT genotypes in population-defined ADHD subtypes. *Journal of Child Psychology & Psychiatry*, *46*(10), 1067–1073.

Tronick, E. Z. (1989). Emotions and emotional communication in infants. *American Psychologist*, *44*(2), 112–119.

van Widenfelt, B. M., Treffers, P. D. A., de Beurs, E., Siebelink, B. M., & Koudijs, E. (2005). Translation and cross-cultural adaptation of assessment instruments used in psychological research with children and families. *Clinical Child and Family Psychology Review*, *8*(2), 135–147.

Visser, J. C., Smeekens, S., Rommelse, N., Verkes, R. J., Van der Gaag, R. J., & Buitelaar, J. K. (2010). Assessment of psychopathology in 2- to 5-year-olds: Applying the Infant-Toddler Social Emotional Assessment. *Infant Mental Health Journal*, *31*(6), 611–629.

Votruba-Drzal, E., Coley, R. L., & Chase-Lansdale, P. L. (2004). Child care and low-income children's development: Direct and moderated effects. *Child Development*, *75*(1), 296–312.

Vygotsky, L. S. (1978). *Mind and society: The development of higher mental processes*. Cambridge, MA: Harvard University Press.

Wakschlag, L. S., Briggs-Gowan, M. J., Carter, A. S., Hill, C., Danis, B., Keenan, K., … Leventhal, B. L. (2007). A developmental framework for distinguishing disruptive behavior from normative misbehavior in preschool children. *Journal of Child Psychology and Psychiatry*, *48*(10), 976–987. doi: 10.1111/j.1469-7610.2007.01786.x

Wakschlag, L. S., Briggs-Gowan, M. J., Choi, S. W., Nichols, S. R., Kestler, J., Burns, J. L., … Henry, D. (2014). Advancing a multidimensional, developmental spectrum approach to preschool disruptive behavior. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(1), 82–96 e83. doi: 10.1016/j.jaac.2013.10.011

Wakschlag, L. S., Briggs-Gowan, M. J., Hill, C., Danis, B., Leventhal, B. L., Keenan, K., … Carter, A. S. (2008). Observational Assessment of Preschool Disruptive Behavior, Part II: Validity of the Disruptive Behavior Diagnostic Observation Schedule (DB-DOS). *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(6), 632–641. doi: 10.1097/CHI.0b013e31816c5c10

Wakschlag, L. S., Choi, S. W., Carter, A. S., Hullsiek, H., Burns, J., McCarthy, K., … Briggs-Gowan, M. J. (2012). Defining the developmental parameters of temper loss in early childhood: Implications for developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *53*(11), 1099–1108. doi: 10.1111/j.1469-7610.2012.02595.x

Wakschlag, L. S., Estabrook, R., Henry, D., Burns, J. L., Perlman, S. B., Pine, D. S., … Briggs-Gowan, M. J. (in press). Clinical application of a dimensional approach: The normal:abnormal spectrum of early irritability. *Journal of the American Academy of Child & Adolescent Psychiatry*.

Wakschlag, L. S., Henry, D. B., Tolan, P., Carter, A. S., Burns, J., & Briggs-Gowan, M. J. (2012). Putting theory to the test: Modeling a multidimensional, developmentally-based approach to preschool disruptive behavior. *Journal of the American Academy of Child & Adolescent Psychiatry*, *51*(6), 593–604.

Wakschlag, L. S., Hill, C., Carter, A. S., Danis, B., Egger, H. L., Keenan, K., … Briggs-Gowan, M. J. (2008). Observational assessment of preschool disruptive behavior, part I: reliability of the Disruptive Behavior Diagnostic Observation Schedule (DB-DOS). *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(6), 622–631. doi: 10.1097/CHI.0b013e31816c5bdb

Wakschlag, L. S., Tolan, P. H., & Leventhal, B. L. (2010). Research review: "Ain't misbehavin": Towards a developmentally-specified nosology for preschool disruptive behavior. *Journal of Child Psychology and Psychiatry*, *51*(1), 3–22.

Wang, M. Zhou, J. (2014). Anxiety disorder symptoms in Chinese preschool children. *Child Psychiatry & Human Development*, *46*(1): 158–66.

Warren, S. L., Emde, R. N., & Sroufe, L. A. (2000). Internal representations: predicting anxiety from children's play narratives. *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*(1), 100–107. doi: 10.1097/00004583-200001000-00022

Warren, S. L., Oppenheim, D., & Emde, R. N. (1996). Can emotions and themes in children's play predict behavior problems? *Journal of the Amercian Academy of Child & Adolescent Psychiatry*, *35*(10), 1331–1337. doi: 10.1097/00004583-199610000-00020

Weitzman, C., Edmonds, D., Davagnino, J., & Briggs-Gowan, M. J. (2014). Young child socioemotional/behavioral problems and cumulative psychosocial risk. *Infant Mental Health Journal*, *35*(1), 1–9. doi: 10.1002/imhj.21421

Wichstrom, L., Berg-Nielsen, T. S., Angold, A., Egger, H. L., Solheim, E., & Sveen, T. H. (2012). Prevalence of psychiatric disorders in preschoolers. *Journal of Child Psychology and Psychiatry*, *53*(6), 695–705. doi: 10.1111/j.1469-7610.2011.02514.x

Wilson, M. N., & Tolson, T. F. (1990). Familial support in the Black community. *Journal of Clinical Child Psychology*, *19*(4), 347–355.

World Health Organization. (2001). *ICF: International Classification of Functioning, Disability, and Health*. Geneva, Switzerland: World Health Organization.

Wu, Y. T., Chen, W. J., Hsieh, W. S., Chen, P. C., Liao, H. F., Su, Y. N., & Jeng, S. F. (2012). Maternal-reported behavioral and emotional problems in Taiwanese preschool children. *Research in Developmental Disabilities*, *33*(3), 866–873. doi: 10.1016/j.ridd.2011.11.018

Yasui, M., & Dishion, T. J. (2007). The ethnic context of child and adolescent problem behavior: Implications for child and family interventions. *Clinical Child and Family Psychology Review*, *10*(2), 137–179. doi: 10.1007/s10567-007-0021-9

Yu, J. W., Buka, S. L., McCormick, M. C., Fitzmaurice, G. M., & Indurkhya, A. (2006). Behavioral problems and the effects of early intervention on eight-year-old children with learning disabilities. *Maternal and Child Health Journal*, *10*(4), 329–338. doi: 10.1007/s10995-005-0066-7

CHAPTER 2

Developmental Issues in Assessment, Taxonomy, and Diagnosis of Psychopathology: Life Span and Multicultural Perspectives

THOMAS M. ACHENBACH and LESLIE A. RESCORLA

LIFE SPAN PERSPECTIVES 47
MULTICULTURAL PERSPECTIVES 47
DEVELOPMENTAL PSYCHOPATHOLOGY 48
THE DEVELOPMENTAL COMPONENT 48
Developmental Theories 49
Developmental Considerations in Assessment, Taxonomy, and Diagnosis 49
Developmental Methodology 49
THE PSYCHOPATHOLOGY COMPONENT 50
Nosologically Based Models 50
Empirically Based Models 51
A FRAMEWORK FOR THE DEVELOPMENTAL STUDY OF PSYCHOPATHOLOGY 52
Developmental Differences 52
Sources of Data 52
Epidemiological Aspects 54
Multivariate Aspects 55
Operational Definitions 55
THE ROLES OF ASSESSMENT AND TAXONOMY IN THE DEVELOPMENTAL STUDY OF PSYCHOPATHOLOGY 56
Assessment 56
Taxonomy 57
Are Behavioral and Emotional Disorders Natural Kinds? 57
Constructing Taxonomies of Behavioral, Emotional, and Social Problems 57
Prototypes as Taxonomic Models 58
DIAGNOSIS 58
Diagnostic Processes 59
Formal Diagnoses 59
Diagnostic Formulations 59
Comorbidity Issues 59
LONGITUDINAL DESIGNS FOR THE DEVELOPMENTAL STUDY OF PSYCHOPATHOLOGY 61
Accelerated Longitudinal Designs 61
Path Designs 65
Growth Curve Designs 67
Growth Mixture Models 70
LIFE SPAN APPLICATIONS 72
STANDARDIZED ASSESSMENT AND TAXONOMIC MODELS IN MULTIPLE LONGITUDINAL STUDIES 72
Zuid Holland Longitudinal Study 73
The TRacking Adolescents' Individual Lives Survey (TRAILS) Study 74
Generation R Longitudinal Study ("R" = Rotterdam) 74
Netherlands Twin Registry (NTR) 75
MULTICULTURAL FINDINGS AND APPLICATIONS 75
Multicultural Tests of Syndromes 75
Multicultural Comparisons of Scale Scores 76
Construction of Multicultural Norms 76
ADVANCING ASSESSMENT, TAXONOMY, AND DIAGNOSIS 77
Challenges to Be Met 78
QUANTITATIVE AIDS TO MEETING THE CHALLENGES 79
Advantages of Quantification for Research 80
Advantages of Quantification for Conceptualizing Psychopathology 80
Advantages of Quantification for Mental Health Services 81
EFFORTS TO QUANTIFY DIAGNOSTIC TAXA 82
Rating Scales for *DSM* Categories 82
DSM-Oriented Scales 82
Bridging Gaps Between Nosologically and Empirically Based Constructs 83
DIRECTIONS FOR CONTINUED RESEARCH 85
Family-Oriented Assessment and Interventions 85
Multicultural Directions 86
Quantification of Diagnostic Taxa 86
Use of Norms to Evaluate Interventions 87
Use of Multisource Data 87
SUMMARY 87
REFERENCES 88

Our chapter in the previous edition of this book culminated with an overview of future directions for the developmental study of psychopathology. These included advancing developmentally oriented research on adult psychopathology and extending such research to more diverse societies. In this chapter, we report numerous new research findings on the assessment of adult as well as child psychopathology in over fifty societies. (For brevity, we use *child* to include ages $1\frac{1}{2}$ to 18.) The research reveals similarities and differences in the patterning and levels of psychopathology at different ages in different societies. It additionally reveals similarities and differences in problems reported for males versus females of different ages, according to multiple sources of data. The research is also yielding practical methods for integrating *multicultural* assessment of children with assessment of their parents to advance *family-oriented* interventions.

A related focus is on progress in strengthening the empirical foundations for developmental understanding of psychopathology. Findings from multiple longitudinal studies that operationalize phenotypes for psychopathology in similar ways are advancing knowledge in terms of evidence-based assessment and taxonomy. In conjunction with life span and multicultural research, the longitudinal studies are expanding our knowledge of multisource assessment, the benefits it confers, and the challenges it raises.

In preparation for considering developmental aspects of assessment, taxonomy, and *diagnosis* of psychopathology, we first outline life span and multicultural perspectives that increasingly contribute to the developmental study of psychopathology. Thereafter, we address conceptual components of developmental psychopathology, the roles of assessment and taxonomy, the multiple meanings of diagnosis, life span, and multicultural findings, the benefits of quantification, and future directions.

LIFE SPAN PERSPECTIVES

Developmental approaches to the study of psychopathology originated with efforts to advance our understanding of maladaptive functioning between birth and maturity (Achenbach, 1974, 1982; Cicchetti, 1984; Rutter & Garmezy, 1983). It is now widely recognized that behavioral and emotional problems in the young need to be understood in relation to developmental sequences, norms, and processes. However, it is also becoming clear that developmental approaches are needed to understand psychopathology at older ages. In many countries, challenges posed by the growing legions of elders are raising awareness of the need to cope with developmental changes in functioning during the last decades of life. Equally important, developmental perspectives can illuminate adaptive and maladaptive functioning in the early and middle adult years, when many changes occur in social roles, relationships, responsibilities, careers, self-concepts, and biological characteristics, including health. Although developmental aspects of assessment, taxonomy, and diagnosis have received more attention regarding children than adults, life span perspectives argue for expanding our horizons to include developmental aspects of adult psychopathology as well.

A particularly important issue concerns the need to obtain multisource data for purposes of assessment, taxonomy, and diagnosis. Most practitioners and researchers now understand that no single source can serve as a gold standard for assessing children's functioning. Because agreement among informants is typically low to moderate (Achenbach, McConaughy, & Howell, 1987; De Los Reyes, 2011), it is widely recognized that assessment of children requires data from multiple informants. The data from multiple informants must then be systematically compared and integrated to identify both the consistencies and variations among reports of each child's functioning.

In the assessment of elders whose mental competence may be questionable, it is common to obtain information from other informants, such as a spouse or grown child. In the assessment of younger adults, however, data are typically obtained solely from the adults who are being assessed, mainly via interviews, tests, and questionnaires. Yet, meta-analyses of cross-informant correlations between reports of adult psychopathology have revealed only modest agreement between adults' self-reports and reports by others (Achenbach, Krukowski, Dumenci, & Ivanova, 2005). A major way to advance assessment, taxonomy, and diagnosis of adult psychopathology is therefore to apply to adults the principles and methods for obtaining multiinformant data that are now commonly applied to children. Other lessons learned from the developmental study of child psychopathology are also applicable to adults.

MULTICULTURAL PERSPECTIVES

Mental health professionals are increasingly expected to help people from diverse backgrounds. In some cases, the need to cope with diversity arises from influxes of foreign refugees and immigrants to host societies. In other cases, the need to cope with diversity arises from migrations within a society. And in still other cases, the need to cope with diversity arises from greater sensitivity to differences

among people who have long coexisted in a particular society.

Diversity raises a variety of challenges for mental health professionals. Perhaps the most obvious is differences in language. Because mental health professionals may not be fluent in the languages of all the groups to be served, interpreters may be needed. Even when language is not a barrier, however, differences related to ethnicity, religion, place of origin, and socioeconomic status (SES) may affect communications between mental health professionals and the people they serve.

For assessment, taxonomy, and diagnosis of psychopathology, a fundamental issue is whether the same standardized procedures can be used to assess people from different backgrounds. This issue raises questions such as the following:

1. Can mental health professionals use the same standardized procedures to assess the problems and adaptive functioning of people from different populations?
2. Do people from different populations accept the same assessment procedures as effective ways of communicating information?
3. What can we learn from comparisons of standardized assessment data obtained from members of different populations?
4. Can mental health professionals use standardized procedures to distinguish between characteristics that are specific to individuals versus characteristics thought to typify populations to which the individuals belong?

To answer these questions, systematic research is needed to compare the results of standardized assessment procedures applied to representative samples of people from different populations. As detailed later, a growing body of research is available to answer these questions for diverse populations. Such research can also help us distinguish between aspects of psychopathology that are relatively consistent across populations versus those that are more variable. Throughout this chapter, we highlight the importance of comparing findings for people who differ in background and developmental level, as they are assessed from different perspectives, to identify important consistencies and differences in patterns and levels of psychopathology.

DEVELOPMENTAL PSYCHOPATHOLOGY

Developmental approaches to the study of psychopathology have become increasingly pervasive over the past few decades. These approaches have greatly advanced our understanding of maladaptive functioning in childhood and adolescence. Developmental approaches to psychopathology have also helped to shape training and practice. However, much remains to be done to integrate the study, prevention, and treatment of psychopathology with the study of development across the life span.

Assessment, taxonomy, and diagnosis are central foci around which developmental approaches to psychopathology can be organized. As a preface to considering assessment, taxonomy, and diagnosis, it is first important to introduce what we mean by *development* and *psychopathology*. We use both of these concepts broadly, with a minimum of theoretical assumptions. Although particular theories of development and psychopathology may help us understand particular phenomena, the utility of these concepts would be diminished by limiting their meanings to the dictates of particular theories.

THE DEVELOPMENTAL COMPONENT

We use *developmental* to encompass processes, changes, sequences, and characteristics that are typically associated with age. Chronological age (CA) is the most obvious developmental metric, but it can serve as a proxy for many other developmental parameters. For some purposes, it may be preferable to use developmental metrics that do not necessarily correlate highly with CA. Mental age (MA) and other indices of cognitive developmental level, for example, would not correlate highly with CA in samples that included children with intellectual disabilities, whose cognitive levels are below their CAs, or very bright children, whose cognitive levels are above their CAs. Biological parameters may also provide important developmental metrics. Nevertheless, even when parameters other than CA provide metrics for developmental variables, CA remains an important common denominator because of CA-related differences in biological maturity, appearance, and social status. Our use of the term *developmental* is intended to stimulate new ideas rather than to mold thinking according to narrow definitions of development.

In addition to identifying characteristics that are associated with developmental parameters, a developmental perspective focuses on adaptation. By identifying adaptive and maladaptive processes, we can shed light on the origins of favorable versus unfavorable outcomes. This, in turn, provides a basis for inferring causal factors in psychopathology and for designing interventions to prevent or treat psychopathology.

Developmental Theories

Some of the most influential theories of human psychological functioning have been developmental. Psychoanalytic theory, for example, interprets adult psychopathology in terms of childhood precursors involving psychosexual stages, fixations, and regressions (Freud, 1940). Piaget's (1983) theory portrays adult cognitive functioning as an outgrowth of stage-like advances and reorganizations occurring from birth to maturity. Learning theories attribute deviant behavior to the environmental contingencies experienced throughout the individual's lifetime (e.g., Bandura, 1977). Theories about infants' attachment to their caregivers have been used to explain later interpersonal functioning in terms of attachment representations (e.g., Cassidy & Shaver, 1999). Epigenetic theories hypothesize biological sequences that start at conception and continue through the eventual emergence of psychopathology (Rutter, 2006). Such theories focus on how life events influence gene expression, which can then predispose a person toward more versus less adaptive ways of handling stress and challenges. Because different theories can help us understand different aspects of development and psychopathology, the developmental study of psychopathology should not be restricted to the tenets of any single theory.

It may seem obvious that an organism's behavior during a particular developmental period is in many respects an outgrowth of its previous history. It is much less obvious how and to what extent the organism's previous history determines successful adaptation versus pathological maladaptation during particular developmental periods. The differences among developmental theories attest to the variety of possible ways to view development and to the multitude of variables potentially associated with developmental periods and levels. Although this chapter cannot delineate all variables posited by all theories of development, the different theories are rich sources of clues and concepts for guiding the developmental study of psychopathology.

Developmental Considerations in Assessment, Taxonomy, and Diagnosis

Knowledge of development should guide assessment, taxonomy, and diagnosis of psychopathology. For example, knowledge of cognitive development would argue against using interviews that require children to communicate evaluations of their own behavioral and emotional problems before they are cognitively capable of making and communicating such evaluations. This may seem obvious. Yet Breton et al. (1995) found that even normal 9- to 11-year-olds failed to understand many questions on the Diagnostic Interview Schedule for Children (DISC; Shaffer, 1992), which is widely used to make psychiatric diagnoses. Knowledge of development is thus needed to tailor assessment methods to the abilities of those who provide the data. Assessment should also be designed to detect possible developmental differences that have not previously been identified.

In designing assessment procedures to detect developmental differences, it is important to be sensitive to developmental variations in the *content* and *patterning* of the relevant phenomena, as well as to parameters that can be measured in a uniform fashion across broad age spans. For example, height, weight, and IQ are expressed as similar parameters for people of all ages. Yet, the physical configurations underlying even the measures of height and weight change markedly across the life span. Thus, children who weigh 40 pounds at age 5 and 120 pounds at age 15 have not merely grown three times as large. Their body proportions have changed, pubertal development has occurred, and their adolescent appearance causes people to treat them differently.

Analogous to height and weight, the cognitive processes that underlie an IQ of 100 at age 5 differ from those that underlie an IQ of 100 at age 15. Unlike height and weight, however, the phenotypic variables that are assessed by IQ tests differ considerably at ages 5 and 15. By the same token, psychopathological conditions that bear the same diagnostic label at different ages, such as attention-deficit/hyperactivity disorder (ADHD), may be phenotypically quite different at different ages, may involve different underlying processes, and may have different consequences for the individual's adaptation. We should therefore be aware that when diagnostic terms such as ADHD are applied to people of different ages they may not actually represent encapsulated disorders that are the same at all ages.

We must also remember that certain behaviors may be considered normal during one developmental period but deviant at earlier or later developmental periods. For example, high levels of motor activity that are normal for preschoolers would be considered deviant in adolescents. Conversely, sexual interests that are normal for adolescents would be considered deviant for preschoolers.

Developmental Methodology

Developmental approaches contribute methodology that is essential for comparing functioning across different

ages and for linking variables assessed at one age with those assessed at other ages. Longitudinal research is a hallmark of developmental methodology that is essential for identifying relations between variables assessed at different ages. Certain classic longitudinal studies have helped to shape views of relations between childhood characteristics and broad aspects of later functioning (e.g., Bayley, 1968; Kagan & Moss, 1962). Other longitudinal studies have revealed developmental relations between childhood characteristics and adult psychopathology (e.g., Reef, Diamantopoulou, van Meurs, Verhulst, & van der Ende, 2010).

Despite their importance, *real-time* longitudinal studies are costly, seldom use broadly representative samples, and take many years to yield results. More practical methods for obtaining longitudinal data have therefore been sought. These include follow-up (or catch-up) studies, in which people assessed at one age are sought for reassessment at a later age, and follow-back studies, which seek to obtain records of the earlier history of people for whom outcomes are known. Follow-up and follow-back studies are usually quicker and cheaper than real-time longitudinal studies. Follow-up and follow-back designs may also be used to select individuals who have low-prevalence risk or outcome characteristics that would be difficult to study with real-time longitudinal designs because it is impractical to repeatedly assess large enough samples of such individuals.

Examples of influential follow-up studies of childhood disorders include Robins's (1966) study of adults who had been assessed at a child guidance clinic many years earlier and a long-term follow-up of autistic children by Kanner, Rodrigues, and Ashenden (1972). Follow-back studies that started with known adult outcomes include comparisons of the early school records of people who differed in adult psychiatric diagnoses (Lewine, Watt, Prentky, & Fryer, 1980).

Because longitudinal research is essential for linking early characteristics with later outcomes, we need practical, cost-effective strategies for longitudinal research on psychopathology. In later sections, we present research designs that are especially useful for illuminating the developmental course of psychopathology.

THE PSYCHOPATHOLOGY COMPONENT

We use the term *psychopathology* broadly to encompass persistent behaviors, thoughts, and emotions that are likely to impede the accomplishment of developmental tasks necessary for long-term adaptation. For children, the important developmental tasks include at least the following: Regulation of biological functions related to eating, sleeping, elimination, and energy expenditure; maintenance of close relationships with family and peers; acquisition of academic and social skills; preparation for adult occupational and social roles; and formation of a stable sense of personal identity. For adults, important developmental tasks include achievement of mature occupational and social roles; maintenance of close personal relationships; responsible citizenship; and for those who have children, responsible parenthood and creation of loving, healthy home environments. For older adults, important developmental tasks include constructive coping with the aging process; changing or giving up occupational roles; facing the loss of friends and loved ones, as well as one's own demise; maintaining a sense of independence; and preserving close attachments without becoming burdensome.

A key aim of this chapter is to advance our ways of thinking about psychopathology. Because psychopathology can be conceptualized in different ways for different purposes, it is important to consider the relative utility of various models for assessment, taxonomy, and diagnosis.

Nosologically Based Models

Nosological models for psychopathology originated in the nineteenth century with efforts to bring psychopathology out of the realm of demonology and into the realm of physical medicine. An important thrust of these efforts was to model the classification of mental disorders on classifications of diseases, which are called *nosologies*.

Contemporary psychiatric nosologies stem largely from Emil Kraepelin's (1883) efforts to construct categories of major adult disorders for which, Kraepelin hoped, different physical causes would eventually be found. Kraepelin's categories included schizophrenia (originally called dementia praecox, i.e., premature or early dementia) and manic-depressive (bipolar) disorder, which have remained cornerstones of psychiatric nosologies ever since. As revealed by the cartoon in Figure 2.1 that Kraepelin drew for the 1896 Heidelberg Bier Zeitung (Beer Newspaper), he did not want diagnoses to be taken too seriously. By the 1915 version of his nosology, Kraepelin included disorders thought to be of psychological origin. He also included personality disorders, which he viewed as occupying a border region between illness and eccentricity.

Kraepelin's nosology was intended to be descriptive in the sense of being based on observations of patients. Some disorders that were initially described in terms of both mental and physical abnormalities were eventually found



Figure 2.1 Cartoon by Emil Kraepelin in the 1896 Heidelberg *Bierzeitung* (Beer Newspaper). In English translation, the caption says, "Psychiatrists of Europe! Protect your most sacred diagnoses!"

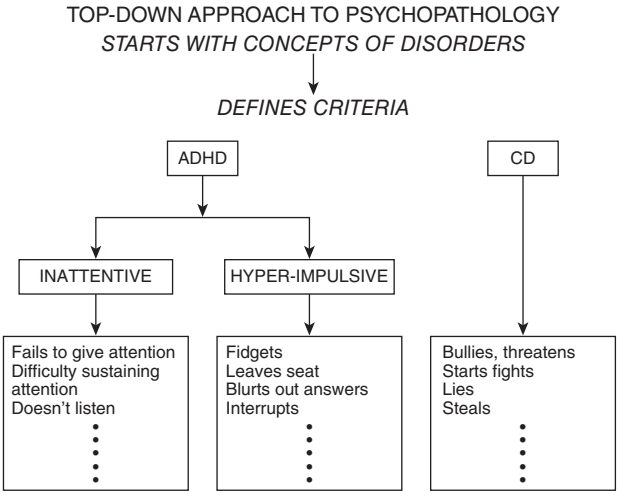


Figure 2.2 The top-down approach to assessment and taxonomy of psychopathology. *Source:* Copyright by T. M. Achenbach. Reproduced by permission.

to have specific physical causes. An example is general paresis, which involves mental and physical deterioration that by the late nineteenth century was found to be caused by syphilitic infection. Another example is Down syndrome, which Langdon Down described in terms of intellectual disability and physical abnormalities in 1866, although it was not until 1959 that the cause was found to be a chromosomal abnormality (Lejeune, Gautier, & Turpin, 1959/1963).

Efforts to describe disorders clinically and then to find physical etiologies have thus yielded some notable successes, especially the discovery that syphilis caused paresis, which supported the physical disease model for psychiatric nosology. On the other hand, discovery of the etiology of Down syndrome had to wait for nearly a century of advances in genetic research.

Kraepelinian diagnostic categories have long molded clinical practice, theory, training, and funding for research and services. In the United States, versions of Kraepelinian nosology have been embodied in successive editions of the American Psychiatric Association's (1952, 1968, 1980, 1987, 1994, 2013) *Diagnostic and Statistical Manual* (*DSM-I, DSM-II, DSM-III, DSM-III-R, DSM-IV, DSM-5*). At the international level, the *International Classification of Diseases (Ninth Edition, ICD-9; Tenth Edition, ICD-10;* World Health Organization, 1978, 1992) has also embodied versions of Kraepelinian nosology.

The Top-Down Strategy

In using nosological models for the *DSM* and *ICD*, experts formulate diagnostic categories and then choose criteria for defining the categories. In effect, the experts start at the top with their concepts of diagnostic categories and then work down to criteria for the categories, as illustrated in Figure 2.2. The diagnostic criteria for some disorders state that symptoms and impairment must be present in more than one context. However, except for including IQ tests in criteria for intellectual disability, neither the *DSM* nor the *ICD* specifies assessment operations for obtaining and using data. Instead, for each case, the diagnostician decides what data to obtain, what assessment methods and sources to use, and how to combine data into yes-or-no decisions about each symptom. The diagnostic data may thus differ greatly from case to case, but the same fixed decision rules are used to make diagnoses for different cases.

Empirically Based Models

For disorders having no known physical cause, an alternative to nosological models is to use statistical procedures to identify problems that tend to co-occur in large samples of people. Sets of statistically co-occurring problems are known as *syndromes*. (The original Greek meaning of *syndrome* was "the act of running together"; Gove, 1971, p. 2320.) The empirical identification of syndromes does not imply any assumptions about the cause of the syndromes. Some syndromes may ultimately be found to have physical causes, while others may be found to have experiential causes or combinations of physical and experiential causes.

Efforts to develop *empirically based models* for child psychopathology began during the 1940s in reaction to the lack of diagnostic categories for childhood disorders

(e.g., Hewitt & Jenkins, 1946). The advent of powerful computers in the 1960s facilitated factor analyses of large pools of problem items scored for large samples of children (e.g., Achenbach, 1966; Quay, 1964). Because the empirically based approach uses statistical analyses to aggregate problems that are then scored in terms of syndromes, it is also known as the *dimensional, quantitative,* or *multivariate* approach.

The Bottom-Up Strategy

The empirically based approach can be described as working from the bottom up because it starts with quantitative ratings of many specific problems obtained for many individuals from informants who see them under various conditions. The item ratings are then analyzed statistically to identify syndromes of problems that tend to occur together, as illustrated in Figure 2.3. Each syndrome is scored by summing ratings of the items that comprise the syndrome. Norms for syndrome scales are then constructed from ratings of large representative samples of people.

Variations on the Bottom-Up Strategy. In addition to empirically based syndrome scales, numerous scales have been constructed according to criteria other than empirically identified associations among problems, even though factor analysis may have been employed as well. As an example, the Behavior Assessment System for Children (BASC) was developed from an initial set of constructs chosen by Reynolds and Kamphaus (1992). Collections of items were written to conform to a priori definitions of the constructs. An item was assigned to a scale if it was "clearly linked in terms of content to the construct underlying its scale" (p. 72). Although factor analyses were performed, items were moved or deleted from the obtained factors on the basis of "reasonableness of content for the hypothesized scale dimension"; "consistency of item placement across age levels and . . . between parent and teacher respondents"; "the need for each scale to have a sufficient number of discriminating items to be reliable"; and "clinical judgment." As an example, of clinical judgment, Reynolds and Kamphaus (p. 73) stated, "The rating scale item "Says 'I want to die' or 'I wish I were dead'" was retained on the Depression scale despite low loadings because of its clear relevance." Also, "Several pairs of final scales initially were part of a single scale. Examples include Depression/Withdrawal and Hyperactivity-Attention Problems" (p. 80).

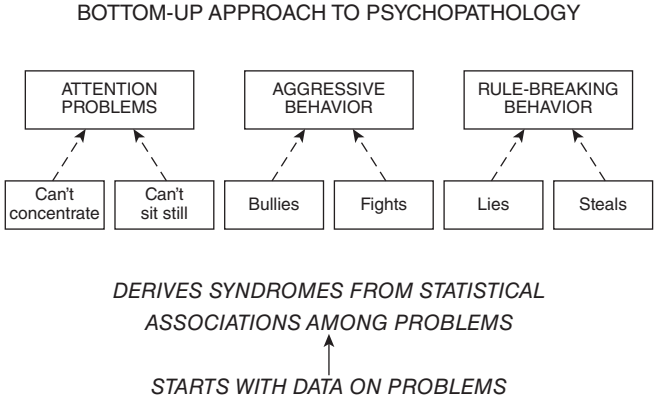


Figure 2.3 The bottom-up approach to assessment and taxonomy of psychopathology. *Source:* Copyright by Achenbach, T. M., Reproduced with permission.

A FRAMEWORK FOR THE DEVELOPMENTAL STUDY OF PSYCHOPATHOLOGY

To advance the developmental study of psychopathology, it is helpful to have a conceptual framework that highlights important issues to be addressed. Because no single research study can simultaneously address all the issues, programmatic research is needed to improve the assessment, taxonomy, and diagnosis of psychopathology.

Developmental Differences

We previously stressed the importance of developmental parameters in all research on psychopathology. To obtain an accurate picture of psychopathology, it is essential to gear assessment procedures to the developmental level of the participants, to determine what features are associated with clinical deviance at each level, and to construct taxonomies that reflect features characterizing each developmental level. The features that discriminate between different kinds of deviance are apt to change with development. It is therefore important for assessment and taxonomy to reflect developmental changes occurring in individuals, so that the same individuals can be studied longitudinally. Both the developmental differences and the links between developmental levels should be rigorously tested.

Sources of Data

Stemming largely from nosological models, clinical interviews have long been the main sources of data for assessing psychopathology. Beginning in the 1970s, standardized diagnostic interviews (SDIs) were developed to make yes-or-no decisions about whether adult patients met specific research diagnostic criteria (RDC) for major mental disorders, such as schizophrenia and bipolar disorders (e.g., Overall & Hollister, 1979). The SDIs included the

semistructured Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) and the highly structured Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981).

The explicit criteria embodied in various versions of RDC laid the foundations for *DSM-III* (American Psychiatric Association, 1980). Departing from the narrative-inferential format of *DSM-I* and *DSM-II* (American Psychiatric Association, 1952, 1968), *DSM-III* defined disorders in terms of lists of symptoms and other criteria, such as age-of-onset and duration of the symptoms. According to *DSM-III* and its successors, *DSM-III-R*, *DSM-IV*, and *DSM-5* (American Psychiatric Association, 1987, 1994, 2013), diagnosticians are to make yes-or-no decisions about whether patients meet each of the criteria required for a particular disorder. If a patient is deemed to meet the requisite criteria, the patient is diagnosed as having the disorder. Diagnostic criteria and decision rules are thus far more explicit than prior to *DSM-III*. However, neither *DSM-III* nor its successors specify assessment operations for determining whether the diagnostic criteria are met, except for including IQ tests in criteria for intellectual disability. As one approach to operationalizing *DSM* diagnostic criteria, the SDIs that had been developed in the 1970s to make RDC diagnoses were subsequently revised to assess *DSM-III*, *DSM-III-R,* and *DSM-IV* criteria (e.g., Shaffer et al., 2000).

Multi-Informant Data for Children

Some of the SDIs for adults were modified for diagnosing children. Examples include the Kiddie-SADS (Ambrosini, 2000; Puig-Antich & Chambers, 1978), which is a child version of the SADS (Endicott & Spitzer, 1978), and the Diagnostic Interview Schedule for Children (DISC; Costello, Edelbrock, Dulcan, Kalas, & Klaric, 1984; Shaffer et al., 2000), which is a child version of the DIS (Robins et al., 1981). When it became apparent that children cannot provide sufficient accounts of their own psychopathology, SDIs were developed to obtain diagnostic information from the children's parents (e.g., DISC-P; Costello et al., 1984; Shaffer et al., 2000).

Unfortunately, the parallel SDIs for children and their parents did not solve the problems of operationalizing *DSM* criteria for child psychopathology. Numerous studies have found little agreement between *DSM* diagnoses made from DISC interviews with children (including adolescents) and their parents (e.g., Costello et al. 1984; Jensen et al., 1999; Verhulst, van der Ende, Ferdinand, & Kasius, 1997). Furthermore, meta-analyses have revealed only low to moderate agreement between most diagnoses obtained from child, parent, and adult SDIs and clinical diagnoses based on other data (Rettew, Doyle, Achenbach, Dumenci, & Ivanova, 2009).

Perhaps we should not be surprised by the poor agreement between diagnoses made from SDIs of children and their parents, or between SDIs and diagnoses made by clinicians who use other data. When required to give yes-or-no answers to questions about psychopathology, children and their parents are apt to draw on different perceptions and memories of the children's functioning, to be differently motivated to report, distort, or withhold information, to understand questions differently, and to be differently affected by interviewers and the interview situation. When making diagnoses, clinicians are also apt to differ from children and parents with respect to their information about the children, their memories of it, how they interpret their information, and how they combine their information and impressions when selecting diagnoses.

Meta-analyses of many studies have yielded a mean Pearson correlation *(r)* of .60 between reports of children's problems by people who play similar roles with respect to the children, such as pairs of parents, pairs of teachers, pairs of mental health workers, and pairs of observers (Achenbach et al., 1987). Between pairs of informants who play different roles with respect to the children, such as parents versus teachers and teachers versus mental health workers, the mean *r* was .28. And between children's self-reports and reports by other people, the mean *r* was .22. Subsequent findings of informant discrepancies have been so consistent with the meta-analytic findings that they are regarded as being among "the most robust findings in clinical child research" (De Los Reyes & Kazdin, 2005, p. 481).

Detailed comparisons of parent versus self-ratings of the same problem items for youths in twenty-five societies and of parent versus teacher ratings of the same problem items for children attending schools in twenty-one societies have revealed great consistency across societies in the directions of discrepancies: Youths tend to rate their own problems higher than their parents rate them, and parents tend to rate their children's problems higher than their teachers rate them (Rescorla et al., 2013, 2014). Although the data for the Achenbach et al. (1987) meta-analyses came mainly from rather similar Western societies, the findings for parent-youth and parent-teacher discrepancies came from samples of very diverse societies in Africa, Asia, Australasia, Europe, Latin America, the Middle East, and North America. The directions of informant discrepancies were quite consistent across societies that vary greatly in culture, economic development, ethnicity, geographical region, language, political system, religion, and schooling.

Furthermore, particular kinds of raters (e.g., parents and youths) agreed very well about which problems were common (i.e., received high ratings) versus rare (i.e., received low ratings). Nevertheless, in each society the discrepancies within particular parent-youth and parent-teacher dyads varied greatly, with some dyads agreeing much better than other dyads. As addressed later in this chapter, numerous other multicultural findings reveal considerable similarity across very different societies, but major variations among members of each society. In any event, the evidence clearly shows that no one informant's reports regarding child psychopathology can substitute for all other reports.

Multi-Informant Data for Adults

Although the need for multi-informant data to assess child psychopathology is now widely recognized, assessment of adult psychopathology still relies mainly on data from the adults who are being assessed. Yet research shows that diagnoses based only on adults' self-reports are not likely to agree with diagnoses based on other sources of data. For example, studies of agreement between adults' self-reports of problems and reports by people who know the adults have yielded average correlations in the .20s to .40s (Achenbach et al., 2005; Meyer, 2002; Meyer et al., 2001). In reviewing research on diagnoses of adult psychopathology, Meyer et al. (2001) concluded that: "There was meager correspondence between diagnoses derived from a single clinician using (clinical interviews) and the diagnoses derived from the multimethod evaluations . . . after correcting for agreements due to chance, about 70% of the interview-based diagnoses were in error" (p. 151). Meyer et al. also found that the mean kappa between diagnoses based on self-reports versus multi-informant data was only .18. Furthermore, even when adults were interviewed twice, once by trained lay interviewers using the DIS and once by psychiatrists using the Schedules for Clinical Assessment in Neuropsychiatry (SCAN), agreement between the two interviews was extremely low, as exemplified by a kappa = .20 for major depressive disorder (Eaton, Neufeld, Chen, & Cai, 2000).

For elders whose mental competence is in question, it is generally recognized that data need to be obtained from people who know the elders, such as spouses, grown children, and caregivers. Consequently, parallel assessment instruments that obtain and systematically compare assessment data from multiple informants should be routinely used to assess elders' functioning (Achenbach, Newhouse, & Rescorla, 2004).

To advance our developmental understanding of psychopathology, we should apply lessons already learned about needs and methods for cross-informant assessment of children to the assessment of adults and elders. The aspects of functioning to be assessed, the sources of data, and the assessment instruments must, of course, be geared to the developmental levels of the people who are being assessed.

Epidemiological Aspects

Many studies of development and psychopathology employ samples chosen for convenience rather than for representativeness with respect to larger populations. This is understandable in light of the need to find people who are willing and able to participate in a particular study. However, convenience samples are often biased by selective factors that limit the generalizability of findings. If our knowledge of psychopathology is derived mainly from people who are conveniently available rather than from representative samples of people, we may be misled by findings for problems that do not actually distinguish people who do not need help from those who do need help.

As an example, both the early psychoanalytic and behavioral literature used children's fears of animals to model theories of the etiology and treatment of psychopathology. Sigmund Freud (1909/1953), for example, used a 145-page case study to illustrate the psychodynamics of Little Hans's horse phobia. In the behavioral tradition, Watson and Rayner (1920) experimentally conditioned Little Albert to fear a white rat, and Jones (1924) used Watson's principles to treat 2-year-old Peter's fear of a white rabbit.

Both the psychodynamic and behavioral traditions thus made children's fears paradigmatic for the subsequent literature on psychopathology. Yet, epidemiological studies have shown that fears of specific animals, situations, or places (other than school) are not necessarily more common among children considered to be clinically deviant than among representative samples of nonreferred children (Achenbach, Howell, Quay, & Conners, 1991; Achenbach & Rescorla, 2000, 2001). Epidemiological research has also shown that specific fears are reported for *fewer* clinically referred than nonreferred adults (Achenbach & Rescorla, 2003).

Other epidemiological findings have indicated that certain problems employed as criteria for nosological categories are not reported more often for clinically referred than nonreferred children. "On the go," for example, is a criterial symptom for the *DSM* category of ADHD (American Psychiatric Association, 1980, 1994, 2013). Yet this item was endorsed more often for a nationally

representative sample of *nonreferred* children than for demographically matched *referred* children (Achenbach et al., 1991). This may reflect favorable connotations of "on the go" as characterizing children who are energetic and busy in constructive ways. Other nosological criteria have likewise been chosen without epidemiological evidence that they validly identify individuals who need help.

Diagnoses of behavioral and emotional disorders seldom depend on a single deviant characteristic. Instead, most disorders involve problems that many people manifest to some degree at some time in their lives. To identify people as being clinically deviant, we therefore need to know the distribution of particular problems among people considered to be relatively normal, as well as among those who are considered to need professional help. Because many selective factors affect referral to a particular service, we also need data on people who are seen in different services and settings. And we need to know the outcomes of particular problems in both referred and nonreferred populations. This requires that epidemiological samples be reassessed longitudinally to determine which problems have relatively good versus poor outcomes when treated versus untreated and what other variables predict outcomes. Such research is important both to determine what problems distinguish people who need help from those who do not, and to identify risk and protective factors on which preventive and therapeutic efforts should focus.

Multivariate Aspects

Because so many different kinds and degrees of problems may evoke concern, it is important to standardize the scoring and analysis of problems. Because it is so hard to assess and aggregate multiple problems for large samples of people, multivariate statistical methods are needed. All scores obtained from self-reports, informants' reports, observations, and tests are subject to sampling and measurement error. However, by using multivariate statistical methods, we can aggregate scores on numerous problems from multiple sources to identify patterns of co-occurring problems. This is analogous to computed tomography (CT) scanning, whereby numerous low-grade x-ray pictures are integrated by computer to produce high-grade images. Once we identify patterns of problems, we can use the patterns to group people for research on the etiology, course, prognosis, and effectiveness of treatment for each pattern. Those multivariate patterns that are found to be reliable and to validly discriminate between people who differ in other important ways can then serve taxonomic functions, as detailed in later sections.

Operational Definitions

Operational definitions of variables provide basic tools for scientific research. Although scientific thought and theory also involve abstractions that are not operationalized, empirical tests of scientific ideas typically require specification of variables in terms of assessment operations. The assessment operations provide the empirical content of the science.

Discussions of psychopathology are replete with terms that are used as if their meaning were self-evident. Terms (and their abbreviations) such as conduct disorder (CD), oppositional defiant disorder (ODD), antisocial personality disorder (APD), and ADHD are common examples from the *DSM* nosology. Terms such as emotional disturbance (ED) and learning disability (LD) are used with similar aplomb in special education circles, reflecting the influence of Public Law 94-142 (P.L. 94-142), the Education of the Handicapped Act (*Federal Register*, 1977, 1981), which specifies eligibility for special education services (reauthorized as P.L. 101-476, Individuals with Disabilities Act, 1990; P.L. 105-17, 1997).

The need for operational definitions has long been recognized in efforts to improve nosologies. In a diagnostic classification of childhood disorders that was proposed half a century ago, the Group for the Advancement of Psychiatry (GAP; 1966) "attempted to set forth operational definitions of clinical categories" (p. 209). *DSM-III* and its successors have also been heralded as providing operational definitions (e.g., Narrow et al., 2013, p. 71; Rapoport & Ismond, 1996, p. 13). However, neither the GAP classification nor the *DSM* has defined behavioral and emotional disorders according to specific assessment operations. But what about the *DSM*'s criteria for each diagnosis? These criteria do not constitute operational definitions, because no assessment operations are specified for determining whether a person manifests each criterial feature. Nor are operations specified for combining often conflicting data from multiple sources. The lack of operational definitions for disorders may be one reason that poor reliability and low cross-informant agreement have been found for GAP and *DSM* diagnoses (e.g., Beitchman, Dielman, Landis, Benson, & Kemp, 1978; Jensen & Weisz, 2002; Lewczyk et al., 2003; Shaffer et al., 2000).

Categories such as those specified by the *DSM* and P.L. 101-476 serve important administrative functions, such as providing guidelines for reimbursement. However, these categories were not derived from empirical findings on distinctions among disorders in actual samples of troubled people. The categories should therefore not

be equated with types of people or types of disorders. Although people may indeed have the kinds of problems implied by such categories, people do not necessarily come packaged according to these categories. In other words, we should not automatically assume that individuals can be validly classified according to categories such as attention-deficit/hyperactivity disorder (ADHD), CD, ODD, APD, ED, or LD. Unless we empirically test (1) the associations among features that are used to define categories and (2) the power of the criterial features to discriminate between individuals who do versus do not need help for their problems, we cannot know how best to distinguish among kinds of disorders. To improve our ability to distinguish between individuals who do versus do not need help, as well as among patterns of clinical deviance, we need programmatic research that yields more valid groupings than most administrative categories provide. Such research is needed to advance our developmental understanding of psychopathology.

THE ROLES OF ASSESSMENT AND TAXONOMY IN THE DEVELOPMENTAL STUDY OF PSYCHOPATHOLOGY

To improve our ways of distinguishing the normal from the deviant and one kind of deviance from another, it is helpful to distinguish between two related tasks. One task is to *assess individuals* to identify their distinguishing features. The second task is to *construct taxonomies* for grouping individuals according to their distinguishing features. These tasks are essential steps in targeting the disorders to be studied in each developmental period. They also form the basis for diagnostic decisions about individuals. To improve diagnostic decision making, we need reliable and valid procedures for identifying the distinguishing features of individuals, for grouping individuals according to their distinguishing features, and for effectively integrating all the relevant data into optimal conclusions. To clarify the different tasks, we first address assessment and taxonomy as scientific endeavors that can be better understood if we avoid confusing them with the term *diagnosis*, whose multiple meanings we address later.

Although diagnosis is an essential part of clinical services, scientific efforts are needed to improve both diagnosis and its links to services. The following sections therefore focus on the scientific process, which "proceeds in endless cycles of observation and measurement, hypothesis testing, and the development of theory, a process dependent on a valid system of classification or taxonomy" (McClellan & Werry, 2000, p. 19).

Assessment

Assessment refers to gathering data with which to identify the distinguishing features of individuals. Every individual can be distinguished from other individuals in many ways. Effective assessment requires selecting optimal features for the kinds of decisions to be made. To identify optimal features, we need data from representative samples of individuals with which to test the distributions, validity, and utility of various features.

Psychometric principles provide guidance for determining the methodological adequacy of assessment data. As applied to the assessment of behavioral/emotional/social problems, these principles can be summarized as follows:

1. Assessment should employ standardized procedures.
2. Multiple items should be used to sample each aspect of functioning.
3. Items should be aggregated to provide quantitative scores for each aspect of functioning.
4. Scores should be normed to indicate how an individual compares with relevant reference groups.
5. To be developmentally sensitive, the normative reference groups should be formed according to age levels or other indices of development.
6. To be considered psychometrically sound, assessment procedures must be reliable and valid, although the types of reliability and validity vary with the type of procedure.

In addition to these principles, psychometric theory prescribes specific standards for constructing sound assessment procedures. Psychometric theory does not, however, tell us *which* of the many features of individuals should be assessed. Instead, we should select features that are found to discriminate among individuals whose behavioral/emotional/social problems differ with respect to etiology, course, prognosis, or the most appropriate intervention. To determine which features specifically discriminate between individuals according to these variables, research is needed to test the discriminative power of various features. Rather than attempting to test every possible feature one-by-one, it is preferable to identify sets of features that tend to co-occur. Because assessment of each feature is subject to random variance, scores for sets of covarying features are likely to be more reliable than scores for each separate feature. Furthermore, sets of covarying features are likely to be more useful than separate features for generating and testing hypotheses, as well as for managing individual cases. The task of aggregating covarying features into sets

with which to identify similar individuals is the task of taxonomy, to which we now turn.

Taxonomy

It is helpful to distinguish between *classification* in general and *taxonomy* in particular. Classification refers to any systematic ordering of phenomena into classes, groups, or types. Many classifications are constructed merely for the convenience of users. Administrative classifications, for example, are designed to meet particular organizational needs. The people who design such classifications may start with knowledge of an organization's needs and the ways in which prospective users of a classification customarily view the phenomena to be classified. They then attempt to mesh the users' classificatory behavior with the needs that the classification must meet.

As an example, health insurers construct their own classifications of disorders to decide on reimbursements for mental health services. A key question is whether a particular classification scheme enables the insurer's actuaries to compute costs accurately enough for the insurer to charge rates that are competitive but high enough to be profitable after covering all costs. For third-party payers that are not funded by premiums based on costs, such as Medicaid, the problem is complicated by unpredictable changes in the size and nature of the covered population, variations in the services obtained, providers' techniques for coping with low reimbursement rates, and political vicissitudes. Considering the variety of factors that affect third-party payers' classification schemes, it is no wonder that so many classifications have been devised for reimbursement of mental health services.

In contrast to the broad concept of classification, taxonomy refers to a subset of classifications that are intended to reflect intrinsic differences between individuals assigned to different classes. Taxonomies of plants and animals, for example, are based on features that are intended to capture important differences among species. Various features can be chosen to distinguish among groups of plants and animals, such as physical characteristics, interbreeding, and hypothesized evolutionary history. Although the concept of species is so familiar as to seem self-evident, species is not an intrinsic property of living things. Instead, species is a taxonomic construct that is imposed on subsets of features, such as having four legs or laying eggs. Criteria for a particular species thus consist of certain features that are selected from all the features that could possibly be used to distinguish among individual plants and animals (Levin, 1979).

Are Behavioral and Emotional Disorders Natural Kinds?

Zachar (2000) distinguished between taxonomies that consist of *natural kinds* and those that consist of *practical kinds*. He defined a natural kind as "an entity that is regular (nonrandom) and internally consistent from one instance to the next" (p. 167). Examples include naturally occurring elements such as gold. By contrast, practical kinds "are stable patterns that can be identified with varying levels of reliability and validity" (p. 167).

Zachar argues that taxonomies of psychopathology involve practical kinds rather than natural kinds. Although some may believe that taxonomies of psychopathology should identify natural kinds, both the empirically based approach and the *DSM*'s nosological approach focus on practical kinds. In other words, they both define taxa in terms of sets of features no one of which is both necessary and sufficient to classify an individual's problems as belonging to a particular taxon. Both approaches are thus *polythetic*. This means that both the empirically based approach and the *DSM* specify sets of features from which various subsets of the features can qualify an individual for a taxon.

Constructing Taxonomies of Behavioral, Emotional, and Social Problems

In constructing taxonomies of behavioral/emotional/social problems, we should be aware that the problems can be aggregated according to different taxonomic models. Certain models may be useful for one stage of knowledge or for one developmental stage but not for other stages of knowledge or development. Furthermore, different models may be useful for different kinds of disorders or for different purposes at the same stage of knowledge. Models based on cognitive measures, for example, may be useful for grouping people according to educational needs, whereas models based on measures of behavioral/emotional/social problems may be useful for grouping the same people for management purposes.

We also need to be aware that the features chosen to characterize individuals are abstractions whereby we link individuals who we expect to be similar in other important ways. When we abstract a subset of features from the many features that could potentially be identified, we form a hypothetical construct of a case—a conceptual abstraction intended to capture useful aspects of the case. If our notion of the individual case and our taxonomic grouping of individuals according to certain shared features both involve hypothetical constructs, how should we match our

construct of the individual case to taxonomic models? To answer this question, we must take account of both the methodological possibilities for assessing individuals and the mental processes involved in matching the features of individuals to the features embodied in taxonomic models.

Effects of Categories on Taxonomic Thinking

Although taxonomies might be viewed as inherently categorical, the effects of classifying individuals in terms of categories need to be empirically evaluated. Cognitive studies have shown that training adult participants to classify pictures of people into experimentally manipulated categories caused the participants to exaggerate *similarities* between pictures of people within a category and also to exaggerate *differences* between pictures of people in different categories (Goldstone, Lippa, & Shiffrin, 2001; Queller, Schell, & Mason, 2006). Furthermore, research with preschool children has shown that labeling pictures of people categorically (e.g., "mean" vs. "nice") promoted stereotyped judgments that could be counteracted by showing the children pictures of people labeled in terms of continuous characteristics (e.g., ranging from "really mean" to "really nice") (Master, Markman, & Dweck, 2012). These findings indicate that, for both children and adults, classification of people categorically *reduces differentiation* and *increases stereotyping* of people's characteristics.

Prototypes as Taxonomic Models

Taxonomic categories have traditionally been defined in terms of criteria that are "singly necessary and jointly sufficient" for classifying individual cases (Cantor, Smith, French, & Mezzich, 1980, p. 182). In other words, a case was assigned to a particular category if, and only if, it met all the criteria for that category. Conversely, all cases that met the criteria for a particular category were ipso facto assigned to that category.

Cognitive research indicates that people's mental use of categories does not conform to the classical model whereby cases are categorized according to features that are both necessary and sufficient. As illustrated by familiar categories such as furniture, for example, the objects that people categorize together do not all share the same defining features (Rosch & Mervis, 1975). Instead, certain objects classified as furniture, such as tables and chairs, are unlike other objects that are also classified as furniture, such as lamps and rugs.

Objects that have the most features of a category are viewed as the most typical of the category and are more quickly and reliably categorized than are objects that have fewer of the category's features (Smith & Medin, 1981). For example, a sparrow is more typical of birds than an ostrich is. Furthermore, objects that have features of multiple categories are especially hard to categorize reliably. As an example, a tomato is hard to categorize as a fruit or vegetable because it has features of both categories.

Instead of being rigidly defined by criterial features that must all be present in all members of a category, mental representations of categories comprise sets of imperfectly correlated features known as *prototypes* (Rosch, 1978). Consequently, people judge category membership according to the degree of overlap between the features of a case and the set of prototypical features that define a category. In other words, people judge cases having many features of a prototype to be very typical of the category represented by that prototype. They judge cases that have few features of a prototype to be less typical of that category. Cases that manifest features of more than one prototype are judged to lie on the border between the categories represented by those prototypes.

According to the prototype view, people make quantitative judgments of the resemblance between particular cases and particular categories. That is, the degree of resemblance between a case and a category is judged according to the number of prototypical features that the case shares with the category. Furthermore, if each prototypical feature of a case and of a category can be scored for the intensity or certainty with which the feature is manifested, then the case's resemblance to the category can be quantified in terms of the sum or other aggregation of these scores.

If the human minds that must use taxonomies actually conceptualize categories in terms of quantifiable prototypes, why not use quantifiable prototypes in taxonomies of psychopathology? Quantification of taxa can help to mesh clinical thinking with taxonomies of psychopathology, as well as counteracting stereotypes promoted by categorical approaches. The possibilities for doing this will be presented after we consider conventional diagnostic thinking.

DIAGNOSIS

The term *diagnosis* conveys an aura of clinical authority, implying that it reveals an essential truth about what is really wrong with a patient. Yet the term has multiple meanings that sow confusion when we move from research tasks conceptualized in terms of assessment and taxonomy to clinical practice conceptualized in terms of diagnoses. To clarify relations between the research tasks and clinical practice, it is helpful to distinguish among the different meanings of diagnosis discussed next.

Diagnostic Processes

Diagnostic processes involve gathering the data needed for diagnostic decisions. Such processes are analogous to what we earlier defined as assessment. However, if data are gathered only to select diagnoses, *diagnostic processes* imply a narrower focus than assessment does.

Formal Diagnoses

Formal diagnoses involve assigning cases to the categories of a diagnostic classification. Accordingly, a leading psychiatric diagnostician, Samuel Guzé (1978), defined diagnosis as "the medical term for classification" (p. 53). This is the sense in which the categories of the *DSM* and *ICD* nosologies are diagnoses. When a clinician states that a child meets the *DSM* criteria for conduct disorder, for example, the clinician is making a formal diagnosis.

Diagnostic Formulations

Diagnostic formulations are efforts to elucidate multiple aspects of an individual's condition. When a clinician interprets a child's problems in terms of biological vulnerabilities, developmental history, family dynamics, stress, and rejection by peers, for example, the clinician makes a diagnostic formulation. A diagnostic formulation should weave all the findings of diagnostic processes into a comprehensive picture of the case. The diagnostic formulation thus provides a broader basis for developing treatment plans than a formal diagnosis does. Yet, it is by means of the formal diagnosis that the individual case is linked to others like it.

A formal diagnosis helps the clinician apply existing knowledge about a class of cases to a diagnostic formulation for a new case. For knowledge about a class of cases to be accurately applied to a new case, reliable procedures are needed for gathering data, integrating the data into a diagnosis, and selecting the appropriate formal diagnosis. Furthermore, the system from which the formal diagnosis is selected must be reliable, must be valid with respect to important correlates of its diagnostic categories, and must encompass cases like the one being diagnosed. To be credible, diagnoses should thus meet the same methodological standards as assessment and taxonomy do. Like assessment and taxonomy, diagnosis involves gathering data to identify the distinguishing features of individuals and grouping individuals according to their distinguishing features. Whichever terminology we use, the value of such procedures ultimately depends on whether they help people.

Comorbidity Issues

Since the introduction of explicit diagnostic criteria by *DSM-III* (American Psychiatric Association, 1980), many people have been found to meet the criteria for more than one diagnostic category (e.g., Costello et al., 1984; Weinstein, Noam, Grimes, Stone, & Schwab-Stone, 1990). Termed *comorbidity*, the tendency for people to meet criteria for multiple categories has a variety of implications. The apparent co-occurrence of two disorders could mean, for example, that one disorder results from the other; or that the same risk factors lead to both disorders; or that the two disorders are not really separate but are manifestations of the same underlying condition (Achenbach, 1991a; Caron & Rutter, 1991).

Neither the *DSM* nor the *ICD* provides evidence that each of their diagnostic categories validly represents a type of disorder that exists independently of disorders represented by other categories. Nor do the *DSM* and *ICD* specify assessment operations for reliably discriminating between disorders. When assessment procedures have been designed to operationalize *DSM* criteria for disorders, high rates of comorbidity have often been found. For example, two studies showed that 96% of boys who met *DSM* criteria for CD also met criteria for ODD (Faraone, Biederman, Keenan, & Tsuang, 1991; Walker et al., 1991). In addition, the *DSM-III-R* field trials found that 84% of clinic-referred children who met criteria for CD also met criteria for ODD (Spitzer, Davies, & Barkley, 1990). The very high rates of overlap between diagnoses suggest that the diagnostic criteria may not validly distinguish between different disorders.

Unidirectional Versus Bidirectional Comorbidity

The specific rates of comorbidity reported in many studies may be misleading, because they reflect computation of the overlap between diagnoses in only one direction. That is, they reflect the percentage of individuals having Diagnosis A who also have diagnosis B. If diagnoses A and B have different prevalence rates in a sample, this unidirectional computation yields a different rate of comorbidity than would be obtained by computing the percentage of individuals having Disorder B who also have Disorder A. For example, suppose that fifty children in a sample of one hundred obtain a diagnosis of CD (Diagnosis A) and that forty-eight of these fifty obtain a diagnosis of ODD (Diagnosis B). The unidirectional comorbidity of ODD (Diagnosis B) among children who have CD (Diagnosis A) is 48/50 = 96%. However, if eighty of the children have ODD and forty-eight of these have CD, the

unidirectional comorbidity of CD among children who have ODD is 48/80 = 60%. By recomputing published comorbidity rates bidirectionally (i.e., as the mean of the comorbidities of A to B and B to A), McConaughy and Achenbach (1994) obtained quite different rates from those based on the unidirectional comorbidities. When comorbidity is conceptualized in terms of the co-occurrence of two categorically defined disorders, it is thus important to consider the bidirectional rates of comorbidity between the disorders.

Berkson's Bias

Apparent comorbidity among disorders may also be misleading in other ways, as detailed elsewhere (Achenbach, 1991a; Caron & Rutter, 1991). Suppose, for example, that individuals manifesting a particular disorder have *X* probability of referral. Suppose, too, that individuals manifesting another disorder have *Y* probability of referral. Even if the disorders do not tend to co-occur in the population as a whole, people who have both disorders have $X + Y - XY$ probability of referral. This is obviously higher than the referral rate for individuals who have only *X* or *Y* probability of referral. If we study only referred people, we may conclude that the two disorders tend to occur together. Yet, if we study the entire population (including nonreferred people), we might find that the two disorders have no more than chance co-occurrence, because individuals having only one disorder without the other are much more common in the nonreferred population than in the referred population.

Known as *Berkson's bias* (Berkson, 1946), the tendency of apparent comorbidity to be inflated by referral biases is one of many artifactual ways in which findings of co-occurrence between even independently verifiable disorders can arise. The different effects of referral biases on different problems has been demonstrated by comparing comorbidities obtained among empirically based syndromes in demographically similar referred and nonreferred samples (McConaughy & Achenbach, 1994).

Implications of Comorbidity

If two disorders are operationally defined according to mutually independent assessment procedures, and if the two disorders are demonstrated to occur together with greater than chance frequency in unbiased samples of the general population, then the comorbidity of these particular disorders may be quite informative. However, if diagnoses are not based on operational definitions of empirically separable disorders, then findings of comorbidity may merely reflect a lack of clear boundaries between diagnostic categories or our inability to validly distinguish between disorders that do not really co-occur with more than chance frequency.

Rather than taking findings of comorbidity at face value as reflecting the co-occurrence of two distinct disorders, we should examine the diagnostic system itself for possible artifactual sources of apparent comorbidity. The high rate of overlap found between diagnoses of ODD and CD (Faraone at al., 1991; Spitzer et al., 1990; Walker et al., 1991), for example, invites scrutiny of the diagnostic system to determine whether ODD and CD truly represent separate disorders. The arbitrariness of the distinctions between the diagnostic categories of ODD and CD is highlighted by the major changes they underwent from *DSM-III* to *DSM-III-R,* with further changes in *DSM-IV*. *DSM-III* (American Psychiatric Association, 1980) listed five criteria for ODD, of which only two criteria needed to be met for the diagnosis. *DSM-III-R* (American Psychiatric Association, 1987), by contrast, listed nine criteria, of which five were needed for the diagnosis. *DSM-IV* listed eight criteria, of which four were needed for the diagnosis. Furthermore, the *DSM* criteria for diagnoses of CD also underwent major changes from *DSM-III* to *DSM-III-R*, with further changes in *DSM-IV*. *DSM-III* provided four distinct categories, plus a residual category of Atypical CD. *DSM-III-R*, by contrast, provided only one set of explicit criteria for CD, although once the prescribed number of criteria were met, the disorder could be categorized as "group type," "solitary aggressive type," or "undifferentiated type," according to the diagnostician's judgment. *DSM-IV* dispensed with these types in favor of "childhood onset type" versus "adolescent onset type." *DSM-5* retains the *DSM-IV* symptom criteria for ODD and CD (American Psychiatric Association, 2013).

Classification systems often need to incorporate changes based on new knowledge. However, the *DSM* revisions have not been derived from empirical tests of the co-occurrence of particular features or of whether the criteria for ODD and CD actually discriminate between distinctly different disorders in representative samples of children. Furthermore, no algorithms have been provided for calibrating diagnoses made from one edition of the *DSM* with those made from previous editions. It should therefore not be surprising that very different distributions of ODD and CD have been obtained when applying criteria from different editions of the *DSM* to the same children (Lahey et al., 1990).

DSM-III-R and *DSM-IV* specified that the diagnosis of ODD was not to be made if the criteria for CD were met.

Did this mean that ODD is a mild version of CD, a developmentally early version, or a by-product? Or did it mean that the two disorders are intrinsically related in some other way? Similar questions arise from overlaps among other diagnostic categories that are not derived from actual data on relations among problems. Based on a detailed examination of these and other issues, Lilienfeld, Waldman, and Israel (1994) cautioned that the application of the term "comorbidity" to psychopathology "encourages the premature reification of diagnostic entities" (p. 71) that have not actually been validated.

In an effort to consider the value of dimensional (quantitative) diagnostic criteria for *DSM-5*, the American Psychiatric Association appointed a task force on dimensional criteria. Evidence supporting dimensional criteria was presented by researchers and clinicians specializing in many areas of psychopathology (Helzer et al., 2008). Despite enthusiasm for dimensional criteria among members of the task force, *DSM-5* does not provide dimensional criteria (American Psychiatric Association, 2013).

LONGITUDINAL DESIGNS FOR THE DEVELOPMENTAL STUDY OF PSYCHOPATHOLOGY

Many kinds of research designs can be used in the developmental study of psychopathology. However, *longitudinal* designs, in which the same people are assessed at two or more points in their development, provide the most direct tests of relations between developmental parameters and patterns of problems. In the following sections, we illustrate some variations on traditional longitudinal designs that may be especially useful for developmental research related to assessment, taxonomy, and diagnosis.

Accelerated Longitudinal Designs

The ability of traditional longitudinal designs to shed light on psychopathology has been limited by the difficulty of maintaining participant samples, research teams, and funding over long periods. Moreover, most longitudinal studies have been handicapped by their failure to compare different birth cohorts and by their use of very different assessment procedures at different ages. Such limitations affect real-time, follow-up, and follow-back studies alike.

Life span developmental psychologists have emphasized the need to take account of differences related to birth cohorts and times of measurement, as well as differences related to age (e.g., Baltes, 1987). Unfortunately, it is difficult to include cohort and time-of-measurement comparisons in traditional longitudinal research designs.

One way to improve longitudinal research on psychopathology is to assess multiple birth cohorts by the same methods over several uniform intervals. Such designs have been variously called *convergence* (Bell, 1953), *mixed-longitudinal* (van't Hoff, Roede, & Kowalski, 1991), and *cohort-sequential* (Baltes, Cornelius, & Nesselroade, 1979). Because several birth cohorts are assessed in the same way over the same period, the longitudinal findings for each cohort can be compared with the findings for each other cohort. We can thus determine whether changes with age are similar in all cohorts or whether the changes differ between cohorts or times of measurement.

An additional strength of multiple birth cohort longitudinal designs is that they can be analyzed so as to reveal developmental sequences over a longer period of time than the real time spanned by the study. When analyzed in this way, they are called *accelerated longitudinal designs*. How can we perform the magic of accelerating longitudinal research? The logic is as follows:

1. At Time 1, individuals from several birth cohorts are assessed in a uniform fashion. For example, in June 2018, a test is administered to cohorts of children who are 6, 7, 8, or 9 years old (designated as Cohorts 6, 7, 8, and 9).
2. At Times 2, 3, and 4, the same children are assessed again in the same way. In other words, Cohorts 6, 7, 8, and 9 are reassessed in 2019, 2020, and 2021.
3. Individuals from one cohort are matched to individuals in another cohort. In our example, children from Cohort 6 are partnered with children in Cohort 7 with respect to demographic variables and to their assessment scores obtained at ages 7 and 8. That is, a particular Cohort 6 middle SES boy named Adam is matched to a particular Cohort 7 middle SES boy named Scott whose test scores at ages 7 and 8 were similar to the scores obtained by Adam at ages 7 and 8.
4. The accuracy of predicting scores obtained by individuals in one cohort from earlier scores obtained by matched partners in another cohort is then tested. In our example, we compute the correlation between scores obtained at age 7 by Cohort 6 children and scores obtained at age 9 by their matched partners from Cohort 7. For purposes of comparison, we then compute the correlation between the scores obtained at age 7 by the Cohort 6 children and their own scores at age 9. Suppose the correlation between age 7 and 9 scores within Cohort 6 is significant. Suppose also that the between-cohort correlation from

scores obtained at age 7 by Cohort 6 children to the age 9 scores obtained by their matched Cohort 7 partners is of the same magnitude as the correlation from age 7 to age 9 within Cohort 7. These findings would indicate that the score obtained at age 7 by a Cohort 6 child, such as Adam, can be used to predict the score obtained at age 9 by a Cohort 7 child, such as Scott, with as much accuracy as prediction of Scott's age 9 score from his own age 7 score.

5. If the findings outlined in Step 4 are replicated for the other combinations of cohorts in the study, it may become possible to create matched sets of partners across the four cohorts to predict scores obtained by Cohort 9 children at age 12 from scores obtained by Cohort 6 children at age 6. An accelerated longitudinal study that requires three years of real time (2018–2021) could thus yield results spanning six years of development (ages 6 to 12).

Although developmental psychologists have long discussed the cohort-sequential concept, its applications were limited mainly to identifying cohort and time-of-measurement effects on personality and cognitive tests (e.g., Nesselroade & Baltes, 1974).

The need for uniform assessment of multiple birth cohorts over several years might seem to make accelerated longitudinal studies of psychopathology prohibitive. On the other hand, accelerated longitudinal studies can be far more cost-effective than traditional longitudinal studies that require as many years to collect data as the developmental period spanned. But would the magic of predicting from early assessments of psychopathology in one cohort to later assessments in another cohort actually work? The answer depends on the specific assessment procedures, participant samples, and developmental periods spanned. The magic has, in fact, worked very well in accelerated longitudinal studies of psychopathology, as illustrated in the following section.

Accelerated Longitudinal Analyses of Aggressive Versus Delinquent Behavior

The nosological category of CD has typically included both aggressive and nonaggressive conduct problems. As an example, the *DSM-5* criteria for CD include 15 symptoms that are grouped under the headings "aggression to people and animals"; "destruction of property"; "deceitfulness or theft"; and "serious violations of rules" (American Psychiatric Association, 2013, pp. 221–222). Because only three symptoms are required for CD, some children may be diagnosed as having CD solely on the basis of three very aggressive behaviors, whereas others may be diagnosed as having CD solely on the basis of three nonaggressive behaviors.

In contrast to the single diagnostic category provided by *DSM* for different kinds of conduct problems, many factor analyses of diverse problem behaviors have shown that aggressive and nonaggressive conduct problems tend to load on different factors (e.g., Quay, 1993; Rescorla et al., 2012). Aggressive conduct problems may certainly be comorbid with nonaggressive conduct problems in many individuals, just as other kinds of problems may be comorbid. However, because the *DSM*'s nosological model places children in the single category of CD whether they manifest only aggressive conduct problems, only nonaggressive conduct problems, or both, the CD category obscures important differences between patterns of conduct problems.

One way to test for developmentally important differences between aggressive and nonaggressive conduct problems is to compare their developmental courses. Accelerated longitudinal analyses have been used to do this over ages 4 to 18 years in a population sample of Dutch children participating in the Zuid (South) Holland Longitudinal Study (Stanger, Achenbach, & Verhulst, 1997). The children were assessed with the Child Behavior Checklist (CBCL; Achenbach, 1991b) completed by their parents five times at two-year intervals. At each assessment, the parents rated their child on diverse behavioral and emotional problem items. Factor analyses of the problem items in other samples had previously produced eight syndromes that included a syndrome designated as *Aggressive Behavior* and one designated as *Delinquent Behavior* (subsequently designated as *Rule-Breaking Behavior*; Achenbach & Rescorla, 2001), which comprises nonaggressive conduct problems such as lying, stealing, running away from home, truancy, and hanging around others who get in trouble.

To compare the developmental courses of the Aggressive and Delinquent syndromes, Stanger et al. (1997) applied an accelerated longitudinal strategy as follows:

1. They used CBCL Aggressive and Delinquent syndrome scores for seven birth cohorts of children who were 4, 5, 6, 7, 8, 9, or 10 years old at Time 1. The children were 12, 13, 14, 15, 16, 17, or 18 years old, respectively, at Time 5, which was eight years later.
2. They matched each child in each of the youngest six birth cohorts to a child ("partner") in the cohort that was two years older. (Cohorts differing by two years of age were matched to take account of the two-year intervals between assessments.)

3. The matched partners were of the same gender and similar SES, and they obtained similar standard scores on the Aggressive and Delinquent syndromes at the two earliest ages at which both members of the pair had been assessed, i.e., at Time 1 and Time 2 for the older cohort of a pair and Time 2 and 3 for the younger cohort of a pair. In other words, Stanger et al. paired children who differed by two years of age, who were of the same gender, and who were similar with respect to SES, Aggressive syndrome scores, and Delinquent syndrome scores at two assessment points, two years apart.
4. Separately for the Aggressive and Delinquent syndromes, Stanger et al. then computed *r*s between children's scores from Time 1 to Time 2, Time 3, Time 4, and Time 5. These *r*s reflected the stability of the rank ordering of the children's own scores on a particular syndrome over periods of two, four, six, and eight years (i.e., the "within-cohort" stability of scores).
5. Stanger et al. also computed analogous *r*s between the syndrome scores obtained at Time 1 by children in the younger cohorts and the syndrome scores obtained by those children's matched partners in the two-year older cohorts at Times 2, 3, 4, and 5. These *r*s thus reflected the stability of the rank ordering of scores when *r*s were computed between one cohort of children at Time 1 and those children's matched partners in another cohort at Times 2, 3, 4, and 5 (i.e., the "between-cohort" stability of scores).

Separately for the Aggressive and Delinquent syndromes, Figure 2.4 displays the mean within-cohort and between-cohort *r*s obtained over intervals of two, four, and six years, plus the within-cohort *r*s over eight years for the seven cohorts of children. (The within-cohort *r*s extended two years beyond the ages that were included in the between-cohort *r*s.)

Figure 2.4 reveals two important findings:

1. Averaged across the seven birth cohorts, the mean between-cohort *r*s were as large as the within-cohort *r*s for both the Aggressive syndrome and the Delinquent syndrome over two-year and four-year periods and for the Aggressive syndrome over six-year periods. The sole exception was a mean between-cohort *r* that was only .01 lower than the mean within-cohort *r* over six years for the Delinquent syndrome, a trivial difference that was not statistically significant. Thus, later scores for the Aggressive and Delinquent syndromes were predicted as well from the early scores of children's matched partners from a different cohort as from the children's own early scores. This indicated that accelerated longitudinal analyses worked for the Aggressive and Delinquent syndromes.
2. At all data points for both the within-cohort and between-cohort *r*s, children's rank orders on the Aggressive syndrome were more stable than on the Delinquent syndrome. The greater stability of Aggressive than Delinquent syndrome scores was shown by mean within-cohort and mean between-cohort *r*s that were .21 higher ($p < .001$ computed by *z* transformation) for the Aggressive syndrome than for the Delinquent syndrome, averaged over all intervals.

To compare the developmental trajectories for the Aggressive and Delinquent syndromes, Stanger et al. averaged scores obtained by children in each cohort at each age for which the cohort was assessed. Separately for each gender, Figure 2.5 displays the mean Aggressive and Delinquent raw scores at ages 4 to 18 (because the seven cohorts were initially assessed at different ages, data were averaged for pairs of adjoining years, e.g., ages 4/5, up to age 18, when data were obtained for only one cohort). By looking at Figure 2.5, you can see that Aggressive syndrome scores declined steadily from ages 4/5 to 18. By contrast, Delinquent syndrome scores showed more complex relations to age, with increases at ages 12/13 for both genders. Girls' Delinquent syndrome scores then declined at age 18, although the data were based on only one cohort at that age.

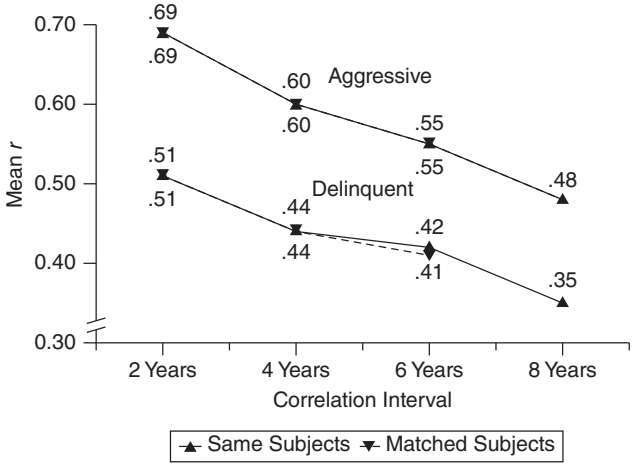


Figure 2.4 Within-cohort (same subjects) predictive correlations over intervals of two to eight years and between-cohort (matched subjects) predictive correlations over six years for Dutch children. Each predictive correlation for the Aggressive Behavior syndrome was significantly greater than for the corresponding Delinquent Behavior syndrome at $p < .001$. *Source:* Stanger, et al. (1997), p. 51. Reprinted with the permission of Cambridge University Press.

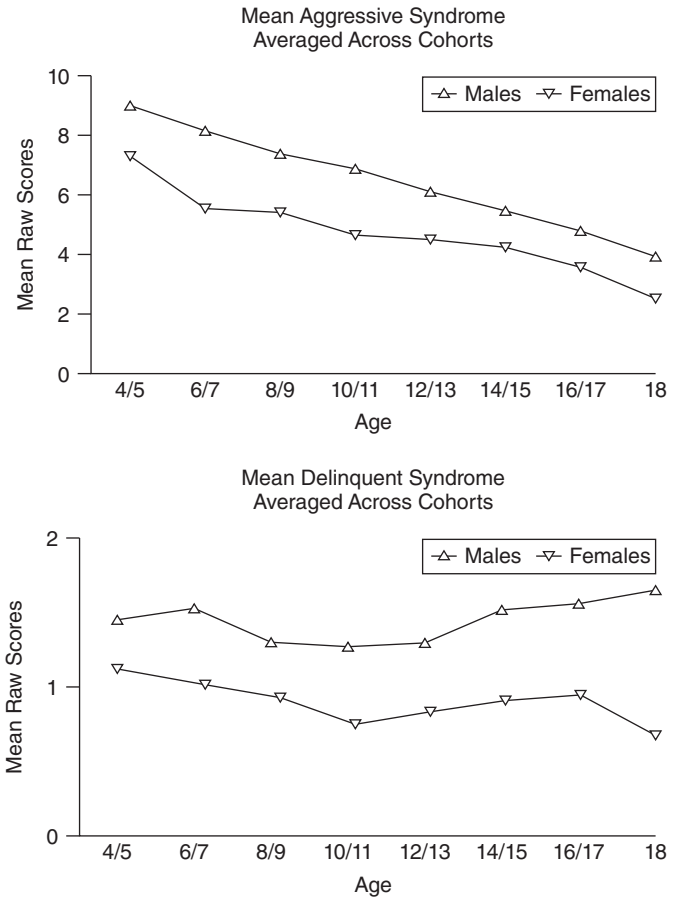


Figure 2.5 Mean Aggressive Behavior and Delinquent Behavior syndrome scores averaged across seven birth cohorts of Dutch children, separately for males and females. *Source:* Stanger et al. (1997), p. 54. Reprinted with the permission of Cambridge University Press.

The Stanger et al. data thus show more linear developmental trends, as well as greater developmental stability in individual differences, for Aggressive syndrome scores than Delinquent syndrome scores. Although Aggressive and Delinquent syndrome scores were significantly associated with each other (*r*s ranged from .53 at ages 4/5 to .63 at age 18), the findings revealed important differences in the developmental courses of aggressive versus nonaggressive conduct problems.

Based on the findings of strong between-cohort predictive relations, predictions could potentially be made from Time 1 scores obtained by one cohort to Time 4 scores obtained by its matched cohort when eight years older. For example, Time 1 scores obtained at age 4 by the Cohort 4 children could be used to predict Time 4 scores obtained at age 12 by their matched partners in Cohort 6. A study spanning six years of real time could thus be used to test predictive relations over eight years of development. Furthermore, tests of predictive relations can be extended to 12 years by matching individuals from successive cohorts to provide predictive correlations between age 4 scores obtained by Cohort 4 members at Time 1 with age 16 scores obtained by Cohort 10 members at Time 4. (To take account of developmental changes in mean scores, the scores need to be standardized within each cohort at each time of assessment.) The potential power of accelerated longitudinal designs greatly increases the value of short-term longitudinal studies, which are usually far more feasible than long-term studies.

Accelerated longitudinal designs are among the many potential methodological contributions of developmental approaches to psychopathology. They illustrate both the promise and the complexity of developmental perspectives for advancing the study of psychopathology. Cohort and time-of-measurement effects, as well as intraindividual developmental sequences, highlight the need for complex multivariate conceptualizations. The complexity should not deter efforts to cope with the challenges of interrelations between development and psychopathology, however,

because powerful research tools are available for coping with such challenges.

Path Designs

Path designs use regression statistics to test predictive pathways between variables assessed at developmentally early points and variables assessed for the same people at developmentally later points. Unlike accelerated longitudinal designs, path designs cannot test relations between variables over developmental periods that are longer than the real-time periods spanned by the assessment of the participants. However, path designs can test the degree to which multiple variables assessed at different early points in development individually and collectively contribute to the prediction of a particular outcome variable assessed at a later point in development. They can also test the indirect effects of developmentally early independent variables predicting subsequent variables that, in turn, predict the outcome variables of interest.

Various statistical techniques are available for conducting path analyses (Lleras, 2005). However, without going into the details of the various statistical techniques, we can illustrate some ways path analyses can contribute to developmental research on the assessment and taxonomy of psychopathology.

Six-Year Predictive Pathways for Aggressive Versus Delinquent Behavior

As reported in our illustration of accelerated longitudinal designs, factor analyses have yielded syndromes designated as Aggressive Behavior and Delinquent Behavior (also called Rule-Breaking Behavior). Both within-cohort and between-cohort correlations have shown significantly greater developmental stability for scores on the Aggressive syndrome than the Delinquent syndrome among Dutch children (Stanger et al., 1997). Another way to evaluate the developmental course of syndromes is via path analyses like those done in the U.S. National Longitudinal Study of a nationally representative sample of American children (Achenbach, Howell, McConaughy, & Stanger, 1995a, 1995b). Eight empirically based syndromes and numerous family variables were assessed at each of three time points, with three-year intervals between Times 1 and 2 and between Times 2 and 3, using methodology similar to that of the Dutch study on which the accelerated longitudinal analyses were based. In addition, at the second and third assessments, information was obtained about potentially important life events that had occurred since the preceding assessment, such as stressful experiences.

Figure 2.6 shows the pathways for Time 1 and Time 2 variables that significantly predicted Time 3 scores on the Aggressive and Delinquent syndromes. The Time 3 syndrome scores were computed by combining parents' ratings on the CBCL, teachers' ratings on the Teacher's Report Form (TRF; Achenbach, 1991b), and adolescents' self-ratings on the Youth Self-Report (YSR; Achenbach, 1991b). (The CBCL, TRF, and YSR are components of the Achenbach System of Empirically Based Assessment, abbreviated as ASEBA.) The Time 1 syndrome scores were computed from parents' ratings on the ACQ Behavior Checklist (Achenbach, Howell, Quay, & Conners, 1991), whereas the Time 2 syndrome scores were computed from parents' ratings on the CBCL.

The numerals on the predictive paths in Figure 2.6 indicate the effect sizes (ES) in terms of the percentage of variance in the Time 3 syndrome scores that was uniquely accounted for by each of the significant predictors, with the effects of the other predictors partialed out. Each ES for boys is printed above the solid and dotted lines, while each ES for girls is printed below the solid and dashed lines (solid lines = effects that were significant for both genders; dotted lines = effects that were significant only for boys; dashed lines = effects that were significant only for girls). For example, if you look under the Time 2 heading for the Delinquent syndrome in Figure 2.6, you can see that the topmost ES was 4% for the prediction of Time 3 Delinquent syndrome scores from Time 2 Delinquent syndrome scores for boys. Beneath the 4% ES for boys, you can see that the corresponding ES for girls was 8%. For both genders, there were also 1% ESs for prediction of Time 3 Delinquent syndrome scores from Time 2 Aggressive syndrome scores. For girls only, there were 1% ESs for prediction of Time 3 Delinquent scores from Time 2 Social Problems syndrome scores (low Time 2 Social Problems scores predicted high Time 3 Delinquent scores) and Attention Problems syndrome scores. There was an additional 1% ES for the number of unrelated adults living in the girls' homes between Time 2 and Time 3.

By looking under the Time 1 heading in Figure 2.6, you can see that, for boys, the following two predictors directly accounted for 1% of variance in Time 3 Delinquent scores: (1) The Time 1 marital status of the boys' parents (Time 3 Delinquent scores were higher if parents were single, separated, or divorced than if they were married or widowed); and (2) the Time 1 Delinquent scores. In summary, for both genders, more variance in Time 3 Delinquent scores was accounted for by earlier scores on the Delinquent syndrome than by any other variable. However, significant variance was also accounted for by other variables,

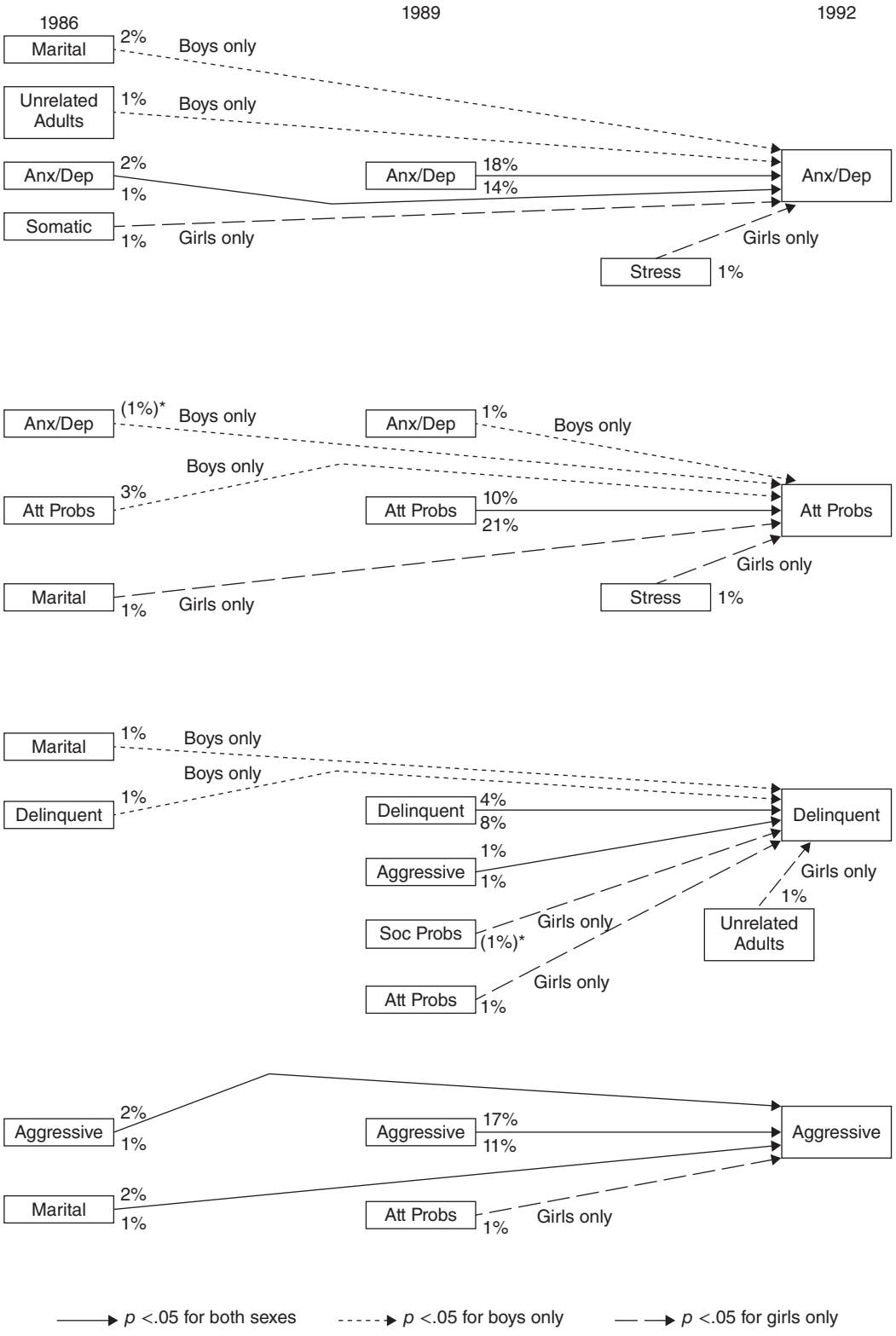


Figure 2.6 Predictive pathways to scores for Aggressive Behavior, Delinquent Behavior, Attention Problems and syndrome scores in an American national sample. *Source:* Achenbach et al. (1995a), p. 343. Adapted with permission of the *Journal of the American Academy of Child and Adolescent Psychiatry*.

including scores on the Aggressive, Social Problems, and Attention Problems syndromes, parents' marital status at Time 1, and the number of unrelated adults living in the home between Time 2 and Time 3.

If you look now at the lower portion of Figure 2.6, you can see that Time 2 Aggressive syndrome scores accounted for 17% of the variance in Time 3 Aggressive syndrome scores for boys and 11% for girls. Time 1 Aggressive syndrome scores accounted for an additional 2% of the variance in Time 3 Aggressive syndrome scores for boys and 1% for girls. The only other significant predictors were parents' Time 1 marital status (2% ES for boys; 1% ES for girls) and Time 2 Attention Problems syndrome scores (1% ES for girls only).

By comparing the predictive paths found for Time 3 Delinquent versus Aggressive syndrome scores, you can see that the ESs from earlier versions of the same syndrome were smaller for the Delinquent than the Aggressive syndrome: The ESs for the Time 1 and Time 2 Delinquent syndromes summed to 5% for boys and 8% for girls, whereas the ESs for the Time 1 and Time 2 Aggressive syndrome summed to 19% for boys and 12% for girls. In addition, more numerous other predictors were found for the Delinquent syndrome than the Aggressive syndrome. The other predictors included the Time 2 Aggressive syndrome scores for both genders, Time 2 Social Problems syndrome scores as negative predictors for girls, and the number of unrelated adults in the home between Times 2 and 3 as positive predictors for girls.

Although path designs differ markedly from accelerated longitudinal designs, findings from our examples of these different designs in the Netherlands and the United States suggest the following conclusions about the taxonomic properties of the Aggressive versus Delinquent syndromes:

1. Early scores on each syndrome are significant predictors of later scores on the same syndrome, indicating developmental stability in the degree to which individuals manifest each kind of problem.
2. The developmental stability of the Aggressive syndrome is greater than the developmental stability of the Delinquent syndrome; that is, individuals' rank orders remain more consistent for Aggressive than Delinquent scores over substantial periods of development.
3. The course of the Delinquent syndrome in relation to age, gender, and other kinds of problems is more variable than the course of the Aggressive syndrome.
4. Overall, the findings suggest that the Aggressive syndrome is more trait-like than the Delinquent syndrome in that individuals maintain more consistent rank orders in relation to peers of the same age and gender. Furthermore, later Aggressive syndrome scores are predicted more exclusively by scores on the same syndrome than is true for the Delinquent syndrome.

Path Analysis via Structural Equation Modeling

Path analysis can also make use of structural equation modeling (SEM) of hypothesized latent variables that are measured in terms of multiple manifest variables. The researcher specifies a model that includes relations between both the latent and manifest variables. Mediator variables are often specified as being affected by developmentally early variables but, in turn, as affecting developmentally later variables. As an example, path analyses were used to test predictors of the latent variable of adult aggression from a variety of measures obtained when the adults were children, 15 years earlier (Huesmann, Moise-Titus, Podolski, & Eron, 2003). The latent variable of adult aggression was derived by SEM from eleven manifest variables, including MMPI scores, plus self-ratings and informant ratings of various kinds of direct and indirect aggression. Childhood measures of peer-nominated aggression and violent television viewing were found to be significant direct predictors of adult aggression. In addition, an initial direct predictor of peer-nominated aggression and violent television viewing—poor school achievement—was found to be an indirect predictor of later adult aggression. Although the results indicated that viewing violent television can increase aggression over long periods, both the preference for viewing violent television and the continuity of aggression from childhood to adulthood may also reflect individual differences in achievement, plus the trait-like aspects of aggression indicated by the accelerated longitudinal and path analyses that we described previously.

Growth Curve Designs

The accelerated longitudinal and path designs illustrated in the preceding sections test changes over time via correlations computed for samples of individuals. In contrast, growth curve designs permit investigation of changes in individuals as well as changes in groups over time (Rogosa, Brandt, & Zimowski, 1982). Latent growth curve analysis uses longitudinal data for each individual to model a growth trajectory for that individual. In addition, an average growth trajectory for all individuals is obtained. Each individual growth curve and the group function are described by a set of parameters. For linear functions, these parameters are the intercept and the slope of the line. When growth is not monotonic, quadratic or cubic functions may

be fit to the data, resulting in parameters for these terms as well. The intercept of the function often represents the initial status of the individual on the dependent measure (i.e., at the first data collection point). The slope measures change in the dependent measure per unit of time over the time period from the first data collection point to the last. The variance estimates for intercept and slope indicate the degree to which individuals in the sample manifest differences in these parameters.

Researchers have developed many statistical procedures to analyze growth in individuals over time. Such procedures include *individual growth curve analysis* (Rogosa et al., 1982), *random effects modeling* (Laird & Ware, 1982), and *hierarchical linear modeling* (HLM; Raudenbush & Bryk, 2002). As HLM is one of the most widely used of these techniques, our summary will focus on this set of procedures.

Hierarchical Linear Modeling

HLM is called a hierarchical model because it describes the data in terms of at least two levels. For growth curve analysis, HLM uses Level 1 to specify a within-subject model for each individual in the sample, based on data collected at different points in time. For example, ordinary least squares (OLS) regression could generate a growth trajectory for each individual based on that individual's score on the CBCL Aggressive Behavior syndrome scale at four time points, such as at ages 9, 12, 15, and 18. If the best fit to the data points is a straight line, then an individual's growth over time will be described by his/her intercept (score at Time 1, or age 9) and his or her slope (rate of change in score per year starting at age 9 and ending at age 18). Because no fitted line perfectly captures all data points, each function also contains an error term. In its simplest form, each participant's growth over time is represented by Equation 2.1.

$$Y_t = a + bX + E \tag{2.1}$$

where Y_t is the dependent measure of the Aggressive Behavior syndrome score at time t, a is Participant A's score at age 9 (the initial data point), b represents the rate of change in scores over time, X represents the number of time units between the initial time point and time t (e.g., age 12 = +3), and E represents random error or residual deviation from the predicted score.

In HLM, the Level 2 or between-subjects model predicts the values of each individual's parameters. Thus, the parameters of the Level 1 model are the outcome measures of the Level 2 model. The first step in this analysis is to determine the total amount of variation in the intercept and slope parameters. When individuals have very different initial scores on the dependent measure and they show very different rates of change in the dependent measure over time, then variance in both the intercept and slope will be high. Variance in the intercept will be minimal when all individuals start at the same point, whereas variance in the slope will be minimal when all individuals decrease (or increase) in the dependent measure at the same rate over time. HLM uses chi-square to test whether the variances in the intercept and slope are significantly different from zero. If one of these parameters is not significantly different from zero, then the model is constrained or fixed so that individuals are not allowed to have their own values on this parameter in subsequent analyses.

In typical HLM analyses, the first Level 2 model has no predictors. This is called an *unconditional* model, because no additional factors, such as gender, SES, diagnosis, referral status, or life experiences, are used to predict individual growth. In this basic unconditional Level 2 model, the parameters from Level 1 (a and b) are predicted only by the population mean (derived from the data) and random deviation. The basic unconditional model at Level 2 appears in Equations 2.2 and 2.3:

$$a = \beta_{00} + \text{ random deviation} \tag{2.2}$$

$$b = \beta_{10} + \text{ random deviation} \tag{2.3}$$

Thus, the unconditional model states that the best estimate of an individual's slope or intercept is the population parameter plus some random deviation or error.

At the next stage in the analysis, HLM contrasts this unconditional model with a conditional model in which some additional factor is entered as a predictor to explain individual differences in either intercept or slope. For example, one might predict that clinically referred children would have higher Aggressive Behavior syndrome scores than nonreferred children (reflected in a higher intercept value). One might also predict that children being treated for aggressive behavior problems might show more improvement over time than children receiving no treatment (steeper negative slope). To test these predictions, the predictor of referral status or group is added to the Level 2 model. Because group in this example is a dichotomous variable, it would be coded using a dummy variable such as 0 = nonreferred and 1 = referred. Equations 2.4 and 2.5 depict the conditional model:

$$a = \beta_{00} + \beta_{01}(\text{group}) + \text{ random deviation} \tag{2.4}$$

$$b = \beta_{10} + \beta_{11}(\text{group}) + \text{ random deviation} \tag{2.5}$$

According to these equations, the individual differences in intercept and slope parameters at Level 1 are determined both by the population values for these parameters and by the child's status as nonreferred or referred (plus random deviation).

HLM next determines whether the conditional model with predictors is a significant improvement over the unconditional model with no predictors. Essentially, HLM determines the percent of total variance explained when predictors are added to the model relative to the total variance in the parameters with no predictors. It also tests the significance of each predictor using *t* tests.

Application of HLM to Developmental Psychopathology

For purposes of explication, we have presented HLM in its simplest form. However, HLM enables analyses that are much more complex. Multiple predictors can be entered at Level 2 and their main effects and interactions can be tested. For example, not only referral status but also gender and SES might be associated with both initial status and change over time in Aggressive Behavior syndrome scores. Some studies might necessitate a three-level model. For example, a study might compare Aggressive Behavior syndrome scores over time in individuals (Level 1) as a function of gender, SES, and referral status (Level 2) in three different countries, such as the United States, the Netherlands, and Korea (Level 3).

Growth curve analysis has become very widely used in developmental research. Because it preserves information about individual trajectories over time and permits examination of multiple predictors of those trajectories, it is very useful for answering major questions in developmental psychopathology. Furthermore, HLM can handle missing data in ways that traditional analysis of variance (ANOVA) and analysis of covariance (ANCOVA) procedures cannot. In a repeated-measures ANOVA, individuals who have missing data at one data point may be eliminated from the analysis or may have missing scores imputed to them. In HLM, however, individuals can remain in the analysis even if they have some missing data. That is, intercepts and slopes can be calculated for all individuals as long as they have scores for most of the data points (e.g., three out of four).

HLM Analyses Testing Moderation Effects

Laurent et al. (2013) used HLM to test whether child hypothalamic pituitary adrenal (HPA) activity moderated the effects of parental depressive symptoms on child CBCL/1½-5 scores over time in 210 adopted children. Mothers and fathers each completed the Beck Depression Inventory (BDI; Beck & Steer, 1993) and the CBCL when the children were 18, 27, and 54 months of age. To avoid possible effects of parental depression on CBCL ratings, each parent's BDI scores were tested as predictors of the child's CBCL Internalizing and Externalizing scores based on the spouse's ratings.

Laurent et al. (2013) measured HPA activity by obtaining morning and evening salivary cortisol over three consecutive days when the children were 54 months old. Because some cortisol data were missing, the authors used HLM to estimate each participant's typical cortisol levels for morning and evening and each participant's day-to-day variability in morning and evening cortisol levels at 54 months. These fitted values of morning and evening cortisol level and cortisol variability were used as markers of stress sensitivity in the child. Interestingly, the correlations between morning and evening cortisol levels (.09) and between morning and evening cortisol variability (.09) were low and not significant. However, cortisol level and variability were correlated modestly with each other in the morning (.31) and substantially in the evening (.81). These findings suggest that the various cortisol measures were not interchangeable or redundant but in fact tapped different aspects of HPA functioning.

In this study, HLM was used to see if variation in cortisol level or variability had a moderating effect on the impact of parental depression on child adjustment. Specifically, HLM modeled CBCL Internalizing and Externalizing scores at Level 1 with an intercept (b_0) and linear slope over time (b_1). Cortisol activity was used as a Level 2 predictor. The authors tested two basic kinds of models, which they called *within-family risk dynamics* and *between-family risk synergy*. The statistical difference between these two models depended on whether parental depression was treated as a Level 1 or Level 2 predictor, as explained below.

To examine within-family risk dynamics, parental BDI scores were added as a time varying covariate at Level 1 (b_2). This within-family risk model tested the effects of variation in parental depression on child adjustment over time as moderated by child stress reactivity measured by cortisol activity at Level 2. The baseline models of child Internalizing and Externalizing problems, which used age 54 months scores as the intercept, indicated that CBCL scores tended to increase from 18 to 54 months. Additionally, the baseline model indicated that there was sufficient within-child variability to add explanatory predictors at Level 1 and sufficient between-child variability in both intercept (54 months score) and slope (change in score from 18 to 54 months) to add explanatory predictors at Level 2.

Results of these analyses indicated that increases in maternal depression predicted increases in child's Internalizing and Externalizing scores over time, with no moderation by child's cortisol activity. In a contrasting pattern, increases in paternal depression showed no main effect on child CBCL Internalizing or Externalizing scores, but child's evening cortisol level and variability had a significant moderating effect. That is, only children who had higher evening cortisol levels and greater evening cortisol variability showed significant increases in Internalizing scores associated with increases in paternal depressive symptoms. In other words, greater child stress reactivity measured by evening cortisol activity increased the within-family effect of paternal depression on child Internalizing scores.

In the between-family risk synergy models, the mean of parent depressive symptoms averaged over the three data points was entered as a Level 2 predictor (rather than entering depressive symptoms as a time-varying covariate at Level 1). As in the within-family models, cortisol variables were entered as Level 2 predictors. The between-family risk synergy models tested whether children's CBCL Internalizing or Externalizing trajectories (intercepts and slopes) were predicted by the synergistic (interaction) effect of parents' average level of depression and the child's stress reactivity. For example, the authors predicted that the combination of high-risk parents and greater child stress reactivity would lead to higher levels of CBCL problems. It was found that Level 2 maternal depression predicted child Internalizing intercepts but the interaction (moderation) by cortisol was not significant. This means that children whose mothers had a high level of depressive symptoms in the period from 18 to 54 months had higher Internalizing scores at 54 months, regardless of their stress reactivity. On the other hand, the interaction of maternal depression and cortisol activity predicted child Internalizing slope, even though neither predictor was significantly associated with Internalizing slope on its own. Specifically, children with low levels of morning cortisol who had mothers with higher levels of depression tended to increase in Internalizing score from 18 to 54 months. For Externalizing, intercepts (i.e., 54 month score) and slopes (change from 18 to 54 months) were both predicted by the combination of high maternal depression and high evening cortisol levels.

Between-family risk synergy models for fathers showed quite similar patterns. Specifically, fathers with higher mean BDI scores had children with higher Internalizing scores at 54 months. The main effects of father's mean BDI scores on Externalizing intercepts and slopes were moderated by evening cortisol level and variability. That is, the combination of high paternal mean depression and high evening cortisol predicted higher child Externalizing scores at 54 months and greater increases in scores from 18 to 54 months.

This study illustrates several benefits of HLM for testing models of developmental psychopathology. Specifically, both linear growth (slope) and final level (intercept) of children's CBCL scores could be modeled in the same analysis. Second, HLM allowed parental BDI scores at 18, 27, and 54 months to be used either as a time-varying coordinate at Level 1 in the within-family risk model or as a mean value over time at Level 2 in the between-family risk synergy model. Additionally, HLM allowed simultaneous tests of within-child effects (what factors accounted for change over time in individual children's CBCL scores) and between-child differences (what factors accounted for individual differences between children in CBCL scores over time and in scores at 54 months). Finally, HLM allowed tests of stress reactivity's moderation of parental depression's impact on both within-child growth and between-child differences in Internalizing and Externalizing problems.

Growth Mixture Models

As described by Jung and Wickrama (2008), growth mixture modeling (GMM) is a person-centered statistical technique designed to capture individual differences in growth trajectories by identifying trajectory subgroups within a population. Like cluster analysis and latent class analysis, growth mixture models classify individuals into classes, categories, or subgroups such that within-class individual differences in scores are smaller than between-class differences in scores. Whereas HLM assumes that all individuals in a given population have the same growth parameters, GMM assumes that subgroups differ in their growth parameters.

A type of GMM described by Nagin (1999) has been called semiparametric mixture model fitting. Nagin points out that semiparametric mixture model fitting is a group-based strategy that can test whether developmental trajectories predicted by theories actually exist in a population. Nagin (1999) noted that unlike HLM and other forms of latent curve modeling that assume a continuous distribution of trajectories in a population, semiparametric mixture modeling aims to identify different groups of individuals within a population, with each group having its own distinct trajectory.

Like HLM, semiparametric mixture modeling uses a polynomial equation to represent the link between age and

the target behavior. For example, the model might assume a quadratic trajectory, which would be defined by an intercept, a linear slope term, and a quadratic term. However, unlike in HLM, the parameters in the equation (model) are left free to differ across groups. In other words, the various trajectory groups are free to differ from each other with respect to intercept, slope, or both, with the possibility that some groups have no growth (i.e., no change over time), others have only linear growth, and others have quadratic or cubic patterns.

The Bayesian Information Criterion (BIC) is typically used to select the optimal number of trajectory groups (i.e., the model with the highest BIC has the optimal number of groups) (Nagin, 1999). Comparison of BIC values for different models can reveal how much better the optimal model fits the data than various alternative models (Raftery, 1995). In addition, for each model solution (e.g., 2, 3, 4, and 5 group solutions), the output includes the probability that this solution is the "true model."

Also included in the output of semiparametric mixture model fitting are estimates of the probability that each individual belongs to each of the groups. This allows each individual to be assigned to the group he or she fits best. Once the groups are thus constructed, they can then be compared with respect to other variables of interest, such as demographic factors, IQ/educational status, biomarkers, or response to treatment.

Developmental Trajectories of Externalizing Problems

Bongers, Koot, van der Ende, and Verhulst (2004) used semiparametric mixture model fitting in a study involving 2,076 children in the Zuid-Holland longitudinal study who were first assessed in 1983. Data used for analyzing developmental trajectories were obtained from ASEBA forms administered at five points over 14 years. Four clusters of Externalizing problems distinguished by Frick et al. (1993) were studied, namely, *Aggression, Opposition, Property Violations,* and *Status Violations*. These constructs were operationalized by Bongers et al. (2004) using items from the CBCL that seemed to best tap the four clusters distinguished by Frick. CFAs were used to test the fit of the selected items to the Frick model at each CBCL time point. The data fit the model well for both males and females, and all item loadings on their respective cluster were significant and large (range from .50 to .91). Bongers et al. (2004) then used multilevel growth curve analysis to determine separately by gender the average trajectory of each of the four externalizing clusters over time as well as the variation around each average trajectory. Results revealed decreasing average trajectories for all clusters except Status Violations, which increased with age.

Following these steps, Bongers et al. (2004) applied semiparametric mixture model fitting. Based on comparison of the BICs, the authors concluded that the best fitting trajectory models were three classes for the Aggression cluster (all with linear slopes), six classes for the Opposition cluster (some with zero slope and some with quadratic slope), four classes for the Property Violations cluster (mix of linear and zero slopes), and four classes for the Status Violation cluster (all linear slopes). Males generally had higher scores than females but the shapes of the trajectories were similar. Trajectory classes were given descriptive labels. For example, within the Aggression cluster, the three classes were designated as "near zero" (71% of the sample), "low decreasers" (21%), and "high decreasers" (8%). The Aggression cluster high and low decreaser groups differed in their starting level of aggression as well as in their aggression scores in adolescence (moderately high vs. near zero). Only the Status Violation cluster had several "increasing" trajectories: 28% ("adolescence increasers") had no status violations before age 10 and then started increasing slightly as they moved into adolescence, 25% ("medium increasers") showed an increasing number of status violations throughout the whole age period, and 1% ("high increasers") showed an increasing number of status violations throughout the whole age period rising to very high levels in adolescence. The Opposition cluster also had one group that increased in adolescence (6%). Males were overrepresented in the more problematic trajectories in each cluster except for Opposition. Interestingly, 64% of individuals in the sample were deviant in only one cluster, leaving 36% deviant in two or more clusters.

The use of semiparametric mixture model fitting allowed the authors to empirically determine trajectory classes in each cluster, with the optimal number of classes (as determined by the BIC) varying across clusters. By using multilevel growth modeling, the authors could determine the average trajectory for each externalizing cluster (separately for males and females). Then, by applying semiparametric mixture model fitting, they could identify trajectory classes that differed in their pathways over time from the prototypic pattern. Some of these classes followed the average trajectory in shape (e.g., generally decreasing over time, but from different starting points, as in the Aggression cluster). However, other classes had a quite different shape (e.g., the average Opposition trajectory decreased over time, but 6% of the sample increased in adolescence).

As noted by Nagin (1999), semiparametric mixture model fitting can be used to test important developmental

hypotheses, such as the distinction between childhood onset and adolescence limited antisocial behavior (Moffitt, 1993). Bongers et al. stated that their model did not identify an adolescence-limited class such as Moffitt described. Although for Opposition and Status Violations there were classes whose scores increased during adolescence, the CBCL scores for these classes were not as high as the scores for the classes who had high scores throughout the 14-year period, which is inconsistent with Moffitt's typology.

LIFE SPAN APPLICATIONS

To apply developmental approaches to psychopathology across the life span, we should consider lessons learned from research on children. Changes in the prevalence, patterning, and possibilities for assessing problems do not end at maturity. Although the changes may be slower and less conspicuously linked with biological development than during childhood, adult psychopathology may be as closely linked to developmental changes as child psychopathology is. However, to test the developmental course of psychopathology, its correlates, and its outcomes, we need assessment procedures and taxonomic constructs that can take account of both the changes and the continuities in psychopathology from childhood to adulthood and old age. Furthermore, it would be desirable to quantify adult assessment and taxonomy in ways that lend themselves to powerful developmental research strategies, such as accelerated longitudinal, path, and growth curve designs.

The value of measures that can track changes and continuities over significant periods of adulthood has been demonstrated in studies of cognitive functioning. As an example, latent growth models were applied to 11 cognitive measures for 550 Swedish adults who were initially tested at ages 44 to 88 years and were then retested at intervals of three and six years thereafter (Finkel, Reynolds, McArdle, Gatz, & Pedersen, 2003). The results revealed different trajectories for different kinds of abilities, including stability into old age for crystallized ability, linear declines in other abilities, and acceleration of linear declines after age 65 for measures that depended on speed. Significant individual variations in growth curves were found for some of the measures.

Similar growth curve analyses can be applied to measures of psychopathology in adults, such as those derived from the ASEBA forms for adults: the Adult Self-Report (ASR) and Adult Behavior Checklist (ABCL) for Ages 18–59, and the Older Adult Self-Report (OASR) and Older Adult Behavior Checklist (OABCL) for Ages 60–90+ (Achenbach, Newhouse, & Rescorla, 2004; Achenbach & Rescorla, 2003). Several problem scales are common to the instruments for both adult age groups. Examples include the empirically based Anxious/Depressed, Somatic Complaints, and Thought Problems syndrome scales and the *DSM*-oriented Depressive Problems, Anxiety Problems, Somatic Problems, and Antisocial Personality Problems scales. One syndrome scale, named Intrusive, is specific to ages 18 to 59. Other scales are specific to ages 60 to 90+, such as the Irritable/Disinhibited, Memory/Cognition Problems, Worries, and Functional Impairment syndrome scales. Many scales for ages 18 to 59 have counterpart scales scored from the CBCL, TRF, and YSR for ages 6 to 18.

Longitudinal research has shown that high problem scores on the adult Intrusive syndrome, as well as on the adult Aggressive Behavior syndrome, are strongly predicted by high scores on the child/adolescent Aggressive Behavior syndrome (Achenbach et al., 1995c). These findings indicate that some aggressive children and adolescents are socially intrusive as adults whereas others remain more overtly aggressive. The same research shows that high child and adolescent scores on the Attention Problems syndrome predict high scores on the adult version of this syndrome. However, the adult version is characterized less by overactivity and more by irresponsibility than the child/adolescent version.

Because analogous assessment instruments are available for ages $1\frac{1}{2}$ to 90+ years, developmental analyses of psychopathology can be extended over lengthy periods of development. In addition, because many of the empirically based and nosologically based scales for adults and older adults are similar to those for children and adolescents, ASEBA instruments facilitate multigeneration family studies. Multigeneration family studies are particularly useful in behavior genetics research designed to identify genetic effects, environmental effects, and their interactions in parents and their children.

STANDARDIZED ASSESSMENT AND TAXONOMIC MODELS IN MULTIPLE LONGITUDINAL STUDIES

Longitudinal studies are essential for identifying and testing developmental courses of psychopathology. However, the payoffs yielded by the large investments, long-term commitments required of investigators and participants, and years needed to obtain results may be limited by a lack of clear generalizability from findings for longitudinal samples to larger populations. One kind of limitation on generalizability stems from unrepresentative samples of

participants who are chosen for their convenient availability and willingness to undergo repeated assessments over long periods. A second kind of limitation stems from assessment procedures that are designed for a particular study or are chosen because they are au courant when the study begins. Results obtained with assessment procedures that are customized for a particular study are of unknown generalizability to larger populations if the procedures are not applied to and normed for those populations. Furthermore, if assessment procedures used in a study go out of fashion by the time they yield results, the results may not be applicable to then-current assessment procedures.

As an example, diagnostic interviews and other assessment instruments based on the 1980 *DSM-III* became obsolete in 1987 with the publication of *DSM-III-R*, which then became obsolete in 1994 with the publication of the *DSM-IV*. The interval between the 2000 *DSM-IV* Text Revision (*DSM-IV-TR*) and the 2013 *DSM-5* has been longer. However, the years needed to develop assessment instruments for each edition of the *DSM* and then to test the *DSM* diagnostic criteria, as well as the lack of standardized algorithms for translating data based on early editions into the diagnostic criteria of later editions of the *DSM*, limit the value of findings based on obsolete diagnostic criteria.

Earlier in this chapter, we illustrated path analyses for testing predictive pathways from variables assessed at developmentally early points to developmentally later scores on Aggressive Behavior and Delinquent Behavior syndromes in the U.S. National Longitudinal Study. The syndromes were scored from age-appropriate versions of ASEBA instruments completed by multiple informants. The sample was representative of the U.S. population, attrition was low in the initial and subsequent assessments, age-appropriate versions of the same items and constructs were assessed at each age with instruments that continue to be widely used, and the scales have age, gender, and informant-specific U.S. national norms. Consequently, the results can be applied to the U.S. population with much greater confidence than if the sample was unrepresentative, or if the assessment instruments were customized for the study, became obsolete, or lacked norms.

Despite the high cost and great value of a longitudinal study on a nationally representative sample, no single study is likely to yield definitive answers by itself. Instead, multiple studies employing standardized assessment procedures and taxonomic models are needed to obtain widely generalizable and useful findings. The following sections provide an overview of four longitudinal studies in the Netherlands using Dutch translations of the assessment instruments that were used to assess the empirically based syndromes in the U.S. National Longitudinal Study.

Zuid Holland Longitudinal Study

The Zuid Holland Longitudinal Study began with assessment of a representative sample of 2,076 4–16-year-olds living in the Province of Zuid Holland. Parents completed CBCLs and reported on child and family variables like those assessed in the U.S. National Longitudinal Study. Teachers completed TRFs and 11–16-year-olds completed YSRs. Age-appropriate ASEBA instruments were subsequently completed by the participants and by various informants at four two-year intervals, followed by six-year and 10-year intervals, spanning a total of 24 years, until the oldest participants had reached the age of 40. Numerous other candidate predictor and outcome variables were also assessed, including *DSM* diagnoses made from SDIs.

Over the age ranges that were similarly assessed in the Zuid Holland and U.S. studies, similar predictive pathways and associations were found from developmentally early to developmentally later scores on the syndromes. Furthermore, despite more universal health insurance coverage for Dutch than U.S. children, as well as other differences between the societies and their mental health services, the developmental course of clinically elevated scores on the syndromes was found to be similar for Dutch and U.S. children (Van Oort, van der Ende, Wadsworth, Verhulst, & Achenbach, 2011). Tendencies for problem scores to be higher for both Dutch and U.S. children from lower than upper SES families were consistent with those found in a review of findings from 15 societies (Achenbach & Rescorla, 2007b).

Over the much longer span of the Zuid Holland than the U.S. study (24 years vs. 9 years), it was found that all the syndromes initially scored from parents' CBCL ratings significantly predicted syndromes scored from the participants' self-ratings on the ASR (Achenbach & Rescorla, 2003) 24 years later (Reef, Diamantopoulou, van Meurs, Verhulst, & van der Ende, 2009). The significant predictive relations revealed homotypic cross-informant (parent to self-ratings) continuity over 24 years for Anxious/Depressed, Withdrawn, Somatic Complaints, Rule-Breaking Behavior, and Aggressive Behavior syndromes. Heterotypic continuity was found for the Thought Problems and Attention Problems syndromes. The parents' initial ratings also significantly predicted *DSM* diagnoses made from SDIs administered to the participants 24 years later (Reef et al., 2010). Especially surprising were findings that parents' initial CBCL ratings also significantly

predicted the participants' CBCL ratings of their own children 24 years later (van Meurs, Reef, Verhulst, & van der Ende, 2009).

The TRacking Adolescents' Individual Lives Survey (TRAILS) Study

The TRAILS study started with a population sample (N = 2,230) and a clinical sample (N = 543) of youths residing in the northern Netherlands. Participants have been assessed at ages 10–12, 12–15, 15–17, and 18–20 and are being assessed again in their 20s (Ormel et al., 2012). Many measures have been used, including the CBCL and YSR, followed by the ASR after the participants' eighteenth birthday. Use of ASEBA instruments to operationalize phenotypes of psychopathology facilitates coordination of findings with other studies that use these instruments. Thus, as done in the Zuid Holland and U.S. longitudinal studies, correlations have been computed between ASEBA ratings of TRAILS participants at successive ages and have been found to be similar in all three studies.

As an example of the many TRAILS findings, parental internalizing disorders significantly predicted internalizing problems among TRAILS participants, while parental externalizing disorders predicted externalizing problems. As another example, maternal postpartum depression predicted internalizing problems, while pregnancy and delivery difficulties predicted externalizing problems. Stressful life events in adolescence were found to predict both internalizing and externalizing problems (Ormel et al., 2012).

Ormel et al. (2012) reported trajectories of scores averaged from CBCL and YSR ratings at ages 10–17 on the Aggressive Behavior, Rule-Breaking Behavior, Anxious/Depressed, and Withdrawn/Depressed syndromes. Significant decreases with age were found for Aggressive Behavior and Anxious/Depressed scores, whereas significant increases with age were found for Rule-Breaking Behavior scores. The decreases in Aggressive Behavior and increases in Rule-Breaking Behavior scores from age 10 to age 17 were consistent with the developmental trajectories shown in Figure 2.5 for CBCL scores averaged across seven birth cohorts. Ormel et al. found that mean Withdrawn/Depressed scores were stable from age 10 to 14, but increased somewhat from age 15 to 17. Gender differences were greatest for Rule-Breaking Behavior, with boys having substantially higher scores than girls at each age from 10 through 17, and for Anxious/Depressed, with girls having higher scores than boys at each age, plus a much smaller decline than boys from age 10 to 17. The authors concluded that "the study of developmental pathways needs to take into account the individual's gender, type of psychopathology, and age" (p. 1032).

Generation R Longitudinal Study ("R" = Rotterdam)

ASEBA instruments are being used to assess psychopathology in another Dutch longitudinal study that began with 8,880 pregnant women living in Rotterdam (Tiemeier et al., 2012). Among many other analyses of relations between developmentally early variables and subsequent psychopathology, Tiemeier et al. tested relations between mothers' self-reported anxiety during pregnancy and again after their child's birth, children's genotypes for the 5-HTTLPR polymorphism in the promoter region of the serotonin transporter gene (5-HTT, SLC6A4), and age 5 Internalizing scores averaged from mothers' and fathers' CBCL ratings. This test was especially important because of previous inconsistencies in findings of whether stressors raised risks for internalizing problems among carriers of the short allele of the 5-HTTLPR polymorphism. In a widely publicized article from a Dunedin, New Zealand, longitudinal study, Caspi et al. (2003) had reported that participants who carried the short allele were found to be at increased risk for developing depression after childhood maltreatment and other stressful experiences. This apparent gene x environment (GxE) interaction was of great interest because it appeared to support GxE mechanisms hypothesized to account for much psychopathology. Subsequent studies (e.g., Risch et al., 2009) failed to support the GxE interaction reported by Caspi et al. Moreover, Kraemer (2012) questioned the methodological basis for drawing either negative or positive conclusions about the GxE interaction from the existing data.

No single study can definitively resolve the very complex issues raised by possible GxE effects on psychopathology. However, the large representative Generation R sample assessed with many measures from the prenatal period onward, including assessment of psychopathology with the same kinds of standardized instruments at successive ages, provides exceptional opportunities for testing GxE effects. The inclusion of prenatal variables in testing the GxE interaction is of particular value, because serotonin appears about 5 weeks after conception. Consequently, individual differences in serotonin transporter availability related to the 5-HTTLPR polymorphism could affect vulnerability to prenatal stressors such as maternal anxiety.

Tiemeier et al. indeed found that children who carried the short allele of the 5-HTTLPR polymorphism and whose mothers reported high prenatal anxiety *or*

high postnatal anxiety (with prenatal anxiety controlled) obtained significantly elevated Internalizing scores at age 5. Furthermore, children who carried two short alleles obtained higher Internalizing scores than children who carried only one allele. However, these effects, as well as many other effects found to date in the highly sophisticated, cutting-edge Generation R study, were very small. The large number of very small effects led the authors to describe their research as representing "bit-by-bit efforts of integrative pluralism with many different perspectives" (p. 1124).

Netherlands Twin Registry (NTR)

The longitudinal studies summarized so far have repeatedly assessed particular birth cohorts of individuals as they developed. The NTR, by contrast, is a unique resource that enrolls large proportions of the twins born in the Netherlands each year. Since 1987, parents of newborn twins have been invited to participate in the NTR, with 40–50% of the parents accepting the invitation. Numerous standardized assessments of the twins and their families have made it possible to do hundreds of cross-sectional studies. Moreover, repeated assessments at successive ages with developmentally appropriate versions of ASEBA instruments have also made it possible to do hundreds of studies of genetic and environmental effects on the development of psychopathology operationalized via the instruments used in the United States, Zuid Holland, TRAILS, Generation R, and other studies.

As an example, Boomsma, van Beijsterveldt, and Hudziak (2005) analyzed Anxious/Depressed syndrome scores computed from mothers' and fathers' ratings of thousands of NTR twins when the twins were 3, 5, 7, 10, and 12 years old. For combined maternal and paternal ratings, heritability estimates for Anxious/Depressed syndrome scores were 76% at age 3, 60% at age 5, 67% at age 7, 53% at age 10, and 48% at age 12. Shared family environment effects were not significant at ages 3 and 7, 16% at age 5, 20% at age 10, and 18% at age 12. Discrepancies between maternal and paternal ratings reflected differences between each parent's information about their children's functioning, which, in turn, reflected significant genetic and shared environmental effects. In other words, rather than reflecting rater error, differences between mothers' and fathers' ratings validly captured different genetically and environmentally influenced variance in their children's behavior, as well as variance common to ratings by both parents.

Longitudinal analyses of NTR data have produced somewhat different findings for genetic and environmental effects on the CBCL Withdrawn, Attention Problems, and Aggressive Behavior syndromes (Hoekstra, Bartels, Hudziak, van Beijsterveldt, & Boomsma, 2008; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2004; van Beisjsterveldt, Hudziak, & Boomsma, 2003). Longitudinal analyses of mothers' and fathers' CBCL ratings of NTR twins have also been used to model distinctions between measurement error versus different genetically and environmentally influenced aspects of children's functioning captured by mother versus father ratings (Bartels, Boomsma, Hudziak, van Beijsterveldt, & van den Oord, 2007).

MULTICULTURAL FINDINGS AND APPLICATIONS

To avoid wrongly assuming that findings in a single society reflect universals, it is important to apply the same standardized assessment procedures for psychopathology to people from a variety of societies. If the assessment procedures are found to function similarly in different societies, they can be used to compare the course and correlates of psychopathology across those societies. Findings from multiple societies warrant more confidence than findings from a single society. An additional advantage of assessment procedures that function similarly in different societies is that they foster multicultural collaborations, which can greatly extend the reach of developmental research on psychopathology.

We use the term *society* in reference to populations living in a particular political jurisdiction in which a particular language is dominant. Many of the societies for which we report findings are countries, but some are regions within countries, such as Flanders (the Flemish-speaking region of Belgium), or are commonwealths or territories that are distinct from the country to which they belong, such as Hong Kong and Puerto Rico. We use the term "multicultural" in reference to findings and applications based on data spanning the cultural variations occurring across multiple societies.

Multicultural Tests of Syndromes

The previously described ASEBA syndromes were derived mainly from ratings of U.S. samples. To test the degree to which the ASEBA syndromes would be supported in other societies, indigenous researchers obtained ratings of samples of people residing in over 50 societies. The ratings obtained in each society were then subjected to confirmatory factor analyses (CFAs). The societies were located in

Africa, Asia, Australasia, the Caribbean, Europe, the Middle East, Central America, and South America. The ratings were obtained with the CBCL/1½-5, Caregiver-Teacher Report Form for Ages 1½-5 (C-TRF), CBCL/6-18, TRF, YSR, ASR, and ABCL, translated into local languages when necessary. CFAs supported the ASEBA syndrome structure in nearly every sample (Ivanova et al., 2007a, 2007b, 2007c, 2010, 2011, 2015a, 2015b; Rescorla et al., 2012b). The findings indicated that the syndrome models are sufficiently applicable in many societies to warrant scoring ratings of ASEBA problem items in terms of these models. Although additional problems might be important in some contexts and societies, the CFA support for the syndrome models in ratings by different kinds of informants in so many societies means that they can provide an evidence-based multicultural taxonomic framework for a broad spectrum of behavioral, emotional, and social problems assessable via self-ratings and collateral ratings.

Multicultural Comparisons of Scale Scores

The CFA support for the ASEBA syndrome models in so many societies justifies multicultural statistical comparisons of the syndrome scale scores. This has been done in age x gender x society ANOVAs of the syndrome scales scored from ratings on the ASEBA forms for ages 1½–5, 6–18, and 18–59 years. In addition to comparisons of syndrome scores, the comparisons have included scores for *DSM*-oriented scales and broad-band Internalizing, Externalizing, and Total Problems scales (Rescorla et al., 2011, 2012a, 2012b, 2015a, 2015b). Age and gender differences in scale scores have been very consistent across very diverse societies. Effect sizes for societal differences in problem scale scores have ranged from small to medium.

As another way of comparing problem ratings across societies, correlations have been computed between the mean ratings for all the problem items in each society and the mean ratings for all the problem items in each other society on the same instrument completed by the same type of informant. For example, the CBCL/6-18 was completed by parents of 69,866 children residing in 42 societies. Using the data from Society 1, Rescorla et al. (2012b) computed the mean of the 0-1-2 Society 1 ratings for each of the 112 problem items that were analyzed for the multicultural comparisons. The same was done for Society 2, Society 3, and so on through Society 42. The formula for Pearson *r* was then used to compute the correlation between the 112 mean item ratings for Society 1 versus Society 2, Society 1 versus Society 3, etc. for all pairs of societies. (In effect, such correlations are *Q* correlations, because they measure the similarity between the mean ratings on the 112 problem items for each pair of societies.)

Rescorla et al. averaged all the correlation coefficients obtained between all pairs of societies to obtain the overall mean correlation as a summary statistic for the degree of multicultural similarity in the items that were rated low, medium, or high. For the CBCL/6-18, the overall mean correlation was .74. For all the other instruments, the overall mean correlations were also in the .70s, indicating a high degree of similarity according to Cohen's (1988) criteria (Rescorla et al., 2011, 2012a, 2012b, 2014a, 2014c). In other words, for ages 1½–5, 6–18, and 18–59 years, informants' ratings were quite consistent with respect to problems that received low, medium, or high ratings across very diverse societies.

Construction of Multicultural Norms

Findings that hundreds of thousands of people in over 50 societies were willing and able to complete the standardized rating instruments indicates their acceptability for assessing members of those societies. The CFA support for the empirically based syndromes, the large multicultural correlations between mean item ratings, and the consistent age and gender effects also indicate that the scales and items function similarly in many societies. However, the small to medium effect sizes for cross-society differences in scale scores argue for different sets of norms with which to evaluate the scale scores obtained by individuals in different societies.

Comparisons of the mean Total Problems scores obtained on each ASEBA instrument in the societies in which the instrument was administered revealed normal distributions of mean Total Problems scores across those societies. Although there were statistically significant differences between the mean Total Problems scores from different societies, the range from the lowest to the highest mean Total Problems score spanned only about 13% of the possible range of Total Problems scores. Furthermore, about 68% of the societies had mean Total Problems scores within one standard deviation (*SD*) of the *omnicultural mean* (the mean of the mean Total Problems scores of all the societies having data for a particular form). The mean Total Problems scores of about 16% of the societies' were >1 *SD* below the omnicultural mean, while about 16% were >1 *SD* above the omnicultural mean. (The percentages were obtained by averaging the percentages found for each instrument.)

Following common psychometric practice, the societies were divided into groups demarcated by 1 *SD* below and 1 *SD* above the omnicultural mean (Achenbach &

Rescorla, 2007, 2010, 2015). Societies whose mean Total Problems scores are >1 *SD* below the omnicultural mean are designated as Group 1, societies whose mean Total Problems scores range from –1 to +1 *SD* from the omnicultural mean are designated as Group 2, and those whose mean Total Problems scores are >1 *SD* above the omnicultural mean are designated as Group 3.

Separately for each instrument, multicultural norms were constructed for Group 1 by computing cumulative frequency distributions of scores on each scale obtained by individuals in each Group 1 society, separately by gender within each age group. For each gender/age group, a new cumulative frequency distribution was then created for each scale by averaging the cumulative frequency distributions from all the Group 1 societies. Percentiles of scores in this mean cumulative frequency distribution were used as the basis for assigning normalized *T* scores to the raw scores. The same procedure was followed for Group 3 societies. Because the U.S. mean Total Problems score was at the middle of the distribution of Group 2 mean Total Problems scores for most instruments and because the U.S. norms had already been used in many research and clinical applications, the U.S. norms are used for Group 2 for those instruments.

Practical Applications of Multicultural Norms

To facilitate applications of the multicultural norms to individuals for both research and practice, the norms are incorporated into computer software for scoring the ASEBA instruments. This enables users to obtain *T* scores based on norms for societies relevant to assessment of each individual and also to display each individual's scale scores on profiles in relation to appropriate multicultural norms.

As an example, if a 14-year-old girl's mother came from Puerto Rico and her father came from Jamaica, scale scores obtained from her mother's CBCL/6-18 ratings could be displayed in relation to norms appropriate for Puerto Rico, while scale scores obtained from her father's CBCL/6-18 ratings could be displayed in relation to norms appropriate for Jamaica. If the girl attended a school in the United States, TRF scale scores from her teachers' ratings could be displayed in relation to U.S. TRF norms. And, if the girl had lived much of her life in the United States, her YSR scale scores could be displayed in relation to U.S. norms. The software would compare the scale scores from each informant's ratings on bar graphs, with each bar standardized for the girl's age, gender, the type of informant (parent, teacher, self), and the appropriate multicultural norms. The user could thus see whether any scale scores were clinically deviant in relation to any of the relevant norms.

ADVANCING ASSESSMENT, TAXONOMY, AND DIAGNOSIS

To set the stage for considering how assessment, taxonomy, and diagnosis can be advanced, Figure 2.7 illustrates the dynamic and continuous relations between them.

To be accurate, diagnosis requires valid ways of conceptualizing the phenomena that are to be diagnosed. Taxonomies provide frameworks for conceptualizing psychopathology in terms of similarities and differences between behavioral/emotional/social problems that can then provide targets for diagnosis. Taxonomic frameworks for problems are commonly conceptualized in terms of syndromes. The value of particular syndromes depends on the degree to which they are found to have important correlates, such as differences in etiology, prognosis, responsiveness to particular interventions, and long-term developmental pathways. To develop reliable, valid, and useful taxonomies, we need reliable and valid assessment data that can be obtained for diverse samples of people. After a taxonomy has been derived from assessment data, the taxonomy can be used to guide assessment of new cases to determine which taxa the cases resemble.

In Figure 2.7, *diagnosis* includes *diagnostic processes, formal diagnoses,* and *diagnostic formulations*. To avoid confusion about diagnosis, it is important to keep in mind these three different aspects of diagnosis. Assessment and taxonomy are important contributors to all three aspects

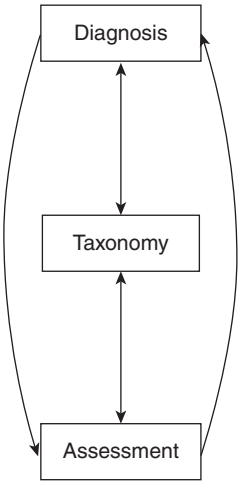


Figure 2.7 Relations between assessment, taxonomy, and diagnosis. As explained in text, *diagnosis* includes *diagnostic processes* (gathering data), *formal diagnoses* (classifying cases and disorders), and *diagnostic formulations* (integrating all relevant data for each case). *Source:* Copyright T. M. Achenbach. Reproduced by permission.

of diagnosis. Diagnostic processes clearly include assessment operations that should be guided by a taxonomy of syndromes. Formal diagnoses involve identification of the taxa that are most relevant to each case. And diagnostic formulations integrate information specific to the case with the type of information on which taxonomic decisions and formal diagnoses are based.

Challenges to Be Met

DSM-III (American Psychiatric Association, 1980) was an important milestone in advancing the nosological approach toward more explicit and testable criteria for psychopathology. Since 1980, *DSM-III* and its successors have generated a tremendous amount of research, as well as major changes in clinical training and practice. An especially important research goal has been to improve the diagnosis of *DSM*-defined disorders via SDIs. Accordingly, enormous resources have been invested in developing, testing, and revising SDIs as ways of operationalizing the *DSM-III*, *DSM-III-R*, and *DSM-IV* criteria (e.g., Costello et al., 1984; Shaffer et al., 2000). Further revisions and testing will be needed to operationalize *DSM-5* criteria.

Efforts to diagnose *DSM*-defined disorders via SDIs have molded research, training, and practice according to the *DSM*'s diagnostic model, which requires yes-or-no decisions about each criterial feature of each diagnostic category. SDIs, such as the DISC for children and DIS for adults, are viewed as quasi-gold standards for assessing psychopathology. However, studies of SDIs have revealed major challenges for assessment, taxonomy, and diagnosis of psychopathology. We first summarize these challenges at the level of specific methodological issues. We then consider possibilities for advancing assessment, taxonomy, and diagnosis to improve understanding, prevention, and treatment of psychopathology.

Challenges Revealed by SDI Studies

The challenges can be summarized as follows:

1. *Test–retest attenuation:* Fewer symptoms are typically reported at second interviews than at initial interviews, causing declines in the number of diagnoses over even very short periods such as a week (e.g., Roberts, Solovitz, Chen, & Casat, 1996).
2. *Low to moderate cross-informant agreement:* People report different symptoms about themselves than others report for them, causing disagreements between diagnoses based on self-reports versus informant-reports (e.g., Achenbach et al., 1987, 2005; Meyer, 2002).
3. *Diagnoses made from SDIs often disagree with clinical diagnoses:* Even when designed to operationalize *DSM* criteria, SDIs such as the DISC and DIS often produce different *DSM* diagnoses than clinical assessments (e.g., Jensen & Weisz, 2002; Lewczyk et al., 2003; Rettew, Doyle, Achenbach, Dumenci, & Ivanova, 2009).
4. *High rates of comorbidity:* Rates of comorbidity between some SDI diagnoses are so high as to raise questions about whether the diagnoses actually represent different disorders (e.g., Faraone et al., 1991; Walker et al., 1991; Wilens et al., 2002).
5. *Great variations in prevalence estimates of DSM diagnoses made from* SDIs *in epidemiological surveys:* When *DSM* criteria are operationalized via SDIs, the SDIs yield much greater variation in prevalence estimates than would be expected from sampling differences (e.g., Achenbach et al., 2012; Breton, Bergeron, Valla, Berthiaume, & Gaudet, 1999).

The challenges raised by SDI studies expose fundamental problems that cut across assessment, taxonomy, and diagnosis. These problems need to be addressed in efforts to improve understanding, prevention, and treatment of psychopathology. Some of the problems revealed by SDI studies may afflict other assessment procedures, as well. For example, test–retest attenuation, low to moderate cross-informant agreement, and high rates of covariation (i.e., comorbidity) among different kinds of problems are found in most procedures for obtaining information about people's problems (e.g., Achenbach & Rescorla, 2001, 2003; Krueger & Piasecki, 2002). Agreement between most assessment procedures and *DSM* clinical diagnoses also tends to be low, although this may be most troubling for procedures that are explicitly designed to correspond to *DSM* diagnoses (e.g., Gadow et al., 2002). If assessment procedures are not specifically designed to correspond to *DSM* diagnoses, their modest agreement with *DSM* clinical diagnoses is less surprising than if they are designed to correspond to *DSM* diagnoses.

Although some of the challenges faced by SDIs are quite general, the problems are exacerbated by the *DSM* requirement to make diagnoses by applying fixed rules to the criteria that must be met. For example, a decline in the number of symptoms endorsed from one occasion to another may reflect the general phenomenon of test–retest attenuation. However, according to *DSM*'s fixed rules for the number of symptoms required for each diagnosis, a decline of even one symptom can change decisions from sick to well for *DSM* diagnoses. Similarly, disagreements between informants' reports for even a single symptom

can cause major differences in diagnoses made on the basis of self-reports versus reports by other informants. Equally important, the dependence of *DSM* diagnoses on the precise definition, number, duration, and co-occurrence of reported symptoms may contribute to the great variations in prevalence estimates found in different epidemiological studies. For example, Roberts, Attkisson, and Rosenblatt (1998) found prevalence estimates ranging from 1% to 50% among epidemiological studies of diagnoses of child and adolescent disorders. Assessment procedures that uniformly operationalize a particular set of taxonomic constructs have yielded more consistent epidemiological findings than SDIs that are intended to operationalize *DSM* diagnostic criteria but that vary considerably in how they obtain judgments of whether each criterion is met (e.g., Achenbach et al., 2012; Rescorla et al., 2012b).

Other Types of Challenges

Assessment, taxonomy, and diagnosis also face challenges raised by the need to identify and combine optimal sources and kinds of assessment data for taxonomy and diagnosis; the need to take account of variations related to age, gender, society, and sources of data; and the need to distinguish between cultural differences versus individual differences. Table 2.1 summarizes the challenges. Progress in meeting the various challenges requires systematic research that can bridge the gaps between nosological and empirically based approaches to provide assessment procedures, taxonomic constructs, and diagnostic targets that can be used by both approaches.

TABLE 2.1 Challenges in Advancing Assessment, Taxonomy, and Diagnosis

1.	Test–retest attenuation: The decline in problems reported from initial interviews to subsequent interviews.
2.	Low to moderate cross-informant agreement: Different informants tend to report different problems.
3.	Limited agreement between diagnoses made from SDIs and clinical evaluations.
4.	High rates of comorbidity: Comorbidity between some diagnoses is so high as to raise questions about whether the diagnoses represent different disorders.
5.	Great variations in prevalence rates of *DSM* diagnoses made from SDIs in epidemiological surveys.
6.	The need to integrate multisource data.
7.	The need to take account of differences related to age, gender, society, and sources of data.
8.	The need to distinguish individual differences from cultural differences.

QUANTITATIVE AIDS TO MEETING THE CHALLENGES

DSM-III, *DSM-III-R*, *DSM-IV*, and *DSM-5* have brought significant advances in making diagnostic criteria more explicit. However, the *DSM*'s diagnostic criteria and categories are based on the assumption that each diagnostic category represents a separate disorder. The assumption that each diagnostic category represents a separate disorder may indeed be tenable for disorders for which specific pathogens or other physical abnormalities have been identified. For example, diagnostic categories for infectious diseases such as tuberculosis, diphtheria, and AIDS can be confidently defined in terms of pathogens that are necessary (though not always sufficient) for the disorder to occur. Diagnostic categories for cancer can be defined in terms of specific kinds and locations of tissue pathology, even if specific etiologies have not been identified.

Some forms of psychopathology may eventually be found to involve physical pathogens or other physical etiological factors. However, until physical etiologies are validated, it may be premature to require yes/no decisions for diagnoses of behavioral, emotional, and social problems. Because even physical disorders may not all be identifiable in terms of yes/no dichotomies (e.g., hypertension), the search for physical etiologies may also be more productive if diagnostic criteria and taxa are quantified. In fact, meta-analyses have indicated that quantitative assessments of psychopathology are substantially more reliable and valid than categorical assessments (Markon, Chmielewski, & Miller, 2011).

Even if a categorical diagnostic format is retained, cut points can be established on distributions of quantitative scores. Rather than being established a priori by committees of experts, the cut points can be based on scores that validly discriminate between representative samples of people who are judged to be relatively normal (i.e., healthy samples) versus people who are judged to need mental health services. To take account of age, gender, societal, and informant differences, cut points can be established on the basis of multicultural norms for people of each gender within particular age ranges, according to assessment data obtained from different kinds of informants. To avoid transferring the disadvantages of purely categorical diagnoses to diagnoses based on scores, the cut points should be based on empirical findings of variations related to age, gender, society, and type of informant. Moreover, levels of deviance can be identified for distinguishing between people who have extreme degrees of the problems encompassed by a diagnostic taxon versus people who have less

extreme degrees of the problems versus people who are in the normal range. Even if cut points are imposed on distributions of scores, the continuous scores should be retained to provide more precise measures of problems than are provided by categorization. Quantitative diagnostic scores also have advantages for measuring change over time, such as by comparing scores before, during, and after treatment and by comparing scores at different points in development.

Advantages of Quantification for Research

Quantification of the criterial features of diagnostic taxa and of the diagnostic taxa themselves offers multiple advantages for most kinds of research on psychopathology, as discussed in the following sections.

Preserving Information

One advantage is that quantification of features typically preserves more information about the features than yes/no categorization does. This is especially true when neither the boundaries of each feature nor of the diagnostic categories are well validated. When the boundaries of diagnostic categories are not well validated, imposition of unvalidated boundaries and categorical assessment procedures may cause both false positives and false negatives. Even if certain disorders are ultimately found to have categorical boundaries, measurement error will prevent assessment procedures from infallibly distinguishing between individuals whose problems fall within those boundaries versus individuals whose problems are not entirely congruent with the categorical boundaries.

Avoiding Forced Choices

Quantified assessment procedures avoid forced choices because they capture the degree to which individuals' problems approximate particular diagnostic taxa and because quantification can take account of measurement error. By indicating the degree to which individuals' problems resemble each relevant diagnostic taxon, quantified assessment can help to prevent the premature closure risked by placing individuals in diagnostic categories that may be wrong.

Increasing Precision

A further advantage of quantification is that it can improve research by increasing the precision of data obtained about each characteristic of each individual. It can also improve research by enabling the use of more varied and powerful analytic tools than are available for categorical data. These analytic tools are especially useful for the following research areas that are increasingly important for advancing assessment, taxonomy, and diagnosis: Identification of patterns of psychopathology and their correlates; life span developmental analyses of adaptive and maladaptive functioning; behavior genetics and epigenetics; epidemiology; efficacy, effectiveness, and outcomes of different kinds of interventions; pharmacokinetics of medications; biochemical correlates of behavioral and emotional problems; and multicultural applications.

Advantages of Quantification for Conceptualizing Psychopathology

Quantitative Aspects of Prototypes

In earlier sections, we outlined prototypes as models for taxonomy. Insofar as people mentally represent categories in terms of prototypes (sets of correlated features), mental representations of even categorical taxonomic constructs involve quantification. That is, a prototype for a category encompasses multiple features that are not perfectly correlated with each other. On the other hand, individuals who manifest many features of a prototype are more easily assigned to the category represented by that prototype than individuals who manifest fewer features of the prototype. Individuals who manifest multiple features of more than one prototype may be especially hard to classify into a single category. If such individuals have enough problems to need mental health services, then their problems can be described in terms of scores on multiple prototypes. According to *DSM*'s rules, individuals who manifest enough prototypical features of two categories to qualify for both categories are assumed to have the disorders represented by both categories, i.e., they have comorbid disorders. (Although some editions of *DSM* have included preemptive rules, such as CD taking precedence over ODD when criteria for both disorders are met, these rules are often suspended in research and practice.)

If diagnostic categories are quantified in terms of the number of their prototypical features, the categories would correspond more closely to people's mental representations in terms of prototypes. In other words, because people use the number of features corresponding to their mental prototypes to help them classify individuals and because the reliability and confidence with which individuals are classified depend on the number of prototypical features manifested, diagnostic categories may be used more effectively if they take advantage of the quantification that is inherent in mental classification processes. Because psychopathology often includes features of multiple diagnostic categories, quantification of each category would allow users of the categories to think about borderline cases and

comorbidity explicitly in terms of the scores obtained for each diagnostic category. Borderline cases, for example, could be operationally defined in terms of scores that are high enough to be somewhat above the normal range but not so high as to be clearly clinically deviant. Furthermore, manifestations of features of multiple categories could be explicitly documented in terms of profiles of scores for all the relevant categories without implying that each elevated score indicates a separate (or comorbid) disorder.

Conceptualizing Cross-informant Variations

Quantification of diagnostic taxa can also facilitate conceptualization of cross-informant variations in assessment data. By allowing for variations in the degree to which particular problems are manifested and reported, quantified taxa can help us use both the differences and the similarities in reports by different informants for determining the overall level of individuals' problems and how consistently they are manifested. For example, if all informants report high levels of attention problems for a child, this would provide consistent evidence for a disorder that involves attention problems, such as ADHD. On the other hand, suppose that a teacher reports attention problems but a parent reports that the child concentrates intently on video games. A purely categorical system forces us to choose between the teacher's and parent's reports to make a yes/no decision about whether the child has ADHD. However, quantified taxa enable us to integrate scores obtained from different informants into the scores for each relevant category. This can be done by considering the possible situational specificity of problems, using weighted means of scores, matching profile patterns from different informants to profile types, and matching scores from different informants to latent classes or latent profiles.

In summary, the quantitative aspects of mental prototypes provide a natural basis for conceptualizing psychopathology in terms of quantified taxa. Quantified taxa facilitate diagnostic decisions about individuals who need help but whose problems are too diverse or situation-specific to meet criteria for a single diagnostic category, as well as individuals who meet criteria for multiple diagnostic categories. Quantified taxa also facilitate thinking about individuals who have significant problems that correspond to a particular diagnostic category but that do not quite reach the threshold required for a categorical diagnosis. Equally important, when different informants report different problems for an individual, profiles of quantified categories scored from each informant can be used to document similarities and differences in how the individual is perceived.

Advantages of Quantification for Mental Health Services

For mental health services, a fundamental advantage of quantification is that decisions about who needs services and what kind of services they need can be based on clients' profiles of adaptive and maladaptive functioning rather than on whether they meet categorical criteria for a particular diagnosis. Excessive reliance on diagnostic categories as a basis for treatment decisions may exclude clients who need help but do not fit the categories. On the other hand, if clients meet criteria for a diagnostic category, it is then too tempting to assume that they are ill even if they are adapting well. Furthermore, excessive reliance on diagnostic categories may cause treatment to focus more narrowly on the categories than is warranted either by the client's needs or by the ability of categorically based treatments to ameliorate broad spectra of problems. This may be especially true when a mental health service specializes in a particular diagnostic category such as depression, anxiety, or ADHD. Because the diagnostic procedures, treatment modalities, and clinicians in such services tend to focus on the target category, clients and their problems may be viewed primarily through the lens of that category. Assessment of multiple quantified taxa can reveal the degree to which individuals' problems really are or are not concentrated in a particular taxon. Table 2.2 summarizes these advantages.

TABLE 2.2 Potential Advantages of Quantifying Criterial Features and Taxonomic Constructs

1.	Avoids yes-versus-no forced choices about the presence of features and diagnosis of disorders.
2.	Allows cut points on distributions of scores that can reflect age, gender, informant, clinical status, and other differences.
3.	Provides more precise measurement of psychopathology than categorization does.
4.	Allows use of more powerful analytic tools than categorization does for research on patterns of psychopathology, developmental trajectories, behavior genetics, epidemiology, effects of interventions, pharmacokinetics, biochemical variables, and cross-cultural variations.
5.	Corresponds to mental representations of taxa in terms of prototypes.
6.	Quantified covariation between problems avoids dilemmas of comorbidity between categorical disorders.
7.	Facilitates aggregation of multisource data in taxonomic constructs.
8.	Provides more precise basis for decisions about treatment and evaluations of progress and outcomes.
9.	Facilitates targeting of specific problems in relevant contexts.
10.	Measures specific problems independently of criteria for diagnoses.

EFFORTS TO QUANTIFY DIAGNOSTIC TAXA

The following sections outline efforts to bring the benefits of quantification to diagnostic taxa.

Rating Scales for *DSM* Categories

One approach to quantifying diagnostic taxa is to reproduce the symptom criteria for individual *DSM* categories in rating scales, such as those described next.

ADHD Rating Scale-IV

DuPaul et al. (1998) constructed the ADHD Rating Scale-IV, which consists of the *DSM-IV* symptom criteria for the Inattentive and Hyperactive-Impulsive types of ADHD. Parents and teachers rate each symptom as *0 = Never or rarely*, *1 = Sometimes*, *2 = Often*, or *3 = Very often*. The ratings are then summed to provide scale scores for Inattention, Hyperactivity-Impulsivity, and Total ADHD. DuPaul et al. (1998) reported that categorical diagnoses of ADHD correlated between .08 and .44 with scores on the scales of the ADHD Rating Scale-IV completed by parents and teachers. However, DuPaul et al. also reported correlations of .76 to .88 between teachers' ratings on the ADHD Rating Scale-IV and the corresponding quantitative scales of the Conners Teacher Rating Scale-39 (C-TRS; Conners, 1997).

Youth's Inventory-4

In an effort to quantify a broader range of *DSM* diagnostic taxa, Gadow and colleagues developed rating scales consisting of symptoms from multiple *DSM-IV* categories. As an example, the Youth's Inventory-4 (YI-4; Gadow et al., 2002) is a self-rating form consisting of *DSM-IV* symptoms that youths rate 0, 1, 2, or 3 according to gradations like those of the ADHD Rating Scale-IV. Symptom severity scores are obtained by summing the numerical ratings on each scale. Versions of a companion rating form, the Adolescent Symptom Inventory-4 (ASI-4), are designed to obtain parent and teacher ratings of many of the same *DSM* symptoms.

Using a sample of 11- to 18-year-olds whose parents requested an outpatient evaluation, Gadow et al. (2002) tested associations between symptom severity scores on the *DSM* scales of the YI-4 and *DSM* diagnoses in the youths' clinic charts. The diagnoses were analyzed in terms of the following five categories: ADHD, ODD, CD, depressive disorders, and anxiety disorders, although many youths qualified for multiple categories. Gadow et al. then identified the YI-4 *DSM* scales whose symptom severity scores had the highest point-biserial *r*s with each category. The highest *r*s ranged from .06 for the YI-4 ODD scale with *DSM* diagnoses of ODD to .38 for the Major Depressive Disorder scale with *DSM* diagnoses of depressive disorders, with mean point–biserial $r = .25$ (computed by Fisher's z transformation).

Although the authors did not report associations between diagnoses and parent or teacher ratings, the Pearson *r*s between the YI-4 and parents' ASI-4 ratings on corresponding *DSM* scales ranged from .05 to .50. All correlations between the YI-4 and teachers' ASI-4 ratings on the corresponding *DSM* scales were extremely low ($r < .18$). However, the YI-4 scales yielded many *r*s in the .60s and .70s with the youths' self-ratings on non *DSM*-based scales scored quantitatively from the Children's Depression Inventory (CDI; Kovacs, 1992), the Multidimensional Anxiety Scale for Children (MASC; March, 1997), and the YSR. Thus, although YI-4 *DSM* symptom severity scores did not yield much agreement with *DSM* clinical diagnoses nor with parent or teacher ratings, self-ratings on the YI-4 *DSM* symptom scales correlated highly with self-ratings on other quantitative measures of problems.

Findings for the ADHD Rating Scale-IV and the YI-4 thus suggest that parent, teacher, and self-ratings of actual *DSM* symptom items do not necessarily agree well with *DSM* diagnoses that are based on the same items. The obstacles to agreement between the *DSM* scales and *DSM* diagnoses could include variations in the data used by the clinicians who made the diagnoses, plus their use of additional criteria such as the duration of symptoms.

DSM-Oriented Scales

Another approach to quantifying *DSM* diagnostic categories is to start with items that are of proven value for obtaining ratings by the relevant informants and then to select those items that are very consistent with *DSM* diagnostic categories, whether or not the items have exact counterparts among the *DSM* symptom items. Because each *DSM* diagnostic category in effect represents a diagnostic construct, experts' understanding of the diagnostic constructs may provide a good basis for selecting items that are appropriately rated by informants, even if the items are not precisely the same as the *DSM* symptom criteria. However, because experts' judgments may vary with such characteristics as their professional training, the settings in which they work, the types of clients they see, and their cultural backgrounds, it is important to identify items on which diverse experts' judgments converge.

To make use of judgments by experts who varied greatly with respect to their training, work settings, clients, and cultures, Achenbach, Dumenci, and Rescorla (2001) invited 22 psychiatrists and psychologists from 16 cultures around the world to rate CBCL/6-18, TRF, and YSR problem items for their consistency with *DSM-IV* diagnostic categories for child and adolescent disorders. The experts had all published on psychopathology and had a mean of 19.1 years of experience since their first doctorate. Five had both an MD and a PhD. The experts were given the *DSM* criteria for nine categories of disorders that are defined largely in terms of behavioral, emotional, and social problems. They then rated each problem item as 0 = not consistent, 1 = somewhat consistent, or 2 = very consistent with each *DSM* category. Because the objective was to obtain the experts' judgments of the diagnostic constructs represented by the *DSM* categories, they could rate problem items as being very consistent with a category even if the items did not have precise counterparts among the *DSM* symptom criteria.

Items that were rated as very consistent with a *DSM* category by at least 64% of the experts were assigned to a scale for that category. The scales are designated as "*DSM*-oriented" because they capture what is common to diverse experts' understanding of the diagnostic constructs underlying *DSM* categories, rather than being verbatim statements of the *DSM* criteria. Enough items met criteria to form *DSM*-oriented scales that were designated as follows: affective problems (including dysthymia and major depression); anxiety problems (including generalized anxiety disorder, separation anxiety disorder, and specific phobia); somatic problems (including somatization and somatoform disorders); ADHD problems (including hyperactive-impulsive and inattentive types); oppositional defiant problems; and conduct problems.

To enable users to judge the degree of deviance indicated by scores on the *DSM*-oriented scales, normative data have been obtained for population samples of 6–18-year-olds living in 42 societies. Scores on the *DSM*-oriented scales are displayed on profiles in relation to multicultural norms for each gender according to particular age ranges, separately for parent ratings on the CBCL, teacher ratings on the TRF, and self-ratings on the YSR (Achenbach & Rescorla, 2001, 2007a; Rescorla, 2012b).

Relations to DSM Diagnoses

Point-biserial *r*s have been reported between *DSM-IV* clinical diagnoses and the *DSM*-oriented scales in a university psychiatry clinic sample (Achenbach & Rescorla, 2001, p. 130). Like the diagnoses reported by Gadow et al. (2002) for youths who completed the YI-4, the diagnoses were obtained from clinical records and were based on multiple sources of data. However, unlike the ages 11 to 18 spanned by the Gadow et al. data for self-ratings on the YI-4, the data for the *DSM*-oriented scales spanned ages 6 to 18 and were obtained from parents' ratings on the CBCL/6-18. For the same diagnostic categories as Gadow et al. used, the correlations between *DSM* clinical diagnoses and the corresponding *DSM*-oriented scales ranged from .34 for CD to .60 for ADHD, with mean point-biserial $r = .45$ (computed by Fisher's z transformation).

As another way of assessing *DSM* diagnoses, clinicians administered the *DSM-IV* Checklist (Hudziak, 1998) to family members who were involved in the clinical evaluation of the children. The *DSM-IV* Checklist consists of questions corresponding to each of the criterial symptoms for each diagnostic category. Although the questions are quoted from the *DSM-IV* symptom criteria, clinical interviewers are free to rephrase the questions for the benefit of the interviewees. The score for each diagnostic category is the sum of symptoms judged to be present. For the same diagnostic groupings as Gadow et al. used, the Pearson *r*s with the corresponding *DSM*-oriented scales ranged from .43 for anxiety disorders to .80 for ADHD, with mean $r = .63$ (computed by Fisher's z transformation). Recall that the *DSM*-oriented scales do not consist of items reproduced verbatim from the *DSM* symptom criteria. Nevertheless, their mean *r* of .45 with *DSM* clinical diagnoses and .63 with scores for *DSM* diagnoses on the clinician-administered *DSM-IV* Checklist were significantly higher ($p<.05$) than the mean *r* of .25 between *DSM* clinical diagnoses and the YI-4 scales that did consist of verbatim *DSM* symptom criteria. This suggests that quantification of *DSM* diagnostic constructs may not be most effectively done by obtaining informants' responses to the verbatim *DSM* symptom criteria. The *DSM*-oriented scales have since been slightly revised on the basis of *DSM-5* diagnostic criteria (Achenbach, 2014).

Bridging Gaps Between Nosologically and Empirically Based Constructs

The top-down and bottom-up strategies that characterize nosologically and empirically based constructs, respectively, are arguably the most common approaches to taxonomy of psychopathology. Each makes use of existing knowledge and concepts in different ways. Both may help to advance assessment, taxonomy, and diagnosis, especially if the potential strengths of both can be combined. One way to combine their strengths is to use a common set of

assessment procedures for nosologically and empirically based constructs. Such a combination is exemplified by the *DSM*-oriented scales that are scored from the same pool of items from which empirically based syndromes are scored (Achenbach, 2014; Achenbach & Rescorla, 2000, 2001, 2003). For children and adults, scores for both types of scales are displayed on profiles in relation to age-, gender-, and informant-specific multicultural norms (Achenbach & Rescorla, 2007a, 2010, 2015). The two types of scales have similar psychometric properties, and there are medium to strong associations between some scales of the two types (Achenbach, Bernstein, & Dumenci, 2005; Achenbach, Dumenci, & Rescorla, 2003b). The ability to score both types of scales from the same items—which are rated by the people being assessed and by informants in about 15 minutes—makes it easy to compare nosologically and empirically based findings in many research and clinical contexts for many purposes.

Longitudinal Research Designs

In previous sections, we presented accelerated longitudinal, path, and growth curve designs as examples of research designs that may be especially useful for developmental research related to assessment, taxonomy, and diagnosis. Some longitudinal studies, such as the Dunedin, New Zealand project (Newman et al., 1996), have reported *DSM* diagnoses for the same participants at different ages. However, application of powerful longitudinal designs to nosological constructs has been limited by the difficulty of operationalizing nosological constructs via SDIs that must be tailored to the different developmental levels of large representative samples of participants repeatedly assessed over long periods. Additionally, major changes in diagnostic criteria from one edition of the *DSM* to another make it hard to link diagnoses based on the different editions. Equally important, although longitudinal growth models can utilize categorical variables such as diagnoses, these categorical variables do not capture as much information about individual differences between people as quantitative scales do.

DSM-oriented scales that can be scored quantitatively from the same brief assessment instruments as empirically based scales facilitate research on nosologically based constructs and comparisons of results obtained for empirically based constructs. Scores on quantitative *DSM*-oriented scales based on ratings from multiple informants can also serve as indicators for latent variable analyses. Latent variables estimated from such indicators can then be tested as predictors, moderators, mediators, and outcomes in path or growth curve analyses. It is thus possible to use accelerated longitudinal, path, and growth curve designs to track relations between developmentally early and later measures of nosologically based constructs to test their developmental stability and the degree to which they predict various outcomes.

Genetic and Other Biological Research

Quantified phenotypic measures of nosological constructs make it possible to test complex quantitative models for genetic and environmental effects on psychopathology. Because these measures can be economically obtained for very large samples of twins and other genetically informative samples, they yield much more powerful genetic analyses than do structured diagnostic interviews, which would be financially prohibitive for such large samples (e.g., Boomsma et al., 2001).

In addition to genetic research, studies of other biological variables can be strengthened by using quantitative measures of nosological constructs. For example, several studies have found significant correlations between the empirically based Aggressive Behavior syndrome and a variety of biochemical measures (e.g., Gabel, Stadler, Bjorn, Shindledecker, & Bowden, 1993; Hanna, Yuwiler, & Coates, 1995; Scerbo & Kolko, 1994). Quantitative scales for measuring nosologically based constructs can provide more powerful and differentiated tests of associations between biochemical variables and diagnostic constructs than categorical diagnoses can.

Research on Secular Trends in Psychopathology

It has been widely noted that the previously rare diagnoses of pervasive developmental disorders (PDD; now called autism spectrum disorder in *DSM-5*), including autism and Asperger's disorder, have become more common (e.g., Chakrabarti & Fombonne, 2001). Reviews of documents have also led to the conclusion that adolescent conduct disorders, substance abuse, depression, and suicide have increased (Rutter & Smith, 1995). Secular (i.e., long-term) changes in rates of psychopathology are apt to be very important, because they may provide clues about etiological factors and protective factors. Secular changes in rates of psychopathology may also argue for new efforts at prevention and treatment. Unfortunately, changing rates of diagnoses may reflect changing diagnostic criteria, diagnostic categories, and even the willingness of mental health workers to make certain diagnoses. As an example, autism was previously blamed on unconscious parental attitudes (e.g., Wolman, 1970, p. vii), which meant that it was a very undesirable diagnosis. Autism has since come to be

regarded as a developmental disability for which intensive services are now provided. Even documentation of suicide may reflect cultural changes, as there is evidence that suicides by young people were previously underreported on death certificates (Males, 1991).

To measure changes in the behavioral and emotional problems on which diagnoses are based, it is necessary to assess representative samples of populations with the same standardized procedures at different points in time. Problem rates can then be compared to measure changes, whether or not there have been changes in diagnostic criteria, categories, or willingness to make certain diagnoses. As an example, studies in the United States and the Netherlands have used the same standardized procedures to obtain CBCL and TRF ratings of different representative samples of children at intervals separated by as much as 23 years (Achenbach, Dumenci, & Rescorla, 2002, 2003a; Tick, van der Ende, & Verhulst, 2007b). In the American samples, empirically based syndromes and *DSM*-oriented scales both showed small but significant increases in problem scores in the 1980s, followed by small but significant decreases in problem scores in the 1990s. From 1983 to 2003, the Dutch samples showed small increases in some kinds of problems. For CBCLs completed by parents of 2–3-year-olds in 1989 and 2003, another Dutch study found little change over the 14-year interval (Tick, van der Ende, Koot, & Verhulst, 2007b). The percentage of children who obtained clinically deviant scores on the *DSM*-oriented pervasive developmental problems scale actually declined slightly, which argues against a general increase in autism spectrum problems.

A study in the United Kingdom (UK) compared parents' reports of problems for 15- and 16-year-olds in cohorts that were assessed in 1974, 1986, and 1999 (Collishaw, Maughan, Goodman, & Pickles, 2004). The assessment instruments differed somewhat among the three studies and only 11 problem items were similar enough for comparison across the three cohorts. Significant increases in conduct problems were found from the 1974 to the 1986 and the 1999 cohorts. Emotional problems and hyperactivity were also significantly higher in 1999 than 1974 or 1986. Although it is certainly possible that secular trends differ among countries, it is difficult to draw firm conclusions from the UK data, because of differences in the assessment instruments and because the completion rates were evidently only 62% in 1974 and 45% in 1986 (Collishaw et al., 2004, p. 1353). The 1999 completion rate was evidently not reported, but samples with high attrition rates often include smaller proportions of people with many problems than do samples with low attrition rates.

DIRECTIONS FOR CONTINUED RESEARCH

Important advances are being made in the developmental study of psychopathology, but much remains to be done. In this section, we outline directions in which assessment, taxonomy, and diagnosis can continue to advance. Although these are not the only directions, they are particularly important for extending the developmental study of psychopathology to adults, to elders, and to more diverse cultures.

Family-Oriented Assessment and Interventions

There are many studies of child/adolescent psychopathology and of adult psychopathology. Research on psychopathology among the elderly is also growing rapidly. However, there is far less research on the developmental course of psychopathology from childhood into adulthood and from adulthood into old age. Several factors may account for the dearth of research that bridges major developmental periods. One factor is that clinicians and researchers tend to specialize in work with children and adolescents, or adults, or the elderly, rather than spanning these different periods. A second factor is that concepts of psychopathology tend to be formulated separately for children and adolescents, for adults, and for the elderly. And a third factor is that assessment, taxonomy, and diagnosis tend to be segmented according to these major life periods.

To fully understand both adaptive and maladaptive functioning within each life period, as well as the continuities and discontinuities between periods, we need assessment procedures that provide continuity across broad age spans. Much of the research on prediction of adult psychopathology from childhood variables has used very different measures of child and adult problems. Because certain behaviors that are considered deviant in childhood would not be deviant in adulthood and vice versa, assessment must be tailored to the developmental level of the people assessed. However, methodologically similar assessments of similar variables for people of different ages are likely to provide better pictures of continuities and discontinuities between developmental periods than are assessments that differ with respect to both the methodology and the variables assessed. Our section on Life Span Applications illustrated some ways in which similar standardized methodology has been used to assess psychopathology across major periods of life. However, far more needs to be done to systematically compare and contrast findings for different life periods and to bridge

the gaps between them with longitudinal research. Use of similar standardized methods to assess people as they grow older makes it possible to apply accelerated longitudinal designs, which can yield developmental findings more quickly and cost-effectively than traditional longitudinal designs.

For clinical as well as research purposes, similar standardized methods can be used to assess different generations within families, such as parents and their children, to identify cross-generational similarities and differences. As an example, factor analyses of ratings of ASEBA problem items have yielded seven similar syndromes for ages 6–18 and 18–59, plus similar broadband Internalizing and Externalizing groupings of syndromes. This makes it possible to compare ratings of 6–18-year-olds and their parents on nine empirically derived scales, as well as on *DSM*-oriented scales that are similar for ages 6–18 and 18–59. The Multicultural Family Assessment Module (MFAM; Achenbach, Ivanova, & Rescorla, 2015) enables researchers and practitioners to display bar graph comparisons of scores for parents and their children on the seven counterpart syndromes, Internalizing, Externalizing, and *DSM*-oriented scales, standardized by age, gender, informant, and multicultural norms. Practitioners in child and family services can show these bar graphs to parents to highlight similarities and differences between self-reported and collateral-reported problems vis-à-vis both the parents themselves and their child. Helping parents see both the variations in problems reported for themselves and for their child and helping them identify parallels between their problems and their child's problems can greatly facilitate family-oriented services.

Multicultural Directions

Globalization has many facets. Although economic globalization may be the most publicized facet, cultural globalization is also important. Cultural globalization stems partly from increasing access to a common set of media for information and entertainment. However, it also stems from mass movements of immigrants and refugees from their indigenous societies to very different societies. Host societies are often ill prepared to provide mental health and related services to immigrants. In addition to language barriers, challenges arise from differences in attitudes toward mental health services, as well as from differences in what is deemed to be normal versus abnormal. Other challenges arise when mental health professionals from developed societies seek to help people in developing societies.

Both to build a more comprehensive science of developmental psychopathology and to use that science to help more people, it is important to use assessment instruments and data that extend across multiple societies. Our section on Multicultural Research reviewed multicultural findings for psychopathology in children and adults, using both bottom-up and top-down assessment procedures.

Quantification of Diagnostic Taxa

Nosological approaches typically require categorical yes/no judgments of each criterial feature of each diagnostic category. These approaches also require yes/no decisions as to whether an individual meets the overall criteria for a diagnosis. However, the categorical, yes/no format is not necessarily an immutable part of the nosological approach. A dimensional approach, whereby symptoms are summed to obtain a score for a diagnosis, is often viewed as the main quantitative alternative to the categorical approach. Yet, summing the number of symptoms is only one of many ways to reap the benefits of quantification for increasing precision, reliability, validity, and statistical power. For example, allowing diagnosticians to rate symptoms and other diagnostic criteria on 3-step scales rather than requiring yes/no forced choices could make the diagnostic process easier, more reliable, and better differentiated. Categorical cut points can still be retained for deciding whether an individual meets criteria for a diagnosis by specifying diagnostic thresholds in terms of the sum of three-step ratings, rather than the number of symptoms. Further benefits of quantification can be obtained by providing age, gender, and multicultural norms for the distributions of scores within each diagnostic category. For categories in which the distributions of scores differ significantly by age, gender, or society, diagnostic thresholds can be chosen to mark a similar degree of deviance for each group, if desired. Quantification can also be used to combine data from multiple sources as a basis for making diagnostic decisions.

As an alternative to dimensional approaches, person-centered quantification can be used to identify categories of individuals who have similar patterns of problems. Methods for using quantitative diagnostic data to identify types or categories of individuals include latent profile analysis, cluster analysis, and latent class analysis. A particularly important use of quantification for individual patterns is known as *patient-focused analysis*. This involves repeated measurement of individuals' scores during and after interventions to detect statistically reliable changes in magnitudes or slope parameters (e.g., Lambert, Hansen, &

Finch, 2001). In short, even elementary quantification, such as rating diagnostic criteria on 3-step scales, would enable the nosological approach to capitalize on the many benefits of quantification that have long been utilized by other approaches to assessment and taxonomy.

Use of Norms to Evaluate Interventions

Numerous studies have been designed to improve prevention and treatment of psychopathology (e.g., Kazdin & Weisz, 2003; Roberts, Kane, Thomson, Bishop, & Hart, 2003). However, when prevention and treatment are targeted on diagnoses that are assumed to reflect enduring characteristics of the individuals who meet diagnostic criteria, as implied by diagnoses such as ADHD and personality disorders, it is difficult to evaluate the effects of the interventions, because the diagnoses are not expected to change. Whether or not particular forms of psychopathology actually involve such enduring conditions, accurate evaluation of intervention effects requires measurement of change from clinical deviance toward the normal range of functioning. To determine whether interventions do produce statistically significant improvements, controlled studies are needed in which norms are used to measure the degree to which participants' preintervention scores move from being clinically deviant to postintervention scores that are more typical of representative normative samples of peers (Achenbach, 2001; Sheldrick, Kendall, & Heimberg, 2001). Because successful adaptation requires not only an absence of problems but the presence of positive competencies, the effects of interventions should be evaluated in relation to norms for competencies as well as for problems.

Use of Multisource Data

Low levels of agreement between clinical diagnoses and SDI diagnoses, as well as the modest correlations between reports by different informants for both adult and child psychopathology, indicate that no single source of data is likely to suffice for comprehensive assessment of psychopathology. Although assessment of children usually includes data from multiple informants, multi-informant data are also needed for adults. Even when multi-informant data are obtained, challenges remain with respect to documenting specific similarities and differences between reports by different informants. As an example of ways to compare reports by multiple informants, we have developed computer software that displays data from multiple informants in terms of side-by-side comparisons of item scores and bar graphs of scale scores (Achenbach & Rescorla, 2015). The software also displays the degree of agreement between informants in terms of *Q* correlations and compares the *Q* correlations obtained for each case with those obtained for large samples of informants. However, much more research is needed on how best to integrate data from different sources as a basis for decisions about diagnoses, interventions, and outcomes.

SUMMARY

Numerous advances are occurring in knowledge and methods related to developmental issues in assessment, taxonomy, and diagnosis of psychopathology. In this chapter, we defined *psychopathology* as persistent behavior, thoughts, and emotions that are likely to interfere with developmental tasks necessary for long-term adaptation. The major themes of our chapter are summarized below.

First, it has become clear that developmental approaches are needed to understand psychopathology across the life span. This means that we need assessment procedures, taxonomic models, and diagnoses geared to the problems characteristic of each developmental period. At the same time, to track developmental sequences and outcomes, continuity is needed between assessment procedures from one developmental period to another.

Second, the developmental study of psychopathology has demonstrated the importance of multisource data for adults as well as children. Research has revealed that correlations between adults' self-reports and reports by other informants are as low as those found for children.

Third, multicultural perspectives are essential. By applying the same standardized multi-informant assessment procedures in different societies, we can identify similarities and differences in patterns of reported problems to learn whether the same assessment procedures, taxonomic constructs, and diagnoses are applicable across societies. Procedures that are found to perform similarly across societies can be used to compare the course and correlates of psychopathology in different societies.

We described developmental methodology that is applicable to the study of psychopathology across the life span. Real-time longitudinal, accelerated longitudinal, path, and growth curve designs are especially useful.

We contrasted nosologically based models of psychopathology that employ a *top-down* strategy (whereby experts formulate diagnostic categories and then choose criteria for the categories) with empirically based models that employ a *bottom-up* strategy (whereby problems rated

for large samples of people are analyzed statistically to derive syndromes of co-occurring problems). We explained that *assessment* refers to gathering data to identify the distinguishing features of individual cases, whereas *taxonomic* models group cases according to their distinguishing features. Prototypes, which represent categories quantitatively in terms of sets of imperfectly correlated features, provide taxonomic models that mesh well with human information processing. We also discussed three different meanings of the term *diagnosis*: *diagnostic processes, formal diagnoses,* and *diagnostic formulations*. In this context, we suggested that the high rates of comorbidity found for *DSM* diagnoses raise questions about whether each diagnostic category truly represents a separate disorder.

To advance assessment, taxonomy, and diagnosis, we need to meet the following challenges: test–retest attenuation, i.e., the tendency for more problems to be reported at initial than subsequent assessments; limited cross-informant agreement; limited agreement between clinical diagnoses and diagnoses made from SDIs; high rates of comorbidity; great variations in prevalence rates of *DSM* diagnoses made from SDIs; the need to integrate multisource data; the need to take account of differences related to age, gender, and data sources; and the need to distinguish individual differences from cultural differences. We suggested that quantification of nosologically based constructs can help to meet many of these challenges. Quantification can also make nosologically based constructs more amenable to powerful longitudinal research designs, genetic and other biological research, and research on secular trends in psychopathology. To continue advancing the developmental study of psychopathology, we outlined directions for further research.

REFERENCES

Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs*, *80* (No. 615). doi: 10.1037/h0093906

Achenbach, T. M. (1974). *Developmental psychopathology*. New York, NY: Ronald Press; 2nd ed. 1982. New York: Wiley.

Achenbach, T. M. (1991a). "Comorbidity" in child and adolescent psychiatry: Categorical and quantitative perspectives. *Journal of Child and Adolescent Psychopharmacology*, *1*, 271–278.

Achenbach, T. M. (1991b). *Integrative guide for the 1991 CBCL/4-18, YSR, and TRF profiles*. Burlington, VT: University of Vermont, Department of Psychiatry.

Achenbach, T. M. (2001). What are norms and why do we need valid ones? *Clinical Psychology: Science and Practice*, *8*, 446–450.

Achenbach, T. M. (2009). *The Achenbach System of Empirically Based Assessment (ASEBA): Development, findings, theory, and applications*. Burlington, VT: University of Vermont Research Center for Children, Youth, and Families.

Achenbach, T. M. (2014). *DSM-oriented guide for the Achenbach System of Empirically Based Assessment (ASEBA)*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Achenbach, T. M., Bernstein, A., & Dumenci, L. (2005). *DSM*-oriented scales and statistically based syndromes for ages 18 to 59: Linking taxonomic paradigms to facilitate multitaxonomic approaches. *Journal of Personality Assessment*, *84*, 47–61.

Achenbach, T. M., Conners, C. K., Quay, H. C., Verhulst, F. C., & Howell, C. T. (1989). Replication of empirically derived syndromes as a basis for taxonomy of child/adolescent psychopathology. *Journal of Abnormal Child Psychology*, *17*, 299–323. doi: 10.1007/ BF00917401

Achenbach, T. M., Dumenci, L., & Rescorla, L. A. (2001). *Ratings of relations between DSM-IV diagnostic categories and items of the CBCL/6-18, TRF, and YSR*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families. Available at www.ASEBA.org

Achenbach, T. M., Dumenci, L., & Rescorla, L. A. (2002). Ten-year comparisons of problems and competencies for national samples of youth: Self, parent, and teacher reports. *Journal of Emotional and Behavioral Disorders*, *10*, 194–203.

Achenbach, T. M., Dumenci, L., & Rescorla, L. A. (2003a). Are American children's problems still getting worse? A 23-year comparison. *Journal of Abnormal Child Psychology*, *31*, 1–11.

Achenbach, T. M., Dumenci, L., & Rescorla, L. A. (2003b). *DSM*-oriented and empirically based approaches to constructing scales from the same item pools. *Journal of Clinical Child and Adolescent Psychology*, *32*, 328–340. doi: 10.1207/S15374424JCCP3203_02

Achenbach, T. M., Howell, C. T., McConaughy, S. H., & Stanger, C. (1995a). Six-year predictors of problems in a national sample of children and youth: I. Cross-informant syndromes. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 336–347.

Achenbach, T. M., Howell, C. T., McConaughy, S. H., & Stanger, C. (1995b). Six-year predictors of problems in a national sample of children and youth: II. Signs of disturbance. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 488–498.

Achenbach, T. M., Howell, C. T., Quay, H. C., & Conners, C. K. (1991). National survey of problems and competencies among 4- to 16-year-olds: Parents' reports for normative and clinical samples. *Monographs of the Society for Research in Child Development*, *56*(3, Serial No. 225).

Achenbach, T. M., Ivanova, M. Y., & Rescorla, L. A. (2015). *Guide to multicultural family based assessment using the Achenbach System of Empirically Based Assessment (ASEBA)*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Achenbach, T. M., Krukowski, R. A., Dumenci, L., & Ivanova, M. Y. (2005). Assessment of adult psychopathology: Meta-analyses and implications of cross-informant correlations. *Psychological Bulletin*, *131*, 361–382. doi: 10.1037/0033-2909.131.3.361

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*, 213–232. doi: 10.1037/0033-2909.101.2.213

Achenbach, T. M., Newhouse, P. A., & Rescorla, L. A. (2004). *Manual for the ASEBA Older Adult Forms & Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.

Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA Preschool Forms & Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the ASEBA School-Age Forms & Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Achenbach, T. M., & Rescorla, L. A. (2003). *Manual for the ASEBA Adult Forms & Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Achenbach, T. M., & Rescorla, L. A. (2007a). *Multicultural supplement to the Manual for the ASEBA School-Age Forms & Profiles*. Burlington, VT: University of Vermont Research Center for Children, Youth, and Families.

Achenbach, T. M., & Rescorla, L. A. (2007b). *Multicultural understanding of child and adolescent psychopathology: Implications for mental health assessment*. New York: Guilford Press.

Achenbach, T. M., & Rescorla, L. A. (2010). *Multicultural supplement to the Manual for the ASEBA Preschool Forms & Profiles*. Burlington, VT: University of Vermont Research Center for Children, Youth, and Families.

Achenbach, T. M., & Rescorla, L. A. (2015). *Multicultural supplement to the Manual for the ASEBA Adult Forms & Profiles*. Burlington, VT: University of Vermont Research Center for Children, Youth, and Families.

Achenbach, T. M., Rescorla, L. A., & Ivanova, M. Y. (2012). International epidemiology of child and adolescent psychopathology: 1. Diagnoses, dimensions, and conceptual issues. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*, 1261–1272. doi: 10.1016/j.jaac.2012.09.010

Ambrosini, P. J. (2000). Historical development and present status of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS). *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 49–58. doi: 10.1097/0000 4583-20000 1000-00016

American Psychiatric Association. (1952; 1968; 1980; 1987; 1994; 2000; 2013). *Diagnostic and statistical manual of mental disorders* (1st ed., 2nd ed., 3rd ed., 3rd ed. rev., 4th ed., 4th ed. text revision., 5th ed.). Washington, DC: American Psychiatric Association.

Baltes, P. B. (1987). Theoretical propositions of life span developmental psychology: On the dynamics between growth and decline. *Developmental Psychology*, *23*, 611–626.

Baltes, P. B., Cornelius, S. W., & Nesselroade, J. R. (1979). Cohort effects in developmental psychology. In J. R. Nesselroade & P. B. Baltes (Eds.), *Longitudinal research in the study of behavior and development*. (pp. 61–87). New York, NY: Academic Press.

Bandura, A. (1977). *Social learning theory*. Englewood Cliffs, NJ: Prentice Hall.

Bartels, M., Boomsma, D. I., Hudziak, J. J., van Beijsterveldt, T. C. E. M., & van den Oord, E. J. C. G. (2007). Twins and the study of rater (dis)agreement. *Psychological Methods*, *12*, 451–466. doi: 10.1037/ 1082-989X.12.4.451

Bayley, N. (1968). Behavioral correlates of mental growth: Birth to thirty-six years. *American Psychologist*, *23*, 1–17.

Beck, A. T., & Steer, R. A. (1993). Manual for the Beck Depression Inventory. San Antonio, TX: Psychological Corporation.

Beitchman, J. H., Dielman, T. E., Landis, J. R., Benson, R. M., & Kemp, P. L. (1978). Reliability of the Group for the Advancement of Psychiatry diagnostic categories in child psychiatry. *Archives of General Psychiatry*, *35*, 1461–1466. doi: 10.1001/archpsyc.1978.0177036006 5008

Bell, R. Q. (1953). Convergence: An accelerated longitudinal approach. *Child Development*, *24*, 145–152.

Berkson, J. (1946). Limitations of the application of fourfold table analysis to hospital data. *Biometrics Bulletin*, *2*, 47–53. doi: 10.2307/3002000

Bollen, K. A. (1989). *Structural equations with latent variables*. New York, NY: Wiley.

Bongers, I. L., Koot, H. M., van der Ende, J., & Verhulst, F. C. (2004). Developmental trajectories of externalizing behaviors in childhood and adolescence. *Child Development*, *75*, 1523–1537. doi: 10.1111/ j.1467-8624.2004.00755.x

Boomsma, D. I., Willemsen, G., Dolan, C. V., Hawkley, L. C., & Cacioppo, J. T. (2005). Genetic and environmental contributions to loneliness in adults: The Netherlands twin register study. *Behavior Genetics*, *35*, 745–752. doi: 10.1007/s10519-005-6040-8

Boomsma, D. I., van Beijsterveldt, T., van Baal, C., Stroet, T., Polderman, T., Groot, A., … Hudziak, J. (July, 2001). *Genetic analysis of* DSM-IV *oriented scales in 3-year-old Dutch twins.* Presented at Behavior Genetics Association, Boulder, CO.

Breton, J. J., Bergeron, L., Valla, J. P., Lepine, S., Houde, L., & Gaudet, N. (1995). Do children aged 9 through 11 years understand the DISC Version 2.5 questions? *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 946–954.

Cantor, N., Smith, E. E., French, R. d. S., & Mezzich, J. (1980). Psychiatric diagnosis as prototype categorization. *Journal of Abnormal Psychology*, *89*, 181–193. doi: 10.1037/ 0021-843X.89.2.181

Caron, C., & Rutter, M. (1991). Comorbidity in child psychopathology: Concepts, issues and research strategies. *Journal of Child Psychology and Psychiatry*, *32*, 1063–1080. doi: 10.1111/j.1469-7610.1991 .tb00350.x

Cassidy, J., & Shaver, P. R. (Eds.). (2008). *Handbook of attachment: Theory, research, and clinical applications*. New York, NY: Guilford.

Chakrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *Journal of the American Medical Association*, *285*, 3093–3099. doi: 10.1001/ jama.285.24.3093

Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development*, *55*, 1–7. doi: 10.2307/1129830

Collishaw, S., Maughan, B., Goodman, R., & Pickles A. (2004). Time trends in adolescent mental health. *Journal of Child Psychology and Psychiatry*, *45*, 1350–1362. doi: 10.1111/ j.1469-7610.2004.00335.x

Conners, C. K. (1997). *Conners' Teacher Rating Scale-Revised*. North Tonawanda, NY: Multi-Health Systems.

Costello, A. J., Edelbrock, C., Dulcan, M. K., Kalas, R., & Klaric, S. H. (1984). *Report on the Diagnostic Interview Schedule for Children (DISC)*. Pittsburgh, PA: University of Pittsburgh, Department of Psychiatry.

De Los Reyes, A., & Kazdin, A. E. (2005). Informant discrepancies in the assessment of childhood psychopathology: A critical review, theoretical framework, and recommendations for further study. *Psychological Bulletin*, *131*, 483–509. doi: 10.1037/0033-2909.131.4.483

DuPaul, G. J., Power, T. J., Anastopoulos, A. D., & Reid, R. (1998). *ADHD Rating Scale-IV: Checklists, norms, and clinical interpretation*. New York, NY: Guilford.

Eaton, W. W., Neufeld, K., Chen, L.-S., & Cai, G. (2000). A comparison of self-report and clinical diagnostic interviews for depression. *Archives of General Psychiatry*, *57*, 217–222. doi: 10.1001/archpsyc.57.3.217

Education of the Handicapped Act. (1977). Public Law 94-142. Federal Register, 42, P. 42478. Amended in Federal Register (1981), 46, p. 3866.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview. *Archives of General Psychiatry*, *35*, 837–844. doi: 10.1001/archpsyc.1978 .01770310043002

Faraone, S. V., Biederman, J., Keenan, K., & Tsuang, M. T. (1991). Separation of *DSM-III* attention deficit disorder and conduct disorder: Evidence from a family-genetic study of American child psychiatric patients. *Psychological Medicine*, *21*, 109–121.

Finkel, D., Reynolds, C. A., McArdle, J. J., Gatz, M., & Pedersen, N. L. (2003). Latent growth curve analyses of accelerating decline in

cognitive abilities in late adulthood. *Developmental Psychology*, *39*, 535–550. doi: 10.1037/0012-1649.39.3.535

Freud, S. (1940). *An outline of psychoanalysis*. New York, NY: Norton.

Freud, S. (1953). Analysis of a phobia in a five-year-old boy (1909). In *Standard edition of the complete works of Sigmund Freud* (Vol. 7). London, UK: Hogarth Press.

Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M. A., … Hanson, K. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review*, *13*, 319–340.

Gabel, S., Stadler, J., Bjorn, J., Shindledecker, R., & Bowden, C. (1993). Dopamine-beta-hydroxylase in behaviorally disturbed youth. Relationship between teacher and parent ratings. *Biological Psychiatry*, *34*, 434–442.

Gadow, K. D., & Sprafkin, J. (1998). *Adolescent Symptom Inventory-4 Norms Manual*. Stony Brook, NY: Checkmate Plus.

Gadow, K. D., & Sprafkin, J. (1999). *Youth's Inventory-4 Manual*. Stony Brook, NY: Checkmate Plus.

Gadow, K. D., Sprafkin, J., Carlson, G. A., Schneider, J., Nolan, E. E., Mattison, R. E., & Rundberg-Rivera, V. (2002). A *DSM-IV* referenced, adolescent self-report rating scale. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 671–679.

Galambos, N. L., Barker, E. T., & Almeida, D. M. (2003). Parents do matter: Trajectories of change in externalizing and internalizing problems in early adolescence. *Child Development*, *74*, 578–594. doi: 10.1111/1467-8624.7402017

Goldstone, R. L., Lippa, Y., & Shiffrin, R. M. (2001). Altering object representations through category learning. *Cognition*, *78*, 27–43. doi: 10.1016/S0010-0277(00)00099-8

Gove, P. (Ed.). (1971). *Webster's third new international dictionary of the English language*. Springfield, MA: Merriam.

Group for the Advancement of Psychiatry. (1966). *Psychopathological disorders in childhood: Theoretical considerations and a proposed classification*. GAP Report No. 62. Washington, DC: Group for the Advancement of Psychiatry.

Guze, S. (1978). Validating criteria for psychiatric diagnosis: The Washington University approach. In M. S. Akiskal & W. L. Webb (Eds.), *Psychiatric diagnosis: Exploration of biological predictors* (pp. 49–59). New York, NY: Spectrum.

Hanna, G. L., Yuwiler, A., & Coates, J. K. (1995). Whole blood serotonin and disruptive behaviors in juvenile obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 28–35.

Helzer, J. E., Kraemer, H. C., Krueger, R. F., Wittchen, H.-U., Sirovatka, P. J., & Regier, D. A. (Eds.). (2008). *Dimensional approaches in diagnostic classification: Refining the research agenda for DSM-V*. Washington, DC: American Psychiatric Association.

Hewitt, L. E., & Jenkins, R. L. (1946). *Fundamental patterns of maladjustment: The dynamics of their origin*. Springfield, IL: State of Illinois.

Hoekstra, R. A., Bartels, M., Hudziak, J. J., Van Beijsterveldt, T. C. E. M., & Boomsma, D. I. (2008). Genetic and environmental influences on the stability of withdrawn behavior in children: A longitudinal, multi-informant twin study. *Behavior Genetics*, *38*, 447–461. doi: 10.1007/s10519-008-9213-4

Hudziak, J. J. (1998). *DSM-IV Checklist for Childhood Disorders*. Burlington, VT: University of Vermont, Department of Psychiatry.

Huesmann, L. R., Moise-Titus, J., Podolski, C.-L., & Eron, L. D. (2003). Longitudinal relations between children's exposure to TV violence and their aggressive and violent behavior in young adulthood: 1977–1992. *Developmental Psychology*, *39*, 201–221. doi: 10.1037/0012-1649.39.2.201

Individuals with Disabilities Education Act. (1990, 1997). Public Law 101-476. 20 U.S. C.1401 et seq. (Reauthorized July, 1997). Public Law 105–17. 20 U.S. C.1400 et seq.

Ivanova, M. Y., Achenbach, T. M., Dumenci, L., Rescorla, L. A., Almqvist, F., Weintraub, S., … Verhulst, F. C. (2007a). Testing the 8-syndrome structure of the CBCL in 30 societies. *Journal of Clinical Child and Adolescent Psychology*, *36*, 405–417. doi: 10.1080/15374410701444363

Ivanova, M. Y., Achenbach, T. M., Rescorla, L. A., Bilenberg, N., Bjarnadottir, G., Denner, S., … Verhulst, F. C. (2011). Syndromes of preschool psychopathology reported by teachers and caregivers in 14 societies using the Caregiver–Teacher Report Form (C-TRF). *Journal of Early Childhood and Infant Psychology*, *7*, 87–103.

Ivanova, M. Y., Achenbach, T. M., Rescorla, L. A., Dumenci, L. Almqvist, F., Bathiche, M., … Verhulst, F. C. (2007b). Testing the Teacher's Report Form syndromes in 20 societies. *School Psychology Review*, *36*, 468–483.

Ivanova, M. Y., Achenbach, T. M., Rescorla, L. A., Dumenci, L., Almqvist, F., Bilenberg, N., … Verhulst, F. C. (2007c). The generalizability of the Youth Self-Report syndrome structure in 23 societies. *Journal of Consulting and Clinical Psychology*, *75*, 729–738. doi: 10.1037/00 22-006X.75.5.729

Ivanova, M. Y., Achenbach, T. M., Rescorla, L. A., Harder, V. S., Ang, R. P., Bilenberg, N., … Verhulst, F. C. (2010). Preschool psychopathology reported by parents in 23 societies. Testing the seven-syndrome model of the Child Behavior Checklist for Ages 1.5-5. *Journal of the American Academy of Child and Adolescent Psychiatry 49*, 1215–1224. doi: 10.1016/j.jaac.2010.08. 019

Ivanova, M. Y., Achenbach, T. M., Rescorla, L. A., Turner, L. V., Ahmeti-Pronaj, A., Au, A., … Zasępa, E. (2015a). Syndromes of self-reported psychopathology for ages 18–59 in 29 societies. *Journal of Psychopathology and Behavioral Assessment*, *37*, 171–183. doi: 10.1007/s10862-014-9448-8

Ivanova, M. Y., Achenbach, T. M., Rescorla, L. A., Turner, L. V., Árnadóttir, H. A., Au, A., … Zasępa, E. (2015b). Syndromes of collateral-reported psychopathology for ages 18–59 in 18 societies. *International Journal of Clinical and Health Psychology*, *15*, 18–28. doi: 10.1016/j.ijchp.2014.07.001

Jensen, A. L., & Weisz, J. R. (2002). Assessing match and mismatch between practitioner-generated and standardized interview-generated diagnoses for clinic-referred children and adolescents. *Journal of Consulting and Clinical Psychology*, *70*, 158–168.

Jones, M. C. (1924). A laboratory study of fear: The case of Peter. *Pedagogical Seminary*, *31*, 308–315.

Jung, T., & Wickrama, K. A. S. (2008). An introduction to latent class growth analysis and growth mixture modeling. *Social and Personality Psychology Compass*, *2*/1, 300–317.

Kagan, J., & Moss, H. A. (1962). *Birth to maturity*. New York, NY: Wiley.

Kanner, L., Rodriquez, A., & Ashenden, B. (1972). How far can autistic children go in matters of social adaptation? *Journal of Autism and Childhood Schizophrenia*, *2*, 9–33. doi: 10.1007/BF01537624

Kazdin, A. E., & Weisz, J. R. (Eds.). (2003). *Evidence-based psychotherapies for children and adolescents*. New York, NY: Guilford.

Kovacs, M. (1992). *Children's Depression Inventory Manual*. North Tonawanda, NY: Multi-Health Systems.

Kraemer, H. C. (2012). Determining gene moderation of environmental risk factors for a mental disorder: A "perfect storm" of methodological problems. *International Journal of Methods in Psychiatric Research*, *21*, 185–194.

Kraepelin, E. (1883). *Compendium der psychiatrie*. Leipzig: Abel.

Krueger, R. F., & Piasecki, T. M. (2002). Toward a dimensional and psychometrically-informed approach to conceptualizing psychopathology. *Behaviour Research and Therapy*, *40*, 485–499. doi: 10.1016/S0005-7967(02)000165

Lahey, B. B., Loeber, R., Stouthamer-Loeber, M., Christ, M. A. G., Green, S., Russo, M.F., … Dulcan, M. (1990). Comparison of *DSM-III* and *DSM-III-R* diagnoses for prepubertal children: Changes in prevalence and validity. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 620–626.

Laird, N. M., & Ware, H. (1982). Random-effects models for longitudinal data. *Biometrics*, *38*, 963–974.

Lambert, M. J., Hansen, N. B., & Finch, A. E. (2001). Patient-focused research: Using patient outcome data to enhance treatment effects. *Journal of Consulting and Clinical Psychology*, *69*, 159–172.

Laurent, H. K., Leve, L. D., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Fisher, P. A., … Reiss, D. (2013). Effects of parental depressive symptoms on child adjustment moderated by hypothalamic pituitary adrenal activity: With and between-family risk. *Child Development*, *84*, 528–542.

Lejeune, J., Gautier, M., & Turpin, R. (1963). Study of the somatic chromosomes of nine mongoloid idiot children (1959). In S. H. Boyer (Ed.), *Papers on human genetics* (pp. 238–240). Englewood Cliffs, NJ: Prentice-Hall.

Levin, D. A. (1979). The nature of plant species. *Science*, *204*, 381–384.

Lewczyk, C. M., Garland, A. F., Hurlburt, M. S., Gearity, J., & Hough, R. L. (2003). Comparing DISC-IV and clinician diagnoses among youths receiving public mental health services. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*, 349–356.

Lewine, R. R. J., Watt, N. F., Prentky, R. A., & Fryer, J. H. (1980). Childhood social competence in functionally disordered psychiatric patients and in normals. *Journal of Abnormal Psychology*, *89*, 132–138.

Lilienfeld, S. O., Waldman, I. D., & Israel, A. C. (1994). A critical examination of the use of the term and concept of comorbidity in psychopathology research. *Clinical Psychology: Science and Practice*, *1*, 71–83.

Lleras, C. (2005). Path analysis. *Encyclopedia of Social Measurement*, *3*, 25–30.

Males, M. (1991). Teen suicide and changing cause-of-death certification, 1953–1987. *Suicide and Life Threatening Behavior*, *21*, 245–259.

March, J. (1997). *MASC: Multidimensional Anxiety Scale for Children*. North Tonawanda, NY: Multi-Health Systems.

Markon, K. E., Chmielewski, M., & Miller, C. J. (2011). The reliability and validity of discrete and continuous measures of psychopathology: A quantitative review. *Psychological Bulletin*, *137*, 856–879. doi: 10.1037/a0023678

Master, A., Markman, E. M., & Dweck, C. S. (2012). Thinking in categories or along a continuum: Consequences for children's social judgments. *Child Development*, *83*, 1145–1163. doi: 10.1111/j.1467-8624.2012.01774.x

McClellan, J. M., & Werry, J. S. (Eds.). (2000). Research psychiatric diagnostic interviews for children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 19–27.

McConaughy, S. H., & Achenbach, T. M. (1994). Comorbidity of empirically based syndromes in matched general population and clinical samples. *Journal of Child Psychology and Psychiatry*, *35*, 1141–1157. doi: 10.1111/j.1469-7610.1994.tb01814.x

Meyer, G. J. (2002). Implications of information gathering methods for a refined taxonomy of psychopathology. In L. E. Beutler & M. L. Malik (Eds). *Rethinking the DSM* (pp. 69–105). Washington, D.C.: American Psychological Association.

Meyer, G. J., Finn, S. E., Eyde, L. D., Kay, G. G., Moreland, K. L., Dies, R. R., … Reed, G. M. (2001). Psychological testing and psychological assessment. *American Psychologist*, *56*, 128–165.

Moffitt, T. E. (1993). Adolescence-limited and life-course persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, *100*, 674–701. doi: 10.1037/0033-295X.100.4.674

Nagin, D. S. (1999). Analyzing developmental trajectories: A semiparametric group-based approach. *Psychological Methods*, *4*, 139–157.

Narrow, W. E., Clarke, D. E., Kuramoto, S. J., Kraemer, H. C., Kupfer, D. J., Greiner, L., & Regier, D. A. (2013). *DSM-5* field trials in the United States and Canada, part III: Development and reliability testing of a cross-cutting symptom assessment for *DSM-5*. *American Journal of Psychiatry*, *170*, 71–82. doi: 10.1176/appi.ajp.2012.12071000

Nesselroade, J. R., & Baltes, P. B. (1974). Adolescent personality development and historical change: 1970–1972. *Monographs of the Society for Research in Child Development*, *39* (*Serial No. 154*). doi: 10.2307/1165824

Newman, D. L., Moffitt, T. E., Caspi, A., Magdol, L., Silva, P., & Stanton, W. R. (1996). Psychiatric disorder in a birth cohort of young adults: Prevalence, comorbidity, clinical significance, and new case incidence from ages 11 to 21. *Journal of Consulting and Clinical Psychology*, *64*, 552–562.

Ormel, J., Oldehinkel, A. J., Sijtsema, J. J., van Oort, F., Raven, D., Veenstra, R., … Verhulst, F. C. (2012). The TRacking Adolescents' Individual Lives Survey (TRAILS). Design, current status and selected findings. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*, 1020–1036. doi: 10.1016/ j.jaac.2012.08.004

Overall, J. E., & Hollister, L. E. (1979). Comparative evaluation of research diagnostic criteria for schizophrenia. *Archives of General Psychiatry*, *36*, 1198–1205. doi: 10.1001/archpsyc. 1979.01780110052006

Piaget, J. (1983). Piaget's theory. In P. H. Mussen (Ed.), *Handbook of child psychology* (4th ed.), *Vol. 1. History, theory, and methods*. W. Kessen (Vol. Ed.). New York, NY: Wiley.

Puig-Antich, J., & Chambers, W. (1978). *The schedule for affective disorders and schizophrenia for school-aged children (Kiddie-SADS)*. New York, NY: New York State Psychiatric Institute.

Quay, H. C. (1964). Personality dimensions in delinquent males as inferred from the factor analysis of behavior ratings. *Journal of Research in Crime and Delinquency*, *1*, 33–37.

Quay, H. C. (1993). The psychobiology of undersocialized aggressive conduct disorder: A theoretical perspective. *Development and Psychopathology*, *5*, 165–180.

Queller, S., Schell, T., & Mason, W. (2006). A novel view of between-categories contrast and within-category assimilation. *Journal of Personality and Social Psychology*, *91*, 406–422.

Raftery, A. E. (1995). Bayesian model selection in social research. *Sociological Methodology*, *25*, 111–164. doi: 10.2307/271063

Rapoport, J. L., & Ismond, D. R. (1996). *DSM-IV training guide for diagnosis of childhood disorders*. New York: Brunner/Mazel.

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data-analysis methods*. Newbury Park, CA: Sage.

Reef, J., Diamantopoulou, S., van Meurs, I., Verhulst, F., & van der Ende, J. (2010). Predicting adult emotional and behavioral problems from externalizing problem trajectories in a 24-year longitudinal study. *Journal of European Child and Adolescent Psychiatry*, *19*, 577–585.

Rescorla, L. A., Achenbach, T. M., Ivanova, M. Y., Bilenberg, N., Bjarnadottir, G., Denner, S., … Verhulst, F. C. (2012a). Behavioral/emotional problems of preschoolers: Caregiver/teacher reports from 15 societies. *Journal of Emotional and Behavioral Disorders*, *20*, 68–81. doi: 10. 1177 /1063426611434158

Rescorla, L.A. Achenbach, T.M., Ivanova, M.Y., Turner, L. V., Althoff, R.R., Arnadottir, H., … Zasepa, E. (2015a). Collateral reports of problems and cross-informant agreement about adult psychopathology in 14 societies. Manuscript submitted for publication.

Rescorla, L. A., Achenbach, T. M., Ivanova, M. Y., Turner, L. V. Althoff, R.R., Arnadottir, H.,… Zasepa, E (2015b). Problems and adaptive functioning reported by adults in 17 societies. Manuscript submitted for publication.

Rescorla, L. A., Bochicchio, L., Achenbach, T. M., Ivanova, M. Y., Almqvist, F., Begovac, I., … Verhulst, F. C. (2014). Parent–teacher agreement on children's problems in 21 societies. *Journal of Clinical Child and Adolescent Psychology*, *43*, 627–642. doi: 10.1080/15374416.2014.900719

Rescorla, L. A., Ginzburg, S., Achenbach, T. M., & Ivanova, M. Y., Almqvist, F., Begovac, I., … Verhulst, F. C. (2013). Cross-informant agreement between parent-reported and adolescent self-reported problems in 25 societies. *Journal of Clinical Child & Adolescent Psychology*, *42*, 262–273. doi: 10.1080/15374416.2012.717870

Rescorla, L. A., Ivanova, M. Y., Achenbach, T. M., Begovac, I., Chahed, M., Drugli, M. B., … Zhang, E. Y. (2012b). International epidemiology of child and adolescent psychopathology: 2. Integration and applications of dimensional findings from 44 societies. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*, 1273–1283. doi: 10.1016/j.jaac.2012.09.012

Rettew, D. C., Doyle, A., Achenbach, T. M., Dumenci, L., & Ivanova, M. (2009). Meta-analyses of agreement between diagnoses made from clinical evaluations and standardized diagnostic interviews. *International Journal of Methods in Psychiatric Research*, *18*, 169–184. doi: 10.1002/mpr.289

Reynolds, C. R., & Kamphaus, R. W. (1992). *Behavior Assessment System for Children Teacher Rating Scales*. Circle Pines, MN: American Guidance Service.

Rietveld, M. J. H., Hudziak, J. J., Bartels, M., Van Beijsterveldt, C. E. M., & Boomsma, D. I. (2004). Heritability of attention problems in children: Longitudinal results from a study of twins, age 3 to 12. *Journal of Child Psychology and Psychiatry*, *45*, 577–588.

Risch, N., Herrell, R., Lehner, T., Liang, K. Y., Eaves, L., Hoh, J.,… Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *Journal of the American Medical Association*, *301*, 2462–2471.

Roberts, C., Kane, R., Thomson, H., Bishop, B., & Hart, B. (2003). The prevention of depressive symptoms in rural school children: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *71*, 622–628. doi: 10.1037/0022-006X.71.3.622

Roberts, R. E., Attkisson, C., & Rosenblatt, A. (1998). Prevalence of psychopathology among children and adolescents. *American Journal of Psychiatry*, *55*,715–725.

Roberts, R. E., Solovitz, B. L., Chen, Y.-W., & Casat, C. (1996). Retest stability of *DSM-III-R* diagnoses among adolescents using the Diagnostic Interview Schedule for Children (DISC-2.1C). *Journal of Abnormal Child Psychology*, *24*, 349–362. doi: 10.1007/ BF01441635

Robins, L. N. (1966). *Deviant children grown up*. Baltimore: Williams and Wilkens.

Robins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, *38*, 381–389.

Rogosa, D., Brandt, D., & Zimowski, M. (1982). A growth curve approach to the measurement of change. *Psychological Bulletin*, *92*, 726–748. doi: 10.1037/0033-2909.92.3.726

Rosch, E. (1978). Principles of categorization. In E. Rosch & B. B. Lloyd (Eds.), *Cognition and categorization*. Hillsdale, NJ: Erlbaum.

Rosch, E., & Mervis, C. B. (1975). Family resemblances: Studies in the internal structure of categories. *Cognitive Psychology, 7,* 573–605. doi:10.1016/0010-0285(75)90024-9

Rounsaville, B. J., Alarcon, R. D., Andrews, G., Jackson, J. S., Kendell, R. E., & Kendler, K. (2002). Basic nomenclature issues for *DSM*-V. In D. J. Kupfer, M. B. First, & D. E. Regier (Eds.), *A research agenda for DSM-V* (pp. 1–29). Washington, DC: American Psychiatric Association.

Rutter, M. (2006). *Genes and behavior: Nature–nurture interplay explained*. Malden, MA: Blackwell Publishing.

Rutter, M., & Garmezy, N. (1983). Developmental psychopathology. In P. Mussen (Ed.), *Handbook of child psychology* (4th ed.) (Vol. 4: pp. 775–911). New York, NY: Wiley.

Rutter, M., & Smith, D. J. (Eds.). (1995). *Psychosocial disorders in young people: Time trends and their causes*. Chichester, UK: Wiley.

Scerbo, A. S., & Kolko, D. (1994). Salivary testosterone and cortisol in disruptive children: Relationship to aggressive, hyperactive, and internalizing behaviors. *Journal of the American Academy of Child and Adolescent Psychiatry*, *33*, 1174–1184.

Shaffer, D. (1992). *Diagnostic Interview Schedule for Children, Version 2.3*. New York, NY: Columbia University Division of Child Psychiatry.

Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 28–38. doi: 10.1097/00004583-200001000-00014

Sheldrick, R. C., Kendall, P. C., & Heimberg, R. G. (2001). The clinical significance of treatments: A comparison of three treatments for conduct disordered children. *Clinical Psychology: Science and Practice*, *8*, 418–430. doi: 10.1093/clipsy.8.4.418

Smith, E. E., & Medin, D. L. (1981). *Categories and concepts*. Cambridge, MA: Harvard University Press.

Spitzer, R. L., Davies, M., & Barkley, R. A. (1990). The *DSM-III-R* field trial of disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 690–697.

Stanger, C., Achenbach, T. M., & Verhulst, F. C. (1997). Accelerated longitudinal comparison of aggressive versus delinquent syndromes. *Development and Psychopathology*, *9*, 43–58. doi: 10.1017/S0954579497001053

Tick, N. T., van der Ende, J., Koot, H. M., & Verhulst, F. C. (2007a). 14-year changes in emotional and behavioral problems of very young Dutch children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*, 1333–1340.

Tick, N. T., van der Ende, J., & Verhulst, F. C. (2007b). Twenty-year trends in emotional and behavioral problems in Dutch children in a changing society. *Acta Psychiatrica Scandinavica*, *116*, 473–482.

Tiemeier, H., Velders, F. P., Szekely, E., Roza, S. J., Dieleman, G., Jaddoe, V. W. V., … Verhulst, F. C. (2012). The Generation R Study: A review of design, findings to date, and a study of the 5-HTTLPR by environmental interaction from fetal life onward. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*, 1119–1135 e7. doi: 10.1016/j.jaac. 2012.08.021

Van Beijsterveldt, C. E. M., Bartels, M., Hudziak, J. J., & Boomsma, D. I. (2003). Causes of stability of aggression from early childhood to adolescence: A longitudinal genetic analysis in Dutch twins. *Behavior Genetics*, *33*, 591–605.

van Oort, F. V., van der Ende, J., Wadsworth, M. E., Verhulst, F. C., Achenbach, T. M. (2011). Cross-national comparison of the link

between socioeconomic status and emotional and behavioral problems in youths. *Social Psychiatry and Psychiatric Epidemiology*, *46*(2), 167–172. doi: 10.1007/s00127-010-0191-5

van't Hoff, M. A., Roede, M. J., & Kowalski, C. (1991). A mixed longitudinal data analysis model. *Human Biology*, *49*, 165–179.

Verhulst, F. C., & van der Ende, J. (1992a). Six-year developmental course of internalizing and externalizing problem behaviors. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 924–931. doi: 10.1097/00004583-199209000-00022

Verhulst, F. C., & van der Ende, J. (1992b). Six-year stability of parent-reported problem behavior in an epidemiological sample. *Journal of Abnormal Child Psychology*, *20*, 595–610. doi: 10.1007/BF00911243

Verhulst, F. C., van der Ende, J. R. A., & Rietbergen, A. (1997). Ten-year time trends of psychopathology in Dutch children and adolescents: No evidence for strong trends. *Acta Psychiatrica Scandinavica*, *96*, 7–13. doi: 10.1111/j.1600-0447.1997.tb09898.x

Walker, J. L., Lahey, B. B., Russo, M. F., Frick, P. J., Christ, M. A. G., McBurnett, K., . . . Green, S. M. (1991). Anxiety, inhibition, and conduct disorder in children: I. Relations to social impairment. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*, 187–191. doi: 10.1097/00004583-199103000-00004

Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, *3*, 1–14. doi: 10.1037/h0069608

Weinstein, S. R., Noam, G. G., Grimes, K., Stone, K., & Schwab-Stone, M. (1990). Convergence of *DSM-III* diagnoses and self-reported symptoms in child and adolescent inpatients. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 627–634. doi: 10.1097/00004583-199007000-00018

Wilens, T. E., Biederman, J., Brown, S., Tanguay, S., Monuteaux, M. C., Blake, C., & Spencer, T. J. (2002). Psychiatric comorbidity and functioning in clinically referred preschool children and school-age youths with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 262–268. doi: 10.1097/00004583-200203000-00005

Wolman, B. B. (1970). *Children without childhood*. New York: Grune & Stratton.

World Health Organization. (1978; 1992). *Mental disorders: Glossary and guide to their classification in accordance with the Tenth Revision of the International Classification of Diseases* (9th ed.; 10th ed.). Geneva, Switzerland: World Health Organization.

Zachar, P. (2000). Psychiatric disorders are not natural kinds. *Philosophy, Psychiatry, Psychology*, *7*, 167–182.

CHAPTER 3

Developmental Epidemiology

E. JANE COSTELLO and ADRIAN ANGOLD

WHAT IS EPIDEMIOLOGY? 94
Scientific Epidemiology 95
Public Health Epidemiology 96
CHARACTERISTICS OF RESEARCH IN PSYCHIATRIC EPIDEMIOLOGY 96
Measuring the Frequency of Disease 96
Defining a Case 97
Taxonomy, Instrumentation, and Mechanisms 97
Using Question-and-Answer Methods to Identify Cases 98
Classifying Cases in the Clinic and the Community 98
Developmental Issues in Case Identification 99
Ascertainment Bias in Case Identification 99
Controlling Bias in Estimating the Influence of Risk Factors 100
Summary 102
WHAT IS DEVELOPMENTAL EPIDEMIOLOGY? 102
The Concept of Development 102
Implications of a Developmental Approach to Child Psychiatric Epidemiology 103
FROM CHILD PSYCHIATRIC EPIDEMIOLOGY TO DEVELOPMENTAL EPIDEMIOLOGY: A BRIEF HISTORY 106
The Origins of Child Psychiatric Epidemiology 106
Distinguishing Psychiatric Disorder from Severe Mental Retardation 106
Distinguishing Among Psychiatric Disorders 106
Psychoanalytic Theory and Developmental Psychopathology 107
Distinguishing Normal from Abnormal 108
Measuring Child and Adolescent Psychopathology 108
Summary 111
EPIDEMIOLOGY AS A DEVELOPMENTAL METHOD 111
Risk, Exposure, and the Meaning of Time 111
Examples from Developmental Psychopathology 112
FUTURE DIRECTIONS: DEVELOPMENTAL EPIDEMIOLOGY 113
Longitudinal Research 114
Impact Studies 117
Mechanisms: Genetic Epidemiology 119
SUMMARY: TRANSLATIONAL EPIDEMIOLOGY 121
REFERENCES 121

WHAT IS EPIDEMIOLOGY?

Epidemiology is the study of patterns of disease in human populations (Kleinbaum, Kupper, & Morgenstern, 1982). Patterns, or nonrandom distributions, of disease occur in both time and space. The task of epidemiology is to understand observed patterns of pathology along these two dimensions and to use this understanding as a basis for prevention and treatment. For example, a pattern has been observed linking exposure to environmental lead in infancy with the development of behavioral and learning problems in later years (Needleman & Bellinger, 1991). The causal relationship appears to be sufficiently strong to justify primary preventive measures to remove lead from gas and domestic paint to protect those children who might otherwise be vulnerable to such exposures (Kleinbaum et al., 1982).

Epidemiology, "an exact and basic science of social medicine and public health" (Earls, 1979, p. 256), emerged as an influential branch of medicine in the nineteenth century. Like the rest of medicine, it is an action-oriented discipline, whose main purpose is intervention to control disease, but which is also concerned with understanding causes. Epidemiology has both similarities to and differences from clinical medicine. Scientific knowledge about the cause and course of disease is one of the tools that both share. Epidemiology reflects clinical medicine also in using two methods of attack: a tactical method, concerned with the practical and administrative problems of disease control at the day-to-day level; and a strategic method, concerned with finding out what causes disease so that new weapons of control and cure can be engineered (Earls, 1980; Susser, 1973). Thus, epidemiologists can be found, in their tactical or public health role, reporting on the

prevalence of AIDS and advising on how to control its spread while simultaneously, as part of a wide strategic plan to control the disease, carrying out multicenter clinical research trials of new vaccines and treatments.

Epidemiology is also like clinical medicine in its understanding of the concept of disease in broad terms. In this chapter, we adopt the definition of *disease* as a pathological process that may have biological, psychological, and social dimensions (Kleinbaum et al., 1982; Susser, 1973). Disease in this sense includes identified syndromes of known etiology. The unifying theme is that, at some point, a decision is made that a pathological process is present in an individual, who is then regarded as a *case*. The thorny problems of case identification in child and adolescent psychopathology are discussed later in this chapter.

Epidemiology diverges from clinical medicine to the extent that it concentrates on understanding and controlling disease processes in the context of the population at risk, whereas the primary focus of clinical medicine is the individual patient. This does not mean that epidemiology is not concerned with the individual; on the contrary, it is very much concerned with understanding the individual's illness and the causes of that illness. The difference lies in the frame of reference. Put at its simplest, clinical medicine asks, What is wrong with this person, and how should I treat him or her? Epidemiology asks, What is wrong with this person, and what is it about him or her that has resulted in this illness? Why is this child depressed, but not her brother? If her mother is also depressed, is the child's depression a cause, a consequence, or an unrelated, chance co-occurrence? Such questions immediately set the individual child within a frame of reference of other children, or other family members, or other people of the same sex or race or social class. As we shall discuss later, this approach to understanding pathology has important effects on the way information is collected, the kind of information collected, the methods used to analyze it, and the conclusions that can be drawn from it.

As the study of patterns of disease distribution in time and space, epidemiology thus encompasses a great deal more than simply counting how many people have smallpox or AIDS. Epidemiology counts (Freedman, 1984), but it does far more than that (Insel & Fenton, 2005). The methods developed to count cases are useful for many purposes; for example, to estimate the need for and probable cost of mental health services or to monitor the effect of a new treatment. However, these methods provide only one part of the information needed to understand the course, causes, and prevention of disease. Figure 3.1 (Kleinbaum et al., 1982) shows how the study of disease patterns in time and space informs both public health and scientific epidemiology (Susser, 1973).

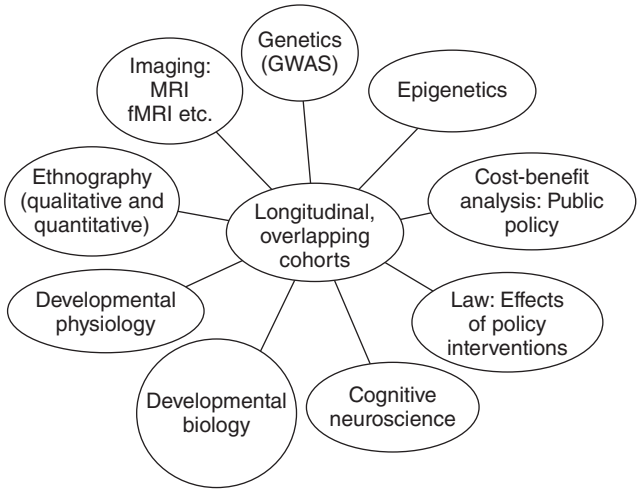


Figure 3.1 Contributions of developmental epidemiology to translational science. *Source:* Kleinbaum et al. (1982).

Scientific Epidemiology

From the strategic or scientific point of view, the issues at each level have to do with identifying the stages by which a disease progresses through its natural course, generating and testing hypotheses about causal pathways, and inventing and testing methods for prevention to be implemented by the tactical, public health arm of the health care professions. The success or failure of attempts to prevent a disorder can then provide important information about the causes of the problem. The course of disease can be divided into three stages: (1) exposure, dealing with causes; (2) disorder, dealing with the manifestation of a disease in clinical signs and symptoms; and (3) outcome, examining what leads to different outcomes for different patients. These three stages invite different kinds of preventive intervention: (1) primary, or universal, prevention tries to reduce the risk of exposure to agents promoting the disease; (2) secondary, or targeted, prevention is directed at saving those who have been exposed from developing the disease; and (3) tertiary, or indicated, prevention focuses on reducing the damage caused when people actually become ill. Tertiary prevention encompasses both reducing the residual damage from an illness and, where necessary, protecting the rest of society from harm, either through infection or from a sick person's dangerous behavior.

Although this approach to disease prevention was first articulated at a time when acute, infectious diseases such as cholera and smallpox were the chief preoccupation of epidemiology, it applies with minor modifications to chronic or episodic diseases, such as heart disease and cancer, which are the main causes of mortality in industrialized countries today. Applied to psychopathology, it also provides a model for exploring the development of psychiatric disorders (Rutter, 1990).

Public Health Epidemiology

At the tactical level, public health and primary care workers are often involved in what has been called *shoe-leather epidemiology* (Kleinbaum et al., 1982). This might take the form of tracing the pathways by which a cluster of suicides spreads through a high school or isolating the source of a neurotoxic element in the water supply, or teaching lifestyle changes to cardiac patients to reduce the risk of stress-related disease. The aim is to identify key points in the development and transmission of a disease at which intervention can act to reduce the prevalence of the disease or harm caused by it. Such interventions may be directed at preventing exposure to the causes of the disorder, at preventing the onset of the disorder in those who are vulnerable to it, or at minimizing death rates or residual impairment in those who survive. Taking depression as an example, primary prevention might include antibullying programs in schools; secondary prevention might be directed at the children of depressed parents, who are known to be a high-risk group (Clark et al., 2002); and tertiary or indicated prevention might examine the ability of adequate treatment to prevent relapse.

There is a great deal of interchange between the public health aspects and the scientific aspects of epidemiology, and also between different levels of investigative and preventive effort. At each of these levels, both strategic and tactical efforts are needed. Indeed, the real-world nature of epidemiology means that the answers to many scientific questions, such as the paths of disease transmission, have at some stage to be tested by regarding a preventive intervention as a scientific experiment. The classic example is the story of how John Snow (1855), by having the handle removed from the Broad Street water pump during the 1853 cholera epidemic in London, tested his hypothesis that (1) cholera was caused by an invisible, self-reproducing agent living in water; (2) the agent flourished in water heavily contaminated with sewage; and (3) therefore, if a community drawing its water from a contaminated supply was forced (by his action in disabling the pump) to use water from a different water company, drawn from a purer source, the rate of disease would fall.

CHARACTERISTICS OF RESEARCH IN PSYCHIATRIC EPIDEMIOLOGY

Epidemiology, as this brief introduction demonstrates, is less a body of substantive knowledge than it is a way of looking at problems. The great historical victories over infectious diseases and the recent achievements in helping to reduce mortality and morbidity from chronic illnesses such as cardiovascular disease (Dawber, 1980) came as a result of looking at clinical problems in a new way. Here we introduce the basic requirements for studying the epidemiology of any disorder (Kleinbaum et al., 1982) and discuss their implications for developmental psychopathology.

Epidemiologic research presupposes the ability to (1) measure disease frequency in the population of interest; (2) make valid generalizations from samples to populations, that is, to avoid sampling or ascertainment bias; and (3) measure the impact of risk factors on disease without confusion among multiple competing risks, that is, to avoid confounding among risk factors. We discuss each of these challenges in turn.

Measuring the Frequency of Disease

The question of how many people exposed to a risk factor for a disease actually become sick, how many avoid disease, and how many die or recover is central to epidemiological research. The most frequently used measures involve the concept of a rate, "an instantaneous potential for change in one quantity per unit change in another quantity" (Kleinbaum et al., 1982, p. 100). Timing is implicit in the idea of a rate. Epidemiology deals most often with incidence rates, which measure the extent to which new cases of a disorder appear in the previously healthy population over a specified period of time, and prevalence rates, which measure all the cases, whether new or previously existing, observed during a specified period. Usually, the epidemiologist is concerned with relative rates of disease onset in two or more groups of people who differ on exposure to a risk or preventive factor. For example, in a Swedish study, the prevalence of drunkenness in adolescence among girls who reached menarche earlier than the norm for the population was found to be high, relative to the rate found in girls of the same age whose menarche occurred within the normal age range (Stattin & Magnusson, 1990). Relative rates thus become the metric for answering questions about a

possible causal role played by putative risk factors in the onset of disorder. In this example, Stattin and Magnusson found that, at age 14, 75% of girls who had reached menarche before age 11 had been drunk at least once, compared with only 29% of girls who reached menarche after age 13. Furthermore, the rate of frequent drunkenness remained higher across adolescence for early than for later maturing girls. This led to speculations about the mechanism by which early puberty could put girls at risk (Moffitt, Caspi, Belsky, & Silva, 1992).

Defining a Case

In any context where decisions have to be made leading to action to prevent or treat illness, categories have to be created. Making a diagnosis means putting an individual into a category: "Has Disorder X" or "Does not have Disorder X." Even if the goal is to eliminate a risk factor that potentially affects everyone in the community, such as environmental pollutants or unsafe automobiles, it is still necessary to define categories of affected and nonaffected individuals to measure the effectiveness of a primary prevention or to calculate costs and benefits. For example, how many cases of learning disability were prevented by reducing environmental lead? How much does it cost to prevent one case of learning disability, and how much is saved by doing so?

The relationship among symptoms, diagnosis, and the concept of disease has exercised many brains (Meehl, 1992; Pickles & Angold, 2003). A diagnosis encapsulates a mixed bag of information: the number and severity of symptoms, the duration of symptomatology, the date of onset, the likelihood of exposure to a causal factor, and the level of functional impairment. All of these have implications for a decision about whether intervention is indicated. For some forms of pathology, there is little ambiguity about how the diagnosis should be made; either it is fairly self-evident, like a broken bone, or the rules have been sanctified by use, like the stages of cervical cancer, or there is a clearly identifiable pathogen that acts as a marker for the disease, as in tuberculosis. Few types of psychopathology can invoke any one of these as a decision rule for diagnosis. There are no reliable pathognomic markers; the boundaries between one disorder and another, or between normality and disorder, are generally ill defined (Pickles & Angold, 2003), as Kendell (1976) demonstrated in his classic exploration of the boundaries of depression. Classification systems arise and pass away, while older classifications may still retain some of their sway. To confuse matters further, psychiatry often makes use of familiar language in its diagnostic terminology (e.g., *depression, anxiety*), not necessarily in the vernacular meaning of the term. In his review of childhood depression, for example, Angold (1988) identified eight different ways the term depression is used in the description of psychopathology:

- As the low end of normal mood fluctuations
- As a description of psychic pain felt in response to some unpleasant situation
- As a trait
- As an individual symptom
- As a syndrome
- As a disorder
- As a disease
- As a cause of handicap or disability

The implications for treatment are likely to differ depending on which definition is being used.

Taxonomy, Instrumentation, and Mechanisms

In identifying cases of a disorder, it is important to distinguish between the problems that can be solved by a well-designed diagnostic instrument and those that have to be solved at the level of the taxonomic system that defines what a case is (Meehl, 1992; Wing, Bebbington, & Robins, 1981). Robins (1989), for example, using both clinical and epidemiological methods, presented evidence that some *DSM* disorders are consistently diagnosed more validly (using several criteria of validity) than others. This, Robins believed, suggests that "part of the source of invalidity lies in the diagnostic grammar of the systems whose criteria standardized interviews evaluate" (p. 57). She used the phrase *diagnostic grammar* because "[diagnosis] is much like a language. The criteria include elements that have special relations to each other like parts of speech—symptoms (nouns?); severity (adjectives?); clustering (verbs?); age of onset, frequency, and duration (adverbs?)" (p. 61). If the grammar is wrong, the sentence will not make sense, however clearly it is articulated. Much of the process of developing a good diagnostic grammar is carried out through careful clinical work. Epidemiology, however, can contribute in several ways. It provides feedback about the relationship between symptom patterns seen in clinical and community samples, which can throw light on possible biases affecting the results (see later discussion). It can also provide information about the prevalence of different disorders in the community, and their patterns of distribution by age, sex, and so on. This information can then influence clinicians' expectations about the relative likelihood that a given patient has one disorder rather than another.

In practice, it is well known that physicians make diagnostic judgments on the basis of very few items of information (Cantwell & Baker, 1988; Meehl, 1954). Good prevalence information can be a very important part of the clinician's database.

Psychiatric epidemiologists have long been active collaborators with clinicians in the work carried out around the world to improve psychiatric taxonomies (Robins, 1989). However, the task facing psychiatric epidemiology is even more difficult than that facing clinical psychiatry. The clinician can assume a certain level of suffering in a patient who has come seeking help; the problem is to figure out exactly where the suffering lies. Epidemiologists, working most of the time in the community, have to identify cases among people most of whom are not seeking treatment. They have to convince themselves and others that the criteria they use are valid: that a clinician would identify this as a case of a given disorder whether or not the individual came seeking treatment. They also have to distinguish between the normal range of feelings and psychopathology; between, for example, what Freud described as *hysterical misery* and *common unhappiness* (Breuer & Freud, 1895).

For child psychiatry, this problem of case definition is even more complex. Children rarely refer themselves for treatment, even when severely symptomatic. Thus, the rule of thumb that equates illness with seeking treatment is even less accurate for children than it is for adults. Referral for treatment may have more to do with characteristics of a child's mother, teacher, or pediatrician than with the child's own behavior or feelings (Costello & Janiszewski, 1990; Dulcan et al., 1990; Shepherd, Oppenheim, & Mitchell, 1971).

A third aspect of case identification is the discovery of mechanisms that underlie the development of psychiatric disorders. These mechanisms exist at many different levels, from genes to brain structure and functioning. Despite much exciting progress in identifying mechanisms (discussed later), none is yet ready for use in everyday clinical or epidemiologic diagnosis. Child psychiatry is still largely dependent for case identification on inferences from responses to verbal questions.

Using Question-and-Answer Methods to Identify Cases

Psychiatry confronts two problems that make it particularly difficult to develop reliable and accurate measures of symptoms and diagnoses using question-and-answer methods. One is that no gold standard measures can be used to evaluate the accuracy of a test (Angold et al., 2012): a postmortem or magnetic resonance image (MRI) will not tell us whether a child really has separation anxiety disorder. The second problem is with the standard procedure for assessing the reliability of a question-and-answer measure. This is known as the test–retest reliability procedure, which uses two interviewers to interview the same subject on two different occasions about the same period of time. Whereas one can take someone's temperature repeatedly without appreciably altering body temperature, interviewing someone at length about emotionally sensitive topics can hardly be done without affecting how the individual thinks or feels about these issues. The first interview is thus likely to affect how the subject responds to the second interview. For example, in a test–retest study in which children were interviewed twice, we found that boys who, at the first interview, admitted that they told a lot of lies, were much more likely than other boys to deny, at the second interview, other kinds of deviant behavior to which they had previously admitted (Angold & Costello, 1995). However, a lot is now known about how to improve accuracy in child psychiatric interviewing, and current methods are remarkably reliable (Shaffer, Lucas, & Richters, 1999).

Classifying Cases in the Clinic and the Community

The problem of equating cases found in community studies with referred cases has been addressed in several ways in child psychiatric epidemiology. First, diagnostic instruments have been developed that meticulously translate clinical diagnostic criteria into methods for identifying the same symptoms in nonreferred cases (Angold & Costello, 2000; Chambers et al., 1985; Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982; Herjanic & Campbell, 1977; Hodges, McKnew, Cytryn, Stern, & Kline, 1982; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000).

Second, most measures for community use now include, or are used with, measures of functional impairment so that the relationship between symptoms and a child's ability to carry out the normal tasks of daily life can become the subject of empirical review (Canino, Costello, & Angold, 1999). At the same time, current attempts to classify psychiatric disorders into a coherent taxonomy have adopted a multiaxial system by which a clinician is encouraged to describe the patient from several points of view, of which psychiatric diagnosis is only one (American Psychiatric Association, 1994, 2013; Rutter et al., 1969, 1979). Several current epidemiological assessment instruments adopt the same multiaxial approach (Angold & Costello, 2000).

Third, the psychometric studies that are carried out as part of the process of instrument development include

studies of referred and nonreferred samples and interviews using both clinicians and trained lay interviewers. Instruments can then be revised so that caseness has the same meaning wherever or by whomever a child is recruited or assessed.

Developmental Issues in Case Identification

One reason why establishing rates of disorder is even more of a problem for child psychiatric epidemiology is that there is a need to reconcile a nondevelopmental psychiatric taxonomy, such as the *DSM* system, with the realities of child development. The *Diagnostic and Statistical Manual, 5th Edition* (*DSM-5*) includes a section on the development and course of each diagnosis that replaces the previous edition's sections on course. However, *DSM-5* uses *development* to refer to the developmental course of the illness, not of the individual. It sticks closely to an implicit medical model according to which a disease, although it has a developmental course along the lines sketched earlier, is defined as a disease by virtue of the fact that every case has roughly the same etiology, pathogenesis, risk factors, presentation, course, and treatment response. Measles is measles is measles. For example, the diagnostic criteria for *DSM-5* major depressive episode are described as being similar in their essential features in children, adolescents, and adults (American Psychiatric Association, 2013); the only differences specified are additions to two of the symptoms: (1) depressed mood. "In children and adolescents, can be irritable mood" (p. 160) and (3) significant weight loss or weight gain. "In children, consider failure to make expected weight gain" (p. 161). Apart from the statement that "the likelihood of onset increases markedly in puberty" (p. 165), no reference is made to any aspect of children's cognitive, social, and bodily development that might influence a child's ability to experience or respond to different symptoms. Neither the similarities nor the differences specified by the *DSM* have a firm basis in developmental data about the manifestation of depression at different stages of life (Angold & Worthman, 1993; Cicchetti & Schneider-Rosen, 1984, 1986; Costello et al., 2002; Digdon & Gotlib, 1985; Ladouceur, Peper, Crone, & Dahl, 2012).

Since 2009, the National Institute of Mental Health has been working on a new way to classify mental illnesses by linking the various layers of systems that contribute to mental function. Called the Research Domain Criteria (RDoC), the goal is to cut across traditional diagnostic categories to identify relationships between behavior and neurobiology. It is too early yet for epidemiologic studies to incorporate RDoC measures, but this is likely to be an important pathway for the future.

In summary, epidemiology shares with clinical psychiatry a concern for and involvement in the problems of case identification. These problems occur at two levels: the level of defining the characteristics of a disorder and the level of developing methods for identifying those who have the disorder so defined. Arguments about the reliability and validity of psychiatric assessment have frequently confused these two levels, as Robins (1985) so clearly demonstrated. Only when we can plainly describe the causal pathway for each disorder, from exposure through onset to outcome, as in Figure 3.1, will we be in a position to be satisfied with the validity of our taxonomy and the reliability of our diagnostic tools.

Ascertainment Bias in Case Identification

Once researchers are able accurately to identify cases of the disorder of interest, the next concern is to ensure that the sample of cases that they recruit for research purposes accurately represents the distribution of cases in the community. This is important because the relationship between the disorder and its putative risk factors needs to be the same in the sample as in the general population. It is the nature of epidemiological research that questions of causality can rarely be answered by laboratory studies in which all but the key variable are carefully controlled or randomly varied. It is not feasible to test the causal role of poverty, for example, by randomly assigning newborns to be raised in high- and low-income households, or the role of maternal temperament by the sort of cross fostering studies that are considered ethical with other primates (Suomi, 1991). One way to compensate is to pay very careful attention to the characteristics of the population from which the study samples are drawn; that is, not only to the numerator of a rate of disease (the number of cases within a given period) but also to the denominator of the rate (the population at risk). Only when we know that the sample selected for study accurately mirrors the population from which it is drawn (or when we know exactly what the biases are and how to control for them) can we specify exactly to whom our causal analyses apply.

This necessity usually rules out samples of convenience and mandates very careful thought about the choice of population for a study. For example, if the question has to do with the causal role played by the peer group in adolescent delinquency, and the sample consists of young people recruited from tenth-grade classrooms, neither the denominator nor the numerator can, by definition, include

persons who have dropped out of school. Thus, the conclusions about causal processes that can be drawn from such a study are limited to those relevant to tenth graders who attend school, not to the age group in general. Because dropping out of school and delinquency are known to be associated, the selection of a school-based sampling frames results in a sample that may be biased in important ways.

The problem of ascertainment bias is even more acute when clinic-based samples are used. There are many reasons people get into treatment, and reasons they do not; these may mislead the researcher trying to understand risk factors for disease. For example, Berkson (1946) used Mayo Clinic records to show that, among people with two illnesses, "each disease is itself aggravated in its symptoms and more likely to be noted by the patient … [the] effect would be to increase relatively the representation of multiple diagnoses in the hospital and in general to increase the discrepancy between hospital and parent population" (p. 50). This led to a spurious belief that cholecystic disease was causally related to diabetes, when in fact, in the general population the association between the two diseases was zero.

Berkson (1946) argued from these analyses that it is more cost-efficient to carry out representative community studies earlier rather than later in the process of studying comorbidity and etiology because bias-free studies have to be done at some point in any case. Meehl (1992) also pointed out that millions of dollars of tax money have been wasted performing studies in clinical settings where the biases inherent in case selection mean that the studies cannot answer the questions about causal association that they are designed to address.

The danger of ascertainment bias is one reason why many epidemiological studies use samples that are carefully selected to be representative of the general population. The disease of interest may be quite rare in the community, which often means that the case-identification stage of such studies is expensive; large numbers of people must be surveyed to identify a relatively small number of cases. With child psychiatric disorders, the issue is compounded because reports from different informants—parents, children, teachers, peers—may identify very different children as cases (Achenbach, McConaughy, & Howell, 1987; Foley et al., 2004, 2005). Depending on the question being studied, it may be necessary to interview three or more people to establish whether a child is or is not a case according to the study criteria. The risk factors for a disorder identified by one informant may also be quite different from the risk factors for the disorder as identified by another informant. For example, in a study of children with symptoms of hyperactivity, inattention, and impulsivity (Costello, Loeber, & Stouthamer-Loeber, 1991), 61% of children identified as hyperactive by both parents and teachers were boys; however, boys made up 77% of children identified as hyperactive by teachers alone but only 38% of children identified by mothers alone.

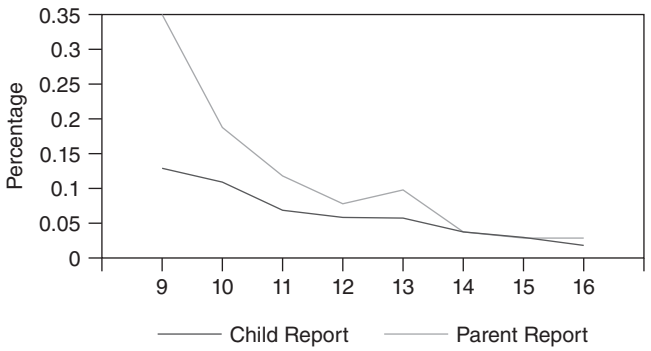


Figure 3.2 Mean separation anxiety symptoms by age and source of information.

For the researcher interested in developmental influences on the expression of psychopathology, the situation becomes even more complex. For example, Figure 3.2 shows the rate of separation anxiety symptoms reported by children between the ages of 7 and 16 and by parents about their children. The parents' report data showed low rates of symptoms and a modest effect of the child's age on the rate of symptoms. The rate of symptoms reported by the children, in contrast, fell sevenfold as the age of the child increased. If the purpose of the study had been to select a sample of cases of separation anxiety disorder, the bias caused by relying exclusively on one informant or the other would have varied depending on the age of the children surveyed.

Controlling Bias in Estimating the Influence of Risk Factors

The third set of threats to the validity of a causal link has to do with competing risk factors or confounders. In most etiological studies, more than one risk factor is involved. The problem is to figure out the relative importance of various risk factors so that decisions about intervention can be focused where they will be most effective. It is useful to distinguish between two distinct characteristics of factors that influence the probability of disease: confounding and effect modification (Miettinen, 1974).

Confounding distorts the impact of a risk factor on the risk of disease because of the presence of some extraneous variable. A factor may act as a confounder in one study but not in another. Consider, for example, two groups

at high and low levels of genetic risk for a disease that is also affected by poverty. If everyone in one group was at high genetic risk, a real relationship between poverty and disease might be obscured in that community; almost everyone who was poor would also be at genetic risk, so it would not be possible to say which factor was causing the disorder. In the second community, where not all the poor were at high genetic risk and not all the group at high genetic risk were poor, it would be possible to look separately at the rates of disorder in four groups (poor nonaffected, rich nonaffected, poor affected, rich affected) and figure the risk associated with poverty.

Effect modification, or synergy, refers to the different impact of a risk factor at different levels of another variable (Rothman, 1976). This relationship is not specific to any particular study; it is a real relationship among two or more risk factors. For example, if both the gene for phenylketonuria (PKU) and a diet high in phenylalanine are necessary for PKU to occur, rates of the disorder will vary in different communities depending on how many people inherit the gene and how many eat a high-phenylalanine diet. The relationship between gene and diet remains constant across sites, but diet will act as an effect modifier, controlling the expression of the gene. Another example is the relationship among peak height velocity (PHV, the growth spurt of early adolescence), change of school, and depressive symptoms. The period of PHV may be a time when youngsters are particularly vulnerable to symptoms of depression (Simmons & Blyth, 1987), particularly when they have to deal with stressful events. It happens that, in the American school system, most children move from middle school to high school between eighth and ninth grades. This coincides with the time when many girls, but few boys, are at PHV. School change could thus be acting as an effect modifier, increasing the risk of depression in girls but not in boys.

Clinical as well as epidemiologic research has to deal with confounders and effect modifiers. Lively debates are in progress about whether and how to derive causal inferences from observational studies with many potential confounders (Kaufman & Cooper, 1999; Pearl, 2013; Robins, 2000). Sometimes it is possible to narrow down the list of potential causes using careful study design. When standard experimental designs are inappropriate or infeasible, quasi-experimental methods may be found that reduce the risk that cases and noncases differ not only on the putative risk factor but also in lots of other ways (Shadish, Cook, & Campbell, 2002). For example, in our study of all 9-, 11- and 13-year-old American Indian children living on a federal reservation, it happened that after we had assessed the children for four years, a casino was opened that gave every enrolled tribal member a sizable income supplement. After another four years, we looked at children's psychiatric symptoms before and after the intervention. We found that children whose families moved out of poverty had reduced conduct disorder symptoms but that the intervention had little effect on depression, anxiety, or attention-deficit/hyperactivity disorder (ADHD; Costello, Compton, Keeler, & Angold, 2003). Because every man, woman, and child on the reservation received the income supplement, there was no possibility that the effect of an increased income could be confounded with the sort of qualities (e.g., hard work, thrift) that could cause both an income increase and a reduction in behavioral symptoms.

The other set of methods to deal with competing risk factors is statistical. Epidemiologists have always been careful to make it clear that nonexperimental studies can show associational but not causal relationships between risk factors and outcomes. They are, however, coming closer to claiming the ability to talk about *causal associations* (Robins, 1997, 2000). Some have gone so far as to claim that properly designed observational studies can produce results that are superior to those based on experimental designs because they often have a level of ecological validity that experimental studies lack (Benson & Hartz, 2000; Hernán, Hernández-Díaz, & Robins, 2013).

The idea that some effect modifiers could operate as protective factors has been widely discussed (Garmezy, 1988; Loeber, Farrington, Stouthamer-Loeber, Moffitt, & Caspi, 1998; Stouthamer-Loeber, 1993). As Rutter (1985) pointed out, there are logical problems in identifying something as a protective factor, except in the presence of a risk or vulnerability; the concept of protection implies the presence of risk. However, in terms of Figure 3.1, it is reasonable to talk about protection from risk factors for a disease.

Measuring the Impact of Risk and Protective Factors

One implication of this discussion of risk factors is that it is possible to rank risk (and protective) factors in terms of their relative importance to the rate of an observed disorder, and thus to make decisions about the most cost-efficient preventive strategies. There are several ways to calculate the importance of a factor's contribution to the prevalence of a disease. Two of these are risk ratio or relative risk and excess or attributable risk. Relative risk is a ratio comparing two risks of disease, often, the risk of a disease in a group exposed to a risk factor compared with the risk in a nonexposed group (e.g., the risk of depression in children of divorced parents compared with the risk to children in intact families). Attributable risk is

an estimate of the extra cases of disease (above the rate in the nonexposed population) attributable to exposure to a particular risk factor. Attributable risk is a function of two factors: the relative risk in groups exposed and not exposed to the risk factor and the frequency of the risk factor in the population. Thus, even if the relative risk associated with a risk factor is high, the attributable risk, and thus the decrease in incidence of the disease if that risk factor were removed, may be low if the proportion of people in the population exposed to that risk factor is low. For example, there might be a high relative risk for lung cancer associated with exposure to a chemical used only in one particular industry, but the attributable risk would be low if only a few people worked in that industry and were exposed to that chemical. From a public health viewpoint, a greater reduction in the number of cases of lung cancer might be achieved by removing a risk factor with a lower relative risk but to which more people are exposed; for example, asbestos or cigarette smoke (Lilienfeld & Lilienfeld, 1980; Rothman, 1976). Measures of relative risk are particularly important for the scientific aspect of epidemiology because they are a way of estimating the causal impact of risk factors. Measures of attributable risk are of particular interest from the practical point of view because they can generate an estimate of the impact of a risk or protective factor in real or proportional terms.

This discussion of causality begs an underlying question: Does it make any sense to think about human behavioral development in terms of continuity and causes? Some developmental psychologists have argued that prediction is not only difficult but also is unnecessary for the study of psychopathology (Lewis, 1990). Epidemiology has not adopted this approach; on the contrary, it has put its money on its ability to control disease through the prediction of causal pathways, and historically this has been an immensely successful bet. The interesting question is whether it is an approach that will prove to be as effective in the area of developmental psychopathology as it has been for infectious diseases.

Summary

In this section, we discussed three functions of epidemiology: (1) to measure disease frequency and risk exposure; (2) to make valid generalizations from samples to populations; and (3) to control bias in relating risk factors to disease. The next question is whether, and how, the methods and ideas that have evolved over the past century, primarily to tackle infectious diseases, can be adapted to the moving target that is developmental psychopathology.

WHAT IS DEVELOPMENTAL EPIDEMIOLOGY?

Epidemiology is inherently a developmental discipline, as shown in Figure 3.1, in the sense that it is concerned with the development of disease and how to intervene in that process. However, we talk about developmental epidemiology as a special variety of epidemiology when we are dealing with child psychopathology. The reason is that in this case, we have to take account of another developmental model: the model that describes the development of the individual. From this point of view, developmental epidemiology can be seen as concerned with the interaction between two developmental processes: of the organism (the child) and of the disease. The importance of understanding this interaction was pointed out by Anna Freud (1965; see also Cicchetti, 1990a; Rutter, 1988), but her view of what develops was somewhat different from the view of both developmental psychology and developmental psychopathology today. In this chapter, our use of the concept of development, which follows that of developmental psychology and biology, will be familiar to developmentalists but may carry different implications for child psychiatrists and pediatricians trained to use the term development either in the context of psychodynamic theories of personality (Dare, 1985) or in reference to specific, mainly physical or cognitive, stages of development (American Psychiatric Association, 1994). For clarification, we offer a brief review of what we understand by development.

The Concept of Development

Earlier in this chapter, we discussed epidemiology as a scientific method of understanding the development of disease (Figure 3.1). This approach shows marked similarities with the way the word development is applied to biological organisms (Hay & Angold, 1993). As Nagel (1957, p. 15) defined it:

> The concept of development involves two essential components: the notion of a system possessing a definite structure and a definite set of preexisting capacities; and the notion of a sequential set of changes in the system, yielding relatively permanent, but novel increments not only in its structure but in its modes of operation.

The process of disease progression has much in common with development: It is programmed by the nature of the transformation of the organism that begins the process, and in general it follows a reasonably regular course, although with wide variations in its rate. Furthermore, there is hierarchical integration as diseases develop. Each stage in the

progress of a given disease builds on the previous stages, and many of the manifestations of earlier stages are integrated into later symptomatology.

This has much in common with the idea of *epigenesis,* as developed by Gottlieb (1991a). Individual development is characterized by an increase of complexity of organization (i.e., the emergence of new structural and functional properties and competencies) at all levels of analysis (molecular, subcellular, cellular, and organismic) as a consequence of horizontal and vertical coactions among the organisms' parts, including organism–environment coactions. Horizontal coactions are those that occur at the same level (gene–gene, organism–organism), whereas vertical coactions occur at different levels (cell–tissue, behavioral activity–nervous system) and are reciprocal, meaning that they can influence each other in either direction, from lower to higher or from higher to lower levels of the developing system.

For example, the genome controls cell structure, but environmentally induced cytoplasmic changes are capable of switching genes on and off (Ho, 1984; Jollos, 1934); neural differentiation in the mammalian visual cortex is dramatically affected by sensory experience (Black & Greenough, 1986); and neuroendocrine mechanisms are powerfully influenced by sociocultural factors (Mineka, Gunnar, & Champoux, 1986; Worthman, 1987). Recent longitudinal epidemiologic studies showed that a functional polymorphism in the gene encoding the neurotransmitter-metabolizing enzyme monoamine oxidase A (MAOA) moderated the effect of early maltreatment so that maltreated children, if they owned the allele conferring high levels of MAOA expression, were less likely to develop antisocial problems in adolescence (Caspi et al., 2002; Kim-Cohen et al., 2006). Thus, the concept of development used here (1) presupposes change and novelty, (2) underscores the importance of timing in behavioral establishment and organization, (3) emphasizes multiple determination, and (4) leads us not to expect invariant relationships between causes and outcomes across the span of development (Cacioppo & Tassinary, 1991; Cairns, 1991).

Behavior is seen as resulting from the dynamic interaction of multiple interdependent systems in both the individual and the environment. As summarized by Eisenberg (1977), "The process of development is the crucial link between genetic determinants and environmental variables, between individual psychology and sociology." It is characteristic of such systems that they consist of feedback and feed-forward loops of varying complexity. Organism and environment are mutually constraining, however, with the result that developmental pathways show relatively high levels of canalization (Angoff, 1988; Cairns, Gariépy, & Hood, 1990; Gottlieb, 1991b; Greenough, 1991; McGue, 1989; Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). For example, consider the well-established path to substance abuse (Kandel & Davies, 1982):

$$\text{Beer or wine} \rightarrow \text{Cigarettes or hard liquor} \rightarrow \text{Marijuana} \rightarrow \text{Other illicit drugs}$$

It is characteristic of this pathway that the number of individuals at each level becomes smaller but that those at the higher levels continue to show behaviors characteristic of the earlier stages. Having described such a pathway, the task is to understand the process by which it is established and to experiment with preventive strategies appropriate to the various stages of the developmental pathway. Such strategies must be appropriate to the developmental stage of both the individual at risk and the disorder.

Research based on this view of development has been going on for several decades, as these volumes attest, but has taken some time to influence child psychiatric epidemiology (Angold & Costello, 1991; Bale et al., 2010). Conversely, until recently developmental psychopathology has paid less attention to some of the key concerns of epidemiology—in particular, the importance of representative samples and controls for confounding. But this is beginning to change.

Implications of a Developmental Approach to Child Psychiatric Epidemiology

Child psychiatry, in its current manifestation in the United States, has modeled its nosology and its concepts of health and disease on adult psychiatry, using the same nomenclature and static assumptions about causality, risk factors, course, and outcome. But children are not scaled-down adults, and this approach causes serious problems for the epidemiologist concerned with explaining patterns of disease distribution in the community. Why, for example, do rates of depressive disorders, which are low in prepubertal children of both sexes, continue low in postpubertal boys but rise very sharply in postpubertal girls (Angold, 2003; Angold, Costello, Erkanli, & Worthman, 1999)? Is the increase causally associated with age, or with pubertal status, or with something else entirely? To understand these phenomena, it seems reasonable to postulate that children are organisms developing at a different rate from adults, organisms in whom developmental changes dramatically alter both the ways they can manifest their

genetic endowment and the nature of their interactions with their surroundings. Here we consider the implications of some basic principles of developmental theory for the epidemiology of child psychiatric disorders.

Implication 1: Development Implies Change

This may seem to be an obvious statement, but child psychiatric epidemiology has not always taken the idea of change very seriously. This is partly the result of the current systems for the classification of diseases, which are essentially nondevelopmental. Most disorders have definitions that take no account of age or developmental level, sometimes in spite of overwhelming evidence that age-dependent changes are a central feature of the phenomenology of the disorders involved. Conduct disorder is a good example. Loeber and colleagues (Loeber & Homish, 2005; Loeber, Keenan, & Zhang, 1997) identified three different developmental pathways involving antisocial behavior. If a cross sectional, snapshot view was taken of behavior at any point in time, children who pursued any of these three pathways shared many characteristics. Longitudinal studies, however, revealed different patterns. Children who followed the authority conflict path tended to show conduct problems early in life, to have trouble at school because of hyperactivity and learning difficulties as well as conduct problems, to explore a wide range of different deviant behaviors, and to remain deviant well into adulthood.

Some of the same behaviors could be found in other children at any given time, but a second group of these followed a different, covert pathway, with a higher remission rate. These children tended to have started showing deviant behavior later in childhood than the first group and to have had fewer problems at school or with peers. Longitudinal studies also suggested the existence of a third group who, like the first, were aggressively deviant in adolescence but who showed little or no early conduct disorder. The evidence for these different developmental patterns has been pieced together from a multitude of different studies, using both clinical and population samples, and is much stronger for boys than for girls (Loeber & Baicker-McKee, 1990). The *DSM,* in contrast, ignores strong evidence that conduct disorder in children is a precursor of antisocial personality disorder in adults (Robins & Price, 1991; Robins & Wish, 1977), to the extent of coding conduct disorder on Axis I (Clinical Disorders) and adult antisocial personality disorder on Axis II (Personality Disorders and Mental Retardation).

Questions about development and change can be addressed using methods developed to identify developmental trajectories (Land & Nagin, 1996; Nagin & Tremblay, 2001). These methods have been used, for example, to show that there are important differences between children who show persistent, rather than time-limited, conduct problems (Nagin & Tremblay, 2001) or obesity (Mustillo et al., 2003).

Implication 2: Development Is Goal Directed

If we accept what Ernst Mayr (1982) called the biological metaphor or heuristic of development, then we accept a teleonomic, goal-directed explanation as part of that metaphor. In Mayr's words:

> A physiological process or a behavior that owes its goal-directedness to the operation of a program can be designated as teleonomic. All the processes of individual development (ontogeny) as well as all seemingly goal-directed behaviors of individuals fall in this category, and are characterized by two components: they are guided by a program, and they depend on the existence of some endpoint or goal that is foreseen in the program regulating the behavior. Each particular program is the result of natural selection and is constantly adjusted by the selective value of the achieved endpoint. (Mayr, 1982, p. 48)

Teleonomic activities are distinguished by Mayr from other types of teleological processes—most important, for our purposes, from those performed by adapted systems (e.g., the cardiovascular or respiratory systems) that owe their adaptedness to a past selectionist process (Hay & Angold, 1993).

A teleonomic approach has particular advantages for developmental epidemiology, given its concern with disease elimination and service provision. It enables us to conceive of disease states in terms of inability to achieve one or more of the goals of development, and it points preventive efforts in the direction of those situations in which a particular process or task seems to be particularly salient at a certain phase of life. For example, Sroufe and Rutter (1984, p. 22) outlined "a series of developmental issues . . . based on the collective experience of numerous developmentalists . . . cutting across affective, cognitive, and social domains":

> Age 0–1: Biological regulations; harmonious dyadic interaction; formation of an effective attachment relationship.
> Age 1–2.5: Exploration, experimentation, and mastery of the object world (caregiver as secure base); individuation and autonomy; responding to external control impulses.
> Age 3–5: Flexible self-control; self-reliance; initiative; identification and gender concept; establishing effective peer contacts (empathy).
> Age 6–12: Social understanding (equity, fairness); gender constancy; same-sex chumships; sense of "industry" (competence); school adjustment.

Age 13+: "Formal operations" (flexible perspective taking; "as if" thinking); loyal friendships (same sex); beginning heterosexual relationships; emancipation; identity.

Sroufe and Rutter's (1984) list has two major implications for epidemiology. First, it implies that if we want to prevent, for example, conduct disorder, we need to define the goal of orderly conduct and the various systems (affect, cognition, impulse, and muscular control) that are most involved with regulating conduct at different developmental stages. This means that we need research to address such questions as the following: Do children who have achieved the goal of forming secure attachments around 1 year of age find behavioral control at age 5 easier than insecurely attached children (Greenberg, Speltz, & DeKlyen, 1993; Lyons-Ruth, 1996)? Intervention designs can be used to test a hierarchical, causal relationship among the various stages (see Cicchetti, 1990b; Cicchetti & Toth, 1992 for a more detailed discussion of this point).

A second, linked implication is that pathology that looks very different at different stages may be causally linked; for example, the *anxious attachment* of a 1-year-old may be causally linked to the emotional lability and superficial friendliness of the same child at age 5 and to an inability to form lasting intimate relationships as an adult. If this were the case, then, from a public health point of view, efforts to support secure attachments in infants would be justified, not just by the manifest anxiety of some 1-year-olds but also by the chance of preventing the social disruption caused by those same children as adolescents and adults.

Implication 3: The Goal of Development Is Normality

Normality is a state not requiring intervention. We would argue the value for developmental epidemiological research of taking the position that the goal of development is normality. That is, among the multiple outcomes of any developmental process there exists a wide range that are not likely to cause a child any serious problems in moving to the next developmental phase or task; any of these outcomes should be defined as normal. Normality and pathology in an epidemiological context can thus be defined in terms of decision making: Is some sort of intervention indicated?

In this respect, epidemiology moves along a different track from much of developmental psychopathology. The latter has tended to adopt the viewpoint of developmental psychology, that many phenomena of interest are best measured on some sort of continuous scale, representing a hypothesized underlying distribution of symptoms or capacities in the population. This makes sense for disciplines whose aim is to map out the pathways by which children's thoughts, feelings, and actions change over time under different internal and external controls (Pickles & Angold, 2003). The specific concern of epidemiology, however, functioning as it does at the interface between scientific understanding and public health, is with those patterns of thought, feeling, and action about which something must be done at a given stage for the sake of the child's present or future well-being or in response to pressures from families, schools, or society in general. Thus, we define a developmental abnormality from an epidemiological point of view as a state in which intervention is indicated, and normality as a state in which intervention is not indicated. These definitions do not necessarily require that an intervention be available or effective, only that pathology has a social meaning as well as a medical and a developmental one, and that a corollary of defining a syndrome as pathological is that one would intervene if it were feasible. In this, public health and clinical medicine are alike.

This view of normality and abnormality requires us to take into account not only the symptom or behavior, but also the developmental stage at which it occurs, in deciding whether to devote major efforts to prevention. Taking an example from externalizing behaviors, Loeber & Le Blanc (1990) pulled together evidence from many studies to argue that behavior that is not highly predictive of later conduct disorder in most children at a certain stage may be highly predictive in a specific child if (1) it first occurs earlier than in normal children; (2) it occurs with greater frequency than in normal children; and (3) it forms part of a more diversified symptom cluster. These developmental abnormalities frequently go together (Cohen, 1990; Farrington, 1983; Tolan, 1987). This implies that, for example, boys younger than 12 who commit minor acts of theft, vandalism, and substance abuse should be treated as showing a serious problem for which intervention is needed, whereas boys who first commit such acts after age 12 should not be taken so seriously. This is based on evidence that children who begin such acts early are likely to show a much higher rate of delinquency in their late teens than those who begin later (Fréchette & Le Blanc, 1987; Tolan, 1987) and to continue showing delinquent behavior for a longer period of time (Le Blanc & Fréchette, 1989).

Thus, the concept of goal-directed change toward developmentally appropriate, normal behavior provides a framework within which to study patterns of pathology in time and space and links the study of human development to the study of disease under the heading of developmental epidemiology.

FROM CHILD PSYCHIATRIC EPIDEMIOLOGY TO DEVELOPMENTAL EPIDEMIOLOGY: A BRIEF HISTORY

In this section, we trace the history of developmental epidemiology from the earliest days of child psychiatry to the present.

The Origins of Child Psychiatric Epidemiology

Work on the epidemiology of child and adolescent psychiatric disorders was first undertaken in the late nineteenth century to answer the most pressing question that faced the emerging discipline of child psychiatry: how to classify and care for children with severe disabilities, many of whom were likely to be a public expense for much of their lives. In this context, the main concerns were to identify the nature of the deficits shown by severely impaired children and to find out how many such children there were.

In the nineteenth century, the disease concept of psychiatric illness replaced the earlier view of psychiatric illness as *moral insanity* (Prichard, 1837). Mental disease was regarded as a thing residing within the affected individual; cases could therefore be counted in the same way as in the epidemiology of physical disease. At the same time, reforms in the administration of psychiatric services (e.g., the assumption by the state of responsibility for mental hospitals) increased the need for statistics and paved the way for epidemiological studies (Jablensky, 1986).

There appear to have been few children in mental hospitals or asylums in the nineteenth century (von Gontard, 1988), but an analogous problem arose here, too: how to identify children who could never be expected to become self-sufficient adults and for whom long-term care, probably at public expense, would be needed (Grob, 1985).

Distinguishing Psychiatric Disorder from Severe Mental Retardation

An important and basic distinction made by the end of the nineteenth century was one between *imbeciles* and *lunatics*. As universal education spread across Europe and the United States in the second half of the century, children who could not handle the demands of the educational system became a visible and troubling group. In their historical overview of child psychiatry, Chess and Habibi (1978) pointed out that the distinction between, and division of responsibility for, idiots and lunatics was far from clear until the twentieth century; in 1876, all the charter members of the American Association on Mental Deficiency were psychiatrists, and the child guidance movement in the United States began at the University of Pennsylvania in a clinic set up by Winner in 1894 primarily to care for the feebleminded. Gradually, over the course of the nineteenth century, specific groups of children were described, and causes for their disabilities were identified. J. L. Down (1867), a psychiatrist and superintendent of a large asylum for idiots, described Down syndrome, and J. C. P. Williams, superintendent of the Scottish National Institution for Imbecile Children, developed a classification into such categories as epileptic, microcephalic, and inflammatory idiocy, demonstrating that the same phenomenon of idiocy could result from many different causes (Ireland, 1877). Mental retardation slowly became recognized as a problem to be treated separately from behavioral and emotional problems. Children with severe developmental deficits began to be seen as lying on a continuum of disability with children who were struggling to survive the new compulsory education system, rather than as simply part of the general class of severely dysfunctional children (although, of course, some children might have both intellectual and psychiatric problems). For example, special classes for the feebleminded were mandated in England by the Elementary Education Act (Defective and Epileptic Children) of 1899. Throughout the industrialized world, as the right of all children to education was acknowledged, the care of the mentally retarded largely moved out of the sphere of psychiatry, unless emotional or behavioral problems were also severe. Causal theories focused on genetics, perinatal insults, and early environmental adversity; treatment centered on pinpointing precise deficits and maximizing children's potential rather than curing them as the pioneers in the treatment of idiocy had hoped to do (von Gontard, 1988).

Distinguishing Among Psychiatric Disorders

From the same period, descriptions can be found of children whose cognitive development was normal but who showed serious emotional or behavioral problems. In 1835, the physician James Prichard (1786–1848) wrote that idiotism and imbecility are observed in childhood, but "insanity, properly so termed, is rare before the age of puberty" (1837, p. 127). Following Pinel, the French psychiatrist who first described *mania without delirium* (Pinel, 1806, p. 150), Prichard distinguished *moral insanity* from, on one hand, "mania, or raving madness … in which the mind is totally deranged" (p. 16)—and which he

attributed to physical causes such as convulsions—and, on the other hand, imbecility or mental retardation. He thus used *moral* in its eighteenth-century sense of pertaining to personality or character. Henry Maudsley (1868), writing 30 years later, used the term in its nineteenth-century sense, referring to ethics and norms. He distinguished between instinctive insanity, which was "an aberration and exaggeration of instincts and passions," and moral insanity, which was a defect of the moral qualities along a dimension of "viciousness to those extreme manifestations which pass far beyond what anyone would call wickedness" (p. 289). In the process, he broadened the realm of child psychiatry to include problems of conduct previously seen to be the responsibility of religion and the law. Maudsley preferred to use the term *affective* (where Prichard used moral) as being "a more general term and expressing more truly the fundamental condition of nerve-element, which shows itself in affections of the mode of feeling generally, not of the special mode of moral feeling only" (p. 280).

When writing about etiology rather than classification, however, Prichard and Maudsley followed the French tradition in distinguishing between moral and physical causes of mental disorder, using moral in the sense of what came to be called exogenous causes. Prichard (1837), quoting Georget, listed among moral causes of insanity domestic grief, disappointment in love, political events, fanaticism, jealousy, poverty or reversal of fortune, reading romances, and excessive study. Mental retardation was seen as stemming exclusively from physical causes: either convulsions of some type in the early years, or some defect transmitted from the parent. This defect might itself be inherited, or it might be "traceable to parental intemperance and excess" (p. 44; see Maudsley, 1879).

The dominant causal theory of psychopathology in the second half of the nineteenth century was genetic: heredity and degeneration caused disease, which started with scarcely perceptible signs in early childhood but took a progressive and irreversible course and would probably be transmitted to future generations if the affected individual were permitted to breed. Even when the proximal cause of insanity was a moral one, "the different forms of insanity that occur in young children . . . are almost always traceable to nervous disease in the preceding generation" (Maudsley, 1868, p. 245).

Psychiatrists in the nineteenth century thus had a developmental causal theory about psychopathology, but it was a narrow form of developmental theory. The development of the disease was progressive and irreversible, tied to the development of the child only in that it manifested itself differently as the child grew, but impervious to other influences, such as treatment or learning. "All one could do was to prevent the most extreme manifestations by strict punishment and to protect those not affected" (von Gontard, 1988). The most effective defense for society was to prevent the procreation of the insane, and eugenics and lifelong segregation in asylums were seen as more effective intervention strategies than attempting treatment or cure. Although a continuum of severity was documented in child psychiatric disorders, as it was in mental retardation, the continuum was interpreted quite differently. It took the form of a continuum of degeneration caused by the disease within the individual across time, rather than a distribution of severity that would remain fairly constant in an individual over time, as was the case for mental retardation. Thus, the prognosis, even for children who presented with mild symptoms, was believed to be gloomy.

Psychoanalytic Theory and Developmental Psychopathology

One of the strengths of the psychoanalytic approach to psychopathology, as its theory and treatment methods developed around the turn of the twentieth century, was that it rejected the therapeutic pessimism of much contemporary child psychiatry. Although Sigmund Freud accepted that individuals had innate or constitutional characteristics, he developed what his daughter, Anna (Freud, 1965), described as an "etiological formula of a sliding scale of internal and external influences: that there are people whose sexual constitution would not have led them into a neurosis if they had not had (certain) experiences, and these experiences would not have had a traumatic effect on them if their libido had been otherwise disposed" (see Freud, 1916–1917, p. 347). Children whose libido disposed them to pathology could be saved by the right environment, or therapy, or both. Thus, although even mild symptoms could be ominous, the course was not inevitable.

Psychoanalytic theory was fundamentally developmental at a time when the term had no place in mainline child psychiatry; as an example, the entries under the heading "Development" in the index of Anna Freud's *Normality and Pathology in Childhood* (1965) take up two columns, whereas there is not a single entry under that heading in two of the classics of mid-twentieth-century American child psychiatry (Chess & Habibi, 1978; Kanner, 1945). Psychoanalytic theory was also developmental in the multiple senses discussed in this chapter and throughout

this volume: It emphasized the multiple determination of outcomes, the transformation and hierarchical integration of behavior, and the emergence of novelty. In the words of Anna Freud (1965, pp. 166–167):

> As we abandon thinking in terms of specific causes of dissociality we become able to think increasingly in terms of successful or unsuccessful transformations of the self-indulgent and asocial trends and attitudes which normally are part of the original nature of the child. This helps to construct developmental lines which lead to pathological results, although these arc more complex, less well defined, and contain a wider range of possibilities than the lines of normal development.

This approach held sway over child psychiatry for almost a century, despite a lack of empirical evidence supporting its interpretation of the etiology of mental disorders (Grunbaum, 1977). In the past two decades, Freudian developmental psychopathology, with its emphasis on etiology, has largely been replaced, particularly in the United States, by a more phenomenological approach whose goal has been to follow the example of medicine, which looks for diseases that have a standard etiology and set of manifestations, and of some branches of psychology, which seek to pin down concepts like intelligence in forms that are deliberately designed to transcend differences that are a function of developmental factors (Cairns & Cairns, 1991). However, although the *content* of psychodynamic theory has not stood up to empirical research, the *form* of the theory, with its emphasis on how the development of the child and of the disease are intertwined, retains considerable attraction as a model for developmental psychopathology and epidemiology.

Distinguishing Normal from Abnormal

One of the great psychiatric dilemmas of our time is the decision as to what is normal and what is abnormal in human behavior. Lacking specific tests to make the distinction, the diagnostician has recourse mainly to clinical judgment which rests on training, experience, perceptiveness, and theoretical persuasion. In child psychiatry, Leo Kanner (1945) pointed out that recorded symptoms "are of necessity those of selected groups and not of the total population of children" (quoted in Lapouse & Monk, 1958). He continued, "This selectiveness, in the absence of 'normal controls' has often resulted in a tendency to attribute to single behavior items an exaggerated 'seriousness' with regard to their intrinsic psychopathologic significance. The seriousness becomes attached to the signal regardless of what it announces and who announces it. The high annoyance threshold of many fond and fondly resourceful parents keeps away from clinics and out of reach of statistics a multitude of early breath holders, nail biters, nose pickers and casual masturbators who, largely because of this kind of parental attitude, develop into reasonably happy and well-adjusted adults" (Lapouse & Monk, 1958, p. 1134).

By 1970 a number of older general population studies (Cullen & Boundy, 1966; Cummings, 1944; Griffiths, 1952; Haggerty, 1925; Lapouse, 1966; Lapouse & Monk, 1958, 1964; Long, 1941; McFie, 1934; Olson, 1930; Wickman, 1928; Young-Masten, 1938; Yourman, 1932) reported prevalences of individual problem behaviors reported by parents and teachers of older children and adolescents. One of the achievements of these early child psychiatric epidemiologists was to document just how common individual abnormal behaviors are in the general population of children. For example, in their survey of a random sample of 6- to 12-year-olds in Buffalo, New York, Lapouse and Monk (1958) found that 43% of children were reported by their mother to have seven or more fears or worries, 49% to be overactive, and 48% to lose their tempers twice a week or more. Similarly, Shepherd et al. (1971) found that, on their scale of 25 deviant behaviors, only 40% of a population sample of elementary school children in Buckinghamshire, England, was not deviant. However, only 2.6% of the children were deviant in seven or more areas. Some of these studies (Cummings, 1944; Griffiths, 1952; Macfarlane, Allen, & Honzik, 1954; Macfarlane et al., 1954) also illustrate a point that seems often to have been forgotten until recently (Loeber & Stouthamer-Loeber, 1997; Tremblay et al., 1999): that some conduct problems now associated with *DSM-5* conduct disorder, such as lying and some forms of aggression, actually have their peak prevalence before age 5.

Measuring Child and Adolescent Psychopathology

The problem with the approach to measuring the prevalence of child psychopathology used in these two studies was illuminated by a review of prevalence studies commissioned for President Carter's Commission on Mental Illness (Gould, Wunsch-Hitzig, & Dohrenwend, 1980). Prevalence estimates of childhood psychopathology varied widely, depending on whether parents or teachers were surveyed (at that time, children were rarely asked about their own problems). Estimates of the prevalence of maladjustment varied widely even when they were based on data from the same informant group: from 6.6% to 22% according to teachers and from 10.9% to 37% according

to mothers. It is difficult either to plan service delivery systems or to examine causal factors if the rate of the disorder itself is so imprecise.

The questionnaires used in the 1950s and 1960s had three other disadvantages for epidemiological research: (1) they were not designed to distinguish clearly among different syndromes or diagnostic clusters of symptoms; (2) they did not take developmental changes into account; and (3) they did not, as a rule, differentiate symptoms of a disorder from any impairment in functioning that might accompany those symptoms but not be a part of the syndrome itself. For example, failing to perform at age-appropriate levels in school could be a symptom of general mental retardation, a specific learning difficulty, or attentional problems associated with ADHD or depression, or it could result from repeated absence from school because of acute separation anxiety or a chronic physical illness. General maladjustment scales did not make it easy to study the relationship between symptoms and impaired functioning in a way that might illuminate causal relationships.

Epidemiologists and clinical researchers have tackled this problem in several different ways in recent decades. The goal of all these approaches is to elicit information that will identify children with psychiatric syndromes or disorders, but the methods differ substantially. Some have developed survey-type questionnaires consisting of questions designed to mirror the symptoms described in a psychiatric taxonomy such as the *DSM* or ICD. Others, wanting to capture more of the detailed information about symptom severity, duration, age at onset, and associated impairment that is needed, together with symptom reports, to make a diagnosis or devise a treatment plan, have developed structured psychiatric interviews (Shaffer et al., 1999). The goal (Angold et al., 2012) has been to mirror as closely as possible the diagnostic skill of experienced child psychiatrists while overcoming the twin problems that clinicians are notoriously unreliable (Zubin, 1978) and are usually too expensive to be used in large or longitudinal community studies. Here we discuss one or two issues about the use of questionnaires that have implications for developmental epidemiology; the area is covered in detail elsewhere (Angold, 2002; Shaffer et al., 1999).

Questionnaires for Assessing Child Psychopathology

There are hundreds of questionnaires designed for the child, teacher, or parent to complete, covering psychopathology in general or specific syndromes. We discuss just one set of these as examples of the strengths and weaknesses of this approach for developmental epidemiology. Achenbach and his colleagues set out to develop a set of interrelated symptom questionnaires (now called the Achenbach System of Empirically Based Assessment [ASEBA]; Achenbach & Rescorla, 2000; Rescorla & Alley, 2001), whose items were selected using a mixture of clinical and epidemiologic methods. The original set of behavioral and emotional problems was compiled from clinical sources, but the process of reducing the list to the number finally selected was based on studies using general population samples. The first of the questionnaires, the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983), was designed for use by parents whose children were 4 through 16. Items endorsed by fewer than 5% or more than 95% of parents were rejected, and language was simplified to be understandable by anyone with a fourth-grade education. Achenbach and Edelbrock carried out extensive factor-analytic studies of how items clustered together in children of different age groups and genders. Many of the syndromes identified in this way showed clear links to the standard diagnostic labels used in psychiatry (Verhulst, van der Ende, Ferdinand, & Kasius, 1997). This is not surprising, as the original set of items was submitted by clinicians familiar with how children's symptoms tend to present. However, the CBCL proved to have much higher test–retest reliability than clinicians did, and items were quick to administer and score.

The work of Achenbach and his colleagues has provided some powerful tools for developmental epidemiology, both practically and conceptually. First, the extensive data collection and careful standardization on large samples set high standards for instrument development. Second, the team developed a closely integrated set of scales associated with the original CBCL, covering infancy to adulthood and available in versions for parent, teacher, and self-report. Third, the list of emotional and behavioral problems is set in the context of questions about the child's social competencies and school performance; basic sociodemographic information also is collected. Fourth, a lot of work has gone into language and layout, and the four-page questionnaires collect a great deal of information in a brief time (about 20 minutes). As a result of these virtues, the instruments are very widely used for both clinical and epidemiological purposes. They have been translated into many languages, encouraging cross-cultural studies of the generalizability of the identified symptoms and syndromes and their correlates (Broidy et al., 2003; Ivanova et al., 2007; van Eldik, Treffers, Veerman, & Verhulst, 2004; Verhulst, Achenbach, Althaus, & Akkerhuis, 1988; Weisz, 1989).

The many virtues of the questionnaire approach adopted by Achenbach and colleagues make it even more

important to recognize its limitations (Achenbach, 1985). First, the strategy of omitting rarely endorsed symptoms means that this method cannot be used to describe some of the most severe and disabling problems, for example, those seen in children with bipolar disorder. Second, it is important to note that the questionnaire method is predicated on the assumption that the respondent knows the child well enough to be able to make ratings on items like "Feels too guilty" or "Lying or cheating." A checklist is thus a very well-designed form for recording information about a child that the respondent already knows, but the data that it provides can only be as good as the respondent's knowledge of the child in question. Third, this method produces a series of scale scores for each child, standardized relative to a population sample matched for age, sex, and social class. These scores cannot be used to answer such public health questions as, "How many children need treatment?" unless certain decision rules are applied to them. A clinical cut point has been identified for the global behavioral problem score of the ASEBA checklists, based on a comparison of the scores of children referred for mental health services during the previous year with the scores of nonreferred children (Achenbach, 1991). However, as we discuss later, there is reliable evidence from national data and community surveys that receipt of services is a poor criterion for defining need for services (Burns et al., 1995; Costello, Burns, Angold, & Leaf, 1993; Costello, Pescosolido, Angold, & Burns, 1998). It is also worth emphasizing that scale scores are standardized to identify a priori a certain percentage of the population as being in the clinical range. Thus, they cannot be used to provide prevalence estimates.

Interviews for Identifying Psychiatric Disorders in Children and Adolescents

The checklist approach does not provide the detailed information about symptom severity, duration, age at onset, and associated impairment that is needed, together with symptom reports, to make a diagnosis or devise a treatment plan. Alternative approaches are needed, ones more closely related to the process of clinical decision making. Beginning with the work of Michael Rutter in the 1960s, a large number of structured psychiatric interviews have been developed. They have much in common. All structured interviews aim to:

- Structure information coverage so that all interviewers will have collected all relevant information (both confirmatory and disconfirmatory) from all subjects
- Define how relevant information is to be collected
- Structure the process by which relevant information is combined to produce a final diagnosis

Interviews vary widely in how they go about these tasks. However, a general distinction can be made between interviewer-based (or investigator-based) interviews, respondent-based interviews, and best-estimate interviews (Angold et al., 2012).

Interviewer-Based Interviews. These interviews require the interviewer to make an informed decision based on what the respondent says. Interviewers are expected to question until they can decide whether a symptom meeting the definitions provided by the interview (or known to them from their training) is present or absent. This group of interviews includes the Anxiety Disorders Interview Schedule (Silverman & Rabian, 1995); the Child and Adolescent Psychiatric Assessment (CAPA; Angold & Costello, 2000); the Child Assessment Schedule (Hodges, 1993; Hodges et al., 1982); the paper-and-pencil (not the computerized) versions of the Diagnostic Interview Schedule for Children and Adolescents (DICA; Reich, 2000) and the Missouri Assessment of Genetics Interview for Children (MAGIC; Reich, 2000), the Interview Schedule for Children and Adolescents (Sherrill & Kovacs, 2000); various versions of the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Ambrosini, 2000); and the Pictorial Instrument for Children and Adolescents (Ernst, Cookus, & Moravec, 2000).

Three of these interviewer-based interviews (the K-SADS-P IVR, the DICA, and the CAPA) provide extensive sets of definitions of symptoms or detailed guidance on the conduct of the interview. Such glossaries are particularly important when an interviewer-based interview is to be used by nonclinician interviewers because they provide detailed guidance as to what the interviewer is supposed to be looking for in making symptom ratings. Nonclinician interviewers have been shown to be able to make such clinical judgments with high reliability when they have received adequate training with such glossaries (Angold & Costello, 1995).

Respondent-Based Interviews. In other psychiatric interviews, it is the respondent who makes the final decision about whether a symptom is present, typically by answering yes or no to each question. The interviewer makes no such decisions but simply reads the questions. Because the decision as to the presence or absence of psychopathology lies with the respondent in such interviews, we refer to them as respondent-based. The DISC (Shaffer et al., 2000), the computer-assisted version of the DICA (Reich, 2000), and the Dominic-R (Valla, Bergeron, & Smolla, 2000) are the three representatives of this approach.

A variant on these two approaches to interviewing is taken in the Development and Well-Being Assessment (Goodman, Ford, Simmons, Gatward, & Meltzer, 2000), which is widely used around the world. Here the interviewer uses a respondent-based approach first and then supplements the information by asking additional questions. The responses to these are integrated with the structured responses by an experienced clinician, who formulates the diagnosis.

The distinction between interviewer- and respondent-based interviews is not hard and fast in actual practice because there has been considerable cross-fertilization between these approaches. For instance, CAPA, which has its roots in the interviewer-based tradition, includes a subset of questions that are to be asked verbatim of all subjects, as in a respondent-based interview, but then allows further questioning for clarification. The DICA, which had previously been a respondent-based interview, now requires interviewers to question much more flexibly and is currently an interviewer-based instrument (Reich, 2000). Although the distinction between interviewer- and respondent-based interviews provides a useful rough-and-ready typology, it is really better to consider interviews as lying at various locations along three dimensions: (1) degree of specification of questions; (2) degree of definition of symptom concepts; and (3) degree of flexibility in questioning permitted to the interviewers (Angold, 2002).

Summary

In this section, our goal has been to see where developmental epidemiology came from and how it got to be where it is today. The problems that had to be formulated and solved were of two kinds: how to conceive of the nature of childhood psychopathology and how to measure it. Progress in the past century has been extraordinary in both areas. However, as we discuss later, this progress has brought developmental epidemiology to a point where radically new concepts, and new methods, are now needed. Before we get there, however, we look at how epidemiologists in other areas of medicine have thought about developmental questions, and give some examples from psychiatry.

EPIDEMIOLOGY AS A DEVELOPMENTAL METHOD

Much of this section is inspired by Breslow and Day's (1980, 1987) two-volume *Statistical Methods in Cancer Research*. As those authors point out: "Most chronic diseases are the result of a process extending over decades, and many of the events occurring in this period play a substantial role" (Breslow & Day, 1987, p. 2).

In the study of physical growth, of mental and hormonal development, and in the process of aging, the essential feature is that changes over time are followed at the individual level. Longitudinal surveillance and recording of these events is therefore a natural mode of study to obtain a complete picture of disease causation.

Many psychiatric disorders fall into the category of chronic diseases, and the methods developed by cancer and cardiovascular epidemiologists to explore causal relationships in such diseases can, we believe, provide at least a useful starting place for thinking about psychiatric disorders.

Chronic disease epidemiologists view the diseases that they study as having inherent developmental processes of their own; processes that obey certain laws and follow certain stages even as they destroy the individual in whom they develop (Hay & Angold, 1993). Developmental epidemiology asks what happens when developmental processes embodied in pathogenesis collide with the processes of human development.

Risk, Exposure, and the Meaning of Time

Many questions can be answered only by methods that take into account temporal characteristics of risk factors, including their onset and the "dose," or level of exposure, over time. Age at first exposure, time since first exposure, duration of exposure, and intensity of exposure are all interrelated aspects of timing that may have different implications for causality and thus for prevention. The kinds of questions we are thinking of include the following:

- Does physical abuse by parental figures cause psychiatric disorders in children? Is a single blow a sufficient cause or does abuse have to go on for a period of time, or happen at a certain level of severity, before it constitutes a risk factor? Are children of different ages or developmental stages differentially vulnerable to physical abuse as a risk factor? What risks are associated with removing children of different ages from home because of physical abuse?
- Why are depressive disorders rare in both prepubertal girls and boys, but much more common in postpuberal girls? What causes the observed sex difference to develop? Is it associated with hormonal, morphological, or social changes occurring around puberty? Why is earlier-than-average maturation apparently a positive event for boys but a negative one, associated with increased risk of behavioral and school problems, for girls?

A risk factor may have a different impact on the risk of disease, depending on when it first occurs, how long it is present, and the level of intensity at which it occurs. For example, insulation workers exposed to asbestos had a cumulative risk of dying of mesothelioma over a 20-year period (controlling for other causes of death) that was the same irrespective of age at first exposure (Peto, Seidman, & Selikoff, 1982). Here, *length of exposure* was the critical aspect of risk. In contrast, the risk of breast cancer following irradiation appears to be highest in girls exposed at ages 0 through 9, falling with age until there is little excess risk for breast cancer associated with exposure to radiation after age 40 (Howe, 1982). Cases of breast cancer attributable to irradiation begin to occur some 10 years after exposure and continue thereafter at a roughly constant level, suggesting that the absolute excess risk increases with time since exposure. Thus, *timing of exposure* is the critical aspect of the risk exposure.

However, different sorts of radiation can have different durations as risk factors for cancer; for example, bone sarcomas occurring after exposure to the radioisotope 224Ra, which has a half-life of 3.6 days, cluster in a period of 5 to 10 years following first exposure, whereas bone sarcomas following exposure to 226Ra, which has a half-life of 1,600 years, occur at a constant rate beginning 5 years after first exposure (Mays & Spiess, 1984). In the latter case, exposure to the decay products of the radioisotope continues at virtually constant levels after absorption of the radium.

A longitudinal study of British male doctors' smoking provides an example of dose-related risk: The annual death rate per 100,000 men, between 1951 and 1971, standardized for age, was 1,317 for nonsmokers, 1,518 for those currently smoking 1 to 14 cigarettes a day, 1,829 for those currently smoking 15 to 24 a day, and 2,452 for those smoking over 24 a day (Doll & Peto, 1976). This was true inspective of age, and risk fell quite rapidly in those who gave up smoking (Breslow & Day, 1987). In this case, *amount of exposure* was the critical factor.

Examples from Developmental Psychopathology

These examples illustrate the importance for developmental epidemiology of thinking about different aspects of development: age at first exposure, time since first exposure, duration of exposure, intensity of exposure. Here we present some examples of how child psychiatric researchers have used ingenious designs to tease out the same issues.

The importance of *age at first exposure* has been studied most intensively of all the aspects of risk over time in child psychopathology because of the theoretical importance attached to early experiences in the Freudian and other psychodynamic models of development. For example, researchers investigating the role of attachment in children's development have concentrated on the very early months and years of life as the crucial period during which the inability to form one or more such relationships may have damaging effects that last into childhood and perhaps even into adulthood (Sroufe, 1988). The critical date of onset of risk appears to occur after 6 months, but the duration of the risk period is not yet clear. Hay (1985) presented evidence that maternal depression, which presumably interferes with mothers' ability to form normal relationships with their infants, affects motor development if it occurs during the 1st year of life, and language development but not motor development if it occurs during the 2nd year of life. This is a case where age at first exposure appears to interact with the developmental processes most salient at a particular age. In another example of the importance of timing, Rutter (1985) pointed out that once children have achieved urinary continence at around age 2, there is a period of risk for relapse into incontinence that appears to coincide with starting school. Once this period of risk is over, the chance of developing enuresis is very slight. In this case, age at exposure is clearly the critical developmental risk factor because no parallel increase in functional enuresis occurs at later times of stress, such as moving to middle or high school, and there is no delay between the stress and the symptoms.

Timing of exposure has rarely been treated separately in studies of child psychopathology. Brown and Harris (1978), in their work on the social origins of depression, argued that women who lost their mother in the 1st decade of life were more vulnerable as adults to depressive episodes in the face of severe life events. However, theirs was a retrospective study that did not address the question of whether these women were also at greater risk of depressive episodes during later childhood and adolescence. It is not clear whether the crucial factor was the length of time since exposure to the risk factor of mother's death, or the age of the child at the time of exposure, or some combination of the two.

Timing of puberty has emerged as an important aspect of risk in relation to both depression and behavioral problems. In a longitudinal study that measured not only age at menarche but also morphological development, Tanner staging (Marshall & Tanner, 1969), and levels of gonadal and steroidal hormones, high levels of testosterone, not timing of puberty, predicted female adolescent depression (Angold et al., 1999). On the other hand, many studies show that girls who are early in developing the morphological signs of puberty, indexed by Tanner stage

or menarche, are at risk for behavioral problems if they have unsupportive families (Ge, Brody, Conger, & Murry, 2002; Ge, Conger, & Elder, 1996; Magnusson, Stattin, & Allen, 1985; Moffitt et al., 1992).

Duration of exposure to poverty was examined by Offord and colleagues (1992) in their repeated surveys of a representative sample of children in Ontario. They showed that children whose families were living below the poverty level at two measurement points were at increased risk of behavioral disorders, compared with children whose families were below the poverty level on one occasion only, or never. In a longitudinal study from New Zealand, Moffitt (1990) found that children identified at age 13 as both delinquent and hyperactive had experienced significantly more family adversity (poverty, poor maternal education, and poor mental health), consistently from the age of 7, than children who were only delinquent or only hyperactive at age 13:

> The most striking increase in the antisocial behavior of ADD+delinquent boys (diagnosed at age 13) occurred between the ages of 5 and 7, when they attained a mean antisocial rating that was not reached by other delinquent boys until 6 years later. School entry and reading failure coincided temporally with this exacerbation of antisocial behavior. These data suggest that the problem behavior of this group, despite being generally persistent, is responsive to experience. The data also reveal a key point of vulnerability that could be a target for intervention: reading readiness. (p. 906).

Another example comes from the Great Smoky Mountains Study. Children who had been assessed over an eight-year period were classified into four groups on the basis of their body mass index (a ratio of weight to height) at each assessment: no obesity (72.8%), childhood-only obesity (5.1%), adolescent-only obesity (7.5%), and chronic obesity (14.8%). Only the chronically obese group was at increased risk of psychiatric disorder (Mustillo et al., 2003). This is an example of *duration of exposure* as the key risk characteristic. It is also an example of the impact of *intensity of exposure* because no effects were found of overweight that fell below the threshold of obesity.

Intensity of exposure to lead (Needleman & Bellinger, 1991) provides an example of a definite dose–response relationship. Another aspect of intensity is the number of different risk factors to which a child is exposed (Seifer, Sameroff, Baldwin, & Balwin, 1989). Rutter (1985) and others have pointed out that children exposed to one risk factor are at increased risk of exposure to others (e.g., no father in the home and poverty) and that the dose–response relationship to an increasing number of different risk factors is not a simple linear one. Most children appear to be able to cope with a single adverse circumstance, but rates of psychopathology rise sharply in children exposed to several adverse circumstances or events (Seifer et al., 1989).

These examples show that it is possible to design studies that at least begin to allow us to tease out the respective roles played by time since first exposure, age or developmental stage at first exposure, duration of exposure, and intensity of exposure. Multistage models of risk, which have been developed to address the complexities of causality in chronic disease, are one way of putting the pieces together. Several such models have been proposed, particularly in the context of carcinogenesis (Peto, 1984), and have been reviewed in terms of developmental psychopathology (Pickles, 1993). Statistical techniques for exploring causality in such multistage models have made great strides recently (Pearl, 2013; Robins, 2001). The challenge is to incorporate all the various aspects of risk into a single model and distinguish the ones that carry the tune from those that are just noise.

FUTURE DIRECTIONS: DEVELOPMENTAL EPIDEMIOLOGY

We have described how child psychiatric epidemiology has developed over the past 50 years as a collaborative activity of basic, clinical, and public health scientists working together to understand the magnitude of the problem of child mental illness and to identify key risk factors. Many of the fundamental problems of conceptualization and measurement have been solved, or at least reduced to manageable proportions.

Up to this point, the role of epidemiology has been mainly a descriptive one: addressing the basic questions, How many? Who? Where? When? We are now in the position of being at least as capable of answering these questions reliably and accurately in child psychiatry as are chronic disease epidemiologists in other areas of medicine. However, child psychiatry is changing, and epidemiology will change as well. The goal is now to understand how risk exposure and vulnerability change over the life course, and how the requirements of so-called normal development at each developmental stage shape the types of psychopathology that emerge if these requirements are not met. The term *developmental epidemiology*, first coined by Kellam in the 1970s (Kellam, Ensminger, & Turner, 1977), is useful to describe what epidemiology is doing these days.

In this concluding section, we describe some rapidly growing research areas that will contribute to the next generation of studies to the shift from child psychiatric epidemiology to developmental epidemiology. The different approaches overlap in many areas, but we discuss the future under three broad headings: longitudinal research; impact studies of burden, cost, and prevention; and studies of mechanisms.

Longitudinal Research

Longitudinal research is almost the sine qua non for testing developmental hypotheses about causes and risks. Here we consider three types: longitudinal studies, life-course studies, and intergenerational studies.

Longitudinal Studies

Although there have been many longitudinal developmental studies, some of them beginning at birth (or even before), longitudinal studies of psychiatric disorders had to await the development of appropriate technology, specifically, data collection methods that validly and reliably translated a given psychiatric taxonomy into instruments that could be used repeatedly with the same subjects (see earlier discussion).

An example of how longitudinal studies can be used to address developmental questions can be found in a recent paper on the Great Smoky Mountains Study (Copeland, Shanahan, Costello, & Angold, 2011). Annual interviews with 1,420 young people over a period from age 9 through 21 provided data to address such questions as the following: Does the prevalence of different disorders increase or decrease with age? Do children with a disorder continue to have the same disorder (homotypic continuity) or a different disorder (heterotypic continuity)? Longitudinal analyses showed that, although the average annual prevalence of psychiatric disorder was around 18%, over the period of the study more than 60% had one or more disorders. Some disorders (ADHD, separation anxiety, functional enuresis) dominated the picture in childhood but had almost disappeared by age 16. Depression and drug abuse were predominantly disorders of adolescence. Anxiety disorders in general had a U-shaped distribution, falling to their lowest point at age 12. Although wave-to-wave continuity was not very high, over the course of the study there was a high degree of continuity of diagnosis (Table 3.1). The increased likelihood of an episode of a disorder following an earlier episode was statistically significant for every diagnosis except specific phobia.

In contrast, there was relatively little prediction from one disorder to another (heterotypic continuity), and it was almost entirely restricted to girls. Anxiety predicted depression, and depression predicted anxiety. Anxiety and conduct disorder predicted substance abuse, and there was some movement among the disruptive behavior disorders,

TABLE 3.1 Homotypic and Heterotypic Continuity, with and Without Controls for Comorbidity

Predicting to	Past depression	Past anxiety	Past conduct disorder	Past oppositional defiant disorder	Past ADUD	Past substance abuse
Depression	7.0 (3.1, 15.9)[c]	3.0 (1.7,5.4)[c]				
Controlling for comorbidity	4.2 (2.1,8.3)[c]	2.7 (1.8, 5.2)[b]				
Anxiety	5.7 (2.2. I4.5)[c]	2.4 (1.6,3.7)[c]				
Controlling for comorbidity	2.8 (1.2,6.5)[a]	2.0 (1.2,3.4)[a]				
Conduct disorder			11.2 (5.9, 21.1)[c]			
Control ling for comorbidity			10.3 (4.3, 24.7)[c]			
Oppositional defiant disorder				3.7 (2.2, 6.2)[c]	2.0 (1.1,3.8)[a]	
Controlling for comorbidity				4.7 (2.7,8.1)[c]	2.1 (1.1, 4.2)[a]	
ADHD			2.2 (1.0.4.5)[a]		10.7 (5.2. 22.3)[c]	
Controlling for comorbidity			1.8 (0.9. 3.9)		9.6 (4.4, 21.2)[c]	
Substance abuse		2.0 (1.2,3.5)[a]	2.7 (1.2,6.5)[a]			21.3 (6.3, 72.5)[a]
Controlling for comorbidity		2.0 (l.l, 3.7)[a]	1.7 (0.6.4.7)			25.7 (7.8, 85.4)[a]

Note: [a]$p < .05$; [b]$p < .01$; [c]$p < .001$; NS = Not significant.
Adapted from "Prevalence and Development of Psychiatric Disorders in Childhood and Adolescence," by E.J. Costello, S. Muslillo, A. Erkanli, G. Keeler, & A. Angold, 2003, *Archives of General Psychiatry, 60,* pp. 837–844.

perhaps suggesting that the *DSM* taxonomy fits boys better than it does girls.

Several research groups have now used their longitudinal data to look at continuities and discontinuities in mental illness from childhood into adolescence and beyond. Some of the longitudinal studies have followed their subjects into adulthood (Caspi, Moffitt, Newman, & Silva, 1996; Cohen, 1996; Fergusson & Woodward, 2000; Reinherz, Giaconia, Hauf, Wasserman, & Silverman, 1999; Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001). These are beginning to show indications of continuity between temperamental characteristics in early childhood and the onset of psychiatric disorders in late adolescence and young adulthood (Caspi et al., 1996). Two examples come from the Dunedin study, a birth cohort of around 1,000 children from New Zealand who have been evaluated every two years since birth.

The first set of analyses (Jaffee et al., 2002) started from hints in the literature that juvenile-onset major depressive disorder (MDD) may be etiologically distinct from adult-onset MDD. In these analyses, early childhood risk factors covered the period from birth to age 9 years. Diagnoses of MDD were made according to *DSM* criteria at three points prior to adulthood (ages 11, 13, and 15 years) and three points during adulthood (ages 18, 21, and 26 years). Four groups were defined: individuals first diagnosed as having MDD in childhood, but not in adulthood ($n = 21$); individuals first diagnosed as having MDD in adulthood ($n = 314$); individuals first diagnosed in childhood whose depression recurred in adulthood by age 26 years ($n = 34$); and never-depressed individuals ($n = 629$). By the time of an age 32 follow-up, 44% of the cohort had experienced at least 1 episode of depression (Moffitt et al., 2007).The two juvenile-onset groups had similar high-risk profiles on the childhood measures. Compared with the adult-depressed group, the juvenile-onset groups experienced more perinatal insults and motor skills deficits, more caretaker instability, criminality, and psychopathology in their family of origin, and more behavioral and socioemotional problems. The adult-onset group's risk profile was similar to that of the never-depressed group with the exception of elevated childhood sexual abuse. These analyses made it clear that the distinction between juvenile- and adult-onset MDD is important for understanding heterogeneity in depression. Similar findings have been seen in clinical samples (Zisook et al., 2004).

A second example from the same study revisited the theory that there are two types of adolescent delinquency, childhood-onset and adolescence-limited; that their manifestations in adolescence are indistinguishable; but that their consequences for persistence into adulthood are different (Moffitt, 1993). At the age-26 follow-up (Caspi et al., 2002), the childhood-onset delinquents were the most elevated on psychopathic personality traits, mental health problems, substance dependence, numbers of children, financial problems, work problems, and drug-related and violent crime, including violence against women and children. The adolescent-onset delinquents were less extreme at age 26 but showed elevated impulsive personality traits, mental health problems, substance dependence, financial problems, and property offenses. A third group of men who had been aggressive as children but not very delinquent as adolescents emerged as low-level chronic offenders who were anxious, depressed, and socially isolated and had financial and work problems. A follow-up at age 32 showed that a family history of behavioral problems distinguished males with persistent problems from those whose behavioral problems were limited to childhood or adolescence (Odgers et al., 2007).

A third line of research from the Dunedin study has begun to link early mental health problems with the early onset of poor physical health and, possibly, risk factors for early mortality. For example, early social isolation had persistent and cumulative detrimental effects on adult health, mediated by childhood overweight, low IQ, and lack of exercise (Caspi, Harrington, Moffitt, Milne, & Poulton, 2006).

These examples serve to show how as developmental epidemiology grows up it can contribute to developmental psychopathology and also shine new light on some of the accepted verities of adult morbidity and mortality.

A next step in the use of descriptive longitudinal studies for developmental epidemiology must be to work out ways of releasing data from these studies for public access so that future researchers can take advantage of these incredibly valuable resources. There are many issues to be worked out, not the least of them the protection of participants' privacy and the rights of the scientists who labored for so many years to collect the data. But these are soluble problems, and the benefits of bringing fresh minds to bear on issues in developmental epidemiology are likely to be tremendous.

Life-Course Studies

Psychiatric disorders are increasingly showing themselves to be chronic diseases. For example, across childhood and adolescence, for every psychiatric disorder except specific phobias, the likelihood of an episode has been shown to be significantly higher in youth with a history of the same disorder than in those with no previous history (Copeland et al., 2011). As data accumulate from prospective studies,

it is becoming clear that the onset of most adult psychiatric disorders lies much further back in childhood than had been suspected from adult studies relying on retrospective recall of onset (Kim-Cohen et al., 2003).

Life-course epidemiology is the study of long-term effects on chronic disease risk of physical and social exposures during gestation, childhood, adolescence, young adulthood, and later adult life. It includes studies of the biological, behavioral, and psychosocial pathways that operate across an individual's life course as well as across generations, to influence the development of chronic diseases (Ben-Shlomo & Kuh, 2002; Kuh & Ben-Shlomo, 1997).

In many ways, life-course and developmental epidemiology mirror one another. Life-course epidemiology, however, has taken on a special concern with the embodiment of social phenomena into the biological (Krieger, 2001), encapsulated in the concept of *health inequalities*. This concern arose historically from work showing that mortality from many diseases is spread unequally across the population (Wilkinson, 1994; Wilkinson & Pickett, 2009) and that these differences in risk can be linked to social inequalities that often go back to infancy or even to the parental generation (Barker, 1998; Davey Smith, Gunnell, & Ben-Shlomo, 2000; Leon, 2000). This body of work has had enormous significance for international thinking about social policy and is having a direct effect on the allocation of public resources in some countries.

Developmental epidemiology needs to take on board the social, economic, and political implications of the fact that children are born with different levels of risk for psychiatric disorder, that their family's socioeconomic status is part of the predictive equation, and that community-level interventions as well as individual treatment can influence how well individuals are able to function (Costello et al., 2003). Future studies will include health disparities (in the broad sense of medical and psychiatric health) as part of their remit.

Global Mental Health

A major shift in the focus of psychiatric epidemiology in the past decade has been from the parochial issues of a single country or continent to the whole world. There are several reasons for this: first, immigration and world travel have opened our eyes to other people's problems. Second, huge multinational studies such as the World Health Organization's World Mental Health Survey (Kessler et al., 2007) have (1) created and tested instruments for use in many language and cultures (Kessler & Üstün, 2004) and (2) permitted comparisons of prevalence rates and risk factors around the world (Demyttenaere et al., 2004). Third, two reports on the global burden of disease, from 1996 (Murray & Lopez, 1996) and 2012 (Lozano et al., 2012), both placed mental illnesses very high in terms of morbidity and disability around the world. Fourth, psychiatry in general is, as noted earlier, beginning to wake up to the reality that most cases of mental illness have their onset or first episode in the first two decades of life (Jaffee, Harrington, Cohen, & Moffitt, 2005). As a result of this confluence of thought and data, the major funding agencies are paying more attention to global mental health, especially of mothers and children (Collins et al., 2011; Patel, Flisher, Hetrick, & McGorry, 2007). This means, for the next generation of epidemiologists, new challenges in developing measures that are usable worldwide, a task that has been tackled for adults but not yet for children (Goodman et al., 2012). Several instruments have been translated into many languages (e.g., the Child Behavior Checklist (Ivanova et al., 2007) and the Development and Well-Being Assessment (Heiervang, Goodman, & Goodman, 2008)), but the hard work of sorting out real and methodological differences between instruments has yet to be done (Angold et al., 2012).

Intergenerational Studies

A life-course approach to developmental epidemiology is not limited to individuals in a single generation but can intertwine biological and social transmission of risk across generations, recognizing that geographic and secular characteristics may be unique to that cohort of individuals (Cairns, Elder, & Costello, 1996; Power, Kuh, & Morton, 2013; Stein, Susser, Saenger, & Marolla, 1975).

Experiences of the previous generation can operate at many different levels of generality. They may be specific to the mother–child dyad (e.g., the effect of drug use during pregnancy) or may affect everyone living in a certain neighborhood (e.g., poverty or exposure to an environmental toxin). All mothers and children may be affected by a particular event, such as a period of famine or disease, or children may be affected by their mother's developmental stage (e.g., children of teen mothers or elderly mothers).

Models for intergenerational research have recently developed (Cairns, Cairns, Rodkin, & Xie, 1998; Rossi, 1989), and statistical methods have become more tractable (Muthen & Muthen, 2000; Robins, 1992; Zeger & Liang, 1992). Undertaking transgenerational studies requires a level of commitment from both researchers and funding agencies that is hard to sustain over the necessary length of time, especially in the United States with its five-year funding cycles. Many American researchers have looked

abroad to find countries that have nurtured such studies and governments that understand their importance (Chasiotis, Scheffer, Restemeier, & Keller, 1998; Granger et al., 1998; Krueger, Caspi, Moffitt, & Silva, 1998). However, we can hope that as policymakers become more aware of the overlapping pattern of risk factors among chronic diseases and the risks associated with very early exposures they will come to understand the immense returns from investment in multigenerational research.

Impact Studies

Prevention Science

The strongest test of a developmental theory about causes of disease and its impact on society is an intervention that manipulates a core etiological factor and tests the outcome. Prevention science is the discipline that has arisen to put this principle into practice. Its strongest growth has been in the area of AIDS prevention, but it has also been an important force in developmental psychopathology, with its own academic societies and research journals.

Prevention science uses theory about the causes of disease to generate interventions, which when tested provide information not only about the effectiveness of the intervention, but also about the etiology of the disease (Dodge, 2001). The famous Broad Street pump intervention described earlier (Snow, 1855) is a classic example of prevention science: If dirty water caused cholera, then removing access to dirty water should both prevent new cases and support the etiologic hypothesis.

Epidemiology traditionally divides prevention into three categories, depending on the mean level of risk in the population of concern. Primary prevention programs like clean water, car seat belts, and parental leave programs are examples of primary or universal prevention. The scientific questions at this level have to do with what is prevented downstream by universal programs upstream. For example, the "Just Say No" drug abstinence program was introduced as a primary prevention for all children in school, designed to stop drug use before it began. Unfortunately, the developmental science behind it was inadequate, and the results were null, if not negative (Lynam et al., 1999). In another careful study of a primary prevention program for school-age children, Stattin and colleagues (Mahoney & Stattin, 2000) evaluated the youth recreation centers that are a ubiquitous feature of Swedish towns. Usually, these are places where young people can hang out, with very little adult supervision or structured activity. Participation in activities with low structure was associated with high levels of antisocial behavior for boys and girls, whereas participation in highly structured leisure activities was linked to low levels of antisocial behavior. Despite several studies, however, community leaders still strongly support these recreation centers: an example of how long it takes for research to influence policy.

On the other hand, primary prevention with children (e.g., the Good Behavior Game; Kellam, Koretz, & Moscicki, 1999), and families (e.g., a family income supplement; Costello et al., 2003) can be effective and suggest etiologic pathways that could be explored in further research. But prevention science has done relatively little in the way of *primary* prevention for mental health problems. It is highly likely that primary prevention programs that improve early nutrition or provide more family leave have effects on the development of psychopathology, but they have not been treated as scientific tests of theory, and so we do not know.

Secondary, or high-risk, intervention programs are much more likely to be subjected to scientifically informative testing, and this is where the bulk of prevention science has been done. A large number of interventions are based on high-risk children, schools, and communities, and many of them have survived the trial by fire of National Institutes of Health review committees and are both theory driven and scientifically sound. This is not the place to review this literature; the main issue for developmental epidemiology is that such studies should be epidemiologically sound. This means that it should be clear how far their results can be generalized. Does an intervention to reduce bullying (Olweus, 1987) work with girls as well as boys? In which age groups? In which ethnic groups? What can we learn about what drives bullying?

A good example of a secondary intervention that yields insights for developmental epidemiology is the Fast Track program for aggressive children in grade school. This was based on clearly articulated theory about cognitive difficulties that could interact with environmental risk to produce aggressive behavior in socially ambiguous situations (Dodge, Pettit, & Bates, 1994). High-risk youth were randomly assigned to an intensive intervention that lasted for 10 years. Hostile attributional bias was indeed found to be a partial mediator of the effect of the intervention on reductions in aggressive behavior. Two years after it ended, youth assigned to preventive intervention had significantly lower use of general and outpatient mental health services but not of inpatient mental health services (Jones et al., 2010). As adolescents and adults, they had fewer arrests (Group, 2010). The intervention was effective but clearly expensive, lasting as it did over 10 years.

Tertiary interventions have hardly been explored in the context of tests of developmental theory. Once children have developed clinically defined psychiatric disorders, interventions focus on clinical treatment rather than tertiary prevention, although pediatricians (Kendrick, 1999), psychologists (Williams, Holmbeck, & Greenley, 2002), and psychiatrists (Pols et al., 1996) are beginning to talk about the need for a focus on tertiary prevention. Given the early onset of most psychiatric disorders, this is clearly a vitally important area for future work.

Within this broad framework there is a wide variety of intervention strategies, some of which may take developmental issues into account. A good example is suicide prevention, which was comprehensively reviewed by Gould and colleagues (Gould, Greenberg, Velting, & Shaffer, 2003). Among the primary prevention efforts they discuss are gun control programs at the national and local level and school-wide suicide awareness curricula. Secondary prevention efforts have included skills-training programs for youth, whether identified through screening or self-referred, screening programs to try to identify children with suicidal ideas, training for gatekeepers such as primary care pediatricians, peer counseling programs, crisis centers, and hotlines. Other approaches include training the media on how to report suicide to minimize copycat behavior, and postsuicide counseling in schools and among the friends of youth who have killed themselves. Tertiary care programs focus on following up suicide attempters in an effort to reduce repeat episodes. Research on the effectiveness of all these strategies is patchy, and some continue to be used despite doubts about their developmental appropriateness and effectiveness (Gould et al., 2003; Gould, Cross, Pisani, Munfakh, & Kleinman, 2013). However, suicide is one of the few areas in child and adolescent mental health in which one can see the skeleton of an integrated prevention strategy beginning to emerge.

It sometimes feels as though progress in implementing prevention programs in the mental health area has been glacially slow, and public policy is often impervious to evidence. But prevention scientists are determined people, and they are building a powerful set of weapons in the form of statistical methods, meta-analytic studies, and cost-benefit analyses that will help them to get their message across in the next decade.

Burden of Illness and Costs and Effectiveness of Interventions

As we described earlier, epidemiology (the study of where, when, why, and to whom diseases occur) has a dual role: to increase our understanding of what causes an illness (scientific epidemiology) and to help institute policies to prevent or control it (public health epidemiology). Implementing policies to prevent or control disease costs money. Before allocating resources to any disease, policymakers need to know how big the problem is, what interventions are available, how useful they are, and how much they cost. So epidemiology has historically been concerned with estimating the burden of disease and the cost and effectiveness of interventions (Angold & Costello, 2000).

Developmental epidemiology has a new role to play in this area, for all the reasons discussed in the section on life-course epidemiology. It is becoming increasingly clear that we cannot afford to wait to intervene until, for example, a child develops conduct disorder. Treatment at that stage is expensive, labor intensive (Costello et al., 2007; Henggeler, Melton, Brondino, Scherer, & Hanley, 1997), and of modest effectiveness (Lipsey & Wilson, 1998). It is imperative that we apply what is known about early predictors to develop interventions that can be used earlier in the development of the disorder. This is an enormous research program and is addressed elsewhere in these volumes.

The role of the developmental epidemiologist is often that of record keeper: to measure the burden of disease (World Health Organization, 1996), the need for (Costello et al., 1993; Rice, Woolston, Stewart, Kerker, & Horwitz, 2002), cost of, and barriers to services (Flisher et al., 1997; Leslie, Rosenheck, & Horwitz, 2001; Owens et al., 2002) at different developmental stages, and the changes in disease incidence as new interventions are introduced (Brown, van Os, Driessens, Hoek, & Susser, 2000; Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1993; Kellam, Rebok, Mayer, Ialongo, & Kalodner, 1994).

None of these activities is new to epidemiology in general, but they have been a long time coming to the area of child psychiatric disorders. The next decade will see a significant expansion of cost, cost-benefit, and cost-effectiveness studies, either as new studies or as additions to ongoing ones.

Mental Health Surveillance

Tracking the prevalence (ongoing cases) and incidence (new cases) of diseases over time is essential if we are to study both the societal impact of a disease, and that of treatment or prevention programs. This is a program of work that has hardly begun in the area of child psychopathology. We rely on agencies of the Centers for Disease Control and Prevention (CDC) to tell us how many cases of SARS there were last year in the United States and whether the incidence of AIDS is increasing or decreasing. Until recently, it would have been of no use to ask CDC the same questions

about prepubertal depression or early-onset schizophrenia, although their social cost may well be as high (Lozano et al., 2012; Murray & Lopez, 1996). Areas where the CDC takes an interest in prevalence include drug use and abuse, on which annual surveys of the school-age population have been available since 1975 from the University of Michigan's Institute for Social Research. Thirty years of data can be tracked to show the rise and fall in use of different drugs. Another exception is the CDC's Youth Risk Behavior Surveillance System, an anonymous population survey set up in 1990 to monitor priority health risk behaviors that contribute markedly to the leading causes of death, disability, and social problems among youth and adults in the United States. These behaviors, often established during childhood and early adolescence, include tobacco use, unhealthy dietary behaviors, inadequate physical activity, alcohol and other drug use, sexual behaviors that contribute to unintended pregnancy and sexually transmitted diseases (e.g., HIV infection), and behaviors that contribute to unintentional injuries and violence.

These data can be used to show, for example, a small but steady decline in the proportion of high school students who report currently using alcohol, from 50.8% in 1991 to 34.9% in 2013 (the latest data available). On the other hand, the proportion making a suicidal attempt serious enough to need medical attention has increased from 1.7% in 1991 to 2.7% in 2013 (http://www.cdc.gov/healthyyouth/yrbs/overall.htm). Because the data come from different (anonymous) samples each year, they cannot be used to track an individual's progression. They do, however, provide hints of what legal or cultural changes might be doing to the development of risk behaviors across adolescence.

Other areas in which CDC has begun to involve itself are autism spectrum disorder (ASD), ADHD, and developmental disabilities. However, the CDC does not publish information on the most common childhood mental disorders: anxiety, depression, oppositional defiant disorder or conduct disorder, or many other disorders.

Clearly, a major task for developmental epidemiology in the next decade will be to help the CDC to shoulder the task of charting national rates of child and adolescent psychiatric disorders over time, as it has done for national rates of other serious conditions. Work is currently in progress to integrate brief psychiatric measures into national surveys such as the National Health Interview Survey (NHIS) and the National Health and Nutrition Examination Survey (NHANES-IV). NHANES has recently been using some modules of the DISC and the DISC Predictive Screener, and the NHIS is using a short questionnaire, the Strengths and Difficulties Questionnaire (Goodman et al., 2000), that is also used in several other countries. This will make international comparisons feasible. More work is needed to evaluate the validity of these measures across the age range and to assess their usefulness as a measure of the burden of child mental illness in the community. It is clear, however, that in the next decade it will be possible to stop treating assessment issues as research problems to be funded by federal research agencies and move them over to being issues to be dealt with by surveillance and monitoring agencies such as the CDC. This will have many advantages. First, it will make it possible to treat psychiatric disorders as on a par with other medical conditions. Second, it will produce national data sets, such as those produced by NHIS, which can be used to look at the sorts of questions raised by life course epidemiology: what environmental, political, and social conditions breed psychiatric disorders, and how societies should address these problems. Third, it will relieve the National Institute of Mental Health scientific research budget of the responsibility for basic prevalence studies and free up research dollars for questions on the scientific side of epidemiology, questions about causes and consequences.

Mechanisms: Genetic Epidemiology

In the past decade genetic epidemiology has become a driving force in the epidemiology of child psychiatric disorders. Three recent revolutions in genetic epidemiology will have a tremendous impact on developmental psychopathology in the next decade: behavior genetics, molecular genetics, and epigenetics.

Psychiatric Behavioral Genetics

The first revolution occurred when the methods of psychiatric epidemiology were applied to behavioral genetics. Psychiatric interviews like those described earlier were used in studies with genetically informative designs, such as twin, adoption, family, and migrant studies. For the first time, researchers explored not only the genetics of scalar characteristics, such as intelligence, but also of categorical disorders such as depression, measured in ways that approximate clinical diagnosis. Furthermore, behavioral geneticists began to take seriously issues of sampling so that they could talk about the contribution of genes to disease in the population as a whole, rather than in highly selected families or groups. Some longitudinal studies have also looked at how genes can have different effects at different developmental stages (Eaves & Silberg, 2003; Jacobson, Prescott, & Kendler, 2002).

An example of this approach is the work emerging from the Virginia Twin Study of Adolescent Behavioral Development (VTSABD), a population-based sample of twin pairs followed through adolescence into adulthood. One set of analyses investigated the role of genetic and environmental factors in the association between depressive symptoms and symptoms of overanxious disorder (OAD), simple phobias, and separation anxiety in 8- to 13-year-old and 14- to 17-year-old girls (Silberg, Neale, Rutter, & Eaves, 2001). There were distinct patterns underlying the association between depression and the different anxiety syndromes during the course of development. First, specific genetic influences on depression after age 14 reflected liability to symptoms of earlier OAD and simple phobias. Second, aspects of the shared environment that influenced symptoms of depression before age 14 contributed to symptoms of separation anxiety and simple phobias later in adolescence. Third, the shared environmental influence on depression in older girls also affected liability to symptoms of concurrent OAD and persistent separation anxiety. These results suggest that depression before and after age 14 may be etiologically distinct syndromes. Further analyses including several other longitudinal twin data sets reinforced the view that environmentally driven differences in stable levels of anxiety and depression continue to increase throughout the life cycle (Kendler et al., 2011).

Molecular Genetics

The second genetic revolution occurred when it became feasible to apply the methods of molecular genetics to epidemiologic samples. This development opens up the opportunity to use not only twin or adoption studies but a wide range of samples to test theories about candidate genes for specific symptoms. Even more exciting is the new opportunity to use the treasure-house of data from longitudinal studies to test for gene–environment interactions. Such studies can answer questions about which genes interact with which environmental factors, at what developmental singe. Two examples provide first glimpses of what is likely to be one of the most fruitful areas of developmental epidemiology in the next decade.

Investigators studied the male children from the Dunedin longitudinal study sample (Caspi et al., 2002) from birth to adulthood to determine why some children who are maltreated grow up to develop antisocial behavior whereas others do not. A functional polymorphism in the gene-encoding MAOA was found to moderate the effect of maltreatment. Maltreated children with a genotype conferring high levels of MAOA expression were less likely to develop antisocial problems. These findings provide epidemiological evidence that genotypes can moderate children's sensitivity environmental insults. The finding has since been replied in the VTSABD (Foley et al., 2004).

In other analyses, the Dunedin team investigated why stressful experiences lead to depression in some people but not in others (Caspi et al., 2003). A functional polymorphism in the promoter region of the serotonin transporter (5-HTT) gene was found to moderate the influence of stressful life events on depression. Individuals with one or two copies of the short allele of the 5-HTT promoter polymorphism exhibited more depressive symptoms, diagnosable depression, and suicidality in relation to stressful life events than individuals homozygous for the long allele. Again, an individual's response to environmental insults is moderated by his or her genetic makeup. The publication has aroused considerable controversy, but a meta-analysis of 55 studies found strong evidence to support the finding (Karg, Burmeister, Shedden, & Sen, 2011).

Geneticists have for generations been telling us that genes express themselves only in an environment, and that the key questions have to do with the relationships between the two over time. Developmental epidemiology and genetic epidemiology are already merging, and the process will continue in the next decade.

In the future, no epidemiologic study will fail ask participants to consent to the collection of biomaterial for DNA. In the immediate future, existing studies are going back and obtaining blood or saliva, or asking participants to consent to a new use of already collected biodata. Collaboration between molecular geneticists and epidemiologists with longitudinal data on representative samples offers an amazing opportunity for progress in understanding the biological and social origins of disease.

Epigenetics

Epigenetics is the study of the way the expression of heritable traits is modified by environmental influences or other mechanisms without a change to the DNA sequence, operationally defined as a stably heritable phenotype resulting from changes in a chromosome without alterations in the DNA sequence (Berger, Kouzarides, Shiekhattar, & Shilatifard, 2009).

Epigenetics can be divided into predetermined and probabilistic epigenesis. Predetermined epigenesis is a unidirectional, scripted, and predictable movement from structural development in DNA to the functional maturation of the protein. Probabilistic epigenesis, on the other hand, is a bidirectional structure-function development with experiences and external molding development. Most of what psychiatric researchers are interested in involves probabilistic epigenesis (Gottlieb, 1991a; Gottlieb

& Willoughby, 2006). A recent methylome-wide association study of blood biomarkers of environmental insults in schizophrenic patients and controls (Aberg et al., 2014) demonstrates that methylation studies can capture signatures of environmental insults even in peripheral tissues. Longitudinal epidemiologic studies with repeated blood samples will find a new use, testing for changes in the methylome associated with identified events.

SUMMARY: TRANSLATIONAL EPIDEMIOLOGY

This chapter has covered a lot of ground: from the first stirrings of understanding about childhood psychiatric disorders to the possibility of using epigenetics to identify gene–environment interactions that can generate psychiatric disorder. There are fuzzy boundaries between developmental epidemiology and developmental psychopathology, life-course epidemiology, genetic epidemiology, services research, and several other areas. Turning this fuzziness into a strength, we suggest adopting the language of *translational epidemiology* (Khoury, Gwinn, & Ioannidis, 2010; Weissman, Brown, & Talati, 2011). Translational science aims to move from bench to bedside, but translational epidemiology looks further, aiming to combine what is learned at the bench and bedside with the results of community-based studies and public health interventions (Khoury et al., 2010).

There is another dimension that current accounts of translational epidemiology omit: development. We have only to look at the age curve of alcohol dependence as an example to see that translation from mechanisms to interventions will be impeded if the effects of development are ignored. In this instance, conduct disorder is a strong predictor of early onset alcohol abuse or dependence only until age 15. Once alcohol misuse becomes developmentally normal, conduct disorder is no longer predictive (Sung, Erkanli, Angold, & Costello, 2004). This developmental pattern has implications not only for bench (molecular genetics) and bedside (high-risk interventions) but also for public policy. Figure 3.1 sketches just some of the ways developmental epidemiology, of which longitudinal community studies are an example, can grow translations across many branches of science.

REFERENCES

Aberg, K. A., McClay, J. L., Nerella, S., Clark, S., Kumar, G., Chen, W. . . . , Gao, G. (2014). Methylome-wide association study of schizophrenia: Identifying blood biomarker signatures of environmental insults. *JAMA Psychiatry*, *71*(3): 255–264.

Achenbach, T. M. (1985). *Assessment and taxonomy of child and adolescent psychopathology*. Beverly Hills, CA: Sage Publications.

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist 4-18 and 1991 Profile*. Burlington, VT: University of Vermont Department of Psychiatry.

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*, 213–232.

Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA Preschool Forms and Profiles: An integrated system of multi-informant assessment*. Burlington, VT: University of Vermont Department of Psychiatry.

Achenbach, T. N., & Edelbrock, C. S. (1983). *Manual for the Child Behavior Checklist and Revised Profile*. Burlington, VT: University of Vermont.

Ambrosini, P. J. (2000). Historical development and present status of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS). *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 49–58.

Angoff, W. H. (1988). The nature-nurture debate, aptitudes, and group differences. *American Psychologist*, *43*(9), 713–720.

Angold, A. (1988). Childhood and adolescent depression II: Research in clinical populations. *British Journal of Psychiatry*, *153*, 476–492.

Angold, A. (2002). Diagnostic interviews with parents and children. In M. Rutter & E. Taylor (Eds.), *Child and adolescent psychiatry: Modern approaches* (4th ed., pp. 32–51). Oxford, UK: Blackwell Scientific Publications.

Angold, A. (2003). Adolescent depression, cortisol and DHEA. *Psychological Medicine*, *33*, 573–581.

Angold, A., & Costello, E. J. (1991). Developing a developmental epidemiology. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium on developmental psychology* (Vol. 3, pp. 75–96). Hillsdale, NJ: Laurence Earlbaum Associates.

Angold, A., & Costello, E. J. (1995). A test–retest reliability study of child-reported psychiatric symptoms and diagnoses using the Child and Adolescent Psychiatric Assessment (CAPA-C). *Psychological Medicine*, *25*, 755–762.

Angold, A., & Costello, E. J. (2000). The Child and Adolescent Psychiatric Assessment (CAPA). *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 39–48.

Angold, A., Costello, E. J., Erkanli, A., & Worthman, C. M. (1999). Pubertal changes in hormone levels and depression in girls. *Psychological Medicine*, *29*(5), 1043–1053.

Angold, A., Erkanli, A., Copeland, W. E., Goodman, R., Fisher, P. W., & Costello, E. J. (2012). Psychiatric diagnostic interviews for children and adolescents: A comparative study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(5), 506–517.

Angold, A., & Worthman, C. W. (1993). Puberty onset of gender differences in rates of depression: A developmental, epidemiologic and neuroendocrine perspective. *Journal of Affective Disorders*, *29*, 145–158.

Bale, T. L., Baram, T. Z., Brown, A. S., Goldstein, J. M., Insel, T. R., McCarthy, M. M., . . . Nestler, E. J. (2010). Early life programming and neurodevelopmental disorders. *Biological Psychiatry*, *68*(4), 314–319. doi: 10.1016/j.biopsych.2010.05.028

Barker, D. J. (1998). *Mothers, babies, and health in later life*. Edinburgh, UK: Churchill Livingstone.

Ben-Shlomo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and

interdisciplinary perspectives. *International Journal of Epidemiology*, *31*, 285–293.

Benson, K., & Hartz, A. (2000). A comparison of observational studies and randomized, controlled trials. *New England Journal of Medicine*, *342*, 1878–1886.

Berger, S. L., Kouzarides, T., Shiekhattar, R., & Shilatifard, A. (2009). An operational definition of epigenetics. *Genes & Development*, *23*(7), 781–783.

Berkson, J. (1946). Limitations of the application of fourfold table analysis to hospital data. *Biometrics Bulletin*, *2*, 47–52.

Black, J. E., & Greenough, W. T. (1986). Induction of pattern in neural structure by experience: Implications for cognitive development. In M. E. Lamb, A. Brown, & B. Rogoff (Eds.), *Advances in developmental psychology* (Vol. 4, pp. 1–50). Hillsdale, NJ: Erlbaum.

Breslow, N. E., & Day, N. E. (1980). *Statistical methods in cancer research: Vol. I. The analysis of case control studies*. Lyon: WHO International Agency for Research on Cancer.

Breslow, N. E., & Day, N. E. (1987). Statistical methods in cancer research: Vol. II. The design and analysis of cohort studies. Lyon: WHO International Agency for Research on Cancer.

Breuer, J., & Freud, S. (1895). *Studies on hysteria*. London: Hogarth Press.

Broidy, L. M., Nagin, D. S., Tremblay, R. E., Bates, J. E., Brame, B., Dodge, K. A., . . . Laird, R. (2003). Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: A six-site, cross-national study. *Developmental Psychology*, *39*(2), 222.

Brown, A., van Os, J., Driessens, C., Hoek, H. W., & Susser, E. (2000). Further evidence of relation between prenatal famine and major affective disorder. *American Journal of Psychiatry*, *157*(2), 190–195.

Brown, G. W., & Harris, T. O. (1978). *The social origins of depression: A study of psychiatric disorder in women*. New York. NY: Free Press.

Burns, B. J., Costello, E. J., Angold, A., Tweed, D., Stangl, D., Farmer, E. M. Z., & Erkanli, A. (1995). Children's mental health service use across service sectors. *Health Affairs*, *14*, 147–159.

Cacioppo, J. T., & Tassinary, L. G. (1991). Inferring psychological significance from physiological signals. *American Psychologist*, *45*, 16–26.

Cairns, R. B. (1991). Multiple metaphors for a singular idea. *Developmental Psychopathology*, *27*, 23–26.

Cairns, R. B., & Cairns, B. D. (1991). Social cognition and social networks: A developmental perspective. In D. J. Pepler & K. H. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 249–278). Hillsdale, NJ: Lawrence Erlbaum Associates, Publishers.

Cairns, R. B., Cairns, B. D., Rodkin, P., & Xie, H. (1998). New directions in developmental research: Models and methods. In R. Jessor (Ed.), *New perspectives on adolescent risk behavior* (pp. 13–40). London, UK: Cambridge University Press.

Cairns, R. B., Elder, G. H., & Costello, E. J. (1996). The making of developmental science. In R. Cairns, G. Elder, & C. E. J. (Eds.), *Developmental science* (Vol. 20, pp. 223–234). New York, NY: Cambridge University Press.

Cairns, R. B., Gariépy, J. L., & Hood, K. E. (1990). Development, microevolution, and social behavior. *Psychological Review*, *97*(1), 49–65.

Canino, G., Costello, E. J., & Angold, A. (1999). Assessing functional impairment and social adaptation for child mental health services research: A review of measures. *Journal of Mental Health Services Research*, *1*, 93–108.

Cantwell, D. P., & Baker, L. (1988). Issues in the classification of child and adolescent psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry*, *27*(5), 521–533.

Caspi, A., Harrington, H., Moffitt, T. E., Milne, B. J., & Poulton, R. (2006). Socially isolated children 20 years later: Risk of cardiovascular disease. *Archives of Pediatrics & Adolescent Medicine*, *160*(8), 805.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., . . . Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 851–854.

Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry*, *53*(11), 1033–1039.

Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I., Harrington, H., . . . Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 386–389.

Chambers, W. J., Puig-Antich, J., Hirsch, M., Paez, P., Ambrosini, P. J., Tabrizi, M. A., & Davies, M. (1985). The assessment of affective disorders in children and adolescents by semistructured interview: Test–retest reliability of the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present Episode Version. *Archives of General Psychiatry*, *42*, 696–702.

Chasiotis, A., Scheffer, D., Restemeier, R., & Keller, H. (1998). Intergenerational context discontinuity affects the onset of puberty: A comparison of parent–child dyads in West and East Germany. *Human Nature*, *9*(3), 321–339.

Chess, S., & Habibi, M. (1978). *Principles and practice of child psychiatry*. New York, NY: Plenum.

Cicchetti, D. (1990a). An historical perspective on the discipline of developmental psychopathology. In J. Rolf, A. Masten, D. Cicchetti, K. Neuchterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 1–28). New York, NY: Cambridge University Press.

Cicchetti, D. (1990b). Perspectives on the interface between normal and atypical development. *Developmental Psychopathology*, *2*, 329–333.

Cicchetti, D., & Schneider-Rosen, K. (1984). Toward a developmental model of the depressive disorders. *New Directions for Child Development*, *26*, 5–27.

Cicchetti, D., & Schneider-Rosen, K. (1986). An organizational approach to childhood depression. In M. Rutter, C. E. Izard, & P. B. Read (Eds.), *Depression in young people: Developmental and clinical perspectives* (pp. 71–134). New York, NY: Guilford Press.

Cicchetti, D., & Toth, S. L. (1992). The role of developmental theory in prevention and intervention. *Developmental Psychopathology*, *4*, 489–493.

Clark, M. S., Sexton, T. J., McClain, M., Root, D., Kohen, R., & Neumaier, J. F. (2002). Overexpression of 5-HT1B receptor in dorsal raphe nucleus using herpes simplex virus gene transfer increases anxiety behavior after inescapable stress. *Journal of Neuroscience*, *22*(11), 4550–4562.

Cohen, J. (1990). Things I have learned (so far). *American Psychologist*, *45*, 1304–1312.

Cohen, J. (1996). Childhood risks for young adult symptoms of personality disorder: Method and substance. *Multivariate Behavioral Research*, *21*, 121–148.

Collins, P. Y., Patel, V., Joestl, S. S., March, D., Insel, T. R., Daar, A. S., . . . Walport, M. (2011). Grand challenges in global mental health. *Nature*, *475*(7354), 27–30.

Copeland, W., Shanahan, L., Costello, E. J., & Angold, A. (2011). Cumulative prevalence of psychiatric disorders by young adulthood: A prospective cohort analysis from the Great Smoky Mountains Study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*(3), 252–261. doi: 10.1016/j.jaac.2010.12.014

Costello, A. J., Edelbrock, C., Kalas, R., Kessler, M. D., & Klaric, S. H. (1982). *The National Institute of Mental Health Diagnostic Interview*

Schedule for Children (DISC). Rockville, MD: National Institute of Mental Health.

Costello, E. J., Burns, B. J., Angold, A., & Leaf, P. J. (1993). How can epidemiology improve mental health services for children and adolescents? *Journal of the American Academy of Child and Adolescent Psychiatry*, *32*(6), 1106–1113.

Costello, E. J., Compton, S. N., Keeler, G., & Angold, A. (2003). Relationships between poverty and psychopathology: A natural experiment. *JAMA*, *290*(15), 2023–2029.

Costello, E. J., & Janiszewski, S. (1990). Who gets treated? Factors associated with referral in children with psychiatric disorders. *Acta Psychiatrica Scandinavica*, *81*, 523–529.

Costello, E. J., Loeber, R., & Stouthamer-Loeber, M. (1991). Pervasive and situational hyperactivity—Confounding effect of informant: A research note. *Journal of Child Psychology and Psychiatry*, *32*, 367–376.

Costello, E. J., Pescosolido, B. A., Angold, A., & Burns, B. J. (1998). A family network-based model of access to child mental health services. In J. Morrissey (Ed.), *Research in community mental health* (Vol. 9, pp. 165–190). Greenwich, CT: JAI.

Costello, E. J., Pine, D. S., Hammen, C., March, J. S., Plotsky, P. M., Weissman, M. M., ... Leckman, J. F. (2002). Development and natural history of mood disorders. *Biological Psychiatry*, *52*, 529–542.

Cullen, K. J., & Boundy, C. A. P. (1966). The prevalence of behavior disorders in the children of 1000 Western Australian families. *Medical Journal of Australia*, *2*, 805–808.

Cummings, J. D. (1944). The incidence of emotional symptoms in school children. *British Journal of Educational Psychology*, *14*, 151–161.

Dare, C. (1985). Psychoanalytic theories of development. In M. Rutter & L. Hersov (Eds.), *Child and adolescent psychiatry: Modern approaches* (2nd ed., pp. 204–215). Oxford, UK: Blackwell Scientific Publications.

Davey Smith, G., Gunnell, D., & Ben-Shlomo, Y. (2000). Life-course approaches to socio-economic differentials in cause specific adult mortality. In D. Leon & G. Walt (Eds.), *Poverty, inequality and health: An international perspective* (pp. 88–124). Oxford, UK: Oxford University Press.

Dawber, T. R. (1980). *The Framingham study: The epidemiology of coronary heart disease*. Cambridge, MA: Harvard University Press.

Demyttenaere, K., Bruffaerts, R., Posada-Villa, J., Gasquet, I., Kovess, V., Lepine, J. P., ... Morosini, P. (2004). Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health Surveys. *JAMA*, *291*(21), 2581–2590.

Digdon, N., & Gotlib, I. H. (1985). Developmental considerations in the study of childhood depression. *Developmental Review*, *5*, 162–199.

Dodge, K. (2001). The science of youth violence prevention: Progressing from developmental epidemiology to efficacy to effectiveness to public policy. *American Journal of Preventive Medicine*, *20*(Suppl 1), 63–70.

Dodge, K. A., Pettit, G. S., & Bates, J. E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development*, *65*, 649–665.

Doll, R., & Peto, R. (1976). Mortality in relation to smoking: 20 years' observations on male British doctors. *British Medical Journal*, *2*, 1525–1536.

Down, J. (1867). Observations on an ethnic classification of idiots. *Journal of Mental Science*, *13*, 121–123.

Dulcan, M. K., Costello, E. J., Costello, A. J., Edelbrock, C., Brent, D., & Janiszewski, S. (1990). The pediatrician as gatekeeper to mental health care for children: Do parents' concerns open the gate? *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*(3), 453–458.

Earls, F. (1979). Epidemiology and child psychiatry: Historical and conceptual development. *Comprehensive Psychiatry*, *20*(3), 256–269.

Earls, F. (1980). Prevalence of behavior problems in 3-year-old children: A cross-national replication. *Archives of General Psychiatry*, *37*, 1153–1157.

Eaves, L. J., & Silberg, J. L. (2003). Modulation of gene expression by genetic and environmental heterogeneity in the timing of a developmental milestone. *Behavior Genetics*, *33*(1), 1–6.

Eisenberg, L. (1977). Development as a unifying concept in psychiatry. *British Journal of Psychiatry*, *131*, 225–237.

Ernst, M., Cookus, B. A., & Moravec, B. C. (2000). Pictorial instrument for children and adolescents (PICA-III-R). *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 94–99.

Farrington, D. P. (1983). Offending from 10 to 25 years of age. In K. T. VanDusen & S. A. Mednick (Eds.), *Prospective studies of crime and delinquency* (pp. 17–37). Boston, MA: Kluwer-Nijhoff.

Fergusson, D., & Woodward, L. (2000). Educational, psychosocial, and sexual outcomes of girls with conduct problems in early adolescence. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *41*(6), 779–792.

Flisher, A. J., Kramer, R. A., Hoven, C. W., Greenwald, S., Alegria, M., Bird, H. R., ... Moore, R. E. (1997). Psychosocial characteristics of physically abused children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 123–131.

Foley, D., Rutter, M., Pickles, A., Angold, A., Maes, H., Silberg, J., & Eaves, L. (2004). Informant disagreement for separation anxiety disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(4), 452–260.

Foley, D., Rutter, M. L., Angold, A., Pickles, A., Silberg, J. L., & Eaves, L. J. (2005). Making sense of informant disagreement for overanxious disorder. *Anxiety Disorders*, *19*, 193–210.

Foley, D. L., Pickles, A., Rutter, M., Gardner, C. O., Maes, H. H., Silberg, J. L., ... Eaves, L. J. (2004). Risks for conduct disorder symptoms associated with parental alcoholism in step-father families versus intact families from a community sample. *Journal of Child Psychology and Psychiatry*, *45*(4), 687–696.

Fréchette, M., & Le Blanc, M. (1987). *Délinquences et délinquants*. Chicoutimi, Québec: Gaetan Morin.

Freedman, D. X. (1984). Psychiatric epidemiology counts. *Archives of General Psychiatry*, *41*, 931–933.

Freud, A. (1965). *Normality and pathology in childhood*. New York, NY: International Universities Press.

Garmezy, N. (1988). Longitudinal strategies, causal reasoning and risk research: A commentary. In M. Rutter (Ed.), *Studies of psychosocial risk* (pp. 29–44). Cambridge, UK: Cambridge University Press.

Ge, X., Brody, G., Conger, R., & Murry, V. (2002). Contextual amplification of pubertal transition effects on deviant peer affiliation and externalizing behavior among African American children. *Developmental Psychology*, *38*(1), 42–54.

Ge, X., Conger, R. D., & Elder, G. H. (1996). Coming of age too early: Pubertal influences on girls' vulnerability to psychological distress. *Child Development*, *67*(6), 3386–3400.

Goodman, A., Heiervang, E., Fleitlich-Bilyk, B., Alyahri, A., Patel, V., Mullick, M. S., ... Goodman, R. (2012). Cross-national differences in questionnaires do not necessarily reflect comparable differences in disorder prevalence. *Social Psychiatry and Psychiatric Epidemiology*, *47*(8), 1321–1331.

Goodman, R., Ford, R., Simmons, H., Gatward, R., & Meltzer, H. (2000). Using the Strength and Difficulties Questionnaire (SDQ) to screen for child psychiatric disorders in a community sample. *British Journal of Psychiatry*, *177*, 534–539.

Gottlieb, G. (1991a). Epigenetic systems view of human development. *Developmental Psychopathology*, *27*, 33–34.

Gottlieb, G. (1991b). Experiential canalization of behavioral development: Theory. *Developmental Psychopathology*, *27*(1), 4–13.

Gottlieb, G., & Willoughby, M. (2006). Probabilistic epigenesis of psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Theory and method* (2nd ed., Vol. 1, pp. 673–700). Hoboken, NJ: John Wiley & Sons, Inc.

Gould, M., Greenberg, T., Velting, D., & Shaffer, D. (2003). Youth suicide risk and preventive interventions: A review of the past 10 years. *Journal of the American Academy Child and Adolescent Psychiatry*, *42*, 386–405.

Gould, M. S., Cross, W., Pisani, A. R., Munfakh, J. L., & Kleinman, M. (2013). Impact of applied suicide intervention skills training on the national suicide prevention lifeline. *Suicide and Life-Threatening Behavior*, *43*(6), 676–691.

Gould, M. S., Wunsch-Hitzig, R., & Dohrenwend, B. P. (1980). Formulation of hypotheses about the prevalence, treatment and prognostic significance of psychiatric disorders in children in the United States. In B. P. Dohrenwend (Ed.), *Mental illness in the United States: Epidemiological estimates* (pp. 9–44). New York, NY: Praeger.

Granger, D. A., Serbin, L. A., Schwartzman, A., Lehoux, P., Cooperman, J., & Ikeda, S. (1998). Children's salivary cortisol, internalising behaviour problems, and family environment: Results from the Concordia Longitudinal Risk Project. *International Journal of Behavioral Development*, *22*, 707–728.

Greenberg, M. T., Speltz, M. T., & DeKlyen, M. (1993). The role of attachment in the early development of disruptive behavior problems. *Development and Psychopathology*, *5*, 191–213.

Greenough, W. T. (1991). Experience as a component of normal development: Evolutionary considerations. *Developmental Psychopathology*, *27*(1), 14–17.

Griffiths, W. (1952). *Behavior difficulties of children as perceived and judged by parents, teachers, and children themselves*. Minneapolis, MN: University of Minnesota Press.

Grob, G. N. (1985). The origins of American psychiatric epidemiology. *American Journal of Public Health*, *75*(3), 229–236.

Group, C. P. P. R. (2010). Fast Track intervention effects on youth arrests and delinquency. *Journal of Experimental Criminology*, *6*(2), 131–157.

Grunbaum, A. (1977). Is psychoanalysis a pseudoscience? Karl Popper versus Sigmund Freud. *Zeitschrift fur Philosophische Forschung*, *31*, 333–353.

Haggerty, M. E. (1925). The incidence of undesirable behavior in public school children. *Journal of Educational Research*, *12*(2), 102–122.

Hay, D. F. (1985). Learning to form relationships in infancy: Parallel attainments with parents and peers. *Developmental Review*, *5*, 122–161.

Hay, D. F., & Angold, A. (1993). Introduction: Precursors and causes in development and pathogenesis. In D. F. Hay & A. Angold (Eds.), *Precursors and causes in development and psychopathology* (pp. 1–21). Chichester, UK: John Wiley and Sons.

Heiervang, E., Goodman, A., & Goodman, R. (2008). The Nordic advantage in child mental health: Separating health differences from reporting style in a cross-cultural comparison of psychopathology. *Journal of Child Psychology and Psychiatry*, *49*(6), 678–685.

Henggeler, S. W., Melton, G. B., Brondino, M. J., Scherer, D. G., & Hanley, J. R. (1997). Multisystemic therapy with violent and chronic juvenile offenders and their families: The role of treatment fidelity in successful dissemination. *Journal of Consulting and Clinical Psychology*, *65*(5), 821–833.

Herjanic, B., & Campbell, W. (1977). Differentiating psychiatrically disturbed children on the basis of a structured interview. *Journal of Abnormal Child Psychology*, *5*, 127–134.

Hernán, M. A., Hernández-Díaz, S., & Robins, J. M. (2013). Randomized trials analyzed as observational studies. *Annals of Internal Medicine*, *159*(8), 560–562.

Ho, M. W. (1984). Environment and heredity in development and evolution. In M. W. Ho & P. T. Saunders (Eds.), *Beyond neo-Darwinism: An introduction to the new evolutionary paradigm* (pp. 267–289). San Diego, CA: Academic Press.

Hodges, K. (1993). Structured interviews for assessing children. *Journal of Child Psychology and Psychiatry*, *34*,1, 49–68.

Hodges, K., McKnew, D., Cytryn, L., Stern, L., & Kline, J. (1982). The Child Assessment Schedule (CAS) Diagnostic Interview: A report on reliability and validity. *Journal of the American Academy of Child Psychiatry*, *21*(5), 468–473.

Howe, G. R. (1982). Epidemiology of radiogenic breast cancer. In J. Boice & J. J. R. Fraumeni (Eds.), *Radiation carcinogenesis: Epidemiology and biological significance* (pp. 119–129). New York, NY: Raven Press.

Ialongo, N., Edelsohn, G., Werthamer-Larsson, L., Crockett, L., & Kellam, S. (1993). Are self-reported depressive symptoms in first-grade children developmentally transient phenomena? A further look. *Development and Psychopathology*, *5*, 433–457.

Insel, T. R., & Fenton, W. S. (2005). Psychiatric epidemiology: It's not just about counting anymore. *Archives of General Psychiatry*, *62*(6), 590–592.

Ireland, W. W. (1877). *On idiocy and imbecility*. London, UK: Churchill.

Ivanova, M. Y., Achenbach, T. M., Dumenci, L., Rescorla, L. A., Almqvist, F., Weintraub, S., ... Dobrean, A. (2007). Testing the 8-syndrome structure of the child behavior checklist in 30 societies. *Journal of Clinical Child and Adolescent Psychology*, *36*(3), 405–417.

Jablensky, A. (1986). Epidemiological surveys of mental health of geographically defined populations in Europe. In M. M. Weissman, J. K. Myers, & C. E. Ross (Eds.), *Community surveys of psychiatric disorders* (pp. 257–313). New Brunswick, NJ: Rutgers University Press.

Jacobson, K. C., Prescott, C. A., & Kendler, K. S. (2002). Sex differences in the genetic and environmental influences on the development of antisocial behavior. *14*(2), 395–416.

Jaffee, S. R., Harrington, H., Cohen, P., & Moffitt, T. E. (2005). Cumulative prevalence of psychiatric disorder in youths. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*(5), 406–407.

Jaffee, S. R., Moffitt, T. E., Caspi, A., Fombonne, E., Poulton, R., & Martin, J. (2002). Differences in early childhood risk factors for juvenile-onset and adult-onset depression [see comment]. *Archives of General Psychiatry*, *59*(3), 215–222.

Jollos, V. (1934). Inherited changes produced by heat treatment in Drosophila melanogaster. *Genetics*, *16*, 476–494.

Jones, D., Godwin, J., Dodge, K. A., Bierman, K. L., Coie, J. D., Greenberg, M. T., ... Pinderhughes, E. E. (2010). Impact of the Fast Track prevention program on health services use by conduct-problem youth. *Pediatrics*, *125*(1), e130–e136.

Kandel, D. B., & Davies, M. (1982). Epidemiology of depressive mood in adolescents: An empirical study. *Archives of General Psychiatry*, *39*(10), 1205–1212.

Kanner, L. (1945). *Child psychiatry*. Springfield, IL: C.C. Thomas.

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, *68*(5), 444.

Kaufman, J. S., & Cooper, R. S. (1999). Seeking causal explanations in social epidemiology. *American Journal of Epidemiology*, *150*, 113–128.

Kellam, S. G., Ensminger, M. E., & Turner, R. J. (1977). Family structure and the mental health of children. *Archives of General Psychiatry*, *34*(9), 1012–1022.

Kellam, S. G., Koretz, D., & Moscicki, E. (1999). Core elements of developmental epidemiologically based prevention research. *American Journal of Community Psychology*, *27*(4), 463–482.

Kellam, S. G., Rebok, G. W., Mayer, L. S., Ialongo, N., & Kalodner, C. R. (1994). Depressive symptoms over first grade and their response to a developmental epidemiologically based preventive trial aimed at improving achievement. *Development and Psychopathology*, *6*, 463–481.

Kendell, R. E. (1976). The classification of depressions: A review of contemporary confusion. *British Journal of Psychiatry*, *129*, 15–28.

Kendler, K. S., Eaves, L. J., Loken, E. K., Pedersen, N. L., Middeldorp, C. M., Reynolds, C., … Gardner, C. O. (2011). The impact of environmental experiences on symptoms of anxiety and depression across the life span. *Psychological Science*, *22*(10), 1343–1352.

Kendrick, T. (1999). Primary care options to prevent mental illness. *Annals of Medicine*, *31*(6), 359–363.

Kessler, R. C., Angermeyer, M., Anthony, J. C., de Graaf, R., Demyttenaere, K., Gasquet, I., … Haro, J. M. (2007). Lifetime prevalence and age-of-onset distributions of mental disorders in the World Health Organization's World Mental Health Survey Initiative. *World Psychiatry*, *6*(3), 168.

Kessler, R. C., & Üstün, T. B. (2004). The world mental health (WMH) survey initiative version of the world health organization (WHO) composite international diagnostic interview (CIDI). *International Journal of Methods in Psychiatric Research*, *13*(2), 93–121.

Khoury, M. J., Gwinn, M., & Ioannidis, J. P. (2010). The emergence of translational epidemiology: From scientific discovery to population health impact. *American Journal of Epidemiology*, *172*(5), 517–524.

Kim-Cohen, J., Caspi, A., Moffitt, T. E., Harrington, H., Milne, B. J., & Poulton, R. (2003). Prior juvenile diagnoses in adults with mental disorder: Developmental follow-back of a prospective-longitudinal cohort. *Archives of General Psychiatry*, *60*(7), 709–717.

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, M. D., Craig, I. W., & Moffitt, T. (2006). MAOA, maltreatment, and gene–environment interaction predicting children's mental health: New evidence and a meta-analysis. *Molecular Psychiatry*, *11*, 903–913.

Kleinbaum, D. G., Kupper, L. L., & Morgenstern, H. (1982). *Epidemiologic research: Principles and quantitative methods*. New York, NY: Van Nostrand Reinhold.

Krieger, N. (2001). Theories for social epidemiology in the 21st century: An ecosocial perspective. *International Journal of Epidemiology*, *30*, 668–677.

Krueger, R., Caspi, A., Moffitt, T., & Silva, P. (1998). The structure and stability of common mental disorders (*DSM-III-R*): A longitudinal–epidemiological study. *Journal of Abnormal Psychology*, *107*(2), 216–227.

Kuh, D. L., & Ben-Shlomo, Y. (1997). *A life course approach to chronic disease epidemiology*. Oxford, UK: Oxford University Press.

Ladouceur, C. D., Peper, J. S., Crone, E. A., & Dahl, R. E. (2012). White matter development in adolescence: The influence of puberty and implications for affective disorders. *Developmental Cognitive Neuroscience*, *2*(1), 36–54.

Land, K. C., & Nagin, D. S. (1996). Micro-models of criminal careers: A synthesis of the criminal careers and life course approaches via semiparametric mixed Poisson regression models, with empirical applications. *Journal of Quantitative Criminology*, *12*(2), 163–191.

Lapouse, R. (1966). The epidemiology of behavior disorders in children. *American Journal of Dysfunctional Children*, *111*, 594–599.

Lapouse, R. L., & Monk, M. A. (1958). An epidemiologic study of behavior characteristics in children. *American Journal of Public Health*, *48*(9), 1134–1144.

Lapouse, R. L., & Monk, M. A. (1964). Behavior deviations in a representative sample of children. *American Journal of Orthopsychiatry*, *34*, 436–447.

Le Blanc, M., & Fréchette, M. (1989). *Male criminal activity from childhood through youth: Multilevel and developmental perspectives*. New York, NY: Springer-Verlag.

Leon, D. A. (2000). Common threads: Underlying components of inequalities in mortality between and within countries. In D. Leon & G. Walt (Eds.), *Poverty, inequality and health: An international perspective* (pp. 58–87). Oxford: Oxford University Press.

Leslie, D. L., Rosenheck, R. A., & Horwitz, S. M. (2001). Patterns of mental health utilization and costs among children in a privately insured population. *Health Services Research*, *36*(1 Pt 1), 113–127.

Lewis, M. (1990). Challenges to the study of developmental psychopathology. In M. Lewis & S. M. Miller (Eds.), *Handbook of developmental psychopathology* (pp. 29–40). New York, NY: Plenum Press.

Lilienfeld, A. M., & Lilienfeld, D. E. (1980). *Foundations of epidemiology* (2 ed.). New York, NY: Oxford University Press.

Lipsey, M. W., & Wilson, D. B. (1998). Effective intervention for serious juvenile offenders: A synthesis of research. In R. Loeber & D. P. Farrington (Eds.), *Serious and violent juvenile offenders* (pp. 313–345). Thousand Oaks, CA: Sage Publications.

Loeber, R., & Baicker-McKee, C. (1990). *The changing manifestations of disruptive/antisocial behavior from childhood to early adulthood: Evolution or tautology?* Unpublished manuscript.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., Moffitt, T. E., & Caspi, A. (1998). The development of male offending: Key findings from the first decade of the Pittsburgh Youth Study. *Studies on Crime and Crime Prevention*, *7*, 141–171.

Loeber, R., & Homish, D. L. (2005). Homicide, violence, and developmental trajectories. In R. E. Tremblay & W. W. Hartup (Eds.), *Developmental origins of aggression* (pp. 202–220). New York, NY: Guilford.

Loeber, R., Keenan, K., & Zhang, Q. (1997). Boys' experimentation and persistence in developmental pathways toward serious delinquency. *Journal of Child and Family Studies*, *6*(3), 321–357.

Loeber, R., & Le Blanc, M. (1990). Toward a developmental criminology. In M. Tonry & N. Morris (Eds.), *Crime and justice: An annual review of research* (Vol.12, pp. 375–473). Chicago, IL: University of Chicago Press.

Loeber, R., & Stouthamer-Loeber, M. (1997). The development of juvenile aggression and violence: Some common misconceptions and controversies. *American Psychologist*, *53*(2), 242–259.

Long, A. (1941). Parents' reports of undesirable behavior in children. *Child Development*, *12*, 43–62.

Lozano, R., Naghavi, M., Foreman, K., Lim, S., Shibuya, K., Aboyans, V., … Murray, C. J. L. (2012). Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: A systematic analysis for the Global Burden of Disease Study 2010. *Lancet*, *380*(9859), 2095–2128.

Lynam, D. R., Milich, R., Zimmerman, R., Novak, S. P., Logan, T. K., Martin, C., … Clayton, R. (1999). Project DARE: No effects at 10-year follow-up. *Journal of Consulting & Clinical Psychology*, *67*(4), 590–593.

Lyons-Ruth, K. (1996). Attachment relationship among children with aggressive behavior problems: The role of disorganized early attachment patterns. *Journal of Consulting and Clinical Psychology*, *64*, 64–73.

Macfarlane, J. W., Allen, L., & Honzik, M. P. (1954). *University of California publications in child development* (Vol. 2). Berkeley, CA: University of California Press.

Magnusson, D., Stattin, H., & Allen, V. L. (1985). Biological maturation and social development: A longitudinal study of some adjustment processes from mid-adolescence to adulthood. *Journal of Youth and Adolescence*, *14*, 267–283.

Mahoney, J. L., & Stattin, H. (2000). Leisure activities and adolescent antisocial behavior: The role of structure and social context. *Journal of Adolescence*, *23*(2), 113–127.

Marshall, W. A., & Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood*, *44*, 291–303.

Maudsley, H. (1868). *The physiology and pathology of mind* (2nd ed.). London, UK: MacMillan.

Mayr, R. (1982). *The growth of biological thought: Diversity, evolution and intelligence*. Cambridge, MA: Harvard University Press.

Mays, C. W., & Spiess, H. (1984). Bone sarcomas in patients given radium-224. In J. Boice & J. F. Fraumeni (Eds.), *Radiation carcinogenesis: Epidemiology and biological significance* (pp. 241–252). New York, NY: Raven Press.

McFie, B. S. (1934). Behavior and personality difficulties in school children. *British Journal of Educational Psychology*, *4*, 34.

McGue, M. (1989). Nature–nurture and intelligence. *Nature*, *340*, 507–508.

Meehl, P. E. (1954). *Clinical versus statistical prediction: A theoretical analysis and review of the evidence*. Minneapolis, MN: University of Minnesota Press.

Meehl, P. E. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality*, *60*, 117–174.

Miettinen, O. S. (1974). Confounding and effect modification. *American Journal of Epidemiology*, *100*, 350–353.

Mineka, S., Gunnar, M., & Champoux, M. (1986). Control and early socioemotional development: Infant rhesus monkeys reared in controllable versus uncontrollable environments. *Child Development*, *57*, 1241–1256.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, *100*, 674–701.

Moffitt, T. E., Caspi, A., Belsky, J., & Silva, P. A. (1992). Childhood experience and the onset of menarche: A test of a sociobiological model. *Child Development*, *63*(1), 47–58.

Moffitt, T. E., Harrington, H., Caspi, A., Kim-Cohen, J., Goldberg, D., Gregory, A. M., & Poulton, R. (2007). Depression and generalized anxiety disorder: Cumulative and sequential comorbidity in a birth cohort followed prospectively to age 32 years. *Archives of General Psychiatry*, *64*(6), 651.

Murray, C. J. L., & Lopez, A. D. (1996). *The global burden of disease* (Vol. 1). Geneva, Switzerland: World Health Organization.

Mustillo, S., Worthman, C., Erkanli, A., Keeler, G., Angold, A., & Costello, E. J. (2003). Obesity and psychiatric disorder: Developmental trajectories. *Pediatrics*, *111*, 851–859.

Muthen, B., & Muthen, L. K. (2000). Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcoholism: Clinical & Experimental Research*, *24*(6), 882–891.

Nagel, E. (1957). Determinism and development. In D. B. Harris (Ed.), *The concept of development* (pp. 15–26). Minneapolis, MN: University of Minnesota Press.

Nagin, D. S., & Tremblay, R. E. (2001). Parental and early childhood predictors of persistent physical aggression in boys from kindergarten to high school. *Archives of General Psychiatry*, *58*, 389–394.

Needleman, H. L., & Bellinger, D. (1991). The health effects of low level exposure to lead. *Annual Review of Public Health*, *12*, 111–140.

Odgers, C. L., Milne, B. J., Caspi, A., Crump, R., Poulton, R., & Moffitt, T. E. (2007). Predicting prognosis for the conduct-problem boy: Can family history help? *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*(10), 1240–1249.

Offord, D. R., Boyle, M. H., Racine, Y. A., Fleming, J. E., Cadman, D. T., Blum, H. M. (1992). Outcome, prognosis, and risk in a longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*(5), 916–923.

Olson, W. C. (1930). *Problem tendencies in children*. Minneapolis, MN: University of Minnesota Press.

Olweus, D. (1987). *Schoolyard bullying - Grounds for intervention*. School Safety, Fall**,** 4–11.

Owens, P. L., Hoagwood, K., Horwitz, S. M., Leaf, P. J., Poduska, J. M., Kellam, S. G., & Ialongo, N. S. (2002). Barriers to children's mental health services. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*(6), 731–738.

Patel, V., Flisher, A. J., Hetrick, S., & McGorry, P. (2007). Mental health of young people: A global public-health challenge. *Lancet*, *369*(9569), 1302–1313. doi: 10.1016/S0140-6736(07)60368-7

Pearl, J. (2013). Interpretation and identification of causal mediation. *Psychological Methods*, *19*(4), 459–481.

Peto, J. (1984). Early- and late-stage carcinogenesis in mouse skin and in man. In M. Börzsönyi, N. E. Day, K. Lapis, & H. Yamasaki (Eds.), *Models, mechanisms and etiology of tumour promotion (IARCScientific Publications No. 56)* (pp. 359–371). Lyon: International Agency for Research on Cancer.

Peto, J., Seidman, H., & Selikoff, I. J. (1982). Mesothelioma mortality in asbestos workers: Implications for models of carcinogenesis and risk assessment. *British Journal of Cancer*, *45*, 124–135.

Pickles, A. (1993). Stages, precursors and causes in development. In D. F. Hay & A. Angold (Eds.), *Precursors and causes in development and psychopathology* (pp. 23–49). Chichester, UK: John Wiley and Sons.

Pickles, A., & Angold, A. (2003). Natural categories or fundamental dimensions: On carving nature at the joints and the rearticulation of psychopathology. *Development and Psychopathology*, *15*, 529–551.

Plomin, R., DeFries, J., & Loehlin, J. (1977). Genotype–environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*(2), 309–322.

Pols, R. G., Sellman, D., Jurd, S., Baigent, M., Waddy, N., Sacks, T., . . . White, A. (1996). What is the psychiatrist's role in drugs and alcohol? *Australian & New Zealand Journal of Psychiatry*, *30*(4), 540–548.

Power, C., Kuh, D., & Morton, S. (2013). From developmental origins of adult disease to life course research on adult disease and aging: Insights from birth cohort studies. *Public Health*, *34*(1), 7.

Prichard, J. C. (1837). *A treatise on insanity and other disorders affecting the mind*. Philadelphia, PA: Haswell, Barrington & Haswell.

Reich, W. (2000). Diagnostic Interview for Children and Adolescents (DICA). *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*(1), 59–66.

Reinherz, H. Z., Giaconia, R. M., Hauf, A. M. C., Wasserman, M. S., & Silverman, A. B. (1999). Major depression in the transition to adulthood: Risks and impairments. *Journal of Abnormal Psychology*, *108*, 500–510.

Rescorla, L., & Alley, A. (2001). Validation of the Language Development Survey (LDS): A parent report tool for identifying language delay in toddlers. *Journal of Speech Language and Hearing Research*, *44*(2), 434–445.

Rice, B. J., Woolston, J., Stewart, E., Kerker, B. D., & Horwitz, S. M. (2002). Differences in younger, middle, and older children admitted to

child psychiatric inpatient services. *Child Psychiatry & Human Development*, *32*(4), 241–261.

Robins, J. (1997). Causal inference from complex longitudinal data. In M. Berkane (Ed.), *Latent variable modeling and applications to causality. Lecture notes in statistics* (pp. 69–117). New York, NY: Springer-Verlag.

Robins, J. M. (1999). Marginal structural models versus structural nested models as tools for causal inference. In M. E. Halloran and D. Berry (Eds.), *Statistical models in epidemiology: The environment and clinical trials* (pp. 95–132). New York, NY: Springer-Verlag.

Robins, J. M. (2001). Data, design, and background knowledge in etiologic inference. *Epidemiology*, *12*(3), 313–320.

Robins, L. N. (1979). Longitudinal methods in the study of normal and pathological development. In K. Kisker, J. Meyer, C. Muller, & E. Stromgren (Eds.), *Psychiatrie der Gegenwart, I: Grundlagen und Methoden der Psychiatrie* (pp. 627–684). New York, NY: Springer-Verlag NY Inc.

Robins, L. N. (1985). Epidemiology: Reflections on testing the validity of psychiatric interviews. *Archives of General Psychiatry*, *42*, 918–924.

Robins, L. N. (1989). Diagnostic grammar and assessment: Translating criteria into questions. *Psychological Medicine*, *19*, 57–68.

Robins, L. N., & Price, R. K. (1991). Adult disorders predicted by childhood conduct problems: Results from the NIMH Epidemiologic Catchment Area Project. *Psychiatry*, *54*, 113–132.

Robins, L. N., & Wish, E. (1977). Childhood deviance as a developmental process: A study of 223 urban Black men from birth to 18. *Social Forces*, *56*, 448–473.

Rohde, P., Lewinsohn, P. M., Kahler, C. W., Seeley, J. R., & Brown, R. A. (2001). Natural course of alcohol use disorders from adolescence to young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 83–90.

Rossi, A. S. (1989). A life-course approach to gender, aging, and intergenerational relations. In K. W. Schaie & C. Schoder (Eds.), *Social structure and aging* (pp. 207–236). Hillsdale, NJ: Erlbaum.

Rothman, K. J. (1976). Reviews and commentary: Causes. *American Journal of Epidemiology*, *104*(6), 587–592.

Rutter, M. (1985). Resilience in the face of adversity: Protective factors and resistance to psychiatric disorder. *British Journal of Psychiatry*, *147*, 598–611.

Rutter, M. (1988). Epidemiological approaches to developmental psychopathology. *Archives of General Psychiatry*, *45*, 486–495.

Rutter, M. (1990). Changing patterns of psychiatric disorders during adolescence. In J. Bancroft & J. M. Reinisch (Eds.), *Adolescence and puberty* (pp. 124–145). New York, NY: Oxford University Press.

Rutter, M., Lebovici, S., Eisenberg, L., Sneznevskij, A. V., Sadoun, R., Brooke, E., & Lin, T. Y. (1969). A tri-axial classification of mental disorders in childhood: An international study. *Journal of Child Psychology and Psychiatry*, *10*, 41–61.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype–environment effects. *Child Development*, *54*(2), 424–435.

Seifer, R., Sameroff, A. J., Baldwin, C. P., & Balwin, A. (1989). *Risk and protective factors between 4 and 13 years of age*. Paper presented at the annual meeting of the Society for Research in Child Development (April), San Francisco, CA.

Shadish, W., Cook, T., & Campbell, D. (2002). *Experimental and quasi-experimental designs for generalized causal inference*. Boston, MA: Houghton Mifflin.

Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 28–38.

Shaffer, D., Lucas, C. P., & Richters, J. E. (1999). *Diagnostic assessment in child and adolescent psychopathology*. New York, NY: Guilford Press.

Shepherd, M., Oppenheim, B., & Mitchell, S. (1971). *Childhood behavior and mental health*. London, UK: University of London Press, Ltd.

Sherrill, J. T., & Kovacs, M. (2000). Interview Schedule for Children and Adolescents (ISCA). *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 67–75.

Silberg, J., Neale, M., Rutter, M., & Eaves, L. (2001). Genetic moderation of environmental risk for depression and anxiety in adolescent girls. *British Journal of Psychiatry*, *179*, 116–121.

Silverman, W. K., & Rabian, B. (1995). Test–retest reliability of the *DSM-III-R* childhood anxiety disorders symptoms using the anxiety disorders interview schedule for children. *Journal of Anxiety Disorders*, *9*(2), 139–150.

Simmons, R. G., & Blyth, D. A. (1987). *Moving into adolescence: The impact of pubertal change and school context*. Hawthorne, NY: Aldine de Gruyter.

Snow, J. (1855). *On the mode of communication of cholera* (2nd ed.). London, UK: Churchill.

Sroufe, L. A. (1988). The role of infant–caregiver attachment in development. In J. Belsky & T. Nezworski (Eds.), *Clinical implications of attachment* (pp. 18–38). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, *55*, 17–29.

Stattin, H., & Magnusson, D. (1990). *Paths through life—Volume 2: Pubertal maturation in female development*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Stein, Z., Susser, M., Saenger, G., & Marolla, F. (1975). *Famine and human development: The Dutch hunger winter of 1944–45*. New York, NY: Oxford University Press.

Stouthamer-Loeber, M. (1993). Optimizing data quality of individual and community sources in longitudinal research. In D. P. Farrington, R. J. Sampson, & P. O. Wikstrom (Eds.), *Integrating individual and ecological aspects of crime* (pp. 259–277). Stockholm, Sweden: Liber-Forlag.

Sung, M., Erkanli, A., Angold, A., & Costello, E. (2004). Effects of age at first substance use and psychiatric comorbidity on the development of substance use disorders. *Drug and Alcohol Dependence*, *75*, 287–299.

Suomi, S. J. (1991). Adolescent depression and depressive symptoms: Insights from longitudinal studies with rhesus monkeys. *Journal of Youth and Adolescence*, *20*, 273–287.

Susser, M. (1973). *Causal thinking in the health sciences: Concepts and strategies in epidemiology*. New York, NY: Oxford University Press.

Tolan, P. H. (1987). Implications of age of onset for delinquency risk. *Journal of Abnormal Child Psychology*, *15*, 47–65.

Tremblay, R. E., Japel, C., Perusse, D., Boivin, M., Zoccolillo, M., Montplaisir, J., & McDuff, P. (1999). The search for the age of "onset" of physical aggression: Rousseau and Bandura revisited. *Criminal Behavior and Mental Health*, *9*, 24–39.

Valla, J.-P., Bergeron, L., & Smolla, N. (2000). The Dominic-R: A pictorial interview for 6- to 11-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 85–93.

van Eldik, T., Treffers, P. D., Veerman, J. W., & Verhulst, F. C. (2004). Mental health problems of deaf Dutch children as indicated by parents' responses to the child behavior checklist. *American Annals of the Deaf*, *148*(5), 390–395.

Verhulst, F. C., Achenbach, T. M., Althaus, M., & Akkerhuis, G. W. (1988). A comparison of syndromes derived from the Child Behavior

Checklist for American and Dutch girls aged 6–11 and 12–16. *Journal of Child Psychology and Psychiatry*, *29*, 879–895.

Verhulst, F. C., van der Ende, J., Ferdinand, R. F., & Kasius, M. C. (1997). The prevalence of *DSM-III-R* diagnoses in a national sample of Dutch adolescents. *Archives of General Psychiatry*, *54*, 329–336.

von Gontard, A. (1988). The development of child psychiatry in 19th century Britain. *Journal of Child Psychology and Psychiatry*, *29*, 569–588.

Weissman, M. M., Brown, A. S., & Talati, A. (2011). Translational epidemiology in psychiatry: Linking population to clinical and basic sciences. *Archives of General Psychiatry*, *68*(6), 600–608.

Weisz, J. R. (1989). Culture and the development of child psychopathology: Lessons from Thailand. In D. V. Cicchetti (Ed.), *Rochester symposium on developmental psychopathology: Volume I*. New York, NY: Cambridge University Press.

Wickman, E. K. (1928). *Children's behavior and teachers' attitudes*. New York, NY: Commonwealth Fund.

Wilkinson, R. G. (1994). Inequalities and health. *Lancet*, *343*, 538.

Wilkinson, R. G., & Pickett, K. E. (2009). Income inequality and social dysfunction. *Annual Review of Sociology*, *35*(1), 493–511. doi:10.1146/annurev-soc-070308-115926

Williams, P. G., Holmbeck, G. N., & Greenley, R. N. (2002). Adolescent health psychology. *Journal of Consulting & Clinical Psychology*, *70*(3), 828–842.

Wing, J. K., Bebbington, P., & Robins, L. N. (1981). *What is a case?* London, UK: Grant McIntyre, Ltd.

Worthman, C. M. (1987). Interactions of physical maturation and cultural practice in ontogeny: Kikuyu adolescents. *Cultural Anthropology*, *2*, 29–38.

Young-Masten, I. (1938). Behavior problems of elementary school children: A descriptive and comparative study. *Genetic Psychology Monographs*, *20*(2), 123–180.

Yourman, J. (1932). Children identified by their teachers as problems. *Journal of Educational Sociology*, *5*, 334–343.

Zeger, S. L., & Liang, K. Y. (1992). An overview of methods for the analysis of longitudinal data. *Statistics in Medicine*, *11*, 1825–1839.

Zisook, S., John Rush, A., Albala, A., Alpert, J., Balasubramani, G., Fava, M., … Wisniewski, S. (2004). Factors that differentiate early vs. later onset of major depression disorder. *Psychiatry Research*, *129*(2), 127–140.

Zubin, J. (1978). Research in clinical diagnosis. In B. B. Wolman (Ed.), *Clinical diagnosis of mental disorders* (pp. 3–14). New York, NY: Plenum Publishing Corporation.

CHAPTER 4

Using Natural Experiments to Test Environmental Mediation Hypotheses

MICHAEL L. RUTTER and ANITA THAPAR

INTRODUCTION 129
WHAT IS MEANT BY A CAUSE? 129
NONCAUSAL ALTERNATIVE EXPLANATIONS FOR AN ASSOCIATION 130
NATURAL EXPERIMENTS 132
Genetically Sensitive Designs 133
Strategies to Identify the Key Environmental Risk Feature 138
Designs for Dealing with Selection Bias 142
Instrumental Variables 144
Regression Discontinuity Designs to Deal with Unmeasured Confounders 146
Adoption and Fostering as a Means of Separating Prenatal from Postnatal Effects 147
ARE NATURAL EXPERIMENTS REALLY NEEDED? 148
OVERVIEW OF NATURAL EXPERIMENT METHODOLOGY 149
REFERENCES 149

INTRODUCTION

The chapter begins by considering what is meant by a cause—noting both equifinality and multifinality. Noncausal alternative explanations for a statistical association are discussed in terms of genetic mediation, social selection, reverse causation, misidentification of the risk feature, and a confounding variable. Unmeasured confounders are discussed by considering propensity scores, growth curve trajectories, structural equation modeling, direct acyclic graphs (DAGs), mediation analyses, and sensitivity analyses. The different varieties of natural experiment are reviewed with respect to many genetically sensitive designs. These include multivariate twin designs, discordant twin pairs, discordant sib pairs, children of twins (CoT) designs, the extended children of twin (eCoT) design, the longitudinal twins and parents (LTaP) design, adoption studies, assisted reproduction technologies (ART), maternal versus paternal smoking contrasts, and migration strategies. The contrasting of two environmental risk factors is discussed with respect to twin–singleton comparisons, adoption as a radical change of environment, time-series designs, and inverse probability of treatment weighting. Designs to deal with social selection involve circumstances that entail either universal introduction of risk or its removal. Instrumental variables are discussed in relation to early puberty, Chinese one-child policy, the sex of the co-twin, and Mendelian randomization (MR). The regression-discontinuity design is examined as the only design that deals adequately with unmeasured confounders. Adoption/fostering is considered as one means of separating prenatal from postnatal effects. Finally, we look at research indicating that natural experiments are truly needed.

This chapter is based on Rutter's (2007, 2012) papers on natural experiments and it includes substantial sections that appear in the same form as they did in those papers. Both authors contributed equally to this chapter. We are most grateful to Camilla Azis-Clauson for great help in searching the literature and in drafting.

WHAT IS MEANT BY A CAUSE?

At first sight, it might be thought unnecessary to ask what is a cause when discussing causal hypotheses. Thus, surely, a cause can simply be considered as anything that brings about whatever outcome is being examined? However, there are several different reasons that the issue is not quite as straightforward as that. First, with respect to either psychopathology or psychological traits, multifactorial causation is the rule. There are very few simple direct

determinative causal effects on any outcome. MacMahon, Pugh, and Ipsen (1960) suggested the metaphor of a web of causation, and Rothman and Greenland (2002) similarly argued that any causal effect will have been brought about by a constellation of components acting in concert. With very few exceptions, there is no such thing as a single necessary and sufficient cause.

Several different causal pathways may all lead to the same end point (Rutter, 1997), what Cicchetti and Cohen (1995) referred to as *equifinality*. Equally, any single starting point may lead to a diverse range of outcomes—what Cicchetti and Cohen called *multifinality*. There can be no single cause of any outcome, for these reasons and also because each causal element is likely to be reducible to a pathway that starts with some other element. Mackie (1965, 1974) referred to causes that are insufficient but necessary components of unnecessary but sufficient causes—now usually referred to as INUS (Schwartz & Susser, 2006). That sounds complicated, but actually it is not. It is simply a way of stating that the overall causal nexus in its entirety is a sufficient causal condition being considered without needing to invoke the operation of any other influences. On the other hand, it is unnecessary because it represents only one of several possible causal pathways. The clear implication is that changing a causal factor will actually reduce the population's burden of disorder (if the outcome being investigated concerns a disorder) (Robins & Greenland, 1996). Hill (1965) famously outlined a set of guidelines that might be used to consider the possible validity of a causal inference. These were helpful, but the most important requirement is the need to consider alternative explanatory hypotheses (Cochran & Chambers, 1965). Simply providing supporting evidence that might bolster the causal inference should never be acceptable (National Research Council, 2002).

Holland (1986) argued that attributes of units can never be causes because causes must be, in principle, open to manipulation. That sounds reasonable, but it is important to note that what seemed to be fixed attributes can often be broken down in a more meaningful way. For example, sex is a fixed attribute, but it is reasonable to pose the question as to whether any effect is a consequence of the person's chromosomes, prenatal hormonal influences, contemporary hormonal influences, and so forth.

The two most influential causal models in recent times have been those of Campbell and of Rubin. Campbell's approach focused on the identification to threats to validity and the inclusion of design features that might prevent those threats from occurring or might render them implausible (Shadish, 2010; West & Thoemmes, 2010). By contrast, Rubin focused on precise specification of possible outcomes and assumptions that are mathematically sufficient to estimate the causal effect. Both have a preference for design over statistical analysis, although analytic approaches are crucial to both. See Berzuini, Dawid, and Bernardinelli (2012) and Foster (2010) for a fuller discussion of the different statistical perspectives needed in studying causality. Pearl (2000) argued for the helpfulness of directed acyclic graphs (DAGs) as a way of making statistically explicit the causal process that is being examined and the assumptions that are involved in its testing (Robins, 2001; Robins, Hernán, & Brumback, 2000).

NONCAUSAL ALTERNATIVE EXPLANATIONS FOR AN ASSOCIATION

Five possibilities have played the greatest role in the development of natural experiments. First, there is the possibility of genetic mediation of the risks stemming from an environmental feature. This possibility arises as a consequence of both passive and active gene–environment correlations. Passive in this connection refers to the fact that parents who pass on their risky genes may also have a tendency to create risky environments. Because a risk factor clearly refers to an environmental feature (as would be the case with poverty or social disadvantage or family conflict and probably also applies to the rearing provided by parents with a mental disorder; Rutter et al., 1997), the causal mediation may, nevertheless, be genetically mediated. Pike, McGuire, Hetherington, Reiss, and Plomin (1996) provided an example of how effects of family conflict are partially genetically mediated. Gene–environment correlations refer to the tendency for individuals (both children and adults) to shape and select their environments. As a consequence, there is evidence of genetic influences on individual variations within a range of different types of risk exposure (Kendler & Baker, 2007). To deal with these possibilities researchers have devised a range of genetically sensitive designs to test environmental–risk mediation hypotheses.

Second, there is the possibility of social selection or allocation bias (Caspi, 2003)—meaning that the association between the risk experience and the outcome reflects the *origins* of the putative risk factor rather than its effects. The possibility arises, for example, in terms of the risk for a variety of psychopathological outcomes for children born to a teenage mother (Maynard, 1997; Moffitt & Team, 2002). Is this a reflection of the causal impact of rearing by a teenage mother, or, rather, does it reflect the qualities of

individuals who have a pregnancy as a teenager? In other words, would the same be found for individuals born to an older mother who, at an earlier point in her life, had a pregnancy as a teenager? Designs using twins or adoptees have been developed to determine the possibility of social selection. Also, designs have been used that capitalize on circumstances in which total populations have been exposed without the opportunity of choice or selection.

Third, there is the possibility of reverse causation. In a now classic article, Bell (1968) argued persuasively that some of the supposed effects of socialization experiences might reflect the effect of children on their social environments (as indexed, for example, by parents and teachers) rather than the effects of the children's rearing environment on them. Thomas, Chess, and Birch (1968) argued much the same when considering children's temperamental features. Such reverse effects have been shown in both experimental (Anderson, Lytton, & Romney, 1986) and naturalistic studies (Bell & Harper, 1977) as well as in studies of the effects of children on their adoptive parents (Ge et al., 1996; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). The notion of reverse causation has also been extended to associations between one form of psychopathology and another type of disorder (McGue & Iacono, 2005).

The fourth possibility is that the risk feature has been misidentified. Most famously, this was the case with the postulate that broken homes were a major cause of antisocial behavior (see Wootton, 1959, for a critique). Research went on to show that the main risk stemmed from either family discord or poor parenting rather than from a family change or breakup as such (Brown, Harris, & Bifulco, 1986; Fergusson, Horwood, & Lynskey, 1992). Similarly, designs using adoptees have been used to separate prenatal from postnatal effects and to differentiate among different possible sources of environmental risk.

Fifth, there is the very real concern that any association might reflect nothing more than some confounding variable—meaning any variable that both differentiates between the groups to be compared and is statistically associated with the outcome being investigated. Traditional multivariate statistical analyses make adjustments for confounders that have been measured. Unfortunately, these methods involve several serious problems (Rutter, Pickles, Murray, & Eaves, 2001). It is evident that statistical adjustments cannot solve the problem. Therein lies the need for natural experiments. First, there is the logical problem of having to use an invariant set of associations to infer what would happen if a key association was different or was changed (McKim & Turner, 1997). That necessitates the assumption that the effect within the existing sample would apply to a different set of circumstances (i.e., without the confounder). The general statistical strategies for dealing with possible confounding variables are discussed in Rosenbaum (2002); Rothman and Greenland (1998); Shadish, Cook, and Campbell (2002); and Susser, Schwartz, Morabia, and Bromet (2006). It would be inappropriate to control for factors that are intermediate on the pathway between exposure to the risk factor and the outcome or those that are caused in part by exposure to the risk experience (see Weinberg, 1993, for a discussion of this issue).

The problem of assuming that effects within the existing sample would apply to a different set of circumstances is particularly great if the two groups being compared do not overlap in key cells (see Melhuish, Belsky, Leyland, & Barnes, 2008, for an example of this kind). They found that scarcely any children in the comparison group came from high-risk families, whereas these constituted a high proportion in the intervention group. Propensity scores are particularly useful in identifying where there is a lack of overlap and the problem can be partially circumvented by dropping the overlap cells before undertaking the statistical controls. They are also helpful in reducing the biasing effects down to a single balancing factor. Note that it is often assumed that if a multivariate analysis shows an effect of A on some outcome that is maintained after taking account of the effects of B or C, this means that necessarily there is a true effect of A in the absence of B or C, but it does not (Rutter, 1983).

An even greater problem is presented by the uncertainties regarding unmeasured confounders (Academy of Medical Sciences, 2007). The main problem does not lie in the control for confounders that have been conceptualized and carefully measured. Rather, the main difficulty arises from unmeasured confounders. The problems in dealing with confounders may be reduced in five main ways (Rutter, 2009). First, propensity scores can be used to determine the features associated with *exposure* to the putative causal influences. They can then be employed to calculate an inverse probability to treatment weighting (IPTW) to create two comparable groups (Robins et al., 2000). The technique involves a minimum of statistical assumptions and Sampson et al. (2006) provided a particularly clear account of its use. Nevertheless, its value is critically dependent on the identification of all the most important features influencing risk exposure.

A second approach is to use growth curve trajectories to take in account of an individual's overall liability to the outcome being considered (Nagin, Barker, Lacourse, & Tremblay, 2008). The logic here is that if the putative causal

influence alters a person's growth curve with respect to the outcome being considered, a causal influence is more probable. The use of this approach requires at least four measurement points over a substantial period of time to create a valid growth curve and requires a time-specific exposure to the putative causal influence. Nagin et al.'s (2008) example showed its value in testing for the effects of gang membership on criminal behavior. Furthermore, as with any other form of cluster analysis the curves represent a model (rather than a real phenomenon at an individual level). The number of curves selected for analysis is a matter of judgment on their likely meaning rather than the result of some single statistical solution, although naturally, statistical criteria should play a crucial part in the choices.

Third, structural equation models (SEMs) can be used to treat confounders as a latent variable, hence allowing a better assessment of their effects after taking into account measurement error. If multimeasurement longitudinal data are available, a two-stage approach can be employed: the first step involves the regression of effects on exposure, and the second step uses the predicted values of the exposure covariate in a regression of the putative cause on outcome (Dawid & Pickles, 2007). Fourth, as already noted, DAGs can use preexisting background knowledge to indicate the ways a possible confounder might operate. The value of this approach is crucially dependent on the plausibility of the model.

The fifth method is provided by mediation analyses (Baron & Kenny, 1986; Fairchild & MacKinnon, 2009; MacKinnon, Fairchild, & Fritz, 2007). On their own, they do not provide a test of causation, but what they do is to isolate a putative key mechanism that could account for the observed statistically significant association. The logic is that when the postulated mediator for C with respect to the effects of A on B is introduced into the overall model, the original correlation of A on B should drop to zero if there is complete mediation or be substantially reduced if there is partial mediation (see Kumsta, Rutter, Stevens, & Sonuga-Barke, 2010, for a discussion of the issues). Care in mediation analyses is required to ensure that there is no confounding between the mediator and the outcome.

Whatever the approach used, there is value in using sensitivity analyses to quantify how strong a confounding effect would have to be to overturn a causal inference based on a case–control comparison. For example, Cornfield et al. (1959) used such an analysis to show that if the association between smoking and lung cancer was due to a confounder, such a confounder would have to be nine times as frequent in heavy smokers as in nonsmokers to undermine the causal inference. Clearly, that was extremely implausible.

Last, it is necessary to consider the mistaken view that randomized controlled trials (RCTs) provide the gold standard for testing for causal influences. RCTs do constitute the gold standard for assessing the effect of a planned intervention. They provide much the best way for providing an unbiased measurement of intervention effects within that sample in relation to a particular intervention. There are three important limitations, however, with respect to studying causation. To begin with, the fact that an intervention has a significant effect does not necessarily mean that this can be generalized to what caused the problem in the first place. Second, RCTs often apply to rather specialized groups that may not be directly relevant to the population of interest. Third, attrition or nonparticipation may be a substantial problem in an RCT, particularly when it is dealing with long-term effects. It has been suggested that prevention trials allow for a good opportunity to test hypotheses about etiology (Howe, Reiss, & Yuh, 2002). However, as Howe et al. (2002) pointed out, for them to be used in this way certain key design features are essential, and these are usually not available.

Propensity scores, growth trajectories, structural equation modeling, mediation analyses, and RCTs provide important elements in the research process, but none constitutes an adequate substitute for natural experiments.

NATURAL EXPERIMENTS

Natural experiments differ fundamentally from many of the conventional statistical approaches in that they do not adjust or control for confounders. Instead, they use naturally occurring circumstances that serve to separate variables that ordinarily go together and, by this means, provide manipulations and not just analysis of crucial variables. Cook and Campbell (1979) and Shadish et al. (2002) were pioneers in developing quasi-experiments or natural experiments. They discussed the vital interplay between designs and statistical analysis. It is important to appreciate that natural experiments are not just a list of designs because the principles may be applied to the analysis of data sets gathered for other purposes. Thus, this may arise, for example, with cohort studies in which it is possible to construct estimators that exploit natural experiments that happen to be embedded within the sample (e.g., siblings and twins). With each of the natural experiments to be described, we consider their possible limitations and the assumptions they have to rely on that may or may not be

justifiable. Each design carries its own threats to internal and external validity and progress is best achieved when different studies, using different designs with different limitations, converge on similar findings.

Genetically Sensitive Designs

There are some nine types of natural experiments designed to differentiate between genetic and environmental mediation of risks through a variety of twin and adoptee designs, through offspring conceived with assisted reproduction technologies, and migration strategies. Twin designs use the contrast between monozygotic (MZ) and dizygotic (DZ) twin pairs to partition the population variance into additive genetic and shared or nonshared environmental effects. This contrast is predicated on the fact that MZ pairs share 100% of their segregating genes, whereas DZ pairs share on average just 50%. By treating some postulated environmental causal factor as a trait or phenotype, it is possible to estimate the heritability of the risk factor trait. By means of cross-twin, cross-trait analyses (the cross being between the risk factor and the outcome), the extent to which mediation is genetically or environmentally mediated can be quantified. Longitudinal data will be required to check the temporal order.

Multivariate Twin Designs

The longest established twin strategy is the use of multivariate twin analyses (see Kendler & Prescott, 2006). Findings using this approach raised doubts with respect to the causal inference made with respect to the substantial association found between an early age of first drinking alcohol and the later development of alcoholism (Grant & Dawson, 1997). Multivariate twin analyses showed no environmentally mediated effect deriving from the age of first drinking. Rather, both the early age of drinking and the alcoholism reflected the same genetic liability. Similarly, Jaffee et al. (2004) used the same strategy to examine the observed association between both corporal punishment and physical child abuse and children's propensity to engage in antisocial behavior. The findings confirmed environmental mediation in the case of abuse but not for corporal punishment, for which the mediation was largely genetic. That is, the implication was that corporal punishment probably mainly occurred as a result of responding to the child's genetically influenced behavior. By contrast, physical abuse did not show this effect and presumably arose from either features in the abusing parent or from features in the social or physical environment. On the other hand, analyses also showed that the regular use of severe physical punishment did involve a propensity for this to escalate into child abuse. The strength of the design is that it allows a means of disentangling different effects but, equally, allows detection of more complex two-way associations.

Much research has proved the value of multivariate twin analyses, but there are several important limitations. For example, the equal environments assumption is probably violated, at least in part, when studying an outcome such as depression or antisocial behavior (Rutter, 2006). But there are three other rather different hazards. First, the operation of parsimony in modeling sometimes means that potentially important features are dropped from the model because they just fail to reach significance. This arises most frequently with shared environmental effects. On occasion, the consequence may be a misleading model (see Maes et al., 1999, for an example of where this occurred). Second, twin studies assume that mating is random, whereas often it may not be. Third, there is rarely the power to deal with the confounding of genetic effects by the inclusion of GxE (when the E involves shared environmental effects).

Discordant Twin Pairs

Discordant same-sex twin pairs reared together represent a rather different twin strategy. The rationale in this case is that by examining pairs that are discordant for both the hypothesized environmental risk factors and the outcome being studied, it will be possible to determine how the two coincide. The design provides a substantial degree of control for shared genetic and environmental liabilities (Kendler & Prescott, 2006). Prescott and Kendler (1999) used this design to examine the supposed causal effect of early drinking on later alcoholism. Statistical modeling showed that the association largely reflected a shared genetic liability, although there was some effect from shared environmental factors. There are two important limitations. First, the discordance within MZ pairs raises the question of *why* they were treated differently within the family or had different experiences outside the family. No robust findings on this important matter are available in the published literature. Second, although the design provides a strong pointer that the within-pair difference in risk exposure exerted a causal influence it cannot completely rule out the possibility that some *other* experience was responsible (Jaffee, 2011; Vitaro, Brendgen, & Arseneault, 2009). Note, too, that, because MZ twins share 100% of their DNA, differences in their experiences cannot logically arise from differences in the structure of their DNA. Nevertheless, it could result from epigenetic differences within a pair (Fraga et al., 2005; Mill et al., 2006;

Wong et al., 2010). This does not invalidate the design, but it does complicate the interpretation because the epigenetic differences are likely to reflect environmental influences but these may operate through effects on gene expression rather than a direct effect of the environment itself.

In dealing with the discordant twin design, Lynskey (Lynskey, Vink, & Boomsma, 2006; Lynskey et al., 2003), first using the Australian twin register and then the Netherlands twin register, examined the so-called gateway hypothesis that the early use of cannabis led, by some form of causal mechanism, to an elevated risk of other elicit drug use. Both studies showed a significant gateway effect—with a two- to fourfold increase in other drug use. Retrospective reporting at age 30 was required to obtain assessments of lifetime use, and the comparisons were based on all twins. The numbers were too small for a full control of genetic influences by focusing on monozygotic pairs. Contrary to the early alcohol study (Prescott & Kendler, 1999), both the Lynskey et al. studies confirmed the likely environmentally mediated causal gateway effect. On the other hand, the mechanisms involved were quite uncertain. They could reflect attitudes induced by early pleasurable cannabis effects or a social effect operating by means of cannabis use providing an entry to groups using other drugs and bringing about contact with drug dealers.

Rutter (2007, 2012) used a study by Caspi et al. (2004) to illustrate how a combination of design features can substantially increase the power of a discordant MZ twin design to test for causal effects. The risk feature being investigated was maternal negative emotion focused on one twin, this having been assessed when the twins were aged 5 years using standardized ratings based on audio-taped interviews with mothers. Outcome was assessed two years later using teachers' reports. This removed the possibility of rater bias, and it also examined effects that spanned a radical change in environment (namely, starting school). The prospective longitudinal design provided measurement of within-individual change and determined the temporal sequence. Because the analysis took account of the children's behavior at age 5, the prospective strategy effectively ruled out any mediating effect of the child's behavior on the mother's emotional expression. The main limitation, as with other quasi-experimental designs, is the uncertainty as to whether the psychopathological risk stemmed from the negative expressed emotion per se or rather from some associated environmental feature impacting one twin only in the MZ pair. Hence, the natural experiment convincingly showed a significant environmental effect, but it could not sort out whether the maternal negative expressed emotion exerted the causal effect as against the possibility that it simply indexed some other environmental influence. In addition, the findings leave open the question of what set of features led the mother to respond so differently to the two twins.

Discordant Sib Pairs

Discordant twins cannot be used to examine prenatal risk effects, but discordant sibling pairs can serve the same purpose. Obel and colleagues (2011) used this design to examine the effects of the fetus' exposure to maternal smoking. Smoking was assessed from maternal report at the time of the child's birth, and attention-deficit/hyperactivity disorder (ADHD) was determined from clinical record register data. In the sample as a whole there was a highly significant odds ratio of 2.2, but the discordant sibling comparison showed a much reduced, statistically non-significant ratio of 1.2, which cast serious doubt on the causal inference with respect to ADHD but again confirming the causal effect on birth weight (which had been supported by all research strategies). It is noteworthy that if a natural experiment had not been used there would be the strong inference of a causal effect because supposedly there had been an adequate statistical control for confounders. Because there is random allocation of genes across sibs, there was good control for genetic liability. See D'Onofrio et al. (2010) for another example of the use of a discordant sib pair design.

Nevertheless, although this is an excellent design for studying prenatal risk factors, three limitations may be relevant. First, sibling comparisons involve the assumption of a stable unit treatment value (SUTVA)—meaning that the effect of each participant's exposure to the risk factor does not influence other unexposed participants (Lahey & D'Onofrio, 2010). That is not a plausible problem with respect to prenatal maternal smoking exposure, but it could well be relevant when examining postnatal influences. Second, the design involves the assumption that any differences between women who quit smoking when they become pregnant and those who continue smoking heavily is unrelated to any risk factors for the child outcome being considered. Although this possibility has been examined in relatively few studies, the large sample (over 18,000) in the UK Millennium Cohort study did provide the possibility of examining this issue (Pickett, Wilkinson, & Wakschlag, 2009). The findings showed that heavy smokers had the most problematic psychosocial context, and the implication is that this could reflect postnatal influences on child outcomes.

This was examined directly by Boutwell and Beaver (2010) using what they called a propensity score approach.

The maternal features used included antisocial behavior, substance abuse, depression, lower level of education, younger age, and family adversity. Three paternal features were also included. Unfortunately, the authors appear to have misunderstood the logic of propensity scores in that they included the child's Apgar score, delivery complications, and labor complications. Not only was it the case that none of these were associated with maternal smoking, but also in the case of first-born children the features will have followed (not preceded) maternal smoking. The paper argued that the propensity score approach reduced the effect of confounders but failed to appreciate that it was an error. The same problem also appears to have applied to a comparable study by McCrory and Layte (2012).

Third, the design does not provide unbiased estimates if the risk factors are not static over time (Jaffee, Strait, & Odgers, 2012). Jaffee and colleagues (2012) used the example of single motherhood as a possible risk factor for children's antisocial behavior. Sibling comparisons might be biased if the mother was younger than 20 years and single when her first child was born but older than 20 years and married when her second child was born. As in all natural experiments, it is crucial to consider the assumptions that have to be made and then decide whether these are actually likely or unlikely to bias the design with respect to the risk factor and outcomes being examined.

Harden et al. (2007) used sibling comparisons to examine whether children born to adolescent mothers had more mental health problems as a result of being reared by an adolescent mother. The alternative possibility is that any such effect was a consequence of the factors associated with being an adolescent mother rather than rearing by such a mother. To address that possibility, children of teenage mothers were compared with the firstborn children of older mothers. There was no difference between children born to teenage mothers and children born to older mothers who had had a birth as a teenager some years previously. A novel feature of this study was its integration with a children of twins (CoT) design. The key comparison here, then, is whether the children born to teenage mothers showed more mental health problems than their first cousins born to older mothers. The findings showed that they did have more problems, and this was consistent with the causal effects of early motherhood rather than the operation of genetic or environmental third variables. It is important to note that Jaffe, Caspi, Moffitt, Belsky, and Silva (2001) using the Dunedin longitudinal study found both an effect of rearing by an adolescent mother and an effect of being born to a mother who had been a teenage parent when younger. Why these two studies produced somewhat different findings is not clear. So far as use of the CoT design is concerned, there is the usual problem of no information on the fathers.

Children of Twins Design

The CoT design provides a useful extension of twin strategies (D'Onofrio et al., 2003; Rutter et al., 2001; Silberg & Eaves, 2004). The rationale of this design is that the offspring of adult MZ twins will be social cousins but genetic half-siblings (half because one parent is part of an MZ pair and the other is not). By contrast, the offspring of DZ twins are cousins both genetically and socially. If the postulated causal parental feature involves a genetically mediated risk, it should apply as much to the offspring of both the MZ twins regardless of whether one is providing the social rearing and the other is not. This will not apply as much to the offspring of DZ twins because, on average, they share just 50% of their segregating genes. The CoT design has been used with good effect to show that harsh forms of physical punishment have an environmentally mediated influence on disruptive behavior and substance use and abuse in the children (Lynch et al., 2006) and have shown that prenatal exposure to maternal smoking does not have an effect on ADHD, at least not in alcoholics (Knopik et al., 2006). The CoT design constitutes a potentially useful strategy for testing for environmental mediation, but it has several limitations. First, as is obvious from the necessity of using the rather small genetic differences between cousins and half-siblings, it requires very large samples. Second, it is quite difficult to take adequate account of the unknowns in both the genetic background and rearing environment provided by the parent who is not a twin. Third, unless the spouse background and rearing behaviors are measured and included in the analysis, the findings for behaviors that involve dyads (as would be the case, e.g., with marital conflict) could be misleading (Eaves, Silberg, & Maes, 2005). To deal with these and other related issues, two extensions of the CoT design have been developed.

ECoT Design

First, the extended children of twins (ECoT) design includes data from adult twins and their offspring and child twins and their parents (Narusyte et al., 2008). The ECoT model has the special advantage of being able to distinguish passive genotype–environment correlations from evocative genotype–environment correlations. The Narusyte study used only cross sectional data, and it would definitely be advantageous to attempt the same with

longitudinal data. Combining two different samples necessarily involves the assumption that the combined samples be comparable in terms of size, participant characteristics, and measured constructs. Because the model excludes the spouse of the parent, assortative mating cannot be taken into account. Also, it appears from simulations that the model performs less well when it includes parental traits influenced by the shared environment.

LTaP Design

The other extension is the longitudinal twins and parents (LTaP) design. An earlier version of the use of combination of twin and family data was described by Meyer et al. (2000). It showed an environmentally mediated effect of family adaptation, but the model used had the substantial disadvantage that the measurement of conduct disorder in the parents had to be reliant on retrospective measurement. The LTaP development obviated that problem by availability of a follow-up into early adult life of child twins. In other words, there was information about twin behavior prior to adulthood and in adulthood with the availability of measures in both age periods. The model upheld the assumption that the same genes influenced parent and offspring phenotypes, provided that there were no sizeable cohort effects. The design also estimated the degree of phenotypic assortment.

Silberg et al. (2010, 2012) used the LTaP design very well to examine the effects of parental depression and parental antisocial behavior on three different forms of psychopathology in the children: ADHD, conduct disturbance, and depression. The findings were striking in showing that the impact of parental antisocial personality on ADHD in the child was entirely genetic. Conversely, the association between parental antisocial behavior (ASB) and child depression was largely environmental. On the other hand, both genetic and family environmental mediation accounted for the association between parental ASB and children's conduct disturbance. The pattern of findings with respect to conduct disorder and depression in the children was much the same when studied in relation to parental depression. Specific parenting behaviors were not included in the published analyses, but separate analyses from the same data set showed the important mediating role of parental neglect on child conduct disturbance over and above any indirect genetic correlation between parents and their children (Eaves, Prom, & Silberg, 2010). As will be appreciated, particular assumptions are involved in several elements in the modeling; however, the main limitation for the design as a whole concerns the lack of information about the twins' spouses and environment, although such information can in principle be obtained and modeled (Jacob et al., 2003).

Adoption Studies

As Rutter et al.'s (1990) review of research strategies clearly showed, there are many different variants of the adoption design. However, all rely on the fact that adoption separates genetic heritage (and prenatal effects) from the rearing environment. In much of the literature, the emphasis has been on the detection of genetic effects, but here we are concerned only with the design as a means of testing environmental mediation. Duyme, Dumaret, and Tomkiewicz's (1999) research may be taken as an example because of its innovative use of measurement of the adoptive environment together with the availability of longitudinal data. The study was designed to examine the effects of the socioeconomic (SES) level of the adoptive family on the IQ gains following adoption between the ages of 4 and 6 years of children who had been compulsorily removed from their families during infancy because of concerns about abuse or neglect. Not surprisingly, there was an overall group increase in IQ following adoption (as would be expected because of the overall environmental improvement). What was novel was the determination of whether the degree of rise in IQ varied according to the SES of the adoptive families. The findings showed that the gains were significantly greater for those placed in high SES families than for those placed in low SES families. It is that finding that was novel in that it provided the best evidence that the gain in IQ was indeed a consequence of the qualities of the adoptive home.

There are, nevertheless, important limitations to adoption designs. First, there may have been selective placement. However, because that can be measured and because it is a relatively straightforward matter to take it into account in data analysis, that is not the main issue. What is a greater concern is the lack of comparability of the biological families and the adopting (rearing) families. In most countries, adopting parents tend to be better educated and more socially advantaged than the rest of the population, and, conversely, biological parents who have given up their children for adoption are much more likely to include a disproportionately high percentage of individuals with genetic risk characteristics.

Stoolmiller (1998, 1999) drew attention to the restricted range of family environments in adoptive families (as a result of the screening of families for what are thought to be environmental risk features). McGue et al. (2007) used a sample of 409 adoptive and 208 nonadoptive families from the Minnesota Sibling Interaction and Behavior

Study (SIBS) to examine this possibility. They found that, relative to nonadoptive families, adoptive families showed a 41% reduction of variance in what the authors called disinhibitory behaviors such as alcohol and substance abuse, plus 18% less variance in socioeconomic status. In short, the findings confirmed the Stoolmiller claims, but, contrary to Stoolmiller, there was no effect of the restricted range on adoptive sibling correlations for delinquency, drug use, and IQ. In other words, perhaps the consequences of the restricted range might have less effect than one would have thought on testing of environmental mediation through adoption designs.

However, as McGue et al. (2007) pointed out, it appears that adoption practices have changed over the last generation in Minnesota, with less screening than there had been in the past. The findings from other independent studies are needed before firm conclusions can be drawn on this particular limitation of the adoption design. From a practical point of view, the limited value of adoption designs, at least in the United Kingdom, is provided by the marked reduction in the number of healthy babies put up for adoption in the infancy period. Also, there are restrictions in many places on the use of adoptee samples for research.

Assisted Reproduction Technologies

Yet another way to exclude genetic mediation of risk, to identify environmental mediation where it occurs, is provided by the use of ARTs (see Thapar et al., 2007). ARTs retain the genetic link between mother and child (as with homologous in vitro fertilization and sperm donation, whereas others do not as with egg or embryo donations). If the maternally provided prenatal risk factor has an environmentally mediated effect on the child's outcome, this should give rise to the same associations irrespective of whether the mother and child are genetically related. Conversely, if the risk is genetically mediated, it should be evident only where there is a genetic link between mother and child. This method has been used to investigate the postulated prenatal risk from maternal smoking associated with both antisocial behavior (Rice et al., 2009) and ADHD (Thapar et al., 2009). Findings showed genetic mediation in relation to both outcomes, but there was environmental mediation in relation to birth weight (as found by animal models as well as by other human research). Importantly, as with other studies the findings showed that statistical control for confounders (including parental ADHD and maternal alcohol consumption) was inadequate to deal with the possibility of genetic mediation. The same design has also been used to test for environmental mediation of hostile parenting on antisocial behavior in the children (Harold, Elam, Lewis, Rice, & Thapar, 2012).

The main practical limitation of this design is the need for an adequate sample size to make the required comparisons. Some have expressed concern as to whether the women who use ARTs are systematically different from those who do not, in ways that might bias the design. We do not see that as a likely problem because the key thing is whether there is a systematic difference between those who have sperm donations compared with those who have egg or embryo donations. Theoretically, that is a possibility, although it is difficult to see how it could arise. With respect to the use of the design to look at the effects of smoking, there is the further problem that secular trends have meant that the number of women smoking in pregnancy is low, with an inevitable reduction in the size of key cells. On the other hand, the sample size was adequate to show a true causal effect of prenatal maternal smoking on the child's birth weight. In the studies undertaken, analyses were based on symptom counts rather than clinical diagnoses and, ideally, it would be useful to have adequate clinical assessment. Nevertheless, in our view the implausibility of biases in this particular design make it a potentially useful one to use, provided there can be the acquisition of appropriate ART samples of adequate size.

Maternal Versus Paternal Smoking Effects

Another way to test the possible causal effect of exposure to maternal smoking during pregnancy is to compare the effects with those of paternal smoking during pregnancy (as the latter cannot have had the same prenatal effect). Langley, Heron, Smith, and Thapar (2012) made this comparison using the Avon Longitudinal Study (ALSPAC) of some 8,000 children. The outcome examined was, again, ADHD. Because of the extensive evidence both from human and animal models that maternal smoking has a causal effect in leading to a reduced birth weight, this comparison was made between maternal and paternal smoking. In line with previous research it was found that maternal smoking was associated with a substantial and significant reduction in birth weight but that paternal smoking was not when the mother did not smoke. Conversely, however, both maternal and paternal smoking was associated with ADHD symptoms in the offspring, and there was no significant difference between the two. This suggested that the prenatal effect was not representing a causal association. It was possible, however, that paternal smoking might have had an intrauterine effect of on the fetus through passive smoking, but there was no evidence to show that this was the case. It is surprising that rather more use has not

been made of this comparison, but presumably it has not because so few studies have examined paternal smoking during the pregnancy on the grounds that it could not have a direct causal effect. The point to make here is that it is a pity to pass up the possibility of using this comparison as an informative natural experiment.

Migration Strategies

The last strategy to control for genetic mediation to mention is provided by migration. Expressed simply, the natural experiment arises because when people move from country to country they carry their genes with them but they may encounter quite different environmental circumstances. The natural experiment therefore is determining what happens when high-risk or low-risk ethnic groups move to a different country and adopt entirely different lifestyles. Two medical examples illustrate the point most clearly. The Pima Indians have long been observed to have an unusually high rate of obesity and obesity-connected conditions. It seems very likely that genetic factors play a role in this but the experimental contrast was provided by comparison of the outcomes between Pima Indians living in affluent circumstances and those living in areas with quite different (less adequate) nutritional opportunities. The findings were striking in showing that, when the lifestyle and dietary conditions varied, so did the rate of medical conditions arising from obesity (Ravussin, Valencia, Esparza, Bennett, & Schulz, 1994; Valencia et al., 1999).

A somewhat parallel, although less dramatic, example is provided by an early study of coronary artery disease in people of Japanese origin living in Japan, Hawaii, and California (Marmot & Syme, 1976). The findings showed that when the Japanese people adopted a Californian lifestyle their rate of coronary artery disease rose to much the same levels as those of Caucasian individuals living in California. In other words, although it is likely that there were genetic factors involved in the vulnerability, the prime risk factor lay in lifestyle features, including diet. Interestingly, in this instance, smoking was not implicated in the migration difference because smoking levels were very high in Japan as well as in California. It should be noted that the findings with respect to hypertension were rather different, and much remains to be explained in the causal nexus in relation to different outcomes.

The best-documented psychopathological example concerns the raised rate of schizophrenia spectrum disorders in people of Afro-Caribbean origin living in either the United Kingdom or the Netherlands—the two countries in which the phenomenon has been most extensively examined (Coid et al., 2008; Jones & Fung, 2005). The design relies on two key comparisons: first, the rate of disorder in migrants compared with the rate of the disorder in their country of origin; and second, the rate in migrants compared with the rate in nonimmigrants in the host country to which they migrated. In the case of schizophrenia spectrum disorders (and, to a lesser extent, some other psychoses) it was found that the rate was substantially higher in people of Afro-Caribbean descent living in the United Kingdom or the Netherlands than in Caucasians living in the same two countries and compared with people of presumably the same ethnic origins living in one of the islands in the Caribbean. A very detailed, carefully controlled study showed that the difference in the incidence of schizophrenia spectrum disorders was not an artifact of recognition or diagnosis but rather seemed to reflect some aspect of environmental adversities associated with migration to the United Kingdom or the Netherlands. Migration as such is not likely to constitute the causal factor because the findings applied to both second- and first-generation migrants and because in other circumstances migration may even bring benefits (Stillman, McKenzie, & Gibson, 2009).

Recently, a parallel study in the United States showed a somewhat similar ethnic difference, with African Americans about three times more likely to be diagnosed with having schizophrenia than White Americans (Bresnahan et al., 2007). The causal inference with respect to some kind of environmental influence is strong, but it would be much stronger with evidence of environmental factors differentially operating within ethnic groups. So far, various possibilities have been ruled out, but the key environmental risk factor has yet to be identified. Fu and Van Landingham (2012) used a migration design in a novel way by comparing Vietnamese immigrants to New Orleans in the United States to never-leavers in Ho Chi Minh City and returnees. These juxtapositions provided a net comparison of selection effects and immigration effects.

Strategies to Identify the Key Environmental Risk Feature

Twin and adoptee strategies have usually been considered solely in terms of their power to examine genetic mediation of what seemed to be environmental risk effects. However, there are circumstances in which they provide the natural experiment opportunity to compare two or more contrasting postulated environmental causes by holding genetic influences constant rather than by determining the possibility of genetic mediation.

Twin–Singleton Comparisons

The starting point for this strategy was the well-established finding that as a group twins lagged behind singletons

in their language development by about three months at 3 years of age (Rutter, Thorpe, Greenwood, Northstone, & Golding, 2003; Thorpe, Rutter, & Greenwood, 2003). The question to be investigated is why there was this language delay. The two leading contenders for the postulated environmental cause were obstetric complications (because they are known to be much common in twins) and altered patterns of parent–child interaction arising from having to deal with two children of the same age at the same time (Rutter & Redshaw, 1991). To focus on influences likely to be important in the general population, children born before 34 weeks of gestation were excluded on the grounds that other research had shown that this was associated with a substantial increase in brain damage (Rutter et al., 2003). As ever, sample selection was crucial in enabling a focus on features that were likely to operate in both twins and singletons as well as in the general population as a whole.

The natural experiment opportunity derived from two main features. First, although genetic influences will operate on individual differences in language, there is no reason to suppose that they will differ in either strength or type between twins and singletons. This meant that the problem of possible genetic mediation could be set aside. Second, overall social disadvantage, which is associated with language impairment in the overall population, is not particularly associated with twinning. Accordingly, the comparison virtually rules out the two major confounding factors found in the general population.

Focus on within-individual change provides a much better assessment of a causative influence than relying entirely on between-group differences. Accordingly, analysis of language performance at 3 years took into account the children's language at age 20 months. In other words, the outcome at 3 years was a reflection of changes between 20 months and 3 years. In this and other connections it is important that both the twins and singletons came from the same general population sampling frame—namely, the Avon Longitudinal Study of Parents and Children (ALSPAC). The findings showed no effect of obstetric complications but a significant effect of parent–child interaction and communication. When a composite score of the latter was introduced into the overall model, it eliminated the twin–singleton difference; that is, it showed complete mediation (Baron and Kenny, 1986). Several methodological steps made the causal inference plausible. Nevertheless, the design involved the limitation stemming from the uncertainty over the measurement of the key features of parent–child interaction as well as from the uncertainties over the generalization of the findings to populations of singletons. There is no reason to doubt the evidence showing the exclusion of any major effect of obstetric complications, but questions remain on the key causal elements of mother–child interaction, particularly as father–child interaction was not measured.

Adoption as a Radical Change of Environment

The English and Romanian Adoptees (ERA) study set out to examine the long-term effects of profound institutional deprivation (Rutter & Sonuga-Barke, 2010). This constituted a natural experiment for at least five different reasons. First, the children were admitted into institutional care in early infancy (thereby eliminating the usual confound of children being admitted when older because of identified impairment). Second, scarcely any children were adopted or returned to their biological families before the Ceauşescu regime fell in 1989 (thereby avoiding the usual biases associated with which children remained and which left institutional care). Third, it was possible to pinpoint accurately the timing of leaving institutional care. Fourth, the move from profoundly depriving institutional care to generally well functioning involved an exceptional marked environmental change. Fifth, the design involved a follow-up to at least the age of 15 years, with multimodal assessments at 6, 11, and 15 years.

Although the adoption created a quite exceptional natural experiment, it was necessary to go on to check whether there could be a bias resulting from variations in the parents' main reasons for adoption (altruism versus a response to infertility). Because none of these variations were associated with outcome, it appeared unlikely that these could constitute a significant bias.

In the 2010 Monograph, the institutional care, as defined, included a few children who had spent short periods of time in institutional care, and main analyses were done with both institutional deprivation and subnutrition. Both of these limitations were dealt with by a further study, based on the same sampling and data, of children who spent the whole of their period prior to adoption in institutional care and who had not shown subnutrition as indexed by body weight at the time of leaving the institution to enter the adoptive family (Rutter, Kumsta, Schlotz, & Sonuga-Barke, 2012). The findings of this group were closely comparable to those in the 2010 Monograph, with the effects of institutional care being, if anything, rather more marked.

The postulated causal effect of institutional deprivation can be tested in two different ways. First, there was the test of developmental recovery following adoption. Findings showed major recovery; these indicated that the initial deficits at the time of leaving institutional care were likely

to have been caused by the institutional deprivation. By the nature of the design, it was not possible to differentiate between the benefits being due to the cessation of a depriving institutional environment or the positive benefits of rearing in an adoptive home. The follow-up to age 15 indicated that variations in outcome were unassociated with variations in the quality of the adoptive home, at least as measured. Nevertheless, it does not rule out the possibility that the massive gains after leaving institutional care were in part contributed to by the quality of the adoptive family environment.

Second, because the recovery was incomplete in many instances, it was necessary to determine whether the deficits were a function of the preadoption institutional environment or variations in the qualities of the postadoption family environment. Several key findings pointed strongly to an institutional effect. First, the associations with institutional deprivation lasting beyond the age of 6 months were as strong at 15 years as they had been at 11 years and before that at 6 years. Second, the deficits were largely of a kind (such as quasi-autism or disinhibited attachment) that is rare in children not experiencing institutional deprivation. Third, the variations in outcome were not accounted for by variations in the postadoption environment. Fourth, institutional deprivation, even in the absence of subnutrition, was associated with a large and significant reduction in brain size that showed partial mediation with respect to the psychological outcomes. Various other methodological checks were also undertaken to rule out nondeprivation causes. As a result of these multiple design features, a causal inference seems well justified. Nevertheless, limitations remain. To begin with, no valid evidence on the biological parents was available, making it difficult to rule out all possibilities of genetic mediation. Also, although the findings were compellingly consistent, there was noteworthy heterogeneity in outcome, and the causes of this variation remain uncertain. Most crucially, although there is good reason to judge the internal validity as very high, questions remain on the extent to which the findings can be generalized to other forms of deprivation in other populations (Rutter & Azis-Clauson, 2011).

Time-Series Designs

Interrupted time-series designs played a major role in the development of quasi-experiments. They required a treatment that could be deliberately manipulated and implemented. In other words, their power lay in the investigation of treatment effects. Shadish et al. (2002) argued that concomitant time-series designs that did not involve introduction of an intervention element were inadequate for testing of causal inferences. That is because, in essence, they simply provide a correlation between two variables that have varied over time, and, on its own, this correlation certainly provides no justification for a causal inference. However, in recent years, improvements and both design and analysis have provided a way of using these as a natural experiment. Two rather different examples illustrate what can be achieved.

The first example examined the question of whether gang membership had a causative environmentally mediated influence on the level of delinquent activities. Many cross sectional studies have documented that gang members have higher delinquency rates than nonmembers (see, e.g., Spergel, 1990), but the question was whether that was a consequence of variables associated with selection into gang membership or socialization as a result of gang membership. Thornberry, Krohn, Lizotte, and Chard-Wiershem (1993) were the first to use longitudinal data to contrast these two hypotheses. They used contrasts between periods when the youths were members of gangs as against those before they entered the gang and after they left the gang. This pioneering early study relied on simple descriptive measures without adjusting for measured or unmeasured characteristics of the boys that might bias the findings. A similar approach had been used earlier by Kandel (1978), who determined whether the similarities in behavior and interest among friends were a reflection of selection or socialization. More recent research has used standard regression models to adjust for measured controls and random effects models to adjust for unmeasured stable characteristics of the boys (Thornberry, 1998; Thornberry, Krohn, Lizotte, Smith, & Tobin, 2003).

Gordon et al. (2004) extended the methodology by estimating fixed effect models to adjust for unmeasured biases, and the possibility that the approach did not control for age. It was possible that a failure to control for age had led to either an over- or underestimate of the effects of gangs. Also, the earlier studies had not considered gang membership that occurred prior to the onset of the study. Gordon et al. dealt with that by using retrospective reports of gang membership before the study began.

Their analyses were based on the Pittsburgh Youth Study. They also looked at whether peer delinquent activity had effects on delinquency that differed from those on drug use. The findings showed that boys who joined gangs were more delinquent before entering the gangs than those who did not join. Nevertheless, even with such selective differences the period of gang membership was associated with a substantial increase in both delinquency

and drug use. Moreover, delinquent activities reduced markedly when the individuals left the gang, although not necessarily right down to the pregang membership level. As the authors point out, the models they employed could not adjust for time varying effects brought about by some third variable associated with both gang participation and increased delinquency in a particular time period. On the other hand, models did adjust for such variables associated with the boys' age, with season and year, or with peer delinquency.

The findings provided convincing evidence that gang membership did have an environmentally mediated effect on delinquent activities. However, in order to understand the mechanisms involved, it would be necessary to have much more detailed data on the boys' peer networks. So it is not possible to rule out entirely the possibility of reverse causation. For example, a boy might exit a gang and reduce his own delinquency because the opportunities for delinquency had diminished. A combination of trajectory analyses and propensity score matching provided a powerful alternative approach (Haviland, Nagin, & Rosenbaum, 2007). This gave rise to similar findings, but it was notable that it was not possible to create matches for the tiny group who showed severe chronic delinquency prior to joining a gang.

Rather comparable parallel issues arise in testing the hypothesis that marriage leads to a reduction in delinquent activities. Again, the question is whether the difference found in other studies reflected selection into marriage or a causal effect of marriage. Using their long-term follow-up to age 70 of the Gluecks' serious adolescent delinquency sample (Glueck & Glueck, 1968), Sampson et al. (2006) addressed this issue by determining whether individuals were less likely, over time, to engage in crime during their periods of being married than during their periods of nonmarriage. The natural experiment aspect arose from the fact that in the United States, there was a very high rate of divorce and also of remarriage. Accordingly, there was a substantial sample of individuals who could be studied before they married, during their marriage, and after their marriage had broken up. In some instances, there were replications of this series of experiences.

The starting point was the use of 10 individual-specific and 10 family and parental background features and a range of adult time-varying covariates to assess selection into marriage. They used an inverse probability of treatment weighting (IPTW), devised by Robins et al. (2000), to weight each person and period by the inverse of the predicted probability of receiving the experience (i.e. marriage) that they actually received in that period. In other words, the model in effect created a pseudo-population of weighted replicates allowing a comparison of status (married versus unmarried) without making distributional assumptions about counterfactuals. The model also allowed age effects to be taken into account and thereby a quantitative estimate of marriage effects on crime.

The findings showed an average crime reduction of some 35% in periods of marriage (using a conservative approach rating ruling out reciprocal effects and imposing a strict causal order). The robustness of the estimate was shown by the demonstration that it was broadly similar across both the shorter time span of 17–32 years and the longer time span of 17–70 years. As with any other nonexperimental study it was impossible entirely to rule out the possibility of the operation of some unconceptualized and unmeasured confounder. Nevertheless, as the authors argued, given the unusually rich range of measures available, it is difficult to imagine what time-stable or time-invariant covariate could overcome the magnitude and robustness of the marriage effect. Propensity scores provide a powerful tool but they can only be as effective as the variables that go into the propensity score. The key predictors are omitted then that will inevitably reduce the benefit of the propensity score approach.

Another example of a time-series approach is provided by Rutter, Maughan, Mortimore, Ouston, and Smith's (1979) study of school effects using changes over time to assess possible environmentally mediated causal effects. The hypothesis being tested was that the qualities of schools influenced the attainments and behavior of the children attending these schools. A competing alternative hypothesis was that any associations found reflected either variations in the intake of pupils to each school or the influence of the pupils on school functioning. The natural experiment element was provided by the fact that the transfer from elementary to high schools in London at that time involved a marked lack of continuity between the two so that the children at any one elementary school moved to a large number of different secondary schools. Data were available for pupil functioning pretransfer (at ages 11–12), at the end of compulsory schooling, and at the end of compulsory schooling five years later. There was also a later follow-up after leaving school. To test the hypothesis, it was of course essential to have detailed measurement of school qualities and these were obtained through observations, interviews, questionnaires, and the like. It was argued that if the within-schools variations observed for pupil outcomes were truly a function of school influences, then the differences should not be accounted for by intake differences. Instead, they should be a function of measured

features in school quality and the effect should increase over the course of schooling.

In other words, the association between school qualities and pupil behavior should be greater at the time of leaving school than it had been at the time of intake. The findings supported this argument. The causal inference was further strengthened by comparable findings in other longitudinal studies and by the beneficial effects on pupil outcomes with the appointment of school principals in two out of three failing schools on the point of closure (see Rutter & Maughan, 2002). The main, substantial limitation is the need to rely on group tendencies rather than within-person change at the individual level for specifics of school experience. That was not available in the research, but, in principle, it could be available and if that were the case it would greatly strengthen the natural experiment.

Designs for Dealing with Selection Bias

Ordinarily, selection biases seriously interfere with any attempts to test environmental causation. That is because most risk experiences derive from situations influenced by people's behavior in selecting or shaping their environments. The strategy for dealing with this relies on the study of experiences that are not open to personal choice because the influences of personal choice have been excluded. These may involve either the introduction of risk experiences or their removal.

Universal Introduction of Risk

Both the examples here concern famine. The first concerns the Dutch famine during World War II (Stein, Susser, Saenger, & Marolla, 1975). The famine was both severe and time limited, and it was imposed on the total population and not just on a socially deprived subgroup. With respect to psychopathology, the main interest derived from the finding of a twofold increase in the risk for schizophrenia (as assessed on a national registry) that was associated with exposure to famine conditions in early gestation. Causal inference was substantially strengthened by a similar effect on congenital anomalies of the central nervous system—thereby suggesting a possible biological pathway (McClellan, Susser, & King, 2006). In addition, studies comparing the epigenetic findings for individuals prenatally exposed to famine and their unexposed same-sex siblings (Heijmans et al., 2008; Tobi et al., 2009, 2012) found that famine exposure resulted in a lower level of methylation of the locus for the insulin-like growth factor II. Again, the possible biological effect supported the causal inference. Causal inferences were also strengthened by the gestational period specificity and the diagnostic specificity. The main limitation of this study concerns the reliance on group exposure, rather than individual exposure. The possibilities for selection bias and selective survival were examined in detail and no support for their operation was found. Nevertheless, it is likely that a few individuals were able to use their initiative in obtaining some food. This could have introduced a very small amount of bias but what it would mean is that it would give rise to a reduction in the case–control difference. Thus, in so far as such a bias occurred, it would lead to an underestimate of the famine effect, and not an overestimate.

The second example is provided by the Chinese famine in 1959–1961 (St Clair et al., 2005), which showed a very similar twofold increase in the risk for schizophrenia. The causal inference was again strengthened by the biological parallels, that is, the reduction in birth weight and the increase in mortality rate associated with the famine. However, as in the Dutch example, there was the limitation of reliance on group differences but with the additional uncertainty in the Chinese case regarding the exact timing of the prenatal exposure. Both of these were good studies, and they provide a strong causal inference regarding environmental mediation of famine effects on child outcomes. Moreover, although not able to show a biological effect at the individual level, the finding did suggest a probably relevant biological pathway.

Universal Removal of Risk

The same basic strategy may be applied to the removal of risks. Costello, Compton, Keeler, and Angold (2003) used a natural experiment in which a casino was opened on an American Indian reservation to determine if the alleviation of poverty brought mental health benefits. Federal law in the United States required that a particular proportion of the profits of all casinos set up on a reservation had to be distributed to all those living on the reservation without any actions by individuals. By good fortune, the casino was set up during a prospective longitudinal study of child mental health—the Great Smoky Mountains study. The natural experiment involved several different elements. First, it was possible to check whether changes over time in the rate of poverty in American Indians were paralleled by similar changes in the White population. It was found that it was not. Second, the change over time allowed for a comparison between those who were not in poverty before or after the casino was set up, those who were poor before and who remained poor afterward, and those who started poor but ceased to be below the poverty line after the

casino was established. The interest, obviously, concerned the third group. The findings showed that the substantial reduction in poverty was followed by the reduction in the rate of some (but not all) kinds of child psychopathology. More detailed post hoc analyses were undertaken to try to identify what were the changes in the family brought about by the reduction of poverty. These showed that the relief of poverty had been accompanied by an increase in parental employment but also by an increase in time spent with the children, with associated greater supervision. Because these family effects were not part of the design, the causal inference is necessarily less certain. However, there was the implication that what mattered was what the relief of poverty did for the family rather than any direct effect on the child.

A further follow-up was undertaken at ages 19 and 21 years, with child interview plus criminality data from the courts and schooling and education information (Akee, Copeland, Keeler, Angold, & Costello, 2010; Costello, Erkanli, Copeland, & Angold, 2010). As is often the case, the much increased duration of follow-up allowed the use of cohort differences to examine the effects of the age of the individual during the period of income supplements and the duration of receiving them to be examined. In addition, mediation analyses in relation to within-individual changes over time became possible. Appropriate detailed analyses were undertaken to check whether findings might have resulted from general secular change; the analyses showed that they were not explicable on these grounds. In brief, it was found that the income supplements led to higher levels of education in early adult life and a lower rate of convictions for minor offenses. Furthermore, income supplements were associated with a lower rate of alcohol abuse and dependence and cannabis abuse and dependence. There were no effects on nicotine or other drug abuse or on emotional or behavioral disorders. The effects were greatest for those who were initially the poorest and for those that experienced the longest time of income supplements. There were no detectable benefits of income supplements that began only after childhood. The findings suggested (but did not prove) that mediation was provided by improved parent–child relationships. There was no indication that families used the lump sums received to buy different levels of long-term benefits.

The findings were striking with respect to the size of effects and their persistence into adult life. Two features stood out as different from other studies. First, the income supplement was very large—amounting to some quarter to a third of the original income—and, second, there was no expected end to the income supplements so that the individuals treated them as permanent. The authors noted that the study was undertaken in a mixed urban–rural area, and it is not known if similar results would occur in deprived inner-city settings. What is clear is that this natural experiment design provided compelling evidence of the causal effect of relief of poverty. As with other designs, the findings are less definitive on the precise mechanisms involved in the causal effect. From a practical point of view, too, the importance of a huge permanent increase in income makes it uncertain whether similar, albeit lesser, benefits might accrue from smaller, less-lasting income increases.

Another example of strategy of a universal intervention applying to the removal of risk is provided by Honda et al. (2005) with the natural experiment concerning the hypothesis that measles, mumps, rubella (MMR) vaccine was responsible for the marked rise in the rate of diagnosed autism spectrum disorder (ASD). The experiment derived from the fact that Japan stopped using the vaccine (because of the concern over the mumps component, which was not any part of the hypothesis about the adverse effects of MMR) at a time when its use was continuing in other parts of the world. The strength of this natural experiment lay in the availability of systematic standardized diagnostic data for a defined geographical area and an exact timing of the total withdrawal of the use of MMR vaccine in the follow-up to age 7 years (to ensure that no cases of autism were missed) and in the fact that the ASD rates elsewhere were still rising. The findings showed that the withdrawal of MMR had no effect on the rising trajectory in the rate of ASD. In addition to a variety of other methodological checks, the findings made the universal causal effect of MMR on ASD implausible, especially in light of the fact that other research strategies all gave rise to the same negative conclusion (Rutter, 2005). Note that, although the finding virtually rules out the possibility of MMR causing an overall rise in ASD, it cannot rule out the possibility of an idiosyncratic effect in a small minority of individuals, although there was no indication that this was the case and there was a complete absence of any known means of identifying such an idiosyncrasy.

Much the same strategy was used, with similarly negative findings, on the withdrawal of Thimerosal (a mercury preservative) from vaccines in Scandinavia in the early 1990s, at a time when the preservative continued to be used in the rest of the world. The initial negative finding (Madsen et al., 2003) was open to the objection that account had not been taken of the change of registry procedures. However, a further later study, on the same data set (Atladóttir et al., 2007), confirmed the negative

finding. With both of these studies, the key thing was that the decision as to whether or not to have the vaccine was a governmental decision with respect to the population as a whole and it was not open to individual choice. Indeed, without getting the vaccines from abroad, the removal of the vaccines would have effectively prevented personal choice.

Instrumental Variables

The use of an instrumental variable as a means of testing causal inferences involves two key design features. First, the instrumental variable must be outside the control of the individual (to avoid allocation bias), and, second, it must affect the outcome by some means that is independent of the usual liability to that outcome (to avoid both possible genetic mediation and possible social confounding). In other words, instrumental variables refer to circumstances that do not affect the outcome being studied but do influence the putative risk factor being considered (Foster & McLanahan, 1996). Instrumental variables are often used as part of a statistical approach designed to deal with omitting confounding variables, two-way influences, and imprecision in measurement (Angrist & Evans, 1996; Angrist, Imbens, & Rubin, 1996). However, here we are considering instrumental variables only in the narrower framework of their role being used in a natural experiment. Two examples are used to illustrate the strategy.

Early Puberty

An unusually early puberty constitutes a natural experiment because it has been associated with an increased rate of drinking alcohol and of drunkenness during the teenage years (Grant & Dawson, 1997). In the general population, early use of alcohol has been strongly associated with an increased risk of alcoholism in adult life. The causal hypothesis, therefore, is that early alcohol use contributes to the causation of later alcoholism. Early puberty constitutes an instrumental variable because it is outside the control of the individual. If early use of alcohol truly predisposes a person to alcoholism, the same early-use effect should be found even if the early use was a result of early puberty rather than an overall alcoholism liability. The findings across three large-scale general population epidemiological studies in Sweden, New Zealand, and Finland are consistent (Caspi & Moffitt, 1991; Pulkkinen, Kaprio, & Rose, 2006; Stattin & Magnusson, 1990), and all showed that early puberty was indeed associated with an increased rate of drinking alcohol and with drunkenness during the teenage years. The test, however, lies in what is found in early adult life. All three studies are consistent in showing that, although there was an effect on alcohol use in adolescence, this was no longer apparent by the mid-20s. In other words, the effects were quite different from those found in ordinary circumstances, carrying the implication that early use was not a causal factor despite its strong correlation. Rather, it reflected the same underlying liability. Longitudinal studies tell the same story. Thus, McGue and Iacono (2005) showed that early alcohol use was associated not only with later alcoholism but also with smoking, the use of drugs, involvement with the police, early sexual activities, conduct problems, and educational underachievement. The inference is that in the case of the timing of first alcohol use it is likely that there is not a causal effect; rather, the early use reflected a shared liability to a broader range of problem behavior.

China's One-Child Policy

A rather different instrumental variable was provided by China's one-child policy (Cameron, Erkal, Gangadharan, & Meng, 2013), which was introduced in 1979. Before that time, the psychological effects of being an only child were confounded by family features associated with a decision to have just one child. This became obsolete post-1979 because the government made the decision. Cameron et al. (2013) compared young people living in Beijing and born in 1975 or 1978 with those from the same area born in 1980 or 1983. Because the one-child policy was anticipated just before its introduction, the 1975 cohort provides a cleaner comparison. The proportion of only children rose from 27% of those born in 1975 to 91% for those born in 1983, and the average number of siblings fell from 0.97 to 0.12. By contrast, there was little difference in the size of the extended family as indexed by the number of cousins. The psychological effects of being an only child had been thought to include self-centeredness, lack of cooperativeness, and difficulty getting on with peers. Cameron et al. (2013) used two standard economic games (i.e., Dictator and Trust) as well as questionnaires to tap the psychological traits. Using the one-child policy as an exogenous instrument, the findings showed that the psychological effects of being an only child were valid and not just a consequence of parental choice. One strength of the study was the use of experimental methods to assess altruism and trustworthiness.

A somewhat comparable approach was used with the Argentine draft lottery to study the effects of military service on later criminal activities (Galiani, Rossi, & Schargrodsky, 2011). The opportunity to make this a natural experiment arose from the practice of random

assignment by lottery to determine who actually was conscripted for compulsory military service. The findings were clear-cut in showing that military service increased the likelihood of a later criminal record.

Sex of Co-Twin

Atterman et al. (2012) used the sex of a co-twin as the instrumental variable for prenatal exposure to testosterone in a study to examine effects on ADHD and ASD traits. The findings went in the opposite direction to prediction, and it seems likely that this reflected either parental reporting bias or confounding. The idea was a good one, but the findings are not really interpretable.

Mendelian Randomization

Mendelian randomization (MR) was first described as a natural experiment strategy by Katan (1986) and then by Gray and Wheatley (1991), but it has been more fully developed by Davey Smith and Ebrahim (2003, 2005). At first sight, the term MR sounds misleading because genes are, of course, not randomly distributed in the sense of everyone having the same chance of receiving particular genetic variants. Nevertheless, the allelic variations in the parental genes are randomly distributed among offspring at the stage of meiosis, as noted in Mendel's second law. What this means is the inheritance of any one trait is independent of the inheritance of other traits. The ingenious point with respect to using MR to test a hypothesis regarding environmental causation is that the allelic variations are likely to be randomly distributed with respect to the environmental risk hypothesis being tested. Cases where the pathway from genotype to phenotype is relatively well understood, and, when the gene in question has functionally varying alleles, allelic status can be used as a randomly distributed proxy for the behavioral differences that emerge from it (Irons, McGue, Iacono, & Oetting, 2007). In other words, the groups defined by the allelic variation known to affect exposure to some intermediate phenotype (such as drinking behavior) can be used to study the effects of variation in that phenotype on some disease or disorder hypothesized to be caused by the intermediate phenotype. The design was employed originally to rule out reverse causation; it does that because the genotype is unaffected by the disease, disorder, or trait being studied. The design is also not invalidated by confounding, provided there is no pleiotropic effect of the regulatory allelic variation (Keavney, 2004).

The design was originally developed for use in internal medicine to determine if the disease being studied had caused the environmental risk factor rather than the other way around. It has had some success both in providing support for a causal hypothesis (Casas, Bautista, Smeeth, Sharma, & Hingorani, 2005; Jokela et al., 2012) and in casting doubt on its validity (Keavney et al., 2006; Lawlor et al., 2011). Nevertheless, it usually requires huge samples, and it is weak if the risk effect is small, if there are difficulties in measuring the intermediate phenotype, or if the risk operates only above some threshold (Meade, Humphries, & De Stavola, 2006).

The strength and limitations of the MR design, compared with randomized controlled experiments, have been well discussed by Nitsch et al. (2006) and by Tobin and colleagues (2004) (see also Davey Smith, 2010; Kivimäki et al., 2011; Tobin, Minelli, Burton, & Thompson, 2004). Glymour, Tchetgen, and Robins (2012) usefully noted the key assumptions of the MR design (and other instrumental variable approaches) as follows: (1) the genotype is associated with the intermediate phenotype (the IV); (2) there are no unmeasured common causes of both the phenotype and the outcome being examined; and (3) every directed pathway from the genotype to the outcome passes through the phenotype (the IV0). They argue for the value of DAGs for spelling out the assumptions and for suggesting ways in which they may be tested. They conclude that usually it is not possible to prove the assumptions, but it is often possible to show that the putative IV is invalid.

Irons et al. (2007) used the MR design to examine the hypothesis that heavy early alcohol use had a gateway effect leading to the later use of hard drugs such as cocaine or heroin and to a broader range of antisocial behavior. That there is a substantial overall statistical association in the general population is not in doubt, but the query is whether it derives from the general liability to disinhibited behavior (whether influenced by either genetic or environmental factors) or whether the intermediate phenotype of heavy drinking causes the other problems. The opportunity to employ the MR design was provided by an allelic variation of a gene that leads to greatly reduced aldohyde dehydrogenase (ALDH) enzyme activity. Individuals with ALDH2 enzyme deficiency have an unpleasant flushing response to any ingestion of alcohol. The relevant gene is found almost exclusively in East Asians and, in this ethnic group, it is associated with a substantially decreased rate of alcoholism compared with those having normal enzyme activity.

Irons et al. (2007) were creative in realizing that the design could be used in their study of adoptees born in Korea and placed in adoptive homes in the United States in infancy, none of the adopting parents being of East Asian descent. Findings showed (as expected) a substantial genetic effect on alcohol abuse, but there was

no significant difference between the ALDH2 deficient and nondeficient groups in either drug abuse or antisocial behavior. The implication was that the association derived from a shared general liability for problem behavior and not from the causal effect of the intermediate phenotype of early alcohol drinking.

As with all natural experiments, it is desirable wherever possible to include more than one design element to separate variables. Adoption provided that further opportunity here. The results showed no effect of adoptive parental alcoholism, which confirmed the twin studies' conclusions of a genetic influence that will not apply when the parentage is nonbiological. On the other hand, it did show a significant effect of drinking by siblings, suggesting that it may well represent an environmental effect. Irons et al. (2007) emphasized that, although the ALDH2 allelic variation has a strong effect on alcoholism, it is not determinative. Not only do some individuals with ALDH2 deficiency become alcoholic, but also there is a gender difference in effect and a secular trend. It was necessary for Irons et al. to take account of these limitations in their analyses, as they did. Caution is needed even when the genetic effect is strong (as it clearly is the ALDH2) if environmental influences also affect the genetic pathway to the phenotype. MD works best when the relevant gene has a strong, highly focused effect; when pleiotropic effects are slight; and when there is a well-defined intermediate phenotype (Katan, 2004; Keavney, 2004).

We conclude that MR constitutes a useful addition to the natural experiments armamentarium, but equally it is likely to be practical in only a few circumstances. When it is necessary to combine samples to obtain the necessary sample size (which it was not in Irons et al., 2007), there are the further problems of ensuring comparability across samples in all relevant respects.

Regression Discontinuity Designs to Deal with Unmeasured Confounders

The only actual natural experiment that can fully deal with unmeasured confounders is the regression discontinuity (RD) design. This was first introduced by Thistlethwaite and Campbell (1960) over half a century ago as an alternative to randomized controlled trials. The key defining feature is that allocation for some planned intervention is by means of an assignment variable that uses a strict predetermined cutoff rather than randomization. In other words, the design capitalizes on a major selection bias, provided, and only provided, that it is under strict control. The basic point is that assignment cannot be caused by the intervention. It does not matter whether or not it is related to the outcome. It does, however, require that all participants belong to the same population prior to assignment. Moreover, the statistical analysis also requires accurate specification of the intervention effect (e.g., whether it is linear or curvilinear) and inclusion of an interaction term when that is appropriate. Unlike an RCT, effects are measured in terms of a discontinuity in regression lines (hence its name) instead of a difference in means. Although, at first sight, it is not obvious that RD allows an unbiased estimate for causal effect, it has been shown mathematically that it does (Rubin, 1977; Shadish et al., 2002) and this constitutes its major advantage (Laird & Mosteller, 1990). Because RD was designed as an alternative to RCT for planned experimental interventions, there is rather limited use as a natural experiment to test prior causes of a nonexperimental variety.

Use of RD to Assess Schooling Influences

Cahan and Cohen (1989) showed the applicability of RD in their study determining whether the amount of formal education, as opposed to increasing chronological age, had an effect on cognitive performance. The design was possible because all children in Jerusalem's Hebrew-language, state-controlled elementary schools (i.e., other than those providing only special education) in 1987 had a single admission date in December. This meant that, within each school year, there was a 12-month age span from the oldest to the youngest child (all of whom would have received the same amount of schooling), and between school classes there was a 12-month difference in duration of schooling (but a similar within-group variation in age). There was a potential problem with the key assumption that there could be no exceptions to the age cutoff. Although grade retention and grade skipping were rare, admission was sometimes delayed and sometimes accelerated. Most crucially, this variation was nonrandom (with variation greatest near the cutoff point) and with delayed admission students tending to be less able, whereas accelerated admission students were more able. To keep this bias to a very low level, Cahan and Cohen (1989) excluded all overage and underage children in each of the three age-specified year classes studied. It also excluded all children born in November and December.

To allow direct comparison between tests, researchers standardized effect sizes by using the pooled within-age standardization in Grade 4 (the youngest age group). The findings were striking in showing that the school effect exceeded the age effect for 10 of the 12 cognitive measures used. The two exceptions were both figural tests.

The greatest school effects were found for verbal tests and also for the one numerical test employed. There was no reason to expect anything other than a linear effect, although it might have been different in a less standardized school system. It should be noted that, although the data were not individually longitudinal, they did represent changes over time. Thus, although technically a cross sectional comparison, the RD design (by ensuring strict comparability of groups) allowed the strong inference of within-individual change.

We have described the study in some detail to emphasize the crucial importance of ensuring that the RD design assumptions are truly met and to illustrate a few of the practical steps that need to be taken to bring this about. The one key assumption that was not explicitly tested was the absence of an age x schooling interaction but the figures provided for the three-grade study gave no indication that there was an interaction. It should be noted that the findings are in keeping with a somewhat different form of natural experiment provided by forced school closure (Rutter & Madge, 1976). None of these schooling experiments tested the possibility of an interaction between schooling effects and the quality of family influences. Nevertheless, the findings from studies of gains and losses in the long U.S. summer school vacation indicated that this was highly likely (Entwisle, Alexander, & Olson, 2004; Heyns, 1978).

Use of RD to Study the Effects of Head Start

The second example of an RD design is provided by a retrospective assessment of the effects of Head Start using a discontinuity in program funding across counties in the United States as the research lever (Ludwig & Miller, 2005). During spring 1965, the US Office of Economic Opportunity provided technical assistance to the 300 poorest counties to develop Head Start funding proposals. Funding rates were found to be 50 to 100% higher in counties with poverty levels just above the cutoff. The treatment group comprised those just above the cutoff, and the control group comprised those just below. Because the cutoff was on a predetermined variable, the design was operative, and it allowed little opportunity for strategic behavior by individuals that could invalidate the required assumptions. The findings derived from somewhat rough county-level funding data and from the National Educational Longitudinal study, which tracked a national sample of eighth graders in 1988. The funding difference persisted through the late 1970s. The findings showed a substantial effect of Head Start on children's health, a lesser but still significant effect on school graduation, but no significant effect on reading or math scores or on noncognitive outcomes.

The validity of the findings was tested by determining whether similar effects were found in age groups that could not have benefited from Head Start (finding that they were not) and whether the health benefits applied to outcomes that could not plausibly be influenced by Head Start (they did not). The important limitation stemmed from reliance on countywide measures, from reliance on residence at follow-up rather than at program initiation, and from the lack of satisfactory data on in and out migration. The study demonstrates the uncertainties that usually operate when seeking to apply the RD design to naturally occurring circumstances outside of experimental control. The RD design provides an important alternative to RCT, but it is quite limited in its applicability to naturally occurring prior risk or protective factors.

McCall and Bielby (2012) provided a useful review of the use of fuzzy RD designs in the educational arena. They indicated that RD can be applied even when requirements are not fully met provided that stringent tests of the assumptions are employed. Whilst we admire the ingenuity of the design we remain very cautious over its general applicability because it has rarely given rise to clear-cut conclusions.

Adoption and Fostering as a Means of Separating Prenatal from Postnatal Effects

With many supposedly prenatal effects, there is the problem of differentiating between risks mediated during the pregnancy and risks that stem from the postnatal environment. For example, some longitudinal studies have shown that fetal cocaine exposure was followed by adverse effects on neurodevelopment and cognitive performance (see, e.g., Jacobson, Jacobson, Sokol, Martier, & Chiodo, 1996; Singer, Arendt, Minnes, Farkas, & Salvator, 2000). However, mothers who take cocaine during the pregnancy are highly likely to take cocaine postnatally, and the sequelae could derive from mother's overall liability to take cocaine. Equally, it could derive from environmentally mediated postnatal effects stemming from adversities in rearing that tend to be more common in drug dependent mothers.

One way forward is to compare drug-exposed infants who are reared by their biological mothers and those reared by a foster or adoptive parents. Thus, Singer et al. (2004), as part of a longitudinal study from birth, compared the outcome at 4 years of age of 48 prenatally cocaine-exposed infants reared by their biological families and 42 reared by adoptive or foster parents. The fostered and adopted children had only a very slightly higher mean IQ, but there was a parallel substantially lower proportion of them

with an IQ below 70 (37% versus 10%). The implication was that the cognitive deficit might be due to the prenatal cocaine exposure—as that will not have been corrected by the postnatal rearing in adoptive and foster families. It was important to check, however, whether the fostered and adopted children had less prenatal cocaine exposure. The findings rule that out because prenatal cocaine exposure was actually twice as high—probably because this was the reason the children had been removed from the biological mother. Genetic risk could not be excluded, but it seemed unlikely to constitute a sufficient explanation. On the other hand, what was problematic is that the prenatal cocaine exposure was also accompanied by a greater use of other drugs (including alcohol and tobacco), so the risk might involve substances other than the cocaine itself.

Moe (2002) used a somewhat similar design in her focus on prenatal drugs and alcohol exposure, but she used a matched control group that had not experienced prenatal exposure. She found that the cognitive functioning at 4.5 years of age in substance-exposed children who were adopted or fostered in infancy was substantially and significantly lower than that in the control group. The findings supported the inference that the adverse effects on cognition and development were primarily a function of prenatal substance exposure rather than an exposure to an adverse rearing environment.

The strategy in these studies provides a useful means of separating prenatal and postnatal effects, but, on its own, it is much less informative regarding either the precise risk feature (because prenatal exposure will often involve several substances) or the particular risk mechanisms.

ARE NATURAL EXPERIMENTS REALLY NEEDED?

We have argued that there are serious limitations to relying on statistical control to deal with confounders. However, we still have to ask what difference it makes in practice. With respect to the effects of exposure in utero to maternal smoking, three studies have used natural experiments (employing three different designs), each of which showed that the effect was likely to be largely, if not entirely, due to genetic mediation. The same three studies also sought to compare the findings using the standard regression techniques for dealing with confounders. All three showed that the effect that the natural experiment results found to be a function of genetic mediation was wrongly shown to be a true environmentally mediated effect using the standard regression approaches.

However, we need to turn to other research to see how far this is a general problem. Duncan, Magnuson, and Ludwig (2004) did a systematic comparison of experimental and nonexperimental estimates for effects of school class size on student test scores. The findings as a whole showed an average bias of the order of about 10 percentile points. This is about the same as the average experimental estimate for the effects of smaller classes and clearly constituted an important bias (Wilde & Hollister, 2002). A further example came from the moving to opportunity (MTO) housing voucher experiment: this randomly assigned residence in a high-poverty neighborhood to either a group that was offered a housing voucher or a control group that received no ability assistance. Because of the well-implemented random assignment it was possible to generate comparisons of nonexperimental and experimental estimates (Ludwig, Duncan, & Hirschfield, 2001).

A third example is provided by a randomized experiment designed to evaluate welfare to work programs in seven sites across the United States. Magnuson and McGroder (2002; cited in Duncan et al., 2004) showed that the nonexperimental estimate did not closely reproduce experimentally based estimates. A systematic review by Glazerman, Levy, and Myers (2002) concluded that regression methods only occasionally produced findings comparable to experimental studies.

While appropriately noting that natural experiments involve problems, Duncan et al. (2004) concluded much greater use needed to be made of them, and they give half a dozen examples of ingenious use of natural experiment strategies—although only some of these used that terminology. Plus, Joy, Kimble, and Zabrack (1986; cited in Duncan et al., 2004) took advantage of the fact that a town in northwestern Canada had a delayed introduction to television because its valley location blinded it to channel transmitters for several years. Two years after the introduction of a television channel, children of all ages showed increased verbal and physical aggression. Rosenbaum (1991; cited in Duncan et al., 2004) compared the effects of relocation to city and suburban addresses for families involved in the Gautreaux program, where favorable outcomes were found for suburban compared with city-based moves. Girotto and Peterson (1999; cited in Duncan et al., 2004) took advantage of the fact that high school students with summer birthdays took drivers' education during the summer (in addition to course work during the academic school year). They found that one additional yearlong academic course was associated with a 0.25 standard deviation increase in standardized test scores. Green, Hoffman, Morse, Hayes, and Morgan

(1964; cited in Duncan et al., 2004) took advantage of the effects of forced school closure as a way of avoiding racial integration and found that a missed year of school resulted in an IQ score that was six points lower. See DeGroot (1951) and Jencks et al. (1972) for other examples of using forced school closure to examine the effects on children or a loss of educational input.

What the studies have done effectively is to make it unlikely that social selection played any part in the effects, thereby doing much to strengthen the causal influence. However, the studies dealt only with the overall effects of schooling, and they were not able to determine what aspect of the schooling it was that made the difference. They made the point that it is important for researchers to be on the look out for natural experiment opportunities to take advantage of opportunities provided. While it is important to make maximal use of approved statistical techniques, it is also crucial to recognize, as pointed out by Shadish and Cook (1999), that design is the key element in natural experiments of all kinds.

OVERVIEW OF NATURAL EXPERIMENT METHODOLOGY

As we indicated in the introduction to this chapter, several rather different methodological hazards can lead to misleading inferences from observational studies that involve no kind of experimental approach. The natural experiments that we have considered differ in which of these hazards they mainly seek to deal with. No strategy is equally appropriate for all the different hazards. Similarly, all research designs involve a range of crucial assumptions. Natural experiments are no exception to that rule. We have sought to note some of the most important assumptions with the different designs and have indicated, where possible, what may be done to test the assumptions. Fortunately, the different forms of natural experiment vary in the assumptions they require. What this means is that conclusions are always likely to be more robust when the same answers are given by several quite different types of natural experiment. The consistent evidence, for example, that prenatal exposure to maternal smoking does not have a causal effect in leading to ADHD or conduct disorder well illustrates that point. Kendler and Gardner (2010) provided another example in which they use both co-twin control and propensity score matching to consider the possible causal impact of stressful life events on major depressive disorder. All natural experiments require appropriate statistical methods, but in some cases statistics are crucial in adding additional means of testing the process being investigated. The example of the effects of membership of a delinquent gang on an individual's engagement in crime constitutes a good example of this kind.

A key limitation of almost all the natural experiments that we have discussed is that the designs are much stronger in showing whether or not there has been environmental risk mediation than they are in showing which particular aspect of the environment provided the mediation. Different designs and different forms of statistical analysis are required to take that absolutely crucial step from identifying a truly causal risk environment to a much more difficult issue of precisely which aspect of the environment provided the risk. That is especially the case when dealing with complex environments and when, as is usually the case, there are bidirectional effects that may vary over time. However, it should no longer be acceptable for researchers (or clinicians) to avoid considering whether a causal inference is justified. Both Rutter (2007) and Foster (2010) pointed out that a high proportion of the literature ignores this important question. Some deal with the problem by arguing that they are dealing only with statistical associations and not causation. However, why anybody should be in the least bit interested in statistical associations, if they carry no implications for causation, remains a mystery. Others make explicit that their research methods cannot test causation; nevertheless, in discussing findings at the end of the paper they suggest that the findings do mean causation. Natural experiments do not provide anything approaching a complete answer to the dilemma of determining when correlation can rightly imply causation, but they do take the field forward in a most important way.

Most crucially, natural experiments should not be thought of as a discrete number of strategies. Rather, they represent an ever-extending list of opportunities open to the alert researcher. Thus, the American Casino Study, the Chinese One Child Policy Study, and the Argentine National Lottery Policy were not designed for the purpose of a natural experiment, but the key researchers spotted that they provided an invaluable natural experiment opportunity. Doubtless, other opportunities are out there waiting to be discovered and acted on.

REFERENCES

Academy of Medical Sciences. (2007). *Identifying the environmental causes of disease: How should we decide what to believe and when to take action?* London, UK: Academy of Medical Sciences

Akee, R., Copeland, W. E., Keeler, G., Angold, A., & Costello, J. (2010). Parents' incomes and children's outcomes: A quasi-experiment with casinos on American Indian reservations. *American Economics Journal: Applied Economics*, *2*, 86–115.

Anderson, K. E., Lytton, H., & Romney, D. M. (1986). Mothers' interactions with normal and conduct-disordered boys: Who affects whom? *Developmental Psychology*, *22*, 604–609.

Angrist, J. D., & Evans, W. N. (1996). *Children and their parents' labor supply: Evidence from exogenous variation in family size*. Working Paper No. 5778. Cambridge, MA: National Bureau of Economic Research.

Angrist, J. D., Imbens, G. W., & Rubin, D. B. (1996). Identification of causal effects using instrumental variables. *Journal of the American Statistical Association*, *91*, 444–455.

Atladóttir, H., Parner, E., Schendel, D., Dalsgaard, S., Thomsen, P., & Thorsen, P. (2007). Time trends in reported diagnoses of childhood neuropsychiatric disorders: A Danish cohort study. *Archives of Pediatric and Adolescent Medicine, 161*, 193–198.

Attermann, J., Obel, C., Bilenberg, N., Nordenbæk, C. M., Skytthe, A., & Olsen, J. (2012). Traits of ADHD and autism in girls with a twin brother: A Mendelian randomization study. *European Child & Adolescent Psychiatry*, *21*, 503–509. doi: 10.1007/s00787-012-0287-4

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.

Bell, R. Q. (1968). A reinterpretation of the direction of effects in studies of socialization. *Psychological Review*, *75*, 81–95.

Bell, R. Q., & Harper, L. V. (1977). *Child effects on adults*. Hillsdale, NJ: Erlbaum.

Berzuini, C., Dawid, P., & Bernardinell, L. (Eds.). (2012). *Causality: Statistical perspectives and applications*. Chichester, UK: John Wiley & Sons Ltd.

Boutwell, B. B., & Beaver, K. M. (2010). Maternal cigarette smoking during pregnancy and offspring externalizing behavioral problems: A propensity score matching analysis. *International Journal of Environmental Research and Public Health*, *7*, 146–163. doi: 10.3390/ijerph7010146

Bresnahan, M., Begg, M. D., Brown, A., Schaefer, C., Sohler, N., Insel, B., . . . Susser, E. (2007). Race and risk of schizophrenia in a US birth cohort: Another example of health disparity? *International Journal of Epidemiology*, *36*, 751–758.

Brown, G. W., Harris, T. O., & Bifulco, A. (1986). Long-term effects of early loss of parent. In M. Rutter, C. Izard, & P. Reed (Eds.), *Depression in young people: Developmental and clinical perspectives* (pp. 251–296). New York, NY: Guilford Press.

Cahan, S., & Cohen, N. (1989). Age versus schooling effects on intelligence development. *Child Development*, *60*, 1239–1249.

Cameron, L., Erkal, N., Gangadharan, L., & Meng, X. (2013). Little emperors: Behavioral impacts of China's one-child policy. *Science*, *340*, 272–273. doi: 10.1126/science.340.6130.272-b

Casas, J. P., Bautista, L. E., Smeeth, L., Sharma, P., & Hingorani, A. D. (2005). Homocysteine and stroke: Evidence on a causal link from mendelian randomisation. *Lancet*, *365*, 224–232.

Caspi, A. (2003). Life-course development: The interplay of social selection and social causation within and across generations. In P. L. Chase-Lansdale, K. Kiernan, & R. J. Friedman (Eds.), *Human development aross lives and generations: The potential for change* (pp. 10–43). New York, NY: Cambridge University Press.

Caspi, A., & Moffitt, T. E. (1991). Individual differences are accentuated during periods of social change: The sample case of girls at puberty. *Journal of Personality and Social Psychology*, *61*, 157–168.

Caspi, A., Moffitt, T. E., Morgan, J., Rutter, M., Taylor, A., Arseneault, L., . . . Polo-Tomas, M. (2004). Maternal expressed emotion predicts children's antisocial behavior problems: Using monozygotic-twin differences to identify environmental effects on behavioral development. *Developmental Psychology*, *40,* 149–161.

Cicchetti, D., & Cohen, D. (1995). *Developmental psychopathology*. New York, NY: John Wiley & Sons, Inc.

Cochran, W. G., & Chambers, S. P. (1965). The planning of observational studies of human populations. *Journal of the Royal Statistical Society. Series A (General), 128*, 234–266.

Coid, J. W., Kirkbride, J. B., Barker, D. J., Cowden, F., Stamps, R., Yang, M., & Jones, P. B. (2008). Raised incidence rates of all psychoses among migrant groups: Findings from the East London first episode psychosis study. *Archives of General Psychiatry*, *65*, 1250–1258. doi: 10.1001/archpsyc.65.11.1250

Cook, T. D., & Campbell, D. T. (1979). *Quasi-experimentation: Design & analysis issues for field settings*. Chicago, IL: Rand-McNally.

Cornfield, J., Haenszel, W., Hammond, E. C., Lilienfeld, A. M., Shimkin, M. B., & Wynder, E. L. (1959). Smoking and lung cancer: Recent evidence and a discussion of some questions. *Journal of the National Cancer Institute*, *22*, 173–203.

Costello, E. J., Compton, S. N., Keeler, G., & Angold, A. (2003). Relationships between poverty and psychopathology: A natural experiment. *Journal of the American Medical Association*, *290*, 2023–2029.

Costello, E. J., Erkanli, A., Copeland, W., & Angold, A. (2010). Association of family income supplements in adolescence with development of psychiatric and substance use disorders in adulthood among an American Indian population. *Journal of the American Medical Association*, *303*, 1954–1960. doi: 10.1001/jama.2010.621

D'Onofrio, B. M., Singh, A. L., Iliadou, A., Lambe, M., Hultman, C. M., Grann, M., . . . Lichtenstein, P. (2010). Familial confounding of the association between maternal smoking during pregnancy and offspring criminality: A population-based study in Sweden. *Archives of General Psychiatry*, *67*, 529–538. doi: 10.1001/archgenpsychiatry.2010.33.

D'Onofrio, B. M., Turkheimer, E. N., Eaves, L. J., Corey, L. A., Berg, K., Solaas, M. H., & Emery, R.E. (2003). The role of the children of twins design in elucidating causal relations between parent characteristics and child outcomes. *Journal of Child Psychology and Psychiatry*, *44*, 1130–1144.

Davey Smith, G. (2010). Mendelian Randomization for strengthening causal inference in observational studies. *Perspectives on Psychological Science*, *5*(5), 527–545.

Davey Smith, G., & Ebrahim, S. (2003). Mendelian randomization: Can genetic epidemiology contribute to understanding environmental determinants of disease? *International Journal of Epidemiology*, *32*, 1–22.

Davey Smith, G., & Ebrahim, S. (2005). What can mendelian randomisation tell us about modifiable behavioural and environmental exposures? *BMJ*, *330*, 1076–1079.

Dawid, A. P., & Pickles, A. (2007). Appendix I: Statistics. In *Identifying the environmental causes of disease: How should we decide what to believe and when to take action?* (pp. 93–102). London, UK: Academy of Medical Sciences.

DeGroot, A. D. (1951). War and the intelligence of youth. *Journal of Abnormal and Social Psychology*, *46*, 596–597.

Duncan, G. J., Magnuson, K. A., & Ludwig, J. (2004). The endogeneity problem in developmental studies. *Research in Human Development*, *1*, 59–80.

Duyme, M., Dumaret, A. C., & Tomkiewicz, S. (1999). How can we boost IQs of "dull children"? A late adoption study. *Proceedings of the National Academy of Sciences, 96*, 8790–8794.

Eaves, L. J., Prom, E. C., & Silberg, J. L. (2010). The mediating effect of parental neglect on adolescent and young adult anti-sociality: A longitudinal study of twins and their parents. *Behavior Genetics*, *40*, 425–437. doi: 10.1007/s10519-010-9336-2

Eaves, L. J., Silberg, J. L., & Maes, H. H. (2005). Revisiting the children of twins: Can they be used to resolve the environmental effects of dyadic parental treatment on child behavior? *Twin Research and Human Genetics*, *8*, 283–290.

Entwisle, D. R., Alexander, K. L., & Olson, L. S. (2004). Young children's achievement in school and socioeconomic background. In D. Conley & K. Albright (Eds.), *After the bell—Family background, public policy and educational success* (pp. 86–108). New York, NY: Routledge.

Fairchild, A. J., & MacKinnon, D. P. (2009). A general model for testing mediation and moderation effects. *Prevention Science*, *10*, 87–99. doi: 10.1007/s11121-008-0109-6

Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1992). Family change, parental discord and early offending. *Journal of Child Psychology and Psychiatry*, *33*, 1059–1075.

Foster, E. M. (2010). Causal inference and developmental psychology. *Developmental Psychology*, *46*, 1454–1480. doi: 10.1037/a0020204

Foster, E. M., & McLanahan, S. (1996). An illustration of the use of instrumental variables: Do neighborhood conditions affect a young person's chance of finishing high school? *Psychological Methods*, *1*, 249–260.

Fraga, M. F., Ballestar, E., Paz, M. F., Ropero, S., Setien, F., Ballestar, M. L., . . . Benitez, J. (2005). Epigenetic differences arise during the lifetime of monozygotic twins. *Proceedings of the National Academy of Sciences, 102*, 10604–10609.

Fu, H., & VanLandingham, M. J. (2012). Mental health consequences of international migration for Vietnamese Americans and the mediating effects of physical health and social networks: Results from a natural experiment approach. *Demography*, *49*, 393–424. doi: 10.1007/s13524-011-0088-2

Galiani, S., Rossi, M. A., & Schargrodsky, E. (2011). Conscription and crime: Evidence from the Argentine draft lotters. *American Economic Journal. Applied Economics*, *3*, 119–136.

Ge, X., Conger, R. D., Cadoret, R. J., Neiderhiser, J. M., Yates, W., Troughton, E., & Stewart, M. A. (1996). The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology*, *32*, 574–589.

Girotto, J., & Peterson, P. (1999). Do hard courses and good grades enhance cognitive skills? In S. Mayer & P. Peterson (Eds.), *Earning and learning: How schools matter* (pp. 205–230). New York, NY: Russell Sage Foundation.

Glazerman, S., Levy, D., & Myers, D. (2002). *Nonexperimental replications of social experiments: A systematic review*. Washington, DC: Mathematica Policy Research.

Glueck, S., & Glueck, E. (1968). *Delinquents and nondelinquents in perspective*. Cambridge, MA: Harvard University Press.

Glymour, M. M., Tchetgen, E. J. T., & Robins, J. M. (2012). Credible Mendelian randomization studies: Approaches for evaluating the instrumental variable assumptions. *American Journal of Epidemiology*, *175*, 332–339. doi: 10.1093/aje/kwr323

Gordon, R. A., Lahey, B. B., Kawai, E., Loeber, R., Stouthamer-Loeber, M., & Farrington, D. P. (2004). Antisocial behavior and youth gang membership: Selection and socialization. *Criminology*, *42*, 55–88.

Grant, B. F., & Dawson, D. A. (1997). Age at onset of alcohol use and its association with *DSM-IV* alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse*, *9*, 103–110.

Gray, R., & Wheatley, K. (1991). How to avoid bias when comparing bone marrow transplantation with chemotherapy. *Bone Marrow Transplantation*, *7*, 9–12.

Green, R. L., Hoffman, L. T., Morse, R., Hayes, M. E., & Morgan, R. F. (1964). *The educational status of children in a district without public schools*. Washington, DC: Office of Education, US Department of Helath, Education and Welfare.

Harden, K. P., Turkheimer, E., Emery, R. E., D'Onofrio, B. M., Slutske, W. S., Heath, A. C., & Martin, N. G. (2007). Marital conflict and conduct problems in children of twins. *Child Development*, *78*(1), 1–18.

Harold, G. T., Elam, K. K., Lewis, G., Rice, F., & Thapar, A. (2012). Interparental conflict, parent psychopathology, hostile parenting, and child antisocial behavior: Examining the role of maternal versus paternal influences using a novel genetically sensitive research design. *Development and Psychopathology*, *24*, 1283–1295. doi: 10.1017/S0954579412000703

Haviland, A., Nagin, D. S., & Rosenbaum, P. R. (2007). Combining propensity score matching and group-based trajectory analysis in an observational study. *Psychological Methods*, *12*, 247–267.

Heijmans, B. T., Tobi, E. W., Stein, A. D., Putter, H., Blauw, G. J., Susser, E. S., . . . Lumey, L. H. (2008). Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proceedings of the National Academy of Sciences, 105,* 17046–17049. doi: 10.1073/pnas.0806560105

Heyns, B. (1978). *Summer learning and the effect of schooling*. New York, NY: Academic Press.

Hill, A. B. (1965). The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine, 58,* 295–300.

Holland, P. W. (1986). Statistics and causal inference. *Journal of the American Statistical Association*, *81*, 945–960.

Honda, H., Shimizu, Y., & Rutter, M. (2005). No effect of MMR withdrawal on the incidence of autism: A total population study. *Journal of Child Psychology and Psychiatry*, *46,* 572–579. doi: 10.1111/j.1469-7610.2005.01425.x

Howe, G. W., Reiss, D., & Yuh, J. (2002). Can prevention trials test theories of etiology? *Development and Psychopathology*, *14*, 673–694.

Irons, D. E., McGue, M., Iacono, W. G., & Oetting, W. S. (2007). Mendelian randomization: A novel test of the gateway hypothesis and models of gene–environment interplay. *Development and Psychopathology*, *19*, 1181–1195.

Jacob, T., Waterman, B., Heath, A., True, W., Bucholz, K. K., Haber, R., . . . Fu, Q. (2003). Genetic and environmental effects on offspring alcoholism: New insights using an offspring-of-twins design. *Archives of General Psychiatry*, *60*, 1265–1272.

Jacobson, S. W., Jacobson, J. L., Sokol, R. J., Martier, S. S., & Chiodo, L. M. (1996). New evidence for neurobehavioral effects of in utero cocaine exposure. *Journal of Pediatrics*, *129*, 581–590.

Jaffee, S., Caspi, A., Moffitt, T. E., Belsky, J., & Silva, P. (2001). Why are children born to teen mothers at risk for adverse outcomes in young adulthood? Results from a 20-year longitudinal study. *Development and Psychopathology*, *13*, 377–397.

Jaffee, S. R. (2011). Genotype–environment correlations: Definitions, methods of measurement, and implications for research on adolescent psychopathology. In K. K. Kendler, S. R. Jaffee, & D. Romer (Eds.), *The dynamic genome and mental health: The role of genes and environments in youth development* (pp. 79–102). New York, NY: Oxford University Press.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Polo-Tomas, M., Price, T. S., & Taylor, A. (2004). The limits of child effects: Evidence for genetically mediated child effects on corporal punishment but not on physical maltreatment. *Developmental Psychology*, *40*, 1047–1058.

Jaffee, S. R., Strait, L. B., & Odgers, C. L. (2012). From correlates to causes: Can quasi-experimental studies and statistical innovations bring us closer to identifying the causes of antisocial behavior? *Psychological Bulletin*, *138*, 272–295. doi: 10.1037/a0026020

Jencks, C., Smith, M., Acland, M., Bane, M. J., Cohen, D., Gintis, H., . . . Michelson, S. (1972). *Inequality: A reassessment of the effect of family and schooling in America*. New York, NY: Basic Books.

Jokela, M., Elovainio, M., Keltikangas-Järvinen, L., Batty, G. D., Hintsanen, M., Seppälä, I., . . . Kivimäki, M. (2012). Body mass index and depressive symptoms: Instrumental-variables regression with genetic risk score. *Genes, Brain and Behavior*, *11*, 942–948. doi: 10.1111/j.1601-183X.2012.00846.x

Jones, P. B., & Fung, W. L. A. (2005). Ethnicity and mental health: The example of schizophrenia in the African-Caribbean population in Europe. In M. Rutter & M. Tienda (Eds.), *Ethnicity and causal mechanisms* (pp. 227–261). New York, NY: Cambridge University Press.

Joy, L. A., Kimball, M. M., & Zabrack, M. L. (1986). Television and children's aggressive behavior. In T. M. Williams (Ed.), *The impact of television: A natural experiment in three communities* (pp. 303–360). New York, NY: Academic Press (cited by Duncan et al., 2004).

Kandel, D. B. (1978). Homophily, selection, and socialization in adolescent friendships. *American Journal of Sociology*, *84*, 427–436.

Katan, M. B. (1986). Apolipoprotein E isoforms, serum cholesterol, and cancer. *Lancet*, *1*, 507–508.

Katan, M. B. (2004). Commentary: Mendelian randomization, 18 years on. *International Journal of Epidemiology*, *33*, 10–11.

Keavney, B. (2004). Commentary: Katan's remarkable foresight: Genes and causality 18 years on. *International Journal of Epidemiology*, *33*, 11–14.

Keavney, B., Danesh, J., Parish, S., Palmer, A., Clark, S., Youngman, L., . . . Collins, R. (2006). Fibrinogen and coronary heart disease: Test of causality by "Mendelian randomization." *International Journal of Epidemiology*, *35*, 935–943.

Kendler, K. S., & Baker, J. H. (2007). Genetic influences on measures of the environment: A systematic review. *Psychological Medicine*, *37*, 615–626. doi: 10.1017/s0033291706009524

Kendler, K. S., & Gardner, C. O. (2010). Dependent stressful life events and prior depressive episodes in the prediction of major depression: The problem of causal inference in psychiatric epidemiology. *Archives of General Psychiatry*, *67*, 1120–1127. doi: 10.1001/archgenpsychiatry.2010.136

Kendler, K. S., & Prescott, C. A. (2006). *Genes, environment, and psychopathology: Understanding the causes of psychiatric and substance use disorders*. New York, NY: Guilford Press.

Kivimäki, M., Jokela, M., Hamer, M., Geddes, J., Ebmeier, K., Kumari, M., . . . Batty, G.D. (2011). Examining overweight and obesity as risk factors for common mental disorders using fat mass and obesity-associated (FTO) genotype-instrumented analysis The Whitehall II Study, 1985–2004. *American Journal of Epidemiology*, *173*, 421–429. doi: 10.1093/aje/kwq444

Knopik, V. S., Heath, A. C., Jacob, T., Slutske, W. S., Bucholz, K. K., Madden, P. A. F., . . . Martin, N. G. (2006). Maternal alcohol use disorder and offspring ADHD: Disentangling genetic and environmental effects using a children-of-twins design. *Psychological Medicine*, *36*, 1461–1472.

Kumsta, R., Rutter, M., Stevens, S., & Sonuga-Barke, E. J. (2010). IX. Risk, causation, mediation, and moderation. *Monographs of the Society for Research in Child Development*, *75*(1), 187–211. doi: 10.1111/j.1540-5834.2010.00556.x

Lahey, B. B., & D'Onofrio, B. M. (2010). All in the family: Comparing siblings to test causal hypotheses regarding environmental influences on behavior. *Current Directions in Psychological Science*, *19*, 319–323.

Laird, N. M., & Mosteller, F. (1990). Some statistical methods for combining experimental results. *International Journal of Technology Assessment in Health Care*, *6,* 5–30.

Langley, K., Heron, J., Smith, G. D., & Thapar, A. (2012). Maternal and paternal smoking during pregnancy and risk of ADHD symptoms in offspring: Testing for intrauterine effects. *American Journal of Epidemiology*, *176*, 261–268. doi: 10.1093/aje/kwr510

Lawlor, D. A., Harbord, R. M., Tybjaerg-Hansen, A., Palmer, T. M., Zacho, J., Benn, M., . . . Nordestgaard, B. G. (2011). Using genetic loci to understand the relationship between adiposity and psychological distress: A Mendelian randomization study in the Copenhagen General Population Study of 53 221 adults. *Journal of Internal Medicine*, *269*(5), 525–537. doi: 10.1111/j.1365-2796.2011.02343.x

Ludwig, J., Duncan, G. J., & Hirschfield, P. (2001). Urban poverty and juvenile crime: Evidence from a randomized housing-mobility experiment. *Quarterly Journal of Economics*, *116*, 655–679.

Ludwig, J., & Miller, D. L. (2005). Does Head Start improve children's life chances? Evidence from a regression discontinuity design. *Quarterly Journal of Economics*, *122*, 159–208.

Lynch, S. K., Turkheimer, E., D'Onofrio, B. M., Mendle, J., Emery, R. E., Slutske, W. S., & Martin, N. G. (2006). A genetically informed study of the association between harsh punishment and offspring behavioral problems. *Journal of Family Psychology*, *20*, 190–198.

Lynskey, M. T., Heath, A. C., Bucholz, K. K., Slutske, W. S., Madden, P. A. F., Nelson, E. C., . . . Martin, N. G. (2003). Escalation of drug use in early-onset cannabis users vs co-twin controls. *Journal of the American Medical Association*, *289*, 427–433.

Lynskey, M. T., Vink, J. M., & Boomsma, D. I. (2006). Early onset cannabis use and progression to other drug use in a sample of Dutch twins. *Behavior Genetics*, *36*(2), 195–200.

Mackie, J. L. (1965). Causes and conditions. *American Philosophical Quarterly*, *2*, 245–264.

Mackie, J. L. (1974). *The cement of the universe: A study of causation*. Oxford, UK: Oxford University Press.

MacKinnon, D. P., Fairchild, A. J., & Fritz, M. S. (2007). Mediation analysis. *Annual Review of Psychology*, *58*, 593–614.

MacMahon, B., Pugh, T. F., & Ipsen, J. (1960). *Epidemiologic methods*. Boston, MA: Little, Brown & Co.

Madsen, K. M., Lauritsen, M. B., Pedersen, C. B., Thorsen, P., Plesner, A. M., Andersen, P. H., & Mortensen, P. B. (2003). Thimerosal and the occurrence of autism: Negative ecological evidence from Danish population-based data. *Pediatrics*, *112*, 604–606.

Maes, H. H., Woodard, C. E., Murrelle, L., Meyer, J. M., Silberg, J. L., Hewitt, J. K., . . . Eaves, L. J. (1999). Tobacco, alcohol and drug use in eight- to sixteen-year-old twins: the Virginia Twin Study of Adolescent Behavioral Development. *Journal of Studies on Alcohol*, *60*(3), 293–305.

Magnuson, K. A., & McGroder, S. (2002). The effect of increasing welfare mothers' education on their young children's academic problems and school readiness. Joint Center for Poverty Research Working Paper no. 280, Northwestern University (cited by Duncan et al., 2004).

Marmot, M. G., & Syme, S. L. (1976). Acculturation and coronary heart disease in Japanese-Americans. *American Journal of Epidemiology*, *104*, 225–247.

Maynard, R. A. (1997). *Kids having kids: Economic costs and social consequences of teen pregnancy*. Washington, DC: Urban Institute Press.

McCall, B. P., & Bielby, R. M. (2012). Regression discontinuity design: Recent developments and a guide to practice for researchers in higher education. In J. C. Smart & M. B. Paulsen (Eds.), *Higher education: Handbook of theory and research* (Vol. 27, pp. 249–290). Dordrecht, The Netherlands: Springer Science.

McClellan, J. M., Susser, E., & King, M. C. (2006). Maternal famine, *de novo* mutations, and schizophrenia. *Journal of the American Medical Association*, *296*, 582–584.

McCrory, C., & Layte, R. (2012). Prenatal exposure to maternal smoking and childhood behavioural problems: A quasi-experimental approach. *Journal of Abnormal Child Psychology*, *40*, 1277–1288. doi: 10.1007/s10802-012-9640-9

McGue, M., & Iacono, W. G. (2005). The association of early adolescent problem behavior with adult psychopathology. *American Journal of Psychiatry*, *162*, 1118–1124.

McGue, M., Keyes, M., Sharma, A., Elkins, I., Legrand, L., Johnson, W., & Iacono, W. G. (2007). The environments of adopted and non-adopted youth: Evidence on range restriction from the Sibling Interaction and Behavior Study (SIBS). *Behavior Genetics*, *37*, 449–462.

McKim, V. R., & Turner, S. P. (1997). *Causality in crisis? Statistical methods and the search for causal knowledge in the social sciences* (Vol. 4). Notre Dame, IN: Univ of Notre Dame Press.

Meade, T. W., Humphries, S. E., & De Stavola, B. L. (2006). Commentary: Fibrinogen and coronary heart disease—test of causality by "Mendelian" randomization by Keavney et al. *International Journal of Epidemiology*, *35*, 944–947.

Melhuish, E., Belsky, J., Leyland, A. H., & Barnes, J. (2008). Effects of fully-established Sure Start Local Programmes on 3-year-old children and their families living in England: A quasi-experimental observational study. *Lancet*, *372*, 1641–1647. doi: 10.1016/S0140-6736(08)61687-6

Meyer, J. M., Rutter, M., Silberg, J. L., Maes, H. H., Simonoff, E., Shillady, L. L., … Eaves, L. J. (2000). Familial aggregation for conduct disorder symptomatology: The role of genes, marital discord and family adaptability. *Psychological Medicine*, *30*, 759–774.

Mill, J., Dempster, E., Caspi, A., Williams, B., Moffitt, T., & Craig, I. (2006). Evidence for monozygotic twin (MZ) discordance in methylation level at two CpG sites in the promoter region of the catechol-O-methyltransferase (COMT) gene. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *141*, 421–425.

Moe, V. (2002). Foster-placed and adopted children exposed in utero to opiates and other substances: Prediction and outcome at four and a half years. *Journal of Developmental & Behavioral Pediatrics*, *23*, 330–339.

Moffitt, T. E., & Team, E-Risk Study. (2002). Teenaged mothers in contemporary Britain. *Journal of Child Psychology and Psychiatry*, *43*, 727–742.

Nagin, D. S., Barker, E. D., Lacourse, E., & Tremblay, R. E. (2008). The interrelationship of temporally distinct risk markers and the transition from childhood physical aggression to adolescent violent delinquency. In P. Cohen (Ed.), *Applied data techniques for turning points research* (pp. 17–36). New York, NY: Routledge.

Narusyte, J., Neiderhiser, J. M., D'Onofrio, B. M., Reiss, D., Spotts, E. L., Ganiban, J., & Lichtenstein, P. (2008). Testing different types of genotype–environment correlation: An extended children-of-twins model. *Developmental Psychology*, *44*, 1591–1603. doi: 10.1037/a0013911

National Research Council. (2002). Scientific research in education. In R. J. Shavelson & L. Towne (Eds.), *Committee on scientific principles for education research*, R.J. Shavelson & L.Towne. Washington, DC: The National Academies Press.

Nitsch, D., Molokhia, M., Smeeth, L., DeStavola, B. L., Whittaker, J. C., & Leon, D. A. (2006). Limits to causal inference based on Mendelian randomization: A comparison with randomized controlled trials. *American Journal of Epidemiology*, *163*, 397–403.

O'Connor, T. G., Deater-Deckard, K., Fulker, D., Rutter, M., & Plomin, R. (1998). Genotype–environment correlations in late childhood and early adolescence: Antisocial behavioral problems and coercive parenting. *Developmental Psychology*, *34*, 970–981.

Obel, C., Olsen, J., Henriksen, T. B., Rodriguez, A., Järvelin, M. R., Moilanen, I., … Ebeling, H. (2011). Is maternal smoking during pregnancy a risk factor for hyperkinetic disorder? Findings from a sibling design. *International Journal of Epidemiology*, *40*, 338–345. doi: 10.1093/ije/dyq185

Pearl, J. (2000). *Causality*. Cambridge, UK: Cambridge University Press.

Pickett, K. E., Wilkinson, R. G., & Wakschlag, L. S. (2009). The psychosocial context of pregnancy smoking and quitting in the Millennium Cohort Study. *Journal of Epidemiology and Community Health*, *63*, 474–480. doi: 10.1136/jech.2008.082594

Pike, A., McGuire, S., Hetherington, E. M., Reiss, D., & Plomin, R. (1996). Family environment and adolescent depressive symptoms and antisocial behavior: A multivariate genetic analysis. *Developmental Psychology*, *32*, 590–604.

Prescott, C. A., & Kendler, K. S. (1999). Age at first drink and risk for alcoholism: A noncausal association. *Alcoholism: Clinical and Experimental Research*, *23*, 101–107.

Pulkkinen, L., Kaprio, J., & Rose, R. J. (2006). *Socioemotional development and health from adolescence to adulthood*. Cambridge, UK: Cambridge University Press.

Ravussin, E., Valencia, M. E., Esparza, J., Bennett, P. H., & Schulz, L. O. (1994). Effects of a traditional lifestyle on obesity in Pima Indians. *Diabetes Care*, *17*, 1067–1074.

Rice, F., Harold, G. T., Boivin, J., Hay, D. F., Van Den Bree, M., & Thapar, A. (2009). Disentangling prenatal and inherited influences in humans with an experimental design. *Proceedings of the National Academy of Sciences, 106*, 2464–2467. doi: 10.1073/pnas.0808798106

Robins, J. M. (2001). Data, design, and background knowledge in etiologic inference. *Epidemiology*, *12*, 313–320.

Robins, J. M., & Greenland, S. (1996). Identification of causal effects using instrumental variables: Comment. *Journal of the American Statistical Association*, *91*, 456–458.

Robins, J. M., Hernán, M. Á., & Brumback, B. (2000). Marginal structural models and causal inference in epidemiology. *Epidemiology*, *11*, 550–560.

Rosenbaum, J. E. (1991). Black pioneers—Do their moves to the suburbs increase economic opportunity for mothers and children? *Housing Policy Debate*, *2*, 1179–1213.

Rosenbaum, P. R. (2002). *Observational studies* (2nd ed.). New York, NY: Springer-Verlag.

Rothman, K. J., & Greenland, S. (1998). *Modern epidemiology* (2nd ed.). Philadephia, PA: Lippincott-Raven.

Rothman, K. J., & Greenland, S. (2002). Causation and causal inference. In R. Detels, J. McEwen, R. Beaglehole, & H. Tanaka (Eds.), *Oxford textbook of public health* (4th ed., pp. 641–653). Oxford, UK: Oxford University Press.

Rubin, D. B. (1977). Assignment to treatment group on the basis of a covariate. *Journal of Educational and Behavioral Statistics*, *2*, 1–26.

Rutter, M. (1983). Statistical and personal interactions: Facets and perspectives. In D. Magnusson & V. L. Allen (Eds.), *Human development, an interactional perspective* (pp. 295–319). New York, NY: Academic Press.

Rutter, M. (1997). Comorbidity: Concepts, claims and choices. *Criminal Behaviour and Mental Health*, *7*, 265–285.

Rutter, M. (2005). Incidence of autism spectrum disorders: Changes over time and their meaning. *Acta Paediatrica*, *94*, 2–15.

Rutter, M. (2006). *Genes and behavior: Nature–nurture interplay explained*. Oxford, UK: Blackwell Scientific.

Rutter, M. (2007). Proceeding from observed correlation to causal inference: The use of natural experiments. *Perspectives on Psychological Science*, *2*, 377–395.

Rutter, M. (2009). Epidemiological methods to tackle causal questions. *International Journal of Epidemiology*, *38*, 3–6.

Rutter, M. (2012). "Natural experiments" as a means of testing causal inferences. In C. Berzuini, P. Dawid, & L. Bernardinelli (Eds.), *Causality: Statistical perspectives and applications* (pp. 253–272). Chichester, UK: Wiley.

Rutter, M., & Azis-Clauson, C. (2011). Extreme deprivation. In P. A. Howlin, T. Charman, & M. Ghaziuddin (Eds.), *The SAGE handbook of developmental disorders* (pp. 529–550). London, UK: SAGE Publications Ltd.

Rutter, M., Bolton, P., Harrington, R., Le Couteur, A., Macdonald, H., & Simonoff, E. (1990). Genetic factors in child psychiatric disorders—I. A review of research strategies. *Journal of Child Psychology and Psychiatry*, *31*, 3–37.

Rutter, M., Dunn, J., Plomin, R., Simonoff, E., Pickles, A., Maughan, B., . . . Eaves, L. (1997). Integrating nature and nurture: implications of person–environment correlations and interactions for developmental psychopathology. *Developmental Psychopathology*, *9*, 335–364.

Rutter, M., Kumsta, R., Schlotz, W., & Sonuga-Barke, E. (2012). Longitudinal studies using a "natural experiment" design: The case of adoptees from Romanian institutions. *Journal of the American Academy of Child & Adolescent Psychiatry*, *51*, 762–770. doi: 10.1016/j.jaac.2012.05.011

Rutter, M., & Madge, N. (1976). *Cycles of disadvantage: A review of research*. London, UK: Heinemann Educational.

Rutter, M., & Maughan, B. (2002). School effectiveness findings 1979–2002. *Journal of School Psychology*, *40*, 451–475.

Rutter, M., Maughan, B., Mortimore, P., Ouston, J., & Smith, A. (1979). *Fifteen thousand hours: Secondary schools and their effects on children*. Cambridge, MA: Harvard University Press.

Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. *Psychological Bulletin*, *127*, 291–324.

Rutter, M., & Redshaw, J. (1991). Annotation: growing up as a twin: Twin–singleton differences in psychological development. *Journal of Child Psychology and Psychiatry*, *32*, 885–895.

Rutter, M., & Sonuga-Barke, E. J. (2010). X. Conclusions: Overview of findings from the E.R.A. study, inferences, and research implications. In *Deprivation-specific psychological patterns: Effects of institutional deprivation* (Vol. 75, pp. 212–229). Oxford, UK: Monographs of the Society for Research in Child Development 75. doi: 10.1111/j.1540-5834.2010.00557.x

Rutter, M., Thorpe, K., Greenwood, R., Northstone, K., & Golding, J. (2003). Twins as a natural experiment to study the causes of mild language delay: I: Design; twin–singleton differences in language, and obstetric risks. *Journal of Child Psychology and Psychiatry*, *44*, 326–341.

Sampson, R. J., Laub, J. H., & Wimer, C. (2006). Does marriage reduce crime? A counterfactual approach to within-individual causal effects, Criminology, *44*, 465–508.

Schwartz, S., & Susser, E. (2006). What is a cause? In E. Susser, S. Schwartz, A. Morabia, & E. J. Bromet (Eds.), *Psychiatric epidemiology: Searching for the causes of mental disorders* (pp. 33–42). New York, NY: Oxford University Press.

Shadish, W. R. (2010). Campbell and Rubin: A primer and comparison of their approaches to causal inference in field settings. *Psychological Methods*, *15*, 3–17. doi: 10.1037/a0015916

Shadish, W. R., & Cook, T. D. (1999). Comment—Design rules: More steps toward a complete theory of quasi-experimentation. *Statistical Science*, *14*, 294–300.

Shadish, W. R., Cook, T. D., & Campbell, D. T. (2002). *Experimental and quasi-experimental designs for generalized causal inference*. Boston, MA: Houghton Mifflin.

Silberg, J. L., & Eaves, L. J. (2004). Analysing the contributions of genes and parent–child interaction to childhood behavioural and emotional problems: A model for the children of twins. *Psychological Medicine*, *34*, 347–356.

Silberg, J. L., Maes, H., & Eaves, L. J. (2010). Genetic and environmental influences on the transmission of parental depression to children's depression and conduct disturbance: An extended children of twins study. *Journal of Child Psychology and Psychiatry*, *51*, 734–744. doi: 10.1111/j.1469-7610.2010.02205.x

Silberg, J. L., Maes, H., & Eaves, L. J. (2012). Unraveling the effect of genes and environment in the transmission of parental antisocial behavior to children's conduct disturbance, depression and hyperactivity. *Journal of Child Psychology and Psychiatry*, *53*, 668–677. doi: 10.1111/j.1469-7610.2011.02494.x

Singer, L. T., Arendt, R., Minnes, S., Farkas, K., & Salvator, A. (2000). Neurobehavioral outcomes of cocaine-exposed infants. *Neurotoxicology and Teratology*, *22*, 653–666.

Singer, L. T., Minnes, S., Short, E., Arendt, R., Farkas, K., Lewis, B., . . . Kirchner, H. L. (2004). Cognitive outcomes of preschool children with prenatal cocaine exposure. *Journal of the American Medical Association*, *291*, 2448–2456.

Spergel, I. A. (1990). Youth gangs: Continuity and change. In M. Tonry & N. Morris (Eds.), *Crime and justice: A review of research* (Vol. 12, pp. 171–275). Chicago, IL: University of Chicago Press.

St Clair, D., Xu, M., Wang, P., Yu, Y., Fang, Y., Zhang, F., . . . Sham, P. (2005). Rates of adult schizophrenia following prenatal exposure to the Chinese famine of 1959–1961. *Journal of the American Medical Association*, *294*, 557–562.

Stattin, H., & Magnusson, D. (1990). *Pubertal maturation in female development* (Vol. 2). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Stein, Z. A., Susser, M., Saenger, G., & Marolla, F. (1975). *Famine and human development: The Dutch hunger winter of 1944–1945*. New York, NY: Oxford University Press.

Stillman, S., McKenzie, D., & Gibson, J. (2009). Migration and mental health: Evidence from a natural experiment. *Journal of Health Economics*, *28*, 677–687. doi: 10.1016/j.jhealeco.2009.02.007

Stoolmiller, M. (1998). Correcting estimates of shared environmental variance for range restriction in adoption studies using a truncated multivariate normal model. *Behavior Genetics*, *28*, 429–441.

Stoolmiller, M. (1999). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior–genetic adoption studies. *Psychological Bulletin*, *125*, 392–409.

Susser, E., Schwartz, S., Morabia, A., & Bromet, E. J. (2006). *Psychiatric Epidemiology: Searching for the causes of mental disorders*. New York, NY: Oxford University Press.

Thapar, A., Harold, G., Rice, F., Ge, X. J., Boivin, J., Hay, D., . . . Lewis, A. (2007). Do intrauterine or genetic influences explain the foetal origins of chronic disease? A novel experimental method for disentangling effects. *BMC Medical Research Methodology*, *7*, 25.

Thapar, A., Rice, F., Hay, D., Boivin, J., Langley, K., van den Bree, M., . . . Harold, G. (2009). Prenatal smoking might not cause attention-deficit/hyperactivity disorder: Evidence from a novel design. *Biological Psychiatry*, *66,* 722–727. doi: 10.1016/j.biopsych.2009.05.032

Thistlethwaite, D. L., & Campbell, D. T. (1960). Regression-discontinuity analysis: An alternative to the ex post facto experiment. *Journal of Educational Psychology*, *51*, 309–317.

Thomas, A., Chess, S., & Birch, H. G. (1968). *Temperament and behaviour disorders in childhood*. New York, NY: New York University Press.

Thornberry, T. P. (1998). Membership in youth gangs and involvement in serious and violent offending. In R. L. Loeber & D. P. Farrington (Eds.), *Serious and violent juvenile offenders: Risk factors and successful interventions* (pp. 147–166). Thousand Oaks, CA: Sage.

Thornberry, T. P., Krohn, M. D., Lizotte, A. J., & Chard-Wiershem, D. (1993). The role of juvenile gangs in facilitating delinquent behavior. *Journal of Research in Crime & Delinquency*, *30*, 55–87.

Thornberry, T. P., Krohn, M. D., Lizotte, A. J., Smith, C. A., & Tobin, K. (2003). *Gangs and delinquency in developmental perspective*. New York, NY: Cambridge University Press.

Thorpe, K., Rutter, M., & Greenwood, R. (2003). Twins as a natural experiment to study the causes of mild language delay: II: Family interaction risk factors. *Journal of Child Psychology and Psychiatry*, *44*(3), 342–355.

Tobi, E. W., Lumey, L. H., Talens, R. P., Kremer, D., Putter, H., Stein, A. D., … Heijmans, B. T. (2009). DNA methylation differences after exposure to prenatal famine are common and timing-and sex-specific. *Human Molecular Genetics*, *18*, 4046–4053. doi: 10.1093/hmg/ddp353

Tobi, E. W., Slagboom, P. E., van Dongen, J., Kremer, D., Stein, A. D., Putter, H., … Lumey, L. H. (2012). Prenatal famine and genetic variation are independently and additively associated with DNA methylation at regulatory loci within IGF2/H19. *PLoS One, 7*(5), e37933. doi: 10.1371/journal.pone.0037933

Tobin, M. D., Minelli, C., Burton, P. R., & Thompson, J. R. (2004). Commentary: Development of Mendelian randomization: From hypothesis test to "Mendelian deconfounding." *International Journal of Epidemiology*, *33*, 26–29.

Valencia, M. E., Bennett, P. H., Ravussin, E., Esparza, J., Fox, C., & Schulz, L. O. (1999). The Pima Indians in Sonora, Mexico. *Nutrition Reviews*, *57*, 55–58.

Vitaro, F., Brendgen, M., & Arseneault, L. (2009). The discordant MZ-twin method: One step closer to the holy grail of causality. *International Journal of Behavioral Development*, *33*, 376–382.

Weinberg, C. R. (1993). Toward a clearer definition of confounding. *American Journal of Epidemiology*, *137*, 1–8.

West, S. G., & Thoemmes, F. (2010). Campbell's and Rubin's perspectives on causal inference. *Psychological Methods*, *15*, 18–37. doi: 10.1037/a0015917

Wilde, E.T., & Hollister, R. (2002). *How close is close enough? Testing nonexperimental estimates of impact against experimental estimates of impact with education test scores as outcomes*. Discussion Paper No. 1242-02. Madison, WI: Institute for Research on Poverty.

Wong, C. C. Y., Caspi, A., Williams, B., Craig, I. W., Houts, R., Ambler, A., … Mill, J. (2010). A longitudinal study of epigenetic variation in twins. *Epigenetics*, *5*, 516–526.

Wootton, B. (1959). *Social science and social pathology*. London, UK: George Allen & Unwin.

CHAPTER 5

Developmental Models and Mechanisms for Understanding the Effects of Early Experiences on Psychological Development

THOMAS G. O'CONNOR

INTRODUCTION 156
ALTERNATIVE DEVELOPMENTAL MODELS AND HYPOTHESES IN PSYCHOLOGICAL RESEARCH ON EARLY EXPERIENCE 157
Sensitive Period Models 157
Developmental Programming 159
Life Span Development or Cumulative Effects Models 160
Alternative Models 161
Summary 163
DEVELOPMENTAL MECHANISMS 163
Stress Physiology 163
Neuroimmunology 164
Neural Development and Neural Circuitry 166
Genetics 167
Psychological Mechanisms 168
Additional Mechanisms 169
Summary 170
CONCEPTUAL AND METHODOLOGICAL CONSIDERATIONS FOR INVESTIGATING DEVELOPMENTAL MODELS AND MECHANISMS 170
Limited Experimental Leverage to Assess Timing, Duration, and Intensity of Exposures 171
Phenotypic and Measurement Stability 171
Individual Differences 172
Sex Differences 173
Translation of Animal Findings to Human Health and Development 174
Summary 175
MODEL SYSTEMS AND PARADIGMS FOR STUDYING DEVELOPMENTAL MODELS AND MECHANISMS 175
Prenatal Stress/Anxiety and Child Development 175
Maternal Immune Activation Paradigm 177
Early and Severe Caregiving Neglect and Deprivation 178
Summary 179
DEVELOPMENTAL MODELS ARE SHAPING CLINICAL AND PUBLIC HEALTH STRATEGIES 179
Early Interventions Can Have Lasting Effects 180
Developmental Hypotheses and Early Intervention 183
Clinical and Public Health Application 184
Summary 185
CONCLUSIONS AND FUTURE DIRECTIONS 185
REFERENCES 186

INTRODUCTION

A rudimentary set of hypotheses in developmental psychopathology seeks to describe the degree to which and to explain the mechanisms by which early exposures and experiences shape psychological development. Many new leads and some changes are evident in this area of research since the publication of the last edition of this volume in 2006. This chapter seeks to provide an updating and integrating of the models and mechanisms that explicitly or implicitly underlie developmental research on normal and abnormal psychological function. More specifically, the aims of the chapter are to (1) outline and differentiate some key developmental models—especially those concerning early experience—for testing hypotheses concerning normal functioning and psychopathology; (2) identify leading mechanisms for these models; (3) articulate methodological challenges for research in this area; (4) review select model systems and research paradigms that have risen to prominence; and (5) discuss how developmental models shape clinical and public health debates and intervention strategies. Concluding comments and future directions end the chapter.

It may be instructive to note some of the major changes in this area of study since the last edition of this volume at the outset of this chapter, to set the stage for the subsequent

discussion and to underscore the pace of change and areas of particular scientific growth. Perhaps the most obvious is that the greater understanding of how genetic mechanisms may modulate and be modulated by (early) experience has stimulated further interest in developmental studies. Epigenetics is a prime example, but evidence suggests that there may be genetic influences on plasticity, developmental timing and susceptibility to environmental exposures. A second major change is a shift toward greater emphasis on integrative or multidisciplinary research. That is seen in the research findings emphasizing how early psychological stress exposure predicts cardiovascular, metabolic, and other physical health outcomes (Felitti et al., 1998); the reverse is also true, reflected in a greater emphasis now on how early diet, inflammatory conditions, and physiology shape neurodevelopment and later psychological outcomes (Brown & Susser, 2008). Findings from these lines of mechanistic research have helped to reformulate broader models of development. A corollary is that this kind of approach will begin to extinguish artificial boundaries in how research is conducted, moving from a perspective derived from medical or scientific disciplinary traditions to an approach that instead focuses on mechanisms of interest across many disciplines. A third change is the increased interest is the intersection of early experience on psychological development and economics, embodied in the increasing interest in a neuroscience-informed social policy agenda. Research evidence in this area is the final theme of the chapter.

Several further preliminary comments are in order. The first is that relevant and allied areas of research on early experience and developmental mechanisms extend too broadly for a single chapter, and so the focus is on exemplary paradigms and findings rather than an exhaustive review. Second, while acknowledging the seminal influence of experimental animal studies, the focus of this chapter is, wherever possible, on human psychological development. Certain areas of research that require mention, such as a consideration of microglia and brain development, are inevitably limited to animal studies. On the other hand, the number and quality of human studies is now substantial and sufficient in some areas to compose a majority of the reviewed research. Third, although the emphasis of the chapter is on psychological development, it is necessary to refer to multiple systems that are linked with psychological development given the inexorable trend toward integrative, multidisciplinary hypotheses in development and psychopathology.

ALTERNATIVE DEVELOPMENTAL MODELS AND HYPOTHESES IN PSYCHOLOGICAL RESEARCH ON EARLY EXPERIENCE

Several conceptual models underlie research on early experiences. Three broad classes of models that have attracted the most attention and have generated considerable research in the animal and human literatures are considered here: sensitive period models, developmental programming models, and life-course models; modified alternatives, adevelopmental and defunct models are then briefly mentioned, partly for historical purposes. Inevitably, given space limitations and the broader aims of this chapter, it is necessary to simplify the definition and description of these models. Many reviews of these models have been provided, along with references. This chapter seeks to cover what is often missing from prior discussions, such as a comparative approach that examines multiple different kinds of developmental models and their distinguishing features, and application to intervention and broader developmental constructs such as resilience.

Sensitive Period Models

The sensitive period model for development has a long history in biology and has been applied across species on phenotypes as complex as bird song and biological processes as basic as cell differentiation (Bornstein, 1989; Knudsen, 2004; MacDonald, 1985). A sensitive period model proposes that there exists an ontogenetic period in the organism's development in which certain specified essential input is needed for normal development to proceed. This is conceptually similar to the experience–expectant model (Greenough, Black, & Wallace, 1987). A *sensitive* period model differs from the related *critical* period model more in terms of degree than in kind. That is, whereas a critical period is generally understood to imply rigid boundaries defining the period with major and uniform effects, a sensitive period model generally implies less rigid periods of ontogenetic vulnerability and possibly less severe consequences and effects. Sensitive period models are often allied with early development, but they need not be. There is, for example, evidence that adolescence and parenthood may also provide a sensitive period for certain exposures and outcomes (Barrett & Fleming, 2011; Dahl & Gunnar, 2009). Importantly, studies of sensitive periods provide links to developmental mechanisms by charting the effects according to timing of exposure, and

cross-tabulating the results with what is known about ontogenetic development. So, for example, if there were a sensitive period of stress exposure on affiliative behavior, then these findings would imply a value in further study of those brain circuits undergoing rapid development during the identified sensitive period.

A distinguishing feature of sensitive period models is their focus on species-typical development rather than more subtle individual differences within normal range of adaptation. That is, sensitive period models are most typically applied when predicting gross pathology that could undermine species survival and reproduction. That is evident in the classic examples of sensitive periods, such as applied to birdsong (Doupe & Kuhl, 1999; Mooney, 1999); aspects of vision (Hubel & Wiesel, 1970); imprinting (Lorenz, 1935); and social attachments and social behavior (Harlow & Suomi, 1970; Martin, Spicer, Lewis, Gluck, & Cork, 1991; O'Connor & Cameron, 2006; Sackett, 1969). The outcome in these studies is not normative variation but rather unambiguous deviation and disorder that could undermine such basic functions as reproduction (e.g., attracting a mate in the case of birdsong). A thematic concern in this line of study is with core principles about the nature of the organism. For example, sensitive period models would consider comparatively extreme nativist hypotheses, or distinguish the dose of exposure that might contrast instinctual behavior from a behavior that required particular environmental input.

A further defining feature of sensitive period models, with particular application to intervention, is the notion that the sensitive period window marks a special period of susceptibility or opportunity in the organism's development. For example, the likelihood of neural tube defects from exposure (e.g., underexposure to folate or overexposure to environmental toxins) is not equally likely at any point in the prenatal period, but only during neural tube formation (e.g., Rice & Barone, 2000). In other words, interventions for neural tube development must occur within a limited time frame defined by the neural tube ontogeny. More broadly, if there were a sensitive period for a particular outcome, then interventions would need to be delivered during that sensitive period; interventions delivered outside the sensitive period would have no effect or, alternatively, would have a different effect. Sensitive period models are therefore distinguished from alternative models in proposing that there is a point in development—outside the period of sensitivity—after which an intervention would not have the intended effect. Moreover, there is little room for recovery or resilience if the necessary and sufficient exposure did not occur within the window of sensitivity.

It may be helpful to elucidate the sensitive period model by contrasting it with contrary concepts, notably plasticity and, particularly for human psychological development, neuroplasticity. Neuroplasticity is the notion that there are ongoing changes in the structure, organization, and function of neurons according to (ongoing) changes in experience (e.g., Licznerski & Duman, 2013). Sensitive period models presume limited or bounded plasticity.

The degree to which there are sensitive periods for human psychological development has generated considerable debate. To be sure, there are examples of sensitive periods not just for classic physical development such as neural tube defects but also for several aspects of brain development affecting language (Flege, Munro, & MacKay, 1995; Johnson & Newport, 1989), hearing (Sharma, Dorman, & Spahr, 2002), or vision (Maurer, Lewis, Brent, & Levin, 1999). Whether or not the clarity of a sensitive period effect can be mimicked in all types of psychological development—behavior in intimate relationships, cognitive problem solving, extraversion, depression—is what stirs debate. Extensions of a sensitive period model to human psychological development have been proposed. Some studies are based on observational designs, as in the case of maternal depression and child behavioral and emotional problems (e.g., Bagner, Pettit, Lewinsohn, & Seeley, 2010); others are based on natural experiments or have other leverage for evaluating sensitive period hypotheses, as in the case of maternal prenatal rubella infection in the first trimester and child autistic-like behaviors (Chess, 1977) or prenatal maternal famine and major mental illness in those offspring in adulthood (Hulshoff Pol et al., 2000). As reviewed in a subsequent section, many purported examples of sensitive periods for human psychological development lack necessary controls and experimental leverage. That does not rule out the possibility of a sensitive period, but it does weaken the strength of the claim. There are also clear instances of misapplying the notion of sensitive periods to human psychological development. These misapplications and their rebuttals have a long history, particularly in the area of attachment and bonding (e.g., Herbert, Sluckin, & Sluckin, 1982) and have generated a disposition toward scepticism (Bruer, 1999).

Importantly, the hypothesis that there may be a sensitive period for certain exposures for specified developmental outcomes is different from proposing that there exists key periods for the emergence of social and cognitive functions. Thus, it is clear that there is a specified period

in development for the emergence of stranger anxiety or self-concept (e.g., Kagan, 1982). A sensitive period hypothesis would take this observation one step further in positing that, for example, the child's self-concept would be especially susceptible to the adverse effects of insensitive caregiving during this period of self-concept emergence.

Most of the empirically demonstrated examples of sensitive (and critical) periods for brain and behavioral development are derived from experimental animal studies. Minimally, that is attributable to the greater experimental leverage and assessment opportunities available in animal compared with human research. And it might also be that sensitive periods are more easily found in rodent and nonhuman models compared with humans. This possibility was illustrated by Berardi and colleagues (Berardi, Pizzorusso, & Maffei, 2000), who plotted the association between life span and sensitive period width across several species from monocular deprivation studies. They found a strong positive relationship between life span of the species and width of the sensitive period; that is, humans have a much wider (and thus possibly harder to detect) sensitive period window than rodents, for example. It is not known if a comparable association between life span length and sensitive period window holds for other kinds of exposures and for psychological outcomes, but that possibility presents a major challenge for research seeking to translate animal (perhaps especially rodent) findings on early exposures to human psychological development, especially where developmental timing may be concerned.

Developmental Programming

Developmental or adaptive programming is an alternative model for understanding how early exposures and experiences may have lasting effects on health and development. Probably the most well-research example of developmental programming is fetal programming, which proposed that in utero exposures instigate an adaptive response in the organism that is carried forward in development with persisting effects on behavior and biology (Amiel-Tison et al., 2004; Barker, 2007; Gluckman & Hanson, 2005; Gluckman et al., 2009). There may be programming effects of postnatal experiences as well, so the broader term developmental programming is used in this chapter.

A key feature of this model is that the biology of the fetus and child adapts to specific features of its environment; these adaptations are referred to as programming because they are thought to set some aspect of the biological system of the organism, with persisting effects that are carried forward into adulthood. The notion that the fetus or child *adapts* to the early exposure is critical in underscoring the importance of organism–environment transactions (e.g., from the fetal period) and for highlighting the explicit evolutionary influence in the model. Furthermore, the concept of adaptation distinguishes the model's focus not so much on normal versus deviant development (as with a sensitive period model) but rather on normative individual differences and the fit between the organism and its environment, that is, the organism's preparedness for its (later) environment. For example, mothers who experience increase stress in pregnancy may be signaling to the offspring and preparing it (i.e., its physiology) for the stressful environment it will encounter (Glover, 2011). Similarly, the thrifty phenotype hypothesis, which derives from the fetal programming model, proposes that poor prenatal nutrition in early life will influence glucose-insulin metabolism of the fetus in preparation for a poor postnatal nutritional environment (Hales & Barker, 2001; Silveira, Portella, Goldani, & Barbieri, 2007). There may be costs associated with certain kinds of adaptations or programming, but it is context dependent and can be judged only in relation to the organism's subsequent fit to its environment. A specific example is the case of the thrifty phenotype. This phenotype is thought to be programmed for a nutritionally poor environment and, if the individual remains in that environment, then the programming may be thought to have provided an adaptive response. In contrast, a programmed thrifty phenotype is ill-suited for a rich western diet and, when exposed to such a context—following migration, for example—may lead to higher rates of metabolic syndrome, type II diabetes, and cardiovascular disease. Accordingly, it is necessary to consider the subsequent environmental context for judging the fit of the programmed response. This component of the programming model clearly distinguishes it from a sensitive period model.

Adaptations or programming that does occur will take place in a particular period in development, that is, during a window of susceptibility or programmability. Much of the research focus to date is on birth weight and early physical growth, which are seen as indicators of early environments that may have programming effects on later somatic health (Barker, 1999; Painter, Roseboom, & Bleker, 2005; Phillips et al., 2000; Roseboom et al., 2000, 2003). Programming has been applied as a model to explain many other outcomes and phenotypes, including stress reactivity and other psychological outcomes. The implication is that there may be programmable effects on several biological

function and mechanisms, including glucose metabolism and stress physiology.

In contrast to the research base on the sensitive period model, which is heavily influenced by animal research, the research base on developmental programming has been substantially shaped by human research. Although much of the attention is on somatic health rather than psychological development, instances of possible programmed psychological outcomes have been suggested (O'Donnell, Glover, Barker, & O'Connor, 2014; Rutter & O'Connor, 2004; Sandman, Davis, & Glynn, 2012; Van den Bergh et al., 2005). One potential limitation to these studies is that the follow-up period rarely extends into or beyond adolescence, so the persistence of a programmed effect is not yet certain.

The application of programmability for intervention has not yet attracted considerable attention. Nonetheless, the clear expectation is that the degree of recovery or change of any sort would be very limited if an exposure such as an intervention occurred outside the period of programmability. This assumes, however, that the window of programming is known, and it may not be. In this context, experimental animal studies of the programmability of the Hypothalamic-Pituitary-Adrenal (HPA) axis from prenatal stress do show that the adverse effects of prenatal stress may be eliminated by postnatal manipulation (Maccari et al., 1995). Interestingly, a human study also showed that the adverse effects of prenatal stress on infant cognitive function could be eliminated if the infant was exposed to sensitive caregiving (Bergman, Sarkar, Glover, & O'Connor, 2010). Furthermore, in a rodent model, some of the effects of postnatal care deprivation could be reduced with an environmental enrichment delivered in the peripubertal period (Francis, Diorio, Plotsky, & Meaney, 2002). These findings leave open key questions about developmental timing and the programming window. For example, results from the Bergman et al. study could be explained if the programming window including both the prenatal period (when the purported risk exposure occurred) and the early postnatal period (when some children experienced a sensitive child–parent relationship). Slightly more challenging are those studies showing that interventions administered much later in development, such puberty, could reverse the presumed programming associated with prenatal stress. Similar to a sensitive period model, the window of programmability is presumed to be limited, although research on the boundaries and limits of the programming window are not always clear and it is rarely itself a target of research. And it may be possible that new periods of programming may open later in development, with implications for earlier programmed effects. That possibility is at least implicit in recent discussions of adolescence as a period during which the effects of early insults might be transformed (Dahl & Gunnar, 2009). Within the developmental programming model, additional research will be needed to examine if there is a point after which the delivery of an intervention would have no effect in resetting or compensating for a programmed response.

Life Span Development or Cumulative Effects Models

Most prominent in developmental studies of psychological development are life span development or life-course models proposing that individual differences in psychological development arise from the individual's particular profile of risk and protective experiences as accumulated through development. Concepts and metaphors such as developmental pathways (e.g., Bowlby, 1988; Sroufe, Egeland, & Kreutzer, 1990) convey the dynamic, unfolding process of development. Life-course models are distinguished from sensitive period and developmental programming models in putting explicit and significant emphasis on both current circumstances and developmental history. Furthermore, any association between early adversity and later outcome would be viewed as probabilistic rather than deterministic; moreover, early experiences would predict longer-term outcomes only insofar as the impact of early exposure is reinforced or maintained by subsequent exposure. So, for example, early adversity would be expected to have little or no—or at a minimum, diminished—effect in the absence of concurrent risk. That is, life-course or cumulative risk models propose that there is a carrying forward in development of early adverse or protective experience or exposure, but the impact is not direct but rather mediated by subsequent exposures. In other words, plasticity may be limited in sensitive period and programming models but is considerable in life span models. A further distinguishing feature of research on life span development models is their emphasis on aging and particularly senescence (Gerstorf et al., 2010; Lovden, Backman, Lindenberger, Schaefer, & Schmiedek, 2010). Reviews and descriptions of the life-course models are widely available (e.g., Baltes, Staudinger, & Lindenberger, 1999; Clarke & Clarke, 2000; Elder, 1998).

Of particular and distinguishing relevance to life span theories are normative and nonnormative transitions, including the transition to adolescence, marriage, retirement, or divorce and remarriage. These are particularly informative points in the developmental pathway; adjustments at these transition points might offer an especially

informative index of future adjustment. In this way, life span models would incorporate well-known psychological developmental stage theories of Erik Erikson, among others. Stage theories are quintessentially life span developmental hypotheses insofar as they propose something specific and distinguishing about the organism at one stage in development that differs from another stage, and that successfully resolving one developmental task increases the likelihood of successfully resolving the next (Sroufe & Rutter, 1984). One stage or developmental task may not necessarily have priority over another, but progressive successes increase the likelihood of continued healthy adaptation. It is not only psychological theorists but also sociological and other social science theorists who emphasis the life-course models for understanding individuals and their fit and adjustment into the family, culture, and economy (Mayer & Schöpflin, 1989).

A still further distinguishing feature of life span models is their focus not on frank pathology but rather on normative variation among individuals. Related to this wide potential for variation are two notions in developmental theory: multifinality, or the idea that a certain risk may lead to diverse outcomes among individuals; and equafinality, which is the notion that individuals may end up at the same (mal)adapted outcome by traversing different pathways or patterns of risk exposure (Cicchetti & Blender, 2006). No such degree of flexibility and focus on individual variation is accommodated within the sensitive period or programming models discussed above.

Whereas animal studies feature centrally in studies of sensitive period and programming hypotheses, they are rarely designed and may not be well-equipped to test life span models; a brief life span in rodents is one obvious reason for this. Alternatively, life span developmental models may be more applicable to behavioral and psychological phenotypes (emphasized in human developmental research) than biological phenotypes (emphasized in animal studies).

It is also in the intervention context that life span developmental models are easily distinguished from sensitive period and programming models. Specifically, life span developmental models emphasize that there is substantial potential for recovery following (early) exposure to adversity. A corollary is that there is not a period in development after which an intervention could not have a salutary effect. (However, to say that there could be an intervention effect is different from proposing that intervention effects will be readily demonstrated.) A life span model proposes that an individual's psychology and by extension biology remains to some important degree malleable and plastic, and this renders current experiences as potentially potent influences on the developmental course and adaptation of the individual. Indeed, empirical examples from naturalistic longitudinal studies that show dramatic life-course changes can be instituted by major transitions occurring in adulthood, including enrolment in the military (Elder, 1986) and marriage (Quinton, Rutter, & Liddle, 1984). Compared with sensitive period and programming models, life-course models place much less emphasis on the timing of the intervention when judging its potential effects.

Alternative Models

There exist other models of development for judging the likely effects of early life experiences and exposures; a few additional examples warrant at least brief discussion. One of these is canalization, which, as discussed by Waddington (1959), described a populations's tendency to produce the same phenotypes despite mild variation in various conditions. That has also been applied to an individual organism, most elaborately by Gottlieb (1991). Put another way, the notion of canalization implies a robustness of the organism's phenotype despite perturbations. The deepness or structural robustness of the canal incorporated in canalization would vary across phenotypes; presumably the more evolutionarily conserved phenotypes may be thought to have a more robust or deeper developmental trajectory in development because they may be more robust to perturbations in the environment. There remains some uncertainty about how well canalization is translated to testable experimental or intervention context, and human research using this model is limited.

In response to findings from studies of children who experienced early institutional deprivation, some authors (Gunnar, 2010; Zeanah, Gunnar, McCall, Kreppner, & Fox, 2011) have suggested that a somewhat modified sensitive period model may be needed. Particularly relevant here is the finding that children who experienced institutional deprivation that was limited to the first six months of life showed no significant differences in many key areas compared with a nondeprived group, and that variation in length of deprivation from six months to more than two years did not reliably predict longer-term outcomes (e.g., Kreppner et al., 2007). Equally important is the observation found in virtually all studies that the experience of institutional care from birth continues to have a persisting mild-moderate effect on several aspects of psychological development long after removal from the institution and placement into a normal-risk family environment. This collection of effects is not easily accommodated by leading

developmental models. There are many limitations to the existing studies, particularly concerning developmental timing effects, and patterns from one sample or design may not extend to another; nonetheless, a stepped function or other nonlinear pattern found in this important collection of studies may require a modified sensitive period model, at least in the case of very extreme deprivation in early development (Zeanah et al., 2011).

A further developmental hypothesis is the so-called sleeper effect. This is the notion that the effects of an exposure may not be apparent immediately or shortly after exposure to the presumed cause, but will become evident later in development. Wallerstein (1985) invoked this notion in suggesting that divorce in childhood may have not only immediate effects, but also effects at a later point in development, such as adolescence, when there is a rekindling of the early developmental struggle or vulnerability. Sleeper effects presume that the (positive or negative) impact caused by the exposure is not readily apparent and measureable because the biological underpinnings of the target behavior were not in place at the time of the exposure, or that the behavior had not yet developed. There are good reasons to expect delayed effects of early exposures, particularly in the context of neurodevelopmental models of psychopathology. That is because the neural circuits associated with stress response and depression may not be developed and integrated at the time of the exposure. On the other hand, one difficulty with the sleeper effect model—and the reason why it is difficult to rule in as a satisfactory explanation—is that the temporal separation of cause and effect could be explained by a cascade of intermediate effects. So, for example, a presumed sleeper effect linking early parental divorce and adolescent sexual behavior could be explained by a chain of indirect effects including increased behavioral disturbance, school difficulties, poor parental monitoring, affiliation with deviant peers, and engagement in high-risk behaviors. This indirect effects model may not only be more accurate, but also more practical for planning interventions. It would be difficult to identify specific intervention options according to a sleeper effect model.

Although influential in studies of risk and psychopathology, the diathesis–stress model is not discussed at length because it is not inherently developmental—although it remains a plausible and valuable null hypothesis for developmental studies. In its basic form diathesis–stress is not fundamentally concerned about how development configures or constitutes a diathesis or vulnerability; rather, the hypothesis is that there is an underlying diathesis is expected to lead to pathology in the context of stress and that this association is essentially invariant across development. This is similar to the experience-dependent model (Greenough et al., 1987).

Finally, it may be worth briefly highlighting, at least for historical purposes, developmental hypotheses that no longer command serious attention. So, for example, the claim that maternal bonding required immediate and extensive post-birth contact was suggested as an evidence-derived human feature some years ago (Kennell, Trause, & Klaus, 1975) but has now been essentially dismissed. It may be that that conclusion was an inappropriate extension from rodent findings, for which there is considerable support. A further example of a now discredited model is the notion of developmental arrest. Disturbances in attachment and social adjustment reported in ex-institutionalized children garnered particular attention because of the strikingly abnormal behavior, including seeking comfort from apparent strangers when distressed, which in some children persisted years after placement in a normal caring environment. The suggestion that the child's psychological development was arrested or frozen in an immature state because of the experience of profound deprivation gained popularity, which persisted for some time. The concept of developmental arrest has been around for some time, viewed by some as an innovation of psychoanalytic theory (e.g., B. Rank, 1949) and considered central to such notions as (infantile) narcissism (Kohut, 1971). To be sure, children who experienced institutional deprivation would have experienced a profound lack of mirroring that is proposed as a basis for healthy psychological development—but this is quite a different matter from the notion of arrested or stopped development. Several authors (Keck & Kupecky, 1995) suggested that the deprived child's arrested development required an extreme form of intervention to unfreeze development. That is where a metaphor inspired radical, coercive, and harmful interventions; some of these radical interventions, such as rebirthing therapy, led to child deaths (Crowder & Lowe, 2000; Mercer, 2005). In response, several professional organizations (Boris & Zeanah, 2005; Chaffin et al., 2006) have taken the extraordinary step of explicitly rejecting as unethical and dangerous coercive and holding therapies. The reason for highlighting this now defunct proposal is not because it is a viable model of development but rather because this dubious model had practical hold on how interventions were practiced, at least for some time and for some individuals. This is not the only discredited model to have had influenced thinking about development and psychopathology. Thus, the notion that birth trauma or the psychic separation from mother at birth is a basis for later anxieties (Rank, 1929) now

seems absurd. The lesson is that it is natural to expect and require that models of development remain in a state of change, requiring further empirical evidence and updating (e.g., Gedo, 1980).

Summary

There is growing recognition that many psychological disorders may be best seen as neurodevelopmental in nature and require a developmental focus (Cicchetti & Rogosch, 2002; Insel & Wang, 2010). Evidence of this takes many forms; one valuable observation is the finding that one-half of those with an adult mental disorder exhibited the problems by early adolescence and that the median age of onset of anxiety and impulse-control disorders is 11 years of age (Kessler et al., 2005). Concepts and findings of this kind underscore the value in formulating and differentiating alternative developmental hypotheses. Accordingly, this opening section sought to draw broad distinctions that might differentiate specific theories of how (early) experiences and exposures may shape psychological development. Rather than focus on individual theories of development, the focus was on broad criteria and features, such as the role of developmental timing, the predominance of animal research, and application to intervention. The three types or classes of models reviewed above may not easily accommodate all specific theories of human development, but might provide a basis for contrasting specific theory hypotheses. For example, in the area of parent–child relationships, the types of models and features previously described may help to distinguish competing theories, such as attachment theory and its focus on life span processes (Ainsworth, 1989; Bowlby, 1988) from alternative theories that place special emphasis on the early relationship (Chodorow, 1978; Winnicott, 1965). The next section identifies key mechanisms that may provide the basis for understanding why (early) experiences and exposure may shape health and development.

DEVELOPMENTAL MECHANISMS

Research findings identify several candidate mechanisms to explain the sources of individual differences underlying psychological development and the mechanisms by which psychological experiences may be transduced to biology and behavior. What constitutes a developmental mechanism can be a matter of some debate, particularly where experimental leverage is limited, as in most human studies and especially those using observational/naturalistic study designs. Indeed, it can be very challenging to identify a factor as a risk mechanism, implying causal effect, rather than merely risk factor, a term which carries much less causal certainty and may merely offer an indirect index of a causal effect. This conceptual issue has been widely discussed in the literature on psychopathology in children and adolescents (e.g., Grant et al., 2003). A further cautionary note on mechanisms is that research may show that a particular factor can be a causal mechanism when considered in isolation or in a very particular sample or context, but the impact may be modest or unknown or unknowable in more typical/normative samples or at the population level. For example, a common experimental animal approach is to study animals in which a particular gene has been experimentally and artificially inactivated; experiments with these genetic knockout animals have been used to implicate a variety of genes in many aspects of development and disease. Such a study may demonstrate that a mouse with inactivated gene X shows disturbance in neurogenesis in the hippocampus and so, by implication, gene X has a role in hippocampal neurogenesis. In fact, what the study has shown is that gene X *can* alter neurogenesis in a very particular (and abnormal) biological context; whether or not gene X *does* govern neurogenesis in the typically developing animal does not necessarily follow. Similarly, findings from several studies strongly suggest that early institutional deprivation *can* be a cause of inattention/hyperactivity (Kreppner, O'Connor, & Rutter, 2001), but the relevance for the population (e.g., in terms of population attributable risk) is uncertain given the rarity and declining numbers of children adopted from institutional settings.

Stress Physiology

Stress physiology is perhaps the most widely studied candidate mechanisms to explain the effects of (early) stress exposure on health and development; of the several stress physiology components, the most popular is probably the hypothalamic-pituitary-adrenal (HPA) axis. The prominent role of the HPA axis in developmental studies in humans is attributable to many factors, including (1) a remarkable evidence base in animal models; (2) the interpretability of markers of the HPA from peripheral samples such as saliva and blood; (3) the modest cost of obtaining and analyzing such samples; (4) the widespread relevance of the HPA axis in many areas of development, making it a quintessentially mind–body biomarker; (5) a now substantial human evidence base implicating the HPA axis and its downstream element cortisol in a wide range of

mental and somatic health outcomes; and (6) accumulating understanding of genetics of the HPA axis.

Human evidence that the HPA axis may be modified by early experiences and may underlie short- and long-term psychological health is substantial (Gunnar, 2003). One line of research has shown that the infant's HPA axis can be modified as a function of early exposures; that list of early exposures includes prenatal maternal distress, variation in early caregiving, and indirect measures of stress exposure such as maternal psychopathology, family conflict, and loss (Brennan et al., 2008; Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; Grant et al., 2009; Hertsgaard, Gunnar, Erickson, & Nachmias, 1995; Nicolson, 2004; O'Connor, Bergman, Sarkar, & Glover, 2013). An important follow-on finding is that perturbations in the HPA axis associated with early stress may have persisting effects, as is shown in studies of adults exposed to early trauma or loss (Heim, Newport, Miller, & Nemeroff, 2000; Luecken & Appelhans, 2006; Nicolson, 2004) and longitudinal follow-up of adolescents exposed to prenatal maternal anxiety (O'Donnell et al., 2013) or maternal postnatal depression (Halligan, Herbert, Goodyer, & Murray, 2007). It should not be surprising that a stress response system does, in fact, respond to significant stress. Rather, the previous findings are important for understanding early experience in two ways: (1) by demonstrating that associations between stress exposures and physiological changes can be found from early development, as even the infant's HPA axis is responsive to social environments; and (2) demonstrating reported evidence that early exposures can have lasting and persisting effects into and beyond adolescence, which in at least some studies is well beyond the period of exposure to the putative cause. To be sure, the effects sizes may be small in many cases, implying wide variation in response and the involvement of many other factors. Nonetheless, these findings help make the HPA axis a natural focus for developmental studies.

An important further observation of the HPA axis for understanding developmental mechanisms is how it responds to prolonged stress exposure. Both animal (Sheridan, Stark, Avitsur, & Padgett, 2000) and human (Miller et al., 2009) research work shows that prolonged stress exposure may lead to glucocorticoid resistance or decreased glucocorticoid receptor sensitivity. This may account for the down-regulation of the stress response following chronic and severe stress exposure, and explains why there is interest both in hyper- and hypo-response to stress in child and adult samples. This process also hints at a mechanism by which alterations in the HPA axis may influence other biological processes, such as dysregulated immune function (Cohen et al., 2012).

A developmental notion relevant to the HPA axis (as well as other physiological systems) is the allostatic load model, which focuses on the wear and tear of stress on the body over a prolonged period of time (Juster, McEwen, & Lupien, 2010; McEwen & Wingfield, 2003; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). The notion here is that adaptive neuroendocrine responses to short-term stress exposure will, if activated persistently, have deleterious effects on the body and hasten the onset of disease. What is not clear is how prolonged the exposure needs to be in order to observe effects, but it is notable that the majority of studies focus on adult outcomes (for exceptions, see Evans, 2003; Evans, Kim, Ting, Tesher, & Shannis, 2007; Flaherty et al., 2009). The implication is that it is the accumulation of stress exposure during the life course rather than stress exposure experienced at a particular point in development—a notion central to sensitive period and programming models—that creates the wear and tear and effect on the HPA and allied physiological systems. Studies based on a life span model have been especially active in incorporating HPA axis mechanisms to explain variation in health and aging (Lupien et al., 1998).

HPA axis-related mechanisms are also main features of developmental programming studies, particularly involving prenatal maternal stress and research on the developmental origins of health and disease (DOHaD) (Hanson & Gluckman, 2011; Swanson, Entringer, Buss, & Wadhwa, 2009). The hypothesis, which has been supported in many studies, is that exposures during particularly sensitive periods in ontogeny program or set glucose metabolism or one of many other functions associated with the HPA axis, with the programming carried forward into adulthood (Gluckman, Hanson, Cooper, & Thornburg, 2008). Most DOHaD-related research examines adult health outcomes, but a few have assessed childhood-onset effects (Ibanez, Ong, Dunger, & de Zegher, 2006). Finally, HPA axis mechanisms have been equally implicated in sensitive period models. One recent report suggested that, in the rodent, corticosterone (the equivalent of cortisol in the human) may influence the sensitive period for attachment learning (Upton & Sullivan, 2010). It would seem that HPA axis-related mechanisms are applicable for the range of developmental models identified in the opening section.

Neuroimmunology

One mechanism for developmental studies that has commanded particularly intense recent interest, particularly concerning early experience and neurodevelopment, is neuroimmunology. In the broadest sense, neuroimmunology is the study of the connections between the nervous

system and the immune system. Psychoneuroimmunology (PNI) expands this area one step further to incorporate behavior. The PNI model grew out of the work of Ader and Cohen several decades ago when they demonstrated that an immune function could be behaviorally conditioned using the classical conditioning paradigm (Ader & Cohen, 1975); that work has since been expanded and reinforced in several ways and in several studies across several decades of investigation (e.g., Ader, Kelly, Moynihan, Grota, & Cohen, 1993).

Research in neuroimmunology and PNI have established robust connections between the immune and the nervous system; specific examples include the finding that (1) there is sympathetic innervation of the spleen and other lymphoid organs; (2) central nervous system manipulations (e.g., lesioning the pituitary) may suppress antibody production; (3) changes in hormone or neurotransmitter levels alter immune function, and the reverse is equally so; (4) lymphocytes express receptors for hormones and neurotransmitters; (5) immunological reactivity is influenced by stress exposure and behavioral interventions. It has been appreciated for some time that stress exposure evokes an immune response, and this has likely evolutionary advantages (Ottaviani & Franceschi, 1996). Other research shows the immune system's role in neural plasticity, such as hippocampal neurogenesis and other areas (Schwartz & Kipnis, 2011; Wolf et al., 2009), and illustrates that the immune system shapes the brain by enacting cellular changes that alter synapse formation, modification, and pruning, thereby shaping neural circuits (Luo & O'Leary, 2005). That means it is reasonable and expectable that influences on the nervous system, such as stress, will have immunological effects and that immunological perturbations may be expected to alter the nervous system and brain development (the most well-known example, the maternal immune activation model, is discussed at some length in section d). These observations justify research on neuroimmune mechanisms as plausible candidates for explaining the early and prolonged effects of stress exposure, variation in neurodevelopment, and the development of psychopathology. The relevance of neuroimmune mechanisms for alternative developmental models in the opening section of this chapter will become clearer as this work unfolds.

Applications of neuroimmunology and psychoneuroimmunology in developmental models and in research with children and adolescents are, so far, quite limited (Ader, 1983; Coe, 1996; O'Connor, Moynihan, & Caserta, 2014). There are several reasons for this. One is the complex nature of CNS-immune connections and, within the immune system, the extraordinary difficulty in understanding the effect of one target amidst a very dynamic system. And, even where immune target effects are demonstrated, the nature of the effects is complex, as in the case of interleukin (IL)-1, which may have both facilitating and impairing effects on learning and memory in the animal model (e.g., Yirmiya & Goshen, 2011). Additionally, candidates for mechanistic research in developmental studies may not be practical research targets in humans. An obvious example is microglia, the resident macrophages of the brain which provide surveillance and maintenance functions. Animal studies provide ample evidence for their role in healthy brain development and repair. A case in point is the study by Paolicelli et al. (2011) who used a knock-out mouse model to demonstrate that microglia played a critical role in synaptic pruning, an essential function for normal development; mice with reduced microglia displayed immature brain circuitry. In this way, microglia may play roles in sensitive period modulation, and in developmental programming of the brain associated with early immune activation (Bilbo, 2013). But, in human developmental research, microglia remain delitescent. That is, human research into microglia rely on the use of post-mortem studies or from recent advances in positron emission tomography (PET) scan studies using specific radiotracers that specifically bind to microglia (Suzuki et al., 2013), but these approaches have evident limitations.

An example of immune function on behavior—and by implication brain—in children and adolescents is Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections (PANDAS), a sudden onset of obsessive-compulsive and tic symptoms following streptococcal infection (Garvey, Giedd, & Swedo, 1998; Swedo & Grant, 2005). In addition, there has been considerable interest in describing immune alterations in autism (e.g., Ashwood et al., 2011; Suzuki et al., 2013), although the etiological and treatment significance of the findings so far reported are unclear, and somewhat difficult to summarize. In general, the extent to which altered immune function is evident in children with behavioral or emotional problems is difficult to estimate because of the limited data available (Birmaher et al., 1994; Keller, El-Sheikh, Vaughn, & Granger, 2010) or reliance on medically ill samples (Mackner, Pajer, Glaser, & Crandell, 2010; Sokal et al., 1998).

In any event, it would appear, based on the current data, that associations between immune markers and psychopathology in children and adolescents may not mimic adult patterns, such as in the case of the cytokine hypothesis in depression (e.g., Mills, Scott, Wray, Cohen-Woods, & Baune, 2013). Indeed, although there is substantial basis for investigating immune mechanisms from early

childhood in relation to stress exposure for what impact it may have on brain and behavior, it is far from clear how the developing immune system may respond to early stress exposure. The young immune system, because it is under development, may be especially vulnerable to the effects of stress; alternatively, it may be less susceptible to long-standing adverse effects because of the greater immunological reserves in younger compared with aged individuals. It is similarly unclear if the immune system early in development would be impaired by stress exposure—the effect readily discernible in adults and especially older adults—or accentuated by stress exposure. Evidence for the latter was demonstrated in several studies, such as an ambulatory sample of preadolescents in whom an index of caregiving and family stress predicted enhanced natural killer (NK) cell functioning (Caserta et al., 2008). In early development, early stress may activate the immune system which may appear, after prolonged exposure, to take the form of down-regulation that has been shown in adults. Later in development, the effect of prolonged inflammatory cytokine elevation may hasten the onset of immunosenescence, which has been operationalized in terms of decreased naïve T cell numbers and declining adaptive responses (Franceschi et al., 2007; McElhaney & Effros, 2009; Pawelec, Larbi, & Derhovanessian, 2010); furthermore, these immune response may be linked with other cognitive and health declines in high stress-exposed individuals. That is, early and chronic psychosocial stress may accelerate the typical process of immunosenescence via exaggerated immune responses. Although the mechanisms in development may not yet be clear, what is certain is that neuroimmune mechanisms could explain why there is a remarkably robust association between early stress exposure and a wide range of disorders, including psychopathology but also autoimmune disease, cardiovascular, periodontal, and respiratory diseases, cancer, and other ailments (Almeida et al., 2010; D. W. Brown et al., 2010; Chen, Miller, Kobor, & Cole, 2010; Dube et al., 2009; Jackson, Kubzansky, Cohen, Weiss, & Wright, 2004; Lemieux, Coe, & Carnes, 2008).

Neural Development and Neural Circuitry

Neural mechanisms in development such as neuronal pruning, specification, consolidation and connectivity, and sculpturing of brain circuits and how these principles underlie such central developmental concepts as plasticity have been discussed at length (Horn, 2004; Knudsen, 2004; Luo & O'Leary, 2005; Sirois et al., 2008; Sur & Rubenstein, 2005). There is obviously a brain basis to the kinds of effects prescribed by a sensitive period, programming, or life-course model as described in the opening segment. The perennial challenge for human psychological research is to document these brain changes, establish their links with behavior, and track intraindividual changes across development to differentiate among competing developmental hypotheses.

One set of findings on human brain development that requires particular attention is the emerging evidence on the amount of brain volume change in the course of the first year, and how the amount of change varies across brain region. For instance, one program of study (Gilmore et al., 2012; Knickmeyer et al., 2008) reported that total brain volume may increase as much as 100% in the first year, and much less in the second; that this increase was largely accounted for by increases in grey matter; that volumetric increases in early infancy may be especially pronounced in the cerebellum; and that changes in specific regions of particular interest in neurodevelopment such as the hippocampus change at rates different from other brain regions from birth to 2 years of age. In addition, other studies suggest that synaptogenesis in the visual and auditory cortex peaks in the early months of life but is more protracted in the prefrontal cortex (e.g., Huttenlocher & Dabholkar, 1997). These findings are obviously central to designing studies, interpreting findings, and developmental theorizing. For example, given these findings it may not be surprising how difficult it is to trace and document stability in stress physiology or effects of stress exposure in early infancy. Variable rates of developmental changes across brain region in early infancy also raise important questions about how to track the formation of brain circuits connecting different brain regions (discussed next); a possible corollary is that no single developmental hypothesis is likely to offer a satisfactory account for such as phenotypes as stress physiology, executive functions, reward learning, or social communication. Also, it remains to be seen how the dominant method in early development of using structural MRI can be translated to more specific questions concerning neural mechanisms involving glia, dendrites, axonal arborisation, and other factors and functions.

A second point concerns the move away from focusing on specific brain regions and links with behavior to systems-level approaches, examining neural circuits and brain connectivity. Conceptual and technical findings and principles have been discussed for normal development (Tau & Peterson, 2010), and examples have been reported for many conditions, including language processing (Gozzo et al., 2009), and in relation to many exposures, such as prenatal maternal anxiety (S. Sarkar et al., 2014).

Movement away from analysis of specific brain regions to the development and change in neural circuits reflects the growing interest in systems neuroscience.

Genetics

There is now no doubt that genetic factors influence a wide array of psychological processes and outcomes across the life span (e.g., Plomin & Rende, 1991). Accordingly, genetic mechanisms are a natural focus for research on how and under what circumstances early exposures may have lasting effects on psychological development. One important line of study has focused on how genetic factors shape behavior that increases exposure to environmental risk, and how genetically influenced biologically factors moderate the effects of exposure. Several findings—insofar as they hint at general principles—are especially relevant to developmental models of psychological development. One is the early emerging genetic influences on behavior, with effects evident for a wide range of behavioral phenotypes from early childhood (Eley et al., 1999; Saudino & Eaton, 1991). A second is the evidence that genetic influences appear to strengthen over time as indexed, for example, by increasing heritability estimates from twin studies or, in an impressive longitudinal analysis, increasing resemblance of adopted-away children to their biological parents (Plomin, Fulker, Corley, & DeFries, 1997).

Molecular genetics research seeks to identify specific genes with putatively direct influence on brain and biology via a cascade of biological processes from brain cells to circuits, e.g., see (Plomin, 2005) and how these allied biological effects modify environmental exposures. Findings from this area of study reinforce the view that there are early-emerging genetically-mediated effects of the environment on behavior apparently associated with specific genes; and, by focusing on specific gene systems, this line of research may also point to biological mechanisms. Many examples with application to developmental models have been reported. For example, in the specific area of child–parent attachment, research findings suggest that children possessing the short variant of the 5-HTTLPR gene were more likely to exhibit an insecure attachment in infancy if they also were exposed to insensitive caregiving (Barry, Kochanska, & Philibert, 2008). The role of dopamine-related genes is also notable in this line of study (e.g., Bakermans-Kranenburg & van Ijzendoorn, 2006; Propper, Willoughby, Halpern, Carbone, & Cox, 2007). Another example with particular relevance to early risk exposure and alcoholism was reported by Barr and colleagues (Barr et al., 2004), who found that the effects of early deprivation on alcohol risk were limited to those female animals at genetic risk according to serotonin transporter gene.

A broader hypothesis with potentially significant impact on development theory proposes that there are genetic influences on susceptibility to the environment; that is, the *same* gene (or more likely, genes) may be associated with better outcomes in the context of positive environmental conditions *and* more negative outcomes in the context of adversity (Bakermans-Kranenburg & van Ijzendoorn, 2007; van Ijzendoorn, Belsky, & Bakermans-Kranenburg, 2012). A recent meta-analysis (van Ijzendoorn et al., 2012) suggested that the serotonin transporter gene may show this susceptibility pattern rather than a vulnerability pattern. Particularly interesting supportive evidence for this model was suggested in a study of children who experienced early institutional deprivation, some of whom also experienced a major early intervention to improve caregiving quality; analyses suggested that the serotonin transporter and brain-derived neurotrophic factor (BDNF) genes may act as susceptibility genes with respect to indiscriminate social behavior (Drury et al., 2012).

Specific genetic influences on the sensitive period phenotype have also been reported, although the evidence is limited to animal models (Hensch, 2004). For example, Huang and colleagues (Huang et al., 1999) demonstrated mice genetically manipulated to overexpress brain-derived neurotrophic factor (BDNF) showed accelerated development and early closure of the critical period in the visual cortex. A complementary proposal that environmental exposure in a sensitive period in development may alter gene expression relevant to NMDA has also been reported (Kinnunen, Koenig, & Bilbe, 2003). Additionally, evidence of ontogenetic changes in glucocorticoid receptor (GR) gene expression in brain development suggests sensitive period of glucocorticoid sensitivity, with application to sensitive period and programming models (Speirs, Seckl, & Brown, 2004). Thus, a picture is emerging that several different gene systems may be relevant for modulating the effects of early exposures on development and perhaps on the sensitivity or susceptibility window. Further work of this kind, insofar as it can be translated to human development, may prove to be especially influential in shaping developmental hypotheses.

More recent research on epigenetic mechanisms offers a powerful method of understanding and redefining genetic influences for shaping developmental models (Meaney, 2010). DNA methylation is one specific epigenetic mechanism that has attracted research attention because the biological processes of methylation and gene silencing

are reasonably well understood (Reik, Dean, & Walter, 2001; Seisenberger et al., 2013), and disrupted patterns of DNA methylation have been associated with specific diseases such as cancer and neurodevelopmental disorders (Urdinguio, Sanchez-Mut, & Esteller, 2009). DNA methylation patterns undergo dramatic developmental change from embryogenesis, suggesting that these systems may be malleable in development (Y. Bergman & Cedar, 2013; Seisenberger et al., 2013). Furthermore, animal models are beginning to demonstrate that exposures in the (early) social environment may induce epigenetic alterations (Champagne et al., 2006; Szyf, Weaver, & Meaney, 2007; Weaver et al., 2004, 2005).

Moving to human research, published reports suggest that childhood maltreatment and traumatic stress are associated with DNA methylation patterns in adulthood (e.g., Mehta et al., 2013); few reports show effects as early as mid- and late adolescence (e.g., Essex et al., 2013). There are important limitations to many of the studies in the area, however. A key question for translating these findings and assessing their reliability derives from the tissue-specific nature of the epigenetic effects, which have been identified in the animal studies but not yet certified in human studies—aside from the limited research on cadavers (e.g., Labonte et al., 2013). There is also limited research including multiple occasions of measurement. That concern is particularly relevant for developmental studies given that normative changes in DNA methylation are particularly notable in pediatric samples (Alisch et al., 2012). What has not yet been reliably documented and replicated is that an hypothesized measure of stress in the social environment is associated with stable methylation patterns. Nonetheless, research has begun to document some of the early predictors of DNA methylation in very young children, including prenatal factors (Herbstman et al., 2013).

A range of plausible candidate gene systems have attracted attention in methylation studies, including the glucocorticoid receptor gene (NR3C1), which has emerged in the animal literature as a leading candidate (Weaver et al., 2004) and is one of the very few replicated human findings across multiple *different* sample types using a range of samples and approaches (Hompes et al., 2013; McGowan et al., 2009; Oberlander et al., 2008). In recognition of evidence for associations between PTSD and immune function, and in support for the review of neuroimmune mechanisms above, immune-related genes are also good candidates for focused gene approaches (Guffanti et al., 2013; Rusiecki et al., 2013). Alternatively, the switch to genome-wide methylation studies will likely gain greater influence in subsequent research (Lam et al., 2012).

A key theme in research on genetic mechanisms, from the earlier work on behavioral genetics to the more recent work on molecular genetics and epigenetics, is on genetic influences modulating development and change. Accordingly, genetic mechanisms will remain an essential component for forming, supporting, and disconfirming models of psychological development.

Psychological Mechanisms

Quite what constitutes a psychological mechanism will likely generate some debate (Bechtel, 2008). But, there should be little debate that attachment theory and its internal working model is an often-referenced candidate for understanding how and if early experience shapes long-term psychological development (Bowlby, 1982; Bretherton, 2005; Steele, Steele, & Johansson, 2002). In infancy, secure internal working models are thought to develop from sensitive and responsive caregiving, from which the child develops a model of the attachment figure as available, protective and caring, and of the self as worthy of love and protection. Children who experience unresponsive and insensitive care form a contrary insecure internal working model. Caregiving behaviors in the early months of life predict security of attachment at one year, as shown in the elaborate and elegant work of Beebe and colleagues (e.g., Beebe et al., 2010). A meta-analysis of some years ago demonstrated that sensitive and responsive care does predict a secure internal working model, at least to a moderate degree (De Wolff & van Ijzendoorn, 1997). Furthermore, the secure versus insecure quality of the internal working model, as derived from several different kinds of attachment measures from infancy through adolescence, is associated with many measures of socio-emotional and behavioral well-being (Allen, Moore, Kuperminc, & Bell, 1998; Arend, Gove, & Sroufe, 1979; Bureau & Moss, 2010; Futh, O'Connor, Matias, Green, & Scott, 2008; Groh et al., 2014; Schneider-Rosen & Cicchetti, 1984; Toth, Rogosch, Sturge-Apple, & Cicchetti, 2009). The above findings underscore two valuable and interlinked lessons for developmental models of early experience: the internal working model mechanism/hypothesis is early emerging and psychologically and behaviorally relevant for current and future clinical outcomes.

Attachment theory and its internal working model mechanism hypothesis, as it is most often expressed, fits squarely within the life-course developmental model, that is, rather than the sensitive period or programming models. That is implicit given the description of the developmental changes in the organization of the child–parent

attachment and explicit in the use of the concept and metaphor of developmental pathway. For example, developmental change is found in the move toward a goal-corrected partnership in preschool-aged and older children (Bowlby, 1982; Marvin & Britner, 2008), reflecting that the child and parent are increasingly able to operate toward a shared set of negotiated goals. Studies of attachment in adolescence have also described important ontogenetic changes and alterations in how attachment qualities and the internal working model in particular may be best assessed (Allen & Miga, 2010; Kobak, Cole, Ferenz-Gillies, Fleming, & Gamble, 1993; Obsuth, Hennighausen, Brumariu, & Lyons-Ruth, 2013; Scott, Briskman, Woolgar, Humayun, & O'Connor, 2011). The life-course model underlying attachment theory and the internal working model mechanism is more explicitly described by and expanded by Bowlby (1988) and Ainsworth (1989). And, in fact, empirical research shows that internal working models of security versus insecurity do, in fact, change in the course of development and (presumably) in response to changes in care arrangements. Although both long-term stability (Waters, Merrick, Treboux, Crowell, & Albersheim, 2000) and instability (Weinfield, Sroufe, & Egeland, 2000) have been reported, the core conceptual feature of the internal working model for this chapter is that it is open to changes in the caregiving arrangement. That is central to the concept of lawful discontinuity and the focus on the developmental history and current state of intimate relationships in attachment theory.

Many other psychological mechanisms implicated in the broad class of activity of information processing (e.g., memory) would also need to be cited here insofar as they too can have own to have carry-forward effects from prior experience. Both the content of memory and the integrity of the brain in the processing and storing of memories are now active areas of study in the early experience literature, particularly in the context of trauma and child maltreatment. Thus, imaging studies suggest that retrospectively reported maltreatment and trauma was associated with a reduction in hippocampal volume (Teicher, Anderson, & Polcari, 2012); other studies suggest that the experience of maltreatment may sensitize, impair and distort memory for specific subsequent experiences, especially where negative affect, trauma, and threat may be salient (Goodman et al., 2011; Goodman, Quas, & Ogle, 2010; cf. Howe, Toth, & Cicchetti, 2011). Quite how these dynamics of memory aid in constructing psychological processes such as attributions and related social learning theory constructs requires is yet unclear and requires further investigation.

The history of psychological theory is richly adorned with theories that placed particular and special attention on the child's experiences in the early years and their ability to predict long-term outcomes. Nonetheless, it is notable that there are few, if any, examples of psychological mechanisms that have been developed and empirically demonstrated to account for a sensitive period or programming effects of early experience on psychological development. Psychological theorizing has adopted life-course model, whether the focus originates in early attachment, resilience, or specific stages in the life course (Erikson, 1968).

Additional Mechanisms

Other candidate mechanisms have been proposed to explain sensitive period, developmental programming, or life span development effects. For example, evolutionary pressures may help explain or provide a context for interpreting the impact of early stress exposures on psychological development. If, for instance, a particular function were so important to the normal development of the organism (e.g., for reproductive fitness), then it might make little evolutionary sense for this function to hinge on very specialized demands from the environment in terms of quality or quantity or timing. That, then, would imply that one may want to be cautious about presuming that windows of sensitivity or programming are narrow and demanding. Somewhat related to this notion is the proposal there are evolutionary advantages to the relatively protracted period of prefrontal cortex development in humans (and nonhuman primates) (Thompson-Schill, Ramscar, & Chrysikou, 2009). Countering this view is the modifier that it might not be adaptive for the organism to be equally and endlessly mutable because of the presumed cost that that would carry, although operationalizing and then testing this hypothesis at the psychological or biological level would be a distinct challenge. An alternative expression of evolutionary pressures is the concept of adaptation, and the corollary that there are evolutionary advantages of adapting, that is, responding to early environmental exposures. Along these lines, a remarkable example of prenatal exposure, specifically population density, triggering growth rates in offspring via glucocorticoids was recently reported in free-ranging red squirrels (Dantzer et al., 2013). The findings underscore how maternal experiences may instigate basic biological changes that can be viewed in the context of survival fitness and evolutionary adaptation. More broadly, integrated neuroendocrine response to stress has likely evolutionary advantages from pregnancy and can at least be traced in evolutionary terms, and

Crespi & Denver (2005) discussed how the ability to alter development and growth, one definition of plasticity, may be seen in evolutionary terms. Specific hypotheses derived from an evolutionary perspective in this area have been proposed, such as the infection–defense hypothesis (Anders, Tanaka, & Kinney, 2013), which offers one example of how connections between immune function and mental disorders may have evolutionary significance. Translating these findings for human health and development and clinical application may not be practical, however.

Specific neurotransmitters have also attracted attention in research on early development. Serotonin is one example. A mouse study reported that inhibiting serotonin transporter expression in early development with fluoxetine was associated with behavior consistent with animal models of anxiety and depression; the pharmacological effect was evident only in animals with a genetic variant of the serotonin transporter associated with reduced activity (Ansorge, Zhou, Lira, Hen, & Gingrich, 2004). Implications of these findings for use of SSRIs in pregnancy (for the fetus) and with young children may be substantial, and require follow-up, and that is now underway (Oberlander, Gingrich, & Ansorge, 2009). In addition, catecholamines such as norepinephrine may be plausible mechanisms mediating the effects of early life exposures because they are stress-responsive from early development and because they have neuromodulating effects in the central nervous system and the peripheral sympathetic nervous system (Rifkin-Graboi, Borelli, & Enlow, 2009; Taylor, Way, & Seeman, 2011). Dopamine-mediated roles in brain development have been equally widely discussed and reviewed in relation to specific early exposures such as prenatal substance use and in more general terms of neurodevelopment (Money & Stanwood, 2013; Ross, Graham, Money, & Stanwood, 2014). As in the case of specific genes, there are good examples in the animal work implying that many specific neurotransmitters may mediate the effects of early stress exposure on brain development and their long-term effects; the task now is not so much to isolate one or the other in developmental studies but rather to attempt to characterize their compiling and conjoint effects.

A more recent area of study is the role of the gut microbiome and its influence on brain development (e.g., Forsythe, Sudo, Dinan, Taylor, & Bienenstock, 2010). Some of the early animal work in this area focused on gut permeability and the consequent potential role that microbiota may play in neurodevelopmental disorders (Hsiao, McBride, Chow, Mazmanian, & Patterson, 2012; Hsiao et al., 2013). Application to human development is not yet clear, but this work has helped to generate interest and synthesize preliminary human findings implying gut microbiome connections with the brain from early development. Somewhat related and much better developed in human research are studies focusing on the micronutrient environment in prenatal or early postnatal period and its potential long-term effects (Brown & Susser, 2008; Coe, Lubach, Bianco, & Beard, 2009; Grantham-McGregor & Ani, 2001; Jacka et al., 2013); the advantage in this area is that many studies are on humans rather than relying on animal models.

Summary

Evidently, many different forms of mechanisms may explain how and to what extend early experiences—both good and bad—may have persisting effects on psychological development. One of the key lessons from research, which is not always evident in any one particular study, is the overlap and interdependence of mechanisms of early stress exposure and psychological development; that has scientific, clinical, and training implications (e.g., Garner & Shonkoff, 2012). Specific examples of the interdependence of developmental mechanisms are numerous. For example, prolonged stress exposure may be associated with glucocorticoid receptor insensitivity, which may lead to excessive innate immune responses and increased inflammation, with implications for neuropsychological and psychological development. Links between stress physiology and neuroimmune mechanisms are abundant and compose just one of many different kinds of coordinated responses in mechanisms of stress and health. The interdependence of these risk mechanisms may make it difficult to determine the particular effect of any risk mechanism in a human in vivo context and to differentiate among competing developmental models of early experience. Perhaps the key point here is that there are now many candidate mechanisms for understanding how early experiences and exposures might have lasting effects on psychological development.

CONCEPTUAL AND METHODOLOGICAL CONSIDERATIONS FOR INVESTIGATING DEVELOPMENTAL MODELS AND MECHANISMS

This section highlights some key conceptual and methodological lessons and challenges for research on developmental models and mechanisms with reference to applications in human psychological development.

Limited Experimental Leverage to Assess Timing, Duration, and Intensity of Exposures

Research efforts to test and differentiate alternative hypotheses about the role of (especially) early risk exposures and experiences on human psychological development are hampered by a general lack of experimental leverage for discerning the particular impact of timing, during of exposure, and dose. In fact, even where experimental leverage is optimized, as in the case of a randomized control trial, there are practical complexities that undermine the experimental control: research subjects do not attend the full complement of purportedly necessary sessions; research subjects may engage in other exposures or experiences that are not part of the intervention but nevertheless influence the targeted outcomes; treatment exposure may vary across interventionist or site; and subjects present with a diverse set of possibly confounding or moderating factors, from age and sex to developmental history and genetics. This situation contrasts sharply with typical experimental animal studies in which the type, start and end points, and intensity of dose of exposure are tightly controlled; furthermore, the developmental and genetic background of the animals is often also tightly controlled, for example, through the use of inbred strains. These widespread differences in experimental control highlight one major the challenge in translating findings in this area from animal to humans.

Of course, most studies in the human literature on early experience and psychological development are not controlled intervention trials but observational studies, leading to still further problems because of the lack of experimental and causal leverage. One of the most intransigent problems in observational studies is stability in risk exposure, which confounds research into timing effects. In fact, the most widely researched exposures of early significant stress such as poverty, maltreatment and other indicators of early caregiving, violence exposure, and parental mental illness are very stable. In response to the wealth of data linking early experience of maltreatment with a range of biological and behavioral disturbances (Cicchetti, Rogosch, Gunnar, & Toth, 2010; Cicchetti, Rogosch, Howe, & Toth, 2010; Heim & Nemeroff, 2002; Newport et al., 2003; Pollak, Cicchetti, Hornung, & Reed, 2000), several groups have sought to examine if these adverse outcomes can be linked specifically to early maltreatment or to exposure at other sensitive periods such as adolescence (Andersen et al., 2008; Bolger & Patterson, 2001). The problem is that, in principle, firm conclusions about timing are essentially impossible to discern using observational designs because of the confounding of exposure at one time point, exposure at other time points, and cumulative exposure. And, although studies which assessed individuals who were exposed to, for example, maltreatment only during a very particular and limited period may have leverage for testing timing hypotheses, these studies would not likely yield clinically importable findings because of the unusual nature of the sample. Importantly, the stability in exposure and consequent difficulty in demonstrating a timing effect is likely also to be the case for other nonpsychosocial contributors to psychological development, such as pathogens in the environment, diet and nutrition.

Intervention study designs could provide greater leverage for assessing causal effects and have been instrumental in contributing to the conceptual models of early experience for later psychological development. Notable examples are the Bucharest Early Intervention Project, discussed in the next section (Nelson et al., 2007), and the set of well-known intervention trials using nurse home visitors or the early school setting, discussed in the final section.

One way of circumventing the limits of observational studies to assess developmental timing is to capitalize on experiments in nature (Bronfenbrenner, 1979) to provide experimental leverage. Natural experiments are conditions that naturally arise in some particular context in which there is a possibility of separating otherwise confounded processes or investigating effects that could not be designed for ethical or practical reasons. The next section of this chapter, which considers several research paradigms, incorporates many examples of natural experiments. Natural experiments have been proposed as something of an antidote to the problems of confounded causes and effects in observational (nonexperimental) studies; they do have advantages and offer purchase on causal hypotheses not provided in a typical observational study. However, the leverage provided is still limited, and there may be features of the natural experiment that may limit the generalizability of the findings. The ability of natural experiments to provide generalizable lessons for clinical practice is a particular matter of debate.

Phenotypic and Measurement Stability

Presumptions in developmental studies include (1) the construct of interest can be stable; (2) change in the level or expression of the phenotype is predictable (i.e., nonrandom responsiveness); and (3) some measurements of the phenotype are suitable across an age span (i.e., there are equally valid indicators of the underlying construct across age).

The first point—that there can be stability of the phenotype across a period of interest—is obviously quite

fundamental. An equally fundamental question concerns the age on onset of phenotypic stability; that is, from what age can a construct (e.g., intelligence, social competence, stress sensitivity) be reliably assessed and expected to predict subsequent measurement? It is important to note that some candidate developmental mechanisms do not show stability from early infancy. One poignant example is the HPA axis, which, as already noted, is a leading candidate mechanism for understanding how psychological stress may influence brain and behavior. The HPA axis undergoes rapid developmental change during infancy, as illustrated by the lack of diurnal rhythm in early infancy; some infants fail to show diurnal rhythm until further along in infancy (de Weerth, Zijl, & Buitelaar, 2003). Regardless of the mechanisms that underlie these changes, an important observation from developmental studies is a lack of stability of individual difference in cortisol reactivity even over comparatively short periods in early infancy (Gunnar, Hertsgaard, Larson, & Rigatuso, 1991; Lewis & Ramsay, 1995; Tollenaar, Jansen, Beijers, Riksen-Walraven, & de Weerth, 2010). When in development there is stability is not yet clear, although one study reported stability of individual differences in reactivity between 12 and 18 months of age (Goldberg et al., 2003). The implication is that studies predicting HPA axis function in early infancy may have identified only a transient effect. Other putative mechanisms in development and psychopathology show no/minimal stability from early to late infancy, including indicators of immune function (O'Connor, Winter, et al., 2013), which, as noted, has gained popularity as an early influence on brain development. A lack of stability from early infancy may not be too surprising and is certainly explicable in terms of rapid brain changes, which vary across brain region (Gilmore et al., 2012). A lack of stability from early infancy is important for hypotheses that place particular emphasis on the role of experiences and exposures in early development: predicting biological or behavioral phenotypes in early infancy may be a valuable observation, but could be transient and may not offer clear direction for later development.

Another version of this challenge is linked with the notion of heterotypic continuity, or the notion that a developmental construct may take on somewhat different forms at different stages of development and so require different measures. One well-known example may be intelligence, as long-term studies show stability of individual differences from quite different seeming measures in infancy, childhood, and adulthood (Fagan, Holland, & Wheeler, 2007; Thompson, Fagan, & Fulker, 1991). Other instances of hypothesized heterotypic continuity exist, such as the shift from behavioral to narrative assessments of attachment; alternatively, the limited stability in some studies (Bar-Haim, Sutton, Fox, & Marvin, 2000) might also raise questions about the extent to which different measures are actually indexing the same underlying construct.

Scientists studying human psychological development are not alone in struggling with phenotypes that may not be stable from childhood or carry the same meaning at different points in development. The US Preventive Services Task Force (USPSTF) recently declared that there is not a sufficient basis to justify screening for primary hypertension in asymptomatic children and adolescents because it had unproven prognostic and clinical value (Moyer, 2013).

Few would expect or require stability in a phenotype or putative causal mechanisms from birth; on the other hand, there is far more uncertainty about whether a presumed key developmental mechanism such as HPA axis function should show stability from infancy or what it would mean if robust stability could not be demonstrated until middle childhood or later.

A related concern about measurement is not so much the stability or instability of a phenotype but how detectable it is and at what age. For example, it may be that early insecure attachment or early institutional deprivation to which a child is exposed in infancy may have major effect on the nuances of social interactions that compose peer relationships (Hodges & Tizard, 1989; J. M. Kreppner et al., 2007), but such impairments would not likely be evident until middle childhood when peer relationships take on particular developmental significance and can be rigorously. Similarly, early deprivation may undermine the ability to provide sensitive care for one's own offspring or predict accelerated cognitive decline associated with aging, but these effects will not be evident if the assessment in limited to childhood and adolescence. (It is an open question if the prodromes or intermediate phenotypes of these later-onset outcomes would be). The implication is that the presumed effects of an early exposure need to be put in the context of the age and developmental stage of the assessment, which will inevitably be limited.

Individual Differences

Strikingly, all of the many human studies of children who experience profound early deprivation amply illustrate that even the most severe form of environmental and caregiving deprivation is followed by wide variation in major psychological outcomes, including attachment relationship quality, cognitive competence, and behavior

and emotional problems (Gunnar & van Dulmen, 2007; Merz & McCall, 2010; Nelson et al., 2007; O'Connor, Marvin, Rutter, Olrick, & Britner, 2003). The magnitude of individual differences in response to even very major stresses—and the presumed resilience that that implies—is one of the more important lessons from human research and presents significant challenges for explanatory models. As outlined in detail in a following section, the consistent finding is that only a minority of children who experienced early institutional deprivation show severe attachment disturbance (although the percentage is obviously higher than what is observed in a typical population) and that the range in cognitive abilities in these children is substantial and includes normal and even above average scores (although the overall average is lower than what would be expected in a typical sample). The observed degree of individual variation and resilience would be inconsistent with a simple sensitive period model of the early years, which would predict substantially higher rates of severe disturbance no matter the (generally high) quality of the adoptive environment into which children were placed following institutionalization. Furthermore, the persistence of effects of early institutional rearing long after removal from the institution and despite adoption into generally low/normal risk and well-resourced families is a challenge to the pattern anticipated by life span hypotheses, which would predicting a diminishing effect. A further imprint of individual differences is that deficits associated with early institutional rearing seem particular to certain outcomes, but whereas some children show deficits in cognitive functioning, others may have deficits in social and attachment behavior (Kreppner et al., 2007). Of course, findings and lessons from this unusual paradigm may not generalize to the kinds of exposures that dominate clinical and developmental research. Nonetheless, any model of human development will need to consider the wide variation in individual differences, including a likely domain-specific pattern of results.

There seems to be an exceptionally long list of possible sources for the individual differences that may confound studies of early exposures and subsequent health and development. Perhaps the most is genetics. Indeed, recent work has considered if genetic factors may account for the variation in social and cognitive outcomes following environments as severe as institutional deprivation. Some positive findings have been reported (e.g., Drury et al., 2010). This will likely play increasingly important roles in studies of early exposures and interventions. Many studies have been and are likely to continue to be underpowered and focused on a very limited number of genes and Single Nucleotide Polymorphisms (SNPs), so replication of findings will be an essential step for extending this form of research to conceptual models and clinical settings.

Several modifications or suggestions for interpreting individual differences have been offered that may prove helpful. Some authors (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011) have suggested that susceptibility to environmental exposures may itself be an individual differences variable; the key point is that there are individual differences in susceptibility rather than only in vulnerability. More specifically, the hypothesis is that some individuals would be more likely affected by, for example, early caregiving neglect as well as early sensitive care; others would be less susceptible to both early neglect and early sensitivity. Tests of this hypothesis continue and have infiltrated observational developmental studies (Pitzer, Jennen-Steinmetz, Esser, Schmidt, & Laucht, 2011); genetic analyses (Belsky & Beaver, 2011); and intervention research (Scott & O'Connor, 2012). Another consideration for individual differences in (early) exposure is the possible role of genetic influences on sensitive periods in development, a topic mentioned already. It is not yet widely supposed that genetic factors such as BDNF may alter sensitive periods in human development but, like the differential susceptibility hypothesis, that could also account for some of the findings obtained. Quite how human research could gain adequate experimental leverage for formally testing this possibility remains to be determined.

Sex Differences

Sex differences in response to stress, which may be further compounded by age, are found at multiple levels of analysis, from stress reactivity (Gallucci et al., 1993; Kirschbaum, Wust, Hallhammer, 1992) to response to early adversity and potentially genetically mediated responses (Barr, Newman, Schwandt, et al., 2004) to behavioral symptoms and disorder (Kessler, 2003). Sex differences do not lead directly or clearly to a mechanism. That is because, at least in humans, sex and gender are proxies for a range of hormonal and social and psychological differences that covary and are therefore difficult to disentangle. Nonetheless, it is significant that many of the mechanisms reviewed above have strong biological bases for sex differences. For example, the HPA and hypothalamic-pituitary-gonadal (HPG) axes show considerable overlap and interaction (Lovejoy & Barsyte-Lovejoy, 2013). Furthermore, progesterone provides many examples of communication with the stress response, including that progesterone is released by the brain and adrenal gland; progesterone

is an indirect precursor to cortisol; both progesterone and cortisol are released in response to adrenocorticotropic hormone (ACTH) and stress; progesterone administration may dampen cortisol response to stress (e.g., Wirth, Meier, Fredrickson, & Schultheiss, 2007). As a result, exposures that affect or program the stress response system could be expected to influence and be influenced by sex steroid hormone production.

Biological evidence for sex steroid involvement in the immune response is also strong. A starting point is the pronounced sex difference in infectious and autoimmune diseases (Guerra-Silveira & Abad-Franch, 2013; Lang, 2004). Mechanistic research makes a comparable point. For example, estrogen enhances humoral (B cell) responses and may enhance the T cell response (Lang, 2004); estrogen regulation of the innate immune response has also been demonstrated, including effects on dendritic cells and the production of TNF-alpha (Carruba et al., 2003; Ito et al., 2001; Kramer, Kramer, & Guan, 2004; Wang, Wang, Zhao, & Qiao, 2005; Wilder & Elenkov, 1999); moreover, a different sex steroid 17-beta estradiol enhances innate responses to challenge (van Eijk et al., 2007).

Animal studies of early stress exposure provide ample evidence of sex moderation of effects. For instance, males and female offspring show different patterns of developmental programming (Bale, 2011); in some cases, only one sex appears to be affected by prenatal stress (Howerton, Morgan, Fischer, & Bale, 2013). Sexually dimorphic effects are also found, as in the case of a rodent study showing that prenatal restraint stress was associated with increased anxiety in males but decreased anxiety in females compared with controls (Zuena et al., 2008). It might be presumed that sex differences would also be readily observed in human studies, but that is not always the case. Notably, studies of children adopted following institutional deprivation do not regularly find marked sex differences (Kreppner et al., 2001), and very large-scale studies of prenatal maternal anxiety or stress often fail to detect sex differences (O'Donnell et al., 2014).

Translation of Animal Findings to Human Health and Development

Given the dominance of experimental animal studies in this area, a major obstacle for constructing models of early experience is to estimate the degree to which findings generalize across species. Many authors have pointed out that demonstrations of sensitive periods are not as clear in primates as they are in birds and other species (Berardi et al., 2000), and that may be especially so for socialization outcomes (Ramey & Sackett, 2000).

Translating findings on early exposures from animal studies to the human context is complicated by specific challenges that likely vary according to risk paradigm. More specifically, there are several areas where there may be poor generalization across species, including the varying maturity of the offspring (e.g., brain size) at birth in species considered in studies of prenatal stress, such as rodents, sheep, nonhuman primates, and humans (Coe et al., 2003; Matthews, 2000); variation in the ease with which glucocorticoids (corticosterone/cortisol) cross the placenta (e.g., Challis, Matthews, Van Meir, & Ramirez, 1995); differential distribution and likely differential functional significance of receptors involved in regulating stress in rodents and primates (Sanchez, Ladd, & Plotsky, 2001); different functions and processes relevant for steroidogenesis (Malassine, Frendo, & Evain-Brion, 2003); and incompatibility in linking immune genes, such as between the mouse and the human (Seok et al., 2013). Compounding this problem is the explicit demonstration of nongeneralizability within species. A well-known example is the striking contrast between the monogamous prairie voles and nonmonogamous montane voles, for whom mating and caregiving behaviors differ dramatically (Young, Wang, & Insel, 1998). Research into within species behavioral differences was instrumental in documenting the role of oxytocin. The key point here is that such remarkable within-species differences in fundamental behaviors should caution against extrapolating behavior and perhaps also biology across species.

Finally, whereas experimental animal research relies on generally precisely timed manipulations to investigate early stress effects, no such control is plausible in human studies, and the comparability of severity of stress is equally uncertain when species and effects are compared or used as a guide. Efforts to develop stress paradigms that might have more application to humans, such as the variable forging demand paradigm (Coplan et al., 1998), may offer greater potential for translation. Another approach is to examine naturally occurring variation in behavior. One example is the work on maltreatment in nonhuman primates (Maestripieri, Tomaszycki, & Carroll, 1999).

The concept of translation has commanded significant attention because of uncertainty about how well research findings may generalize across species given inevitable biological differences, wide variation in research paradigms used with different species, and substantial difficulties in operationalizing compatible outcomes across species.

Some findings in the animal literature are certain to have no application to human health; other findings may have application but could be impossible to investigate in humans because of ethical or practical limitations. Inevitably, this means that conceptual models for early experience and human psychological development, and certainly those that shape prevention and treatment must be informed principally by human studies.

Summary

It is no longer seriously questioned if early experiences have particular and lasting effects on behavioral and biology in the animal. The challenge for human health is to document how well, and if, lessons from experimental animal studies generalize to human development. A presumption of some degree of translation from animal to human is typically assumed, and there are clearly some key biological processes that are conserved across species. On the other hand, as reviewed in this section, there are specific reasons why generalization may not occur. Furthermore, difficulties in developing research designs to differentiate alternative developmental hypotheses are matched by data analytic challenges. So, for example, data analytic strategies, however sophisticated, cannot overcome in-built confounds: demonstrating that there may be an independent effect of one particular risk or exposure period from another may not carry much practical application if the nature of the risks is to covary in development. Accordingly, it may not be too surprising that a range of ideas in the clinical and research settings persist about the role of early experience on psychological development in humans.

MODEL SYSTEMS AND PARADIGMS FOR STUDYING DEVELOPMENTAL MODELS AND MECHANISMS

A question continually framing discussions of developmental theory and developmental mechanisms is whether or not there are persisting effects of early (stress) exposures on behavioral disturbance and psychopathology. The early effects hypotheses are scientifically important and have potentially substantial practical application. Influential paradigms for understanding the relevance and distinctiveness of developmental models are reviewed in this section. Paradigms reviewed below were chosen because they have generated considerable experimental animal and preclinical and clinical human research, suggest important areas of convergence among studies of several varieties, demonstrate that there can be persisting effects of early experiences on psychological development, and offer general lessons for research into developmental mechanisms.

Prenatal Stress/Anxiety and Child Development

Research findings on prenatal maternal anxiety/stress are considered in detail because it has emerged from the experimental animal and human literatures as one of the leading examples of developmental programming. As such, it offers an extensive set of findings relevant for developmental theories of early experience; more recently, this research paradigm has begun to shape clinical decision-making. Importantly, because of its recognized scientific and public health significance, the maternal prenatal anxiety/stress paradigm is being pursued by many research groups in several countries.

The notion that the mother's mental state in pregnancy may influence the health of the baby has a rich cultural history. Scientific interest in this topic is also long-standing (Sontag, 1941). Experimental animal studies using the prenatal stress paradigm date back decades (Ader & Plaut, 1968; Hockman, 1961; Joffe, 1965; Keeley, 1962), and this paradigm continues to shape experimental animal research (Henry, Kabbaj, Simon, Le Moal, & Maccari, 1994; Matthews, Owen, Banjanin, & Andrews, 2002; Schneider et al., 1998; Wilson, Schade, & Terry, 2012). The current human model proposes that prenatal maternal anxiety or, in some studies, stress is associated with altered maternal physiology; most attention focuses on cortisol, a downstream product of the HPA axis, but many physiological candidates effects might be identified (Evans, Myers, & Monk, 2008; Kivlighan, DiPietro, Costigan, & Laudenslager, 2008; O'Connor, Tang, et al., 2014; Sarkar, Bergman, Fisk, O'Connor, & Glover, 2007b). Furthermore, this altered physiology is communicated to the fetus to shape fetal brain development directly by maternal transfer or indirectly by altering placental function (Monk et al., 2011; O'Donnell et al., 2012; Sarkar, Bergman, Fisk, O'Connor, & Glover, 2007a). Consistent with the developmental programming model there is an additional premise that increased exposure to cortisol by the fetus may program biological systems with effects observable on physiology, metabolism, immune function, brain and behavior. The intensity of findings in this area suggests that maternal mental health in pregnancy may be, like diet and health behaviors, a factor that requires routine attention in prenatal care.

Many reviews of the empirical evidence supporting links between prenatal anxiety of stress and child outcomes are available (Glover, O'Connor, & O'Donnell, 2010; Huizink, Mulder, & Buitelaar, 2004; Sandman, Davis, Buss, & Glynn, 2011b; Talge, Neal, & Glover, 2007), and new evidence suggesting prediction to behavioral and emotional symptoms, stress physiology, neurodevelopment, and many other outcomes appears regularly from many different studies and sites (Buss et al., 2012; King et al., 2009; Laplante, Brunet, Schmitz, Ciampi, & King, 2008; O'Donnell et al., 2014; Robinson et al., 2011; Sandman, Davis, Buss, & Glynn, 2011a). The breadth of effects in human research is quite striking, and also includes some of the developmental mechanisms cited above, including immune competence and altered cytokines expression (Entringer et al., 2008; O'Connor, Winter, et al., 2013). Particularly impressive are studies suggesting that maternal prenatal anxiety, depression, or stress may extend to more severe mental illness and neurodevelopmental outcomes such as schizophrenia (Khashan et al., 2008; King et al., 2009; Kinney, Miller, Crowley, Huang, & Gerber, 2008). That is in addition to several obstetric and birth outcomes and complications, such as preterm delivery (Hedegaard, Henriksen, Sabroe, & Secher, 1993), which may also contribute to long-term psychological development.

Investigation into the possible mechanisms accounting for the apparent widespread programming effects on psychological and biological development from prenatal maternal anxiety/stress emphasizes stress physiology, and the HPA axis in particular. Much the strongest evidence derives from animal studies because of the substantially greater leverage to test mechanisms that is available. So, for example, nonhuman primate research has shown that injection of ACTH, a precursor of cortisol, in nonstressed pregnant animals has comparable effects on offspring outcomes as uncontrollable environmental stress (Schneider, Coe, & Lubach, 1992).

What particularly distinguishes the findings in this area is not so much the extent of replication across sites or the range of biobehavioral outcomes so far predicted from prenatal maternal anxiety—although that is impressive—but rather the persistence of effects long after removal from exposure to risk (i.e., prenatal anxiety/stress). Data from the Avon Longitudinal Study of Parents and Children (ALSPAC) provide some of the strongest evidence that prenatal anxiety may have bona fide programming effects on behavioral and emotional problems in children and adolescents (O'Connor, Heron, Golding, & Glover, 2003; O'Donnell et al., 2014). Particularly striking was that growth curve analyses indicated no growth or change in the magnitude of prediction across a decade of developmental data, offering a formidable operationalization of a programmed effect. Also, follow-up to age 15 years indicated that prenatal maternal anxiety continued to influence diurnal HPA axis function, leading to flatter slope that has been found in other samples of chronic risk-exposed individuals (O'Donnell et al., 2013); the effect size in this case was very small, however, so the degree of persisting or programming on the HPA axis must be seen as limited and unable to account for observed effects on psychological symptoms.

Findings from other cohort studies provide additionally important results. Data from the Danish Civil Registration System indicated that the death of the father during the pregnancy was associated with a notable 6.5 point reduction in standard IQ points compared with children whose mothers did not experience the death of a spouse/partner (Virk, Obel, Li, & Olsen, 2014). That study has helped to expand the range of outcomes and the developmental mechanisms of interest, given that bereavement also predicted type-1diabetes (Virk et al., 2010) and cranial-neural-crest malformation (Hansen, Lou, & Olsen, 2000). Epidemiological and large-scale cohort studies are beginning to have particular influence in this area of study because they reliably document small to modest links between prenatal maternal stress and many disease states and symptoms in the child long after birth (Tegethoff, Greene, Olsen, Schaffner, & Meinlschmidt, 2011).

Attempts to establish a causal effect in human studies is naturally limited by the constraints of observational or naturalistic study designs that compose virtually all of the reports so far, but a few instances in this area have capitalized on natural experiments. One potentially powerful example was an in vitro fertilization (IVF) study, which was able to dismiss passive genetic transmission as a confound. The findings indicated that prenatal maternal stress significantly predicted conduct problems but not attention or emotional problems in their genetically unrelated children. The design is a potentially valuable one (Rice, Jones, & Thapar, 2007), although findings from women undergoing assisted reproduction may not generalize to other populations, and the measures of stress were from a single item using retrospective report. An important but less compelling sort of natural experiment in the literature contrasts prenatal maternal with prenatal paternal anxiety. Evidence of an in utero programming effect is suggested because the effects on child behavioral and emotional symptoms were far stronger and more robust for maternal prenatal anxiety than for paternal prenatal anxiety (O'Donnell et al., 2014).

A different kind of natural experiment capitalized on a natural disaster that occurred in Quebec. Project Ice Storm has followed mothers and their children and categorized women according to their state of pregnancy at the time of the disaster (King & Laplante, 2005). The study has the advantage that the stress—a dramatic ice storm with widespread and catastrophic effects in the region—was a random occurrence and not obviously confounded with maternal characteristics (although timing, that is, in early pregnancy, is still confounded with duration). Findings from that program support other research in linking stress exposure in pregnancy with behavioral and neurodevelopmental outcomes in the child (King et al., 2009; Laplante et al., 2008). Other research on natural experiments and prenatal anxiety/stress concerning earthquakes (Glynn, Wadhwa, Dunkel-Schetter, Chicz-Demet, & Sandman, 2001) and the events of September 11 (Yehuda et al., 2005) attest to the influence that the prenatal maternal anxiety/stress paradigm is gaining in developmental studies of risk mechanisms. Indeed, there are many very powerful examples of natural experiments that have ruled in the prenatal exposure period as having lasting effects on psychiatric, metabolic, and neurodevelopmental outcomes (Hines, 2003; Stein, Pierik, Verrips, Susser, & Lumey, 2009; Thurner et al., 2013).

Results from several studies imply that there may be important individual differences in the programming effects associated with prenatal maternal anxiety. If replicated, these results would mark an important shift, from a process that describes a general mechanism operating across species to a process that is better conceptualized as varying among individuals. In the case of the ALSPAC cohort, the moderation is from BDNF polymorphisms on internalizing symptoms (O'Donnell et al., in press); in the case of a smaller German cohort, the moderation derives from the 7-repeat allele of a variable number tandem repeat polymorphism in exon III of dopamine receptor DRD4 on conduct problems (Zohsel et al., 2014); in the Dutch Generation R study, a moderation of prenatal anxiety in predicting emotional problems in young children is associated with the serotonin transporter gene (5-HTTLPR) (Tiemeier et al., 2012). To date, the list of examples provided is too short to know if there are robust effects, and few studies consider a broad network of genes rather than a small number of candidates. Nonetheless, the important point is that genetics work in humans may have a sizable impact on the concept of developmental programming and programmability and may suggest that this mechanisms may be something on which individuals may differ from one another according to genetic makeup.

Maternal Immune Activation Paradigm

A second research paradigm, also involving the prenatal period, that has substantially contributed to developmental models of psychological outcomes is the maternal immune activation (MIA) model. Numerous findings on the MIA paradigm have been reported in animal and human studies. Results from this line of study have been important in demonstrating that an immune insult may have long-term neurodevelopmental effects; that immune mechanisms can play major roles in psychological development; and that prenatal immunization strategies offer a sizable public health approach to mental disorders.

Experimental animal studies provide ample evidence that prenatal (or early postnatal) immune activation is associated with brain volume and neurogenesis (Bilbo et al., 2005; Kohman & Rhodes, 2013; Monje, Toda, & Palmer, 2003); this cross-talk is an essential starting point for this avenue of study. Nonhuman primate studies confirmed that infecting pregnant rhesus monkeys with influenza reduced grey and white matter in the offspring in magnetic resonance imaging analyses (Short et al., 2010). Evidence of the MIA hypothesis is not reliant on animal studies, however. A long program of study of Brown, Susser, and colleagues links neurodevelopmental disorders such as schizophrenia to influenza infection during pregnancy; some of these results imply that MIA may account for a sizable percentage of schizophrenia cases (Brown et al., 2004; Brown et al., 2004; Brown, Susser, Lin, & Gorman, 1995).

The MIA model has also been applied to autism. A recent population-based Danish study of over 96,000 individuals indicated that the likelihood of a child being diagnosed with autism spectrum disorder in middle childhood and adolescence was associated with prolonged fever in the mother in pregnancy and with maternal prenatal influenza; effect sizes that have clinical and public health significance (Atladottir, Henriksen, Schendel, & Parner, 2012). Just as interesting was the additional prediction from antibiotics in pregnancy.

Although the MIA model is so far mostly limited to more severe neurodevelopmental outcomes and the precise mechanisms are not yet clear in human research, it is likely that, as neuroimmune mechanisms become more familiar in research on development and psychopathology, this paradigm will be applied to a broader array of developmental and clinical outcomes and yield valuable and practical results.

Early and Severe Caregiving Neglect and Deprivation

A third paradigm singled out for particular attention has the most detailed and complex history in developmental psychopathology, the effects of early caregiving adversity for later psychological adjustment. Research on this topic was central to some early theorizing about sensitive and perhaps even critical periods of caregiving experience for psychological health (Bowlby, 1950, 1951; Spitz, 1945). Experimental animal work of Harlow and colleagues was instrumental in developing some of the early principles for psychological development (Harlow & Suomi, 1970; Suomi, Harlow, & McKinney, 1972; Young, Suomi, Harlow, & McKinney, 1973). These have been taken on board and expanded upon in a series of studies of children who experienced early institutional care. The value of these studies may not be so much in terms of clinical application; given the unusual nature of institutional deprivation, findings from these studies may not generalize to other clinical contexts. Instead, the value of the many studies of children who experienced institutional rearing is what they have to say about the principles of early deprivation, sensitive periods, and the capacity to recover following very severe early care. Indeed, recent years have seen no fewer than three Society for Research in Child Development (SRCD) monographs devoted to studies of children who experienced institutional deprivation.

Probably the most notable set of findings in this area for many years derive from the Bucharest Early Intervention Project (BEIP), a long-term follow-up of an ambitious intervention to improve the caregiving arrangements of a randomly selected sample of children who had been reared in institutions. The study has been instrumental in providing detailed descriptions of biology and behavior that respond to changes in caregiving structures in young children, and in offering a model for improving caregiving arrangements for children without stable caregivers. One report from the study indicated that the intervention—moving children from the institutional setting to a foster care setting—was associated with significant improvements in cognitive outcomes, and that this was especially so for children under two years of age (Nelson et al., 2007). Documented intra-individual improvements in other areas of functioning, such as attachment relationships and social functioning, provide strong causal evidence for the role of caregiving manipulations and, to some extent, hint at potentially sensitive periods for these interventions to occur (Smyke, Zeanah, Fox, Nelson, & Guthrie, 2010).

The finding that early institutional care is associated with significant and lasting impact on several aspects of psychological development has been well described (Bruce, Tarullo, & Gunnar, 2009; Chisholm, 1998; Kreppner et al., 2010; Rutter, Kreppner, & O'Connor, 2001; Vorria et al., 2006; Zeanah, Smyke, Koga, & Carlson, 2005). Detailed analyses have sought to document duration of exposure to deprivation as a measure of developmental timing. Although the effects have varied slightly across studies, there is little doubt that, for some, the effects of early deprivation are strong and persistent (Zeanah et al., 2011). As Zeanah and colleagues note, whether or not a sensitive period model explains the diversity of results so far reported partly depends on how widely the sensitive period is defined. Thus, some studies and some outcomes may imply a sensitive period of less than six months, whereas others may imply a wider period of sensitivity up to two years. It may be unreasonable to expect considerable convergence on a construct as complex as a sensitive period, particularly where data are derived from very divergent research designs such as observational follow-up and randomized controlled trial. In any event, it is reasonably clear that a sensitive period is intrinsically limited because of the wide variation in outcomes that have been reported in all studies at practically all degrees of deprivation, from a few months to several years. That is, mean scores on a scale of symptoms or impairment or overall percentages of affected individuals may show a nonlinear/sensitive period or other type of stepped function pattern; nevertheless, a strict sensitive period model does not easily accommodate the diversity of adjustments among those who experienced brief or prolonged deprivation. Comparable concerns could be expressed in relation to a programming model that could be applied to these data (Rutter & O'Connor, 2004). The individual variation in outcome is every bit as important as the evidence suggesting that prolonged early deprivation can have lasting effects on biobehavioral outcomes. On the other hand, the findings do not fit into a life-course model, given that evidence of persisting and significant impairment has been consistently described in a sizable subset of children long after removal from the depriving environment and in the apparent absence of very significant stress exposure in the adoptive environment.

One limitation in most studies that have followed children who experienced institutional rearing is that the children were placed at birth. As a result it is not possible to know if the effects of institutional care are found only when deprivation is experienced in the early weeks or months of life. In this context, two studies of children who experienced institutional care postinfancy require particular attention (Vorria, Rutter, Pickles, Wolkind, & Hobsbaum, 1998; Wolkind, 1974). For example, some years ago Wolkind

(1974) reported that disinhibition toward strangers, a key behavioral marker of children who experienced institutional care, was associated with early institutional care but not institutional care that was initiated post infancy. That sort of finding and this sort of research design is especially valuable for testing timing effects, a theme that will re-emerge in the following section on intervention.

As noted, the corpus of data on children who experienced early institutional care do not easily conform to a sensitive period, programming, or life-course model. Research into the underlying mechanisms underlying these associations might help to resolve some of this uncertainty. Several candidate mechanisms have been investigated. Stress physiology has attracted considerable attention, and there is some evidence (Gunnar & Vazquez, 2001) that children who experienced prolonged deprivation show the kind of dampened response that other high stress populations display. The involvement of other candidates, such as the neuropeptide oxytocin, may be seen as a natural target (Wismer Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005), although its links with the HPA axis and other biological processes means that it would not operate in isolation. Similarly, involvement of dopaminergic pathways might be relevant here, to explain not only disturbances in attachment behavior but also problems in attention that frequently get reported in children who experienced deprivation. And, a separate line of investigation on a genetic knock-out mice model demonstrated that animals lacking the μ-opioid receptors exhibited disturbances in social behaviors (Moles, Kieffer, & D'Amato, 2004), offering a further biological focus for further study.

Imaging studies also offer some clues about brain regions and functions that may be driving the observed behavioral effects associated with institutional deprivation. One of the earliest reports was from Chugani and colleagues (2001) who described reduced glucose metabolism in children adopted into the United States from institutions in Romania compared with adult and child epileptic comparisons. Affected brain regions included the orbital frontal cortex, prefrontal cortex, and medial temporal structures that include the amygdala and hippocampus. In one of the larger imaging studies of children who experienced institutional rearing, Tottenham and colleagues (2010) found an association between prolonged deprivation and larger amygdala volume, an effect that might accommodate some of the behavioral and symptom pattern expression found in this population. Imaging data from the BEIP suggested that institutional rearing was associated with smaller cortical gray matter volume compared with children who did not experience institutionalization (Sheridan, Fox, Zeanah, McLaughlin, & Nelson, 2012). Furthermore, other brain-based assessment techniques, such as electroencephalogram (EEG; McLaughlin et al., 2010), have similarly identified deficits that may be linked with duration of deprivation.

Collectively, the findings so far reported suggested a predictably wide range of biological mechanisms that might help to locate an effect of institutional deprivation underlying the range of behavioral and health effects. It seems implausible that the effects from deprivation would not have wide-ranging biological effects, so it remains for further study to develop integrative models to synthesize complementary and overlapping biological effects, and to track intra-individual biological changes in response to behavioral changes (Vanderwert, Marshall, Nelson, Zeanah, & Fox, 2010).

Studies of children who experienced institutional rearing and were then placed into normal risk adoptive homes provide a natural experiment to examine the effects of early caregiving deprivation because exposure to deprivation was relatively precisely timed and restricted to the first months or years of life; completing these natural experiments are more formal intervention experiments, such as the BEIP. These studies have had a major impact in pushing forward the notion that, in some circumstances and for some children, there are persisting effects of early severe deprivation. The challenge—which is not yet resolved—is to decide if the lessons from these extraordinary studies can generalize to other settings and samples sufficiently well to inform theories of normal and (less severely) abnormal development, and clinical application.

Summary

Available data from human studies of prenatal maternal anxiety/stress, maternal immune activation, and postnatal deprivation are consistent with some of the key findings from animal studies and offer some of the strongest human evidence that there can be lasting effects of early exposures on psychological development. Results from studies in these areas are less clear about the kinds of mechanisms that may carry these effects. How these findings and uncertainties may be applied to intervention and prevention is the focus of the final section of the chapter.

DEVELOPMENTAL MODELS ARE SHAPING CLINICAL AND PUBLIC HEALTH STRATEGIES

The final section provides applications for research on early experience, and considers how prevention and intervention

studies have informed core conceptual issues. The major themes are (1) many intervention studies starting from early childhood have demonstrated long-term effects on psychological development, although very few have been specifically designed to contrast differing views about developmental timing; (2) prevention and intervention studies, as well as natural experiments, have importantly contributed to the early experiences debate; (3) intervention and prevention studies are increasingly targeted as mediators and outcomes those biological mechanisms that may have particular significance in understanding early stress exposure; and (4) integration of the early experience concepts and findings and its neuroscience basis are beginning to be applied to public and policy debate and treatment rationales, usually with mixed success.

Early Interventions Can Have Lasting Effects

A starting point for this section is that there are several intervention programs that have been administered to young children that show lasting effects on psychological development. School-based interventions are among the best known and widely researched (Barnett, 1995; Yoshikawa, 1995). The Abecedarian project is a longitudinal study of the effectiveness of a school-based intervention with high-risk children randomly assigned to treatment or control conditions (Campbell et al., 2012; Campbell, Pungello, Miller-Johnson, Burchinal, & Ramey, 2001; Campbell & Ramey, 1994; Ramey & Campbell, 1984). One group of children were given the intervention from infancy through to 3 years of schooling; a second group received the preschool treatment only; a third group received only the school-based treatment; the third group constituted the control condition, that is, the comparison/control condition. Analyses of the age 12 data (Campbell & Ramey, 1994) indicated that the children who received the continuous intervention from infancy attained the highest scores on intellectual and achievement tests; children who received only the preschool intervention performed lower, and lower still were children who received the intervention only from school age. Although, on average, children in each intervention group performed better than the control children, their scores were still lower than children from a nonrisk comparison sample. Extending the follow-up period to age 21 years, the investigators found that children in the early intervention group continued to have long-term benefits on cognitive and academic measures. Analyses of the cognitive and achievement data are instrumental in showing that early interventions can have lasting effects, long after the exposure period (i.e., the intervention) was ended.

One of the recent trends in intervention and prevention work is a focus on outcomes extending beyond cognitive, social, and emotional development; that model has been adopted in the Abecedarian project (Campbell et al., 2014). Analyses of select biological data in adulthood, when this component of the study started, indicated that males who received the cognitive and social stimulation intervention in early childhood had, in their mid-30s, fewer signs of prehypertention based on systolic and diastolic blood pressure readings; they were also less likely to show multiple health risk factors. No significant effects were found for females, however. Importantly, the treatment effect differences for males were not explained by the better health care experienced by those who had received the early treatment (e.g., greater health insurance coverage). The follow-up study was of a comparatively small sample and the range of physical health outcomes was limited, but the report is important because it offers one of the few experimental studies assessing early psychological environment and later somatic health, which was not targeted in the intervention. The experimental leverage provided by the intervention trial is far greater than the large number of naturalistic long-term follow-up studies showing associations between psychological risk and exposures with adult health outcomes.

A second very well-known example of an early school-oriented intervention having lasting effects is the Chicago Child-Parent Center (CPC), which provided a comprehensive educational, family and health service intervention to children living in dense areas of poverty (Reynolds, Temple, Robertson, & Mann, 2001). The CPC contrasted an index group of 989 children who received preschool and school-age services with a comparison group of 550 children who received alternative early intervention programs. Effects 15 years after intervention delivery were retained and impressive. Specifically, children in the preschool program had higher rates of high-school completion by age 20 years than comparison children (49.7% versus 38.5%); were less likely to drop out than comparison children (46.7% versus 55%); and were less likely to have been arrested by age 18 years (16.9% versus 25.1%). Additionally, whereas 14.4% of the index children required special education placement, the rate in the comparison group was 24.6%.

A further valuable feature of the study is that not all children received the intervention from preschool through school age. Accordingly, as with the Abecedarian project, it is possible to contrast the outcomes of children who received only early intervention (through preschool) with those children who received a more protracted intervention

(into third grade). Findings showed that school-age participation did not predict additional gain over the preschool intervention for educational attainment or delinquency outcomes, but was associated with additional benefit on the measure of use of remedial services: participation in the school program (for at least one additional year) predicted modestly lower rates of special education, grade retention, and years of special education services compared with children whose receipt of the intervention stopped after preschool (Reynolds et al., 2001). Collectively, the CPC and the Abecedarian project offer some of the strongest evidence for a persisting effect of an early school-based project on cognitive, social, and perhaps even somatic health outcomes and have rightly played a major role in advocating for more widespread use of intensive school-based interventions for high-need children.

The nurse home visiting program (Eckenrode et al., 2000; Kitzman et al., 1997, 2000, 2010; Olds, Henderson, & Kitzman, 1994) is a very different kind of early intervention that has also attracted considerable attention for what it may say about the persisting effects of a fairly mild manipulation to the child's early environment on broadband effects indexed by achievement, behavioral outcomes, and health. Academic achievement, behavioral and emotional problems including more severe disturbances, and domestic violence and other forms of risk exposure are just some of the areas in which the intervention has had replicated and significant effects; to a considerable extent, many of the outcomes associated with intervention were not formally targeted in the intervention. As a result of these persuasive findings, the nurse home visiting program has been extended to many settings.

Other kinds of early interventions have been aggressively pursued in studies of child psychological development; parenting interventions among the most commonly assessed. Whether or not early parenting interventions have long-standing effects on child psychological development is perhaps less clear. That is partly because of the limited number of long-term follow-up studies and the typically modest (one–three-year) follow-up period, although there are exceptions. The Incredible Years' intervention program, which generally targets preschool or early school-age children and seeks to promote parental support and decrease harsh parenting practices, has likely attracted the most widespread interest because it has wide dissemination across many countries and settings (Jones, Daley, Hutchings, Bywater, & Eames, 2008; Webster-Stratton, Rinaldi, & Jamila, 2011). One recently reported follow-up study is of interest because it assessed both an indicated clinic-attending sample and a selective high-risk community sample, and because the follow-up was comparatively long, at approximately 6–7 years (Scott, Briskman, & O'Connor, 2014). Findings indicated long-term retained gains in the treated children relative to the comparison children in behavioral problems, psychopathy, and reading outcomes as well as some measures of parenting in the indicated sample into adolescence. Nonetheless, the overlap levels of disturbance in this clinical sample still exceeded those found in the community sample. In contrast, gains initially observed in the selective group from the parenting intervention were entirely absent at the long-term follow-up assessment. The finding that the intervention effects were most persistent among the more behavioral disturbed children is consistent with other studies, including the large-scale fast track program (CPPRG, 2011). The lesson here is that somewhat early and targeted interventions can alter the longer-term developmental course of high-risk children, but there are likely strong determinants of individual variation in treatment response. It is too soon to know if parenting interventions research can inform questions about early exposures and developmental timing, however, because timing is not itself a core feature of the research design in these studies.

Other changes in caregiving arrangements have been linked with positive outcomes for young children; effects may be found even where the overall environment is extremely poor and the change in arrangement would appear more structural than psychological—at least in terms of the childcare arrangement and intervention focus. For example, Zeanah and colleagues (Smyke, Dumitrescu, & Zeanah, 2002; Zeanah, Smyke, & Dumitrescu, 2002) found that improving the child:staff ratio and increasing staff availability and consistency predicted decreases in severely disturbed attachment behavior, a common and significant disturbance in children reared in institutions.

Even more radical caregiving arrangement interventions have been investigated and may inform discussions of early experience and developmental timing. The most obvious are those of children who were adopted into normal risk families following early institutional deprivation; some of these results were reviewed previously. The BEIP was a formal intervention and moved some children from institutional to foster family care, and assessed the effects. More broadly, though, all of the studies that followed children who were removed from institutional placement provide a powerful assessment of early intervention—although not based on formal randomization. Indeed, there are few interventions as radical as changing the caregiving environment. The beneficial effects of this radical form of intervention were reviewed previously; virtually all studies

identify substantial developmental catch-up and clinical improvement with home placement, and that this occurs at least to some degree for all children. As noted, the implication is that adoption even at a later age (preschool in most of these cases) had salutary effects on child health and behavior. Given these findings, establishing a distinct sensitive period hypothesis for developmental catch-up effects associated with removal from institution and placement in an adoptive family has been difficult, as it may vary across study, age at assessment, and domain of interest; in addition, there are sizable individual differences that confound these efforts (Nelson et al., 2007; Zeanah et al., 2011).

One study that capitalized on a change in placement from a maltreating family to foster placement is noteworthy for suggesting that a sensitive period in the formation of new attachment relationships may extend well into middle childhood or later. Joseph and colleagues (Joseph, O'Connor, Briskman, Maughan, & Scott, 2014) found that nearly half of children who were placed with a foster carer in late childhood exhibited a secure attachment based on a semi-structured attachment interview when the children were early adolescents; evidence of observed parent–adolescent quality and its association with attachment security helped substantiate this finding. This rate of secure attachment is potentially remarkable given that the adolescents had histories of early maltreatment so severe as to require removal from the home, and that their discussions of their biological parents during the attachment interview indicated marked insecurity. Another study investigated late-placed caregiving changes and also found notable gains in children's psychological development, in this case for IQ. Duyme, Dumaret, and Tomkiewicz (1999) found that children with history of neglect and maltreatment and low IQ who were adopted between 4 and 6 years of age showed sizable gains in IQ by early adolescence; gains of eight points in the low and 20 points in the high socioeconomic status (SES) demonstrate that improvements can be particularly large and dependent on the new caregiving situation.

The Joseph et al. and the Duyme et al. (1999) studies are unusual in assessing late placement changes and, more significantly, in demonstrating that sensitive periods for attachment and IQ extend well beyond the period implied by findings from children reared in institutions. It may be that early institutional rearing may limit the extent and duration and degree that developmental catch-up is possible; it may also be that findings implying a sensitive period window from severe deprivation may not extend to less severe forms of stress and trauma. And, it may be that windows of sensitivity or programming may be too particular to exposure type, sample, and outcome to derive general lessons.

An interesting example of how the effects of early school- or cognitive-based intervention may be moderated health status factors was recently reported by Tofail and colleagues (2013). They found that a fairly standard psychosocial stimulation program in Bangladesh to children aged 6–24 months was associated with gains in the Bayley Scales of Infant Development approximately 9 months later; moreover, there was a suggestion—a statistical trend—that the gains were weaker among those children who were iron deficient. That is notable, given that iron (and other biological context variables) are rarely accounted for in studies of psychological interventions and outcomes. The lesson from this study, and an emerging theme throughout this chapter, is that studies of early experience will be limited and may misspecify effects if they adopt a narrow, unidisciplinary conceptual and biological framework. Tofail and colleagues identified one health risk that might alter intervention effectiveness in young children. Incorporating more than a single targeted factor does impose demands and costs and complexities on the research design, which may exceed practical limits. And, it is significant that the iron deficient/anemia condition which had a modest moderating effect on the stimulation intervention in the Tofail et al. study was engineered as part of the research design. That is, it was likely necessary to design iron deficiency into the sampling framework to detect its effect on treatment outcomes. That contrasts with the more typical situation in which biological measures of risk, such as genetics, diet, or physiology, are collected post hoc rather than as part of the sampling frame, which may result in reduced power because of insufficient numbers or ranges of biological risk.

Factors external to the treatment or clinical condition may also moderate treatment effects. Initial severity of disturbance is frequently considered as a moderator of treatment. An example in the early intervention literature was reported as part of an eight-year follow-up of a large sample of children who received an early intervention to compensate for low birth weight. There were no lasting overall effects, but where effects were detected they were limited to the heavier low birth weight premature children (McCarton et al., 1997). It is significant that there are sizable individual differences in treatment effects even when the treatment was delivered so early in development—as early as delivery.

A small but important number of studies in the early effects literature have begun to target specific biological mechanisms alongside key behavioral outcomes. Predictably, the list of biological targets overlaps with the

candidate mechanisms of risk reviewed in the second section of the chapter. Although it is not yet clear that there are robust findings, several sets of results stand out at this early stage. One line of study seeks to examine if disrupted HPA axis function that may result from early adverse care responds to psychological treatment and may mediate treatment effects on behavior. In one instance, Dozier and colleagues (Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008) found that children in an early intervention group exhibited lower initial values at a lab visit than children in the treatment control group. In a separate study, researchers found that children involved in child protective services but remained at home showed flatter cortisol patterns—similar to what has been reported in chronically stressed samples—compared with children who were placed into a foster care setting (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010). Other reports targeting HPA axis and other mechanism that may be especially sensitive to early caregiving adversity are available (Fisher, Gunnar, Dozier, Bruce, & Pears, 2006). It would be premature to make firm conclusions at this stage of the research, and it may even be premature to distinguish the specific optimal physiological outcome that would be targeted in a successful treatment for some of these biological candidates. Further studies with a range of samples and conditions are needed, and this will likely constitute a major topic of further study.

Results from even the small number of selected studies of early interventions reviewed above are challenging to summarize for what they may imply about the role of early experience on psychological development and what they can say about developmental timing. The broadest conclusion is that early interventions can have persisting effects on multiple areas of psychological function, and long after the intervention exposure ceased. Some additional specifics are worth noting, including the finding that at least some of the school-based interventions did not start especially early (although it is not always clear what is meant by early); other interventions with dramatic effects were instituted in middle childhood. Clearly, interventions need not start in early infancy if they are to have lasting effects on the trajectories of psychological development into adulthood, but it is not yet clear how important age (as a short-hand proxy for many aspects of development) is for deciding when the intervention needs to be administered. However, to say that early interventions can have lasting effects is not to imply that it is expectable that all well-constructed interventions would have salutary impact (unsuccessful early interventions are not reviewed at length in this chapter). What the findings reviewed above do imply is that many factors determine the long-term effectiveness of an intervention program, from nutritional factors, to initial severity, to a host of largely untapped sources of individual differences such as genetics. Movement toward research strategies to link interventions to specific biological mechanisms that may carry presumed beneficial effects of the intervention are under way and should provide some clarity for shaping future intervention strategies.

Developmental Hypotheses and Early Intervention

Several authors (Bruer, 1999; Clarke & Clarke, 2000) have summarized some of the main features of the early intervention literature and some of its limitations, and others have put forward a case for greater emphasis on early intervention (Kirp, 2007). Bruer's book of some years ago generated considerable controversy for the intervention field; some of his concerns remain, including an uncertain connection between claims about the critical need for early intervention and the neuroscience that might substantiate that claim. It is fair to say that research designs in the early intervention literature are not, by and large, designed to tackle specific questions of developmental timing. That is, children have not typically been assigned to an intervention according to age with, say, some children getting 12 months of intervention in infancy and others getting 12 months of intervention in middle childhood. As a result, evidence of specific timing effects—whether that is early infancy or any period—is limited.

On the other hand, there are, to be sure, interesting research examples implying timing effects that capitalize on natural experiments. Some of these have very direct practical application. The age at which children enter kindergarten, because of arbitrary age or calendar cutoff or parental choice, is one such example. Data from the NICHD (2007) day care study indicated that children who entered kindergarten at younger ages scored higher on word recognition but lower on other scales, including mathematics, than children who entered later; by follow-up in third grade, children who entered kindergarten (comparatively) later performed better than earlier-entering children on applied problems and picture vocabulary. Although this is a popular kind of natural experiment to assess timing effects, the role of parental choice and planning—and how it has changed over time—mean that the experimental leverage to assess timing effects in this type of study has substantially diminished.

Intervention timing may be examined in other contexts. A further very practical clinical example is treatment for psychological disorders. Interventions using

cognitive-behavioral therapy are especially interesting in this regard because it may be that earlier is not always better if the treatment requires some degree of matured cognitive sophistication. There is, in fact, some evidence that older children respond more positively to CBT, possibly as result of differences in cognitive ability and executive functioning that are evident later in development and mediate treatment response but that has not yet been formally demonstrated (Durlak, Fuhrman, & Lampman, 1991). Another practical application for intervention concerns the question of when preventive interventions for substance abuse might be most effective in engaging and reducing use, either in terms of age or in terms of drug use experimentation (Dishion, Kavanagh, Schneiger, Nelson, & Kaufman, 2002). A still further example for developmental timing studies focuses on the course of the disorder. In this regard, Post and colleagues (Post, 1992; Post, Leverich, Xing, & Weiss, 2001; Weiss & Post, 1994) suggested that first episodes of depressive illness may be linked with psychosocial factors but that subsequent episodes may be more autonomous from psychosocial risk, perhaps because of the neurobiological changes brought on by the illness. In this case, the early treatment imperative links to stage of illness rather than to age. As these examples document, the concept of timing as it relates to intervention may take many forms, and is not always equated with age or maturation. And, as previously noted, there is considerable interest in adolescence as a period during which stress exposures and interventions may have particular effect, perhaps because there is a reopening of sensitive periods.

An application of the developmental timing question is to ask if there is an age after which the intervention would not be effective, or would be substantially less so (i.e., compared with earlier exposures). More entrenched developmental problems will pose additional and significant challenge for interventions delivered late in development and thus may require more intensive effort. In other words, age of the child may predict the intensity required for an intervention to work rather than the ultimate likelihood of success as such (Ramey & Ramey, 1998) because of a bona fide timing effect than can be traced to individual psychology or biology. Clearly, plasticity is a key concept underlying these questions and debates, but it has proven difficult to operationalize in empirical research.

Intervention studies have, for some time, focused on the question, For whom are interventions effective? Another question now also needs to be posed: When in development is the intervention likely to have the strongest effect (and why)? The form of developmental translational research requiring additional attention is what links brain, biology, and behavior in intervention designs that could be positioned to address questions of developmental timing. The needs of such an approach are clear enough: we need to understand not only that the intervention effects a behavioral change, but also what the underlying and sustaining mechanisms are, and if there is particular value in when it may be delivered. Early intervention and prevention studies may be especially well-suited to this sort of translational research, and candidates for biological investigation and the techniques to capture the relevant data are being improved and added to regularly. The conceptual case for multivariate, multilevel, biopsychosocial models of applied research is compelling and will necessitate creative scientific and financial approaches.

Clinical and Public Health Application

There is growing recognition that understanding the effects of early experience on later development and general questions of developmental timing have sizable clinical and public health significance. Identifying the age at kindergarten entry, deciding when and if to remove a child from an abusive caregiving setting, formulating an intervention for early symptom expression versus waiting for diagnostic threshold, and considering prenatal screening and intervention for significant maternal prenatal anxiety are all practical clinical issues that have a core developmental timing component.

One essential element to these discussions that infrequently appears in developmental studies is cost-effectiveness. Cost effectiveness estimates will depend on numerous assumptions, and may well vary across site, and will be limited by the duration of follow-up period. Nonetheless, such analyses have now been calculated for many programs, with generally positive and impressive effects for many model programs (Barnett & Masse, 2007; Olds et al., 2010). That is the sort of outcome that may require greater attention in developmental studies, so that clinical and public health impact—operationalized in monetary terms—are seen as part of the study design. Equally important, there is the matter of research approaches that attend to other areas equally neglected in developmental science, such as implementation science (Smits & Denis, 2014).

National organizations are active in emphasizing the impact and importance of early experiences for health psychological and somatic development, including the National Center for Infants, Toddlers, and Families, and others (e.g., National Scientific Council on the Developing Child, 2012; Sawhill, Thomas, & Monea, 2010), and many reviews and policy-oriented discussions are now available

(Kirp, 2007). Efforts to continue to inform policy makers about early experience and brain development, and the importance of (early) socioemotional development for general health have been informed by a wide range of studies, and the relevant empirical base is clearly growing steadily and quickly, as implied by research covered earlier in the chapter. How it is that scientific concerns of developmental timing and early experience seep into and shape policy debate is complex and hardly linear. As Kirp (2007) suggested, allowing for scientific study to shape so private an enterprise as caregiving, a common focus in studies of early exposure and early intervention, would present a major cultural paradigm shift that may be resisted. The strength of scientific research is only one factor influencing policy and cultural perceptions, and that is so even where the research evidence is undeniably watertight. (We are not yet there in many regards in this field of study.)

Difficult and practical questions will continue to arise and shape clinically oriented research into early exposures and developmental models of health and disease. So, for example, attempts to identify biological risk for later illness, in the absence of frank pathology, pose questions about when in the course of the illness intervention might be warranted, clinically effective, and socially acceptable. Recent findings for cognitive decline and dementia provide one good example (Mapstone et al., 2014), but research findings may soon nominate other illnesses and conditions for these debates.

Summary

A theme of this section is that intervention studies have and will continue to play a key role in differentiating alternative hypotheses concerning the impact of early experience on long-term adjustment. However, there are limits of existing designs, the role of developmental timing is not often formally tested, and the focus on mediating mechanisms is still somewhat limited. Developmental timing, as it relates to intervention and prevention, may take many forms and may be operationalized according to maturation, disease state, or other factors. Research along these lines would provide practical cues about when in development interventions may be most effectively delivered, and these observations could further efforts in monetizing, (de-)politicizing, and disseminating effective practice.

CONCLUSIONS AND FUTURE DIRECTIONS

Advances in research techniques make possible and reports of research findings make plausible multiple active programs of mechanistic studies to identify how and under what conditions exposures to both stress and protective elements early in development may have lasting effects on psychological development. To that end, this chapter provided illustrations of some intriguing findings from research paradigms that have been particularly influential in supporting the hypothesis that early exposures may have lasting effects on psychological and biological development. Furthermore, the chapter identified some of the more prevalent candidate mechanisms that might explain a carrying-forward of effects on longer-term health and development. Countering this scientific enthusiasm and optimism was a section detailing the inherent difficulties of gaining causal leverage to resolve questions about which conceptual models may offer the strongest fit to the available data.

Opportunities for future research were distributed throughout discussions in each section and can be summarized as follows. One is to make more explicit in the research plan both the developmental model being tested and its specific operationalization in measurement and analysis. In this regard, studies seeking to test for the effects of early exposures without accounting for recent and cumulative exposures (i.e., to the same or relevant sources of stress) are especially unhelpful but can nonetheless be found in published reports. Placing competing and plausibly tested developmental models and expectations in competition to account for obtained data may help to differentiate research models and point to further avenues of inquiry, and offer practical applications. In addition, as emphasized throughout the chapter, much more research is needed to document the limits of generalizing or translating findings from one context (e.g., species, sample, research design, scientific paradigm) to another. Human studies to test early experience hypotheses for clinical import clearly do have important scientific limitations but, as noted, influential models and programs have developed significantly, and can provide a basis for clinical research. Making relevant the neuroscience of early experience work will require greater use of plausible clinical phenotypes and mechanisms that are accessible in a clinic context; reciprocally, engaging clinical and public health settings may well be required for leveraging causal evidence. Finally, future research is needed that builds up from research in which a specific and (sometimes artificially) isolated—or just presumably isolated—candidate mechanism is considered and offered undue explanatory power. Many candidate genes, nutrients, brain regions, cytokines, hormones, and psychological processes have currency in research on early experience. It is now necessary to design into investigations their interplay in development.

REFERENCES

Ader, R. (1983). Developmental psychoneuroimmunology. *Developmental Psychobiology*, *16*(4), 251–267. doi: 10.1002/dev.420160402

Ader, R., & Cohen, N. (1975). Behaviorally conditioned immunosuppression. *Psychosomatic Medicine*, *37*(4), 333–340.

Ader, R., Kelly, K., Moynihan, J. A., Grota, L. J., & Cohen, N. (1993). Conditioned enhancement of antibody production using antigen as the unconditioned stimulus. *Brain, Behavior, and Immunity*, *7*(4), 334–343. doi: 10.1006/brbi.1993.1033

Ader, R., & Plaut, S. M. (1968). Effects of prenatal maternal handling and differential housing on offspring emotionality, plasma corticosterone levels, and susceptibility to gastric erosions. *Psychosomatic Medicine*, *30*(3), 277–286.

Ainsworth, M. D. S. (1989). Attachments beyond infancy. *American Psychologist*, *44*(4), 709–716.

Alisch, R. S., Barwick, B. G., Chopra, P., Myrick, L. K., Satten, G. A., Conneely, K. N., & Warren, S. T. (2012). Age-associated DNA methylation in pediatric populations. *Genome Research*, *22*(4), 623–632. doi: 10.1101/gr.125187.111

Allen, J. P., & Miga, E. M. (2010). Attachment in adolescence: A move to the level of emotion regulation. *Journal of Social and Personal Relations*, *27*(2), 181–190. doi: 10.1177/0265407509360898

Allen, J. P., Moore, C., Kuperminc, G., & Bell, K. (1998). Attachment and adolescent psychosocial functioning. *Child Development*, *69*(5), 1406–1419.

Almeida, N. D., Loucks, E. B., Kubzansky, L., Pruessner, J., Maselko, J., Meaney, M. J., & Buka, S. L. (2010). Quality of parental emotional care and calculated risk for coronary heart disease. *Psychosomatic Medicine*, *72*(2), 148–155. doi: 10.1097/PSY.0b013e3181c925cb

Amiel-Tison, C., Cabrol, D., Denver, R., Jarreau, P. H., Papiernik, E., & Piazza, P. V. (2004). Fetal adaptation to stress. Part I: acceleration of fetal maturation and earlier birth triggered by placental insufficiency in humans. *Early Human Development*, *78*(1), 15–27. doi: 10.1016/j.earlhumdev.2004.03.002

Anders, S., Tanaka, M., & Kinney, D. K. (2013). Depression as an evolutionary strategy for defense against infection. *Brain, Behavior, and Immunity*, *31*, 9–22. doi: 10.1016/j.bbi.2012.12.002

Andersen, S. L., Tomada, A., Vincow, E. S., Valente, E., Polcari, A., & Teicher, M. H. (2008). Preliminary evidence for sensitive periods in the effect of childhood sexual abuse on regional brain development. *Journal of Neuropsychiatry and Clinical Neuroscience*, *20*(3), 292–301. doi: 10.1176/appi.neuropsych.20.3.292

Ansorge, M. S., Zhou, M., Lira, A., Hen, R., & Gingrich, J. A. (2004). Early-life blockade of the 5-HT transporter alters emotional behavior in adult mice. *Science*, *306*(5697), 879–881. doi: 10.1126/science.1101678

Arend, R., Gove, F. L., & Sroufe, L. A. (1979). Continuity of individual adaptation from infancy to kindergarten: a predictive study of ego-resiliency and curiosity in preschoolers. *Child Development*, *50*(4), 950–959.

Ashwood, P., Krakowiak, P., Hertz-Picciotto, I., Hansen, R., Pessah, I. N., & Van de Water, J. (2011). Altered T cell responses in children with autism. *Brain, Behavior, and Immunity*, *25*(5), 840–849. doi: 10.1016/j.bbi.2010.09.002

Atladottir, H. O., Henriksen, T. B., Schendel, D. E., & Parner, E. T. (2012). Autism after infection, febrile episodes, and antibiotic use during pregnancy: an exploratory study. *Pediatrics*, *130*(6), e1447–1454. doi: 10.1542/peds.2012-1107

Bagner, D. M., Pettit, J. W., Lewinsohn, P. M., & Seeley, J. R. (2010). Effect of maternal depression on child behavior: a sensitive period? *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*(7), 699–707. doi: 10.1016/j.jaac.2010.03.012

Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2006). Gene–environment interaction of the dopamine D4 receptor (DRD4) and observed maternal insensitivity predicting externalizing behavior in preschoolers. *Developmental Psychobiology*, *48*(5), 406–409. doi: 10.1002/dev.20152

Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2007). Research Review: genetic vulnerability or differential susceptibility in child development: the case of attachment. *Journal of Child Psychology and Psychiatry*, *48*(12), 1160–1173. doi: 10.1111/j.1469-7610.2007.01801.x

Bale, T. L. (2011). Sex differences in prenatal epigenetic programming of stress pathways. *Stress*, *14*(4), 348–356. doi: 10.3109/10253890.2011.586447

Baltes, P. B., Staudinger, U. M., & Lindenberger, U. (1999). Lifespan psychology: theory and application to intellectual functioning. *Annual Review of Psychology*, *50*, 471–507. doi: 10.1146/annurev.psych.50.1.471

Bar-Haim, Y., Sutton, D. B., Fox, N. A., & Marvin, R. S. (2000). Stability and change of attachment at 14, 24, and 58 months of age: behavior, representation, and life events. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *41*(3), 381–388.

Barker, D. J. (1999). Fetal origins of cardiovascular disease. *Annals of Medicine, 31 Supplement 1*, 3–6.

Barker, D. J. (2007). The origins of the developmental origins theory. *Journal of Internal Medicine*, *261*(5), 412–417. doi: 10.1111/j.1365-2796.2007.01809.x

Barnett, W. S. (1995). Long-term effects of early childhood programs on cognitive and school outcomes. *Future of Children*, *5*, 25–50.

Barnett, W. S., & Masse, L. N. (2007). Comparative benefit–cost analysis of the Abecedarian program and its policy implications. *Economics and Education Review*, *26*, 113–125.

Barr, C. S., Newman, T. K., Lindell, S., Shannon, C., Champoux, M., Lesch, K. P., … Higley, J. D. (2004). Interaction between serotonin transporter gene variation and rearing condition in alcohol preference and consumption in female primates. *Archives of General Psychiatry*, *61*(11), 1146–1152. doi: 10.1001/archpsyc.61.11.1146

Barrett, J., & Fleming, A. S. (2011). Annual research review: All mothers are not created equal: neural and psychobiological perspectives on mothering and the importance of individual differences. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *52*(4), 368–397. doi: 10.1111/j.1469-7610.2010.02306.x

Barry, R. A., Kochanska, G., & Philibert, R. A. (2008). G x E interaction in the organization of attachment: mothers' responsiveness as a moderator of children's genotypes. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *49*(12), 1313–1320. doi: 10.1111/j.1469-7610.2008.01935.x

Bechtel, W. (2008). Mechanisms in cognitive psychology: What are the operations? *Philosophy of Science*, *75*, 983–994.

Beebe, B., Jaffe, J., Markese, S., Buck, K., Chen, H., Cohen, P., … Feldstein, S. (2010). The origins of 12-month attachment: a microanalysis of 4-month mother infant interaction. *Attach Human Development*, *12*(1–2), 3–141. doi: 919621246 [pii]10.1080/14616730903338985

Belsky, J., & Beaver, K. M. (2011). Cumulative-genetic plasticity, parenting and adolescent self-regulation. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *52*(5), 619–626. doi: 10.1111/j.1469-7610.2010.02327.x

Berardi, N., Pizzorusso, T., & Maffei, L. (2000). Critical periods during sensory development. *Current Opinion in Neurobiology*, *10*(1), 138–145.

Bergman, K., Sarkar, P., Glover, V., & O'Connor, T. G. (2010). Maternal prenatal cortisol and infant cognitive development: Moderation by infant-mother attachment. *Biological Psychiatry*, *67*(11), 1026–1032. doi: 10.1016/j.biopsych.2010.01.002

Bergman, Y., & Cedar, H. (2013). DNA methylation dynamics in health and disease. *Nature Structural & Molecular Biology*, *20*(10), 1236. doi: 10.1038/nsmb1013-1236a

Bernard, K., Butzin-Dozier, Z., Rittenhouse, J., & Dozier, M. (2010). Cortisol production patterns in young children living with birth parents vs children placed in foster care following involvement of Child Protective Services. *Archives of Pediatric and Adolescent Medicine*, *164*(5), 438–443. doi: 10.1001/archpediatrics.2010.54

Bilbo, S. D. (2013). Frank A. Beach award: programming of neuroendocrine function by early-life experience: A critical role for the immune system. *Hormones and Behavior*, *63*(5), 684–691. doi: 10.1016/j.yhbeh.2013.02.017

Bilbo, S. D., Levkoff, L. H., Mahoney, J. H., Watkins, L. R., Rudy, J. W., & Maier, S. F. (2005). Neonatal infection induces memory impairments following an immune challenge in adulthood. *Behavioral Neuroscience*, *119*(1), 293–301. doi: 10.1037/0735-7044.119.1.293

Birmaher, B., Rabin, B. S., Garcia, M. R., Jain, U., Whiteside, T. L., Williamson, D. E., … Ryan, N. D. (1994). Cellular immunity in depressed, conduct disorder, and normal adolescents: Role of adverse life events. *Journal of the American Academy of Child Adolescent Psychiatry*, *33*(5), 671–678. doi: 10.1097/00004583-199406000-00008

Bolger, K. E., & Patterson, C. J. (2001). Developmental pathways from child maltreatment to peer rejection. *Child Development*, *72*(2), 549–568.

Boris, N. W., & Zeanah, C. H. (2005). Practice parameter for the assessment and treatment of children and adolescents with reactive attachment disorder of infancy and early childhood. *Journal of the American Academy of Child Adolescent Psychiatry*, *44*(11), 1206–1219. doi: 10.1097/01.chi.0000177056.41655.ce

Bornstein, M. H. (1989). Sensitive periods in development: Structural characteristics and causal interpretations. *Psychological Bulletin*, *105*(2), 179–197.

Bowlby, J. (1950). Research into the origins of delinquent behaviour. *Br Med J*, *1*(4653), 570–573.

Bowlby, J. (1951). Maternal care and mental health. *Bull World Health Organ*, *3*(3), 355–533.

Bowlby, J. (1982). *Attachment and loss, volume 1: Attachment (2nd ed)*. New York, NY: Basic Books.

Bowlby, J. (1988). Developmental psychiatry comes of age. *American Journal of Psychiatry*, *145*(1), 1–10.

Brennan, P. A., Pargas, R., Walker, E. F., Green, P., Newport, D. J., & Stowe, Z. (2008). Maternal depression and infant cortisol: influences of timing, comorbidity and treatment. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *49*(10), 1099–1107. doi: 10.1111/j.1469-7610.2008.01914.x

Bretherton, I. (2005). In pursuit of the internal working model construct and its relevance to attachment relationships. In K. G. K. E. Grossmann, & E. Waters (Ed.), *Attachment from infancy to adulthood: The major longitudinal studies* (pp. 13–47). New York, NY: Guilford Press.

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design*. Cambridge, MA: Harvard University Press.

Brown, A. S., Begg, M. D., Gravenstein, S., Schaefer, C. A., Wyatt, R. J., Bresnahan, M., … Susser, E. S. (2004). Serologic evidence of prenatal influenza in the etiology of schizophrenia. *Archives of General Psychiatry*, *61*(8), 774–780. doi: 10.1001/archpsyc.61.8.774

Brown, A. S., Hooton, J., Schaefer, C. A., Zhang, H., Petkova, E., Babulas, V., … Susser, E. S. (2004). Elevated maternal interleukin-8 levels and risk of schizophrenia in adult offspring. *The American journal of psychiatry*, *161*(5), 889–895.

Brown, A. S., & Susser, E. S. (2008). Prenatal nutritional deficiency and risk of adult schizophrenia. *Schizophrenia Bulletin*, *34*(6), 1054–1063. doi: 10.1093/schbul/sbn096

Brown, A. S., Susser, E. S., Lin, S. P., & Gorman, J. M. (1995). Affective disorders in Holland after prenatal exposure to the 1957 A2 influenza epidemic. *Biological Psychiatry*, *38*(4), 270–273. doi: 10.1016/0006-3223(95)00241-8

Brown, D. W., Anda, R. F., Felitti, V. J., Edwards, V. J., Malarcher, A. M., Croft, J. B., & Giles, W. H. (2010). Adverse childhood experiences are associated with the risk of lung cancer: A prospective cohort study. *BMC Public Health*, *10*, 20. doi: 10.1186/1471-2458-10-20

Bruce, J., Tarullo, A. R., & Gunnar, M. R. (2009). Disinhibited social behavior among internationally adopted children. *Development and Psychopathology*, *21*(1), 157–171. doi: 10.1017/S0954579409000108

Bruer, J. T. (1999). *The myth of the first three years*. New York, NY: Free Press.

Bureau, J. F., & Moss, E. (2010). Behavioural precursors of attachment representations in middle childhood and links with child social adaptation. *British Journal of Developmental Psychology*, *28*(Pt 3), 657–677.

Buss, C., Davis, E. P., Shahbaba, B., Pruessner, J. C., Head, K., & Sandman, C. A. (2012). Maternal cortisol over the course of pregnancy and subsequent child amygdala and hippocampus volumes and affective problems. *Proceedings of the National Academy of Sciences of the United States of America*, *109*(20), E1312–1319. doi: 10.1073/pnas.1201295109

Campbell, F., Conti, G., Heckman, J. J., Moon, S. H., Pinto, R., Pungello, E., & Pan, Y. (2014). Early childhood investments substantially boost adult health. *Science*, *343*(6178), 1478–1485. doi: 10.1126/science.1248429

Campbell, F. A., Pungello, E. P., Burchinal, M., Kainz, K., Pan, Y., Wasik, B. H., … Ramey, C. T. (2012). Adult outcomes as a function of an early childhood educational program: An Abecedarian Project follow-up. *Developmental Psychology*, *48*(4), 1033–1043. doi: 10.1037/a0026644

Campbell, F. A., Pungello, E. P., Miller-Johnson, S., Burchinal, M., & Ramey, C. T. (2001). The development of cognitive and academic abilities: growth curves from an early childhood educational experiment. *Developmental Psychology*, *37*(2), 231–242.

Campbell, F. A., & Ramey, C. T. (1994). Effects of early intervention on intellectual and academic achievement: a follow-up study of children from low-income families. *Child Development*, *65*(2 Spec No), 684–698.

Carruba, G., D'Agostino, P., Miele, M., Calabro, M., Barbera, C., Bella, G. D., … Cillari, E. (2003). Estrogen regulates cytokine production and apoptosis in PMA-differentiated, macrophage-like U937 cells. *Journal of Cellular Biochemistry*, *90*(1), 187–196. doi: 10.1002/jcb.10607

Caserta, M. T., O'Connor, T. G., Wyman, P. A., Wang, H., Moynihan, J., Cross, W., … Jin, X. (2008). The associations between psychosocial stress and the frequency of illness, and innate and adaptive immune function in children. *Brain Behav Immunity*, *22*(6), 933–940. doi: 10.1016/j.bbi.2008.01.007

Chaffin, M., Hanson, R., Saunders, B. E., Nichols, T., Barnett, D., Zeanah, C., … Miller-Perrin, C. (2006). Report of the APSAC task force on attachment therapy, reactive attachment disorder, and attachment problems. *Child Maltreatment*, *11*(1), 76–89. doi: 10.1177/1077559505283699

Challis, J. R., Matthews, S. G., Van Meir, C., & Ramirez, M. M. (1995). Current topic: The placental corticotrophin-releasing hormone-adrenocorticotrophin axis. *Placenta*, *16*(6), 481–502.

Champagne, F. A., Weaver, I. C., Diorio, J., Dymov, S., Szyf, M., & Meaney, M. J. (2006). Maternal care associated with methylation of the estrogen receptor-alpha1b promoter and estrogen receptor-alpha expression in the medial preoptic area of female offspring. *Endocrinology*, *147*(6), 2909–2915. doi: 10.1210/en.2005-1119

Chen, E., Miller, G. E., Kobor, M. S., & Cole, S. W. (2010). Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. *Molecular Psychiatry*. doi: 10.1038/mp.2010.53

Chess, S. (1977). Follow-up report on autism in congenital rubella. *Journal of Autism and Childhood Schizophrenia*, *7*(1), 69–81.

Chisholm, K. (1998). A three year follow-up of attachment and indiscriminate friendliness in children adopted from Romanian orphanages. *Child Dev*, *69*(4), 1092–1106.

Chodorow, N. (1978). *The reproduction of mothering: psychoanalysis and the sociology of gender* Berkeley, CA: University of California Press.

Chugani, H. T., Behen, M. E., Muzik, O., Juhasz, C., Nagy, F., & Chugani, D. C. (2001). Local brain functional activity following early deprivation: a study of postinstitutionalized Romanian orphans. *Neuroimage*, *14*(6), 1290–1301. doi: 10.1006/nimg.2001.0917

Cicchetti, D., & Blender, J. A. (2006). A multiple-levels-of-analysis perspective on resilience: Implications for the developing brain, neural plasticity, and preventive interventions. *Ann N Y Acad Sci*, *1094*, 248–258. doi: 1094/1/248 [pii]10.1196/annals.1376.029

Cicchetti, D., & Rogosch, F. A. (2002). A developmental psychopathology perspective on adolescence. *Journal of Consulting and Clinical Psychology*, *70*(1), 6–20.

Cicchetti, D., Rogosch, F. A., Gunnar, M. R., & Toth, S. L. (2010). The differential impacts of early physical and sexual abuse and internalizing problems on daytime cortisol rhythm in school-aged children. *Child Development*, *81*(1), 252–269. doi: 10.1111/j.1467-8624.2009.01393.x

Cicchetti, D., Rogosch, F. A., Howe, M. L., & Toth, S. L. (2010). The effects of maltreatment and neuroendocrine regulation on memory performance. *Child Development*, *81*(5), 1504–1519. doi: 10.1111/j.1467-8624.2010.01488.x

Clarke, A. M., & Clarke, A. D. B. (2000). *Early experience and the life path*. London, UK: Jessica Kingsley.

Coe, C. L. (1996). Developmental Psychoneuroimmunology Revisited. *Brain, Behavior, and Immunity*, *10*(3), 185–187.

Coe, C. L., Kramer, M., Czeh, B., Gould, E., Reeves, A. J., Kirschbaum, C., & Fuchs, E. (2003). Prenatal stress diminishes neurogenesis in the dentate gyrus of juvenile rhesus monkeys. *Biological Psychiatry*, *54*(10), 1025–1034.

Coe, C. L., Lubach, G. R., Bianco, L., & Beard, J. L. (2009). A history of iron deficiency anemia during infancy alters brain monoamine activity later in juvenile monkeys. *Developmental Psychobiology*, *51*(3), 301–309. doi: 10.1002/dev.20365

Cohen, S., Janicki-Deverts, D., Doyle, W. J., Miller, G. E., Frank, E., Rabin, B. S., & Turner, R. B. (2012). Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proceedings of the National Academy of Sciences U S A*, *109*(16), 5995–5999. doi: 10.1073/pnas.1118355109

Coplan, J. D., Trost, R. C., Owens, M. J., Cooper, T. B., Gorman, J. M., Nemeroff, C. B., & Rosenblum, L. A. (1998). Cerebrospinal fluid concentrations of somatostatin and biogenic amines in grown primates reared by mothers exposed to manipulated foraging conditions. *Archives of General Psychiatry*, *55*(5), 473–477.

CPPRG. (2011). The effects of the fast track preventive intervention on the development of conduct disorder across childhood. *Child Development*, *82*(1), 331–345. doi: 10.1111/j.1467-8624.2010.01558.x

Crespi, E. J., & Denver, R. J. (2005). Ancient origins of human developmental plasticity. *American Journal of Human Biology: The Official Journal of the Human Biology Council*, *17*(1), 44–54. doi: 10.1002/ajhb.20098

Crowder, C., & Lowe, P. (2000, October 29). Her name was Candace. *Rocky Mountain News*.

Dahl, R. E., & Gunnar, M. R. (2009). Heightened stress responsiveness and emotional reactivity during pubertal maturation: Implications for psychopathology. *Development and Psychopathology*, *21*(1), 1–6. doi: 10.1017/S0954579409000017

Dantzer, B., Newman, A. E., Boonstra, R., Palme, R., Boutin, S., Humphries, M. M., & McAdam, A. G. (2013). Density triggers maternal hormones that increase adaptive offspring growth in a wild mammal. *Science*, *340*(6137), 1215–1217. doi: 10.1126/science.1235765

Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., & Cummings, E. M. (2008). Adrenocortical underpinnings of children's psychological reactivity to interparental conflict. *Child Development*, *79*(6), 1693–1706. doi: 10.1111/j.1467-8624.2008.01219.x

de Weerth, C., Zijl, R. H., & Buitelaar, J. K. (2003). Development of cortisol circadian rhythm in infancy. *Early Human Development*, *73*(1–2), 39–52.

De Wolff, M. S., & van Ijzendoorn, M. H. (1997). Sensitivity and attachment: A meta-analysis on parental antecedents of infant attachment. *Child Development*, *68*(4), 571–591.

Dishion, T. J., Kavanagh, K., Schneiger, A., Nelson, S., & Kaufman, N. K. (2002). Preventing early adolescent substance use: A family-centered strategy for the public middle school. *Prevention Science: The Official Journal Of the Society for Prevention Research*, *3*(3), 191–201.

Doupe, A. J., & Kuhl, P. K. (1999). Birdsong and human speech: common themes and mechanisms. *Annual Review of Neuroscience*, *22*, 567–631. doi: 10.1146/annurev.neuro.22.1.567

Dozier, M., Peloso, E., Lewis, E., Laurenceau, J. P., & Levine, S. (2008). Effects of an attachment-based intervention on the cortisol production of infants and toddlers in foster care. *Development and Psychopathology*, *20*(3), 845–859. doi: 10.1017/S0954579408000400

Drury, S. S., Gleason, M. M., Theall, K. P., Smyke, A. T., Nelson, C. A., Fox, N. A., & Zeanah, C. H. (2012). Genetic sensitivity to the caregiving context: The influence of 5HTTLPR and BDNF VAL66MET on indiscriminate social behavior. *Physiology & Behavior*, *106*(5), 728–735. doi: 10.1016/j.physbeh.2011.11.014

Drury, S. S., Theall, K. P., Smyke, A. T., Keats, B. J., Egger, H. L., Nelson, C. A., . . . Zeanah, C. H. (2010). Modification of depression by COMT VAL158MET polymorphism in children exposed to early severe psychosocial deprivation. *Child Abuse and Neglect*, *34*(6), 387–395. doi: 10.1016/j.chiabu.2009.09.021

Dube, S. R., Fairweather, D., Pearson, W. S., Felitti, V. J., Anda, R. F., & Croft, J. B. (2009). Cumulative childhood stress and autoimmune diseases in adults. *Psychosomatic Medicine*, *71*(2), 243–250. doi: 10.1097/PSY.0b013e3181907888

Durlak, J. A., Fuhrman, T., & Lampman, C. (1991). Effectiveness of cognitive-behavior therapy for maladapting children: A meta-analysis. *Psychological Bulletin*, *110*(2), 204–214.

Duyme, M., Dumaret, A. C., & Tomkiewicz, S. (1999). How can we boost IQs of "dull children"?: A late adoption study. *Proceedings of the National Academy of Sciences of the United States of America*, *96*(15), 8790–8794.

Eckenrode, J., Ganzel, B., Henderson, C. R., Jr., Smith, E., Olds, D. L., Powers, J., … Sidora, K. (2000). Preventing child abuse and neglect with a program of nurse home visitation: the limiting effects of domestic violence. *Journal of the American Medical Association*, *284*(11), 1385–1391.

Elder, G. H. (1986). Military time and turning points in men's lives. *Developmental Psychology*, *22*, 233–245.

Elder, G. H., Jr., (1998). The life course as developmental theory. *Child Development*, *69*(1), 1–12.

Eley, T. C., Bishop, D. V., Dale, P. S., Oliver, B., Petrill, S. A., Price, T. S., … Plomin, R. (1999). Genetic and environmental origins of verbal and performance components of cognitive delay in 2-year-olds. *Developmental Psychology*, *35*(4), 1122–1131.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology*, *23*(1), 7–28. doi: 10.1017/S0954579410000611

Entringer, S., Kumsta, R., Nelson, E. L., Hellhammer, D. H., Wadhwa, P. D., & Wust, S. (2008). Influence of prenatal psychosocial stress on cytokine production in adult women. *Developmental Psychobiology*, *50*(6), 579–587. doi: 10.1002/dev.20316

Erikson, E. H. (1968). *Identify: Youth and crisis*. New York, NY: W.W. Norton and company.

Essex, M. J., Boyce, W. T., Hertzman, C., Lam, L. L., Armstrong, J. M., Neumann, S. M., & Kobor, M. S. (2013). Epigenetic vestiges of early developmental adversity: childhood stress exposure and DNA methylation in adolescence. *Child Development*, *84*(1), 58–75. doi: 10.1111/j.1467-8624.2011.01641.x

Evans, G. W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology*, *39*(5), 924–933.

Evans, G. W., Kim, P., Ting, A. H., Tesher, H. B., & Shannis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology*, *43*(2), 341–351. doi: 10.1037/0012-1649.43.2.341

Evans, L. M., Myers, M. M., & Monk, C. (2008). Pregnant women's cortisol is elevated with anxiety and depression—but only when comorbid. *Archives of Womens Mental Health*, *11*(3), 239–248. doi: 10.1007/s00737-008-0019-4

Fagan, J. F., Holland, C. R., & Wheeler, K. (2007). The prediction, from infancy, of adult IQ and achievement. *Intelligence*, *35*(3), 225–231.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., … Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, *14*(4), 245–258. doi: 10.1016/S0749-3797(98)00017-8

Fisher, P. A., Gunnar, M. R., Dozier, M., Bruce, J., & Pears, K. C. (2006). Effects of therapeutic interventions for foster children on behavioral problems, caregiver attachment, and stress regulatory neural systems. *Ann N Y Acad Sci*, *1094*, 215–225. doi: 10.1196/annals.1376.023

Flaherty, E. G., Thompson, R., Litrownik, A. J., Zolotor, A. J., Dubowitz, H., Runyan, D. K., … Everson, M. D. (2009). Adverse childhood exposures and reported child health at age 12. *Academic Pediatrics*, *9*(3), 150–156. doi: 10.1016/j.acap.2008.11.003

Flege, J. E., Munro, M. J., & MacKay, I. R. (1995). Factors affecting strength of perceived foreign accent in a second language. *Journal of the Acoustical Society of America*, *97*(5 Pt 1), 3125–3134.

Forsythe, P., Sudo, N., Dinan, T., Taylor, V. H., & Bienenstock, J. (2010). Mood and gut feelings. *Brain, Behavior, and Immunity*, *24*(1), 9–16. doi: 10.1016/j.bbi.2009.05.058

Franceschi, C., Capri, M., Monti, D., Giunta, S., Olivieri, F., Sevini, F., … Salvioli, S. (2007). Inflammaging and anti-inflammaging: a systemic perspective on aging and longevity emerged from studies in humans. *Mech Ageing Dev*, *128*(1), 92–105. doi: 10.1016/j.mad.2006.11.016

Francis, D. D., Diorio, J., Plotsky, P. M., & Meaney, M. J. (2002). Environmental enrichment reverses the effects of maternal separation on stress reactivity. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *22*(18), 7840–7843.

Futh, A., O'Connor, T. G., Matias, C., Green, J., & Scott, S. (2008). Attachment narratives and behavioral and emotional symptoms in an ethnically diverse, at-risk sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*(6), 709–718. doi: 10.1097/CHI.0b013e31816bff65

Gallucci, W. T., Baum, A., Laue, L., Rabin, D. S., Chrousos, G. P., Gold, P. W., & Kling, M. A. (1993). Sex differences in sensitivity of the hypothalamic-pituitary-adrenal axis. *Health Psychology*, *12*(5), 420.

Garner, A. S., & Shonkoff, J. P. (2012). Early childhood adversity, toxic stress, and the role of the pediatrician: Translating developmental science into lifelong health. *Pediatrics*, *129*(1), e224–231. doi: 10.1542/peds.2011-2662

Garvey, M. A., Giedd, J., & Swedo, S. E. (1998). PANDAS: the search for environmental triggers of pediatric neuropsychiatric disorders. Lessons from rheumatic fever. *Journal of Child Neurology*, *13*(9), 413–423.

Gedo, J. E. (1980). Reflections on some current controversies in psychoanalysis. *Journal of the American Psychoanalytic Association*, *28*(2), 363–383.

Gerstorf, D., Ram, N., Mayraz, G., Hidajat, M., Lindenberger, U., Wagner, G. G., & Schupp, J. (2010). Late-life decline in well-being across adulthood in Germany, the United Kingdom, and the United States: Something is seriously wrong at the end of life. *Psychology and Aging*, *25*(2), 477–485. doi: 10.1037/a0017543

Gilmore, J. H., Shi, F., Woolson, S. L., Knickmeyer, R. C., Short, S. J., Lin, W., … Shen, D. (2012). Longitudinal development of cortical and subcortical gray matter from birth to 2 years. *Cerebral Cortex*, *22*(11), 2478–2485. doi: 10.1093/cercor/bhr327

Glover, V. (2011). Annual research review: Prenatal stress and the origins of psychopathology: An evolutionary perspective. *Journal of Child Psychology and Psychiatry*, *52*(4), 356–367. doi: 10.1111/j.1469-7610.2011.02371.x

Glover, V., O'Connor, T. G., & O'Donnell, K. (2010). Prenatal stress and the programming of the HPA axis. *Neuroscience and Biobehavior Reviews*, *35*(1), 17–22. doi: 10.1016/j.neubiorev.2009.11.008

Gluckman, P., & Hanson, M. (2005). *The fetal matrix: Evolution, development, and disease*. Cambridge, UK: Cambridge University Press.

Gluckman, P. D., Hanson, M. A., Bateson, P., Beedle, A. S., Law, C. M., Bhutta, Z. A., … West-Eberhard, M. J. (2009). Towards a new developmental synthesis: Adaptive developmental plasticity and human disease. *Lancet*, *373*(9675), 1654–1657. doi: 10.1016/S0140-6736(09)60234-8

Gluckman, P. D., Hanson, M. A., Cooper, C., & Thornburg, K. L. (2008). Effect of in utero and early-life conditions on adult health and disease. *New England Journal of Medicine*, *359*(1), 61–73. doi: 10.1056/NEJMra0708473

Glynn, L. M., Wadhwa, P. D., Dunkel-Schetter, C., Chicz-Demet, A., & Sandman, C. A. (2001). When stress happens matters: Effects of earthquake timing on stress responsivity in pregnancy. *Am J Obstet Gynecol*, *184*(4), 637–642. doi: 10.1067/mob.2001.111066

Goldberg, S., Levitan, R., Leung, E., Masellis, M., Basile, V. S., Nemeroff, C. B., & Atkinson, L. (2003). Cortisol concentrations in 12- to 18-month-old infants: stability over time, location, and stressor. *Biological Psychiatry*, *54*(7), 719–726.

Goodman, G. S., Ogle, C. M., Block, S. D., Harris, L. S., Larson, R. P., Augusti, E. M., … Urquiza, A. (2011). False memory for trauma-related Deese-Roediger-McDermott lists in adolescents and adults with histories of child sexual abuse. *Development and Psychopathology*, *23*(2), 423–438. doi: 10.1017/S0954579411000150

Goodman, G. S., Quas, J. A., & Ogle, C. M. (2010). Child maltreatment and memory. *Annual Review of Psychology*, *61*, 325–351. doi: 10.1146/annurev.psych.093008.100403

Gottlieb, G. (1991). Experiential canalization and behavioral development: Theory. *Developmental Psychology*, *27*, 4–13.

Gozzo, Y., Vohr, B., Lacadie, C., Hampson, M., Katz, K. H., Maller-Kesselman, J., … Ment, L. R. (2009). Alterations in neural connectivity in preterm children at school age. *Neuroimage*, *48*(2), 458–463. doi: 10.1016/j.neuroimage.2009.06.046

Grant, K. A., McMahon, C., Austin, M. P., Reilly, N., Leader, L., & Ali, S. (2009). Maternal prenatal anxiety, postnatal caregiving and infants' cortisol responses to the still-face procedure. *Developmental Psychobiology*, *51*(8), 625–637. doi: 10.1002/dev.20397

Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin*, *129*(3), 447–466.

Grantham-McGregor, S., & Ani, C. (2001). A review of studies on the effect of iron deficiency on cognitive development in children. *Journal of Nutrition*, *131*(2S-2), 649S–666S; discussion 666S–668S.

Greenough, W. T., Black, J. E., & Wallace, C. S. (1987). Experience and brain development. *Child Development*, *58*(3), 539–559.

Groh, A. M., Fearon, R. P., Bakermans-Kranenburg, M. J., van Ijzendoorn, M. H., Steele, R. D., & Roisman, G. I. (2014). The significance of attachment security for children's social competence with peers: A meta-analytic study. *Attachment & Human Development*, *16*(2), 103–136. doi: 10.1080/14616734.2014.883636

Guerra-Silveira, F., & Abad-Franch, F. (2013). Sex bias in infectious disease epidemiology: patterns and processes. *PLoS One*, *8*(4), e62390. doi: 10.1371/journal.pone.0062390

Guffanti, G., Galea, S., Yan, L., Roberts, A. L., Solovieff, N., Aiello, A. E., … Koenen, K. C. (2013). Genome-wide association study implicates a novel RNA gene, the lincRNA AC068718.1, as a risk factor for post-traumatic stress disorder in women. *Psychoneuroendocrinology*. doi: 10.1016/j.psyneuen.2013.08.014

Gunnar, M. R. (2003). Integrating neuroscience and psychological approaches in the study of early experiences. *Annals of the New York Academy of Sciences*, *1008*, 238–247.

Gunnar, M. R. (2010). A commentary on deprivation-specific psychological patterns. *Monographs of the Society for Research in Child Development*, *75*(1), 232–247. doi: 10.1111/j.1540-5834.2010.00559.x

Gunnar, M. R., Hertsgaard, L., Larson, M., & Rigatuso, J. (1991). Cortisol and behavioral responses to repeated stressors in the human newborn. *Developmental Psychobiology*, *24*(7), 487–505. doi: 10.1002/dev.420240704

Gunnar, M. R., & van Dulmen, M. H. (2007). Behavior problems in postinstitutionalized internationally adopted children. *Development and Psychopathology*, *19*(1), 129–148. doi: 10.1017/S0954579407070071

Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology*, *13*(3), 515–538.

Hales, C. N., & Barker, D. J. (2001). The thrifty phenotype hypothesis. *British Medical Bulletin*, *60*, 5–20.

Halligan, S. L., Herbert, J., Goodyer, I., & Murray, L. (2007). Disturbances in morning cortisol secretion in association with maternal postnatal depression predict subsequent depressive symptomatology in adolescents. *Biological Psychiatry*, *62*(1), 40–46. doi: 10.1016/j.biopsych.2006.09.011

Hansen, D., Lou, H. C., & Olsen, J. (2000). Serious life events and congenital malformations: A national study with complete follow-up. *Lancet*, *356*(9233), 875–880. doi: 10.1016/S0140-6736(00)02676-3

Hanson, M., & Gluckman, P. (2011). Developmental origins of noncommunicable disease: Population and public health implications. *Am J Clin Nutr*. doi: 10.3945/ajcn.110.001206

Harlow, H. F., & Suomi, S. J. (1970). Nature of love—simplified. *American Psychologist*, *25*(2), 161–168.

Hedegaard, M., Henriksen, T. B., Sabroe, S., & Secher, N. J. (1993). Psychological distress in pregnancy and preterm delivery. *BMJ*, *307*(6898), 234–239.

Heim, C., & Nemeroff, C. B. (2002). Neurobiology of early life stress: clinical studies. *Seminars in Clinical Neuropsychiatry*, *7*(2), 147–159. doi: 10.1053/scnp.2002.33127

Heim, C., Newport, D. J., Miller, A. H., & Nemeroff, C. B. (2000). Long-term neuroendocrine effects of childhood maltreatment. *JAMA*, *284*(18), 2321.

Hensch, T. K. (2004). Critical period regulation. *Annual Review of Neuroscience*, *27*, 549–579.

Henry, C., Kabbaj, M., Simon, H., Le Moal, M., & Maccari, S. (1994). Prenatal stress increases the hypothalamo-pituitary-adrenal axis response in young and adult rats. *Journal of Neuroendocrinology*, *6*(3), 341–345.

Herbert, M., Sluckin, W., & Sluckin, A. (1982). Mother-to-infant bonding. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *23*(3), 205–221.

Herbstman, J. B., Wang, S., Perera, F. P., Lederman, S. A., Vishnevetsky, J., Rundle, A. G., … Tang, D. (2013). Predictors and consequences of global DNA methylation in cord blood and at three years. *PLoS One*, *8*(9), e72824. doi: 10.1371/journal.pone.0072824

Hertsgaard, L., Gunnar, M., Erickson, M. F., & Nachmias, M. (1995). Adrenocortical responses to the strange situation in infants with disorganized/disoriented attachment relationships. *Child Dev*, *66*(4), 1100–1106.

Hines, M. (2003). Sex steroids and human behavior: prenatal androgen exposure and sex-typical play behavior in children. *Annals of the New York Academy of Sciences*, *1007*, 272–282.

Hockman, C. H. (1961). Prenatal maternal stress in the rat: Its effects on emotional behavior in the offspring. *Journal of Comparative and Physiological Psychology*, *54*, 679–684.

Hodges, J., & Tizard, B. (1989). Social and family relationships of ex-institutional adolescents. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *30*(1), 77–97.

Hompes, T., Izzi, B., Gellens, E., Morreels, M., Fieuws, S., Pexsters, A., … Claes, S. (2013). Investigating the influence of maternal cortisol and emotional state during pregnancy on the DNA methylation status of the glucocorticoid receptor gene (NR3C1) promoter region in cord blood. *Journal of psychiatric research*, *47*(7), 880–891. doi: 10.1016/j.jpsychires.2013.03.009

Horn, G. (2004). Pathways of the past: The imprint of memory. *Nature Reviews. Neuroscience*, *5*(2), 108–120. doi: 10.1038/nrn1324

Howe, M. L., Toth, S. L., & Cicchetti, D. (2011). Can maltreated children inhibit true and false memories for emotional information?

Child Development, *82*(3), 967–981. doi: 10.1111/j.1467-8624.2011.01585.x

Howerton, C. L., Morgan, C. P., Fischer, D. B., & Bale, T. L. (2013). O-GlcNAc transferase (OGT) as a placental biomarker of maternal stress and reprogramming of CNS gene transcription in development. *Proceedings of the National Academy of Sciences of the United States of America*, *110*(13), 5169–5174. doi: 10.1073/pnas.1300065110

Hsiao, E. Y., McBride, S. W., Chow, J., Mazmanian, S. K., & Patterson, P. H. (2012). Modeling an autism risk factor in mice leads to permanent immune dysregulation. *Proceedings of the National Academy of Sciences of the United States of America*, *109*(31), 12776–12781. doi: 10.1073/pnas.1202556109

Hsiao, E. Y., McBride, S. W., Hsien, S., Sharon, G., Hyde, E. R., McCue, T., … Mazmanian, S. K. (2013). Microbiota modulate behavioral and physiological abnormalities associated with neurodevelopmental disorders. *Cell*, *155*(7), 1451–1463. doi: 10.1016/j.cell.2013.11.024

Huang, Z. J., Kirkwood, A., Pizzorusso, T., Porciatti, V., Morales, B., Bear, M. F., … Tonegawa, S. (1999). BDNF regulates the maturation of inhibition and the critical period of plasticity in mouse visual cortex. *Cell*, *98*(6), 739–755.

Hubel, D. H., & Wiesel, T. N. (1970). The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *Journal of Physiology*, *206*(2), 419–436.

Huizink, A. C., Mulder, E. J., & Buitelaar, J. K. (2004). Prenatal stress and risk for psychopathology: specific effects or induction of general susceptibility? *Psychological Bulletin*, *130*(1), 115–142. doi: 10.1037/0033-2909.130.1.1152003-11000-005

Hulshoff Pol, H. E., Hoek, H. W., Susser, E., Brown, A. S., Dingemans, A., Schnack, H. G., … Kahn, R. S. (2000). Prenatal exposure to famine and brain morphology in schizophrenia. *American Journal of Psychiatry*, *157*(7), 1170–1172.

Huttenlocher, P. R., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *Journal of Comparative Neurology*, *387*(2), 167–178.

Ibanez, L., Ong, K., Dunger, D. B., & de Zegher, F. (2006). Early development of adiposity and insulin resistance after catch-up weight gain in small-for-gestational-age children. *Journal of Clinical Endocrinology and Metabolism*, *91*(6), 2153–2158. doi: 10.1210/jc.2005-2778

Insel, T. R., & Wang, P. S. (2010). Rethinking mental illness. *Journal of the American Medical Association*, *303*(19), 1970–1971. doi: 10.1001/jama.2010.555

Ito, A., Bebo, B. F., Jr., Matejuk, A., Zamora, A., Silverman, M., Fyfe-Johnson, A., & Offner, H. (2001). Estrogen treatment down-regulates TNF-alpha production and reduces the severity of experimental autoimmune encephalomyelitis in cytokine knockout mice. *Journal of Immunology*, *167*(1), 542–552.

Jacka, F. N., Ystrom, E., Brantsaeter, A. L., Karevold, E., Roth, C., Haugen, M., … Berk, M. (2013). Maternal and early postnatal nutrition and mental health of offspring by age 5 years: a prospective cohort study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*(10), 1038–1047. doi: 10.1016/j.jaac.2013.07.002

Jackson, B., Kubzansky, L. D., Cohen, S., Weiss, S., & Wright, R. J. (2004). A matter of life and breath: childhood socioeconomic status is related to young adult pulmonary function in the CARDIA study. *International Journal of Epidemiology*, *33*(2), 271–278. doi: 10.1093/ije/dyh00333/2/271

Joffe, J. M. (1965). Genotype and prenatal and premating stress interact to affect adult behavior in rats. *Science*, *150*(3705), 1844–1845.

Johnson, J. S., & Newport, E. L. (1989). Critical period effects in second language learning: The influence of maturational state on the acquisition of English as a second language. *Cognitive Psychology*, *21*(1), 60–99.

Jones, K., Daley, D., Hutchings, J., Bywater, T., & Eames, C. (2008). Efficacy of the Incredible Years Programme as an early intervention for children with conduct problems and ADHD: long-term follow-up. *Child: Care, Health and Development*, *34*(3), 380–390. doi: 10.1111/j.1365-2214.2008.00817.x

Joseph, M. A., O'Connor, T. G., Briskman, J. A., Maughan, B., & Scott, S. (2014). The formation of secure new attachments by children who were maltreated: an observational study of adolescents in foster care. *Development and Psychopathology*, *26*(1), 67–80. doi: 10.1017/S0954579413000540

Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, *35*(1), 2–16. doi: 10.1016/j.neubiorev.2009.10.002

Kagan, J. (1982). The emergence of self. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *23*(4), 363–381.

Keck, G. C., & Kupecky, R. (1995). *Adopting the hurt child*. Colorado Springs, CO: Pinon Press.

Keeley, K. (1962). Prenatal influence on behavior of offspring of crowded mice. *Science*, *135*(3497), 44–45.

Keller, P. S., El-Sheikh, M., Vaughn, B., & Granger, D. A. (2010). Relations between mucosal immunity and children's mental health: The role of child sex. *Physiol Behav*, *101*(5), 705–712. doi: 10.1016/j.physbeh.2010.08.012

Kennell, J. H., Trause, M. A., & Klaus, M. H. (1975). Evidence for a sensitive period in the human mother. *Ciba Foundation Symposium* (*33*), 87–101.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of *DSM-IV* disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602. doi: 10.1001/archpsyc.62.6.593

Khashan, A. S., Abel, K. M., McNamee, R., Pedersen, M. G., Webb, R. T., Baker, P. N., … Mortensen, P. B. (2008). Higher Risk of Offspring Schizophrenia Following Antenatal Maternal Exposure to Severe Adverse Life Events. *Archives of General Psychiatry*, *65*(2), 146–152. doi: 10.1001/archgenpsychiatry.2007.20

King, S., & Laplante, D. P. (2005). The effects of prenatal maternal stress on children's cognitive development: Project Ice Storm. *Stress*, *8*(1), 35–45. doi: 10.1080/10253890500108391

King, S., Mancini-Marie, A., Brunet, A., Walker, E., Meaney, M. J., & Laplante, D. P. (2009). Prenatal maternal stress from a natural disaster predicts dermatoglyphic asymmetry in humans. *Development and Psychopathology*, *21*(2), 343–353. doi: 10.1017/S0954579409000364

Kinney, D. K., Miller, A. M., Crowley, D. J., Huang, E., & Gerber, E. (2008). Autism prevalence following prenatal exposure to hurricanes and tropical storms in Louisiana. *Journal of Autism and Developmental Disorders*, *38*(3), 481–488.

Kinnunen, A. K., Koenig, J. I., & Bilbe, G. (2003). Repeated variable prenatal stress alters pre- and postsynaptic gene expression in the rat frontal pole. *J Neurochem*, *86*(3), 736–748.

Kirp, D. L. (2007). *The sandbox investment. The preschool movement in kids-first politics*. Cambridge, MA: Harvard University Press.

Kitzman, H., Olds, D. L., Henderson, C. R., Jr., Hanks, C., Cole, R., Tatelbaum, R., … Barnard, K. (1997). Effect of prenatal and infancy home visitation by nurses on pregnancy outcomes, childhood injuries, and repeated childbearing. A randomized controlled trial. *Journal of the American Medical Association*, *278*(8), 644–652.

Kitzman, H., Olds, D. L., Sidora, K., Henderson, C. R., Jr., Hanks, C., Cole, R., … Glazner, J. (2000). Enduring effects of nurse home visitation on maternal life course: A 3-year follow-up of a

randomized trial. *Journal of the American Medical Association*, *283*(15), 1983–1989.

Kitzman, H. J., Olds, D. L., Cole, R. E., Hanks, C. A., Anson, E. A., Arcoleo, K. J., … Holmberg, J. R. (2010). Enduring effects of prenatal and infancy home visiting by nurses on children: Follow-up of a randomized trial among children at age 12 years. *Archives of Pediatrics & Adolescent Medicine*, *164*(5), 412–418. doi: 10.1001/archpediatrics.2010.76

Kivlighan, K. T., DiPietro, J. A., Costigan, K. A., & Laudenslager, M. L. (2008). Diurnal rhythm of cortisol during late pregnancy: Associations with maternal psychological well-being and fetal growth. *Psychoneuroendocrinology*, *33*(9), 1225–1235. doi: 10.1016/j.psyneuen.2008.06.008

Knickmeyer, R. C., Gouttard, S., Kang, C., Evans, D., Wilber, K., Smith, J. K., … Gilmore, J. H. (2008). A structural MRI study of human brain development from birth to 2 years. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *28*(47), 12176–12182. doi: 10.1523/JNEUROSCI.3479-08.2008

Knudsen, E. I. (2004). Sensitive periods in the development of the brain and behavior. *Journal of Cognitive Neuroscience*, *16*(8), 1412–1425. doi: 10.1162/0898929042304796

Kobak, R. R., Cole, H. E., Ferenz-Gillies, R., Fleming, W. S., & Gamble, W. (1993). Attachment and emotion regulation during mother-teen problem solving: A control theory analysis. *Child Development*, *64*(1), 231–245.

Kohman, R. A., & Rhodes, J. S. (2013). Neurogenesis, inflammation and behavior. *Brain, Behavior, and Immunity*, *27*(1), 22–32. doi: 10.1016/j.bbi.2012.09.003

Kohut, H. (1971). *The analysis of the self*. New York, NY: International Universities Press.

Kramer, P. R., Kramer, S. F., & Guan, G. (2004). 17 beta-estradiol regulates cytokine release through modulation of CD16 expression in monocytes and monocyte-derived macrophages. *Arthritis and Rheumatism*, *50*(6), 1967–1975. doi: 10.1002/art.20309

Kreppner, J., Kumsta, R., Rutter, M., Beckett, C., Castle, J., Stevens, S., & Sonuga-Barke, E. J. (2010). IV. Developmental course of deprivation-specific psychological patterns: early manifestations, persistence to age 15, and clinical features. *Monographs of the Society for Research in Child Development*, *75*(1), 79–101. doi: 10.1111/j.1540-5834.2010.00551.x

Kreppner, J. M., O'Connor, T. G., & Rutter, M. (2001). Can inattention/overactivity be an institutional deprivation syndrome? *Journal of Abnormal Child Psychology*, *29*(6), 513–528.

Kreppner, J. M., Rutter, M., Beckett, C., Castle, J., Colvert, E., Groothues, C., … Sonuga-Barke, E. J. (2007). Normality and impairment following profound early institutional deprivation: A longitudinal follow-up into early adolescence. *Developmental Psychology*, *43*(4), 931–946. doi: 10.1037/0012-1649.43.4.93

Labonte, B., Suderman, M., Maussion, G., Lopez, J. P., Navarro-Sanchez, L., Yerko, V., … Turecki, G. (2013). Genome-wide methylation changes in the brains of suicide completers. *American Journal of Psychiatry*, *170*(5), 511–520. doi: 10.1176/appi.ajp.2012.12050627

Lam, L. L., Emberly, E., Fraser, H. B., Neumann, S. M., Chen, E., Miller, G. E., & Kobor, M. S. (2012). Factors underlying variable DNA methylation in a human community cohort. *Proceedings of the National Academy of Sciences of the United States of America*, *109* Suppl 2, 17253–17260. doi: 10.1073/pnas.1121249109

Lang, T. J. (2004). Estrogen as an immunomodulator. *Clinical Immunology*, *113*(3), 224–230. doi: 10.1016/j.clim.2004.05.011

Laplante, D. P., Brunet, A., Schmitz, N., Ciampi, A., & King, S. (2008). Project Ice Storm: Prenatal maternal stress affects cognitive and linguistic functioning in 5 1/2-year-old children. *Journal of the American Academy of Child Adolescent Psychiatry*, *47*(9), 1063–1072. doi: 10.1097/CHI.0b013e31817eec80

Lemieux, A., Coe, C. L., & Carnes, M. (2008). Symptom severity predicts degree of T cell activation in adult women following childhood maltreatment. *Brain Behav Immun*, *22*(6), 994–1003. doi: 10.1016/j.bbi.2008.02.005

Lewis, M., & Ramsay, D. S. (1995). Developmental change in infants' responses to stress. *Child Development*, *66*(3), 657–670.

Licznerski, P., & Duman, R. S. (2013). Remodeling of axo-spinous synapses in the pathophysiology and treatment of depression. *Neuroscience*, *251*, 33–50. doi: 10.1016/j.neuroscience.2012.09.057

Lorenz, K. (1935). The companion in the bird's world. *The fellow-member of the species as releasing factor of social behavior. Journal fur Ornithologie Beiblatt (Leipzig)*, *83*, 137–213.

Lovden, M., Backman, L., Lindenberger, U., Schaefer, S., & Schmiedek, F. (2010). A theoretical framework for the study of adult cognitive plasticity. *Psychological Bulletin*, *136*(4), 659–676. doi: 10.1037/a0020080

Lovejoy, D. A., & Barsyte-Lovejoy, D. (2013). Systems approaches to genomic and epigenetic inter-regulation of peptide hormones in stress and reproduction. *Progress in Biophysics and Molecular Biology*. doi: 10.1016/j.pbiomolbio.2013.02.002

Luecken, L. J., & Appelhans, B. M. (2006). Early parental loss and salivary cortisol in young adulthood: The moderating role of family environment. *Development and Psychopathology*, *18*(1), 295–308. doi: 10.1017/S0954579406060160

Luo, L., & O'Leary, D. D. (2005). Axon retraction and degeneration in development and disease. *Annual Review of Neuroscience*, *28*, 127–156. doi: 10.1146/annurev.neuro.28.061604.135632

Lupien, S. J., de Leon, M., de Santi, S., Convit, A., Tarshish, C., Nair, N. P., … Meaney, M. J. (1998). Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nature Neuroscience*, *1*(1), 69–73. doi: 10.1038/271

Maccari, S., Piazza, P. V., Kabbaj, M., Barbazanges, A., Simon, H., & Le Moal, M. (1995). Adoption reverses the long-term impairment in glucocorticoid feedback induced by prenatal stress. *Journal of Neuroscience*, *15*(1 Pt 1), 110–116.

MacDonald, K. (1985). Early experience, relative plasticity, and social development. *Developmental Review*, *5*, 99–121.

Mackner, L., Pajer, K., Glaser, R., & Crandell, W. (2010). *Relationships between depression, cytokines, and cortisol in pediatric inflammatory bowel disease*. Paper presented at the AGA abstracts. San Diego, CA October 2014.

Maestripieri, D., Tomaszycki, M., & Carroll, K. A. (1999). Consistency and change in the behavior of rhesus macaque abusive mothers with successive infants. *Developmental Psychobiology*, *34*(1), 29–35.

Malassine, A., Frendo, J. L., & Evain-Brion, D. (2003). A comparison of placental development and endocrine functions between the human and mouse model. *Human Reproduction Update*, *9*(6), 531–539.

Mapstone, M., Cheema, A. K., Fiandaca, M. S., Zhong, X., Mhyre, T. R., Macarthur, L. H., … Federoff, H. J. (2014). Plasma phospholipids identify antecedent memory impairment in older adults. *Nature Medicine*, *20*(4), 415–418. doi: 10.1038/nm.3466

Martin, L. J., Spicer, D. M., Lewis, M. H., Gluck, J. P., & Cork, L. C. (1991). Social deprivation of infant rhesus monkeys alters the chemoarchitecture of the brain: I. Subcortical regions. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *11*(11), 3344–3358.

Marvin, R. S., & Britner, P. A. (2008). Normative development: The ontogeny of attachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook*

of attachment: Theory, research and clinical applications (pp. 269–294). New York, NY: Guilford.

Matthews, S. G. (2000). Antenatal glucocorticoids and programming of the developing CNS. *Pediatric Research*, *47*(3), 291–300.

Matthews, S. G., Owen, D., Banjanin, S., & Andrews, M. H. (2002). Glucocorticoids, hypothalamo-pituitary-adrenal (HPA) development, and life after birth. *Endocrine Research*, *28*(4), 709–718.

Maurer, D., Lewis, T. L., Brent, H. P., & Levin, A. V. (1999). Rapid improvement in the acuity of infants after visual input. *Science*, *286*(5437), 108–110.

Mayer, K. U., & Schöpflin, U. (1989). The state and the life course. *Annual Review of Sociology*, *15*, 187–209.

McCarton, C. M., Brooks-Gunn, J., Wallace, I. F., Bauer, C. R., Bennett, F. C., Bernbaum, J. C., … Meinert, C. L. (1997). Results at age 8 years of early intervention for low-birth-weight premature infants. The Infant Health and Development Program. *Journal of the American Medical Association*, *277*(2), 126–132.

McElhaney, J. E., & Effros, R. B. (2009). Immunosenescence: what does it mean to health outcomes in older adults? *Curr Opin Immunol*, *21*(4), 418–424. doi: 10.1016/j.coi.2009.05.023

McEwen, B. S., & Wingfield, J. C. (2003). The concept of allostasis in biology and biomedicine. *Hormones and Behavior*, *43*(1), 2–15.

McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonte, B., Szyf, M., … Meaney, M. J. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience*, *12*(3), 342–348. doi: 10.1038/nn.2270

McLaughlin, K. A., Fox, N. A., Zeanah, C. H., Sheridan, M. A., Marshall, P., & Nelson, C. A. (2010). Delayed maturation in brain electrical activity partially explains the association between early environmental deprivation and symptoms of attention-deficit/hyperactivity disorder. *Biol Psychiatry*, *68*(4), 329–336. doi: 10.1016/j.biopsych.2010.04.005

Meaney, M. J. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, *81*(1), 41–79. doi: 10.1111/j.1467-8624.2009.01381.x

Mehta, D., Klengel, T., Conneely, K. N., Smith, A. K., Altmann, A., Pace, T. W., … Binder, E. B. (2013). Childhood maltreatment is associated with distinct genomic and epigenetic profiles in posttraumatic stress disorder. *Proceedings of the National Academy of Sciences of the United States of America*, *110*(20), 8302–8307. doi: 10.1073/pnas.1217750110

Mercer, J. (2005). Coercive restraint therapies: a dangerous alternative mental health intervention. *MedGenMed*, *7*(3), 6. doi: 508956 [pii]

Merz, E. C., & McCall, R. B. (2010). Behavior problems in children adopted from psychosocially depriving institutions. *Journal of Abnormal Child Psychology*, *38*(4), 459–470. doi: 10.1007/s10802-009-9383-4

Miller, G. E., Chen, E., Fok, A. K., Walker, H., Lim, A., Nicholls, E. F., … Kobor, M. S. (2009). Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proceedings of the National Academy of Sciences U S A*, *106*(34), 14716–14721. doi: 10.1073/pnas.0902971106

Mills, N. T., Scott, J. G., Wray, N. R., Cohen-Woods, S., & Baune, B. T. (2013). Research review: The role of cytokines in depression in adolescents: a systematic review. *Journal of Child Psychology and Psychiatry*, *54*, 816–835.

Moles, A., Kieffer, B. L., & D'Amato, F. R. (2004). Deficit in attachment behavior in mice lacking the mu-opioid receptor gene. *Science*, *304*(5679), 1983–1986. doi: 10.1126/science.1095943

Money, K. M., & Stanwood, G. D. (2013). Developmental origins of brain disorders: Roles for dopamine. *Frontiers in Cellular Neuroscience*, *7*, 260. doi: 10.3389/fncel.2013.00260

Monje, M. L., Toda, H., & Palmer, T. D. (2003). Inflammatory blockade restores adult hippocampal neurogenesis. *Science*, *302*(5651), 1760–1765. doi: 10.1126/science.1088417

Monk, C., Fifer, W. P., Myers, M. M., Bagiella, E., Duong, J. K., Chen, I. S., … Altincatal, A. (2011). Effects of maternal breathing rate, psychiatric status, and cortisol on fetal heart rate. *Developmental Psychobiology*, *53*(3), 221–233. doi: 10.1002/dev.20513

Mooney, R. (1999). Sensitive periods and circuits for learned birdsong. *Current Opinion in Neurobiology*, *9*(1), 121–127.

Moyer, V. A. (2013). Screening for primary hypertension in children and adolescents: U.S. Preventive Services Task Force recommendation statement. *Pediatrics*, *132*(5), 907–914. doi: 10.1542/peds.2013-2864

National Scientific Council on the Developing Child. (2012). The science of neglect: The persistent absence of responsive care disrupts the developing brain, Working Paper 12. http://www.developingchild.harvard.edu

Nelson, C. A., III, Zeanah, C. H., Fox, N. A., Marshall, P. J., Smyke, A. T., & Guthrie, D. (2007). Cognitive recovery in socially deprived young children: the Bucharest Early Intervention Project. *Science*, *318*(5858), 1937–1940. doi: 318/5858/1937 [pii]10.1126/science.1143921

Newport, D. J., Heim, C., Owens, M. J., Ritchie, J. C., Ramsey, C. H., Bonsall, R., … Nemeroff, C. B. (2003). Cerebrospinal fluid corticotropin-releasing factor (CRF) and vasopressin concentrations predict pituitary response in the CRF stimulation test: A multiple regression analysis. *Neuropsychopharmacology*, *28*(3), 569–576. doi: 10.1038/sj.npp.1300071

NICHD, E. C. C. R. N. (2007). Age of entry to kindergarten and children's academic achievement and socioemotional development. *Early Education and Development*, *18*(2), 337–368. doi: 10.1080/10409280701283460

Nicolson, N. A. (2004). Childhood parental loss and cortisol levels in adult men. *Psychoneuroendocrinology*, *29*(8), 1012–1018. doi: 10.1016/j.psyneuen.2003.09.005

O'Connor, T. G., Bergman, K., Sarkar, P., & Glover, V. (2013). Prenatal cortisol exposure predicts infant cortisol response to acute stress. *Developmental Psychobiology*, *55*(2), 145–155. doi: 10.1002/dev.21007

O'Connor, T. G., & Cameron, J. L. (2006). Translating research findings on early experience to prevention: animal and human evidence on early attachment relationships. *American Journal of Preventive Medicine*, *31*(6 Suppl 1), S175–181. doi: 10.1016/j.amepre.2006.07.005

O'Connor, T. G., Heron, J., Golding, J., & Glover, V. (2003). Maternal antenatal anxiety and behavioural/emotional problems in children: A test of a programming hypothesis. *Journal of Child Psychology and Psychiatry*, *44*(7), 1025–1036.

O'Connor, T. G., Marvin, R. S., Rutter, M., Olrick, J. T., & Britner, P. A. (2003). Child–parent attachment following early institutional deprivation. *Development and Psychopathology*, *15*(1), 19–38.

O'Connor, T. G., Moynihan, J. A., & Caserta, M. T. (2014). Annual research review: The neuroinflammation hypothesis for stress and psychopathology in children—Developmental psychoneuroimmunology. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *55*(6), 615–631. doi: 10.1111/jcpp.12187

O'Connor, T. G., Tang, W., Gilchrist, M. A., Moynihan, J. A., Pressman, E. K., & Blackmore, E. R. (2014). Diurnal cortisol patterns and psychiatric symptoms in pregnancy: Short-term longitudinal study. *Biological Psychology*, *96*, 35–41. doi: 10.1016/j.biopsycho.2013.11.002

O'Connor, T. G., Winter, M. A., Hunn, J., Carnahan, J., Pressman, E. K., Glover, V., … Caserta, M. T. (2013). Prenatal maternal anxiety predicts reduced adaptive immunity in infants. *Brain, Behavior, and Immunity*, *32*, 21–28. doi: 10.1016/j.bbi.2013.02.002

O'Donnell, K. J., Bugge Jensen, A., Freeman, L., Khalife, N., O'Connor, T. G., & Glover, V. (2012). Maternal prenatal anxiety and downregulation of placental 11beta-HSD2. *Psychoneuroendocrinology*, *37*(6), 818–826. doi: 10.1016/j.psyneuen.2011.09.014

O'Donnell, K. J., Glover, V., Barker, E. D., & O'Connor, T. G. (2014). The persisting effect of maternal mood in pregnancy on childhood psychopathology. *Development and Psychopathology*, *26*(2), 393–403. doi: 10.1017/S0954579414000029

O'Donnell, K. J., Glover, V., Jenkins, J., Browne, D., Ben-Shlomo, Y., Golding, J., & O'Connor, T. G. (2013). Prenatal maternal mood is associated with altered diurnal cortisol in adolescence. *Psychoneuroendocrinology*, *38*(9), 1630–1638. doi: 10.1016/j.psyneuen.2013.01.008

Oberlander, T. F., Gingrich, J. A., & Ansorge, M. S. (2009). Sustained neurobehavioral effects of exposure to SSRI antidepressants during development: molecular to clinical evidence. *Clinical Pharmacology and Therapeutics*, *86*(6), 672–677. doi: 10.1038/clpt.2009.201

Oberlander, T. F., Weinberg, J., Papsdorf, M., Grunau, R., Misri, S., & Devlin, A. M. (2008). Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR3C1) and infant cortisol stress responses. *Epigenetics*, *3*(2), 97–106.

Obsuth, I., Hennighausen, K., Brumariu, L. E., & Lyons-Ruth, K. (2013). Disorganized behavior in adolescent–parent interaction: Relations to attachment state of mind, partner abuse, and psychopathology. *Child Development*. doi: 10.1111/cdev.12113

Olds, D. L., Henderson, C. R., Jr., & Kitzman, H. (1994). Does prenatal and infancy nurse home visitation have enduring effects on qualities of parental caregiving and child health at 25 to 50 months of life? *Pediatrics*, *93*(1), 89–98.

Olds, D. L., Kitzman, H. J., Cole, R. E., Hanks, C. A., Arcoleo, K. J., Anson, E. A., … Stevenson, A. J. (2010). Enduring effects of prenatal and infancy home visiting by nurses on maternal life course and government spending: Follow-up of a randomized trial among children at age 12 years. *Archives of Pediatrics & Adolescent Medicine*, *164*(5), 419–424. doi: 10.1001/archpediatrics.2010.49

Ottaviani, E., & Franceschi, C. (1996). The neuroimmunology of stress from invertebrates to man. *Progress in Neurobiology*, *48*(4–5), 421–440.

Painter, R. C., Roseboom, T. J., & Bleker, O. P. (2005). Prenatal exposure to the Dutch famine and disease in later life: An overview. *Reproductive Toxicology*, *20*(3), 345–352. doi: 10.1016/j.reprotox.2005.04.005

Paolicelli, R. C., Bolasco, G., Pagani, F., Maggi, L., Scianni, M., Panzanelli, P., … Gross, C. T. (2011). Synaptic pruning by microglia is necessary for normal brain development. *Science*, *333*(6048), 1456–1458. doi: 10.1126/science.1202529

Pawelec, G., Larbi, A., & Derhovanessian, E. (2010). Senescence of the human immune system. *J Comp Pathol*, *142* Suppl 1, S39–44. doi: 10.1016/j.jcpa.2009.09.005

Phillips, D. I., Walker, B. R., Reynolds, R. M., Flanagan, D. E., Wood, P. J., Osmond, C., … Whorwood, C. B. (2000). Low birth weight predicts elevated plasma cortisol concentrations in adults from 3 populations. *Hypertension*, *35*(6), 1301–1306.

Pitzer, M., Jennen-Steinmetz, C., Esser, G., Schmidt, M. H., & Laucht, M. (2011). Differential susceptibility to environmental influences: The role of early temperament and parenting in the development of externalizing problems. *Comprehensive Psychiatry*, *52*(6), 650–658. doi: 10.1016/j.comppsych.2010.10.017

Plomin, R. (2005). Finding genes in child psychology and psychiatry: When are we going to be there? *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *46*(10), 1030–1038. doi: 10.1111/j.1469-7610.2005.01524.x

Plomin, R., Fulker, D., Corley, R., & DeFries, J. C. (1997). Nature, nurture, and cognitive development from 1 to 16 years: A parent-offspring adoption study. *Psychological Science*, *8*, 442–447.

Plomin, R., & Rende, R. (1991). Human behavioral genetics. *Annual Review of Psychology*, *42*, 161–190. doi: 10.1146/annurev.ps.42.020191.001113

Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: developmental effects of child abuse and neglect. *Developmental Psychology*, *36*(5), 679–688.

Post, R. M. (1992). Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. *American Journal of Psychiatry*, *149*(8), 999–1010.

Post, R. M., Leverich, G. S., Xing, G., & Weiss, R. B. (2001). Developmental vulnerabilities to the onset and course of bipolar disorder. *Development and Psychopathology*, *13*(3), 581–598.

Propper, C., Willoughby, M., Halpern, C. T., Carbone, M. A., & Cox, M. (2007). Parenting quality, DRD4, and the prediction of externalizing and internalizing behaviors in early childhood. *Developmental Psychobiology*, *49*(6), 619–632. doi: 10.1002/dev.20249

Quinton, D., Rutter, M., & Liddle, C. (1984). Institutional rearing, parenting difficulties and marital support. *Psychological Medicine*, *14*(1), 107–124.

Ramey, C. T., & Campbell, F. A. (1984). Preventive education for high-risk children: Cognitive consequences of the Carolina Abecedarian Project. *American Journal of Mental Deficiency*, *88*(5), 515–523.

Ramey, C. T., & Ramey, S. L. (1998). Early intervention and early experience. *American Psychologist*, *53*(2), 109–120.

Ramey, C. T., & Sackett, G. P. (2000). The early caregiving environment: Expanding views on nonparental care and cumulative life experiences. In A. Sameroff, M. Lewis & S. M. Miller (Eds.), *Handbook of Developmental Psychopathology* (2nd ed., pp. 365–380). New York, NY: Kluwer Academic/Plenum Publishers.

Rank, B. (1949). Adaptation of the psychoanalytic technique for the treatment of young children with atypical development. *American Journal of Orthopsychiatry*, *19*(1), 130–139.

Rank, O. (1929). *The trauma of birth*. London, UK: Kegan Paul, Trench, Trubner, and Co., Ltd.

Reik, W., Dean, W., & Walter, J. (2001). Epigenetic reprogramming in mammalian development. *Science*, *293*(5532), 1089–1093. doi: 10.1126/science.1063443

Reynolds, A. J., Temple, J. A., Robertson, D. L., & Mann, E. A. (2001). Long-term effects of an early childhood intervention on educational achievement and juvenile arrest: A 15-year follow-up of low-income children in public schools. *Journal of the American Medical Association*, *285*(18), 2339–2346.

Rice, D., & Barone, S., Jr., (2000). Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. *Environmental Health Perspectives*, *108* Suppl 3, 511–533.

Rice, F., Jones, I., & Thapar, A. (2007). The impact of gestational stress and prenatal growth on emotional problems in offspring: A review. *Acta Psychiatr Scand*, *115*(3), 171–183. doi: ACP895 [pii]10.1111/j.1600-0447.2006.00895.x

Rifkin-Graboi, A., Borelli, J. L., & Enlow, M. B. (2009). Neurobiology of stress in infancy. In C. H. Zeanah (Ed.), *Handbook of infant mental health* (3rd ed., pp. 59–79). New York, NY: Guilford.

Robinson, M., Mattes, E., Oddy, W. H., Pennell, C. E., van Eekelen, A., McLean, N. J., … Newnham, J. P. (2011). Prenatal stress and

risk of behavioral morbidity from age 2 to 14 years: the influence of the number, type, and timing of stressful life events. *Development and Psychopathology*, *23*(2), 507–520. doi: 10.1017/S0954579411000241

Roseboom, T. J., van der Meulen, J. H., Osmond, C., Barker, D. J., Ravelli, A. C., Schroeder-Tanka, J. M., … Bleker, O. P. (2000). Coronary heart disease after prenatal exposure to the Dutch famine, 1944–45. *Heart*, *84*(6), 595–598.

Roseboom, T. J., Van Der Meulen, J. H., Ravelli, A. C., Osmond, C., Barker, D. J., & Bleker, O. P. (2003). Perceived health of adults after prenatal exposure to the Dutch famine. *Paediatric and Perinatal Epidemiology*, *17*(4), 391–397.

Ross, E. J., Graham, D. L., Money, K. M., & Stanwood, G. D. (2014). Developmental consequences of fetal exposure to drugs: What we know and what we still must learn. Neuropsychopharmacology*: Official Publication of the American College of Neuropsychopharmacology*. doi: 10.1038/npp.2014.147

Rusiecki, J. A., Byrne, C., Galdzicki, Z., Srikantan, V., Chen, L., Poulin, M., … Baccarelli, A. (2013). PTSD and DNA methylation in select immune function gene promoter regions: A repeated measures case-control study of U.S. military service members. *Frontiers in Psychiatry*, *4*, 56. doi: 10.3389/fpsyt.2013.00056

Rutter, M., Kreppner, J. M., & O'Connor, T. G. (2001). Specificity and heterogeneity in children's responses to profound institutional privation. *Br J Psychiatry*, *179*, 97–103.

Rutter, M., & O'Connor, T. G. (2004). Are there biological programming effects for psychological development? Findings from a study of Romanian adoptees. *Developmenal Psychology*, *40*(1), 81–94. doi: 10.1037/0012-1649.40.1.812003-10667-007

Sackett, G. P. (1969). The persistence of abnormal behaviour in monkeys following isolation rearing. *International Psychiatry Clinics*, *6*(1), 3–37.

Sanchez, M. M., Ladd, C. O., & Plotsky, P. M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: Evidence from rodent and primate models. *Development and Psychopathology*, *13*(3), 419–449.

Sandman, C. A., Davis, E. P., Buss, C., & Glynn, L. M. (2011a). Exposure to Prenatal psychobiological stress exerts programming influences on the mother and her fetus. *Neuroendocrinology*. doi: 10.1159/000327017

Sandman, C. A., Davis, E. P., Buss, C., & Glynn, L. M. (2011b). Prenatal programming of human neurological function. *International Journal of Peptides*, 2011, 837596. doi: 10.1155/2011/837596

Sandman, C. A., Davis, E. P., & Glynn, L. M. (2012). Prescient human fetuses thrive. *Psychological Science*, *23*(1), 93–100. doi: 10.1177/0956797611422073

Sarkar, P., Bergman, K., Fisk, N. M., O'Connor, T. G., & Glover, V. (2007a). Ontogeny of foetal exposure to maternal cortisol using midtrimester amniotic fluid as a biomarker. *Clinical Endocrinology (Oxf)*, *66*(5), 636–640. doi: CEN2785 [pii]10.1111/j.1365-2265.2007.02785.x

Sarkar, P., Bergman, K., Fisk, N. M., O'Connor, T. G., & Glover, V. (2007b). Ontogeny of foetal exposure to maternal cortisol using midtrimester amniotic fluid as a biomarker. *Clinical Endocrinology*, *66*(5), 636–640. doi: 10.1111/j.1365-2265.2007.02785.x

Sarkar, S., Craig, M. C., Dell'acqua, F., O'Connor, T. G., Catani, M., Deeley, Q., … Murphy, D. G. (2014). Prenatal stress and limbic-prefrontal white matter microstructure in children aged 6-9 years: A preliminary diffusion tensor imaging study. *The World Journal of Biological Psychiatry: The Official Journal of the World Federation of Societies of Biological Psychiatry*, *15*(4), 346–352. doi: 10.3109/15622975.2014.903336

Saudino, K. J., & Eaton, W. O. (1991). Infant temperament and genetics: An objective twin study of motor activity level. *Child Development*, *62*(5), 1167–1174.

Sawhill, I., Thomas, A., & Monea, E. (2010). An ounce of prevention: Policy prescriptions to reduce the prevalence of fragile families. *The Future of Children/Center for the Future of Children, the David and Lucile Packard Foundation*, *20*(2), 133–155.

Schneider-Rosen, K., & Cicchetti, D. (1984). The relationship between affect and cognition in maltreated infants: Quality of attachment and the development of visual self-recognition. *Child Dev*, *55*(2), 648–658.

Schneider, M. L., Clarke, A. S., Kraemer, G. W., Roughton, E. C., Lubach, G. R., Rimm-Kaufman, S., … Ebert, M. (1998). Prenatal stress alters brain biogenic amine levels in primates. *Development and Psychopathology*, *10*(3), 427–440.

Schneider, M. L., Coe, C. L., & Lubach, G. R. (1992). Endocrine activation mimics the adverse effects of prenatal stress on the neuromotor development of the infant primate. *Developmental Psychobiology*, *25*(6), 427–439. doi: 10.1002/dev.420250604

Schwartz, M., & Kipnis, J. (2011). A conceptual revolution in the relationships between the brain and immunity. *Brain Behav Immun*, *25*(5), 817–819. doi: 10.1016/j.bbi.2010.12.015

Scott, S., Briskman, J., & O'Connor, T. G. (2014). Early prevention of antisocial personality: Long-term follow-up of two randomized controlled trials comparing indicated and selective approaches. *American Journal of Psychiatry*. doi: 10.1176/appi.ajp.2014.13050697

Scott, S., Briskman, J., Woolgar, M., Humayun, S., & O'Connor, T. G. (2011). Attachment in adolescence: overlap with parenting and unique prediction of behavioural adjustment. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *52*(10), 1052–1062. doi: 10.1111/j.1469-7610.2011.02453.x

Scott, S., & O'Connor, T. G. (2012). An experimental test of differential susceptibility to parenting among emotionally-dysregulated children in a randomized controlled trial for oppositional behavior. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *53*(11), 1184–1193. doi: 10.1111/j.1469-7610.2012.02586.x

Seeman, T., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: Cumulative allostatic load. *Ann N Y Acad Sci*, *1186*, 223–239. doi: 10.1111/j.1749-6632.2009.05341.x

Seisenberger, S., Peat, J. R., Hore, T. A., Santos, F., Dean, W., & Reik, W. (2013). Reprogramming DNA methylation in the mammalian life cycle: Building and breaking epigenetic barriers. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *368*(1609), 20110330. doi: 10.1098/rstb.2011.0330

Seok, J., Warren, H. S., Cuenca, A. G., Mindrinos, M. N., Baker, H. V., Xu, W., … Tompkins, R. G. (2013). Genomic responses in mouse models poorly mimic human inflammatory diseases. *Proceedings of the National Academy of Sciences of the United States of America*. doi: 10.1073/pnas.1222878110

Sharma, A., Dorman, M. F., & Spahr, A. J. (2002). A sensitive period for the development of the central auditory system in children with cochlear implants: Implications for age of implantation. *Ear and Hearing*, *23*(6), 532–539. doi: 10.1097/01.AUD.0000042223.62381.01

Sheridan, J. F., Stark, J. L., Avitsur, R., & Padgett, D. A. (2000). Social disruption, immunity, and susceptibility to viral infection. *Role of glucocorticoid insensitivity and NGF. Annals of the New York Academy of Sciences*, *917*, 894–905.

Sheridan, M. A., Fox, N. A., Zeanah, C. H., McLaughlin, K. A., & Nelson, C. A., III., (2012). Variation in neural development as a result of exposure to institutionalization early in childhood. *Proceedings of*

the National Academy of Sciences of the United States of America, *109*(32), 12927–12932. doi: 10.1073/pnas.1200041109

Short, S. J., Lubach, G. R., Karasin, A. I., Olsen, C. W., Styner, M., Knickmeyer, R. C., … Coe, C. L. (2010). Maternal influenza infection during pregnancy impacts postnatal brain development in the rhesus monkey. *Biological Psychiatry*, *67*(10), 965–973. doi: 10.1016/j.biopsych.2009.11.026

Silveira, P. P., Portella, A. K., Goldani, M. Z., & Barbieri, M. A. (2007). Developmental origins of health and disease (DOHaD). *Jornal de pediatria*, *83*(6), 494–504. doi: 10.2223/JPED.1728

Sirois, S., Spratling, M., Thomas, M. S., Westermann, G., Mareschal, D., & Johnson, M. H. (2008). Precis of neuroconstructivism: How the brain constructs cognition. *Behavioral and Brain Sciences*, *31*(3), 321–331; discussion 331–356. doi: 10.1017/S0140525X0800407X

Smits, P. A., & Denis, J. L. (2014). How research funding agencies support science integration into policy and practice: An international overview. *Implementation Science: IS*, *9*, 28. doi: 10.1186/1748-5908-9-28

Smyke, A. T., Dumitrescu, A., & Zeanah, C. H. (2002). Attachment disturbances in young children. I: The continuum of caretaking casualty. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*(8), 972–982. doi: 10.1097/00004583-200208000-00016

Smyke, A. T., Zeanah, C. H., Fox, N. A., Nelson, C. A., & Guthrie, D. (2010). Placement in foster care enhances quality of attachment among young institutionalized children. *Child Development*, *81*(1), 212–223. doi: 10.1111/j.1467-8624.2009.01390.x

Sokal, E. M., Conjeevaram, H. S., Roberts, E. A., Alvarez, F., Bern, E. M., Goyens, P., … Hoofnagle, J. (1998). Interferon alfa therapy for chronic hepatitis B in children: a multinational randomized controlled trial. *Gastroenterology*, *114*(5), 988–995. doi: S0016508598004375

Sontag, L. W. (1941). The significance of fetal environmental differences. *American Journal of Obstetrics and Gynecology*, *42*, 996–1003.

Speirs, H. J., Seckl, J. R., & Brown, R. W. (2004). Ontogeny of glucocorticoid receptor and 11beta-hydroxysteroid dehydrogenase type-1 gene expression identifies potential critical periods of glucocorticoid susceptibility during development. *Journal of Endocrinology*, *181*(1), 105–116.

Spitz, R. A. (1945). Hospitalism; an inquiry into the genesis of psychiatric conditions in early childhood. *Psychoanalytic Study of the Child*, *1*, 53–74.

Sroufe, L. A., Egeland, B., & Kreutzer, T. (1990). The fate of early experience following developmental change: Longitudinal approaches to individual adaptation in childhood. *Child Development*, *61*(5), 1363–1373.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, *55*(1), 17–29.

Steele, M., Steele, H., & Johansson, M. (2002). Maternal predictors of children's social cognition: an attachment perspective. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *43*(7), 861–872.

Stein, A. D., Pierik, F. H., Verrips, G. H., Susser, E. S., & Lumey, L. H. (2009). Maternal exposure to the Dutch famine before conception and during pregnancy: quality of life and depressive symptoms in adult offspring. *Epidemiology*, *20*(6), 909–915. doi: 10.1097/EDE.0b013e3181b5f227

Suomi, S. J., Harlow, H. F., & McKinney, W. T., Jr., (1972). Monkey psychiatrists. *American Journal of Psychiatry*, *128*(8), 927–932.

Sur, M., & Rubenstein, J. L. (2005). Patterning and plasticity of the cerebral cortex. *Science*, *310*(5749), 805–810. doi: 10.1126/science.1112070

Suzuki, K., Sugihara, G., Ouchi, Y., Nakamura, K., Futatsubashi, M., Takebayashi, K., … Mori, N. (2013). Microglial activation in young adults with autism spectrum disorder. *JAMA Psychiatry*, *70*(1), 49–58. doi: 10.1001/jamapsychiatry.2013.272

Swanson, J. M., Entringer, S., Buss, C., & Wadhwa, P. D. (2009). Developmental origins of health and disease: Environmental exposures. *Seminars in Reproductive Medicine*, *27*(5), 391–402. doi: 10.1055/s-0029-1237427

Swedo, S. E., & Grant, P. J. (2005). Annotation: PANDAS: A model for human autoimmune disease. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *46*(3), 227–234. doi: 10.1111/j.1469-7610.2004.00386.x

Szyf, M., Weaver, I., & Meaney, M. (2007). Maternal care, the epigenome and phenotypic differences in behavior. *Reproductive Toxicology*, *24*(1), 9–19. doi: 10.1016/j.reprotox.2007.05.001

Talge, N. M., Neal, C., & Glover, V. (2007). Antenatal maternal stress and long-term effects on child neurodevelopment: How and why? *Journal of Child Psychology and Psychiatry*, *48*(3–4), 245–261. doi: 10.1111/j.1469-7610.2006.01714.x

Tau, G. Z., & Peterson, B. S. (2010). Normal development of brain circuits. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *35*(1), 147–168. doi: 10.1038/npp.2009.115

Taylor, S. E., Way, B. M., & Seeman, T. E. (2011). Early adversity and adult health outcomes. *Development and Psychopathology*, *23*(3), 939–954. doi: 10.1017/S0954579411000411

Tegethoff, M., Greene, N., Olsen, J., Schaffner, E., & Meinlschmidt, G. (2011). Stress during pregnancy and offspring pediatric disease: A National Cohort Study. *Environmental Health Perspectives*, *119*(11), 1647–1652. doi: 10.1289/ehp.1003253

Teicher, M. H., Anderson, C. M., & Polcari, A. (2012). Childhood maltreatment is associated with reduced volume in the hippocampal subfields CA3, dentate gyrus, and subiculum. *Proceedings of the National Academy of Sciences of the United States of America*, *109*(9), E563–572. doi: 10.1073/pnas.1115396109

Thompson-Schill, S. L., Ramscar, M., & Chrysikou, E. G. (2009). Cognition without control: When a little frontal lobe goes a long way. *Current Directions in Psychological Science*, *18*(5), 259–263. doi: 10.1111/j.1467-8721.2009.01648.x

Thompson, L. A., Fagan, J. F., & Fulker, D. W. (1991). Longitudinal prediction of specific cognitive abilities from infant novelty preference. *Child Development*, *62*(3), 530–538.

Thurner, S., Klimek, P., Szell, M., Duftschmid, G., Endel, G., Kautzky-Willer, A., & Kasper, D. C. (2013). Quantification of excess risk for diabetes for those born in times of hunger, in an entire population of a nation, across a century. *Proceedings of the National Academy of Sciences of the United States of America*, *110*(12), 4703–4707. doi: 10.1073/pnas.1215626110

Tiemeier, H., Velders, F. P., Szekely, E., Roza, S. J., Dieleman, G., Jaddoe, V. W., … Verhulst, F. C. (2012). The Generation R Study: A review of design, findings to date, and a study of the 5-HTTLPR by environmental interaction from fetal life onward. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(11), 1119–1135 e1117. doi: 10.1016/j.jaac.2012.08.021

Tofail, F., Hamadani, J. D., Mehrin, F., Ridout, D. A., Huda, S. N., & Grantham-McGregor, S. M. (2013). Psychosocial stimulation benefits development in nonanemic children but not in anemic, iron-deficient children. *Journal of Nutrition*, *143*(6), 885–893. doi: 10.3945/jn.112.160473

Tollenaar, M. S., Jansen, J., Beijers, R., Riksen-Walraven, J. M., & de Weerth, C. (2010). Cortisol in the first year of life: Normative

values and intra-individual variability. *Early Human Development*, *86*(1), 13–16. doi: 10.1016/j.earlhumdev.2009.12.003

Toth, S. L., Rogosch, F. A., Sturge-Apple, M., & Cicchetti, D. (2009). Maternal depression, children's attachment security, and representational development: An organizational perspective. *Child Dev*, *80*(1), 192–208. doi: 10.1111/j.1467-8624.2008.01254.x

Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., … Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and difficulties in emotion regulation. *Dev Sci*, *13*(1), 46–61. doi: 10.1111/j.1467-7687.2009.00852.x

Upton, K. J., & Sullivan, R. M. (2010). Defining age limits of the sensitive period for attachment learning in rat pups. *Developmental Psychobiology*, *52*(5), 453–464. doi: 10.1002/dev.20448

Urdinguio, R. G., Sanchez-Mut, J. V., & Esteller, M. (2009). Epigenetic mechanisms in neurological diseases: Genes, syndromes, and therapies. *Lancet Neurology*, *8*(11), 1056–1072. doi: 10.1016/S1474-4422(09)70262-5

Van den Bergh, B. R., Mennes, M., Oosterlaan, J., Stevens, V., Stiers, P., Marcoen, A., & Lagae, L. (2005). High antenatal maternal anxiety is related to impulsivity during performance on cognitive tasks in 14- and 15-year-olds. *Neurosci Biobehav Rev*, *29*(2), 259–269. doi: 10.1016/j.neubiorev.2004.10.010

van Eijk, L. T., Dorresteijn, M. J., Smits, P., van der Hoeven, J. G., Netea, M. G., & Pickkers, P. (2007). Gender differences in the innate immune response and vascular reactivity following the administration of endotoxin to human volunteers. *Critical Care Medicine*, *35*(6), 1464–1469. doi: 10.1097/01.CCM.0000266534.14262.E8

van Ijzendoorn, M. H., Belsky, J., & Bakermans-Kranenburg, M. J. (2012). Serotonin transporter genotype 5HTTLPR as a marker of differential susceptibility? A meta-analysis of child and adolescent gene-by-environment studies. *Translational Psychiatry*, *2*, e147. doi: 10.1038/tp.2012.73

Vanderwert, R. E., Marshall, P. J., Nelson, C. A., 3rd, Zeanah, C. H., & Fox, N. A. (2010). Timing of intervention affects brain electrical activity in children exposed to severe psychosocial neglect. *PLoS One*, *5*(7), e11415. doi: 10.1371/journal.pone.0011415

Virk, J., Li, J., Vestergaard, M., Obel, C., Lu, M., & Olsen, J. (2010). Early life disease programming during the preconception and prenatal period: Making the link between stressful life events and type-1 diabetes. *PLoS One*, *5*(7), e11523. doi: 10.1371/journal.pone.0011523

Virk, J., Obel, C., Li, J., & Olsen, J. (2014). In-utero exposure to bereavement and offspring IQ: A Danish national cohort study. *PLoS One*, *9*(2), e88477. doi: 10.1371/journal.pone.0088477

Vorria, P., Papaligoura, Z., Sarafidou, J., Kopakaki, M., Dunn, J., Van Ijzendoorn, M. H., & Kontopoulou, A. (2006). The development of adopted children after institutional care: A follow-up study. *Journal of Child Psychology and Psychiatry*, *47*(12), 1246–1253. doi: 10.1111/j.1469-7610.2006.01666.x

Vorria, P., Rutter, M., Pickles, A., Wolkind, S., & Hobsbaum, A. (1998). A comparative study of Greek children in long-term residential group care and in two-parent families: I. Social, emotional, and behavioural differences. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *39*(2), 225–236.

Waddington, C. H. (1959). Canalization of development and genetic assimilation of acquired characters. *Nature*, *183*(4676), 1654–1655.

Wallerstein, J. S. (1985). Children of divorce: Preliminary report of a ten-year follow-up of older children and adolescents. *Journal of the American Academy of Child Psychiatry*, *24*(5), 545–553.

Wang, Y., Wang, L., Zhao, J., & Qiao, Z. (2005). Estrogen, but not testosterone, down-regulates cytokine production in nicotine-induced murine macrophage. *Methods and Findings in Experimental and Clinical Pharmacology*, *27*(5), 311–316. doi: 10.1358/mf.2005.27.5.893666

Waters, E., Merrick, S., Treboux, D., Crowell, J., & Albersheim, L. (2000). Attachment security in infancy and early adulthood: A twenty-year longitudinal study. *Child Development*, *71*(3), 684–689.

Weaver, I. C., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., … Meaney, M. J. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience*, *7*(8), 847–854. doi: 10.1038/nn1276

Weaver, I. C., Champagne, F. A., Brown, S. E., Dymov, S., Sharma, S., Meaney, M. J., & Szyf, M. (2005). Reversal of maternal programming of stress responses in adult offspring through methyl supplementation: Altering epigenetic marking later in life. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *25*(47), 11045–11054. doi: 10.1523/JNEUROSCI.3652-05.2005

Webster-Stratton, C., Rinaldi, J., & Jamila, M. R. (2011). Long-term outcomes of incredible years parenting program: Predictors of adolescent adjustment. *Child and Adolescent Mental Health*, *16*(1), 38–46. doi: 10.1111/j.1475-3588.2010.00576.x

Weinfield, N. S., Sroufe, L. A., & Egeland, B. (2000). Attachment from infancy to early adulthood in a high-risk sample: Continuity, discontinuity, and their correlates. *Child Development*, *71*(3), 695–702.

Weiss, S. R., & Post, R. M. (1994). Caveats in the use of the kindling model of affective disorders. *Toxicology and Industrial Health*, *10*(4–5), 421–447.

Wilder, R. L., & Elenkov, I. J. (1999). Hormonal regulation of tumor necrosis factor-alpha, interleukin-12 and interleukin-10 production by activated macrophages. A disease-modifying mechanism in rheumatoid arthritis and systemic lupus erythematosus? *Annals of the New York Academy of Sciences*, *876*, 14–31.

Wilson, C. A., Schade, R., & Terry, A. V., Jr., (2012). Variable prenatal stress results in impairments of sustained attention and inhibitory response control in a 5-choice serial reaction time task in rats. *Neuroscience*, *218*, 126–137. doi: 10.1016/j.neuroscience.2012.05.040

Winnicott, D. W. (1965). *The maturational processes and the facilitating environment*. New York, NY: International Universities Press.

Wirth, M. M., Meier, E. A., Fredrickson, B. L., & Schultheiss, O. C. (2007). Relationship between salivary cortisol and progesterone levels in humans. *Biological Psychology*, *74*(1), 104–107. doi: 10.1016/j.biopsycho.2006.06.007

Wismer Fries, A. B., Ziegler, T. E., Kurian, J. R., Jacoris, S., & Pollak, S. D. (2005). Early experience in humans is associated with changes in neuropeptides critical for regulating social behavior. *Proceedings of the National Academy of Sciences of the United States of America*, *102*(47), 17237–17240. doi: 10.1073/pnas.0504767102

Wolf, S. A., Steiner, B., Akpinarli, A., Kammertoens, T., Nassenstein, C., Braun, A., … Kempermann, G. (2009). CD4-positive T lymphocytes provide a neuroimmunological link in the control of adult hippocampal neurogenesis. *Journal of Immunology*, *182*(7), 3979–3984. doi: 10.4049/jimmunol.0801218

Wolkind, S. N. (1974). The components of affectionless psychopathy in institutionalized children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *15*(3), 215–220.

Yehuda, R., Engel, S. M., Brand, S. R., Seckl, J., Marcus, S. M., & Berkowitz, G. S. (2005). Transgenerational effects of posttraumatic stress disorder in babies of mothers exposed to the World Trade

Center attacks during pregnancy. *J ournal of Clinical Endocrinology and Metabolism*, *90*(7), 4115–4118. doi: 10.1210/jc.2005-0550

Yirmiya, R., & Goshen, I. (2011). Immune modulation of learning, memory, neural plasticity and neurogenesis. *Brain, Behavior, and Immunity*, *25*(2), 181–213. doi: 10.1016/j.bbi.2010.10.015

Yoshikawa, H. (1995). Long-term effects of early childhood programs on social outcomes and delinquency. *The Future of Children/Center for the Future of Children, the David and Lucile Packard Foundation*, *5*(3), 51–75.

Young, L. D., Suomi, S. S., Harlow, H. F., & McKinney, W. T., Jr., (1973). Early stress and later response to separation in rhesus monkeys. *American Journal of Psychiatry*, *130*(4), 400–405.

Young, L. J., Wang, Z., & Insel, T. R. (1998). Neuroendocrine bases of monogamy. *Trends in Neurosciences*, *21*(2), 71–75.

Zeanah, C. H., Gunnar, M. R., McCall, R. B., Kreppner, J. M., & Fox, N. A. (2011). VI. Sensitive Periods. *Monographs of the Society for Research in Child Development*, *76*(4), 147–162. doi: 10.1111/j.1540-5834.2011.00631.x

Zeanah, C. H., Smyke, A. T., & Dumitrescu, A. (2002). Attachment disturbances in young children. II: Indiscriminate behavior and institutional care. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*(8), 983–989. doi: 10.1097/00004583-200208000-00017

Zeanah, C. H., Smyke, A. T., Koga, S. F., & Carlson, E. (2005). Attachment in institutionalized and community children in Romania. *Child Development*, *76*(5), 1015–1028. doi: 10.1111/j.1467-8624.2005.00894.x

Zohsel, K., Buchmann, A. F., Blomeyer, D., Hohm, E., Schmidt, M. H., Esser, G., … Laucht, M. (2014). Mothers' prenatal stress and their children's antisocial outcomes–a moderating role for the Dopamine D4 Receptor (DRD4) gene. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *55*(1), 69–76. doi: 10.1111/jcpp.12138

Zuena, A. R., Mairesse, J., Casolini, P., Cinque, C., Alema, G. S., Morley-Fletcher, S., … Maccari, S. (2008). Prenatal restraint stress generates two distinct behavioral and neurochemical profiles in male and female rats. *PLoS One*, *3*(5), e2170. doi: 10.1371/journal.pone.0002170

CHAPTER 6

Emotional Security Theory and Developmental Psychopathology

PATRICK T. DAVIES, MEREDITH J. MARTIN, and MELISSA L. STURGE-APPLE

THE SUBSTANCE OF EMOTIONAL SECURITY IN HISTORICAL PERSPECTIVE 201
The Goal-Corrected System of Emotional Security in EST 201
Conceptual Pitfalls and Barriers in the EST Conceptualization of Emotional Security 203
Advances in the Characterization of the Emotional Security System in EST-R 204
EMOTIONAL SECURITY AS A MEDIATOR OF CHILDREN'S ADJUSTMENT TO INTERPARENTAL CONFLICT 209
Research on the Mediational Role of Emotional Security 209
Interparental Sources of Children's Emotional Insecurity 214
The Potency of Signs of Emotional Insecurity in the Mediational Pathways 216
The Multitude of Sequelae of Children's Insecurity 217
Developmental Cascade Mechanisms 217
CONTEXTUAL CHARACTERISTICS 223
Family Processes 224
Child Characteristics 229
Ecological Characteristics 232
DEVELOPMENTAL PARAMETERS 235
Transactions of Insecurity 236
Experiential Histories 237
Sensitive Periods 239
Developmental Pathways and Trajectories 241
FUTURE DIRECTIONS 243
Person-Based Taxonomy 243
Expanding Emotional Security Beyond the Interparental Dyad 246
Protection and Resilience in the Face of Insecurity 247
Prevention, Intervention, and Public Policy 249
CONCLUSIONS 251
REFERENCES 252

As a cornerstone of the family, relationship dynamics between parents are highly significant in the lives of children (Amato, 2000). Although some forms of interparental conflict and discord (e.g., resolution) may have benign or salubrious consequences for children, destructive ways of managing conflict pose a substantial public health concern by virtue of their prevalence and risk for child bystanders. Dissolution of interparental relationships is a common occurrence, with over 30% of children experiencing disruption in their parents' relationships by nine years of age (Kennedy & Bumpass, 2008). Moreover, conflict and turmoil between parents often accompanies the family transition (Birditt, Brown, Orbuch, & McIlvane, 2010). Even among parents who remain together, rates of discord and violence are relatively high. Between 20% and 40% of parents in intact dyads experience clinically significant levels of relationship distress (Cummings & Davies, 1994). The proportion of parents who report experiencing physical aggression from a romantic partner within the last year is estimated to be as high as 49% (Slep & O'Leary, 2005). Exposure to high levels of discord and conflict, in turn, has been repeatedly documented to increase children's risk for a wide array of psychological problems including internalizing symptoms (e.g., depression, anxiety), externalizing problems (e.g., aggression, conduct problems), social impairments (e.g., poor peer relations), and academic difficulties (Dunn & Davies, 2001; Harold & Leve, 2012). Moreover, the magnitude of vulnerability experienced by children increases considerably in contexts of interparental aggression and violence (Evans, Davies, & DiLillo, 2008; Kitzmann, Gaylord, Holt, & Kenny 2003).

In drawing on developmental psychopathology as a guide toward better understanding the risk faced by children from high conflict homes, emotional security theory was initially developed to address precisely how, when,

In preparing the chapter, Patrick Davies was supported by the James McKeen Cattell Fund Sabbatical Fellowship.

[1]Color versions of Figure 6.5 are available at http://onlinelibrary.wiley.com/book/10.1002/9781119125556

and why interparental discord is associated with children's trajectories of mental health (Davies & Cummings, 1994). Because witnessing hostile and unresolved conflicts are emotionally arousing and threatening events for children, mounting and regulating emotional responses are regarded as central processes in understanding how children adapt to interparental conflict. In these contexts, preserving emotional security is an important goal that organizes children's emotional experiences (e.g., fear), action tendencies (e.g., withdrawal, intervention), and appraisals of themselves and their families. Thus, a primary thesis of the theory is that witnessing destructive interparental conflict increases children's vulnerability to psychological problems by undermining their ability to preserve emotional security within various family relationships.

In this chapter, our overarching objective is to provide a review and analysis of emotional security theory. Within the developmental psychopathology perspective (Cicchetti & Toth, 2009), the risk posed by interparental conflict is specifically conceptualized as a manifestation of a vibrant undercurrent of evolving exchanges between the dynamic child in a shifting, multilayered constellation of biopsychosocial liabilities and resources in the family. Thus, the developmental course of each child is to some degree unique due to the specific transactions between their prior and current experiences in family contexts and their own attributes, resources, and histories of adaptation. The derivative assumption, then, is that changes in child adjustment can be lawfully predicted and explained by elucidating the nature of transactions between the child and environment.

To organize and represent the inherent complexity of a developmental psychopathology approach, Figure 6.1 offers a framework for the primary substantive themes and pathways that we will cover in the chapter. Throughout the chapter, the main principles of developmental psychopathology will serve as a template for addressing important theoretical and research directions in characterizing associations between interparental conflict and child functioning. Consistent with Path #1 in the figure, the first part of the paper reviews the conceptualization of emotional security within a historical perspective, with a focus on highlighting the components of the system that distinguish it from other prevailing family process models. Next, as a core theoretical assumption of EST, we examine the hypothesis that interparental conflict, in certain forms, increases children's risk for psychopathology by magnifying their concerns about security interparental

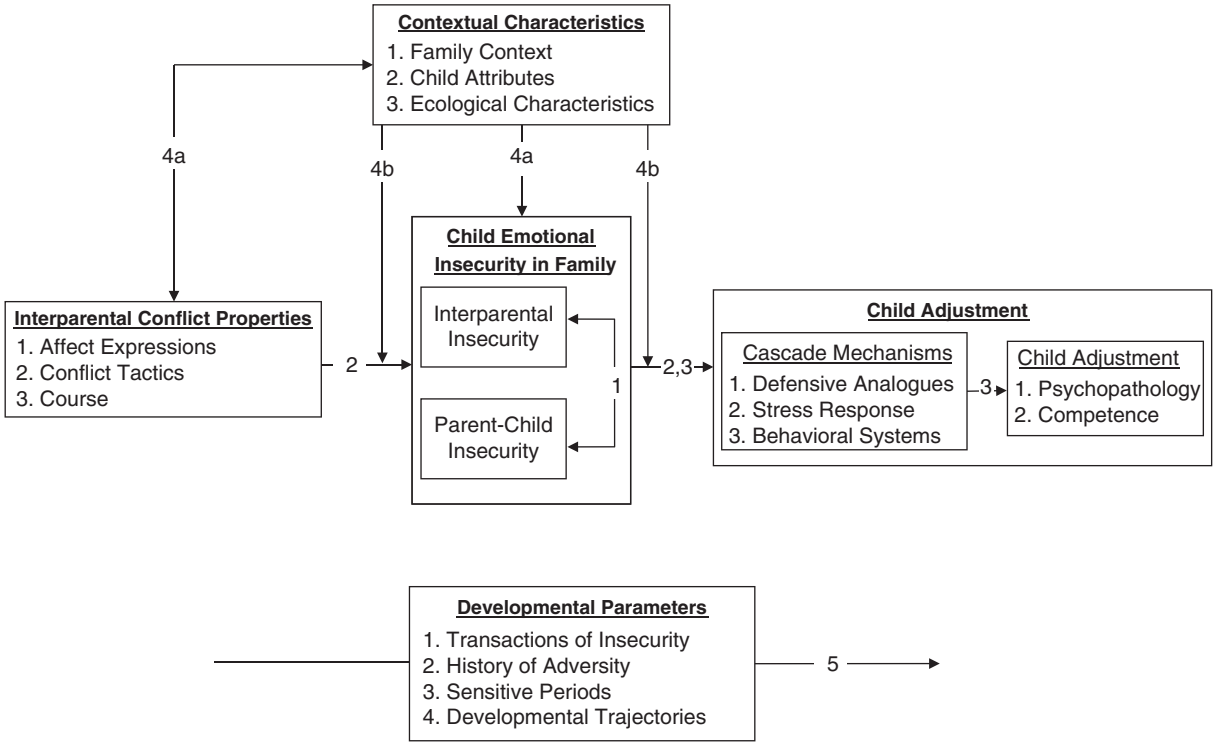


Figure 6.1 Key pathways and processes in emotional security theory that are proposed to inform an understanding of children's heightened vulnerability to interparental conflict.

relationship (see #2 Paths in Figure 6.1). Delving deeper into understanding the multitude of adjustment trajectories of children from high conflict homes, we then explore the developmental processes that are proposed to explain why emotional insecurity increases the likelihood of psychopathology (see #3 Paths in Figure 6.1). The fourth part of the paper illustrates how embedding the study of emotional security in the interparental relationship within the broader contextual landscape can further account for the diverse pathways experienced by children from high conflict homes (see #4 Paths in Figure 6.1). As the final component of our conceptual model illustrated by Path 5 in Figure 6.1, we examine how a fuller appreciation of developmental concepts in developmental psychopathology offers a more nuanced view of the conditions and processes underlying children's adaptation to interparental conflict. Finally, our concluding section of the paper selectively describes the translational implications and future directions of EST.

Throughout the chapter, we distinguish between two primary versions of emotional security theory to adequately characterize the significant conceptual developments in the history of the theory. The original emotional security theory, which we designate with the acronym EST, was first developed by Davies and Cummings (1994) and subsequently refined in several papers (e.g., Cummings & Davies, 1996; 2010; Davies, Harold, Goeke-Morey, & Cummings, 2002). In response to several barriers to advancing EST, we then developed a reformulation of emotional security theory (EST-R; Davies & Martin, 2013; Davies & Sturge-Apple, 2007; Davies, Sturge-Apple, & Martin, 2013). Although EST-R accepts many of the principles of EST, it is designed to provide greater theoretical depth, precision, and novelty by utilizing evolutionary and ethological frameworks as guides. Thus, whenever applicable in the chapter, we will highlight the primary distinctions between the EST and EST-R and any resulting differences in hypotheses and approaches. As a first step in this endeavor, we now turn to providing a historical account of the conceptual developments in EST over the last two decades.

THE SUBSTANCE OF EMOTIONAL SECURITY IN HISTORICAL PERSPECTIVE

Emotional security theory is built, in part, on an enduring conceptual foundation of earlier models of security in the parent–child relationship. As described by Ainsworth and Bowlby (1991), decades of laborious theoretical and empirical work culminated in groundbreaking advances in understanding the significance of relationship security in models of typical and atypical child development during the 1960s and 1970s (e.g., Ainsworth, Blehar, Waters, & Wall, 1978; Blatz, 1966; Bowlby, 1969; 1973). As a primary conceptual product of this progress, attachment theory has continued to be highly influential in understanding the developmental precursors, composition, and implications of individual differences in parent–child relationship security. Consistent with attachment theory, EST shares the assumption that histories of family interactions have a long-term impact on children's development by setting the stage for differences in children's ability to achieve safety and security in close family relationships. Accordingly, EST accepts the hypothesis that security in parent–child relationships serve as key explanatory mechanisms between child-rearing practices of socialization agents (e.g., sensitivity, responsiveness) and children's developmental outcomes (Bretherton, Ridgeway, & Cassidy, 1990; Thompson, 2000).

However, commonality between the theories begs the question of what differentiates EST from attachment security. A fundamental source of distinction between the theories lies in the novel EST hypothesis that a broader array of family characteristics may shape developmental trajectories by altering children's security in multiple family relationships. Because EST was originally developed to explain the developmental sequelae of interparental relationship dynamics, conflict between parents assumed center stage as a contextual factor that may enhance or undermine children's emotional security in family relationships. Although this premise generates several hypotheses regarding the interplay between interparental conflict and children's attachment security in parent–child relationships (see the "Parent–child relationship processes" section for more details), originality in EST is rooted in its proposition that children also develop a relatively distinct sense of emotional security in the interparental relationship. Thus, as shown by Path #1 of Figure 6.1, EST maintains that the unique composition of emotional security cannot simply be reduced into attachment security. Of particular relevance to developmental psychopathology, the distinctive operation of emotional security is theorized to provide incremental leverage in predicting children's trajectories of psychological adjustment in models of parent–child relationship processes.

The Goal-Corrected System of Emotional Security in EST

Toward the goal of delineating similarities and differences between the theories, early conceptual efforts in EST

focused on characterizing how the emotional security system operates in the context of interparental conflict (e.g., Cummings & Davies, 1996; Davies & Cummings, 1994; Davies, Harold, et al., 2002). According to EST, emotional security is a latent goal that is inferred from three observable classes of response processes that are commonly referred to as component processes. As illustrated in Figure 6.2, difficulties preserving security are reflected in the component processes of (1) emotional reactivity, characterized by intense, prolonged, and dysregulated distress (e.g., fear, vigilance) reactions to interparental conflict; (2) regulation of exposure to parent affect, as evidenced by protracted, rigid attempts to become involved in or avoid parental conflicts; and (3) internal representations of interparental relations, as indexed by children's evaluations of the potential consequences parental conflicts have for their well-being. Although each of these responses is hypothesized to serve as a measurable sign of insecurity in the interparental relationship, EST follows the principles of the organizational perspective in developmental psychopathology by proposing that each response process is part of a complex constellation of responses (e.g., Cicchetti & Rogosch, 2002). Thus, deciphering the meaning of any one domain of responding is advanced by examining it within the broader organization of insecure signs of reactivity to conflict.

Guided by developmental psychopathology, the integration of an organizational perspective within EST is instantiated in the model of emotional security as a goal-corrected system. In this behavioral system, preserving emotional security in the interparental relationship is specifically conceptualized as a latent goal that regulates and is regulated by the three component processes (Cummings & Davies, 1996; Davies & Cummings, 1994). Reflecting one part of this transactional feedback process, threats to the goal of emotional security triggered by hostility and conflict between parents organizes children's heightened emotional reactivity, forms of reducing exposure to parent affect, and negative representations of the consequences conflict has for their welfare. Likewise, in illustrating the other component of the reciprocal interplay, the three classes of response processes are designed to regulate children's sense of security. First, the fearful, vigilant forms of emotional reactivity are theorized to serve several protective functions of the goal system, including (1) priming children to rapidly identify and process potential threats, (2) increasing accessibility to psychophysiological resources necessary to mount an effective response to threatening stimuli, and (3) facilitating acquisition and prompt recall of aversive interparental cues in scanning subsequent interparental conflicts (Davies, Forman, Rasi, & Stevens, 2002; Davies, Harold, et al., 2002). Second, as

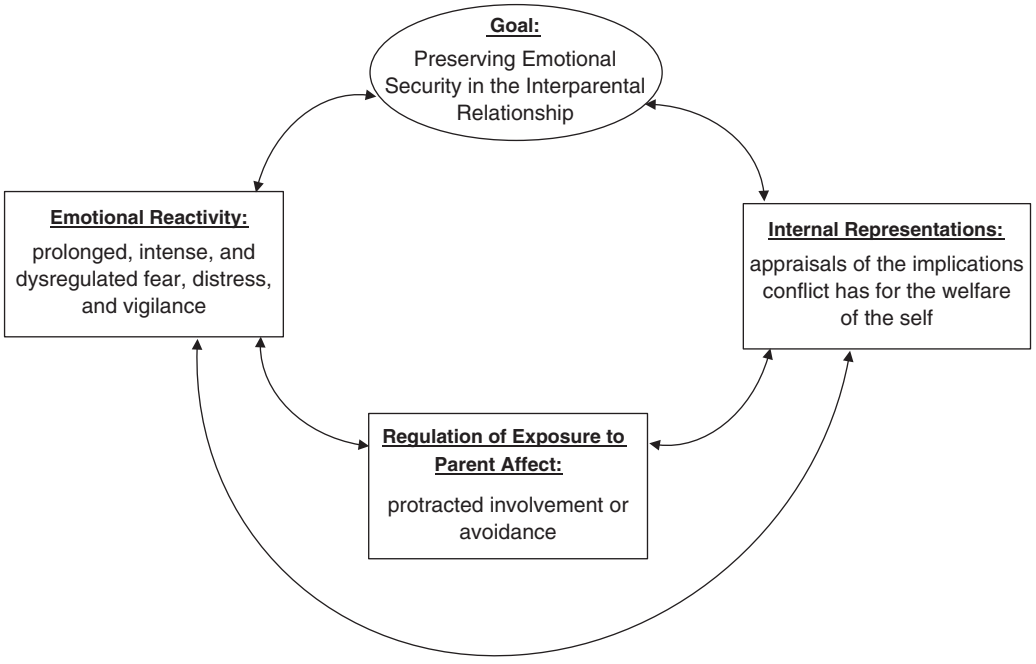


Figure 6.2 A graphical depiction of the bidirectional associations between the latent goal of preserving emotional security and the three component processes of emotional reactivity, regulation of exposure to parent affect, and internal representations.

ways of regulating parent affect, intervention and avoidance are theorized to serve direct protective functions of attenuating children's exposure to the threatening characteristics of interparental conflict. For example, although involvement in conflicts may run the risk of increasing children's initial exposure to interparental difficulties, the underlying attempts to regulate the course of conflict may provide them with a means to modulate parental emotionality and behaviors before they escalate and result in more serious consequences for the family (Cummings & Davies, 1996). Third, in reflecting ways children process the interpersonal meaning and consequences of the ways parents manage conflicts, internal representations may prepare children for potential danger by providing them with templates for scanning, understanding, and responding quickly to imminent interparental threats (Davies, Harold, et al., 2002).

As illustrated by the bidirectional arrows linking each of the three component processes together in Figure 6.2, the original formulation of EST hypothesizes that indicators within and across the three component processes are interdependent in reflecting the superordinate goal of preserving emotional security. Based on the assumptions of interdependency in the security system, a central hypothesis is that the magnitude of associations among the multiple signs of insecurity will generally fall in the moderate range. In accord with this prediction, research has generally reported moderate empirical overlap. For example, in a review of fifteen studies, Davies and Martin (2013) concluded that the mean overlap of response processes proposed to serve the goal of emotional security was approximately 17% ($r = .41$).

Conceptual Pitfalls and Barriers in the EST Conceptualization of Emotional Security

Although EST provided initial guidance in describing the composition and operation of the emotional security system (Davies, Harold, et al., 2002), it still lacks the clarity and richness necessary to fully characterize how the security system operates. Signs of nagging imprecision in conceptual and operational definitions are specifically manifested in considerable inconsistency between studies in the measurement of emotional insecurity in the interparental relationship. At the extreme, problems grasping the conceptual definition of emotional insecurity in the literature are evident in assessments of general behavioral or response styles across multiple contexts that more closely resemble personality and temperament traits (e.g., neuroticism, mood). For example, a prior study utilized self-report measures of "poor regulation of affect (e.g., 'I lose my temper at the slightest offense'); behavior indicating insecurity in a broad array of close relationships (e.g., 'I find myself clinging to people I'm close to because I'm afraid they'll leave me'); and negative expectances about the stability of relationships (e.g., 'I don't feel that important relationships will last. I expect them to end.')" (Mann & Gilliom, 2004, pp. 258–259). Likewise, Su, Simons, and Simons (2011) purported to provide a test of emotional security theory by examining the mediational role of children's generalized anxiety disorder symptomatology as a mediator in the link between interparental aggression and their antisocial behavior problems. In both examples, the use of trait-like assessments is tapping constructs that expand well beyond the circumscribed focus in EST on children's response patterns in the specific relational context of the interparental subsystem. Thus, using inhibited temperament or neurotic personality indices as proxies of emotional security is akin to empirically disconfirmed notions that patterns of parent–child attachment security are reducible to child temperamental attributes (see Sroufe, 1985).

Other scholars view emotional security as a relational construct rather than a trait-like attribute, but experience difficulties in conveying how emotional security differs from parent–child attachment. For example, Crockenberg and Langrock's (2001) description of emotional security as "the belief that one is loved and cared for" (p. 139) closely resembles a dimension of parent–child relationship quality. Likewise, Rothbaum, Rosen, Ujie, and Uchida (2002) described EST as postulating that "child wellbeing is fostered when children have a secure model of the parents' relationship. That is, children's security may depend in part on parents' attachment to one another" (p. 338). In fact, a need for greater clarity in disentangling the two constructs is even apparent in the descriptions of emotional security offered by the original developers of EST. For example, emotional security in the interparental relationship has been portrayed as serving as a "secure base" for children and a metaphorical "bridge between the child and the world" (Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006, p. 134; see also El-Sheikh, Cummings, Kouros, Elmore-Staton, & Buckhalt, 2008). Thus, it is not uncommon for emotional security to be characterized as closely related to, if not synonymous with, attachment security.

Even when empirical efforts have focused more precisely on children's responses to interparental conflict, the relatively vague characterization in EST of children's emotional security in the interparental relationship has generated considerable inconsistencies in the identification of signs of

insecurity. At the one extreme, emotional reactivity has been defined in very broad terms as an aggregation of multiple forms of negative affect and diminished positive affect (e.g., Cummings, Goeke-Morey, Papp, & Dukewich, 2002; Du Rocher Schudlich & Cummings, 2007; Goeke-Morey, Cummings, Harold, & Shelton, 2003). At another extreme, other research has utilized narrower definitions of emotional reactivity as consisting specifically of fearful distress reactions to conflict (e.g., Davies, Cicchetti, & Martin, 2012). In falling between the two poles of this wide continuum of emotional reactivity definitions, other studies have targeted emotional reactivity constructs through indices of relatively global emotional distress in which fear, sadness, and anger were each given relatively equal weight as signs of insecurity (Crockenberg & Langrock, 2001; Davies & Cummings, 1998). To resolve the multiple problems in understanding and assessing emotional security in EST, EST-R was introduced to provide a deeper portrayal of the properties of the security system (Davies & Martin, 2013; Davies & Sturge-Apple, 2007; Davies & Woitach, 2008). Thus, as part of our historical account, the next section provides a synopsis of how the recent reformulation of the theory offers a novel characterization of how the goal-corrected system of security operates.

Advances in the Characterization of the Emotional Security System in EST-R

Consistent with the interdisciplinary nature of developmental psychopathology (Cicchetti & Toth, 2009), EST-R was designed to provide a richer characterization of the properties of the emotional security system based on a growing corpus of psychobiological and animal research in ethological and evolutionary frameworks (Davies & Sturge-Apple, 2007; Davies, Sturge-Apple, & Martin, 2013). According to ethological and evolutionary frameworks, distinct control systems or ethological modules of responding to environmental cues are postulated to have emerged and persisted throughout phylogeny based on their success in promoting survival and reproduction within developmental and ecological niches in our evolutionary past. Therefore, the behavioral and mental processes of all, or virtually all, individuals within a species should be organized in relation to these modules. Each module or system is characterized by an integrated set of processing algorithms, neurobiological processes, and psychological and behavioral repertoires that increase in salience as organizers of functioning when contemporaneous environmental input corresponds with the selective pressures and conditions of ancestral environments (Konner, 2010).

Ethological Differentiation of Emotional Security and Parent–Child Security

Building on this conceptual foundation in ethology, a fundamental premise of EST-R is that children's security in the interparental and parent–child relationships is largely regulated by two different ethological modules: the social defense (SDS) and attachment systems. Table 6.1 provides a comparison of the two types of security based on the characteristics of each of these ethological modules. As depicted in the table, the two systems share the broad adaptive function of protection from harm. However, the security systems organized by SDS and attachment are

TABLE 6.1 Comparison of Security in the Interparental and Parent-Child Relationships Along Ethological Parameters

	Security in the interparental relationship	Parent–child relationship security
Control system	Social defense system	Attachment system
Broad adaptive function	Protection from harm	Protection from harm
External/observed goal	Defuse or avoid threats by conspecifics	Maximize sensitivity and protection of caregiver
Action tendencies	Vigilance, heightened perceptual-cognitive sensitivity to environmental signs of danger, fear, distress, freezing, flight strategies, cut off behaviors (e.g., covering eyes), camouflaging behaviors (e.g., inhibiting verbal and overt emotional expressions; concealing face), long-term demobilization (i.e., dysphoria, vegetative state, fatigue, inferiority, hopelessness, and helplessness), social de-escalation strategies (e.g., appeasing, submissive disengagement, coy or ingratiating behavior), affected behavior (e.g., demonstrative display of distress)	Distress; bids for comfort and support; proximity-seeking; clinging behavior; monitor whereabouts of caregiver
Conditions and environmental cues	Frightening (e.g., hostility, yelling, aggression) and frightened behavior by parents or attachment figures in the family	Internal (e.g., fatigue, sickness) or external (e.g., aloneness, darkness, strangers, novel objects or settings) cues to danger that do not originate with the parental or attachment figure

postulated to be fundamentally different from each other based on (1) distinct external goals, (2) unique conditions that increase their saliency as organizers of behavior, and (3) action tendencies that regulate their external goals. In accord with broader evolutionary frameworks, a primary assumption guiding the delineation of each system is that its external goal is defined by its original, specific function in regulating the ongoing relationship between organism and environment in ways that ultimately contributed to survival and reproduction within ancestral environments (Hilburn-Cobb, 2004). As the primary organizer of security in the interparental relationship, EST-R proposes that the external goal of the SDS system is avoiding or defusing threat from hostile conspecifics. Underlying this proposal is the assumption that the high cost of conflict between members of social groups throughout our phylogenetic history put selective pressure on the development of a behavioral system (i.e., SDS) capable of efficiently identifying social signals indicative of the potential threat (e.g., yelling, dominant posturing) and responding by organizing behavioral strategies to defend against threat (Davies & Sturge-Apple, 2007). Despite obvious differences between the family structures of today and those of our evolutionary past (e.g., extended family units, clans), most, if not all contemporary families are headed by dominant adults who carry disproportionate power in shaping the structure, stability, and safety of the family unit. Because relationship difficulties between these adults can have threatening implications for children and the family as a whole, the SDS and its goal of protecting oneself from interpersonal harm is posited to be a central source of children's response patterns to interparental conflict. Thus, the environmental cues and conditions that increase the salience of the social defense system are mainly composed of displays of frightened or frightening behavior by caregivers.

Conversely, efforts to preserve security in the parent–child relationship are largely regulated by the attachment system and its behavioral goal of maximizing the sensitivity and protection of caregivers in times of distress of threat (Bowlby, 1969). Ethological perspectives on attachment theory within the developmental literature propose that the attachment system evolved out of an evolutionary history in which ecological conditions were characterized by a high risk of infant mortality (e.g., fatal injury in perilous living conditions, predation, infanticide). In these contexts, inclusive fitness increased for those individuals who successfully adopted a strategy of maintaining proximity to and garnering protection from a primary caregiver (Bowlby, 1969; Chisholm, 1996). Consequently, circumstances that activate the attachment system are largely proposed to be different from those that trigger the SDS. In particular, attachment system is viewed as increasing in salience under conditions of internal (e.g., fatigue, sickness) or external (e.g., aloneness, darkness, interpersonal discord, novel people, stimuli, and settings) cues to potential danger or threat. Although interpersonal discord is regarded as a cue that increases the salience of the attachment system, it is only likely to be the primary organizer of children's behavior when attachment figures are physically and psychologically accessible. Thus, increasing proximity to caregivers is unlikely to be the primary external goal underlying children's behavior when the attachment figures are otherwise unavailable by virtue of their engagement in conflict. In most cases, the displays of frightened and frightening behaviors by combatant parents during conflicts will likely inhibit children's motivation to approach and seek support. Under these conditions, EST-R posits that protecting oneself from harm should be prioritized as a predominating goal, manifested in the relatively high salience of SDS as an organizer of children's responses to interparental conflict.

Due to the latent and unobservable nature of the external goals, evaluation of the degree to which the SDS is prioritized in stimulating reaction patterns requires an analysis of the action tendencies that serve each ethological module. For the attachment system, clinging, seeking comfort, verbal bids for support, behavioral signals of distress, and regular monitoring of the whereabouts of the parent are all potential means toward increasing accessibility to a supportive caregiver (Bowlby, 1969; Hilburn-Cobb, 2004). Conversely, minimizing threat accompanying interparental conflict requires that information-processing capacities be predominantly geared toward monitoring the aversive stimuli rather than divided between danger and attachment figure cues. Thus, SDS, at its core, is manifested in increased vigilance or, more specifically, heightened perceptual-cognitive sensitivity to the interparental conflict. Moreover, consistent with the organizational perspective in developmental psychopathology (Davies & Cicchetti, 2004), regulating exposure to the diverse properties and changing course of interparental conflicts can be flexibly achieved through the enlistment of a number of action tendencies and behavioral strategies. Efforts to safeguard against interpersonal harm may be expressed through freezing, cutoff behaviors (e.g., covering eyes), or camouflaging tendencies (e.g., inhibiting verbal and overt emotional expressions; concealing face) in some developmental contexts. Under other conditions, the goal may be achieved through response tendencies that include social de-escalation strategies (e.g., submissive disengagement,

coy behavior, ingratiation), long-term demobilization (e.g., dysphoria, inferiority, hopelessness, helplessness), or affected responses characterized by demonstrative displays of vulnerability. In illustrating the other side of the feedback loop in the goal system, the enlistment of specific configurations of these action tendencies are designed to facilitate the goal of defusing threat.

At this early stage of theoretical development, research has yet to systematically distinguish between the predominant action tendencies of the two ethological systems in order to definitively test whether the SDS is prioritized over attachment system in contexts of interparental conflict. However, trained coders of maternal narratives of toddler responses as bystanders to angry expressions by family members revealed that concerned facial expressions were one of the most consistent ways of responding to both naturally occurring (19%) and simulated anger (20%) incidents, far exceeding the incidence of attachment behaviors such as pick up bids (3% to 6%) (Cummings, Zahn-Waxler, & Radke-Yarrow, 1981). Likewise, maternal and paternal diary reports of preadolescent and early adolescent reactions to interparental conflict in the home revealed a high incidence of frightened or sad responses (18–28%) and avoidance (25–29%). Limited assessments of distinctive attachment behaviors do not allow for a thorough comparison of the relative propensity of interparental conflict to activate SDS and attachment systems. However, in support of EST-R, the distress and avoidance behaviors proposed to serve the SDS increased exponentially for children with histories of exposure to more threatening forms of interparental conflict characterized by violence. For example, children who witnessed violence between parents were two to four times more likely to exhibit fearful, sad, or avoidant reactions than were their counterparts from nonviolent homes (Garcia O'Hearn, Margolin, & John, 1997).

Differences in the Conceptualization of Emotional Security Across EST-R and EST

EST and EST-R differ in how they conceptualize emotional security in the interparental relationship. Without any clearly articulated external goal beyond the colloquial term of *security*, the definition of insecurity in EST has remained underdeveloped. As a case in point, Crockenberg and Langrock (2001) noted that emotional insecurity in EST is largely defined by "general emotional distress . . . [and] the nature of the negative emotion is of no significance to the model" (p. 140). Due the lack of a more precise characterization of the affective underpinnings of emotional security in EST, assessments of security have also been stretched into broad affective constructs reflecting that the degree to which an array of negative (i.e., angry, scared, sad) emotional responses outweigh positive (i.e., happy) emotional responses (e.g., Cummings et al., 2002; Du Rocher Schudlich & Cummings, 2007; Goeke-Morey et al., 2003).

In contrast, the external goal of defending against interpersonal threat in EST-R translates to a more circumscribed definition of insecurity that is organized around the defensive, inhibitory system. Thus, the presence of positive affect in EST-R is not viewed as an unconditional sign of greater security. Rather, the locus of evaluation is centered on the intensity and salience of heightened vigilance and perceptual sensitivity to signs of threat in the interparerntal relationship. Notably, narrower definitions of security as reflecting the absence of efforts to defend against social threat are more consistent with earlier security and attachment theories from which EST and EST-R are derived (Ainsworth et al., 1978; Blatz, 1966; Bowlby, 1969). For example, the Latin root of security translates to "without anxiety" or "without care" (Ainsworth & Bolwby, 1991), and the opposite of fear, alarm, or apprehension underlying insecurity is a calm state that does not require the presence of happiness or joy (Goldberg, Grusec, & Jenkins, 1999). In fact, within the organizational perspective in developmental psychopathology (Cicchetti & Rogosch, 2002), happiness cannot be conceptualized as an intrinsically positive or secure response without deciphering its deeper meaning or function within the broader organization of children's patterns of reactivity to conflict. For example, in EST-R, responses that appear to be positive based on their form or morphology (e.g., praising or entertaining the parents, comforting the parent) are commonly interpreted as signs of insecurity rather than security when they occur in a broader context of fear or vigilance to interparental conflict (Davies & Martin, 2013; Davies & Sturge-Apple, 2007).

Differences between the definitions are not simply minor and semantic in nature. Rather, the distinction provides a fundamental foundation for identifying the nature and substance of emotional insecurity in the face of interparental conflict. From an evolutionary perspective, the inclusion of positive affect in the definition of security adulterates any analysis of the protective function of the SDS and its organization around vigilance and fear. Building a broad construct of emotional security that houses an amalgamation of positive and negative affect blights the ability to distinguish differences in how children defend against interparental threat from the functioning of other distinct behavioral systems that improve fitness through the regulation of positive emotionality and approach (e.g.,

affiliation, exploration). From the perspective of EST-R, winnowing positive affect and diffuse forms of negative affect from the definition of emotional security is essential to understanding the specific operation of the SDS, its coordination with other ethological systems, and its specific place in ontogenesis. Thus, rather than accepting the metaphor of security as a "bridge between the child and the world," EST-R proposes that security (i.e., the absence of anxiety or fear) is the figurative road that paves the way for other approach-oriented systems of exploration and affiliation to serve as the symbolic bridge connecting the child to the broader world.

Due to the early stages of theoretical development in EST-R, research has yet to definitively determine whether this more circumscribed definition provides a better characterization of the emotional security system. Although authoritative tests of the viability of the security conceptualization in EST-R will require collection of close observations and assessments of children's responses to interparental conflict, there is some initial evidence to suggest that positive responses and perceptual sensitivity to threat are not simply gradations of security and insecurity nested on the same continuum. For example, behavioral signs of children's vigilance to interparental conflict in a simulated phone argument task involving the mother and father were unrelated to their reports of happiness at either kindergarten ($r = -.05$) or first grade ($r = -.03$) assessments of a longitudinal study (Davies, Sturge-Apple, Winter, Cummings, & Farrell, 2006). Moreover, factor analyses of children's self-reported reactivity to interparental conflict on the Security in the Interparental Subsystem (SIS) scale over a year revealed that positive behavioral and emotional responses either loaded positively onto higher order insecurity construct (e.g., "When my parents have an argument, I try to be on my best behavior . . . ") or failed to cohere with any dimension of emotional insecurity (e.g., "When my parents have an argument, I laugh or smile") (Davies, Forman, et al., 2002). Likewise, daily diary reports of preadolescent responses to interparental conflict over a six-week period indicated that children's fear and sadness were positively associated with becoming unusually well behaved (Garcia O'Hearn et al., 1997). Finally, Davies and Martin (2013) provided some preliminary evidence for the confluence of vigilance to threat and ingratiating, well-behaved, appeasing, and overbright behaviors within higher order patterns of insecure responding to interparental conflict (see the Person-Based Taxonomy section in Future Directions).

If the data continue to support the more precise definition of security in EST-R, then what are the implications for the whole generation of research that was predicated on the old conceptualization of security in EST? Although it is premature to make any definitive conclusions until more research is conducted, we would like to caution against the practice of throwing the baby out with the bathwater. Even if research consistently supports the characterization of security in EST-R, the methodological and analytic approaches adopted by many past studies may still yield sufficiently valid measures of security. In contrast to the questionable discriminant validity of creating broad manifest composites of negativity and diminished positivity, existing latent approaches that more heavily utilize manifest indicators of fear and vigilance may offer a psychometrically sound approach to assessing insecurity (e.g., Davies, Cicchetti, & Martin, 2012; Davies, Sturge-Apple, & Martin, 2013). By capturing common variance between fearful responses and other action tendencies (e.g., hostility, involvement, behavioral dysregulation) that serve both the SDS and other goal systems, these latent approaches increase construct validity by maximizing the likelihood that fear and defense represent a common underlying source of children's reaction patterns to interparental conflict.

Distinctions Between Emotional Security and Child Temperament and Personality

Defining emotional security as the operation of the social defense system within the bounded relationship context of the interparental subsystem also provides a clear set of conceptual bases for differentiating it from temperament, personality, and psychopathology. As a relational construct housed within the setting of the interparental dyad, children's security in the interparental relationship is theorized to serve as a precursor of subsequent trajectories of personality and adjustment over time. Consistent with transactional models of developmental psychopathology (Sameroff & MacKenzie, 2003), EST-R postulates that temperament and personality can, in turn, affect the calibration of the goal-corrected system of emotional security (e.g., Davies & Sturge-Apple, 2007). However, according to theory, emotional security is not synonymous with dimensions of personality or temperament. That is, children's security in the interparental relationship cannot be regarded as simply a by-product of higher order temperamental (e.g., inhibition or shyness), personality (e.g., neuroticism), or psychopathology (e.g., anxiety) constructs.

Research has consistently supported the distinctive nature of emotional security measures in relation to indices of temperament, personality, and psychopathology measures. During early childhood, various indices

of difficult temperament have evidenced negligible to modest associations with children's emotional insecurity in the interparental relationship. For example, toddlers' temperamental distress derived from maternal surveys was unrelated to coder ratings of their concurrent emotional insecurity based on maternal narratives from a semistructured interview (Davies, Manning, & Cicchetti, 2013). Likewise, as indices of emotional insecurity, measures of fearful reactivity to interparental conflict were not associated with observations of toddler inhibited temperament and were modestly related to lower, rather than higher, levels of observed temperamental irritability (Davies, Cicchetti, & Martin, 2012). As children progress through childhood and adolescence, empirical evidence continues to support the hypothesis that emotional security is distinct from personality or psychopathology constructs. For example, associations among emotional security and indices of personality (e.g., trait negative affect) and psychopathology have typically been in the moderate range during middle childhood (e.g., Davies & Cummings, 1998; El-Sheikh et al., 2008) and adolescence (e.g., Cummings, George, McCoy, & Davies, 2012; Davies, Harold, et al., 2002).

Additional distinctions can be drawn between emotional security and attributes of temperament and personality in the degree of stability of individual differences in the constructs (i.e., differential stability) over time. Whereas personality and adjustment constructs are defined by relatively high temporal consistency, the relational and coping properties of emotional security are proposed to evidence lower developmental stability that typically will fall in the modest to moderate range depending on the length of the developmental span. From a developmental psychopathology perspective, the emergence and intensification of adjustment trajectories are, under many conditions, gradual products of sustained and persistent developmental processes (Sroufe & Rutter, 1984). Thus, in translating these assumptions to our theory, differences between children in their responses to conflict must evidence some coherence over time if they increase the likelihood of intensification and proliferation into patterns of psychopathology (Cummings & Davies, 2010). Continuity in children's experiences of security is specifically theorized to result from (1) some degree of temporal stability in their exposure to properties of interparental conflict that activate the social defense system, and (2) their own tendencies utilize previously developed patterns as guides for interpreting and reacting to subsequent threatening events in the interparental subsystem (Davies, Winter, & Cicchetti, 2006; Davies et al., 2013). However, against this backdrop of stability, adaptation to interparental conflict is assumed to be the product of the dynamic interplay between actively changing children and family contexts. Thus, consistent with the documented plasticity of similar relational constructs (e.g., parent–child attachment; Belsky, Campbell, Cohn, & Moore, 1996), the emotional security system remains at least somewhat open to alteration across time.

Although more research on the temporal consistency of emotional security is needed to make authoritative comparisons with the differential stability of temperament and personality constructs, the emerging empirical evidence is consistent with our hypotheses. For example, with an average developmental span for calculating developmental stability of seven years, a meta-analysis indicated that continuity fell within the moderate to high range for temperamental and personality traits from preschool through late adolescence (e.g., mean $r = .49$; range = .45 to .52; Roberts & Del Vecchio, 2000; also see Nepple et al., 2010). Conversely, correlations between signs of insecurity over a comparable six to seven year span in childhood to adolescence were in the modest to moderate range (mean $r = .23$, range = .17–.30; Davies, Sturge-Apple, Bascoe, & Cummings, 2014). Analyses of differential stability within smaller developmental spans of one or two years commonly yield higher coefficients (e.g., *r*s largely in the .30s to .40s) that fall more squarely in the moderate range (e.g., Davies, Martin, & Cicchetti, 2012; Davies, Sturge-Apple, Bascoe, & Cummings, 2014). However, the stability is still significantly lower than the continuity indices reported for personality and temperament constructs (*r*s =.60s and .70s) of personality and temperament constructs over comparable time periods (e.g., McCrae et al., 2000).

Summary

In sum, the first decade following the inception of EST in 1994 advanced an understanding of the behavioral and psychological processes that regulate children's goal of preserving emotional security in the interparental relationship (e.g., Cummings & Davies, 1996; Davies & Cummings, 1994). Although emotional reactivity, regulation of exposure to parent affect, and internal representations were identified as classes of response processes serving the goal of emotional security, the original formulation did not provide sufficient conceptual precision or depth in characterizing the architecture or operation of the emotional security system. Against a diverse landscape of other social and emotional constructs (e.g., personality and temperamental attributes, parent–child attachment security, broader forms of responding to interparental

conflict), EST offered little theoretical justification for why emotional security should be considered a distinct process in its own right. Guided by evolutionary models, EST-R was developed, in part, to distinguish emotional security from related constructs by precisely describing the ethological underpinnings (i.e., operation of the SDS), primary properties (e.g., vigilance to interpersonal threat as a central process), and developmental characteristics (e.g., differential stability and change) of the emotional security system (Davies & Martin, 2013; Davies & Sturge-Apple, 2007; Davies, Sturge-Apple, & Martin, 2013; Davies & Woitach, 2008). Although definitive empirical tests of all EST-R hypotheses have yet to be conducted at this early scientific stage, the existing research findings have produced consistent support for characterizing the goal of preserving emotional security in the interparental relationship as a distinct relational construct that is regulated by a different ethological module than the parent–child attachment system.

EMOTIONAL SECURITY AS A MEDIATOR OF CHILDREN'S ADJUSTMENT TO INTERPARENTAL CONFLICT

Establishing emotional security in the interparental relationship as a distinct construct is only a beginning step in the scientific process. From a developmental psychopathology perspective, determining the value of a theory also hinges on its utility in elucidating the mechanisms by which risk factors increase children's vulnerability for maladjustment (Cicchetti & Toth, 1998; Sroufe & Rutter, 1984). By enlisting the concept of developmental cascades (Cicchetti, 2002; Masten & Cicchetti, 2010), developmental psychopathology has specifically characterized children's mental health as a product of an unfolding series of processes that proliferate across multiple developmental systems. Drawing on this important conceptual tool, EST proposes that emotional insecurity is part of a mediational chain that ultimately shapes children's trajectories of adjustment (see #2 paths in Figure 6.1). In the first part of the mediational chain, witnessing destructive forms of interparental conflict characterized by hostility, aggression, and conflict escalation is proposed to progressively increase children's concerns about their security and safety over time and in the context of the interparental subsystem. In the second part of the unfolding series of processes, difficulties preserving emotional security are proposed to intensify and proliferate to increase children's vulnerability to poor adjustment and psychopathology (Cummings & Davies, 1996; Davies & Cummings, 1994). Thus, evaluating the developmental significance of EST requires examining whether emotional security serves as a consistent explanatory process in developmental pathways between interparental conflict and child adjustment even after taking into account alternative, viable mechanisms. In the following sections, we describe the progress in testing these pathways, including delineation of (1) the mediational role of emotional security, (2) interparental sources of emotional insecurity, (3) the potency of specific signs of emotional insecurity in the developmental cascade, and (4) the multitude of sequelae of insecurity.

Research on the Mediational Role of Emotional Security

Children have several sound reasons for being concerned about their safety in the context of interparental relationship difficulties. First, conflict between parents may undermine children's security by increasing the likelihood that the unresolved hostility stemming from the conflicts may proliferate into parenting practices, interactions with children, and the broader family unit. Second, disagreements between parents are commonly manifestations of underlying power struggles between adult dominants in the family and, as a result, may signify chaotic collapses and perturbations in the social hierarchy. Third, parental expressions of hostility and distress may reflect a loved one's unhappiness and emotional instability and raise the possibility of family dissolution (e.g., parental break up) and its accompanying hardships (Johnston, Roseby, & Kuehnle, 2009). Accordingly, children's emotional insecurity in the interparental relationship is proposed to develop out of protracted histories of their exposure to destructive properties of interparental conflict, including high levels of anger, verbal aggression, violence, gridlock, and escalation of hostility (Davies & Cummings, 1994). In turn, children's recurrent needs to prioritize the goal of regaining and preserving security in the interparental relationship, set the stage for increased vulnerability to psychopathology through multiple processes that collectively reduce the opportunities for children to successfully engage in other prominent developmental challenges and life goals.

Table 6.2 provides a synopsis of the results from empirical tests of emotional insecurity as a mediator of associations between destructive interparental conflict and children's psychological and physical difficulties. To maximize the construct validity of emotional insecurity in our literature review, studies were only included in the table if they explicitly measured and tested signs of emotional security in the interparental relationship in

TABLE 6.2 Synopsis of Findings from Studies Examining Multiple Signs of Emotional Insecurity as Mediators of Associations Between Interparental Conflict and Child Adjustment

Study	Sample	Approach	Relevant findings
Buehler, Lange, & Franck (2007)	416 11- to 14-year-old children	SEM analyses using multiple methods (i.e., observation, survey) and informants (i.e., child, parent, teacher) within a longitudinal design	Marital hostility predicted adolescent insecurity in the interparental relationship one year later ($\beta = .34$). Insecurity, in turn, predicted greater externalizing ($\beta = .27$) and internalizing ($\beta = .38$) symptoms one year later after controlling for prior symptoms. Due to multicollinearity between emotional security and cognitive appraisals from the cognitive-contextual framework, SEM analyses with both constructs as simultaneous mediators did not produce an admissible solution.
Cummings, George, McCoy, & Davies (2012)	235 5- to 7-year-old children	Multiple methods (i.e., observation, survey) and informants (i.e., mothers, fathers, children) nested within three time points using SEM	Children's exposure to interparental conflict during kindergarten predicted greater emotional insecurity two years later after controlling for prior insecurity, $\beta = .30$. Insecurity in second grade was associated with increases in children's internalizing ($\beta = .25$) and externalizing ($\beta = .21$) symptoms in seventh grade after specifying the autoregressive path involving prior symptoms.
Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings (2006), Study 1	226 8- to17-year-old children	SEM analyses using multiple informants (i.e., mother, father, children) nested within a longitudinal design	Interparental conflict predicted greater emotional insecurity in the interparental relationship ($\beta = .36$) one year later. Emotional insecurity, in turn, was concurrently associated with greater internalizing ($\beta = .35$) and externalizing ($\beta = .20$) symptoms after controlling for children's initial symptomatology and age.
Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings (2006), Study 2	232 5- to 7-year-old children	SEM analyses using multiple methods (i.e., observation, survey) and informants (i.e., mother, father) within a prospective design	Interparental conflict predicted children's emotional insecurity across a one-year period ($\beta = .23$). Insecurity, in turn, predicted greater internalizing ($\beta = .14$) and externalizing ($\beta = .12$) symptoms one year later after controlling for initial levels of symptoms.
Davies, Cicchetti, & Martin (2012)	201 2-year-old children	SEM analyses conducted with multiple methods (i.e., observation, semistructured interview, survey) and informants (i.e., mothers, observers) within a longitudinal design with two annual time points	Interparental aggression predicted concurrent signs of child insecurity as reflected in fearful reactivity to conflict, $\beta = .27$. Insecurity, in turn, significantly predicted: mother reports of internalizing, $\beta = .17$, and, $\beta = .16$, and externalizing symptoms; and observer ratings of internalizing, $\beta = .23$, and externalizing, $\beta = .19$, symptoms. Results were significant even after controlling for initial levels of internalizing and externalizing symptoms in the model, including child inhibited and irritable temperamental characteristics as covariates, and specifying angry and sad reactivity to conflict as alternative mediators in the model.
Davies & Cummings (1998)	56 6- to 9-year-old children	Latent variable path analysis with partial least squares estimation of cross sectional data derived from multiple informants (mothers, children) and methods (observation, semistructured interview, surveys)	Interparental discord was associated with greater emotional reactivity to a simulated conflict involving the mother ($\beta = .34$) and more negative representations of interparental relationships ($\beta = .29$). Emotional reactivity, in turn, was associated with greater internalizing ($\beta = .29$) and externalizing symptoms ($\beta = .26$) whereas internal representations related only to internalizing symptoms ($\beta = .27$). Regulation of exposure to parent affect failed to mediate associations between interparental discord and child problems.
Davies, Harold, Goeke-Morey, & Cummings (2002), Study 2	285 11- to 13-year-olds	SEM analyses of multiple informant (i.e., mothers, fathers, children, teachers) survey data across two measurement occasions spaced two years apart	In separate models conducted for internalizing and externalizing symptoms, discord between parents significantly predicted greater emotional insecurity as indexed by emotional reactivity, behavioral dysregulation, and negative representations of interparental relationships two years later (βs= .16 and .17) Greater insecurity, in turn, was significantly associated with concurrent assessments of child internalizing, $\beta = .47$, and externalizing, $\beta = .25$, symptoms. Results were obtained after controlling for initial child symptoms and specifying cognitive-contextual framework processes of child self-blame and perceived threat as alternative mediators.

TABLE 6.2 (*Continued*)

Study	Sample	Approach	Relevant findings
Davies, Harold, Goeke-Morey, & Cummings (2002), Study 3	173 10 to 15-year-olds	Cross sectional design with multiple informants (i.e., parents, children) using SEM analyses	Destructive interparental conflict was significantly associated with a latent composite of emotional insecurity, $\beta = .25$. Emotional insecurity also predicted child internalizing, $\beta = .54$, and externalizing, $\beta = .23$, symptoms. Findings were part of a larger model that also specified parenting difficulties and parent–child attachment security as predictors.
Davies, Martin, & Cicchetti (2012), Study 1	250 10- to 15-year-olds graders (M age = 13)	SEM analyses of survey data collected from multiple informants (i.e., parents, children) at a single time point	Destructive interparental conflict (i.e. frequency, verbal aggression, child-related conflict) was associated with a multi-indicator latent construct of children's insecurity, $\beta = .32$. Insecurity, in turn, was associated with greater child problems, $\beta = .61$. These results remained significant even after specifying the negligible effects of exposure to constructive interparental conflict and including child negative affectivity, parent–child relationship quality, holistic family functioning, and SES as covariates.
Davies, Martin, & Cicchetti (2012), Study 2	201 2-year-old children	SEM analyses of multimethod (i.e., surveys, observations, semistructured interviews) of three annual waves of data in a longitudinal design	Destructive interparental conflict (i.e., frequency, verbal aggression, violence) predicted increases in children's emotional insecurity over a one year period, $\beta = .37$. Increasing insecurity, in turn, was associated with less steep declines in the latent growth curve slope of child adjustment problems over the three measurement occasions. Findings were consistent after estimating the minimal effects of constructive interparental conflict and covariates of child negative affectivity, parent–child relationship quality, and SES.
Davies, Sturge-Apple, Bascoe, & Cummings (2014)	230 children (M age = 13)	SEM analyses of multimethod (i.e., surveys, observations) and multi-informant (i.e., mother, father, child, teacher) data derived from three annual waves	Destructive interparental conflict predicted increases in children's insecurity over a one year period ($\beta = .31$) which, in turn, predicted greater psychological adjustment problems over the next year, $\beta = .34$. Results were robust after taking into account prior levels of insecurity and psychological adjustment problems, family SES, and child sex.
Du Rocher Schudlich & Cummings (2007)	262 8- to 16-year-old children	SEM analyses conducted with a cross sectional design with multiple methods (i.e., surveys, interviews, observations) and informants (i.e., mother, father, child)	Depressive interparental conflict tactics characterized by parental vulnerability were concurrently associated with emotional insecurity ($\beta = .31$), which in turn predicted children's psychological maladjustment ($\beta = .27$). Results were significant in a broader model of predictor involving maternal and paternal parenting and depressive symptoms, constructive and destructive (i.e., hostile) marital conflict tactics, and child age.
El-Sheikh, Cummings, Kouros, Elmore-Staton, & Buckhalt (2008)	251 children with a M age of 8 years	Multiple informants (i.e., mother, father, child) within a cross sectional design that employed SEM analyses	Six analyses were conducted examining the mediational role of insecurity in models of child internalizing difficulties, PTSD symptoms, externalizing problems, aggression directed toward mother and father, and physical health. Emotional insecurity in the interparental relationship was predicted by both maternal directed aggression toward the father (*Mean* $\beta = .26$) and paternal directed aggression toward the mother (*mean* $\beta = .54$). In the second link in the mediational chain, emotional insecurity was concurrently associated with three of the six outcomes, including child internalizing ($\beta = .45$), PTSD ($\beta = .51$), and externalizing ($\beta = .63$) symptoms. These results were produced in analyses that also included family SES and child age, gender, and race as covariates.
El-Sheikh, Buckhalt, Cummings, & Keller (2007)	166 children with a M age of 9 years	SEM analyses of cross sectional data derived from multiple informants (i.e., mother, father, child, teacher) and methods (i.e., survey, actigraphy)	Children's perceptions of conflict were associated with a multi-indicator construct of emotional insecurity ($\beta = .45$) which, in turn, was associated with diminished sleep quality in models of parent ($\beta = -.33$) and teacher ($\beta = -.32$) adjustment problems. In the final part of the multichain cascade, lower levels of sleep quality were associated with parent reports of emotional ($\beta = -.59$) and behavioral ($\beta = -.54$) problems and teacher reports of emotional problems ($\beta = -.40$) and academic functioning ($\beta = .30$), but not child behavior problems.

(*continued*)

TABLE 6.2 (*Continued*)

Study	Sample	Approach	Relevant findings
Harold, Shelton, Goeke-Morey, & Cummings (2004)	181 11- to 12-year-old children	SEM analyses using multiple methods (i.e., surveys, analog methods) over two measurement occasions	Interparental conflict was significantly associated with concurrent insecurity indices of children's emotional reactivity, $\beta = .29$, and cognitive representations, $\beta = .30$, but was unrelated to children's regulation of exposure to parent affect. However, associations involving insecurity and subsequent child internalizing and externalizing symptoms one year later were all nonsignificant in a broader model that incorporated autoregressive pathways of child symptoms and children's emotional security about parenting at Time 2.
Kelly & El-Sheikh (2013)	176 children with *M* age of 9 years	SEM analyses of three waves of longitudinal data over a five-year span with use of multiple informants (i.e., mother, father, child) and methods (i.e., survey, actigraphy)	Interparental aggression predicted greater a multiindicator latent construct of children's emotional insecurity two years later, $\beta = .20$. Child insecurity, in turn, predicted sleeping problems three years later, βs = .20. Results were significant with autoregressive paths included for emotional insecurity and child reports of sleeping problems. Emotional insecurity did not mediate actigraphy assessments of sleep duration or efficiency.
McCoy, Cummings, & Davies (2009)	235 5- to 7-year-old children	Multiple informant (i.e., mothers, fathers, teachers) measures over three time points analyzed through SEM	In separate analytic models, constructive (e.g., resolution) and destructive (e.g., hostility) interparental conflict were each associated with children's emotional security one year later, βs = .32 and -.34, respectively. Children's emotional security was associated with their prosocial behavior in each model, βs = .26 and .30. These findings were generated in the context of larger analytic models testing parental warmth as another possible mediator and including autoregressive paths for prosocial behavior.

mediational models of interparental conflict. Sixteen of the seventeen studies in the table support the hypothesis that interparental conflict increases children's vulnerability to psychological problems by undermining their emotional security in the interparental relationship. That is, 94% of the studies in the table reported at least one significant mediational pathway whereby interparental conflict was associated with at least one sign of insecurity, with insecurity, in turn, evidencing a significant relationship with at least one measure of child psychological functioning. In some of the papers, multiple mediational pathways were specified within single studies due to the inclusion of multiple forms of conflict, signs of insecurity, forms of developmental outcomes, or some combination thereof. Evaluation of all the various pathways in these studies continued to yield consistent support for each link in the mediational cascade. Of relevance to the first link, 81% (i.e., 21 of 26) of the pathways between interparental conflict and children's signs of emotional insecurity were reported to be significant in the studies in Table 6.1. Likewise, pathways tested among children's signs of emotional insecurity and their forms of psychological difficulties were also significant in the majority of cases (59%; 29 of 49).

Confidence in the findings is bolstered by an evaluation of the methodological characteristics of the studies. In highlighting the scientific rigor in the empirical approaches, all the studies included in our review employed, at a minimum, sophisticated latent variable approaches (i.e., most were SEM) with designs that capitalized on multiple informants, multiple methods, or both. Nevertheless, the common observation of an inverse association between methodological rigor and the strength of substantive findings in literature reviews in the developmental sciences (e.g., Kitzmann, Gaylord, Holt, & Kenny, 2003; Rhoades, 2008) raises questions about whether the significant findings may be an artifact of shared method or informant variance. Our analysis, however, does not suggest that this conclusion applies to tests of the mediational role of emotional security. First, if the findings were an artifact of shared method or informant variance, systematic variability in the strength of the findings would be evident across different measurement approaches, with the results favoring less rigorous designs. However, in the studies in Table 6.2, the proportion of significant mediational findings did not differ as a function of whether the design incorporated (1) either multiple informants or multiple methods (16/30; 53%) or (2) both multiple informants and methods (17/29; 59%).

Second, documentation of mediational pathways using temporally ordered lagged analyses of interparental conflict, emotional security, and child developmental outcomes would further strengthen conclusions about the

usefulness of our theory. Consistent with this criterion, the proportion of significant mediational pathways identified in studies utilizing prospective lagged analyses of change in children's health outcomes or their insecurity were actually higher (i.e., 19/28; 68%) than the results from cross sectional designs (i.e., 14/31; 45%). Moreover, the findings from the prospective analyses did not differ considerably as a function of whether the lagged models utilized changes in child outcomes only (i.e., 14/20; 70%) or a more rigorous approach that quantified latent change in both their emotional insecurity and outcomes (i.e., 5/8; 63%).

Third, from a general methodological perspective, the two studies in Table 6.2 that utilized simulations of adult anger as proxies for assessing children's insecurity in the interparental relationship should evidence lower levels of validity than assessments of children's reactivity to actual interparental conflict (i.e., Davies & Cummings, 1998; Harold, Shelton, Goeke-Morey, & Cummings, 2004). Although anger simulations may achieve some degree of psychological realism by activating many of the same processes (e.g., defending against interpersonal threat) that occur in actual interparental conflicts (Aronson, Wilson, & Brewer, 1998; Cummings, 1995), they are unlikely to evidence a one-to-one correspondence with how children respond to conflict between their parents. Consistent with this thesis, assessments of insecurity derived from contexts of interparental conflict produced considerably more support for the mediational pathways (i.e., 31/49; 63%) than did the analog measures of insecurity (i.e., 2/10; 20%). In addition, the power of analog designs to evoke processes approximating insecurity in the interparental relationship is likely to increase when simulations are more affectively charged and personally involving. Thus, empirical bases for EST would be strengthened if support for emotional security is stronger with increases in the psychological realism of the analog design. Consistent with these conditions, the more engaging and involving simulation of live anger involving a mother and experimenter in the Davies and Cummings (1998) yielded evidence for half (i.e., 2 of 4) of the mediational pathways tested. Conversely, the videotaped presentation of conflicts between adult strangers in the study by Harold and colleagues (2004), which was likely to evidence lower psychological realism, provided no support for any of the six mediational pathways examined.

Fourth, evidence for the primary hypothesis in EST would be further garnered if it can be demonstrated that mediational pathways involving interparental conflict, emotional insecurity, and child maladjustment are not simply byproducts of third variables and their more proximal explanatory power. In further supporting the validity of EST, the comparison of studies in Table 6.2 revealed that the proportion of significant mediational pathways involving emotional security did not differ appreciably as a function of the inclusion (i.e., 22/38, 58%) or exclusion (i.e., 11/21; 52%) of covariates in the analytic models. Thus, the robustness of emotional security as a mediator remained even after specifying of a wide array of demographic (e.g., parent education, income, occupational prestige); family-level (e.g., cohesion, instability); parent–child (e.g., warmth, attachment security, relationship quality, role reversal); and child characteristics (e.g., sex, age, temperament) (e.g., Cummings et al., 2006; Davies, Cicchetti, & Martin, 2012; Davies, Martin, & Cicchetti, 2012; Davies, Cicchetti, Hentges, & Sturge-Apple, 2013; El-Sheikh et al., 2008; McCoy, Cummings, & Davies, 2009). Moreover, the results from studies in Table 6.2 (e.g., Davies, Cicchetti, & Martin, 2012; Davies, Harold, et al., 2002) indicated that emotional security remained a significant mediator of interparental conflict while simultaneously specifying the mediating roles of alternative forms of conflict reactivity outlined within the cognitive-contextual framework (e.g., perceived threat, self-blame; Grych & Fincham, 1990), specific emotions theory (e.g., dysphoric conflict responses, Crockenberg & Langrock, 2001), and social learning theory (e.g., angry, hostile responding to conflict; Cox, Paley, & Harter, 2001; Margolin, Oliver, & Medina, 2001; Zimet & Jacob, 2001). Therefore, in accord with our premise that emotional security is a distinctive psychological construct in its own right, the empirical evidence indicates that emotional security was a unique and consistent mediator of associations between interparental conflict and children's psychological functioning even in the context of other alternative contextual and psychological processes.

In conclusion, our systematic review of the literature indicates that there is a clear pattern of empirical support for the role of emotional security as a mediator of associations between interparental conflict and children's psychological adjustment. Support for emotional security as an explanatory mechanism was garnered under the most methodologically rigorous conditions that employed multiple informants and methods, assessments of child reactivity in naturalistic contexts of interparental conflict, designs that carefully examined prospective changes in children's adaptation over time, and statistical approaches that incorporated multiple third variables as covariates. Building on the strength of the findings, the next sections in our analysis are devoted to describing the specific interparental sources of children's emotional insecurity, tracing the implications of insecurity, and outlining the developmental significance of more deeply characterizing the responses underlying emotional insecurity.

Interparental Sources of Children's Emotional Insecurity

Research on the mediational role of emotional security has consistently identified exposure to heightened interparental conflict as a predictor of insecurity. However, common empirical treatment of interparental conflict as a broad homogenous construct in the literature has hindered the identification of specific expressions of interparental conflict that pose a sustained threat to children's emotional security in the interparental relationship. Although this approach is advantageous in maximizing parsimony and retaining a practical (rather than dispersive) scope of inquiry, it is not sensitive to the extensive scientific and clinical acknowledgment that couples vary widely in how they approach and manage conflicts. For example, some parents are able to marshal efforts to work in a collaborative way toward reaching a compromise or resolution to their problems. For other partners, conflicts are marked by tendencies to resort to escalation of hostility and aggression, and, still other couples respond to disagreements by disengaging and withdrawing. From a developmental psychopathology perspective (Cicchetti & Rogosch, 1996), the diversity of interparental conflict expressions highlights the potential operation of equifinality and the underlying notion that a single outcome (i.e., emotional insecurity) may result from multiple factors (i.e., conflict characteristics). Accordingly, a critical step in advancing process-oriented models is to better understand the scope of equifinality in associations among interparental conflict characteristics and children's emotional insecurity.

In attempting to define the boundaries of equifinality, EST and EST-R offer different predictions regarding the range of interparental conflict characteristics that are proposed to affect children's emotional insecurity. According to EST, children's emotional insecurity in the interparental relationship is a product of a wide array of conflict expressions. A primary proposal is that parental conflict tactics can be distinguished in terms of their constructive and destructive properties based on the degree to which they increase or reduce insecurity (Goeke-Morey et al., 2003). Interparental conflict characterized by escalating hostility, aggression, fear, dysphoria, and detachment is defined as destructive because it is posited to increase signs of insecurity. In contrast, parental displays of cooperation, collaboration, and resolution during conflicts are postulated to enhance children's security, in part, by reducing their signs of distress (McCoy et al., 2009). Taken together, EST assigns relatively equal empirical weight to constructive and destructive types of conflict as independent contributors to children's emotional insecurity (Cummings & Davies, 1996; Davies & Cummings, 1994). Thus, equifinality is ubiquitous in the assumption that virtually any form of interparental conflict may impact children's concerns for safety and security for better or for worse.

Although EST-R proposes that there is some degree of equifinality, it was designed in part to more precisely identify the specific characteristics of interparental stimuli that are particularly potent in engendering insecurity. Evolutionary models conceptualize the social defense system as an encapsulated module that responds selectively to a delimited set of contextual stimuli that are comparable in form to recurring cues presaging significant threats to survival throughout human evolution (e.g., angry expressions, loud noises, aggression, dominant posturing) (Davies & Sturge-Apple, 2007; Öhman & Mineka, 2001). As the function of the social defense system is to defuse conspecific threat, children's emotional insecurity in the context of interparental conflict should selectively reflect their sensitivity to histories of exposure to these same interpersonal threat cues displayed by parents in their disagreements (Davies & Sturge-Apple, 2007; Hofmann, Moscovitch, & Heinrichs, 2002). Because diminished displays of mutual happiness, support, and cohesiveness are far less reliable as signals of danger if they are not accompanied by hostile threat cues, these parameters of constructive conflict are hypothesized to carry minimal weight in signifying threat and organizing children's social defense strategies (Öhman & Mineka, 2001). Therefore, EST-R places significantly greater weight on destructive forms of conflict as etiological agents versus constructive or benign conflict tactics (Davies & Sturge-Apple, 2007; Davies & Woitach, 2008).

In further highlighting the importance of more fine-grained differentiations of destructive conflict, EST-R also proposes that other forms of destructive conflict assume varying degrees of salience in undermining children's insecurity. Sensitivity to danger cues is unlikely to simply be limited to the identification of angry, hostile displays by conspecifics. Rather, the ability to decipher fearful expressions holds similar adaptive significance to anger processing capabilities as a mechanism for detecting imminent danger in the social group (Davies & Sturge-Apple, 2007; LoBue, 2009). Thus, exposure to parental fearful and frightened behaviors during interparental conflict is posited to evoke children's concerns about their safety and security that approximate the potent effects of angry, hostile conflicts. In contrast, the strength of interparental dsyphoria and disengagement as predictors of children's emotional insecurity should fall in the intermediate range between the minimal impact of constructive conflict and the potent effects of conflicts containing heightened anger or fear. On the one hand, dysphoria and disengagement

signify abandonment, rejection, and interpersonal struggle within the social hierarchy (Gilbert, 2001). Therefore, repeated exposure to these conflict properties is more likely to undermine children's security in the interparental relationship than variations in constructive conflict by signalling the potential for threat (Dixon, 1998). On the other hand, bouts of disengagement and dysphoria are less reliable prognosticators of imminent interpersonal threat. Thus, it follows that exposure to parental hostile and fearful displays during conflict represent more immediate threat risk than disengaged or dysphoric conflict tactics.

Although studies have examined children's responding to different forms of interparental conflict, direct empirical tests of the comparative utility of predictions derived from EST and EST-R are still relatively rare. Scientific approaches to addressing this gap specifically require the simultaneous examination of the joint interplay among destructive and constructive properties of interparental conflict in the prediction of children's insecurity. However, most studies exploring multiple dimensions of interparental conflict have isolated the predictive power of each class of conflict tactics (e.g., constructive, hostile-destructive) in separate analytic models of emotional insecurity (e.g., McCoy et al., 2009; Cummings et al., 2002; Cummings, Goeke-Morey, & Papp, 2004; Goeke-Morey et al., 2003; Goeke-Morey, Cummings, & Papp, 2007). For example, a recent investigation reported that the association between constructive interparental conflict tactics and children's prosocial behavior was mediated by signs of diminished child insecurity in the interparental relationship (McCoy et al., 2009). However, documentation of heightened insecurity as a mediator of the relation between destructive interparental conflict and prosocial behavior in a separate analysis does not address the question of whether both forms of conflict operate uniquely as predictors (i.e., EST) or whether one class of tactics (i.e., constructive) is a spurious predictor by virtue of its association with a more operative class of conflict expressions (i.e., destructive) in undermining children's security.

Despite the need for more systematic analysis of the interplay between multiple dimensions of interparental conflict in the prediction of child insecurity, the sparse existing studies provide some initial support for the added precision of EST-R in identifying interparental sources of insecurity. For example, Fosco and Grych (2007) reported that children's exposure to destructive interparental conflict was the only significant predictor of their appraisals of threat within a multivariate model that also examined parental positive emotional expressiveness as a predictor. As a more direct test, a multistudy investigation revealed that children's emotional insecurity mediated associations between destructive interparental conflict and children's psychological problems even after specifying the negligible role of constructive interparental conflict and a wide array of family, parenting, and child covariates (Davies, Martin, & Cicchetti, 2012; also see Table 6.2). Replication of emotional security as a unique explanatory process underlying exposure to destructive conflict was especially striking in light of the variation in measures (i.e., multiple informants; multiple methods), methodological designs (i.e., cross sectional; longitudinal), and the risk (i.e., high risk; community sample) and SES (i.e., economically disadvantaged; working and middle class) characteristics of the samples across studies. Thus, these results are consistent with the evolutionary reformulation of emotional security theory and the resulting primacy ascribed to destructive interparental conflict in accounting for individual differences in children's emotional insecurity and its pathogenic implications (Davies & Sturge-Apple, 2007).

More fine-grained differentiation of the relative primacy of frightened (e.g., hostility, aggression) and vulnerable (e.g., worry, dysphoria, disengagement) forms of destructive conflict in studies has produced more complex results. Findings have shown that interparental hostility and disengagement were both uniquely associated with domains of children's insecure responding to interparental conflict (e.g., behavioral indices of distress, negative internal representations) in concurrent and prospective analyses (Davies, Sturge-Apple, et al., 2006). Although this study supports the value of distinguishing between the two types of destructive conflict, it does not provide a strong test of the comparative utility of EST and EST-R because both theories propose that equifinality is operating within the bounds of disengaged and hostile forms of interparental conflict. Likewise, in another study, multivariate analyses indicated that depressive conflict tactics (e.g., sadness, distress) were significantly related to children's insecurity while destructive (e.g., hostility) and constructive (e.g., problem solving) conflict tactics failed to explain any unique variance in child concerns about security (Du Rocher Schudlich & Cummings, 2007; see Table 6.2). Whereas the primacy of depressive conflict expressions over constructive tactics in the prediction of insecurity is more consistent with EST-R than EST, the negligible associations between hostile parental conflict and child insecurity run counter to predictions from both theories.

Complexity in interpretation is further compounded by the use of insecurity measures that fall outside the circumscribed definitional boundaries of the conceptual

construct in EST-R. In contrast to the characterization of emotional security as consisting of defensive, inhibitory behaviors, the two aforementioned studies utilized amorphous assessments of insecurity that included indices of anger and diminished positive affect. Therefore, due to concerns about the fidelity between the conceptualization of security in EST-R and its measurement in existing studies, definitive tests of the salience of different forms of destructive interparental conflict in EST-R (e.g., fear, dysphoria, hostility) will require more fine-grained approaches in future research. Likewise, similar refinements will also be necessary in distinguishing between the forms of destructive conflict that are proposed in EST-R to vary in their threat value for children. For example, in the Du Rocher Schudlich and Cummings (2007) study, the aggregation of both dysphoric (e.g., sadness, withdrawal) and frightened (i.e., fear) parental displays into the assessment of depressive conflict tactics did not permit sharp discrimination of the evolutionary hypothesis that fearful parental expressions would pose a greater threat to children's security than dysphoric expressions. In spite of the complexity of findings, there are compelling bases for expecting that hypotheses offered by EST-R will be supported in future research. Results from experimental research on emotion processing support the notion that there is a potent biological proneness to respond defensively to conspecific anger and fear (e.g., LoBue, 2009; Öhman & Mineka, 2001). For example, LoBue (2009) found consistent evidence for a bias in both young children and adults to more quickly process negative facial cues than positive facial stimuli. Moreover, processing of negative emotion stimuli varied in the same hypothesized way as outlined in EST-R, with more rapid identification of threat relevant emotions of anger and fear than sad stimuli.

The Potency of Signs of Emotional Insecurity in the Mediational Pathways

If emotion is a central organizer of children's efforts to preserve emotional security as emotional security theory proposes, then it should assume primacy in measurement models that utilize multiple indicators of children's insecurity. Given that the majority of papers in Table 6.2 employ latent approaches to assessing children's insecurity and report on their loadings (Cummings et al., 2006; 2012; Davies, Harold, et al., 2002; Davies, Martin, & Cicchetti, 2012; Davies, Sturge-Apple, Bascoe, & Cummings, 2014; El-Sheikh, Buckhalt, Cummings, & Keller, 2007; El-Sheikh et al., 2008; McCoy et al., 2009), they provide a means of examining the viability of this thesis. Consistent with predictions, calculations of the mean standardized loading for each of the dimensions of reactivity onto the latent construct of insecurity across the 10 studies was highest for emotional reactivity (.73) relative to negative representations of interparental relations (.69), ways of regulating (i.e., involvement, avoidance) exposure to parent affect (.66), and behavioral dysregulation (.58). As a further testament to the central role of emotional reactivity, all ten studies utilized an emotional reactivity variable as an indicator of emotional insecurity. In contrast, the studies evidenced greater inconsistency in the inclusion of the other indicators: negative representations (60%), regulation of exposure to parent affect (80%), and behavioral dysregulation (60%). Thus, the consistent support for emotional security as a mediator from the results of these studies can be attributed to a significant degree by the focus on emotionality as a core underlying experience of insecurity in the interparental relationship.

Methodological approaches that examine the relative roles of multiple signs of insecurity in models of interparental conflict and child adjustment offer additional evidence for the centrality of children's affective experiences in the face of interparental difficulties. For example, Davies and Cummings (1998) found that children's emotional reactivity was the most consistent mediator of associations between interparental conflict and child internalizing and externalizing symptoms in a model that also specified children's regulation of exposure to parent affect and internal representations as mediating mechanisms. In a more elaborate model containing nine dimensions of child reactivity as explanatory mechanisms, findings indicated that emotion dysregulation and internalization of distress in contexts of interparental conflict both uniquely mediated the prospective pathway between interparental conflict and children's internalizing symptoms (Buehler, Lange, & Franck, 2007). Meta-analytic results further revealed that negative affective reactions to conflict involving parents was a more consistent predictor of child problems relative to patterns of regulating exposure to parent affect (e.g., avoidance) (Rhoades, 2008).

By the same token, designation of emotional reactivity as a principal organizer of efforts to regulate the goal of preserving security may require additional refinement. Many of the earlier studies have focused on relatively broad assessments of emotional reactivity that contain indices of fear in conjunction with sadness, general emotional upset, anger, and even diminished happiness (Du Rocher Schudlich & Cummings, 2007; Rhoades, 2008). Although EST-R proposes that difficulties preserving emotional security may be manifested in a variety of responses

(e.g., anger, general distress), fear and vigilance are postulated to represent a common underlying source of defending against threat in the reformulated theory (Davies & Sturge-Apple, 2007; Davies & Woitach, 2008). Stimulated by refinements in the theory, some scientific attention has turned to developing more precise measurement strategies for capturing fearful and vigilant reactions than existing measures (e.g., parallel child and parent report assessments of Security in the Interparental Subsystem and Security in the Marital Subsystem–Parent Report; Cummings & Davies, 2010; Davies, Forman, et al., 2002). For example, with the development of the Interparental Disagreement Interview, systems for coding parent narratives derived from the semistructured interview discriminate between multiple forms of children's emotional expressions, including fearful, angry, and dysphoric reactivity to interparental conflict (IDI; Davies, Cicchetti, & Martin, 2012; Davies, Martin, & Cicchetti, 2012). These studies have shown that fearful forms of emotional and behavioral reactivity to conflict mediate prospective associations between destructive interparental conflict and children's psychological problems even after controlling for sad and angry forms of emotional reactivity, indices of temperamental emotionality, and a wide array of contextual variables (e.g., parenting, family functioning, socioeconomic risk). Thus, although it is too early in the research process to draw firm conclusions, these new measurement developments may offer greater flexibility and precision in capturing key emotional processes underlying insecurity.

The Multitude of Sequelae of Children's Insecurity

According to the principle of *multifinality* in developmental psychopathology (Cicchetti & Rogosch, 1996; Cicchetti & Toth, 2009), a diverse set of outcomes can be assumed to result from a common factor or pathway. In research on emotional security, the utility of this concept is illustrated in associations between children's concerns about security and a wide array of mental and physical health outcomes. Inspection of the findings from the studies in Table 6.2 reveals that emotional insecurity in the interparental relationship is specifically associated with internalizing symptoms, externalizing problems, and physical health and somatic (e.g., sleep) difficulties. Beyond domains of psychopathology, insecurity has also predicted a number of poor adjustment outcomes in the form of school difficulties, social and peer problems, attention impairments, and emotion dysregulation and interpersonal dependency (e.g., Bascoe, Davies, Sturge-Apple, & Cummings, 2009; Davies, Manning, & Cicchetti, 2013; Davies, Sturge-Apple, Bascoe, & Cummings, 2014; Davies, Woitach, Winter, & Cummings, 2008; McCoy et al., 2009; Sturge-Apple, Davies, Winter, Cummings, & Schermerhorn, 2008).

Although the list of outcomes associated with insecurity may be impressive in its scope, it begs the question of whether the prognostic value of insecurity varies across domains of functioning. Understanding the relative magnitude of risk for a wide array of outcomes is a critical part of the translational initiatives not only in developmental psychopathology (Cicchetti & Toth, 2006) but also in attempts to understand the unfolding cascade of processes that eventuate in distinctive adjustment patterns for children (Davies, Sturge-Apple, & Martin, 2013). The meta-analysis by Rhoades (2008) provides some of the strongest clues to date regarding potentially differential pathways that are moderated by form of adjustment. Although the system for characterizing dimensions of responding in her study was not specifically defined by dimensions of insecurity, forms of conflict reactivity characterized by negative affect, avoidance, and involvement can be regarded as close approximations of insecurity signs. Notably, when findings from multiple studies were pooled together, each of these parameters of reactivity more strongly predicted children's internalizing symptoms than externalizing symptoms. Analyses of results from studies specifically assessing insecurity in Table 6.2 provide additional, albeit modest, evidence to support this conclusion, with insecurity evidencing somewhat greater consistency in predicting internalizing (12/18, 67%) than externalizing (10/19, 53%) symptoms.

If future research more definitively confirms that insecurity is an especially robust precursor of internalizing symptoms, then it may provide clues in identifying the pathogenic mechanisms of effect. However, complexity is likely to be the rule rather than the exception in light of the plurality of outcomes emerging from the mediational pathways (Cicchetti & Toth, 2009). Regardless of whether insecurity evidences differences in the ability to predict subsequent forms of adjustment, the question still remains as to how and why multifinality prevails. Accordingly, in the next section, we describe the latest theory and research initiatives for identifying the explanatory mechanisms underlying the diversity of outcomes exhibited by children with high levels of insecurity.

Developmental Cascade Mechanisms

In keeping with the concept of developmental cascades in developmental psychopathology (Cox, Mills-Koonce, Propper, & Ganepy, 2010; Masten, 2005; Masten & Cicchetti, 2010), EST posits that the cumulative stress of coping with prolonged insecurity in the interparental

relationship sets in motion an array of cognitive, affective, and biological responses that have a snow-balling impact on adjustment across multiple contexts (e.g., school, peers, romantic partners). Over time, transactions between interparental conflict, emotional insecurity, and child adjustment problems may solidify their interrelationships and in the process spiral and proliferate into widespread child difficulties (Cummings et al., 2012; Kouros, Cummings, & Davies, 2010). Although the exploration of these cascading paths is still in its early stages, theoretical and empirical evidence point to several related processes as likely candidate mechanisms, including (1) the proliferation of defensive analogs to novel social situations beyond the family, (2) the sensitization of the stress response systems, and (3) disruption to other ethological systems that promote survival.

Proliferation of Defensive Analogs

Defensive analogs refer to models children utilize to filter, interpret, and affectively respond to threat and stress in challenging settings (Davies, Sturge-Apple, & Martin, 2013; Johnston et al., 2009). Thus, analogs encompass not only internal representations or working models of the meaning of close relationships but also sensorimotor schemes and action tendencies for coping with difficulties (e.g., submissiveness) that are often implicit and reflexive in nature. A central proposal of EST-R is that the children who experience insecurity in the interparental relationship are likely to become increasingly proficient in processing threat and recruiting resources necessary to guard against threat (e.g., Davies, Sturge-Apple, & Martin, 2013). As part of this process, children tend to utilize this defensive orientation as a guide to coping with novel or challenging social contexts (Bascoe et al., 2009; Cassidy, Kirsch, Scolton, & Parke, 1996; Davies et al., 2006; Johnston et al., 2009). Thus, more stable negative representations of family relationships may engender increasingly greater inflexibility and intractability in approaching subsequent interpersonal contexts (Cassidy et al., 1996; Johnston et al., 2009; Sturge-Apple et al., 2008). For example, psychobiological conceptualizations propose that greater proficiencies in processing and interpreting conflict in the family may, over time, sensitize children to perceiving and identifying threatening interpersonal stimuli (e.g., hostility) while restricting or blunting their recognition of benign or harmonious features (e.g., smiling, friendly approach) of their social environment (e.g., Pollak & Tolley-Schell, 2004).

In support of this hypothesis, research has shown that hostile social information processing of peer transgressions served as a mediator of the prospective link between insecurity in the interparental relationship and children's difficulties adjusting to school one year later (Bascoe et al., 2009). As a testament to the distinctive nature of this pathway, the mediational findings remained robust even after taking into consideration other potential contextual factors, including trait dispositions to experience negative affect, insecure representations of parent–child relationships, and socioeconomic characteristics. Interpreted within our cascade model, the results suggest that negative representations of the interparental relationship may increasingly generalize to other interpersonal relationships with time. From a process-oriented objective, addressing the question of why this generalization process occurs is a critical research direction. For example, based on the clinical work of Johnston and colleagues (2009), EST-R proposes that children's representations develop from scripts that consist of highly rapid and automatic rules for filtering, amplifying, and interpreting characteristics of conflicts between parents. Whereas benign or secure scripts of interparental relationships are likely to pave the way for more approach-oriented goals that engender greater diversity, flexibility, and openness to adapting to new social contexts, insecure scripts of the family are proposed to engender a defensive perceptual processing style that is significantly weighted toward filtering out benign stimuli and selectively identifying and amplifying the threatening stimuli. Through this process, children with insecure representations are proposed to reflexively and intransigently applied to interpersonal contexts, as they continue to scan new settings for old dangers (Davies, Sturge-Apple, & Martin, 2013).

Consistent with the organizational perspective in developmental psychopathology, negative internal representations are theorized to be an inextricable part of broader affective analog that is applied to an increasingly wider array of contexts. As negative processing biases proliferate across interpersonal contexts, children may evidence increasingly malevolent attributions of others, difficulties regulating negative affect, and self-protective behavioral strategies characterized by submissiveness or aggression (e.g., Dodge, 2006; Granot & Mayseless, 2012; Luebbe, Bell, Allwood, Swenson, & Early 2010). In reflecting the amplification of a negative transactional process, these extreme or atypical response styles in social contexts may evoke aversive responses from peers and adults and, in the process, become the only viable strategy for coping with pervasive social threats (e.g., teasing, bullying, rejection; Bierman, 2004;

Eggum et al., 2012; Lansford, Malone, Dodge, Pettit, & Bates, 2010). Through this canalization process (Cicchetti & Cohen, 1995; Gottlieb, 1991), plasticity and change may become progressively more limited as children become ensnared in an aversive transactional web and experience protracted forms of psychological problems characterized by internalizing and externalizing symptoms (Dodge, 2006; Granot & Mayseless, 2011, 2012; Luebbe et al, 2010; Prinstein, Cheah, & Guyer, 2005).

Alterations in Stress Response Systems

Applying a multiple-levels-of-analysis conceptualization to developmental cascades highlights the potential for physiological processes to operate as explanatory mechanisms in linkages between children's emotional insecurity and child psychopathology (Davies, Winter, & Cicchetti, 2006). As a framework for exploring the role of physiological mechanisms in developmental cascades, EST-R draws on evolutionary conceptualizations of the stress response system (SRS) as fulfilling an evolved neurobiological function of coordinating responses to environmental challenges in ways that promoted fitness in ancestral environments (Davies, Sturge-Apple, & Martin, 2013; Del Giudice, Ellis, & Shirtcliffe, 2011; Korte, Koolhaas, Wingfield, & McEwen, 2005). Physiological models highlight three modules that subserve the SRS: the sympathetic nervous system, the parasympathetic nervous system, and the hypothalamic-pituitary-adrenocortical axis (e.g., Repetti, Robles, & Reynolds, 2011). Although the parasympathetic nervous system serves to shift physiological resources toward restorative, homeostatic functioning and reduce physiological arousal (Porges, 1995), it also allows for the other physiological systems to more easily mount responses to threat by dampening its operation in stressful contexts. As the parasympathetic stress system adjusts to the stressful event, the sympathetic nervous system is charged with the task of priming the body to respond to the stressor over relatively short temporal spans (e.g., seconds) by increasing the availability of resources (e.g., cardiac output, oxygen flow, blood glucose levels) (Porges, 2006). Finally, the hypothalamic-pituitary-adrenocortical axis and its end product of cortisol constitutes a third wave of responding to stressful events that mobilizes energy (e.g., glocuose, oxygen) and modulates processing, encoding, and memory consolidation of emotionally significant events over relatively longer periods of time (e.g., minutes; Gunnar & Quevedo, 2007; Juster et al., 2011).

Allostasis and allostatic load are primary concepts in developmental psychopathology that help to inform an understanding of the role of the SRS in typical and atypical development (Cicchetti, 2011). Allostasis refers to the process by which biological set points in homeostasis are temporarily altered to meet the challenging dynamics of the environment and preserve the internal integrity of the organism. However, in highlighting the operation of allostatic load, successive cycles of allostasis engendered by repeated exposure to environmental adversity are theorized to result in wear and tear on the body that undermine the integrity of multiple domains of functioning. Thus, in socialization environments characterized by low levels of interpersonal threat, models of allostasis (e.g., Evans, 2004; McEwen, 2002) propose that healthy activation of SRS components consist of (1) mounting psychophysiological responses that are commensurate with the challenge posed by the stressor, and (2) facilitating the return to homeostatic functioning following the termination of the stressor through disengagement of stress-responsive physiological processes. Conversely, shifts in the operation of the SRS beyond of the homeostatic zones of functioning carry the advantage of increasing children's capacity to adapt to the formidable physiological demands of coping with family adversity. Although these alterations in SRS may take the form of either dampened or heightened physiological arousal, both types of allostatic load are posited to compromise physical and mental health by undermining immune, metabolic, cardiovascular, and central nervous system functions (Ganzel & Morris, 2011; Juster et al., 2011; McEwen, 2006; Turnbull & Rivier, 1999).

Building on allostatic load models, EST-R proposes two primary pathways through which children's prolonged concerns about insecurity may eventuate in psychopathology (Davies, Sturge-Apple, & Martin, 2013). As the first component of the multifinality process (Cicchetti & Rogosch, 1996), insecurity in the interparental relationship may increase children's vulnerability to psychopathology by amplifying their physiological arousal in one or more components of the SRS. The bases for this hypothesis are rooted in earlier behavioral conceptualizations of sensitization and its prediction that experiential histories of adversity in the interparental relationship pose a risk to children's adjustment by progressively intensifying their emotional and behavioral distress in the face of subsequent interparental conflicts (Cummings, 1998; Davies, Meyers, Cummings, & Heindel, 1999; Goeke-Morey, Papp, & Cummings, 2013). As a second component for the multifinality process, translation of the attenuation hypothesis to EST-R generates the prediction that insecurity may adversely impact

children's mental health by inhibiting the operation of physiological responses to threat (Susman, 2006). The dampening of neurobiological systems (e.g., sympathetic nervous system, hypothalamic-pituitary-adrenal axis) may specifically reflect the activation of processes designed to thwart the toxic effects of chronic physiological arousal to threat (Susman, 2006).

Despite the formulation of theoretically guided predictions, studies have yet to directly delineate SRS reactivity and activity as mediating mechanisms in associations between insecurity and children's developmental outcomes. Rather, most of the work has focused on understanding how children's physiological reactivity to interparental events may inform an understanding of the link between interparental conflict and child adjustment without consideration of children's signs of insecurity (e.g., Davies, Sturge-Apple, & Cicchetti, 2011; Davies, Sturge-Apple, Cicchetti, & Cummings, 2007; El-Sheikh, 2005; Hinnant, El-Sheikh, Keiley, & Buckhalt, 2013). Moreover, the rare empirical efforts to delineate the developmental implications of the multivariate interplay between signs of insecurity and physiological functioning have generated a complex, inconsistent pattern of results. For example, as a central indicator of children's insecurity, toddler fearful reactivity to conflict predicted attenuated sympathetic nervous system activity but evinced no associations with cortisol or parasympathetic nervous system functioning (Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009). In contrast, observer and maternal ratings of children's fearful distress reactivity to interparental conflict during the early elementary school years were both associated with higher, current levels of cortisol reactivity to conflict even after taking into account other forms of responding (i.e., hostility, involvement) and demographic characteristics (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; also see Koss et al., 2013).

Solving the inherent complexity generated by the multilevel cascade models of insecurity will likely require careful attention to multiple parameters in the proposed pathways. First, physiological activity and stress reactivity are likely to play distinct roles in elucidating the sequelae of insecurity. Thus, part of the inconsistency in the aforementioned findings may be attributable to differences in whether the study focused on baseline physiological activity (Davies, Sturge-Apple, Woitach, & Cummings, 2009) or physiological reactivity (Davies et al., 2008). Second, if atypically low or high patterns of physiological functioning may be pathogenic as EST-R postulates, then the conventional approach of utilizing linear conceptualizations of physiological functioning may not sensitively discriminate between normal and abnormal neurobiological processes. As the work of Koss and colleagues (2013) illustrated, pattern-based methodological approaches such as growth mixture modeling offer powerful new tools for identifying qualitatively different types of psychophysiological responses that are more amenable to capturing multifinality in models of security. Third, although there is extensive regulation among the three components of the stress response system via a central control center located in higher order limbic structures (e.g., Gold & Chrousos, 2002), different systems can assume dominance within the response hierarchy. Accordingly, one implication of the distinctive nature of the physiological systems is that uniformity in the mediational pathways should not be expected across the modules of the SRS. Moreover, this also suggests that consideration of individual differences in the higher order interrelationships among the SRS modules may offer further insight into the specific physiological patterns of responding that may mediate insecurity (Davies, Sturge-Apple, & Martin, 2013; El-Sheikh et al., 2009). Fourth, sensitizing and attenuating pathways may be part of differing strategies children use to defend against interparental threat. For example, insecurity manifested in high levels of vigilance, freezing, and masking forms of distress may be accompanied by different patterns of physiological reactivity than tendencies to express insecurity by directly engaging the threatening stimuli through aggression and hostility (Davies & Martin, 2013; Davies, Sturge-Apple, & Martin, 2013).

Open systems conceptualizations of developmental psychopathology further highlight the significance of identifying characterological, developmental, and contextual sources of multiplicity in physiological pathways (Davies & Cicchetti, 2004; Richters, 1997). At the characterological level, different psychophysiological cascades of insecurity may be foreshadowed by individual differences in children's earlier temperamental dispositions. In support of this hypothesis, interparental conflict was associated with greater cortisol reactivity to interparental conflict over time, which in turn was associated with concurrent increases in internalizing symptoms only for children with inhibited temperament. Conversely, for children with bold temperaments, exposure to interparental conflict predicted dampened cortisol reactivity to conflict and, in turn, greater attention difficulties and hyperactivity (Davies, Sturge-Apple, & Cicchetti, 2011). At a developmental level, allostatic load models in developmental psychopathology emphasize the possibility of sensitive periods of plasticity and susceptibility in the context of adverse environments (e.g., Curtis & Cicchetti, 2007; Ganzel & Morris, 2011; Mead, Beauchaine, & Shannon, 2010). For example, it is possible that the confluence of neural,

hormonal, and brain changes may bias physiological systems toward attenuating or sensitizing patterns of reactivity at different developmental periods (Ganzel & Morris, 2011). At the level of contextual conditions, prevailing neurodevelopmental conceptualizations posit that calibration of physiological systems may vary as a function of the chronicity of the adversity (Ganzel & Morris, 2011; Susman, 2006). For example, sensitization of the hypothalamic-pituitary-adrenocortical axis may be natural response in the early stages of defending against threat in the interparental relationship. However, as insecurity persists over a longer developmental span, inhibition of the axis may occur to prevent chronic overarousal of the stress system in response to danger (Susman, 2006).

Cascading Effects on Ethological Systems of Approach

Prolonged wear and tear on the stress systems is not the only psychobiological consequence of emotional insecurity. Resource depletion models in human behavioral ecology and developmental neuroscience underscore the importance of efficiently and equitably distributing a limited reservoir of psychobiological resources to multiple developmental and cognitive tasks necessary to support survival and reproductive success across the lifespan (Baumeister, Vohs, & Tice, 2007; Borgerhoff Mulder & Schacht, 2012; Kaplan & Berman, 2010; Posner, 2012; Van Der Meer et al., 2010). Thus, biases toward pursuing selective goals will necessarily limit the resources that can be devoted to other significant domains of functioning (Ford, 2009). In integrating resource depletion conceptualizations, a central thesis in our theory is that prolonged experiences with insecurity in the interparental relationship will tip the balanced allocation of psychobiological resources toward investing in immediate personal safety at the cost of equal investment of resources towards mastery of other important goal pursuits (Davies, Winter, & Cicchetti, 2006).

Although enlisting resource depletion models provided a general foundation for understanding how and why insecurity may be associated with a multitude of negative sequelae, it offers little direction in identifying specific cascade processes. To address this significant conceptual gap, EST-R offers a novel framework that considers the interplay between the operation of the SDS and other ethological modules that play a significant role in organizing approach behaviors necessary to promote fitness (Figure 6.3). These evolved modules represent discrete neurophysiological systems, each organizing specific patterns of affect, information processing, and behavior in service of a unique adaptive function. This broader evolutionary view supports several assumptions, including (1) the common phylogenetic heritage of nonhuman animals and humans produced similarly functioning ethological systems that allow for cross-species comparisons; (2) each behavioral system will be responsive to particular environmental contexts and perceptual cues providing information relevant to their function; and (3) individual differences in psychological adjustment are likely due to differences in the functioning within specific behavioral systems (e.g., prototypic phenotypes) and the relationships between behavioral systems (Buss, 2008; Davies & Sturge-Apple, 2007; MacDonald, 2012; MacDonald & Leary, 2005).

In further elaborating on the last principle, the interplay between behavioral systems with incompatible functions reflect mutually inhibiting relationships with one another that require organisms to negotiate competing motivational demands (Buss, 2008; Davies & Sturge-Apple, 2007; MacDonald, 2012; MacDonald & Leary, 2005). Translated to EST-R, SDS and its behavioral goal of defusing threat are fundamentally at odds with the functions of approach systems or ethological modules. Given that protecting oneself from threat is prioritized in the hierarchy of goal systems, activation of the SDS system is theorized to supersede the salience of approach systems as organizers of behavior. During childhood, the protracted operation of the SDS in the interparental relationship may specifically increase the likelihood of psychopathology by undermining the operation of three approach systems: exploration, affiliation, and caregiving. In the following sections, we offer a synopsis of how these systems may serve as mediators of insecurity in the developmental cascade.

The Exploratory System. As depicted in Figure 6.3, the broad adaptive function of the exploratory system of accessing basic survival materials is achieved through the more specific behavioral or external goal of gaining familiarity and mastering of the physical world (Davies & Sturge-Apple, 2007). Efficient functioning of the exploratory system and increased likelihood of success in achieving the external goal is reflected in action tendencies characterized by intrinsic motivation and behavioral efforts to approach, manipulate, and understand the workings of the physical world. From the multilevel lens of developmental psychopathology (Cicchetti & Curtis, 2007), the exploratory system is catalyzed by neurobiological substrates (e.g., dopaminergic reward mechanisms) that incentivize and reinforce curiosity, approach to novelty, and other exploratory behaviors (Depue & Fu, 2012; Panskepp, 1998; Panskepp & Moskal, 2008).

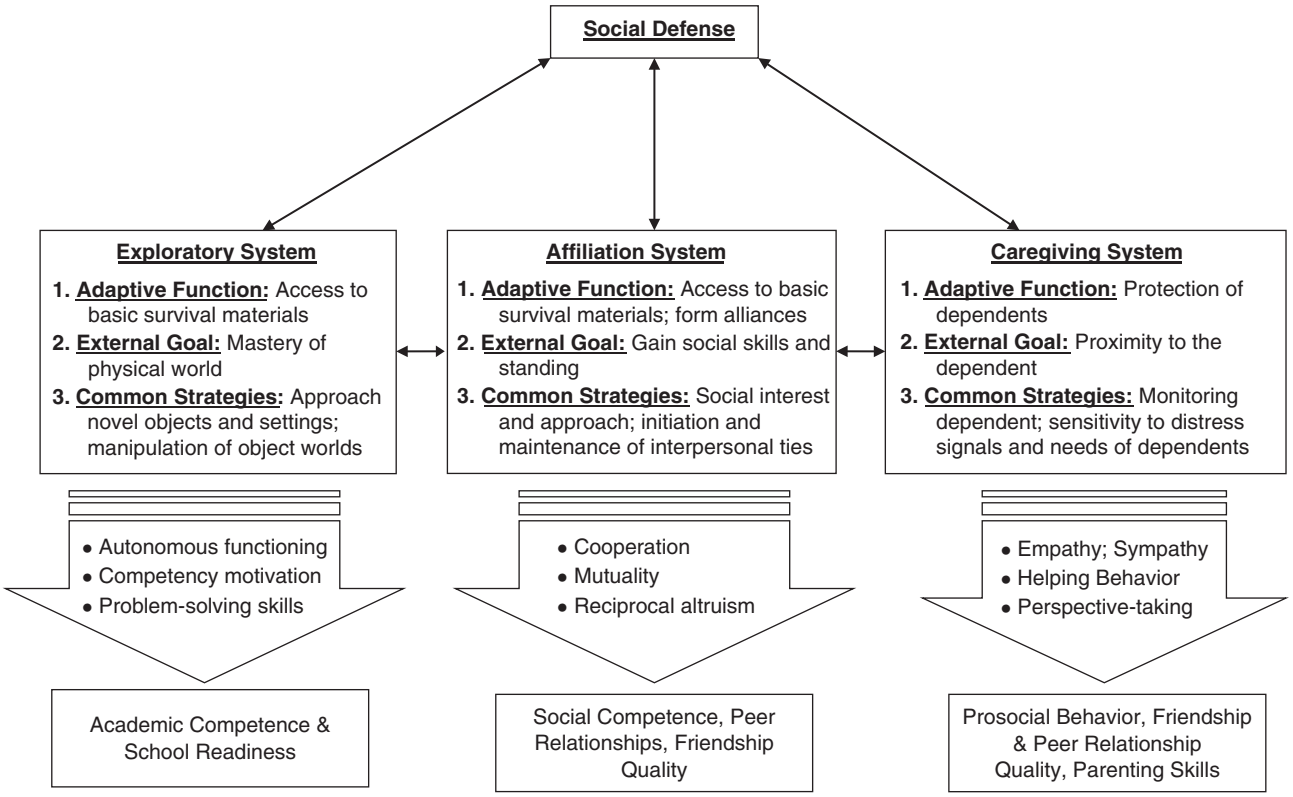


Figure 6.3 A model illustrating how the SDS impacts children's competence in multiple domains by altering the operation of ethological systems that organize approach motives and behaviors.

Given the dialectical opposition of the SDS and exploratory systems, EST-R proposes that recurrent experiences with insecurity in the interparental relationship substantially undermine children's capacities to explore the environment. As part of this process, defensive, reflexive, and automatic reactivity to threat in the interparental relationship may compromise executive functioning and regulatory abilities by reducing neural activity in the brain regions (e.g., prefrontal cortex) organizing reflective engagement with environmental stimuli (Blair & Raver, 2012). Thus, over time, diminished engagement in the physical environment is specifically proposed to compromise resourceful and flexible problem-solving (e.g., set shifting, planning), perceived academic efficacy, and ultimately intellectual performance (Blair & Diamond, 2008; Davies, Woitach, Winter, & Cummings, 2008; Quinkert et al., 2011). Although research has yet to systematically test these hypotheses, initial empirical tests are promising. For example, children's insecure representations of the interparental relationship were associated with increasing attention problems over a one-year period. In the second part of the cascade, attention problems, in turn, predicted broader school adjustment problems (Davies et al., 2008).

The Affiliative System. Figure 6.3 also illustrates that the SDS may limit the opportunities of insecure children to pursue affiliative goals of achieving access to survival materials and social standing through formation and maintenance of cooperative alliances (Irons & Gilbert, 2005; Markiewicz, Doyle, & Brendgen, 2001). The affiliative system is specifically designed to lubricate and sustain cooperative social interactions through the organization of affect expressions (e.g., warmth, affection, trust) and behavioral displays of affiliation and play (e.g., smiling, cooperative play, humor) (Davies & Sturge-Apple, 2007; Furman, 1999; Gordon, Martin, Feldman, & Leckman, 2011). Dopaminergic and opiate systems and hormones such as oxytocin subserve the affiliative system as the foundation for social behavior by stimulating and regulating social bonding, shared attention, eye gaze, behavioral synchrony, and trust (Depue & Collins, 1999; Depue & Morrone-Strupinsky, 2005; Feldman, 2012; Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005). The antithetical nature of the relationship between the inhibitory function of SDS and the approach goals of the affiliative system is supported by research demonstrating that anxiety and hypervigilance interfere with the processing of positive

social cues (Clark, 2001; Frewen, Dozois, Joanizze, & Neufeld, 2008; Hofmann, 2007; Taylor, Bomyea, & Amir, 2010). Difficulties in the processing of social information (e.g., hostile attribution biases, threat perceptions), in turn, increase children's vulnerability to victimization, peer rejection, and poor social competence (e.g., Bascoe et al., 2009; Crick & Dodge, 1994; Taylor, Sullivan, & Kliewer, 2013). Thus, the primacy of the SDS for insecure children is likely to contribute to deficits in social competence by directly interfering with the affective, information processing, and behavioral functioning of the affiliative system.

Recurring activation of the SDS in the interparental relationship is also theorized to indirectly undermine children's social competence by limiting the contextual experiences that might promote the elaboration and efficient functioning of the affiliative system. Experiences with family adversity in conjunction with tendencies to employ fight, flight, or avoidance behaviors as defense strategies are theorized to constrain insecure children's opportunities to elaborate and practice behavioral strategies for forming and maintaining cooperative alliances (Irons & Gilbert, 2005; Markiewicz, Doyle, & Brendgen, 2001). Early acquisition and refinement of social skills for establishing companionship, cooperation, mutualism, and reciprocal altruism are the key building blocks for social competence and harmonious, mutually beneficial peer relationships (Deater-Deckard & Petrill, 2004; Lindsey, Cremeens, Colwell, & Caldera, 2009). Poor social competence, externalizing and internalizing problems, and impulsive or anxious behaviors, in turn, reduce the child's value as a potential play partner and contribute to peer experiences (e.g., rejection, victimization) that serve to sustain insecurity in the long run (Beirman, 2004; Cook, Williams, Guerra, Kim, & Sadek, 2010; Hanish & Guerra, 2000; 2004).

The Caregiving System. The caregiving system functions to organize feelings of empathy, attunement to dependents, and behavior that serves the adaptive function to protect dependents and reduce their distress (Brown, Brown, & Preston, 2011; Davies & Sturge-Apple, 2007; Swain et al., 2012). Although the caregiving system is largely considered in relation to its role in parenting and likely remains relatively underdeveloped during childhood and early adolescence, its adaptive role in organizing the care of offspring requires the development of sensitivity and responsiveness to others' needs early in the lifespan. Due to their common phylogenetic history, the affiliative and caregiving systems share some physiological, affective, and behavioral strategies (e.g., the neuropeptides oxytocin and vasopressin) in motivating and rewarding behaviors that assist others. However, other underlying biological agents (e.g., estrogen) are distinct in serving the caregiving system (Depue & Morrone-Strupinsky, 2005; Galbally, Lewis, van IJzendoorn, & Permezel, 2011; Panskepp, 1998). Thus, relative to the affiliative system and its regulation of mutualism, reciprocity, and shared identity in social relationships, the caregiving system is sensitive to contextual cues that indicate others' vulnerability (e.g., sadness, fear, illness, child-like features) and organizes cognitions and affect (e.g., compassion, sense of efficacy, protectiveness, empathy, sympathy) that bolster the individual's ability to provide protection and care (Goetz, Keltner, & Simon-Thomas, 2010; Swain et al., 2012). The neurophysiological components of the caregiving system work to both motivate approach to those in distress, while simultaneously inhibiting competing stress responses that might interfere with caregiving ability (Brown et al., 2011; Numan, 2006).

When personal distress and SDS responding overwhelm the individual's capacity to regulate or inhibit self-concern, the likelihood that children will exhibit helping behavior is drastically reduced (Batson, 1991; Eisenberg, Fabes, & Spinrad, 2006). Thus, a highly sensitive SDS system may disrupt the normative development of the caregiving system through emotional distress, egoistic concern with safety, and difficulties regulating one's behavior (Collins, Guichard, Ford, & Feeney, 2004; Shaver, Mikulincer, & Shemesh-Iron, 2010; Shields, Cicchetti, & Ryan, 1994). Reductions in the capacity for empathic responding and prosocial behavior may, in turn, contribute to poor social competence, peer rejection, and poor quality friendships (Eisenberg, Huerta, & Edwards, 2012; Eisenberg et al., 2006). Difficulties forming and maintaining positive close relationships (e.g., best friendships, romantic affiliations) during adolescence and early adulthood may further limit opportunities to practice and refine the caregiving strategies that are ultimately critical to developing competent parenting skills (Davies, Sturge-Apple, Woitach, & Cummings, 2009).

CONTEXTUAL CHARACTERISTICS

Although our literature review documents the empirical support for the mediational role of emotional security in pathways between interparental conflict and child adjustment, the research also indicates that there is substantial heterogeneity in children's reactivity to conflict. Thus, a

key question remains: Why is there considerable diversity in these mediational pathways? From a developmental psychopathology perspective, individual development is regarded as part of an ongoing interplay between the child and a constellation of family and extrafamilial processes (Cicchetti, 1993; Davies & Cicchetti, 2004). In applying this notion to emotional security models, a derivative hypothesis is that individual differences in the strength of mediation will lawfully vary as a function of the broader multilevel matrix of contextual characteristics. In the following sections, we describe the theoretical and empirical progress made in contextualizing the study of emotional security across several units of analysis, including: family processes, child attributes, and ecological characteristics.

Family Processes

The significance of understanding the developmental implications of broader family processes in EST is evidenced by the core assumption that children derive emotional security from multiple relationships in the family. Consistent with family systems theory (Cox & Paley, 1997; Davies & Cicchetti, 2004), EST further posits that comprehensively understanding the dynamics of children's emotional security within any specific family relationship (i.e., interparental dyad) is contingent on embedding the study of security within the broader family unit (Cummings & Davies, 1996; Davies, Harold, et al., 2002). To illustrate the utility of this approach, the following sections describe the theory and research on three classes of family processes that may help to account for individual differences in children's pathways of security: parent–child relationship properties, parent psychopathology, and family-level characteristics.

Parent–Child Relationship Properties

Consistent with the emphasis on the interplay between family relationships in family systems theory (Cox & Paley, 1997), EST conceptualizes the interparental subsystem as the cornerstone of the family unit. A derivative assumption is that parental frustration, distress, and relationship preoccupation in the wake of interparental conflicts negatively influence parental interactions with their children (Easterbrooks & Emde, 1988; Grych, 2002). Consistent with these predictions, interparental conflict has been associated with parenting difficulties across both short-term experimental and diary (e.g., minutes, days) designs (Almeida, Wethington, & Chandler, 1999; Jouriles & Farris, 1992) and longitudinal methodologies spanning longer time (e.g., years) periods (Sturge-Apple, Davies, & Cummings, 2006a; 2006b). Moreover, numerous parenting difficulties have been linked with high levels of interparental conflict, including emotional unavailability, diminished warmth, inconsistent discipline, psychological control, disengagement, and harsh discipline (e.g., Benson, Buehler, & Gerard, 2008; Levendosky, Leahy, Bogat, Davidson, & von Eye, 2006; Margolin & John, 1997; Owen, Thompson, & Kaslow, 2006; Sturge-Apple, Davies, & Cummings, 2006b). These parenting difficulties, in turn, have been shown to serve as partial mediators of the link between interparental conflict and children's psychological adjustment (Cummings & Davies, 2010). EST is designed to build on the mediational pathways involving interparental conflict, parenting difficulties, and child adjustment by advancing an understanding of why parenting serves as an explanatory mechanism for interparental conflict. In moving beyond our previous documentation of emotional insecurity in the interparental relationship as a mediator of the link between interparental conflict and child maladjustment, Figure 6.3 depicts three additional pathways that are generated by the integration of parent–child processes into models of emotional security.

Drawing on attachment theory (Bowlby, 1969; Goldberg et al., 1999; McElwain & Booth-LaForce, 2006), Path 1 illustrates the hypothesis that the mediational role of parenting impairments in links between interparental conflict and parenting difficulties is further explained by children's attachment insecurity in parent–child relationships. That is, destructive interparental conflict may undermine children's confidence in parents as sources of protection and support by increasing parental displays of harsh, unavailable, frightening, and frightened caregiving behaviors in interactions with their children. Resulting declines in attachment security, in turn, are proposed to increase children's risk for psychological problems (Davies, Harold, et al. 2002; Davies, Sturge-Apple, & Martin 2013). In support of this hypothesis, findings from a multiple informant study indicated that parenting difficulties characterized by low levels of warmth, emotional rejection, and poor monitoring of children's activities mediated the link between interparental conflict and child-parent attachment insecurity. Attachment insecurity, in turn, was associated with adolescent internalizing and externalizing symptoms even after specifying child insecurity in the interparental relationship as a predictor (Davies, Harold, et al., 2002). In a subsequent multimethod study, observations of diminished responsiveness by parents in interactions with their children served as an intervening mechanism in the link between destructive interparental

conflict and children's insecure representations of parents as supportive figures. In turn, insecure representations of parent–child relationships mediated associations between diminished parental responsiveness and teacher reports of poor academic engagement (Sturge-Apple et al., 2007).

As shown in Path 2, EST postulates that interparental conflict may also increase children's vulnerability to psychopathology by increasing children's attachment insecurity with parents. Thus, even after taking into account the mediational role of caregiving difficulties in parent–child interactions, exposure to interparental conflict is proposed to undermine children's confidence in parents as protective and supportive figures and, in the process, increase their risk for experiencing mental health problems. The few studies examining this pathway have yielded inconsistent results. In accord with the hypothesis, destructive interparental conflict has been shown to be indirectly related to child adjustment through its association with indices of parent–child attachment insecurity (Lindsey, Caldera, & Tankersley, 2009; Sturge-Apple et al., 2008). Conversely, other studies have found that interparental conflict is unrelated to parent–child attachment security in broader multivariate models including parenting variables as predictors (e.g., Davies, Harold, et al., 2002). Resolving the empirical inconsistencies in testing attachment security as a mediator of interparental conflict is a central part of the future agenda for emotional security theory. As a potential conceptual approach to overcoming this barrier, we argue that the complexity in the findings may be attributable, in large part, to the need for greater specificity in conceptualizing and testing the mediational pathways.

In increasing the specificity of hypotheses, EST-R specifically proposes that disorganized and disoriented (D) attachment may be the most potent explanatory mechanism among the various forms of attachment insecurity (Davies, Sturge-Apple, & Martin, 2013). In EST-R, D attachment behaviors are products of the conjoint operation of both the attachment and social defense systems. For example, children's contradictory D behaviors characterized by simultaneous or successive approach and avoidance displays can be interpreted as reflecting an impasse between attachment and defense systems. Likewise, freezing and inhibition of overt distress expressions may specifically reflect self-protective camouflaging efforts that may serve the organized SDS function of reducing children's salience as targets of oppressive adult dominants (Davies & Sturge-Apple, 2007). Thus, although children have a hard-wired, phylogenetic tendency to approach the attachment figure, the D strategy signifies that the attachment relationship is simultaneously a source of threat worthy of activation of social defense strategies. It follows, then, that the high threat value associated with witnessing frightening (e.g., hostile, aggressive), vulnerable (e.g., distressing, fearful), and volatile (e.g., emotionally labile) parental behaviors during intense bouts of interparental conflict may amplify children's social defense responses to caregivers in subsequent parent–child interactions. Consistent with this hypothesis, several studies have identified associations between high levels of interparental conflict and signs of D attachments (Johnston & Campbell, 1988; Leon, 2003; Moss, St-Laurent, Dubois-Comtois, & Cyr, 2005; Zeanah, Boris, & Larrieu, 1997). Moreover, research distinguishing between levels of disorganized and insecure patterns of attachment has shown that interparental conflict is a more consistent predictor of D attachment for mothers and fathers than general forms of attachment insecurity (Owen & Cox, 1997).

Path 3 of Figure 6.4 further illustrates the hypothesis that some parenting behaviors may play a distinct role in increasing children's worries about security in the interparental relationship. Although dimensions of interparental discord are proposed to be among the most robust risk factors of children's insecurity in interparental contexts, parental interactions with children may also increase children's vulnerability by uniquely predicting child concerns about their security in the interparental relationship. Expressions of parental disengagement, unresponsiveness, and rejection directed toward children in high conflict homes are specifically theorized to undercut the establishment of fundamental mechanisms for preserving a sense of security in the interparental relationship, including the development of emotion regulation skills, self-confidence, and coping efficacy. Although parenting practices are theorized to be modest predictors of insecurity in the interparental relationship, tests of this hypothesis have yielded variable results. For example, Sturge-Apple and colleagues (2008) reported that low levels of parental warmth were uniquely associated with children's insecure representations of the interparental relationships even after taking into account interparental conflict. Children's insecure representations of the interparental relationship, in turn, predicted higher levels of their adjustment problems. However, other studies have failed to identify significant pathways between parenting difficulties and children's insecurity in the interparental relationship (e.g. Davies, Harold, et al., 2002; Davies, Sturge-Apple, Cicchetti, Manning, & Vonhold, 2012).

Toward the goal of addressing these empirical discrepancies, tests of the mediational linkages in Path 3 could benefit from the greater conceptual depth afforded by

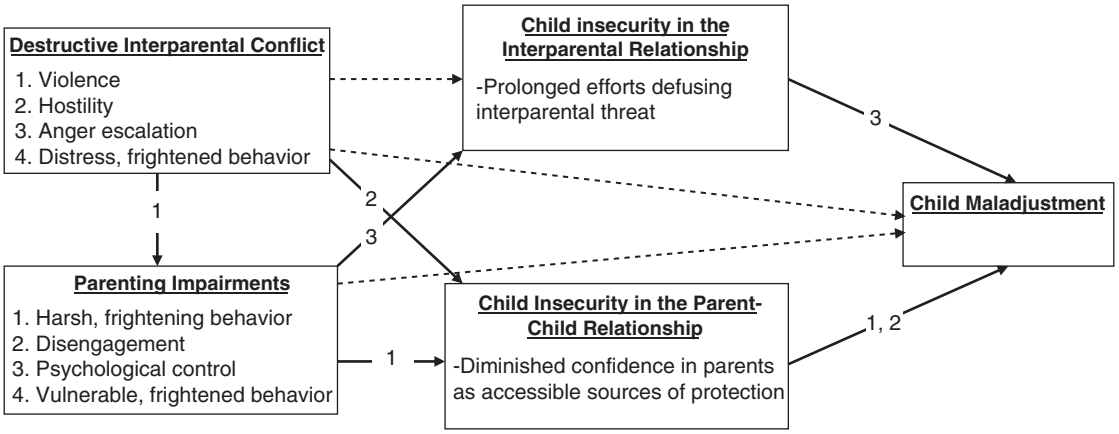


Figure 6.4 A model illustrating how parent–child relationship processes may inform how interparental conflict increases children's risk for psychopathology though their experiences with insecurity in the interparental and parent–child relationships.

EST-R. If child concerns about insecurity in the interparental relationship are underpinned by the goal of defusing parental threat, then it is plausible that children's exposure to frightening or frightened caregiver behaviors in parent–child interactions may be the central etiological agent that increases children's sensitivity to the interpersonal dangers of subsequent bouts of interparental conflict. Evaluated in this context, assessments of parental availability, warmth, and sensitivity in previous studies may be less relevant than parental behaviors that more closely signify imminent interpersonal danger. Thus, EST-R proposes that volatile (e.g., high emotional lability), highly vulnerable (e.g., intense anxiety or dysphoria), or oppressive (e.g., violent, overtly rejecting) parenting behaviors may not only increase psychopathology by undermining attachment security in the parent–child relationship but also by increasing the salience of the SDS as an organizer of behavior in interparental contexts (Davies & Martin, 2013; Davies & Sturge-Apple, 2007). In spite of the strong theoretical basis for this hypothesis, the comparative utility of different forms of parenting behavior as predictors of insecurity in the interparental relationship has yet to be systematically examined in studies. Therefore, comprehensively testing these pathways is a critical direction in future research.

Parental Psychopathology

Developmental psychopathology models of the intergenerational transmission of psychological problems from parent to offspring testify to the importance of parent psychopathology in understanding children's experiences with emotional insecurity (Cicchetti & Toth, 1998; Goodman & Gotlib, 1999). In fact, the high threat value ascribed to atypical and extreme parental behaviors in EST-R suggests that parental symptomatology may impact the mediational role of insecurity in the interparental relationship in several ways (Davies & Martin, 2013). First, parental psychological problems may serve as distal predictors of children's insecurity in the interparental relationship through its association with high levels of family adversity in the form of children's exposure to interparental conflict and frightening and vulnerable caregiving behaviors (Goodman & Gotlib, 1999; Davies, Sturge-Apple, Cicchetti, Manning, & Vonhold, 2012). For example, interparental aggression and hostility has been identified as a consistent correlate of parental depressive symptoms (Cummings, Keller, & Davies, 2005; Du Rocher Schudlich & Cummings, 2003; Shelton & Harold, 2008), antisocial personality traits (e.g., Davies et al., 2012; Jaffee, Belsky, Harrington, Caspi, & Moffitt, 2006), alcohol problems (e.g., Keller, El-Sheikh, Keiley, & Liao, 2009), and general psychological distress (Papp, Goeke-Morey, & Cummings, 2007). In further advancing an understanding of the developmental cascades of parent psychopathology, research is also beginning to identify the unfolding security processes underlying the interplay between interparental conflict and child psychopathology. For example, studies have shown that interparental conflict mediates associations between various forms of parental symptomatology and children's insecurity. Insecurity, in turn, was a robust predictor of children's psychological problems (e.g., Davies et al., 2012; Du Rocher Schudlich & Cummings, 2007).

Second, interaction models propose that parent psychopathology serves as a potentiating factor in family process models of child adjustment (Cummings, Davies, & Campbell, 2000; Goodman & Gotlib, 1999; Leve et al., 2007). Parent psychopathology may specifically create a

double whammy in magnifying the pathogenic mediational pathways involving interparental conflict, children's emotional security, and their adjustment. By virtue of the heritability of parental psychological difficulties, children of parents with psychopathology may possess genetic vulnerabilities that increase their susceptibility to adverse family experiences and processes (Leve et al., 2010; Reiss & Leve, 2007). Likewise, because higher levels of parental psychological difficulties are associated with children's exposure to maladaptive caregiving behaviors, the toxic nature of the family environment may compound the threat children experience in the interparental relationship (Davies & Sturge-Apple, 2013; Goodman & Gotlib, 1999). Against this conceptual backdrop and the extensive research on associations between parent and offspring psychopathology (e.g., Frick et al., 1992; Goodman et al., 2011), it is surprising that very little research has examined parental psychological symptoms as possible moderators in mediational pathways involving family conflict and child coping and adjustment. Notably, only one study to our knowledge has tested whether the strength of children's emotional security as a mediator of interparental conflict varies as a function of parental psychopathology. In supporting this research direction, Kouros, Merriliees, and Cummings (2008) found that paternal depression selectively moderated the first link in the mediational chain of emotional insecurity. Moderated-mediation analyses revealed that interparental conflict was a stronger predictor of emotional insecurity for children whose fathers evidenced clinical levels of depression than for children whose fathers had lower levels of depressive symptoms. In addition, maternal depression failed to moderate emotional security, but was associated with emotional insecurity in the interparental relationship, which in turn increased children's risk for experiencing psychological problems.

Toward the goal of advancing this line of research in the future, greater precision may be achieved by identifying the specific forms of parental psychopathology that serve as contextual factors in models of insecurity. For example, parent antisocial personality disorder, which is characterized by a stable pattern of intentionally violating the basic rights of others in a callous, impulsive, manipulative, and aggressive manner, may be a particularly robust risk factor in organizing children's emotional insecurity. Relative to other forms of psychopathology, parental antisocial personality symptomatology is a robust predictor of interparental aggression (Humbad, Donnellan, Iacono, & Burt, 2010; Jaffee et al., 2006; Magdol et al., 1998). In tracing its implications for children's emotional insecurity, research has also shown that maternal antisocial personality symptomatology was the only predictor of interparental aggression in a model that contained other forms of maternal psychopathology including depressive, anxiety, and substance use problems. Moreover, interparental aggression, in turn, was associated with greater signs of child emotional insecurity (Davies et al., 2012). In contrast, low self-worth, hopelessness, loss of interest, and depressed affect underlying depressive syndromes may engender more disengaged and dysphoric expressions of interparental conflict. For example, Du Rocher Schudlich and Cummings (2007) revealed that both paternal and maternal depressive symptoms were indirectly associated with children's emotional insecurity through the mediational role of depressive (e.g., sadness, withdrawal, distress) styles of interparental conflict. Although studies have not examined the impact of parent psychopathology on emotional security within the parameters of EST-R, it is plausible that specific features of parental psychopathology may result in children enlisting different insecure strategies to defend against interparental threats (see Person-Based Taxonomy section). Taken together, the conceptual and empirical progress provides foundations for future research to consider how the unique features and facets of psychopathology in parents may differentially operate in the process models of interparental discord and children's emotional insecurity.

Family-Level Characteristics

Family systems theory offers a useful heuristic for integrating the study of children's emotional security and developmental psychopathology in a broader family context (Davies & Cicchetti, 2004). According to the theory, broader stressors and perturbations in the family unit may provoke reorganizations that change the nature of interdependencies among family relationships and the functioning of individuals (Cox & Paley, 1997; Grych, 2002). Family-level characteristics are theorized to be central agents in regulating transactions between family members because they reflect higher order operations in the family that transcend individual and dyadic relationships (Davies & Sturge-Apple, 2014). Thus, in keeping with the potentiating and protective frameworks in developmental psychopathology, interrelationships among the interparental subsystem, children's emotional security, and child development may vary depending on properties of the larger family system (Erel & Burman, 1995; Grych, 2002).

Family instability and cohesion constitute two central family-level factors that may assist in deciphering how children cope and adjust to interparental conflict. Characterized by the accumulation of disruptive family events

(e.g., caregiver changes, job/income loss, family deaths, caregiver romantic relationship changes), family instability functionally reflects breakdowns in the ability of the family unit to serve as a source of stability, continuity, and predictability in the lives of children (Ackerman, Kogos, Youngstrom, Schoff, & Izard, 1999; Forman & Davies, 2003). Conversely, family cohesion is conceptualized as the degree of emotional closeness, warmth, and harmony in the family unit (Olson, Russell, & Sprenkle, 1983; Minuchin, 1974). Given their designation as processes at the whole family unit, family instability and cohesion are theorized to regulate the strength of mediational pathways involving emotional insecurity. Consistent with this thesis, family instability has been empirically identified as a moderator in the first link in the mediational chain through its effect on potentiating or amplifying associations between interparental conflict and children's emotional insecurity. Family cohesion, in contrast, moderated the second stage of the unfolding mediational pathway as a protective factor. More specifically, children's insecurity in the interparental relationship was more weakly associated with their psychological problems when they experienced higher levels of family cohesion (Davies, Harold, et al., 2002). Although additional analyses indicated that associations between interparental conflict and child insecurity did not vary depending on the level of cohesion (Davies, Harold, et al., 2002), other research has shown that arguments between parents more weakly predict children's perceptions of threat in the interparental relationship when there is higher warmth and cohesion in the family unit (Lindahl & Malik, 2011). Thus, it is premature to settle on any definitive conclusions without additional research.

Family systems frameworks also underscore the usefulness of analyzing interpersonal boundaries to more firmly contextualize interparental conflict and child emotional security in the whole family system (Davies, Cummings, & Winter, 2004). Boundaries in family relationships are defined by characteristic ways of exchanging resources, information, and materials in the family unit. Although theory and research on family systems has identified a number of different configurations of emotional and relational functioning in the family, cohesive, disengaged, triangulated, and enmeshed patterns of communication have been most consistently delineated in empirical work (Davies et al., 2004; Johnson, 2010; Kerig, 1995; Kretchmar & Jacobvitz, 2002; Minuchin, 1974). Flexible, well-defined boundaries characteristic of cohesive families provide children with ready access to resources (e.g., warmth, support, guidance) while respecting their autonomy and individuality. Conflict and distress among family members tend to be mild, well managed, and encapsulated within interparental, parent–child, and sibling relationships and are substantially outweighed by warmth, affection, and autonomy support. Thus, children in these families are theorized to be more likely to experience trajectories of security and healthy psychological functioning.

Children growing up in families with the other types of boundaries are proposed to fare significantly worse than children in cohesive families. Overly rigid, thick, and inflexible boundaries in disengaged families block access to support, protection, and other resources across family subsystems. Consequently, high levels of emotional detachment, apathy, and alienation are commonly accompanied by bouts of hostility that collectively serve to increase or maintain psychological distance between family members. In contrast, enmeshed families are characterized by weak metaphorical boundaries in which children's access to resources commonly comes at a price of a loss of autonomy and undue exposure to discord. Thus, any displays of warmth and support commonly occur in a larger context of family expressions of psychological control, intrusiveness, and hostility that tend to proliferate seamlessly across individuals and relationships (Davies et al., 2004; Jacobvitz, Hazen, Curran, & Hitchens, 2004; Kerig, 1995). Likewise, triangulation in families reflects various blends of enmeshment and disengagement across family subsystems and individuals in which family members form defensive alliances (i.e., enmeshed component) against another individual or subsystem (i.e., disengaged component). For example, in detouring families, children's difficulties intensify as they serve to increase closeness between parents who are in an otherwise unhappy relationship. Conversely, the psychological burdens of serving as caretaker, confidante, or guardian in parent–child coalitions pose its own set of risks for children (Buehler & Welsh, 2009; Johnson, 2010; Kerig, 1995).

As a first test of the utility of family boundary conceptualizations for understanding children's emotional security, Davies et al. (2004) utilized pattern-based analyses to identify both enmeshed and disengaged family typologies. Whereas each type of boundary disturbance experienced elevated levels of interparental conflict and distress, enmeshed families exhibited greater coparenting disagreements and higher levels of parent acceptance, inconsistent discipline, and psychological control than did disengaged families. Although children in enmeshed and disengaged families exhibited higher levels of child insecurity in the interparental relationship relative to cohesive families, the family enmeshment profile was associated with the most widespread signs of emotional insecurity.

Moreover, emotional insecurity was a more consistent mediator of psychological problems experienced by children in enmeshed families than it was for their counterparts in disengaged families. Thus, one possibility is that diffuse boundaries evident in enmeshed families fuel concerns about insecurity by "breaking down any affective distance between the children and the parents' distress" (Coyne, Downey, & Boergers, 1992, pp. 222). Future progress depends on more comprehensively capturing family level processes and their implications for security pathways. For example, research has yet to examine whether the mediational role of emotional security may vary depending on the occurrence and type of triangulating relationships in the family (e.g., detouring, parent–child coalitions). Likewise, the earlier findings by Davies et al. (2004) raised the possibility that enmeshed and disengaged family dynamics may engender different expressions of children's insecurity in the interparental relationship. Thus, as we outline in the Future Directions section, EST-R may be used as a guide in examining whether qualitatively distinct SDS profiles of insecurity evolve from different constellations of interparental and family-level functioning (Davies & Sturge-Apple, 2007).

Child Characteristics

Although it is important to note that a variety of child attributes may figure prominently in the variability of children's adaptation to interparental conflict, we selectively focus on two primary dimensions for the sake of illustration: gender and temperament.

Gender

Although child gender has garnered considerable scientific attention in models of interparental conflict (e.g., Davies & Lindsay, 2001; Emery, 1982; Rutter, 1970; Snyder, 1998), its significance in advancing an understanding of emotional security has yet to be compellingly demonstrated. Part of this knowledge gap can be attributed to the tendency in the literature to ignore child gender in reporting findings (Cummings & Davies, 2010). In spite of calls to move the study of gender from its background or nuisance variable designation to a more central role in models of interparental conflict (Davies & Lindsay, 2001; Snyder, 1998), gender has not been systematically incorporated in empirical tests of emotional security. For example, a small subset of studies in Table 6.2 reports on findings from analyses of child gender as a covariate (29%) or moderator (35%) in mediational tests of emotional security. Tendencies to relegate gender to a peripheral variable in emotional security research are understandable to some degree. Research over the past two decades has largely focused on testing emotional security as an explanatory mechanism within increasingly sophisticated models of family relationship processes. Thus, advancing to the use of complex blends of mediation and moderation required to incorporate gender in analyses hinges first on the compellingly documenting that emotional security mediates interparental conflict. Moreover, the small sample sizes of many of the earlier studies were ill equipped to sensitively examine the role of gender in models of emotional security.

The recent accumulation of evidence of emotional security as a mediator of interparental conflict now raises a critical question: What role, if any, might gender play in elucidating child emotional insecurity as a mediator? Our tentative answer is that child gender, in itself, is unlikely to assume center stage in the objective of identifying the multiple pathways between family processes, emotional insecurity, and child adjustment. Our conceptualization of security does not regard gender as a primary source of variability in children's vulnerability to interparental conflict. Within EST-R, the survival value of guarding against social threat and competition is theorized to be comparable for males and females (Davies & Sturge-Apple, 2007). As a result, the function, developmental origins, and implications of the SDS are not expected to vary widely across child gender, particularly in comparison to other potential contextual factors. Rather, gender is conceptualized as a proxy for a set of more operative mechanisms that more directly shape children's vulnerability to interparental conflict (Davies & Lindsay, 2001).

It may still be tempting to posit that gender plays a significant role in altering the power of emotional security as an explanatory mechanism. After all, early studies underscored the possibility that boys and girls may express their insecurity in different ways. For example, prior to adolescence, some studies have reported that girls respond to conflict between adults through subtle channels of distress, worry, and fear, whereas boys react with greater anger and aggression (Davies & Lindsay, 2001; Snyder, 1998). Research during adolescence further indicated that girls exhibited higher levels of emotional and behavioral involvement in conflicts between their parents (e.g., Buchanan, Maccoby, & Dornbusch, 1991; Davies & Lindsay, 2001; Gore, Aseltine, & Colten, 1993). Consistent with this work, gender comparisons in the psychometric analyses of the first survey of emotional insecurity indicated that adolescent girls reported higher levels of emotional reactivity, avoidance, involvement, and destructive internal representations than boys did (Davies, Forman, et al., 2002).

However, in the context of the broader literature on gender effects, these findings have not been replicated with enough regularity to make any firm conclusions. For example, in the five studies in Table 6.2 that explicitly examined associations between gender and signs of emotional insecurity, gender failed to predict emotional security or alter its power as a mediator (Davies, Cicchetti, & Martin, 2012; Davies, Sturge-Apple, Bascoe, & Cummings, 2014; Du Rocher Schudlich & Cummings, 2007; El-Sheikh et al., 2008; Kelly & El-Sheikh, 2013).

Even if future research identifies gender as a unique predictor of signs of insecurity, it does not address the qualitatively different question of whether mediational pathways involving interparental conflict and children's emotional insecurity and psychological problems differ significantly for boys and girls. That is, identifying gender differences in absolute levels of children's reactivity to conflict is orthogonal to understanding whether the developmental precursors and implications of children's concerns about emotional security vary as a function of their gender. In more directly addressing this question, the six studies in Table 6.2 that explored child gender as a moderator of insecurity failed to find any evidence that the mediational paths involving emotional security varied by gender (Cummings et al., 2006, 2012; Davies, Cicchetti, & Martin, 2012; El-Sheikh et al., 2008; Kelly & El-Sheikh, 2013). Moreover, the null findings do not appear to be attributable to statistical power issues, as the average sample size in these studies was 220. Thus, consistent with EST-R, the most parsimonious interpretation of the findings is that child gender offers little in the way of uniquely explaining children's security-related vulnerability to interparental conflict.

However, at this juncture, it may be premature to completely discard child gender from models of emotional security. Null, inconsistent, and weak findings on gender may still simply be the result of a failure to fully consider gender processes within conceptualizations of insecurity (Snyder, 1998). Interpreted within a developmental psychopathology framework, gender-linked characteristics may confer both potentiating and protective factors that may offset each other. First, tendencies for girls (particularly in adolescence) to experience higher levels of communion or interpersonal connectedness may signify that they have stronger emotional stakes in monitoring family relationship dynamics (Davies & Lindsay, 2004). Thus, when they are repeatedly exposed to destructive interparental conflict, they may be more vulnerable than boys to experiencing emotional insecurity. Yet high levels of interpersonal connectedness may also signify greater acuity in identifying and processing family adversity. Thus, in counteracting their greater sensitivity to interparental anger, the resulting advantage of more sophisticated social information processing abilities may be manifested in the ability to respond to interparental conflict with more effective strategies of coping (Cummings & Davies, 2010). Second, the predominant assessment of broad composites of interparental conflict and emotional insecurity may be masking important gender differences in pathways involving distinct parameters of conflict, specific signs of emotional insecurity, and child maladjustment. For example, the moderating role of child gender in associations between interparental conflict and children's reactivity to conflict may actually vary depending on the gender of the parent, the form of the conflict (e.g., physical aggression), and the specific way insecurity is expressed (e.g., mediation, avoidance) (e.g., Crockenberg & Forgays, 1996; Davies & Lindsay, 2001; Shelton, Harold, Goeke-Morey, & Cummings, 2006). Thus, identifying gender-differentiated pathways of emotional security in future research may benefit from more fine-grained models that are specifically designed to capture gender processes.

Temperament

In capturing individual differences in characteristic ways of behaving that are largely constitutional in origin and stable across time and context (McCrae et al., 2000; Rothbart & Posner, 2006), temperament has been consistently regarded as source of heterogeneity in the adjustment of children from high-conflict homes (Crockenberg & Langrock, 2001; Davies & Cummings, 1994; Grych & Fincham, 1990). However, research on the role of temperament as a moderator of interparental conflict has produced inconsistent, complex results. For example, whereas some studies find support for the role of negative emotionality as a vulnerability factor that exacerbates the risk posed by interparental conflict (e.g., Pauli-Pott & Beckmann, 2007), other findings indicate that negative temperament is not a significant moderator of interparental conflict (e.g., Crockenberg, Leerkes, & Lekka, 2007). Still other studies report that associations between interparental conflict and infant affect dysregulation are more pronounced for children with low negative emotionality (Du Rocher Schudlich, White, Fleischhauer, & Kitzgerald, 2011).

Why might empirical inconsistencies exist in the literature? According to EST-R, the scientific complexity can be attributed, in large part, to the predominant focus on assessing children's distal outcomes that are temporally removed from the original threatening experiences of witnessing destructive interparental conflict. Repeated

exposure to interparental conflict is conceptualized as slowly setting in motion the dynamic emergence of risk processes characterized by regularity in child response patterns in specific contexts that, over time, intensify, broaden, and crystallize into the development of children's negative outcomes. Thus, temperamental attributes may play a more powerful role in altering the cascade of insecurity processes that underlie links between exposure to interparental conflict with more distal mental and physical health outcomes (Masten & Cicchetti, 2010).

Although little is known empirically about how temperament operates within models of security, EST-R proposes that temperamental attributes play a significant role in shaping the multiple mediational pathways involving interparental conflict, child insecurity, and child developmental outcomes (Davies & Sturge-Apple, 2007). As a particularly informative taxonomy of temperament for EST-R, Rothbart and colleagues parsimoniously classify child attributes into three higher order classes of behavior: (1) negative affectivity; (2) surgency; and (3) effortful control (Rothbart, Ellis, Rueda, & Posner, 2003). Whereas negative affectivity consists of intense, prolonged experiences of fear, frustration, or sadness, surgency is composed of approach behaviors that are generally manifested in high activity level, impulsivity, and intense pleasure. As forms of reactivity, negative affectivity and surgency are each defined as hard-wired, early emerging, and reflexive responses to environmental cues (Garstein & Rothbart, 2003; Putnam, Ellis, & Rothbart, 2001). In contrast, effortful control dimensions of temperament reflect voluntary, deliberate efforts to suppress and regulate forms of reactivity. According to EST-R, consideration of each of these classes of attributes may provide insight into the multiple ways temperament may alter security pathways (Davies & Martin, 2013; Davies & Sturge-Apple, 2007).

Dimensions of negative affectivity are specifically posited to calibrate the SDS to be highly sensitive to the threat cues accompanying recurrent interparental conflict (Davies & Windle, 2001). Therefore, indices of negative emotional reactivity may alter the first link in the proposed mediational cascade of insecurity by serving as both: (a) additive precursors that catalyze the SDS system in organizing insecure responses to interparental conflict, and (b) moderators that specifically increase their sensitivity to exposure to interparental conflict (Davies & Sturge-Apple 2007). However, greater precision may be necessary to discriminate between dimensions of negative emotionality. In drawing on evolutionary perspectives, Marshall and Stevenson-Hinde (2005) conceptualized fearful inhibition as a manifestation of a highly sensitive fear behavior or defense system. Thus, fearful inhibition may play a particularly strong role in increasing the saliency of the SDS as an organizer of behavior in contexts of interparental conflict (Davies & Martin, 2013). Another critical task will involve identifying whether the multiplicative interplay between temperament and interparental conflict corresponds more closely with the operation of a vulnerability or susceptibility factor. Whereas diathesis–stress models postulate that temperamental negativity is a vulnerability factor that compounds the risk associated with interparental conflict (Rothbart, 2011; Zuckerman, 1999), the differential susceptibility hypothesis proffers that children who are high in negative emotionality exhibit *susceptibility* or plasticity to both negative and supportive interparental contexts (Belsky & Pluess, 2009; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011).

Several temperamental attributes may also serve to amplify or dilute the sequelae of children's emotional insecurity. For example, difficulties preserving security may be particularly likely to proliferate and broaden across contexts for children who are predisposed to respond to challenges and stressors with intense, lengthy bouts of negative affect. Studies have provided support for associations between inhibited, reactive temperament and greater vigilance to threat and novelty, broader generalization of conditioned fear responses to other stimuli, and slower extinction of acquired fear responses (Barker, Reeb-Sutherland, & Fox, 2014; Helfinstein, Fox, & Pine, 2012; Schwartz, Stutz, & Lederman, 2012). Thus, from a developmental cascade framework, inhibited temperamental characteristics may potentiate pathways between insecurity in the interparental relationship and children's mental health difficulties by catalyzing the development of extensive negative representations of familial and extrafamilial relationships. As another example, effortful control and surgency dimensions (e.g., extraversion) may protect insecure children from developing psychopathology by bolstering the operation of approach-oriented ethological systems. For example, children who are more sociable and extraverted may be better able to weather the prolonged operation of the SDS and its pernicious impact on the social competence and peer relationships because they naturally devote greater efforts toward the affiliative system and its external goal of acquiring social skills and standing. Likewise, children with higher levels of effortful control may provide the constitutional tools (e.g., attention shifting and focusing, inhibitory control) to sufficiently manipulate and acquire knowledge about objects and establish intellectual and academic competencies even in the context of diminished resources resulting from a highly

active SDS. Consequently, the exploratory system and its goal of mastering the physical world may be relatively intact among insecure children with high effortful control.

Ecological Characteristics

Consistent with the transactional, ecological systems approach of developmental psychopathology (e.g., Cicchetti & Lynch, 1993; Cicchetti & Toth, 1997), a contextual understanding of emotional security in the interparental relationship requires consideration of the child's broader social ecology beyond the family. In highlighting the urgency of this goal, numerous calls across multiple disciplines have been made to further contextualize the study of family and developmental processes within community and cultural settings (e.g., Garcia Coll, 2005; Kazak, 2004; Parke, 1998). Because the theoretical and empirical agenda for emotional security theory over the past two decades has largely been organized around explicating the composition, family correlates, and developmental implications of emotional security as a goal-corrected system, little progress has been made in integrating the study of emotional security within an ecological framework. As a first step toward advancing this research direction, the next sections offer a preliminary conceptual heuristic for developing more formal theoretical assumptions and hypotheses by selectively focusing on three representative ecological characteristics: (1) socioeconomic, neighborhood, and community contexts; (2) race, ethnicity, and culture; and (3) peer relations.

Socioeconomic, Neighborhood, and Community Contexts

The interplay between family processes and community and socioeconomic factors are theorized to play a significant role in children's concerns about security and ultimately their adjustment (Cummings & Davies, 2010). With respect to socioeconomic difficulties, the family stress model proposes that economic upheaval and impoverishment may indirectly increase children's risk for psychopathology by undermining family dynamics (Conger & Conger, 2002; Conger et al., 2002). Empirical tests of the family stress model have shown that low income, financial instability, or economic problems are associated with lower levels of marital quality (e.g., Amato, Booth, Johnson, & Rogers, 2007; Dakin & Wampler, 2008; Falke & Larson, 2007) and child adjustment (e.g., Ackerman et al., 1999; Conger & Conger, 2002; Conger, Conger, Matthews, & Elder, 1999). Process-oriented research has also demonstrated that socioeconomic strain may undermine child coping with stress through its association with family conflict (e.g., Conger & Donnellan, 2007; Wadsworth & Compas, 2002). In signifying a complementary set of processes, other socio-ecological models have theorized that neighborhood and community characteristics play a critical role in shaping family processes that are relevant to children's concerns about safety and security (Cummings, Goeke-Morey, Schermerhorn, Merrilees, & Cairns, 2009; Margolin & Gordis, 2000; Martinez & Richters, 1993). Experiences with community violence and chaos are relatively common in the lives of families both in the U.S. and the broader world (Barber, 2008; Feerick & Prinz, 2003; Margolin & Gordis, 2000). Exposure to high levels of community and political violence place families at greater risk for high levels of conflict and a lack of cohesion (Cooley, Turner, & Beidel, 1995) and heightened risk for children's maladaptive functioning (Bradley & Corwyn, 2002; Lynch & Cicchetti, 1998).

Although research has documented the prevalence and negative family repercussions of community violence and socioeconomic difficulties, little is known about how family and economic factors operate jointly to alter child coping and adjustment. Process-oriented accounts delineating the role of children's emotional security within ecological models may be useful in advancing an understanding of precisely how and why the confluence community and family adversity may undermine child adjustment (Cummings & Davies, 2010; Cummings et al., 2009). As a primary hypothesized pathway, these frameworks postulate that community violence and socioeconomic problems may increase insecurity in the interparental relationship and ultimately its effects on child psychopathology by taxing interparental resources in ways that increase destructive interparental conflict and discord. Research by Cummings and colleagues has demonstrated that children's emotional security in various family relationships served as unique mediators between community violence exposure and their adjustment problems (e.g., Cummings et al., 2009, 2010a, 2010b). In testifying to the value of distinguishing between different forms of community violence and their implications for family violence, this programmatic work has shown that sectarian violence (i.e., between ethnic, religious, or cultural groups) is a more robust predictor of family and child functioning than other forms of violence. Consistent with the multiple pathways outlined in their socioecological model, their findings indicated that sectarian violence was related to children's emotional insecurity indirectly through its association with the greater interparental conflict. Insecurity, in turn, was associated with children's increased risk for internalizing symptoms (e.g., Cummings et al., 2010b).

Socioeconomic and community factors may not only leave indelible imprints on children by altering the very family dynamics that impact child security, but also by serving as conditions that regulate or modify mediational pathways of security (Cummings & Davies, 2010). For example, exposure to socioeconomic difficulties or community discord may prime the social defense system, resulting in high sensitivity to the subsequent deleterious effects of exposure to interparental conflict. Consistent with this premise, research has documented associations between ecological sources of stress (e.g., socioeconomic difficulties, community violence) and children's concerns about their security in family and extrafamilial contexts (e.g., Cummings et al., 2010b, 2011). However, a necessary condition for supporting this hypothesis involves demonstrating that the exposure to adversity in ecological settings is a moderator that magnifies the role of interparental conflict as a predictor of children's insecurity. Thus, future research is required to fully test this hypothesis. In providing a complementary perspective that emphasizes the operation of resilience in the face of risk conditions, it is also plausible that support and cohesion in the neighborhood (e.g., caring, reliable adult) or community (e.g., school) settings may interrupt the pathogenic processes set in motion by insecurity. For example, connection with caring adults in the community may provide the structure necessary for insecure children to accomplish goals organized by the other ethological systems (e.g., exploratory, affiliative, caregiving) and thereby may promote desirable outcomes.

Cultural Contexts

Investigations on children's emotional security have primarily focused on White populations within the United States. In the rare cases in which research samples evidenced appreciable diversity in race and ethnicity, studies typically ignore these characteristics in their conceptual and analytic approaches to testing processes models. Overcoming the dominance of Eurocentric theory and research has been highlighted as a primary goal for the field of developmental psychopathology (Garcia Coll, Akerman, & Cicchetti, 2000) and continues to be raised as a significant issue in the literature on emotional security theory (Cummings & Davies, 2002; 2010). In spite of these recommendations, limited progress has been made in integrating the study of race, ethnicity, or culture in tests of emotional security theory. Substantiating the urgency of this issue, growing Latino and African American populations in the United States are proposed to continue increase substantially for the next half century (Flores et al., 2002; U.S. Census Bureau, 2012). Moreover, ethnic and racial minority children continue to face significant challenges and risks (Kerr, Beck, Downs Shattuck, Kattar, & Uriburu, 2003; McLoyd, Harper, & Copeland, 2001). For example, African-American families are at increased risk for interparental discord and dissolution compared with European American families (McLoyd, Harper, & Copeland, 2001).

Integrating emotional security theory into a broader framework that systematically considers race, ethnicity, and culture as key processes requires consideration of the potential value of two different models: (1) ethnic equivalence and (2) cultural values (Lamborn & Felbab, 2003). Whereas an ethnic equivalence orientation yields predictions that the mediational process associations involving children's insecurity will be comparable across ethnic, racial, and cultural groups, cultural value models theorize that culture may alter the mediational pathways of emotional security. The few studies comparing the relative viability of these models have yielded inconsistent findings. In support of cultural value approaches, Parke and colleagues (2004) reported that interparental conflict was a significantly stronger predictor of psychological problems for Mexican-American children compared to White samples. Similarly, Cummings, Wilson, and Shamir (2003) reported that interparental hostility was more strongly associated with adjustment problems in Chilean children than in American children. However, American children exposed to interparental violence were significantly more vulnerable to psychopathology than were Chilean children. In adding to the inconsistencies, other studies have reported no significant differences between non-Latino and Latino samples in associations between interparental conflict and negative child outcomes (Buehler & Gerard, 2002; Stutzman et al., 2011). Examinations of racial differences in the risk posed by interparental conflict have also yielded comparable sets of complex results. For example, Nievar and Luster (2006) found that associations between interparental conflict and child adjustment problems were significant for non-Latino White families but not for African American families. Conversely, other findings have indicated that associations between interparental conflict and child psychopathology were comparable for married White and African American families (Krishnakumar, Buehler, & Barber, 2003). Thus, the small corpus of inconsistent findings does not provide evidence that substantially favors either the ethnic equivalence or cultural values models.

Even less is known about whether the mediational role of emotional security varies significantly as a function of race or ethnicity. Empirical tests of emotional security as

a mediator have yet to be directly conducted with Latino families. However, at this early juncture, the scant findings suggest that the mediational pathways involving family conflict, emotional security, and child adjustment evidence more similarities than differences across racial and ethnic groups. For example, the predictive role of interparental conflict in models of perceived threat appears to be similar in Mexican American and European American families (Tschann Flores, Pasch, & Marin, 1999). Similarly, in the only direct analysis conducted on race as a moderator of children's emotional security, El-Sheikh and colleagues (2008) reported that mediational pathways between interparental conflict, children's emotional security, and their adjustment problems did not vary for African American and White families. Further highlighting the generalizability of the findings across different races, research with predominantly Black, impoverished families have demonstrated that emotional security mediates associations between destructive interparental conflict and children's psychological difficulties (e.g., Davies, Cicchetti, & Martin, 2012; Davies, Martin, & Cichetti, 2012; Davies, Sturge-Apple, Cicchetti, Manning, & Vonhold, 2012). Thus, although it is premature to draw any definitive conclusions about the role of race and ethnicity in models of emotional security, the sparse research findings have documented the generalizability of emotional security as a mediator for families with different racial and ethnic backgrounds.

Predictions regarding race and ethnicity as moderators are commonly predicated on the assumption that they reflect fundamental differences in cultural processes. However, wide and meaningful individual differences exist within racial and ethnic groups in the adoption of cultural beliefs and practices. For example, in a sample of Mexican American families, individual differences in maternal and paternal endorsement of familism each uniquely predicted lower levels of interparental conflict (Taylor, Larsen-Rife, Conger, & Widaman, 2012). Moreover, race and ethnicity co-occur with other ecological characteristics (e.g., economic factors, community processes, family structure, racial discrimination), which may, themselves, serve as unique regulatory conditions in models of emotional security. Thus, although race and ethnicity may offer expedient starting points for the study of culture, they are, at best, unrefined markers of more significant psychocultural processes. To address this significant barrier, a critical step in advancing culturally sensitive models of emotional security is to understand how specific cultural beliefs and practices may shape the meaning of family adversity and introduce distinctive constellations of protective and patenting factors (Garcia Coll et al., 2000; McLoyd et al., 2001; Tschann et al., 2009).

In facilitating this research direction, McLoyd et al. (2001) proposed that the tendency for ethnic and racial minority families to place greater value on extended family and extrafamilial networks of support may actually serve to attenuate the risk trajectories of children from high conflict homes (Nievar & Luster, 2006). Extended kinship networks may not only be an adaptive strategy for minority families to cope with considerable social and economic disparities (Hatchet & Jackson, 1993; McAdoo, 1978; Wilson, 1989), but they may also help to minimize the repercussions of interparental conflict for children. From an emotional security perspective, interparental conflict may engender fewer child concerns about security when it occurs within a larger unit of family members. Moreover, the larger number of adult and older sibling caregivers in these families may provide children who do experience insecurity with the emotional support and guidance necessary to promote resilient developmental outcomes. In support of this hypothesis, research has demonstrated that kinship emotional support, advice, and counseling had positive effects on family relationships, parental psychological wellbeing and adolescent adjustment (Taylor, Seaton, & Dominguez, 2008).

Capturing the traditional cultural values and practices of Latino families may also advance tests of the cultural specificity and generalizability of emotional security as a mediator of interparental conflict. Greater emphasis on the importance of the family over individual differences (i.e., familism) and child dutifulness and deference to parents (i.e., respeto) may specifically change the meaning of interparental conflict for children and families who follow conventional Latino traditions (Halgunseth, Ipsa, & Rudy, 2006; Lindahl, Malik, Kaczynski, & Simons, 2004). Due to the high value placed on the family and the maintenance of order and harmony, exposure to high levels of interparental conflict may potentiate children's concerns about their security and safety in families endorsing higher levels of familism and respeto. In supporting the plausibility of this prediction, Cummings and colleagues (2003) found that Chilean children responded to videotaped simulations of unresolved conflict between adults with greater distress and more negative expectations about interadult relationship quality than did American children. However, the high value placed on the family may predispose family members to provide support and protection to children who experience insecurity, thereby counteracting any vulnerability to psychopathology. As an alternative hypothesis, differences between families in the adoption of

Latino cultural traditions may shape how children express their insecurity rather than directly amplify or dilute emotional security as an explanatory mechanism. For example, proper respect to parents and broader devotion to the family may prompt children to suppress any overt, blatant expressions of distress and insecurity in favor of more veiled displays of fear and wariness.

Peer Relations

Peer relationships represent increasingly important sources of both opportunity and challenge as children age and attempt to navigate their complex social world (Parker, Rubin, Erath, Wojslawowicz, & Buskirk, 2006; Rubin, Bukowski, & Parker, 2006). In highlighting the interplay between multiple interpersonal contexts, research has linked interparental conflict with poor peer relationships and maladaptive social behavior (e.g., more aggression, less prosocial behavior) (Gottman & Katz, 1989; Kouros, Cummings, & Davies, 2010; Lindsey et al., 2009; Lindsey, Colwell, Frabutt, & MacKinnon-Lewis, 2006; McCoy et al., 2009; Stocker & Youngblade, 1999). Consistent with this research, the ethological analysis of developmental cascades highlights how the SDS can undermine peer relationships processes through the proliferation of defensive analogs into extrafamilial settings and reductions in the salience of the affiliative system as an organizer of behavior (see Developmental Cascade Mechanisms section). Nevertheless, it is plausible that some dimensions of peer and friendship quality may serve as moderators of the mediational role of emotional security. Although some studies have failed to identify peer relationship characteristics as moderators of interparental conflict (e.g., Jenkins & Smith, 1990), research has shown that children's perceptions of peer availability and active solicitation of friendship support reduced their likelihood of exhibiting adjustment problems in the context of heightened interparental conflict (Rogers & Holmbeck, 1997). Moreover, this protective function has been documented across a range of peer relationship experiences including friendship quality, sociometric popularity, and peer support (Wasserstein & LaGreca, 1996; Xin, Chi, & Yu, 2009). In highlighting the potentiating role of friendship problems, there is also some evidence to indicate that the magnitude of the linkage between interparental conflict and psychopathology was increased for adolescents with low quality friendships (Larsen, Branje, van der Valk, & Meeus, 2007).

However, challenges and barriers remain in identifying how peer and friendship characteristics may inform an understanding of children's coping with interparental conflict. Empirical inconsistencies are evidenced by null findings on the moderational role of peer relationship qualities in models of interparental conflict (e.g., Jenkins & Smith, 1990). A large part of this gap in knowledge may be due to the failure to examine how peer and friendship experiences alter the unfolding cascade of processes in the pathways between interparental conflict and children's health outcomes. Many peer relationship and friendship experiences may buffer children from the negative consequences of exposure to interparental conflict by offering an alternative source of support and warmth. Yet, without theory, there is an insufficient base to identify the precise point in the cascade at which friendships and peer relationships serve to interrupt the unfolding pathogenic processes experienced by children from high conflict homes. In offering some conceptual direction toward this goal, EST-R proposes that the moderating roles of friendship and peer relationships are most pronounced in the second link in the mediational pathways involving interparental conflict, emotional security, and child functioning. A primary hypothesis is that supportive friendship and peer relationships may protect children who have concerns about security from developing psychopathology. From a process-oriented perspective, supportive, prosocial peer networks may counter the tendency of the SDS system to undermine the functioning of the affiliative system and propagate defensive analogs and representations across multiple social contexts. Conversely, histories of peer rejection or involvement with antisocial peers may catalyze the processes by which children's insecurity increase their psychopathology by magnifying their experiences with threatening social contexts and synergistically undermining the approach (e.g., affiliative, caregiving) systems. Consistent with many of the other dimensions of contextual characteristics in emotional security theory, efforts to understand how peer relationship processes may inform an understanding of the antecedents and consequences of emotional insecurity are still in their scientific infancy. Therefore, future research is critical for testing key predictions derived from EST-R.

DEVELOPMENTAL PARAMETERS

Integrating the psychopathology component and its accompanying consideration of risk and protective processes within the "developmental" element of developmental psychopathology requires understanding of security as a dynamic process over broad spans of time. Consistent with this theme in developmental psychopathology, an ultimate goal of EST is to characterize how children's concerns

about emotional security operate within a broader model of the shifting constellation of risk and protective processes across specific developmental periods. Although achieving this complex objective requires multiple approaches and steps, it can be concisely divided into four primary scientific directions. As outlined in Path 5 of Figure 6.1, these directions include (1) delineation of transactions of children's insecurity in broader developmental contexts; (2) characterization of the history and duration of experiences in understanding pathways of insecurity; (3) the significance of sensitive periods in understanding emotional insecurity; and (4) identification the developmental trajectories of insecurity.

Transactions of Insecurity

Developmental psychopathology strongly emphasizes that child adaptation is an evolving product of the interactions between an active, changing child within a dynamic, multilayered ecological context (Sameroff & MacKenzie, 2003). The active role of children specifically translates to the assumption that the interplay between family and child characteristics is reciprocal in nature over time. In support of this premise, there is growing evidence that children's functioning is both a precursor and product of interparental conflict. For example, Jenkins, Simpson, Dunn, Rasbash, and O'Connor (2005) found that child externalizing problems subsequently increased arguments between parents two years later, particularly in families facing additional challenges (e.g., children with high externalizing symptoms, stepfamilies). Likewise, Cui, Donnellan, and Conger (2007) examined the reciprocal interplay among interparental conflict over child-rearing and adolescent symptomatology in a series of cross-lagged autoregressive analyses over three time periods each spaced one year apart. Consistent with transactional models, adolescent depressive and delinquency symptoms were identified as both outcomes and predictors of interparental conflict.

These initial findings point to the plausibility of viewing child functioning as an antecedent and consequence of interparental relationship dynamics within a spiraling bidirectional cycle. However, little is known about how children's concerns about security may operate in shaping the course of interparental conflicts. Toward further advancing precision in the domain of children's responses, EST specifically proposes that the different forms of reactivity assume distinct roles in the unfolding bidirectional influences between interparental conflict and children's emotional insecurity (Davies & Cummings, 1994; Schermerhorn, Cummings, DeCarlo, & Davies, 2007). In the early stages of the transactional process, EST proposes that exposure to interparental conflict increases children's concerns about insecurity in the form of negative emotional reactivity. Negative emotional reactivity, in turn, increases the likelihood that children may increase their efforts to interrupt the course of escalating conflict before it substantially threatens the family system (Davies & Cummings, 1994). Relative to veiled forms of fearful distress (e.g., avoidance, freezing), more demonstrative displays of conflict reactivity manifested in involvement, mediation, or disruptive behavior are more salient to parents who are otherwise occupied with their ongoing conflict and, as a result, may have a particularly potent influence on the course of interparental disagreements. In keeping with this premise, Emery (1989) proposed a transactional cycle of effects between children and the interparental subsystem that designates children's coercive reactions to conflict as primary architects of conflict. In the initial series of unfolding processes, children respond to witnessing violence with higher levels of distress that, in turn, precipitate specific attempts on the part of children to alleviate their distress through disruptive (e.g., temper tantrums) behaviors. At the subsequent phases of this reciprocal cycle, children's misbehavior is posited to broaden and intensify in subsequent conflicts because it serves a function of distracting parents from engagement in the ongoing, aversive conflict (Davis, Hops, Alpert, & Sheeber, 1998).

Questions remain as to whether the effectiveness of children's forms of involvement in reducing conflict is sustained over broader developmental spans of days, months, or years. Rare empirical inquiries into this issue highlight the possibility that it may depend on the specific approach children use to regulate the conflict (Schermerhorn et al., 2005, 2007). Consistent with EST, these studies have shown that interparental conflict increases tendencies to exhibit forms of involvement through its association with children's negative emotional reactivity to conflict. Although forms of involvement, in turn, predicted subsequent changes in the frequency of destructive conflicts over a two-year period, whether conflicts subsided or improved varied as a function of the form of involvement (Schermerhorn et al., 2007). Higher levels of coercive involvement (e.g., yelling, causing trouble) were associated with increases in interparental conflict over a two-year span. Thus, the proposed utility of involvement in reducing contemporaneous bouts of interparental conflict was paradoxically related to greater long-term disruptions in the interparental relationship. Conversely, children's greater use of facilitative intervention behaviors

designed to diminish conflict was associated with decreases in interparental conflict over two years.

Although the mechanisms for these differential associations have yet to be identified, transactional models have posited that disruptive forms of involvement are part of a coercive family process that, while temporarily alleviating conflict, ultimately increase distress, ill-will, and hostility in both the children and parents (Emery, 1989; Patterson, 1982; Schermerhorn et al., 2007). In contrast, when children exhibit more constructive, facilitative forms of involvement, parents may be in a more reflective emotional state that allows them to sensitively respond to their children's burdensome involvement by working together in the long run to reduce their disagreements. Regardless of the explanatory mechanisms underlying the different interparental sequelae of children's forms of reactivity, advancing precision in charting transactional processes will require careful consideration of how the reciprocal interplay between child reactivity to conflict and interparental conflict over time may shift progressively over time (e.g., minutes, days, months, years). In addition, future research would be facilitated by considering the possibility that the impact of signs of child insecurity on interparental conflict may differ in magnitude or even quality across dimensions of interparental conflict. From a family systems perspective, child-related conflicts reflect perturbations in the coparental relationship, a subsystem that is theorized to be more susceptible to variations in child behavior than the more encapsulated marital subsystem (Feinberg, 2003; McHale, 1995; Lindsey, Caldera, & Colwell, 2005). Moreover, frequent disagreements about the children commonly signify extensive proliferation of stress across family subsystems (Davies, Cummings, & Winter, 2004; Kretchmar & Jacobvitz, 2002). Consequently, children's insecure reactions may be more strongly related to parental disagreements over child-related issues than conflicts about marital or adult issues. Although research has yet to systematically test children's insecurity as a predictor of different forms of interparental conflict, children's psychological difficulties have been shown to predict greater conflict parental conflicts only for arguments centered on child-related issues (Cui et al., 2007; Jenkins et al., 2005).

Experiential Histories

Developmental psychopathology is also a useful heuristic for examining how early histories of adversity may inform an understanding of children's later adaptation to interparental conflict (Davies, Sturge-Apple, Bascoe, & Cummings, 2014). According to the definition of hierarchical motility, continuity in functioning may result from the tendency for older psychological patterns to be integrated in subsequent forms of functioning. Even after considering subsequent family experiences between earlier adaptation and subsequent outcomes, hierarchical motility models translate to the assumption that early experience has a unique and persistent impact on developmental sequelae (Fraley, Goisman, & Haltigan, 2013; Roisman, Collins, Sroufe, & Egeland, 2005; Sroufe, Egeland, & Kreutzer, 1990). In the context of emotional security, this assumption raises the plausible hypothesis that earlier histories of insecurity in the interparental relationship will continue to predict subsequent trajectories later in childhood and adolescence even after taking into account contemporaneous experiences with insecurity and family adversity.

Longitudinal studies have shown that emotional insecurity is a persistent predictor of children's maladjustment across developmental periods. For example, Kelly and El-Shiekh (2013) reported that children's insecurity in preadolescence and early adolescence was associated with greater sleep problems three years later in middle adolescence. In another study, children's insecurity in second grade was associated with increases in children's internalizing and externalizing symptoms five years later in seventh grade (Cummings et al., 2012). Although these investigations provide preliminary support, they were not designed to test the enduring effects hypothesis. Because neither study incorporated family and insecurity experiences that were concomitant with the adjustment outcomes, they cannot rule out the possibility that contemporaneous adversity fully mediated links between early insecurity and child adjustment. In addressing this gap in the literature, a recent investigation reported that child interview assessments of their insecurity over a two year span in the early elementary school period uniquely predicted a multi-informant assessment of their adjustment difficulties six years later in adolescence even after controlling for concurrent and prior adolescent emotional insecurity, adolescent exposure to interparental conflict, contemporaneous family socioeconomic background, and children's sex (Davies, Sturge-Apple, Bascoe, & Cummings, 2014).

In building on this small corpus of research, key research questions still remain. What is the unique set of pathogenic processes set in motion by childhood histories of insecurity that were not accounted for by more temporally proximal experiences with interparental adversity during adolescence? As children assume greater social roles and challenges during the early school years, might this be a sensitive period for increasingly utilizing ways of

responding to family adversity (i.e., defensive analogs) as a guide in coping with complex or stressful social contexts (e.g., peer interactions)? If this developmental cascade process is delineated in future research, it may specifically provide clues into how defensive strategies displayed by insecure children may take on a "life of their own" by evolving into intractable patterns of heightened sensitivity to extrafamilial stressors and, in the process, uniquely impact their later adjustment (Bascoe et al., 2009). Likewise, it is possible that the substantial increases in social perspective taking capacities during the early school years precipitated new ways of responding to interparental conflict by sensitizing children to concerns about the safety of their caregivers (Cummings & Davies, 2010). A developmental psychopathology perspective also serves as a heuristic for proposing that insecure histories earlier (e.g., toddler or preschool period) or later (e.g., adolescence) in the life span may engender unique mechanisms that generate enduring effects of insecure experiences at different developmental periods. For example, EST-R proposes that insecurity in early childhood is largely characterized by preverbal, sensory motor patterns of emotional reactivity that are largely inaccessible to conscious processing (Davies & Sturge-Apple, 2007). Thus, the encapsulated, automatic forms of defending against threat may incur psychological burdens on children across subsequent developmental periods by impeding processing, assimilation, and integration into patterns of effortful, regulated engagement with the environment (Johnston et al., 2009).

Our developmental model of security further posits that histories of insecurity and experiences with family discord modify and shape the mediational role of emotional security (Davies, Sturge-Apple, Bascoe, & Cummings, 2014). Insecure histories may potentiate either part of the subsequent mediational cascades involving insecurity, including associations between (a) interparental conflict and emotional insecurity and (b) insecurity and the ensuing probability of developing psychopathology. Prior experiences with family adversity (e.g., interparental conflict earlier in the life span) and psychological problems are also proposed to operate in similar roles as moderators of the mediational role of emotional insecurity. For the sake of illustration, the remainder of this section addresses the legacy of earlier insecure experiences as a moderator of the association between interparental conflict and children's subsequent sense of security. Prevailing models in developmental psychopathology have offered some guidance on three plausible ways early adversity primes children to become increasingly sensitive to subsequent stress. As shown in Figure 6.5a, the stress amplification or dual-risk model proposes that early adversity serves as a risk factor that becomes disproportionately magnified following exposure to subsequent stressful experiences (Rudolph & Flynn, 2007). Support for this model in EST would be evidenced by findings indicating that childhood histories of insecurity amplify the associations between interparental conflict (i.e., the stressful event) and adolescent emotional insecurity (Davies et al., 2006). The ordinal nature of the interaction depicted in Figure 6.5a further illustrates that children with insecure histories will exhibit relatively higher vulnerability to subsequent problems even when interparental conflict is low or benign.

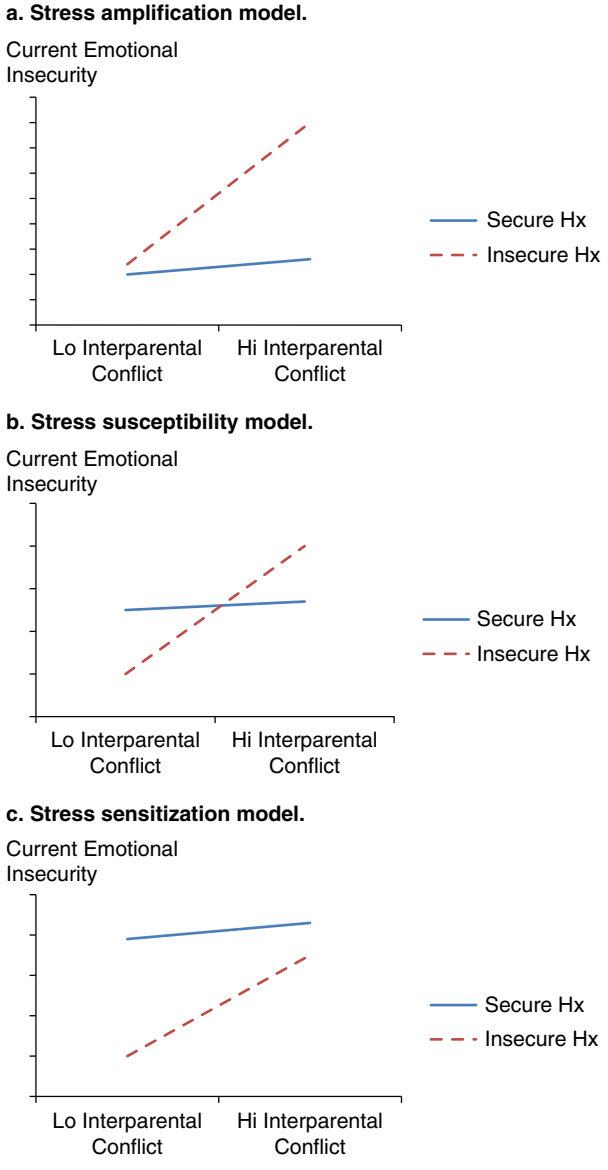


Figure 6.5 Models in developmental psychopathology. See footnote 1.

According to the second conceptualization, a stress susceptibility model postulates that children with histories of insecurity are more reactive to subsequent interparental conflict for better or for worse. Biological sensitivity to context theory and the adaptive calibration model both propose that children's early exposure to stress may amplify their biological and stress sensitivity to challenges in a way that may increase their openness to subsequent environmental input (Del Giudice et al., 2011; Ellis, Essex, & Boyce, 2005). Translated to EST, the dotted line in Figure 6.5b demonstrates that increases in destructive interparental conflict predict progressively higher levels of emotional insecurity for children with prior adverse histories of insecurity. Thus, similar to the stress amplification conceptualization, the stress susceptibility model hypothesizes that children with histories of insecurity suffer disproportionately in the face of high interparental conflict (i.e., dotted line) relative to their counterparts with secure histories (i.e., solid line). However, as the primary locus of distinction between the two models, the stress susceptibility model postulates that the greater sensitivity to environmental cues exhibited by children with insecure histories is manifested in substantially fewer concerns about their security in the context of more benign and constructive conflicts between parents. In sum, synthesis of stress susceptibility models within EST yields the prediction that insecure histories increase children's sensitivity to interparental relationship quality such that they fare substantially better than normal in benign interparental contexts, but suffer disproportionately worse fates in the face of high interparental conflict.

As a third form of moderation, the risk sensitization model proposes that histories of vulnerability lower children's subsequent thresholds of sensitivity to family difficulties (Harkness, Bruce, & Lumley, 2006; Rudolph & Flynn, 2007). Through this kindling process (Monroe & Harkness, 2005), the level of family adversity necessary to evoke children's psychological difficulties becomes progressively more mild. Thus, in models of security, the hypersensitivity of children grappling with earlier vestiges of insecurity is reflected in experiences of greater security concerns than their counterparts with secure histories only at low or mild levels of interparental conflict (Figure 6.5c). Conversely, exposure to frequent, destructive forms of interparental conflict (i.e., high levels) poses such a palpable threat that children have a general tendency to experience insecurity regardless of their prior developmental histories.

At this early stage of research, it is premature to draw firm conclusions about the role of early histories of adversity in altering the mediational pathways of children's emotional insecurity. However, a recent study provides some preliminary support for the fruitfulness of examining histories of insecurity for understanding how children contend with later conflicts (Davies, Sturge-Apple, Bascoe, & Cummings, 2014). Moderator findings specifically indicated that children who experienced high levels of insecurity during the early elementary school years exhibited greater sensitivity to subsequent exposure to interparental conflict in a for-better-or-for-worse manner predicted by the stress susceptibility framework. On the worse side of the plot (Figure 6.5b), adolescents with childhood histories of insecurity evinced greater emotional insecurity in contexts of high interparental conflict than teens with secure histories. Conversely, on the better side of the plot, higher childhood insecurity was actually associated with lower adolescent insecurity when interparental conflict was low. Testing the specificity and generalizability of these results across developmental periods, types of experiential histories, and forms of sequelae will be an important direction for future research.

Sensitive Periods

EST posits that emotional security should be associated with psychological adjustment across the lifespan. However, this moderate stability is also proposed to occur in the context of developmental transformations and burgeoning stage-salient challenges during the course of childhood and adolescence that may serve to attenuate or intensify the pathways among interparental conflict, child emotional insecurity, and adjustment (Cummings & Davies, 2010). Thus, advances in understanding emotional security in developmental context will require examining possible sources of developmental stability and change. The most common approach to addressing this issue is to focus on age as a marker of developmental differences between children. However, the analyses generally indicate that children in any one age group or developmental period do not evince heightened susceptibility to interparental conflict (Buehler, Anthony, Krishnakumar, & Stone, 1997; Buehler et al., 1998; 2007; Gerard & Buehler, 1999).

Difficulties in identifying periods of developmental susceptibility may result from the tendency to focus on associations between interparental conflict and psychopathology without consideration of how children cope in high conflict homes. For example, null findings on age as a moderator in a previous meta-analysis may be an artifact of the selective focus on examining child internalizing and externalizing symptoms as outcomes of interparental

conflict (Buehler et al., 1997). A more nuanced analysis of the unfolding processes in links between interparental conflict and child psychological symptoms raises the possibility that age may play a more significant role as a moderator of the mediational role of emotional security. Simply examining age as a moderator of associations between interparental conflict and child outcomes may miss the possibility that age-linked vulnerabilities shift across the cascade of insecurity. For example, a developmental period evidencing a potentiation in associations between interparental conflict and child insecurity may actually be counteracted by its protective function in the subsequent pathway from insecurity to adjustment problems. For example, meta-analytic results indicated that associations between interparental violence and children's distress reactions to simulated conflict were most pronounced for preschool girls in comparison to older children (Kitzmann et al., 2003). Conversely, in a meta-analysis investigating the second part of the mediational pathway, proxies of insecurity (e.g., negative emotional reactivity, perceived threat) were the weakest predictors of internalizing and externalizing symptoms for younger children in samples ranging in age from five to nineteen (Rhoades, 2008). However, in adding to the complexity, these patterns are not always replicated in studies that specifically utilize assessments of insecurity in the context of actual interparental conflicts. For example, running counter to the meta-analytic results, Cummings and colleagues (2006) found the magnitude of the link between interparental conflict and emotional security was significantly stronger for older children in a sample of 8- to 17-year-olds.

Given the nagging empirical inconsistencies, any comprehensive developmental analysis must drill to a conceptually deeper level than age or developmental period. Age is a proxy for a complex undercurrent of social, cognitive, emotional, and physiological processes that directly alter children's coping and adjustment in the face of interparental conflict. Due to the wide-ranging developmental changes across childhood, emerging psychological or physiological vulnerabilities at any one age may be offset by a comparable set of buffers and competencies. For example, interparental conflict may more easily overwhelm the regulatory capacities of preschool children. In comparison to older children, preschool children respond to conflict between adults with greater fear, distress, and threat, poorer perceived competence, and limited abilities to utilize coping strategies (e.g., Cummings, Vogel, Cummings, & El-Sheikh, 1989; Davies, Dumenci, & Windle, 1999; El-Sheikh & Cummings, 1995; Grych, 1998). Although this evidence suggests that the mediational role of insecurity may be particularly pronounced for younger children, their vulnerabilities may be offset by their lower sensitivity to adult problems and diminished social perspective taking abilities (Davies et al., 1999).

Conversely, adolescent concerns about their security and their pathogenic implications may be amplified by their greater awareness of subtle interparental difficulties, stronger dispositions to mediate conflicts, and their longer histories of exposure to interparental conflict (Cummings, Ballard, El-Sheikh, & Lake, 1991; Davies, Myers, & Cummings, 1996; Davies et al., 1999). Underscoring the salience of another developmental period, evolutionary models highlight the juvenile period (i.e., middle childhood) as a potential sensitive period or "switch point" for the translation of early stress experiences into specific strategies for regulating stress and coping with threat and conflict (Del Giuidice, Angeleri, & Manera, 2009; Del Giudice & Belsky, 2011). However, in the context of interparental conflict, these adrenarche-linked increases in children's susceptibility may be counterbalanced by tendencies to exhibit more effective emotion regulation and coping strategies than younger children and diminished sensitivity to subtle interpersonal problems relative to adolescents. Thus, no one age group or developmental period appears to be uniformly more vulnerable to experiencing insecurity and adjustment problems in the face of interparental conflict. Rather, it is likely that each age ushers in specific vulnerabilities that are counteracted by newly emerging developmental competencies and buffers.

Our analysis collectively begs the question of whether there may be an alternative way of comprehending how insecurity is linked with these age-graded vulnerabilities and competencies. The substantive emphasis on stage-salient or developmental tasks in models of developmental psychopathology offers a fruitful approach to addressing this knowledge gap (Cicchetti, 1991; 1993; Sroufe & Rutter, 1984). Within these models, development is conceptualized as a series of stage salient tasks that emerge as prominent in a particular developmental period and subsequently remain important throughout the lifespan. At each developmental stage, children are faced with resolving a number of significant tasks across multiple domains of functioning (Chase-Lansdale, Wakschlag, & Brooks-Gunn, 1995; Cicchetti, 1991; Sroufe & Rutter, 1984). Table 6.3 provides a listing of examples of major developmental challenges from infancy through adolescence. Given that these tasks are already challenging in themselves, their successful resolution may be particularly sensitive to the risk associated with children's concerns about their security (Davies et al., 2006). Moreover,

TABLE 6.3 Developmental Challenges Faced by Children From Infancy Through Adolescence

Developmental period	Stage-salient challenges
Infancy	Modulation of arousal
	Physiological regulation
	Harmonious, synchronous interactions with caregivers
	Secure attachments with primary caregivers
	Interpersonal trust
Toddler	Awareness of self as distinct
	Exploration of social and object worlds
	Emotion regulation and management
	Awareness and responsivity to standards
	Mastery, persistence, and problem solving
	Autonomy
	Understanding of internal emotion states of others
Preschool	Flexible self-control
	Self-reliance
	Initiative
	Awareness of social roles
	Gender role development
	Establishing effective peer relations
	Empathy and prosocial behavior
Childhood	Social understanding (equity, fairness)
	Gender constancy
	Same-sex friendships
	Peer relations and reputation
	Sense of industry (competence)
	Social agency
	School adjustment
	Internalization of standards of conduct
Adolescence	Flexible perspective taking
	Abstract thinking and moral reasoning
	Reconciling ideal world with real world
	Loyal same-sex friendships
	Establishing romantic relationships
	Balancing emotional autonomy with relatedness to parents
	Identity development
	Interpersonal intimacy
	Sexuality
	Risk management (e.g., substance use, sexual promiscuity)

because mastery of early developmental tasks serves as building blocks for successfully achieving subsequent tasks, difficulties in resolving the predominant challenges at one period in development are theorized to lay the foundation for subsequent problems mastering stage-salient tasks that collectively increase children's risk for mental health problems over time.

In the context of a stage-salient task framework, insecurity may be hypothesized to increase vulnerability to psychopathology by undermining children's progress in resolving distinctive developmental challenges within well-defined sensitive periods. For example, as a first test of stage-salient task functioning in elucidating the sequelae of insecurity, Davies, Manning, and Cicchetti (2013) reported that toddler insecurity predicted their difficulties resolving stage-salient tasks (i.e., emotion regulation, autonomy, problem-solving) one year later after controlling for initial stage-salient task performance. Stage-salient task difficulties, in turn, were associated with increases in children's behavior problems one year later. Table 6.3 may be a useful guide for future empirical efforts to identify stage-salient cascade processes involving insecurity. For example, given the importance of physiological regulation and the rapid changes in cardiac functioning related to the regulation of distress during infancy (Bar-Haim, Marshall, & Fox, 2000; Graham, Ablow, & Measelle, 2010), it is possible that insecurity may ultimately confer risk during the first year of life by disrupting physiological reactivity to stress. Likewise, childhood is characterized by numerous social challenges, including establishing and maintaining harmonious relationships with peers and the development of empathy and prosocial behavior strategies (Eisenberg, Fabes, Schaller, & Miller, 1989). Thus, insecurity may be particularly likely to increase children's adjustment problems by disrupting their ability to resolve these challenges (Bascoe et al., 2009; Sroufe, Egeland, & Carlson, 1999).

Developmental Pathways and Trajectories

In conceptualizing development as a product of transactions between a multifaceted, changing child and the multidimensional, dynamic context, developmental psychopathology frameworks commonly assume that there are multiple, diverse developmental trajectories for both family and child functioning (Sroufe, 1997). Accordingly, at the level of child functioning, a critical task is to identify the multitude of developmental pathways by examining interindividual differences in intraindividual change. At the level of family functioning, a central objective is to understand children's functioning within a developmental-contextual framework that underscores the continuity, variability, and transformation of family processes over time.

Although treatment of interparental conflict as a static, stable trait is common in the literature, a developmental perspective considers risk factors, like interparental conflict, as potentially dynamic processes (Cui, Conger, & Lorenz, 2005; Cummings & Davies, 2010; Fincham, Grych, & Osborne, 1994). Conceptual distinctions between enduring and transient risk factors within developmental psychopathology models provide a useful blueprint in advancing an understanding of how children adapt to family contexts (Cicchetti & Toth, 1995). In signifying

different ends of a temporal risk continuum, enduring factors are defined by their relatively long life span, whereas transient factors refer to temporary conditions. Because different couples face unique arrays of dispositions, challenges, and stressors, it is likely that there is considerable interfamilial variability in the enduring or transient nature of interparental conflict. Thus, one derivative hypothesis in EST is that the degree of risk posed by destructive interparental conflict for children's safety and well-being is a direct function of the chronicity of their exposure to parental problems. By the same token, EST also posits that this general rule of thumb only applies for children who are experiencing comparable forms and levels of interparental conflict. Consequently, under some conditions, the enduring bouts of interparental discord may not be universally more powerful than transient bouts of conflict in predicting child problems, especially if transient conflicts are reflecting severe forms of destructive conflict (e.g., violence) (Cummings & Davies, 2010).

Propositions in EST-R may offer a more nuanced perspective on the interplay between temporal dimensions of interparental conflict and children's insecurity. If human response systems are guided by a complex set of algorithms to cope with the multiplicity of prehistoric environmental niches as evolutionary accounts postulate (Klein, Cosmides, Tooby, & Chance, 2002), then the association between duration of destructive interparental conflict and child reactivity may not be linear in form as earlier sensitization models postulated. Developmental-evolutionary models specifically postulate that variability in environments with high fitness implications (e.g., threat, reproduction) has been associated with greater developmental plasticity and malleability to subsequent environmental input (e.g., Frankenhuis & Del Giudice, 2012). Thus, rather than proposing that prolonged exposure to relatively unchangeable levels of destructive interparental conflict will progressively increase children's fearful reactivity to later conflicts, EST-R draws on evolutionary models in positing that fluctuations in the balance between destructive and benign forms of interparental conflict over extended developmental periods (e.g., years) may engender maximal sensitization to subsequent conflicts.

How, then, do we reconcile this hypothesis with repeated empirical documentation that recurring exposure to destructive interparental conflict is associated with progressively greater signs of insecurity? Our interpretation is that this is likely an artifact of the tendency to only examine interparental conflict within narrow temporal periods in samples of families who are relatively advantaged (e.g., middle class) and, unlikely, on the whole, to experience highly stable forms of aggression and discord. Although empirical tests have yet to examine whether variability rather than stability of destructive conflict is the key parameter underlying children's sensitization, existing research has produced some promising results. For example, in moving beyond narrow snapshots of interparental functioning to more dynamic assessments of change in children's experiences with destructive interparental conflict, Goeke-Morey and colleagues (2013) reported that changes in destructive conflict in annual assessments over a three-year period continued to predict concomitant increases in children's negative emotional reactivity and threat responses to parental conflict even after taking into account children's average exposure to interparental conflict over the duration of the study.

EST-R eschews the tacit assumption that the sensitization process can be sustained in a constant dose-response fashion over periods over several years. Consistent with the attenuation hypothesis in the psychophysiological literature (Gold & Chrousos, 2002; Susman, 2006), there must be an ill-defined upper limit in experiencing fear and distress that is likely to precipitate qualitative differences in how children respond to subsequent threats in the interparental relationship. EST-R proposes that protracted exposure to high levels of aggression, violence, and intimidation between parents with little source of solace, protection, and support in the family may, initially amplify fear and anxiety as they grapple with concerns about their security. However, over longer developmental histories of exposure to this highly adverse context, children are theorized to develop a "demobilizing" pattern characterized by helplessness, dysphoria, anhedonia, lassitude, and pervasive inhibition of exploratory and affiliative behavior (Davies & Sturge-Apple, 2007). This qualitative shift in behavior is specifically proposed to serve the function of reducing harm by lying low and signaling submissiveness to conspecifics (Gilbert, 2006; Sloman, Atkinson, Milligan, & Liotti, 2002). Moreover, because a "demobilizing" profile is an adaptive solution to a highly stable context that, within our evolutionary past (Del Giudice et al., 2011), commonly signified protracted future exposure to interpersonal violence and threat, it is likely to be highly resistant to modification even with significant changes in constellations of family threat. Thus, in accord with stress autonomy and experiential canalization models in developmental psychopathology (Morris, Ciesla, & Garber, 2010; Sroufe, 1997), it is plausible that the highly automatic and intractable nature of some forms of reactivity to conflict may become

canalized and take on a life of their own, which progressively diminishes their sensitivity to subsequent interparental conflicts.

FUTURE DIRECTIONS

Throughout our discussion of emotional security theory and research, we have attempted to address specific substantive gaps in the literature and their implications for future research. However, it is also important to step back and contemplate broader thematic issues in beginning to formulate an integrative agenda for future research. Toward this goal, the remaining part of this chapter is devoted to sketching some rough blueprints for future scientific work on emotional security theory.

Person-Based Taxonomy

The original version of EST places conceptual emphasis on the hypothesis that indicators within and across the three component processes are interdependent (i.e., intercorrelated), reflecting the common goal of preserving emotional security. Consequently, empirical tests of EST have predominantly utilized variable-based measurement approaches that aggregate different dimensions of conflict reactivity (e.g., distress, vigilance, involvement, negative representations) into a single, additive composite of insecurity. However, questions can be raised about the viability of searching for linear correspondence among the three component processes of emotional security. At an empirical level, the average shared variance among signs of insecurity was modest to moderate in magnitude in a review of existing research (Davies & Martin, 2013). At a conceptual level, the additive approach to combining forms of reactivity into a single composite of insecurity does not fully capture the assumption that emotional security is a dynamic, nonlinear control system. By designating inherently negative types of responding as carrying relatively equal weight as signs of greater insecurity, linear quantitative approaches to assessing security are also inconsistent with the organizational assumption that the meaning of morphologically identical responses to conflict cannot be deciphered unless examined in relation to children's larger constellation of responses (Cummings, 1987; Davies & Forman, 2002; Maughan & Cicchetti, 2002). For example, within the context of high levels of overt emotional reactivity and excessive regulation of conflict exposure, children's low levels of self-reported distress may reflect an insecure strategy of suppressing subjective threat. Alternatively, if low subjective distress occurs in the context of well-regulated concern and low levels of involvement and avoidance, it may be regarded as part of a secure pattern of coping with conflict (Davies & Forman, 2002).

To remedy incongruities between theoretical and empirical approaches to characterizing emotional insecurity, EST-R has recently proposed a person-based taxonomy that is designed to identify the qualitatively different profiles. Each profile possesses unique functional utility in defending against threat under specific family and developmental conditions. Thus, although the SDS is universally designed with the external goal of neutralizing interpersonal threat, an ethological approach assumes that humans adopt a limited number of distinctive subroutines or strategies for achieving this overarching goal that were exquisitely designed, over evolutionary history, to manage and defuse specific configurations of recurring interpersonal threat (Crittenden, Kozlowska, & Landini, 2010; Del Guidice et al., 2011). Likewise, adopting specific strategies to defend against threats is proposed to also advance developmental psychopathology initiatives of more precisely charting the sequelae of coping styles in adverse family environments. Thus, in an attempt to move beyond the nondescript, pathogenic meaning of insecurity in EST and its laundry list of proposed negative outcomes, EST-R is designed to offer greater depth and pinpoint precision in identifying how each pattern or profile of insecurity is associated with a distinctive constellation of developmental vulnerabilities and competencies. In the following sections, we provide a synopsis of the hypothesized form and function of four fundamental social defense strategies and their hypothesized developmental antecedents and consequences (also see Davies & Martin, 2013; Davies & Sturge-Apple, 2007).

Secure Patterns

A secure profile of responding to interparental conflict is characterized by minimal or mild displays of distress that largely reflect mitigated, empathetic concern for the parents rather than egoistic worries about safety. Subjective experiences accompanying this behavioral profile are postulated to consist of benign internal representations of parental relationship challenges, relatively low impulses to regulate interparental conflict, and minimal or mild experiences of distress. The pattern of behavior is organized by an SDS system that is designed to appropriate substantial psychobiological resources toward defusing threat only in relatively circumscribed instances of direct, interpersonal danger (Davies & Sturge-Apple, 2007; Gilbert, 2001).

Thus, regulated by children's underlying confidence in their parents to effectively resolve interparental disagreements, the SDS assumes salience as an organizer of behavior only under dire interparental conflicts and, even in these rare occasions, operates efficiently to preserve security.

Confidence in the parents to encapsulate and regulate their conflicts is posited to evolve from witnessing well-managed parental disputes (e.g., minimal anger, resolution) within a family context that is sufficiently resilient to intermittent interparental conflict (Davies & Sturge-Apple, 2007). Accordingly, the broader family system is likely to be characterized by harmony, child trust in parents as sources of support in times of distress (i.e., secure attachment), and parental psychological health (e.g., high well-being; low psychopathology). Likewise, temperamental characteristics of the child reflecting effortful control and a high threshold for negative affect are theorized to bias the operation of the SDS towards developing a secure profile (e.g., Davies & Martin, 2013). In turn, the high threshold of activation and the efficiency of the SDS system in contexts of interparental conflict allows for balanced resource investment of psychobiological resources to other significant goals organized by ethological systems (e.g., affiliative, exploratory) (Davies, Sturge-Apple, & Martin, 2013). In this way, emotional security may lay the foundation for the development of a wide range of social and academic skills, including problem-solving abilities, social skills, cooperation, empathy, and prosocial behavior, and ultimately low risk for psychopathology. However, EST-R proposes that security may not confer uniformly advantageous outcomes. For example, as described in Davies and Martin (2013), the relatively simple and underdeveloped system for identifying and defending against threat may increase children's risk for victimization and bullying in some developmental contexts.

Mobilizing Patterns

Responses to interparental conflict organized around a mobilizing profile consist of overt, blatant, and demonstrative expressions of distress and fear that are commonly accompanied by active de-escalation strategies (e.g., appeasing, overbright behavior), submissiveness, and vulnerable forms of involvement (e.g., caretaking). At a functional level, the considerable stakes in monitoring and managing interparental conflicts reflect the goal of sustaining vigilance to emerging patterns of threat while also remaining prepared to capitalize on sporadic opportunities for gaining resources in the family (Davies & Sturge-Apple, 2007; Trower, Gilbert, & Sherling, 1990). The high emotional entanglement of mobilizing children in the interparental relationship is also reflected in subjective experiences characterized by considerable negative affect, pessimistic appraisals of the impact of interparental conflict on the family and their own welfare, and considerable impulses to regulate interparental difficulties through fight (e.g., intervention) or flight strategies. In light of the proposal that the mobilizing strategy serves to simultaneously reduce threat and garner attention and sympathy from family members, children who exhibit these strategies are theorized to experience heightened destructive interparental conflict and family discord but also periodic, conditional expressions of warmth and support from family members. Blurred family boundaries (e.g., parental psychological control, resistant attachment, family enmeshment) may further fuel mobilizing strategies by emotionally pulling or coaxing children into interparental difficulties (Davies & Sturge-Apple, 2007). High stakes in guarding against threat and cultivating connectedness evident in the mobilizing profile may be further amplified by temperamental dispositions to exhibit high sensitivity to both punishment (e.g., high distress proneness) and reward (e.g., impulsivity, approach) and impairments in effortful control that prevent inhibition of prepotent, reactive responses to conflict (Davies & Martin, 2013).

Consistent with our ethological account of the cascade mechanisms of insecurity (for details, see the Developmental Cascade Mechanisms section), the mobilizing pattern along with the remaining two insecure profiles may broadly increase adjustment problems by depleting resources that could otherwise be devoted to developing skills and behaviors organized by other behavioral systems (Davies & Martin, 2013). However, the incremental utility of the person-based taxonomy of EST-R also lies in the specificity of the hypothesis that each profile is associated with a distinct portfolio of developmental costs and advantages. On the harmful side of the equation for mobilizing children, acute awareness of the threatening implications of interparental conflict, perseveration on their interpersonal standing in the family, and reflexive alternations between emotion dysregulation and vulnerability (e.g., appeasing behaviors) are proposed to increase vulnerability to anxiety problems, attention difficulties, and borderline personality symptoms (e.g., unstable sense of self, interpersonal dependency, emotional lability) (Gilbert, 2001; Gratz et al., 2009; Jensen et al., 2006). On the socially desirable side, persistent motivation to cultivate and maintain interpersonal relationships may set the stage for trajectories characterized by social interest, interpersonal engagement, openness to intimacy, and empathetic orientations in some developmental contexts.

Dominant Patterns

The functional utility of the dominant strategy consists of enlisting strategies designed to directly defeat threat. Because direct engagement of the source of potential harm requires keen identification of aversive stimuli, children adopting dominant strategies evidence prolonged activation of the SDS characterized by high levels of wariness and vigilance. However, unlike the mobilizing pattern, minimizing the experience and expression of vulnerability (e.g., fear, submissiveness, sadness) and downplaying the significance of family relationships permits the enactment of dominance posturing, coercion, callous indifference, and aggression in an effort to directly confront threat and regain power in the family (Davies & Sturge-Apple, 2007; Dixon, 1998; Hilburn-Cobb, 2004). High levels of destructive interparental conflict are likely to engender dominant strategies when significant collapses in family power hierarchies lead to obscured guidelines for accessing family resources and inconsistent consequences for violations of interpersonal conduct (Davies & Sturge-Apple, 2007; Sloman, Farvolden, Gilbert, & Price, 2006). Ill-defined power hierarchies are most commonly reflected in hostile detachment in the interparental relationship, cold, disengaged family contexts, low parental investment in the lives of children (e.g., apathy, passivity), and vulnerable forms of adult psychopathology (e.g., anxiety, depression). Likewise, temperamental characteristics comprised of bold approach to novelty, low sensitivity to punishment, poor effortful control, and impulsivity may further calibrate the SDS system toward a dominant strategy in contexts of threat (Davies & Martin, 2013).

In serving as a guide to more precisely identifying the sequelae of dominant strategies, the taxonomy in EST-R specifically proposes that the blunting of vulnerable emotions and devaluation of family relationships may be especially likely to take a toll on the affiliative and caregiving systems. Over time, the confluence of dampened interpersonal processes (e.g., empathy, communion) required for the operation of caregiving and affiliative systems and the depletion of resources to devote to goals organized by these systems are theorized to engender poor social competency, hostile attribution biases, callousness, and the rigid, reflexive reliance on aggressive behaviors across social contexts. As this hostile, disenfranchised interpersonal orientation continues to proliferate beyond the interparental and family contexts, children adopting dominant strategies may be specifically vulnerable to developing conduct problems, delinquency, and antisocial symptomatology (Davies & Sturge-Apple, 2007). However, in highlighting the developmental tradeoffs, dominant strategies may also increase the likelihood, in some contexts, of adaptive trajectories characterized by self-confidence, agency, and a bold readiness to experience novelty and challenge (Hawley, Little, & Rodkin, 2007; Sih, Bell, & Johnson, 2004).

Demobilizing Patterns

Designated as an involuntary defeat strategy in ethological models, demobilizing profiles are functionally designed to reduce children's salience as targets of hostility and ire from dominant conspecifics in the social group (Gilbert, 2001; Gilbert & Allan, 1998; Marks & Nesse, 1994; Sloman et al., 2006). The lie-low function of demobilizing strategies may be expressed in a wide array of forms, including freezing, vigilance, quiet disengagement, appeasing behavior (e.g., head down, gaze aversion, unexplainable and intense smiling), sadness, anhedonia, helplessness, fatigue, postural slumping, or downtrodden behaviors. High- (e.g., tenseness, freezing, gingerly moving away) and low-arousal behaviors (e.g., postural slumping, fatigue) may each serve the overarching function of limiting children's exposure to concurrent or future threat. Because demobilizing profiles are last resort strategies for diffusing threat in our evolutionary past, EST-R postulates that this profile evolves from exposure to very dire, agonic family niches. Agonic family contexts are typically characterized by recurrent displays of aggression, intimidation, and oppression by adults that can be manifested in interparental aggression and coercion, family hostility, intimidating forms of parental functioning (e.g., antisocial personality disorder, anger dysregulation), and disorganized attachment. Moreover, unlike the mobilizing strategy, children with demobilizing profiles are theorized to grow up in family contexts that offer minimal (if any) opportunities or resources to mitigate threat through the provision of support, protection, or warmth (Davies & Martin, 2013; Davies & Sturge-Apple, 2007; Gilbert, 1993).

Children adopting a demobilizing strategy are predicted to bear the most significant long-term mental and physical health burdens of any of SDS strategy (Gilbert, 2006; Sloman et al., 2006). The collateral damage of utilizing demobilizing strategies to limit exposure to interpersonal threat is likely to be revealed in disproportionate increases in internalizing symptoms (e.g., depression, anxiety). The attenuation of approach motivations may be most pronounced in deficits within the affiliative and exploratory systems. As a result, demobilizing tendencies should be associated with ample impairments in social skills, agency, and problem-solving abilities (Davies & Sturge-Apple, 2007; Gilbert, 2001; Sloman et al., 2002). However, even

though children with demobilizing profiles may experience considerable and widespread susceptibility to adjustment problems, their underlying aversion to risk may protect them from the development of disruptive, risky, and oppositional behavior problems. Moreover, in reflecting a potential bright spot in tracing the development trajectories, evolutionary models have postulated that some of the components of a demobilizing profile persisted across evolution by promoting a reflective orientation toward the environment and greater inhibitory control (Aksan & Kochanska, 2004; Bell, 2007; Davies, Cicchetti, Hentges, & Sturge-Apple, 2013; Sih et al., 2004).

Summary

The person-based taxonomy in EST-R is designed to increase novelty and precision in the identification of the developmental precursors and consequence of different patterns of defending against threat in the interparental relationship. Although our preliminary findings provide support for the prevalence of each of the four profiles, research has yet to systematically test the hypothesized origins and outcomes of the profiles (Davies & Martin, 2013). Thus, empirically testing the utility of the EST-R taxonomy remains an important direction for future research. However, in addressing this gap, it is important to recognize that the hypothesized relations among family and developmental conditions, SDS strategies, and outcomes are probabilistic rather than certain. Consistent with the open systems model in developmental psychopathology (Cicchetti & Rogosch, 1996; Davies & Cicchetti, 2004), EST-R acknowledges child and ecological characteristics are likely to serve as regulatory conditions (i.e., moderators, predictors) that increase heterogeneity in the mediational pathways of the security profiles. Thus, equifinality is evidenced in expectation that different experiences may give rise to any given SDS profile and pattern of adjustment, whereas multifinality is reflected in the hypothesis that comparable family experiences will produce different forms of adjustment.

Expanding Emotional Security Beyond the Interparental Dyad

In underscoring its potential to cultivate multiple growing points in developmental psychopathology, the conceptualization of the SDS in EST-R may have broad translational implications in other family and ecological contexts. In contrast to the circumscribed focus in EST on security within the interparental subsystem, EST-R offers greater flexibility in defining emotional security as a product of the operation of the SDS. By accepting the premise that the SDS organizes strategies that were designed to defuse configurations of threat in social networks, EST-R has the potential to break the conceptual shackles of EST and expand the scope of inquiry to multiple interpersonal contexts. Careful attention to form and function of patterns of defending against threat in EST-R may specifically offer new insights into the developmental course of individual differences in preserving safety in other ecologies (e.g., parent–child and triadic contexts, peer relationships, community violence). To illustrate the potential translational implications for developmental psychopathology (Cicchetti, 2010), we provide a brief and selective overview of how EST-R may advance an understanding of how children cope with both familial and extrafamilial settings (for more details, see Davies & Martin, 2013).

The interparental relationship is not the only source of conflict, anger, and aggression in the family unit. Children are both bystanders and recipients of hostility and conflict by parents and siblings (Bush & Peterson, 2013; Margolin & Gordis, 2000; Straus, 2001). Despite empirical evidence that these family relationships can serve as sources of threat that may organize children's defense strategies (Davies & Martin, 2013), traditional approaches to understanding the processes by which parent–child and sibling discord impact children's mental health and adjustment have predominantly focused on social learning (e.g., modeling) and attachment (e.g., support) theories (e.g., McElhaney, Allen, Stephenson, & Hare, 2009; Gass, Jenkins, & Dunn, 2007; Volling & Blandon, 2005). Thus, EST-R may offer a unique theoretical window into understanding how and why children exhibit specific defense strategies in contending with threat in parent–child, sibling, or broader family relationship contexts. Because the taxonomy in EST-R is guided by evolutionary and ethological work on individual differences in coping with conspecific threat, it has the strong potential to provide a heuristic for identifying the nature, origins, and sequelae of defense strategies in multiple family contexts. In support of this research direction, our preliminary observations have provided solid evidence for the occurrence of the four SDS patterns (i.e., secure, mobilizing, dominant, demobilizing) in triadic conflicts involving children and both of their parents (see Davies & Martin, 2013). Future research is needed to explore whether each defense profile develops from the hypothesized unique constellations of early family threats, supports, and temperamental dispositions. Likewise, predictions regarding the distinctive sequelae of defense strategies in parent–child, sibling, triadic, and family-level contexts remain to be tested.

Exposure to threatening interpersonal contexts is also common outside of the family. Children regularly grapple with a wide array of peer stressful events, including victimization, rejection, conflict, and competition in peer and school settings (Asher & Coie, 1990; Craig & Pepler, 1998; Juvonen & Graham, 2001; Laursen & Pursell, 2009). Despite increased recognition of the significance of peer relationships for children's mental health, research on the nature, precursors, and implications of children's coping with stressful peer interactions remains underdeveloped at multiple levels. At the conceptual level, no formal theories exist to guide the identification of patterns of individual differences in how children organize behavioral responses to threatening peer events (Asher & MacDonald, 2009). Predominant conceptual and empirical focus on the morphological properties (i.e., form, structure) of behavior has resulted in limited attention to the functional utility (i.e., purpose) of behavior within person-based patterns of children's reactivity to peer threat. Thus, little is known about how children cope with and respond to stressful peer interactions beyond broad indices of interpersonal functioning (e.g., rejected status, aggressive/withdrawn behavior). The field continues to grapple with the challenge of generating process-oriented hypotheses about the origins, course, and sequelae of distinct patterns of behavior in response to challenging peer interactions.

Given our account of the contextual versatility of EST-R, our person-based taxonomy and its broader array of hypotheses may be useful in addressing this gap. In supporting the significance of EST-R for peer contexts, pilot research has identified that the four SDS profiles are commonly employed by children in peer settings (Davies & Martin, 2013). The focus on functional utility and its leverage in precisely identifying developmental precursors and implications of SDS profiles in EST-R is also readily applicable to the study of peer relationships. For example, if the mobilizing profile functions to garner support, sympathy, and alliances as a way of defending against peer threat, then it may be most evident when exposure to threat and conflict are intermingled with intermittent access to resources in peer ecologies. Thus, the approach–avoidance dialectic evidenced in mobilizing responses is hypothesized to be cultivated in interpersonal contexts containing moderate levels of peer hostility in conjunction with some relationship dynamics (e.g., friendship, support) that motivate children to maintain or increase social ties (Davies & Martin, 2013; Martin, Davies, & MacNeill, 2014). Consistent with our taxonomy, emotion dysregulation and interpersonal preoccupation resulting from mobilizing tendencies is posited to coalesce into anxiety, attention, and hyperactivity difficulties as the stress response associated with prolonged SDS activity undermines developing regulatory systems (Hilburn-Cobb, 2004; Sloman et al., 2006).

In conclusion, by casting emotional security theory within an ethological framework of social defense, EST-R may serve as a useful guide for increasing precision and novelty in numerous areas of developmental psychopathology that extend well beyond the interparental dyad. At the level of the family, social defense conceptualizations may be usefully adapted to understanding the developmental trajectories that evolve from the common occurrence of conflict in parent–child, sibling, triadic, and family-level interactions. In moving beyond the family, EST-R may also serve as a useful tool for formulating and testing hypotheses on children's adaptation to peer, neighborhood, and community adversity (see Davies & Martin, 2013).

Protection and Resilience in the Face of Insecurity

Emotional security theory has largely been centered on characterizing the vulnerability and maladjustment of children facing histories of adversity (Davies et al., 2006; McCoy et al., 2009). However, identifying maladaptive outcomes without balanced consideration of resilience does not adequately represent the primary tenets of developmental psychopathology. One of the foundational principles of developmental psychopathology consists of the importance of considering, with equal weight, both risk and resilience in development (Cicchetti, 1989; Luthar, Cicchetti, & Becker, 2000; Masten & Tellegen, 2012). Resiliency is specifically defined as the ability of children to adapt fairly well in spite of their exposure to considerable adversity (Luthar et al., 2000).

In integrating resiliency into emotional security theory, conceptualizations and assessments must be able to capture the dynamic, multidimensional nature of resilience, with particular recognition that some children may evidence shifting levels of competence in some areas of functioning and psychopathology in others (Luthar et al., 2000). Evolutionary models of development underscore the significance of conditional adaptation based on the goodness of fit between children's characteristics and their ecological niches. Some seemingly maladaptive behaviors may specifically be sustained within the population because they confer advantages in supporting survival and, in some cases, healthy development under specific social and ecological conditions (Belsky & Pluess, 2009; Ellis et al., 2011). For example, differential susceptibility theory proposes that children who are high in sensitivity

to environmental input fare substantially better than their peers in resource-rich environments but experience disproportionately greater problems under highly adverse developmental conditions (Belsky & Pluess, 2009). In further highlighting the potential costs that may accompany resilience, the person-based taxonomy in EST-R posits that specific patterns of insecurity commonly result in developmental trade-offs in which proficiency or resilience achieved in one domain occurs at the cost of undesirable outcomes in other areas of functioning (Davies & Martin, 2013). For example, whereas mobilizing children are theorized to be at increased risk for experiencing some forms of psychopathology, the risk is balanced to some degree by the potential for these children to follow developmental trajectories characterized by high communion and social attunement.

After sufficiently characterizing resilience in a multidimensional framework, the next challenge is to further delineate the protective factors and mechanisms that explain why some insecure children are able to develop along competent developmental trajectories. Taxonomies that distinguish between various forms of protective effects will be valuable tools in precisely characterizing how a given factor might contribute to the adaptation of children with high levels of insecurity (Luthar, 1993; Luthar et al., 2000). For example, *protective-reactive* factors confer substantially greater competence when insecurity is low, with its buffering potency waning significantly at high levels of insecurity. In contrast, *protective*-stabilizing *factors* produce uniformly high levels of adaptation across increasing levels of insecurity. Alternatively, *protective-enhancing* factors operate in a way that actually promotes better functioning with increasing experiences with insecurity.

Investigations have yet to identify protective factors that mitigate the deleterious effects of insecurity. However, due to its conceptual and empirical links with emotional security, research on interparental conflict may provide a starting point for specifying the form of protective effects. For example, a series of studies identified vagal regulation (i.e., suppression of vagal tone) during adult arguments as a protective factor in associations between interparental aggression and children's adjustment problems (El-Sheikh, Harger, & Whitson, 2001; El-Sheikh & Whitson, 2006). To more precisely identify the form of protection for vagal regulation, additional analyses indicated that vagal suppression was a protective-stabilizing factor, conferring consistently low internalizing symptoms across levels of interparental aggression (El-Sheikh & Whitson, 2006). Even existing findings on moderating factors that do not explicitly pinpoint the forms of protection may be usefully interpreted within these taxonomies. For example, results on the role of maternal communications about conflicts in moderating the risk posed by interparental conflict were not initially framed in a protective factor classification scheme (Gomulak-Cavicchio, Davies, & Cummings, 2006). However, post hoc inspection of the findings indicates that various forms of maternal communication served distinct protective functions in associations between interparental conflict and child externalizing symptoms. Whereas the moderating effects of maternal remorseful apologies to children were expressed in a protective-stabilizing form, communications denying or limiting child exposure to conflicts (i.e., renunciation) were consistent with a protective-reactive effect. Given its documented value in developmental psychopathology research, future identification of protective effects in models of insecurity would benefit from the precision afforded by systems for classifying protection.

The identification of protective factors is commonly regarded as the final scientific objective in research on resilience (Cummings et al., 2000). However, from a process-oriented perspective, simply ending the inquiry at cataloguing protective factors and their specific effects is conceptually unsatisfying. This approach does little to advance an understanding of why individuals with certain characteristics or experiences benefit from the protective factor. Addressing questions of why protective factors buffer insecure children from developing adjustment difficulties will require more sophisticated analytic approaches (e.g., mediated-moderation) than the standard moderator models. For example, in drawing from the broader literature on interparental conflict, Manning, Davies, and Cicchetti (2013) first identified that maternal sensitive parenting served as a protective factor in associations between interparental aggression and preschooler psychological functioning. However, in moving beyond the simply delineating protective factors, the use of mediated-moderation analyses specifically revealed that maternal sensitivity buffered children from violent homes through its association with diminished levels of child angry reactivity to interparental conflict.

In accord with multiple levels of analysis perspectives in developmental psychopathology (Cicchetti & Curtis, 2007), the search for mechanisms underlying protective factors can be facilitated by analyzing the interplay between risk and protective factors across biological, psychological, and interpersonal units of analysis. Returning to our example of vagal regulation as a protective factor (El-Sheikh & Whitson, 2006; El-Sheikh et al., 2001), it is possible that future research might identify vagal

suppression as a protective factor in pathways between children's insecurity and psychological adjustment. A multilevel, process-oriented approach may consist of examining the psychological manifestations of vagal suppression that serve to mediate of its moderating effects. For example, vagal suppression is theorized to function as a physiological substrate for effective emotion regulation strategies and successful adaptations to stressful events threat (Bornstein & Suess, 2000; Graziano & Derefinko, 2013; Porges, 2003). Therefore, testing emotion regulation and coping strategies as potential mechanisms that account for how and why vagal regulation protects insecure children may further an understanding of resilience within a multiple levels of analysis framework. The resulting advances in the interplay among multiple levels of protective factors may, in turn, provide greater strength and versatility in delineating clinical targets and therapeutic tools for treatment initiatives.

Prevention, Intervention, and Public Policy

Our ultimate goal in advancing emotional security theory is to facilitate the translation of theoretically guided empirical knowledge into practice. Consistent with this objective, progress in testing and refining EST and EST-R over the past two decades has provided a base of scientific findings to guide clinical practice and public policy initiatives. Although outlining the translational implications of our theory for clinical and policy programs is a pressing research direction, successfully achieving this goal remains a formidable challenge. Generating policy, legislation, and services for children from high conflict homes is still in its infancy as public health, community, and legal systems continue to grapple with treatment implications of a growing and complex body of data. Given the state of the literature on children's emotional security, it is premature to offer any definitive recommendations for treatment or public policy. Accordingly, we outline, at a broad level, the potential translational implications of emotional security theory for alleviating the burden of child mental illness.

Identifying Children at Greatest Risk for Psychopathology

Identification of children's sense of security as a mediating mechanism of the vulnerability experienced by children who are exposed to interparental conflict has important implications for clinical assessment. The process-oriented approach of EST emphasizes the importance of utilizing clinical assessments designed to carefully observe children's behavior under conditions of conflict and threat in the interparental and broader family unit. Although capturing children's conscious responses to interparental conflict should be a fundamental part of clinical assessment process, EST and EST-R are unique relative to other prevailing theories of interparental conflict (e.g., Crockenberg & Forgays, 2001; Grych & Fincham, 1990) in underscoring that children's experiences with insecurity in the interparental relationship are commonly rooted in affective structures outside the child's conscious awareness or verbal articulation (Davies & Sturge-Apple, 2007). Moreover, the resulting expressions of child behaviors organized by these affective structures commonly exhibit modest correspondence with reports of their subjective emotional experiences and appraisals (Davies & Forman, 2002). Consequently, obtaining a comprehensive profile for identifying children at high risk for psychopathology within an emotional security framework would be facilitated by use of other informants (e.g., parents) or methods (e.g., observations) in the assessment process. In addition to the availability of structured and semistructured methods for obtaining children's self-report measures of their security (Davies, Forman, et al., 2002; Davies, Sturge-Apple, et al., 2006, 2013), clinicians have, at their disposal, a number of other new approaches for implementing a multi-informant, multimethod measurement battery, including caregiver surveys (Davies, Harold, et al., 2002), semistructured interviews with parents (Davies, Manning, & Cicchetti, 2013), and observational systems (Davies et al., 2004).

Scientific progress in characterizing the substance and operation of the goal-corrected system of emotional security also offers clinical blueprints for precisely identifying patterns of reactivity to conflict with high prognostic utility. Advances in articulating how children express their insecurity in EST-R specifically highlight the significance of fear and vigilance as a common underlying sources of children's reactivity to interparental difficulties. By the same token, EST-R also recognizes that the vigilance and fear may organize a diversity of ways of defending against the threat accompanying interparental conflict, including anger, flight, fight, submissive disengagement, appeasing behavior, camouflaging behavior, demobilizing, and demonstrative displays of distress (Davies & Sturge-Apple 2007). Although the myriad of different expressions of insecurity may increase the challenges and complexities in clinical assessments of family process, EST-R offers a parsimonious taxonomy of identifying the profiles of security through the articulation of the four SDS profiles.

Greater precision afforded by EST-R is also balanced by the integration of children's trajectories of coping and adjustment within a broader, complex developmental

system. The schematic in Figure 6.1 embeds children's emotional security within a dynamic constellation of family, ecological, child, and developmental processes. In some parts of the model, the contextualization of emotional security highlights the operation of other complementary processes (e.g., parenting, parent–child attachment) that may help to increase the ability to identify children from high conflict homes who are at risk for experiencing psychopathology. In other parts of the conceptualization, family, ecological, and developmental processes may serve as moderators that may be useful clinical sources of information for determining which children may evidence disproportionately greater or diminished risk for experiencing problems. Finally, our objective of explicating how and why insecurity may increase risk for mental health problems may help clinicians to identify areas of concern that play more proximal roles in developmental cascades to psychopathology. Collectively, the contextualization of emotional security underscores the value (whenever feasible) of obtaining more comprehensive assessments of adversity, strengths, and opportunities of at-risk children within family, developmental, and ecological levels of analysis.

Delineating Clinical Targets and Approaches

Given the central role of children's emotional security in the interparental relationship as a mediating factor contributing to their long-term mental health, it makes intuitive sense that reducing signs of emotional insecurity should be a primary treatment goal in programs aiming to reduce the negative public health consequences of family conflict. However, a singular, myopic focus on reducing specific expressions of insecurity in the interparental relationship may have unintended, negative consequences of stripping children of the very strategies that commonly prove to be successful in defending against threat in high conflict homes (Davies & Martin, 2013; Davies et al., 2006). From this perspective, any therapeutic approach that is designed to reduce risk for psychopathology by altering children's reactivity to conflict must be accompanied by a complementary focus on ameliorating the destructive interparental family processes (e.g., interparental aggression, family disengagement) that foster insecurity. Without the comparable family intervention component, children are likely to revert to their old, insecure strategy as a way of tailoring their response to the threatening environmental input. Moreover, failing to institute more comprehensive family-wide programs may create new sets of risks for children who are ill equipped to cope with the continuing perturbations in the family system.

An alternative approach may consist of programs designed to prevent or reduce destructive family conflict characterized by hostility, aggression, and violence between parents (Cummings & Davies, 2010; Cummings, Faircloth, Mitchell, Cummings, & Schermerhorn, 2008; Cummings & Shatz, 2012; Shifflett & Cummings, 1999). Numerous therapeutic prevention and intervention programs exist to help distressed couples decrease their destructive conflict tactics (Cowan, Cowan, Ablow, Johnson, & Measelle, 2005; Feinberg & Kan, 2008; Halweg & Richter, 2010; Loew et al., 2012; Markman & Rhoades, 2012). However, these programs typically vary in their efficacy, are rarely tested within randomized-control trials, and seldom consider the perspective of the child (Cummings & Schatz, 2012; Markman et al., 2010). Toward addressing these gaps, the Happy Couples, Happy Kids (HCHK) program is a psychoeducational program that translates EST principles into treatment models that are designed to foster child well-being by reducing destructive interparental conflict (Cummings & Schatz, 2012; Faircloth & Cummings, 2008; Faircloth, Schermerhorn, Mitchell, Cummings, & Cummings, 2011). As a brief program delivered to parents in a small group setting, HCHK has three primary objectives: (1) to provide couples with evidenced-based knowledge on distinctions between destructive from constructive conflict strategies; (2) to inform them of the influence interparental conflict on children and on their own ability to successfully parent; and (3) to explain the importance of maintaining a sense of security across multiple family relationships in supporting child mental health (Faircloth & Cummings, 2008). HCHK program has been shown to reduce the destructive conflict tactics, increase marital satisfaction, and enhance child adjustment for up to two years after the program (Cummings & Merilees, 2010; Cummings & Shatz, 2012; Faircloth & Cummings, 2008; Faircloth et al., 2011).

For practitioners who have significant resources available, complementary family-wide approaches may also provide significant clinical leverage in reducing children's insecurity in the interparental relationship. Consistent with developmental psychopathology and family systems approaches (Davies & Cicchetti, 2004), EST proposes that children's insecurity is a product of complex associations between interparental, family, developmental, and ecological factors. For example, EST posits that the nature of the interplay between interparental conflict and parenting difficulties may increase children's vulnerability to psychopathology by undermining insecurity in multiple (e.g., interparental, parent–child) relationships (Sturge-Apple et al., 2008). Thus, EST may provide a useful framework for

integrating couple-centric therapeutic approaches into the broader family system (Coatsworth, 2013). Consistent with this recommendation, a growing number of family-wide programs are designed, in part, to minimize interparental conflict and its deleterious developmental consequences (Kaminiski et al., 2008; Sanders, 2008). In keeping with this clinical direction, adaptations to the HCHK now include additional educational components aimed at improving communication and reducing destructive conflict in both interparental and parent–child subsystems (Cummings & Schatz, 2012). By directing clinical focus to issues of multifinality and equifinality in the emergence of children's concerns about security (Cicchetti & Rogosch, 1996), these types of programs for targeting multiple pathways of risk across family relationships may be particularly effective in reducing child insecurity across different family subsystems (Cowan & Cowan, 2002; Cummings & Davies, 2010; Cummings & Shatz, 2012; Liddle & Schwartz, 2002).

Although the opportunities to improve children's security and adjustment through family interventions are encouraging, implementation of these programs is not always feasible. In addition to not always yielding meaningful, sustainable improvements in child adjustment, family-wide interventions require considerable expenditure of professional resources and, in some cases, are impossible to implement in families who are unwilling or unable to participate in treatment. To increase the flexibility and accessibility of intervention programs informed by emotional security theory, EST and EST-R can also assist in identifying alternative points for intervention that do not require the engagement of parents or family. As knowledge continues to accumulate on the downstream processes set in motion by children's insecurity (Davies & Martin, 2013), we will be in an increasingly stronger position to interrupt the pathogenic processes before they crystallize into broader trait-like patterns of psychopathology. Although still in its early stages, research has delineated several cascade mechanisms that account for why insecurity poses a risk for children, including proliferation of defensive representations and analogs to other interpersonal relationships (e.g., Bascoe, et al., 2009), disruption of biological processes (e.g., sleep activity; Kelly & El-Sheikh, 2013), difficulties focusing and sustaining attention (e.g., Davies et al., 2008), and failure to resolve stage-salient developmental tasks (e.g., Davies, Manning, & Cicchetti, 2013). Thus, specific clinical goals for insecure children may include (1) managing hostile attributions and rigid negative analogs by developing safe, supportive, and trusting interpersonal havens for more openly processing interpersonal problems (Dodge & Godwin, 2013; Johnston et al., 2009); (2) supporting healthy sleep patterns through parental establishment of regular sleep hygiene and bedtime routines (Mindell & Owens, 2009; Mindell et al., 2011); (3) increasing child attention and executive functioning capacities (DuPaul, Helwig, & Slay, 2011); and (4) enhancing mastery of stage-salient tasks across childhood and adolescence (Schulz & Kerig, 2012; Ursache, Blair, & Raver, 2012).

With future empirical testing, EST-R may also deliver a unique lens for refining and tailoring intervention strategies to the individual needs and capacities of insecure children. Although extreme caution should be exercised in translating the largely untested hypotheses to clinical initiatives, the value of EST-R lies in its potential to refine treatment targets by (1) identifying specific developmental and ecological conditions that serve as precursors of each of the four SDS profiles; and (2) offering greater precision in delineating the advantageous and disadvantageous sequelae of each profile (see Davies & Martin, 2013). By selectively adapting therapeutic components to address the distinctive experiences, needs, and strengths of each profile, it may be possible to maximize the efficacy of intervention and prevention efforts. For example, because children with mobilizing tendencies tend to value interpersonal connectedness in the context of exhibiting poor social skills and emotion regulation difficulties, the effectiveness of intervention programs may be enhanced by treatment modules that rely heavily on social skills exercises, emotion regulation training, and pairings with competent peers. Conversely, as part of a dominant profile, efforts to defeat threats in the family through oppositional strategies are likely to be sustained by ambiguity in family power structures and tendencies to downplay the significance of interpersonal relationships. Thus, interventions tailored to this profile may be more successful if they are integrated into a therapeutic context that offers clear communication of guidelines for conduct, consistent disciplinary consequences for transgressions, and material (rather than simply social rewards such as praise) reinforcement for desirable behavior.

CONCLUSIONS

In conclusion, the integration of emotional security theory and principles of developmental psychopathology has advanced our understanding of the implications of children's processing and responding to family conflict in the typical and atypical development of children. As our review of research in this chapter documents, there is now

firm evidence that emotional insecurity is a unique and robust mediator of multiple pathways involving family adversity and children's adjustment problems. Moreover, strides in contextualizing the study of the mediational pathways of insecurity in the interparental relationship are evidenced by the conceptualization of a multilayered framework that highlights the roles of broader family characteristics, developmental processes, child attributes, and ecological parameters. Complementary progress is also being made in characterizing the pathogenic cascade processes underlying the vulnerability experienced by children with high levels of insecurity. Based on preliminary research, we are cautiously optimistic that the EST-R will offer incremental degrees of precision and novelty in characterizing the origins, nature, and sequelae of children's patterns of responding to interparental conflict. Through a balanced approach to testing hypotheses generated by EST and EST-R, it is hoped that the coming decades generate a knowledge base to further guide intervention and public policy initiatives and reduce the burdens experienced by children and families in high-conflict homes.

REFERENCES

Ackerman, B. P., Kogos, J., Youngstrom, E., Schoff, K., & Izard, C. (1999). Family instability and the problem behaviors of children from economically disadvantaged families. *Developmental Psychology*, *35*, 258–268. doi: 10.1037//0012-1649.35.1.258

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the Strange Situation*. Hillsdale, NJ: Erlbaum.

Ainsworth, M. D. S., & Bowlby, J. (1991). An ethological approach to personality development. *American Psychologist*, *46*, 333–341. doi: 10.1037/0003-066X.46.4.333

Aksan, N., & Kochanska, G. (2004). Links between systems of inhibition from infancy to preschool years. *Child Development*, *75*, 1477–1490. doi: 10.1111/j.1467-8624.2004.00752.x

Almeida, D. M., Wethington, E., & Chandler, A. L. (1999). Daily transmission of tensions between marital dyads and parent–child dyads. *Journal of Marriage and the Family*, *61*, 49–61. doi: 10.2307/353882

Amato, P. R. (2000). The consequences of divorce for adults and children. *Journal of Marriage and Family*, *62*, 1269–1287. doi: 10.1111/j.1741-3737.2000.01269.x

Amato, P. R., Booth, A., Johnson, D. R., & Rogers, S. J. (2007). *Alone together: How marriage in America is changing*. Cambridge, MA: Harvard University Press.

Aronson, E., Wilson, T. D., & Brewer, M. B. (1998). Experimentation in social psychology. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., pp. 143–179). Boston, MA: McGraw-Hill.

Asher, S. R., & Coie, J. D. (1990). *Peer rejection in childhood*. New York, NY: Cambridge University Press.

Asher, S. R., & MacDonald, K. L. (2009). The behavioral basis of acceptance, rejection, and perceived popularity. In K. H. Rubin, W. M. Bukowski, & B. Laursen (Eds.), *The handbook of peer interactions, relationships, and groups* (pp. 232–248). New York, NY: Guilford Press.

Barber, B. K. (2008). Contrasting portraits of war: Youths' varied experiences with political violence in Bosnia and Palestine. *International Journal of Behavioral Development*, *32*, 298–309. doi: 0.1177/0165025408090972

Bar-Haim, Y., Marshall, P. J., & Fox, N. A. (2000). Developmental changes in heart period and high-frequency heart period variability from months to 4 years of age. *Developmental Psychobiology*, *37*, 44–56. doi: 10.1002/1098-2302

Barker, T. V., Reeb-Sutherland, B. C., & Fox, N. A. (2014). Individual differences in fear potentiated startle in behaviorally inhibited children. *Developmental Psychobiology*, *56*, 133–141.

Bascoe, S. M., Davies, P. T., Sturge-Apple, M. L., & Cummings, E. M. (2009). Children's representations of family relationships, peer information processing, and school adjustment. *Developmental Psychology*, *45*, 1740–1751. doi: 10.1037/a0016688

Batson, C. D. (1991). *The altruism question: Toward a social-psychological answer*. Hillsdale, NJ: Lawrence Erlbaum Associated, Inc.

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science*, *16*, 396–403. doi: 10.1111/j.1467-8721.2007.00534.x

Bell, A M. (2007). Future directions in behavioural syndromes research. *Proceedings of the Royal Society*, *274*, 755–761. doi: 10.1098/rspb.2006.0199

Belsky, J., Campbell, S. B., Cohn, J. F., & Moore, G. (1996). Instability of infant–parent attachment security. *Developmental Psychology*, *32*, 921–924.

Belsky, J., & Pluess, M. (2009). Beyond diathesis–stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*, 885–908. doi: 10.1037/a0017376

Benson, M. J., Buehler, C., & Gerard, J. M. (2008). Interparental hostility and early adolescent problem behavior spillover via maternal acceptance, Harshness, inconsistency, and intrusiveness. *Journal of Early Adolescence*, *28*, 428–454. doi: 10.1177/0272431608316602

Bierman, K. L. (2004). *Peer rejection: Developmental processes and intervention strategies*. New York, NY: Guilford.

Birditt, K. S., Brown, E., Orbuch, T. L., & McIlvane, J. M. (2010). Marital conflict behaviors and implications for divorce over 16 years. *Journal of Marriage and Family*, *72*, 1188–1204. doi: 10.1111/j.1741-3737.2010.00758.x

Blair, C., & Diamond, A. (2008). Biological processes in prevention and intervention: The promotion of self-regulation as a means of preventing school failure. *Development and Psychopathology*, *20*, 899–911. doi: 10.1017/S0954579408000436

Blatz, W. E. (1966). *Human society: Some reflections*. Toronto, Ontario: University of Toronto Press.

Borgerhoff Mulder, M., & Schacht, R. (2012). Human behavioural ecology. In eLS: John Wiley & Sons, Ltd: Chichester. doi: 10.1002/9780470015902.a0003671.pub2

Bornstein, M. H., & Suess, P. E. (2000). Child and mother cardiac vagal tone: Continuity, stability, and concordance across the first 5 years. *Developmental Psychology*, *36*, 54–65. doi: 10.1037//0012-1649.36.1.54

Bowlby, J. (1969). *Attachment and loss. Vol. I: Attachment*. New York, NY: Basic Books.

Bowlby, J. (1973). *Attachment and loss. Vol.II: Separation*. New York, NY: Basic Books.

Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annual Review of Psychology*, *53*, 371–399. doi: 10.1146/annurev.psych.53.100901.135233

Bretherton, I., Ridgeway, D., & Cassidy, J. (1990). Assessing internal working models of the attachment relationship. In M. T. Greenberg, D. Cicchetti, D., & E. M. Cummings (Eds.), *Attachment in preschool years: Theory, research, and intervention* (pp. 273–308). Chicago, IL: University of Chicago Press.

Brown, S. L., Brown, R. M., & Preston, S. (2011). A model of human caregiving motivation. In S. L. Brown, R. M. Brown, & L. Penner (Eds.), *Moving beyond self interest: Perspectives from evolutionary biology, neuroscience, and the social sciences*. New York, NY: Oxford University Press.

Buchanan, C. M., Maccoby, E. E., & Dornbusch, S. M. (1991). Caught between parents: Adolescents' experience in divorced homes. *Child Development*, *62*, 1008–1029. doi: 10.1111/j.1467-8624.1991.tb01586.x

Buehler, C., Anthony, C., Krishnakumar, A., & Stone, G. (1997). Interparental conflict and youth problem behaviors: A meta-analysis. *Journal of Child and Family Studies*, *6*, 223–247. doi: 10.1023/A:1025006909538

Buehler, C., & Gerard, J. M. (2002). Marital conflict, ineffective parenting, and children's and adolescents' maladjustment. *Journal of Marriage and Family*, *64*, 78–92. doi: 10.1111/j.1741-3737.2002.00078.x

Buehler, C., Krishnakumar, A., Stone, G., Anthony, C., Pemberton, S. Gerard, J., et al. (1998). Interparental conflict styles and youth problem behaviors: A two-sample replication study. *Journal of Marriage and Family*, *60*, 119–132. doi: 10.2307/353446

Buehler, C., Lange, G., & Franck, K. L. (2007). Adolescents' cognitive and emotional responses to marital hostility. *Child Development*, *78*, 775–789. doi: 10.1111/j.1467-8624.2007.01032.x

Buehler, C., & Welsh, D. P. (2009). A process model of adolescents' triangulation into parents' marital conflict: The role of emotional reactivity. *Journal of Family Psychology*, *23*, 167–180. doi: 10.1037/a0014976

Bush, K. R., & Peterson, G. W. (2013). Parent–child relationships in diverse contexts. Handbook of marriage and the family. In G. W. Peterson & K. R. Bush (Eds.) *Handbook of Marriage and the Family* (pp. 275–302). New York, NY: Springer

Buss D. M. (2008). *Evolutionary psychology: The new science of the mind* (3rd ed.). Boston: Allyn & Bacon.

Cassidy, J., Kirsh, S. J., Scolton, K. L., & Parke, R. D. (1996). Attachment and representations of peer relationships. *Developmental Psychology*, *32*, 892–904. doi: 10.1037/0012-1649.32.5.892

Chase-Lansdale, P. L., Wakschlag, L. S., & Brooks-Gunn, J. (1995). A psychological perspective on the development of caring in children and youth: The role of family. *Journal of Adolescence*, *18*, 515–556. doi: 10.1006/jado.1995.1037

Chisholm, J. S. (1996). The evolutionary ecology of attachment organization. *Human Nature*, *7*, 1–37.

Cicchetti, D. (Ed.). (1989). *The emergence of a discipline: Rochester symposium on developmental psychopathology*. Hillsdale, NJ: Lawrence Earlbaum.

Cicchetti, D. (1991). Fractures in the crystal: Developmental psychopathology and the emergence of self. *Developmental Review*, *13*, 471–502. doi: 10.1016/0273-2297

Cicchetti, D. (1993). Developmental psychopathology: Reactions, reflections, projections. *Developmental Review*, *13*, 471–502. doi: 10.1006/drev.1993.1021

Cicchetti, D. (2002). The impact of social experience on neurobiological systems: Illustration from a constructivist view of child maltreatment. *Cognitive Development*, *17*, 1407–1428. doi: 10.1016/S0885-2014(02)00121-1

Cicchetti, D. (2010). Resilience under conditions of extreme stress: A multilevel perspective. *World Psychiatry*, *9*, 145–154. doi: 10.1002/j.2051-5545.2010.tb00297.x

Cicchetti, D. (Ed.). (2011). Allostatic Load, [Special Issue]. *Development and Psychopathology, Part 1*, *23(3)*, 723–954; *Part 2, 23(4)*, 955–1212.

Cicchetti, D., & Cohen, D. J. (1995). Perspectives on developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 1. Theory and methods* (pp. 3–20). New York, NY: Wiley.

Cicchetti, D., & Curtis, W. J. (2007). Multilevel perspectives on pathways to resilient functioning. *Development and Psychopathology*, *19*, 627–629. doi: 10.1017/S0954579407000314

Cicchetti, D., & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences of children's development. *Psychiatry*, *56*, 96–118.

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597–600. doi: 10.1017/S0954579400007318

Cicchetti, D., & Rogosch, F. A. (2002). A developmental psychopathology perspective on adolescence. *Journal of Consulting and Clinical Psychology*, *70*, 6–20. doi: 10.1037/0022-006X.70.1.6

Cicchetti, D., & Toth, S. L. (1995). A developmental psychopathology perspective on child abuse and neglect. *Journal of American Academy of Child and Adolescent Psychiatry*, *34*, 541–565. doi: 10.1097/00004583-199505000-00008

Cicchetti, D., & Toth, S. L. (1997). Transactional ecological systems in developmental psychopathology. In S. S. Luthar, J. A. Burack, D. Cicchetti, & J. R. Weisz (Eds.), *Developmental psychopathology: Perspectives on adjustment, risk, and disorder* (pp. 317–349). New York, NY: Cambridge University Press.

Cicchetti, D., & Toth, S. L. (1998). Perspectives on research and practice in developmental psychopathology. In W. Damon (Ed.), *Handbook of child psychology* (5th ed., Vol. *4*, pp. 479–583). New York, NY: Wiley.

Cicchetti, D., & Toth, S. L. (2006). Editorial: Building bridges and crossing them: Translational research on developmental psychopathology. *Development and Psychopathology*, *18*, 619–622. doi: 10.1017/S0954579406060317

Cicchetti, D., & Toth, S. L. (2009). A developmental psychopathology perspective on adolescent depression. In S. Nolen-Hoeksema & L. Hilt (Eds.) *Handbook of adolescent depression* (pp. 3–31). New York, NY: Taylor & Francis.

Clark, D. M. (2001). A cognitive perspective on social phobia. In: W. R. Crozier & L. E. Alden (Eds.), *International handbook of social anxiety: Concepts, research and interventions relating to the self and shyness* (pp. 405–430). New York, NY: John Wiley & Sons.

Coatsworth (2013). Could emotional security theory help advance family-focused prevenetative interventoins? In N. S. Lansdale, S. M. McHale, & A. Booth (Eds.), *Families and child health* (pp. 95–104). New York, NY: Springer.

Collins, N. L., Guichard, A. C., Ford, M. B., & Feeney, B. C. (2004). Working models of attachment: New developments and emerging themes. In: W. S. Rholes & J. A. Simpson (Eds.), *Adult attachment: Theory, research, and clinical implications* (pp. 196–239). New York, NY: Guildford Publications.

Cooley, M. R., Turner, S. M., & Beidel, D. C. (1995). Assessing community violence: The children's report of exposure to violence. *Journal of the American Academy of Child & Adolescent Psychiatry*, *34*, 201–208. doi: 10.1097/00004583-199502000-00015

Conger, R. D., & Conger, K. J. (2002). Resilience in Midwestern families: Selected findings from the decade of prospective, longitudinal

study. *Journal of Marriage and Family*, *64*, 361–373. doi: 10.1111/j.1741-3737.2002.00361.x

Conger, R. D., Conger, K. J., Matthews, L. S., & Elder, G. H. (1999). Pathways of economic influence on adolescent adjustment. *American Journal of Community Psychology*, *27*, 519–541. doi: 10.1023/A:1022133228206

Conger, R. D., & Donnellan, M. B. (2007). An interaction perspective on the socioeconomic context of human development. *Annual Review of Psychology*, *58*, 175–199. doi: 10.1146/annurev.psych.58.110405.085551

Conger, R. D., Wallace, L. E., Sun, Y., Simons, R. L., McLoyd, V. C., & Brody, G. H. (2002). Economic pressure in African American families: A replication and extension of the Family Stress Model. *Developmental Psychology*, *38*, 179–193. doi: 10.1037/0012-1649.38.2.179

Cook, C. R., Williams, K. R., Guerra, N. G., Kim, T. E., & Sadek, S. (2010). Predictors of bullying and victimization in childhood and adolescence: A meta-analytic investigation. *School Psychology Quarterly*, *25*, 65–83. doi: 10.1037/a0020149

Cowan, P. A., & Cowan, C. P. (2002). Interventions as tests of family systems theories: Marital and family relationships in children's development and psychopathology. *Development and Psychopathology*, *14*, 731–759. doi: 10.1017/S0954579402004054

Cowan, P. A., Cowan, C. P., Ablow, J. C., Johnson, V. K., & Measelle, J. R. (2005). *The family context of parenting in children's adaptation to elementary school*. Hillsdale, NJ: Erlbaum.

Cox, M. J., Mills-Koonce, R., Propper, C., & Gariépy, J. (2010). Systems theory and cascades in developmental psychopathology. *Development and Psychopathology*, *22*, 497–506. doi: 10.1017/S0954579410000234

Cox, M. J., & Paley, B. (1997). Families as systems. *Annual Review of Psychology*, *48*, 243–267. doi: 10.1146/annurev.psych.48.1.243

Cox, M. J., Paley, B., & Harter, K. (2001). Interparental conflict and parent–child relationships. In J. H. Grych & F. D. Fincham (Eds.), *Interparental conflict and child development: Theory, research, and applications* (pp. 249–272). New York, NY: Cambridge University Press.

Coyne, J. C., Downey, G., & Boergers, J. (1992). Depression in families: A systems perspective. In D. Cicchetti & S. L. Toth (Eds.), *Rochester symposium on developmental psychology: Developmental perspectives on depression, vol. 4* (pp. 211–250). Rochester, NY: University of Rochester Press.

Craig, W. M., & Pepler, D. J. (1998). Observations of bullying and victimization in the school yard. *Canadian Journal of School Psychology*, *13*, 41–59. doi: 10.1177/082957359801300205

Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information processing mechanisms in children's adjustment. *Psychological Bulletin*, *115*, 74–101. doi: 10.1037/0033-29091.15.1.74

Crittenden, P. M., Kozlowska, K., & Landini, A. (2010). Assessing attachment in school-age children. *Clinical Child Psychology and Psychiatry*, *15*, 185–208. doi: 10.1177/1359104509356741

Crockenberg, S., Forgays, D. (1996). The role of emotion in children's understanding and emotional reactions to marital conflict. *Merrill-Palmer Quarterly*, *42*, 22–47.

Crockenberg, S., & Langrock, A. (2001). The role of emotion and emotional regulation in children's responses to interparental conflict. In J. H. Grych & F. D. Fincham (Eds.), *Interparnetal conflict and child development: Theory, research, and applications* (pp. 129–156). New York, NY: Cambridge University Press.

Crockenberg, S. C., Leerkes, E. M., & Lekka, S. K. (2007). Pathways from marital aggression to infant emotion regulation: The development of withdrawal in infancy. *Infant Behavior and Development*, *30*, 97–113. doi: 10.1016/j.infbeh.2006.11.009

Cui, M., Conger, R. D., & Lorenz, F. O. (2005). Predicting change in adolescent adjustment from change in marital problems. *Developmental Psychology*, *41*, 812–823. doi: 10.1037/0012-1649.41.5.812

Cui, M., Donnellan, M. B., & Conger, R. D. (2007). Reciprocal influences between parents' marital problems and adolescent internalizing and externalizing behavior. *Developmental Psychology*, *43*, 1544–1552. doi: 10.1037/0012-1649.43.6.1544

Cummings, E. M. (1987). Coping with background anger in early childhood. *Child Development*, *58*, 976–984. doi: 10.2307/1130538

Cummings, E. M. (1995). Usefulness of experiments for the study of the family. *Journal of Family Psychology*, *9*, 175–185. doi: 10.1037/0893-3200.9.2.175

Cummings, E. M. (1998). Stress and coping approaches and research: The impact of marital conflict on children. *Journal of Aggression, Maltreatment & Trauma*, *2*, 31–50. doi: 10.1300/J146v02n01_03

Cummings, E. M., Ballard, M., El-Sheikh, M., & Lake, M. (1991). Resolution and children's responses to interadult anger. *Developmental Psychology*, *27*, 462–470. doi: 10.1037/0012-1649.27.3.462

Cummings, E. M., & Davies, P. T. (1994). *Children and marital conflict: The impact of family dispute and resolution*. New York, NY: Guilford.

Cummings, E. M., Davies, P. T. (1996). Emotional security as a regulatory process in normal development and the development of psychopathology. *Development and Psychopathology*, *8*, 123–139. doi: 10.1017/S0954579400007008

Cummings, E. M., & Davies, P. T. (2002). Effects of marital conflict on children: recent advances and emerging themes in process-oriented research. *Journal of Child Psychology and Psychiatry*, *43*, 31–63. doi: 10.1111/1469-7610.00003

Cummings, E.M., & Davies, P. T. (2010). *Marital conflict and children: An emotional security perspective*. New York, NY: Guilford Press.

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2000). *Developmental psychopathology and family process: Theory, research, and clinical implications*. New York, NY: Guilford Press.

Cummings, E. M., Faircloth, W. B., Mitchell, P. M., Cummings, J. S., & Schermerhorn, A. C. (2008). Evaluating a brief prevention program for improving marital conflict in community families. *Journal of Family Psychology*, *22*, 193–202. doi: 10.1037/0893-3200.22.2.193

Cummings, E. M., George, M. R. W., McCoy, K. P., & Davies, P. T. (2012). Interparental conflict in kindergarten and adolescent adjustment: Prospective investigation of emotional security as an explanatory mechanism. *Child Development*, *83*, 1703–1715. doi: 10.1111/j.1467-8624.2012.01807.x

Cummings, E. M., Goeke-Morey, M. C., & Papp, L. M. (2004). Everyday marital conflict and child aggression. *Journal of Abnormal Child Psychology*, *32*, 191–202. doi: 10.1023/B:JACP.0000019770.13216.be

Cummings, E. M., Goeke-Morey, M. C., & Papp, L. M., & Dukewich, T. L. (2002). Children's responses to mothers' and fathers' emotionality and conflict tactics during marital conflict in the home. *Journal of Family Psychology*, *16*, 478–492. doi: 10.1046/j.1467-8624.2003.00646.x

Cummings, E. M., Goeke-Morey, M. C., Schermerhorn, A. C., Merrilees, C. E., & Cairns, E. (2009). Children and political violence from a social ecological perspective: Implications from research on children and families in Northern Ireland. *Clinical Child and Family Psychology Review*, *12*, 16–38. doi: 10.1007/s10567-009-0041-8

Cummings, E. M., Keller, P. S., & Davies, P. T. (2005). Towards a family process model of maternal and paternal depressive symptoms: Exploring multiple relations with child and family functioning.

Journal of Child Psychology and Psychiatry, *46*, 479–489. doi: 10.1111/j.1469-7610.2004.00368.x

Cummings, E. M., & Merrilees, C. E. (2010). Identifying the dynamic processes underlying links between marital conflict and child adjustment. In M. S. Schulz, M. K. Pruett, P. K. Kerig, & R. D. Parke (Eds.), *Strengthening couple relationships for optimal child development: Lessons from research and intervention* (pp. 27–40). Washington, DC: American Psychological Association. doi: 10.1037/12058-003

Cummings, E. M., Merrilees, C. M., Schermerhorn, A. C., Goeke-Morey, M. C., Shirlow, P., & Cairns, E. (2010a). Testing a social ecological model for relations between political violence and child adjustment in Northern Ireland. *Development and Psychopathology*, *22*, 405–418. doi: 10.1017/S0954579410000143

Cummings, E. M., Merrilees, C. M., Schermerhorn, A. C., Goeke-Morey, M. C., Shirlow, P., & Cairns, E. (2010b). Political violence and child adjustment in Northern Ireland: Testing pathways in a social-ecological model including single- and two-parent families. *Developmental Psychology*, *46*, 827–841. doi: 10.1037/a0019668.

Cummings, E. M., Merrilees, C. E., Schermerhorn, A. C., Geoke-Morey, M. C., Shirlow, P., & Cairns, E. (2011). Longitudinal pathways between political violence and child adjustment: The role of emotional security about the community in Northern Ireland. *Journal of Abnormal Child Psychology*, *39*, 213–224. doi: 10.1007/s10802-010-9457-3

Cummings, E. M., & Schatz, J. N. (2012). Family conflict, emotional security, and child development: Translating research findings into a prevention program for community families. *Clinical Child and Family Psychology Review*, *15*, 14–27. doi: 10.1007/s10567-012-0112-0

Cummings, E. M., Schermerhorn, A. C., Davies, P. T., Goekey-Morey, M. C., & Cummings, J. S. (2006). Interparental discord and child adjustment: Prospective investigations of emotional security as an explanatory mechanism. *Child Development*, *77*, 132–152. doi: 10.1111/j.1467-8624.2006.00861.x

Cummings, E. M., Vogel, D., Cummings, J. S., & El-Sheikh, M. (1989). Children's responses to different forms of expression of anger between adults. *Child Development*, *60*, 1392–1404. doi: 10.2307/1130929

Cummings, E. M., Wilson, J., & Shamir, H. (2003). Reactions of Chilean and US children to marital discord. *International Journal of Behavioral Development*, *27*, 437–444. doi: 10.1080/01650250344000055

Cummings, E. M., Zahn-Waxler, C., & Radke-Yarrow, M. (1981). Young children's responses to expressions of anger and affection by others in the family. *Child Development*, *52*, 1274–1282. doi: 10.2307/1129516

Curtis, W. J., & Cicchetti, D. (2007). Emotion and resilience: A multilevel investigation of hemispheric electroencephalogram asymmetry and emotion regulation in maltreated and nonmaltreated children. *Development and Psychopathology*, *19*, 811–840. doi: 10.1017/S0954579407000405

Dakin, J., & Wampler, R. (2008). Money doesn't buy happiness, but it helps: Marital satisfaction, psychological distress, and demographic differences between low- and middle-income clinic couples. *American Journal of Family Therapy*, *36*, 300–311. doi: 10.1080/01926180701647512

Davies, P. T., & Cicchetti, D. (2004). Editorial: Toward an integration of family systems and developmental psychopathology approaches. *Development and Psychopathology*, *16*, 477–481. doi: 10.1017/S0954579404004626

Davies, P. T., Cicchetti, D., Hentges, R., & Sturge-Apple, M. L. (2013). the genetic precursors and the advantageous and disadvantageous sequelae of inhibited temperament: An evolutionary perspective. *Developmental Psychology*, *49*, 2285–2300. doi: 10.1037/a0032312

Davies, P. T., Cicchetti, D., & Martin, M. J. (2012). Towards greater specificity in identifying associations among interparental aggression, child emotional reactivity to conflict, and child problems. *Child Development*, *83*, 1789–1804. doi: 10.1111/j.1467-8624.2012.01804.x

Davies, P. T., & Cummings, E. M. (1994).Marital conflict and child adjustment: An emotional security hypothesis. *Psychological Bulletin*, *116*, 387–411. doi: 10.1037/0033-2909.116.3.387

Davies, P. T., & Cummings, E. M. (1998). Exploring children's emotional security as a mediator of the link between marital relations and child adjustment. *Child Development*, *69*, 124–139. doi: 10.1111/j.1467-8624.1998.tb06138.x

Davies, P. T., Cummings, E. M., & Winter, M. A. (2004). Pathways between profiles of family functioning, child security in the interparental subsystem, and child psychological problems. *Development and Psychopathology*, *16*, 525–550. doi: 10.1017/S0954579404004651

Davies, P. T., Dumenci, L., & Windle, M. (1999). The interplay between maternal depressive symptoms and marital distress in the prediction of adolescent adjustment. *Journal of Marriage and the Family*, *61*, 238–254. doi: 10.2307/353897

Davies, P. T., & Forman, E. M. (2002). Children's patterns of preserving emotional security in the interparental subsystem. *Child Development*, *73*, 1880–1903. doi: 10.1111/1467-8624.t01-1-00512

Davies, P. T., Forman, E. M., Rasi, J. A., & Stevens, K. I. (2002). Assessing children's emotional security in the interparental subsystem: The Security in the Interparental Subsystem (SIS) scales. *Child Development*, *73*, 544–562. doi: 10.1111/1467-8624.00423

Davies, P. T., Harold, G. T., Goeke-Morey, M., Cummings, E. M. (2002). Children's emotional security and interparental conflict. *Monographs of the Society for Research in Child Development*, *67*, 1–129. doi: 10.1111/1540-5834.00205

Davies, P. T., & Lindsay, L. L. (2001). Does gender moderate the effects of conflict on children? In J. Grych and F. Fincham (Eds.), *Interparental conflict and child development: Theory, research, and applications* (pp. 64–97). New York, NY: Cambridge University Press.

Davies, P. T., & Lindsay, L. L. (2004). Interparental conflict and adolescent adjustment: Why does gender moderate early adolescent vulnerability? *Journal of Family Psychology*, *18*, 170–180. doi: 10.1037/0893-3200.18.1.160

Davies, P. T., Manning, L. G., & Cicchetti, D. (2013). Tracing the developmental cascade of children's insecurity in the interparental relationship: The role of stage-salient tasks. *Child Development*, *84*, 297–312. doi: 10.1111/j.1467-8624.2012.01844.x

Davies, P. T., & Martin, M. J. (2013). The reformulation of emotional security theory: The role of children's social defense in developmental psychopathology. *Development and Psychopathology*, *25*, 1436–1454. doi:10.1017/S0954579413000709

Davies, P. T., Martin, M. J., & Cicchetti, D. (2012). Delineating the sequelae of destructive and constructive interparental conflict for children within an evolutionary framework. *Developmental Psychology*, *48*, 939–955. doi: 10.1037/a0025899

Davies, P. T., Myers, R. L., & Cummings, E. M. (1996). Responses of children and adolescents to marital conflict scenarios as a function of the emotionality of conflict endings. *Merrill-Palmer Quarterly*, *42*, 1–21.

Davies, P. T., Myers, R. L., Cummings, E. M., & Heindel, S. (1999). Adult conflict history and children's subsequent responses to conflict: An experimental test. *Journal of Family Psychology*, *13*, 610–628. doi: 10.1037/0893-3200.13.4.610

Davies, P. T., & Sturge-Apple, M. L. (2007). Advances in the formulation of emotional security theory: An ethologically-based perspective. *Advances in Child Behavior and Development*, *35*, 87–137.

Davies, P. T., & Sturge-Apple, M. L. (2014). Family context in the development of psychopathology. In M. Lewis & K. Rudolph (Eds), *Handbook of developmental psychopathology* (3rd ed., pp. 143–161). New York, NY: Springer.

Davies, P. T., Sturge-Apple, M. L., Bascoe, S. M., & Cummings, E. M. (2014). The legacy of early insecurity histories in shaping adolescent adaptation to interparental conflict. *Child Development*, *85*, 338–354. doi: 10.1111/cdev.12119

Davies, P. T., Sturge-Apple, M. L., & Cicchetti, D. (2011). Interparental aggression and children's adrenocortical reactivity: Testing an evolutionary model of allostatic load. *Development and Psychopathology*, *23*, 801–814. doi: 10.1017/S0954579411000319

Davies, P. T., Sturge-Apple, M. L., & Cicchetti, D., & Cummings, E. M. (2007). The role of child adrenocortical functioning in pathways between interparental conflict and child maladjustment. *Developmental Psychopathology*, *43*, 918–930. doi: 10.1037/0012-1649.43.4.918

Davies, P. T., Sturge-Apple, M. L., & Cicchetti, D., & Cummings, E. M. (2008). Adrenocortical underpinnings of children's psychological reactivity to interparental conflict. *Child Development*, *79*, 1693–1706. doi: 10.1111/j.1467-8624.2008.01219.x

Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., Manning, L. G., & Vonhold, S. E. (2012). Pathways and processes of risk in associations among maternal antisocial personality symptoms, interparental aggression, and preschooler's psychopathology. *Development and Psychopathology*, *24*, 807–832. doi: 10.1017/S0954579412000387

Davies, P. T., Sturge-Apple, M. L., & Cicchetti, D., Manning, L., & Zale, E. (2009). Children's patterns of emotional reactivity to conflict as explanatory mechanisms in links between interpartner aggression and child physiological functioning. *Journal of Child Psychology and Psychiatry*, *50*, 1384–1391. doi: 10.1111/j.1469-7610.2009.02154.x

Davies, P. T., Sturge-Apple, M. L., & Martin, M. J. (2013). Family discord and child health: An emotional security formulation. In N. S. Lansdale, S. M. McHale, & A. Booth (Eds.), *Families and child health* (pp. 45–74). New York, NY: Springer.

Davies, P. T., Sturge-Apple, M. L., Winter, M. A., Cummings, E. M., & Farrell, D. (2006). Child adaptational development in contexts of interparental conflict over time. *Child Development*, *77*, 218–233. doi: 10.1111/j.1467-8624.2006.00866.x

Davies, P. T., Sturge-Apple, M. L., Woitach, M. J., & Cummings, E. M. (2009). A process analysis of the transmission of distress from interparental conflict to parenting: Adult relationship security as an explanatory mechanism. *Developmental Psychology*, *45*, 1761–1773. doi: 10.1037/a0016426

Davies, P. T., & Windle, M. (2001). Interparental discord and adolescent adjustment trajectories: The potentiating and protective role of intrapersonal attributes. *Child Development*, *72*, 1163–1178. doi: 10.1111/1467-8624.00340

Davies, P. T., Winter, M. A., & Cicchetti, D. , D. (2006). The implications of emotional security theory for understanding and treating childhood psychopathology. *Development and Psychopathology*, *18*, 707–735. doi: 10.1017/S0954579406060354

Davies, P. T., & Woitach, M. J. (2008). Children's emotional security in the interparental relationship. *Current Directions in Psychological Science*, *17*, 269–274. doi: 10.1111/j.1467-8721.2008.00588.x

Davies, P. T., Woitach, M. J., Winter, M. A., & Cummings, E. M. (2008). Children's insecure representations of the interparental relationshp and their school adjustment: The mediating role of attention difficulties. *Child Development*, *79*, 1570–1582. doi: 10.1111/j.1467-8624.2008.01206.x

Davis, B. T., Hops, H., Alpert, A., & Sheeber, L. (1998). Child responses to parental conflict and their effect on adjustment. *Journal of Family Psychology*, *12*, 163–177. doi: 10.1037/0893-3200.12.2.163

Deater-Deckard, K., & Petrill, S. A. (2004). Parent–child dyadic mutuality and child behavior problems: An investigation of gene–environment processes. *Journal of Child Psychologyand Psychiatry*, *45*, 1171–1179. doi: 10.1111/j.1469-7610.2004.00309.x

Del Giudice, M., Angeleri, R., & Manera, V. (2009). The juvenile transition: A developmental switch point in human life history. *Developmental Review*, *29*, 1–31. doi: 10.1016/j.dr.2008.09.001

Del Giudice, M., & Belsky, J. (2011). The development of life history strategies: Toward a multistage theory. In D. M. Buss & P. H. Hawley (Eds.), *The evolution of personality and individual differences* (pp. 154–176). New York, NY: Oxford University Press.

Del Giudice, M., Ellis, B. J., Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience and Biobehavioral Reviews*, *35*, 1562–1592. doi: 10.1016/j.neubiorev.2010.11.007

Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences*, *22*, 491–569. doi: 10.1017/S0140525X99002046

Depue, R. A., & Fu, Y. (2012). Neurobiology and neurochemistry of temperament in adults. In M. Zentner & R. L. Shine (Eds.), *Handbook of temperament* (pp. 368–402). New York: Guilford Press.

Depue, R. A., & Morrone-Strupinsky, J. V. (2005). A neurobehavioral model of affiliative bonding: Implications for conceptualizing a human trait of affiliation. *Behavior and Brain Sciences*, *28*, 313–395. doi: 10.1017/S0140525X05000063

Dixon, A. K. (1998). Ethological strategies for defence in animals and humans: their role in some psychiatric disorders. *British Journal of Medical Psychology 71*, 417–445. doi: 10.1111/j.2044-8341.1998.tb01001.x

Dodge, K.A. (2006). Translational science in action: Hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology*, *18*, 791–814. doi: 10.1017/S0954579406060391

Dodge, K. A., & Godwin, J. (2013). Social-information-processing patterns mediate the impact of preventive intervention on adolescent antisocial behavior. *Psychological Science*, *24*, 456–465. doi: 10.1177/0956797612457394

DuPaul, G. J., Helwig, J. R., & Slay, P. M. (2011). Classroom interventions for attention and hyperactivity. In M. A. Bray & T. J. Kehle (Eds.), *The Oxford handbook of school psychology* (pp. 428–441). New York, NY: Oxford University Press.

Du Rocher Schudlich, T. D., & Cummings, E. M. (2003). Parental dysphoria and children's internalizing symptoms: Marital conflict styles as mediators of risk. *Child Development*, *74*, 1663–1681. doi: 10.1046/j.1467-8624.2003.00630.x.

Du Rocher Schudlich, T., & Cummings, E. M. (2007). Parental dysphoria and children's adjustment: Marital conflict styles, children's emotional security, and parenting as mediators of risk. *Journal of Abnormal Child Psychology*, *35*, 627–639. doi: 10.1007/s10802-007-9118-3

Du Rocher Schudlich, T. D., White, C. R., Fleischhauer, E. A., & Kitzgerald, K. A. (2011). Observed infant reactions during live interparental conflict. *Journal of Marriage and Family*, *73*, 221–235. doi: 10.1111/j.1741-3737.2010.00800.x

Dunn, J. & Davies, L. (2001). Sibling relationships and interparental conflict. In J. Grych & F. Fincham (Eds.), *Child Development and Interparental Conflict* (pp. 273–290). New York, NY: Cambridge University Press.

Easterbrooks, M. A., & Emde, R. N. (1988). Marital and parent–child relationships: The role of affect in the family system. In R. Hinde & J. Stevenson-Hinde (Eds.), *Relationships within families* (pp. 83–103). Oxford, UK: Clarendon Press.

Eggum, N. D., Eisenberg, N., Reiser, M., Spinrad, T. L., Valiente, C., Salquist, J., Michalik, N. M., & Liew, J. (2012). Relations over time among children's shyness, emotionality, and internalizing problems. *Social Development*, *21*, 109–129. doi: 10.1111/j.1467-9507.2011.00618.x

Eisenberg, N., Fabes, R. A., Schaller, M., & Miller, P. A. (1989). Sympathy and personal distress: Development, gender differences, and interrelations of indexes. *New Directions for Child Development, Summer*, 107–126. doi: 10.1002/cd.23219894408

Eisenberg, N., Fabes, R. A., & Spinrad, T. L. (2006). Prosocial development. In: N. Eisenberg & W. Damon (Eds.). *Handbook of child psychology, Vol. 3: Social, emotional, and personality development* (6th ed., pp. 646–718). New York, NY: Wiley.

Eisenberg, N., Huerta, S., & Edwards, A. (2012). Relations of empathy-related responding to children's and adolescents' social competence. In J. Decety (Ed.), *Empathy: From bench to bedside* (pp. 147–164). Cambridge, MA: Massachusetts Institute of Technology.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. *Development and Psychopathology*, *23*, 7–28. doi: 10.1017/S0954579410000611

Ellis, B. J., Essex, M. J., & Boyce, W. T. (2005). Biological sensitivity to context: II. Empirical explorations of an evolutionary-developmental theory. *Development and Psychopathology*, *17*, 303–328. doi: 10.1017/S0954579405050157

El-Sheikh, M. (2005). The role of emotional responses and physiological reactivity in the marital conflict–child functioning link. *Journal of Child Psychology and Psychiatry*, *46*, 1191–1199. doi: 10.1111/j.1469-7610.2005.00418.x

El-Sheikh, M., Buckhalt, J. A., Mark Cummings, E., & Keller, P. (2007). Sleep disruptions and emotional insecurity are pathways of risk for children. *Journal of Child Psychology and Psychiatry*, *48*, 88–96. doi: 10.1111/j.1469-7610.2006.01604.x

El-Sheikh, M., & Cummings, E. M. (1995). Children's responses to angry adult behavior as a function of experimentally manipulated exposure to resolved and unresolved conflict. *Social Development*, *4*, 75–91. doi: 10.1111/j.1467-9507.1995.tb00052.x

El-Sheikh, M., Cummings, E. M., Kouros, C. D., Elmore-Staton, L., & Buckhalt, J. (2008). Marital psychological and physical aggression and children's mental and physical health: Direct, mediated, and moderated effects. *Journal of Consulting and Clinical Psychology*, *76*, 138–148. doi: 10.1037/0022-006X.76.1.138

El-Sheikh, M., Harger, J., & Whitson, S. M. (2001). Exposure to interparental conflict and children's adjustment and physical health: The moderating role of vagal tone. *Child Development*, *72*, 1617–1636. doi: 10.1111/1467-8624.00369

El-Sheikh, M., Kouros, C. D., Erath, S., Cummings, E. M., Keller, P., & Staton, L. (2009). Marital conflict and children's externalizing behavior: Interactions between parasympathetic and sympathetic nervous system activity. *Monographs of the Society for Research in Child Development*, *74* (1, Serial No. 292), 1–79. doi: 10.1111/j.1540-5834.2009.00501.x

El-Sheikh, M. & Whitson, S. A. (2006). Longitudinal relations between marital conflict and child adjustment: Vagal regulation as a protective factor. *Journal of Family Psychology*, *20*, 30–39. doi: 10.1037/0893-3200.20.1.30

Emery, R. E. (1982). Interparental conflict and the children of discord and divorce. *Psychological Bulletin*, *92*, 310–330. doi: 10.1037/0033-2909.92.2.310

Emery, R. E. (1989). Family violence. *American Psychologist*, *44*, 321–328. doi: 10.1037/0003-066X.44.2.321

Erel, O., & Burman, B. (1995). Interrelatedness of marital relations and parent–child relations: A meta-analytic review. *Psychological Bulletin*, *118*, 108–132. doi: 10.1037/0033-2909.118.1.108

Evans, G. W. (2004). The environment of childhood poverty. *American Psychologist*, *59*, 77–92. doi: 10.1037/0003-066X.59.2.77

Evans, S. E., Davies, C., & DiLillo, D. (2008). Exposure to domestic violence: A meta-analysis of child and adolescent outcomes. *Aggression and Violent Behavior*, *13*, 131–140. doi: 10.1016/j.avb.2008.02.005

Faircloth, W. B., & Cummings, E. M. (2008). Evaluating a parent education program for preventing the negative effects of marital conflict. *Journal of Applied Developmental Psychology*, *29*, 141–156. doi: 10.1016/j.appdev.2007.12.004

Faircloth, W. B., Schermerhorn, A. C., Mitchell, P. M., Cummings, J. S., & Cummings, E. M. (2011). Testing the long-term efficacy of a prevention program for improving marital conflict in community families. *Journal of Applied Developmental Psychology*, *32*, 189–197. doi: 10.1016/j.appdev.2011.05.004

Falke, S. I., & Larson, J. H. (2007). Premarital predictors of remarital quality: Implications for clinicians. *Contemporary Family Therapy*, *29*, 9–23. doi: 10.1007/s10591-007-9024-4

Feerick, M. M., & Prinz, R. J. (2003). Next steps in research on children exposed to community violence or war/terrorism. *Clinical Child and Family Psychology Review*, *6*, 303–305. doi: 10.1023/B:CCFP.0000006296.19932.c3

Feinberg, M. E. (2003). The internal structure and ecological context of coparenting: A framework for research and intervention. *Parenting: Science and Practice*, *3*, 95–131. doi: 10.1207/S15327922PAR0302_01

Feinberg, M. E., & Kan, M. L. (2008). Establishing family foundations: Intervention effects on coparenting, parent/infant well-being, and parent–child relations. *Journal of Family Psychology*, *22*, 253–263. doi: 10.1037/0893-3200.22.2.253

Feldman, R. (2012). Oxytocin and social affiliation in humans. *Hormones and Behavior*, *61*, 380–391. doi: 10.1016/j.yhbeh.2012.01.008

Fincham, F. D., Grych, J. H., & Osborne, L. N. (1994). Does marital conflict cause child maladjustment? Directions and challenges for longitudinal research. *Journal of Family Psychology*, *8*, 128–140. doi: 10.1037/0893-3200.8.2.128

Flores, G., Fuentes-Afflick, E., Barbot, O., Carter-Pokras, O., Claudio, L., Lara, M., … Weitzman, M. (2002). The health of Latino children. *Journal of the American Medical Association*, *288*, 82–90. doi: 10.1001/jama.288.1.82

Ford, J. D. (2009). Neurobiological and developmental research: Clinical implications. In C. A. Courtois & J. D. Ford (Eds.), *Treating complex traumatic stress disorders: An evidenced-based guide* (pp. 31–58). New York, NY: Guildford Press.

Forman, E. M., & Davies, P. T. (2003). Family instability and young adolescent maladjustment: The mediating effects of parenting quality and adolescent appraisals of family security. *Journal of Clinical Child and Adolescent Psychology*, *32*, 94–105. doi: 10.1207/S15374424JCCP3201_09

Fosco, G. M., & Grych, J. H. (2007). Emotional expression in the family as a context for children's appraisals of interparental conflict. *Journal of Family Psychology*, *21*, 248–258. doi: 10.1037/0893-3200.21.2.248

Fraley, R. C., Roisman, G. I., & Haltigan, J.D. (2013). The legacy of early experiences in development: Formalizing alternative models of how early experiences are carried forward in time. *Developmental Psychology*, *49*, 109–126. doi: 10.1037/a0027852

Frankenhuis, W. E., & Del Giudice, M. (2012). When do adaptive developmental mechanisms yield maladaptive outcomes. *Developmental Psychology*, *48*, 628–642. doi: 10.1037/a0025629

Frewen, P. A., Dozois, D. J. A., Joanisse, M. F, & Neufeld, R. W. J. (2008). Selective attention to threat versus reqRD: Meta-analysis and neural-network modeling of the dot-probe task. *Clinical Psychology Review*, *28*, 307–337. doi: 10.1016/j.cpr.2007.05.006

Frick, P. J., Lahey, B. B., Loeber, R., Stouthamer-Loeber, M., Christ, M. A. G., & Hanson, K. (1992). Familial risk factors to oppositional defiant disorder and conduct disorder: parental psychopathology and maternal parenting. *Journal of Consulting and Clinical Psychology*, *60*, 49–55. doi: 10.1037/0022-006X.60.1.49

Furman, W. (1999). The role of peer relationships in adolescent romantic relationships. In W. A. Collins & B. Laursen (Eds.), *Minnesota Symposium on Child Development: Vol 29. Relationships as developmental contexts* (pp. 133–154). Hillsdale, NJ: Erlbaum.

Galbally, M., Lewis, A. J., van IJzendoorn, M., & Permezel, M. (2011). The role of oxytocin in mother-infant relations: A systematic review of human studies. *Harvard Review of Psychiatry*, *19*, 1–14. doi: 10.3109/10673229.2011.549771

Ganzel, B. L., & Morris, P. A. (2011). Allostasis and the developing human brain: Explicit consideration of implicit models. *Development and Psychopathology*, *23*, 955–974. doi: 10.1017/S0954579411000447

Garcia Coll, C. (2005). Editorial. *Developmental Psychology*, *41*, 299–300.

Garcia Coll, C., Akerman, A., & Cicchetti, D. (2000). Cultural influences on developmental processes and outcomes: Implications for the study of development and psychopathology. *Development and Psychopathology*, *12*, 333–356. doi: 10.1017/S0954579400003059

Garcia-O'Hearn, H., Margolin, G., & John, R. S. (1997). Mothers' and fathers' reports of children's reaction to naturalistic marital conflict. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 1366–1373. doi: 10.1097/00004583-199710000-00018

Gartstein, M. A., & Rothbart, M. K. (2003). Studying infant temperament via the revised infant behavior questionnaire. *Infant Behavior and Development*, *26*, 64–86. doi: 10.1016/S0163-6383(02)00169-8

Gass, K., Jenkins, J., & Dunn, J. (2007). Are sibling relatinoships protective? A longitudinal study. *Journal of Child Psychology and Psychiatry*, *48*, 167–175. doi: 10.1111/j.1469-7610.2006.01699.x

Gerard, J. G., & Buehler, C. (1999). Multiple family risk factors and youth problem behaviors. *Journal of Marriage and Family*, *61*, 343–361.

Gilbert, P. (1993). Defence and safety: Their function in social behavior and psychopathology. *British Journal of Clinical Psychology*, *32*, 131–153. doi: 10.1111/j.2044-8260.1993.tb01039.x

Gilbert, P. (2001). Evolutionary approaches to psychopathology: the role of natural defences. *Australian & New Zealand Journal of Psychiatry*, *35*, 17–27. doi: 10.1046/j.1440-1614.2001.00856.x

Gilbert, P. (2006). Evolution and depression: Issues and implications. *Psychological Medicine*, *36*, 287–297. doi: 10.1017/S0033291705006112

Gilbert, P., & Allan, S. (1998). The role of defeat and entrapment (arrested flight) in depression: An exploration of an evolutionary view. *Psychological Medicine*, *28*, 584–597. doi: 10.1017/S0033291798006710

Goeke-Morey, M. C., Cummings, E. M., Harold, G. T., & Shelton, K. H. (2003). Categories and continua of destructive and constructive marital conflict tactics from the perspective of U.S. and Welsh children. *Journal of Family Psychology*, *17*, 327–338. doi: 10.1037/0893-3200.17.3.327

Goeke-Morey, M. C., Cummings, E. M., & Papp, L. M. (2007). Children and marital conflict resolution: Implications for emotional security and adjustment. *Journal of Family Psychology*, *21*, 744–753. doi: 10.1037/0893-3200.21.4.744

Goeke-Morey, M. C., Papp, L. M., & Cummings, E. M. (2013). Changes in marital conflict and youths' responses across childhood and adolescence: A test of sensitization. *Development and Psychopathology*, *25*, 241–251. doi: 10.1017/S0954579412000995

Goetz, J. L., Keltner, D., & Simon-Thomas, E. (2010). Compassion: An evolutionary analysis and empirical review. *Psychological Bulletin*, *136*, 351–374. doi: 10.1037/ a0018807

Gold, P. W., & Chrousos, G. P. (2002). Organization of the stress system and its dysregulation in melancholic and atypical depression: High vs low CRH/NE states. *Molecular Psychiatry*, *7*, 254–275. doi: 10.1038/sj.mp.4001032

Goldberg, S., Grusec, J. E., & Jenkins, J. (1999). Confidence in protection: Arguments for a narrow definition of attachment. *Journal of Family Psychology*, *13*, 475–483. doi: 10.1037/0893-3200.13.4.475

Gomulak-Cavicchio, B. M., Davies, P. D., & Cummings, E. M. (2006). The role of maternal communication patterns about interparental disputes in associations between interparental conflict and child psychological maladjustment. *Journal of Abnormal Child Psychology*, *34*, 757–771. doi: 10.1007/s10802-006-9050-y

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, *106*, 458–490. doi: 10.1037/0033-295X.106.3.458

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical Child and Family Psychology Review*, *14*, 1–27. doi: 10.1007/s10567-010-0080-1

Gordon, I., Martin, C., Feldman, R., & Leckman, J. F. (2011). Oxytocin and social motivation. *Developmental Cognitive Neuroscience*, *1*, 471–493. doi: 10.1016/j.dcn.2011.07.007

Gore, S., Aseltine Jr., R. H., & Colten, M. E. (1993). Gender, social-relationship involvement, and depression. *Journal of Research on Adolescence*, *3*, 101–125. doi: 10.1207/s15327795jra0302_1

Gottlieb, G. (1991). Experiential canalization of behavioral development: Theory. *Developmental Psychology*, *27*, 4–13. doi: 10.1037/0012-1649.27.1.4

Gottman, J. M., & Katz, L. F. (1989). Effects of marital discord on young children's peer interactions and health. *Developmental Psychology*, *25*, 273–281. doi: 10.1037/0012-1649.25.3.373

Graham, A. M., Ablow, J. C., & Measelle, J. R. (2010). Interparental relationship dynamics and cardiac vagal functioning in infancy. *Infant Behavior and Development*, *33*, 530–544. doi: 10.1016/j.infbeh.2010.07.005

Granot, D., & Mayseless, O. (2012). Representations of mother–child attachment relationships and social-information processing of peer relationships in early adolescence. *The Journal of Early Adolescence*, *32*, 537–564. doi: 10.1177/0272431611403482

Gratz, K. L., Tull, M. T., Reynolds, E. K., Bagge, C. L., Latzman, R. D., Daughters, S. B., & Lejuez, C. W. (2009). Extending extant models of the pathogenesis of borderline personality disorder to childhood borderline personality symptoms: The roles of affective dysfunction, disinhibition, and self-and emotion-regulation deficits. *Development and Psychopathology*, *21*, 1263–1291. doi: 10.1017/S0954579409990150

Graziano, P., & Derefinko, K. (2013). Cardiac vagal control and children's adaptive functioning: A meta-analysis. *Biological Psychology*, *94*, 22–37. doi: 10.1016/j.biopsycho.2013.04.011

Grych, J. H. (1998). Children's appraisals of interparental conflict: Situational and contextual influences. *Journal of Family Psychology*, *12*, 437–453. doi: 10.1037/0893-3200.12.3.437

Grych, J. H. (2002). Marital relationships and parenting. In M. H. Bornstein (Ed.), *Handbook of parenting: Vol. 4: Social conditions and applied parenting* (2nd ed., pp. 203–225). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Grych, J. H., & Fincham, F. D. (1990). Marital conflict and children's adjustment: A cognitive-contextual framework. *Psychological Bulletin*, *111*, 434–454. doi: 10.1037/0033-2909.108.2.267

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145–173. doi: 10.1146/annurev.psych.58.110405.085605

Halgunseth, L. C., Ispa, J. M., & Rudy, D. (2006). Parental control in Latino families: An integrated review of the literature. *Child Development*, *77*, 1282–1297. doi: 10.1111/j.1467-8624.2006.00934.x

Hahlweg, K., & Richter, D. (2010). Prevention of marital instability and distress. Results of an 11-year longitudinal follow-up study. *Behaviour Research and Therapy*, *48*, 377–383. doi: 10.1016/j.brat.2009.12.010

Hanish, L. D., & Guerra, N. G. (2000). Predictors of peer victimization among urban youth. *Social Development*, *9*, 521–543. doi: 10.1111/1467-9507.00141

Hanish, L.D., & Guerra, N. G. (2004). Aggressive victims, passive victims, and bullies: Developmental continuity or developmental change. *Merrill-Palmer Quarterly*, *50* 17–38. doi: 10.1353/mpq.2004.0003

Harkness, K. L., Bruce, A. E., & Lumley, M. N. (2006). The role of childhood abuse and neglect in the sensitization to stressful life events in adolescent depression. *Journal of Abnormal Psychology*, *115*, 730–741. doi: 10.1037/0021-843X.115.4.730

Harold, G. T., & Leve, L. D. (2012). Parents as partners: How the parental relationship affects children's development. In A. Balfour, M. Morgan, & C. Vincent (Eds.), *How couple relationships shape our world: Clinical practice, research, and policy perspectives* (pp. 25–56). London, UK: Karnac.

Harold, G. T., Shelton, K. H., Goeke-Morey, M. C., & Cummings, E. M. (2004). Marital conflict, child emotional security about family relationships, and child adjustment. *Social Development*, *13*, 350–376. doi: 10.1111/j.1467-9507.2004.00272.x

Hatchett, S. J., & Jackson, J. S. (1993), African American extended kin systems: An assessment. In H. P. McAdoo (Ed.), *Family ethnicity: Strength in diversity* (pp. 90–108). Newbury Park, CA: Sage.

Hawley, P. H., Little, T. D., & Rodkin, P. C. (Eds.) (2007). *Aggression and adaptation: The bright side to bad behavior*. New York, NY: Lawrence Erlbaum Associates.

Helfinstein, S. M., Fox, N. A., & Pine, D. S. (2012). Approach–withdrawal and the role of the striatum in the temperament of behavioral inhibition. *Developmental Psychology*, *48*, 815–826. doi: 10.1037/a0026402

Hilburn-Cobb, C. (2004). Adolescent psychopathology in terms of multiple behavioral systems: The role of attachment and controlling strategies and frankly disorganized behavior. In L. Atkinson & S. Goldberg (Eds), *Attachment issues in psychopathology and intervention* (pp. 95–135). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Hinnant, J. B., El-Sheikh, M., Keiley, M., & Buckhalt, J. A. (2013). Martial conflict, allostatic load, and the development of children's fluid cognitive performance. *Child Development*, *84*, 2003–2014. doi: 10.1111/cdev.12103

Hofmann, S. G., Moscovitch, D. A., & Heinrichs, N. (2002). Evolutionary mechanisms of fear and anxiety. *Journal of Cognitive Psychotherapy*, *16*, 317–330.

Hofmann, S. G. (2007). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy*, *36*, 193–209. doi: 10.1080/16506070701421313

Humbad, M. N., Donnellan, M. B., Iacono, W. G., & Burt, S. A. (2010). Externalizing psychopathology and marital adjustment in long-term marriages: Results from a large combined sample of married couples. *Journal of Abnormal Psychology*, *119*, 151–162. doi: 10.1037/a0017981

Irons, C., & Gilbert, P. (2005). Evolved mechanisms in adolescent anxiety and depression symptoms: The role of the attachment and social rank systems. *Journal of Adolescence*, *28*, 325–341. doi: 10.1016/j.adolescence.2004.07.004

Jacobvitz, D., Hazen, N., Curran, M., & Hitchens, K. (2004). Observations of early triadic family interactions: Boundary disturbances in the family predict symptoms of depression, anxiety, and attention-deficit/hyperactivity disorder in middle childhood. *Development and Psychopathology*, *16*, 577–592. doi: 10.1017/S0954579404004675

Jaffee, S. R., Belsky, J., Harrington, H., Caspi, A., & Moffitt, T. E. (2006). When parents have a history of conduct disorder: How is the caregiving environment affected? *Journal of Abnormal Psychology*, *115*, 309–319. doi: 0.1037/0021-843X.115.2.309

Jenkins, J., Simpson, A., Dunn, J., Rasbash, J., & O'Connor, T. G. (2005). Mutual influence of marital conflict and children's behavior problems: Shared and nonshared family risks. *Child Development*, *76*, 24–39. doi: 10.1111/j.1467-8624.2005.00827.x

Jenkins, J. M., & Smith, M. A. (1990). Factors protecting children living in disharmonious homes: Maternal reports. *Journal of American Academy of Child and Adolescent Psychiatry*, *29*, 60–69. doi: 10.1097/00004583-199001000-00011

Jensen, P. S., Mrazek, D. A., Knapp, P., Steinberg, L., Pfeffer, C. R., Schowalter, J., & Shapiro, T. (2006). Application of evolutionary models to attention deficit/hyperactivity disorder. In P. Jensen, P. Knapp, & D. A. Mrazek (Eds.), *Toward a new diagnostic system for child psychopathology: Moving beyond the DSM* (pp. 96–110). New York, NY: Guilford Press.

Johnson, V. K. (2010). From early childhood to adolescence: Linking family functioning and school behavior. *Family Relations*, *59*, 313–325. doi: 10.1111/j.1741-3729.2010.00604.x

Johnston, J. R., & Campbell, L. E. G. (1988). *Impasses of divorce: The dynamics and resolution of family conflict*. New York, NY: Free Press.

Johnston, J., Roseby, V., & Kuehnle, K. (2009). *In the name of the child: A developmental approach to understanding and helping children of conflict and violent divorce*. New York, NY: Springer Publishing.

Jouriles, E. N., & Farris, A. M. (1992). Effects of marital conflict on subsequent parent-son interactions. *Behavior Therapy*, *23*, 355–374. doi: 10.1016/S0005-7894(05)80163-7

Juster, R. P., Sindi, S., Marin, M. F., Perna, A., Hashemi, A., Pruessner, J. C., & Lupien, S. J. (2011). A clinical allostatic load index is associated with burnout symptoms and hypocortisolemic profiles in healthy workers. *Psychoneuroendocrinology*, *36*, 797–805. doi: 10.1016/j.psyneuen.2010.11.001

Juvonen, J., & Graham, S. (2001). *Peer harassment in school: The plight of the vulnerable and victimized*. New York, NY: Guilford Press.

Kaczynski, K. J., Lindahl, K. M., Malik, N. M., & Laurenceau, J. P. (2006). Marital conflict, maternal and paternal parenting, and child adjustment: A test of mediation and moderation. *Journal of Family Psychology*, *20*, 199–208. doi: 10.1037/0893-3200.20.2.199

Kaminski, J. W., Valle, L. A., Filene, J. H., & Boyle, C. L. (2008). A meta-analytic review of components associated with parent training program effectiveness. *Journal of Abnormal Child Psychology*, *36*, 567–589. doi: 10.1007/s10802-007-9201-9

Kaplan, S., & Berman, M. G. (2010). Directed attention as a common resource for executive functioning and self-regulation. *Perspectives on Psychological Science*, *5*, 43–57. doi: 10.1177/1745691609356784

Kazak, A. E. (2004). Editorial: Context, collaboration, and care. *Journal of Family Psychology*, *18*, 3–4. doi: 10.1037/0893-3200.18.1.3

Keller, P. S., El-Sheikh, M., Keiley, M., & Liao, P. J. (2009). Longitudinal relations between marital aggression and alcohol problems. *Psychology of Addictive Behaviors*, *23*, 2–13. doi: 10.1037/a0013459

Kelly, R. J., & El-Sheikh, M. (2013). Longitudinal relations between marital aggression and children's sleep: The role of emotional insecurity. *Journal of Family Psychology*, *27*, 282–292. doi: 10.1037/a0031896

Kennedy, S., & Bumpass, L. (2008). Cohabitation and children's living arrangements: New estimates from the United States. *Demographic Research*, *19*, 1663–1692.

Kerig, P. K. (1995). Triangles in the family circle: Effects of family structure on marriage, parenting, and child adjustment. *Journal of Family Psychology*, *9*, 28–43. doi: 10.1037/0893-3200.9.1.28

Kerr, M. H., Beck, K., Downs Shattuck, T., Kattar, C., & Uriburu, D. (2003). Family involvement, problem and prosocial behavior outcomes of Latino youth. *American Journal of Health Behavior*, *27*, S55–S65.

Kitzmann, K. M., Gaylord, N. K., Holt, A. R., & Kenny, E. D. (2003). Child witnesses to domestic violence: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *71*, 339–352. doi: 10.1037/0022-006X.71.2.339

Klein, S. B., Cosmides, L., Tooby, J., & Chance, S. (2002). Decisions and the evolution of memory: multiple systems, multiple functions. *Psychological Review*, *109*, 306–329. doi: 10.1037/0033-295X.109.2.306

Konner, M. (2010). *The evolution of childhood: Relationships, emotion, mind*. Cambridge, MA: Harvard University Press.

Korte, S. M., Koolhaas, J. M., Wingfield, J. C., & McEwen, B. S. (2005). The Darwinian concept of stress: Benefits of allostasis and costs of allostatic load and the trade-offs in health and disease. *Neuroscience and Biobehavioral Reviews*, *29*: 3–38. doi: 10.1016/j.neubiorev.2004.08.009

Kosfeld, M., Heinrichs, M., Zak, P. J., Fischbacher, U., & Fehr, E. (2005). Oxytocin increases trust in humans. *Nature*, *435*, 673–676. doi: 10.1038/nature03701

Koss, K. J., George, M. R. W., Davies, P. T., Cicchetti, D., Cummings, E. M., & Sturge-Apple, M. L. (2013). Patterns of children's adrenocortical reactivity to interparental conflict and associations with child adjustment: A growth mixture modeling approach. *Developmental Psychology*, *49*, 317–326. doi: 10.1037/a0028246

Kouros, C. D., Cummings, E. M., & Davies, P. T. (2010). Early trajectories of interparental conflict and externalizing problems as predictors of social competence in preadolescence. *Development and Psychopathology*, *22*, 527–537. doi: 10.1017/S0954579410000258

Kouros, C. D., Merrilees, C. E., & Cummings, E. M. (2008). Marital conflict and children's emotional security in the context of parental depression. *Journal of Marriage and Family*, *70*, 684–697. doi: 10.1111/j.1741-3737.2008.00514.x

Kretchmar, M. D., & Jacobvitz, D. B. (2002). Observing mother–child relationships across generations: Boundary patterns, attachment, and the transmission of caregiving. *Family Process*, *41*, 351–374. doi: 10.1111/j.1545-5300.2002.41306.x

Krishnakumar, A., Buehler, C., & Barber, B. K. (2003). Youth perceptions of interparental conflict, ineffective parenting, and youth problem behaviors in European-American and African-American families. *Journal of Social and Personal Relationships*, *20*, 239–260. doi: 10.1177/02654075030202007

Lamborn, S. D., & Felbab, A. J. (2003). Applying ethnic equivalence and cultural values models to African-American teens' perceptions of parents. *Journal of Adolescence*, *26*, 601–618. doi: 10.1016/S0140-1971(03)00059-9

Lansford, J. E., Malone, P. S., Dodge, K. A., Pettit, G. S., & Bates, J. E. (2010). Developmental cascades of peer rejection, social information processing biases, and aggression during middle childhood. *Development and Psychopathology*, *22*, 593–602. doi: 10.1017/S0954579410000301

Larsen, H., Branje, S. J., van der Valk, I., & Meeus, W. H. (2007). Friendship quality as a moderator between perception of interparental conflicts and maladjustment in adolescence. *International Journal of Behavioral Development*, *31*, 549–558. doi: 10.1177/0165025407080578

Laursen, B., & Pursell, G. (2009). Conflict in peer relationships. In K. H. Rubin, W. M. Bukowski, & B. Laursen (Eds.), *Handbook of peer interactions, relationships, and groups* (pp. 267–286). New York, NY: Guilford.

Leon, K. (2003). Risk and protective factors in young children's adjustment to parental divorce: A review of the research. *Family Relations*, *52*, 258–270. doi: 10.1111/j.1741-3729.2003.00258.x

Leve, L. D., Kerr, D. C., Shaw, D., Ge, X., Neiderhiser, J. M., Scaramella, L. V., . . . Reiss, D. (2010). Infant pathways to externalizing behavior: evidence of genotype × environment interaction. *Child Development*, *81*, 340–356. doi: 10.1111/j.1467-8624.2009.01398.x

Leve, L. D., Neiderhiser, J. M., Ge, X., Scaromella, L. V., Conger, R. D., Reid, J. B., Shaw, D. S. & Reiss, D. (2007). The early growth and development study: A prospective adoption design. *Twin Research and Human Genetics*, *10*, 84–95. doi: 10.1375/twin.10.1.84

Levendosky, A. A., Leahy, K. L., Bogat, G. A., Davidson, W. S., & von Eye, A. (2006). Domestic violence, maternal parenting, maternal mental health, and infant externalizing behavior. *Journal of Family Psychology*, *20*, 544–552.

Liddle, H. A., & Schwartz, S. J. (2002). Attachment and family therapy: Clinical utility of adolescent-family attachment research. *Family Process*, *41*, 455–476. doi: 10.1111/j.1545-5300.2002.41311.x

Lindahl, K. M., & Malik, N. M. (2011). Marital conflict typology and children's appraisals: The moderating role of family cohesion. *Journal of Family Psychology*, *25*, 194–201. doi: 10.1037/a0022888.

Lindsey, E. W., Caldera, Y., & Colwell, M. (2005). Correlates of coparenting during infancy, *Family Relations*, *54*, 346–359. doi: 10.1111/j.1741-3729.2005.00322.x

Lindsey, E. W., Caldera, Y. M., & Tankersley, L. (2009). Marital conflict and the quality of young children's peer play behavior: The mediating and moderating role of parent–child emotional reciprocity and attachment security. *Journal of Family Psychology*, *23*, 130–145. doi: 10.1037/a0014972

Lindsey, E. W., Colwell, M. J., Frabutt, J. M., & MacKinnon-Lewis, C. (2006). Family conflict in divorced and non-divorced families: Potential consequences for boys' friendship status and friendship quality. *Journal of Social and Personality Relationships*, *23*, 45–63. doi: 10.1177/0265407506060177

Lindsey, E. W., Cremeens, P. R., Colwell, M. J., & Caldera, Y. M. (2009). The structure of parent–child dyadic synchrony in toddlerhood and children's communication competence and self-control. *Social Development*, *18*, 375–396. doi: 10.1111/j.1467-9507.2008.00489.x

LoBue, V. (2009). More than just another face in the crowd: Superior detection of threatening facial expressions in children and adults. *Developmental Science*, *12*, 305–313. doi: 10.1111/j.1467-7687.2008.00767.x

Loew, B., Rhoades, G., Markman, H., Stanley, S., Pacifici, C., White, L., & Delaney, R. (2012). Internet delivery of PREP-based

relationship education for at-risk couples. *Journal of Couple & Relationship Therapy*, *11*, 291–309. doi: 10.1080/15332691.2012.718968

Luebbe, A. M., Bell, D., Allwood, M., Swenson, L., & Early, M. (2010). Social information processing in children: Specific relations to anxiety, depression, and affect. *Journal of Clinical Child and Adolescent Psychology*, *39*, 386–399. doi: 10.1080/15374411003691685

Luthar, S. S. (1993). Annotation: Methodological and conceptual issues in research on childhood resilience. *Journal of Child Psychology and Psychiatry*, *34*, 441–453. doi: 10.1111/j.1469-7610.1993.tb01030.x

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, *71*, 543–562. doi: 10.1111/1467-8624.00164

Lynch, M., & Cicchetti, D. (1998). An ecological-transactional analysis of children and contexts: The longitudinal interplay among child maltreatment, community violence, and children's symptomatology. *Development and Psychopathology*, *10*, 235–257. doi: 10.1017/S095457949800159X

MacDonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin*, *131*, 202–223. doi: 10.1037/0033-2909.131.2.202

MacDonald, K. (2012). Cutting nature at its joints: Toward an evolutionarily informed theory of natural types of conduct disorder. *Journal of Social, Evolutionary, and Cultural Psychology*, *6*, 260–291.

Magdol, L., Moffitt, T. E., Caspi, A., & Silva, P. A. (1998). Developmental antecedents of partner abuse: A prospective-longitudinal study. *Journal of Abnormal Psychology*, *107*, 375–389. doi: 10.1037/0021-843X.107.3.375

Mann, B. J., & Gilliom, L. A. (2004). Emotional security and cognitive appraisals mediate the relationship between parents' marital conflict and adjustment in older adolescents. *Journal of Genetic Psychology*, *165*, 250–271. doi: 10.3200/GNTP.165.3.250-271

Manning, L. G., Davies, P. T., & Cicchetti, D. (2013). Interparental violence and early childhood adjustment: A mediated moderation examination of maternal sensitivity as protective. Manuscript submitted for publication.

Margolin, G., & Gordis, E. B. (2000). The effects of family and community violence on children. *Annual Review of Psychology*, *51*, 445–479. doi: 10.1146/annurev.psych.51.1.445

Margolin, G., & John, R. S. (1997). Children's exposure to marital aggression: Direct and mediated effects. In G. K. Kantor & J. L. Jasinki (Eds.), *Out of darkness: Contemporary perspectives on family violence* (pp. 90–104). Thousand Oaks, CA: Sage Publications.

Margolin, G., Oliver, P. H., & Medina, A. M. (2001). Conceptual issues in understanding the relation between interparental conflict and child adjustment: Integrating developmental psychopathology and risk/resilience perspectives. In J. H. Grych & D. Fincham (Eds.), *Interparental conflict and child development: Theory, research, and applications* (pp. 9–38). New York, NY: Cambridge University Press.

Markman, H. J., & Rhoades, G. K. (2012). Relationship education research: Current status and future directions. *Journal of Marital and Family Therapy*, *38*, 169–200. doi: 10.1111/j.1752-0606.2011.00247.x

Markman, H. J., Rhoades, G. K., Delaney, R., White, L., & Pacifici, C. (2010). Extending the reach of research-based couples interventions: The role of relationship education. In K. Hahlweg, M. Grawe-Gerber, & D. H. Baucom (Eds.), *Enhancing couples: The shape of couple therapy to come* (pp. 128–141). Cambridge, MA: Hogrefe Publishing.

Marks, I. M., & Nesse, R. M. (1994). Fear and fitness: An evolutionary analysis of anxiety disorders. *Ethology and Sociobiology*, *15*, 247–261. doi: 10.1016/0162-3095(94)90002-7

Markiewicz, D., Doyle, A., & Brendgen, M. (2001). The quality of adolescents' friendships: Associations with mothers' interpersonal relationships, attachments to parents and friends, and prosocial behaviors. *Journal of Adolescence*, *24*, 429–445. doi: 10.1006/jado.2001.0374

Marshall, P., & Stevenson Hinde, J. (2005). Behavioral inhibition: Physiological correlates. In W. R. Crozier & L. E. Alden (Eds.), *The essential handbook of social anxiety for clinicians* (pp. 57–80). Sussex, UK: John Wiley & Sons.

Martin, M. J., Davies, P. T., & MacNeill (2012). Social defense: An evolutionary-developmental model of children's strategies for coping with threat in the peer group. Manuscript submitted for publication.

Martinez, P., & Richters, J. E. (1993). The NIMH community violence project: II. Children's distress symptoms associated with violence exposure. *Psychiatry: Interpersonal Biological Processes*, *56*, 22–35.

Masten, A. S. (2005). Ordinary magic: Resilience processes in development. In M. E. Hertzig & E. A. Farber (Eds.), *Annual progress in child psychiatry and child development 2002* (pp. 115–142). New York, NY: Routledge.

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*, 491–495. doi: 10.1017/S0954579410000222

Masten, A. S., & Tellegen, A. (2012). Resilience in developmental psychopathology: contributions of the Project Competence Longitudinal Study. *Development and Psychopathology*, *24*, 345–361. doi: 10.1017/S095457941200003X

Maughan, A., & Cicchetti, D. (2002). Impact of child maltreatment and interadult violence on children's emotion regulation abilities and socioemotional adjustment. *Child Development*, *73*, 1525–1542. doi: 10.1111/1467-8624.00488

McAdoo, H. P. (1978). Factors related to stability in upwardly mobile black families. *Journal of Marriage and the Family*, *40*, 761–776.

McCrae, R. R., Costa, P. T., Jr., Ostendorf, F., Angleitner, A., Hřebíčková, M., Avia, M. D., . . . Smith, P. B. (2000). Nature over nurture: temperament, personality, and life span development. *Journal of Personality and Social Psychology*, *78*, 173–186. doi: 10.1037/0022-3514.78.1.173

McCoy, K., Cummings, E. M., & Davies, P. T. (2009). Constructive and destructive marital conflict, emotional security, and children's prosocial behavior. *Journal of Child Psychology and Psychiatry*, *50*, 270–279. doi: 10.1111/j.1469-7610.2008.01945.x

McElhaney, K. B., Allen, J. P., Stephenson, J. C., & Hare, A. L. (2009). Attachment and autonomy during adolescence. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology* (pp. 358–403). Hoboken, NJ: Wiley.

McElwain, N. L., & Booth-LaForce, C. (2006). Maternal sensitivity to infant distress and nondistress as predictors of infant-mother attachment security. *Journal of Family Psychology*, *20*, 247–255. doi: 10.1037/0893-3200.20.2.247

McEwen, B. (2002). Estrogen actions throughout the brain. *Recent Progress in Hormone Research*, *57*, 357–384. doi: 10.1210/rp.57.1.357

McEwen, B. S. (2006). Sleep deprivation as a neurobiologic and physiologic stressor, allostasis and allostatic load. *Metabolism*, *55*, 20–23. doi: 10.1016/j.metabol.2006.07.008

McHale, J. P. (1995). Coparenting and triadic interactions during infancy: The role of marital distress and child gender. *Developmental Psychology*, *31*, 985–996. doi: 10.1037/0012-1649.31.6.985

McLoyd, V. C., Harper, C. I., & Copeland, N. L. (2001). Ethnic minority status, interparental conflict, and child adjustment. In J. H. Grych & F. D. Fincham (Eds.), *Interparental conflict and child development: Theory, research, and application* (pp. 98–125). New York, NY: Cambridge University Press.

Mead, H. K., Beauchaine, T. P., & Shannon, K. E. (2010). Neurobiological adaptations to violence across development. *Development and Psychopathology*, *22*, 1–22. doi: 10.1017/S0954579409990228

Mindell, J., & Owens, J. A. (2010). *A clinical guide to pediatric sleep: Diagnosis and management of sleep problems* (2nd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Mindell, J. A., Owens, J., Alves, R., Bruni, O., Goh, D. Y., Hiscock, H., Kohyama, J., & Sadeh, A. (2011). Give children and adolescents the gift of a good night's sleep: A call to action. *Sleep Medicine*, *12*, 203–204. doi: 10.1016/j.sleep.2011.01.003

Minuchin, S. (1974). *Families and family therapy*. Cambridge, MA: Harvard University Press.

Monroe, S. M., & Harkness, K. L. (2005). Life stress, the "kindling" hypothesis, and the recurrence of depression: considerations from a life stress perspective. *Psychological Review*, *112*, 417–444. doi: 10.1037/0033-295X.112.2.417

Morris, M. C., Ciesla, J. A., & Garber, J. (2010). A prospective study of stress autonomy versus stress sensitization in adolescents at varied risk for depression. *Journal of Abnormal Psychology*, *119*, 341–354. doi: 10.1037/a0019036

Moss, E., St-Laurent, D., Dubois-Comtois, K., & Cyr, C. (2005). Quality of attachment at school age. In K. A. Kerns & R. A. Richardson (Eds.), *Attachment in middle childhood* (pp. 189–211). New York, NY: Guilford Press.

Nievar, M. A., & Luster, T. (2006). Developmental processes in African American families: An application of McLoyd's theoretical model. *Journal of Marriage and Family*, *68*, 320–331. doi: 10.1111/j.1741-3737.2006.00255.x

Nepple, T. K., Donnellan, M. B., Scaramella, L. V., Widaman, K. F., Spillman, S. K., Ontai, L. L., & Conger, R. D. (2010). Differential stability of temperament and personality from toddlerhood to middle childhood. *Journal of Research in Personality*, *44*, 386–396. doi:10.1016/jrp2010.04.004

Numan, M. (2006). Hypothalamic neural circuits regulating maternal responsiveness toward infants. *Behavioral and Cognitive Neuroscience Reviews*, *5*, 163–190. doi: 10.1002/dev.20198

Öhman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review*, *108*, 483–522. doi: 10.1037//0033-295x.108.3.483

Olson, D. H., Russell, C. S., & Sprenkle, D. H. (1983). Circumplex model of marital and family systems: Vl. Theoretical update. *Family Process*, *22*, 69–83. doi: 10.1111/j.1545-5300.1983.00069.x

Owen, M. T., & Cox, M. J. (1997). Marital conflict and the development of infant–parent attachment relationships. *Journal of Family Psychology*, *11*, 152–164. doi: 10.1037/0893-3200.11.2.152

Owen, A. E., Thompson, M. P., & Kaslow, N. J. (2006). The mediating role of parenting stress in the relation between intimate partner violence and child adjustment. *Journal of Family Psychology*, *20*, 505–513. doi: 10.1037/0893-3200.20.3.505

Panksepp, J. (1998). *Affective neuroscience: The foundation of human and animal emotions*. New York, NY: Oxford University Press.

Panksepp, J., & Moskal, J. (2008). Dopamine and SEEKING: Subcortical "reward" systems and appetitive urges. In A. Elliot (Ed.), *Handbook of approach and avoidance motivation* (pp. 67–87). New York, NY: Taylor & Francis Group, LLC.

Papp, L. M., Goeke-Morey, M. C., & Cummings, E. M. (2007). Linkages between spouses' psychological distress and marital conflict in the home. *Journal of Family Psychology*, *21*, 533–537. doi: 10.1037/0893-3200.21.3.533

Parke, R. D. (1998). Editorial. *Journal of Family Psychology*, *12*, 3–6.

Parke, R. D., Coltrane, S., Duffy, S., Buriel, R., Dennis, J., Powers, J., French, S., & Widaman, K. F. (2004). Economic stress, parenting, and child adjustment in Mexican American and European American families. *Child Development*, *75*, 1632–1656. doi: 10.1111/j.1467-8624.2004.00807.x

Parker, J. G., Rubin, K. H., Erath, S. A., Wojslawowicz, J. C., & Buskirk, A. A. (2006). Peer relationships, child development, and adjustment: A developmental psychopathology perspective. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (pp. 419–493). Hoboken, NJ: Wiley & Sons.

Patterson, G. R. (1982). *Coercive family process*. Eugene, OR: Castalia.

Pauli-Pott, U., & Beckmann, D. (2007). On the association of interparental conflict with developing behavioral inhibition and behavior problems in early childhood. *Journal of Family Psychology*, *21*, 529–532. doi: 10.1037/0893-3200.21.3.529

Pollak, S. D., & Tolley-Schell, S. (2004). Attention, emotion, and the development of psychopathology. In M. I. Posner (Ed.), *Cognitive neuroscience of attention* (pp. 357–368). New York, NY: Guilford.

Porges, S. W. (1995). Orienting in a defensive world: mammalian modifications of our evolutionary heritage: A polyvagal theory. *Psychophysiology*, *32*, 301–318. doi: 10.1111/j.1469-8986.1995.tb01213.x

Porges, S. W. (2003). Social engagement and attachment. *Annals of the New York Academy of Sciences*, *1008*, 31–47. doi: 10.1196/annals.1301.004

Porges, S.W. (2006). Asserting the role of biobehavioral sciences in translational research: the behavioral neurobiology revolution. *Developmental Psychopathology*, *18*, 923–933. doi: 10.1017/S0954579406060457

Prinstein, M. J., Cheah, C. S. L., & Guyer, A. E. (2005). Peer victimization, cue interpretation, and internalizing symptoms: Concurrent and longitudinal findings for children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, *34*, 11–24. doi: 10.1207/s15374424jccp3401_2

Putnam, S. P., Ellis, L. K., & Rothbart, M. K. (2001). The structure of temperament from infancy through adolescence. In A. Eliasz & A. Angleitner (Eds.), *Advances in research on temperament* (pp. 165–182). Berlin, Germany: Pabst Scientist Publisher.

Quinkert, A. W., Vimal, V., Weil, Z. M., Reeke, G. N., Schiff, N. D., Banavar, J. R. & Pfaff, D. W. (2011). Quantitative descriptions of generalized arousal, an elementary function of the vertebrate brain. *Proceedings of the National Academy of Sciences*, *108*, 15617–15623.

Reiss, D., & Leve, L. D. (2007). Genetic expression outside the skin: Clues to mechanisms of genotype × environment interaction. *Development and Psychopathology*, *19*, 1005–1027. doi: 10.1017/S0954579407000508

Repetti, R. L., Robles, T. F., & Reynolds, B. (2011). Allostatic processes in the family. *Development and Psychopathology*, *23*, 921–938. doi: 10.1017/S095457941100040X

Rhoades, K. A. (2008). Children's responses to interparental conflict: A meta-analysis of their associations with child adjustment. *Child Development*, *79*, 1942–1956. doi: 10.1111/j.1467-8624.2008.01235.x

Richters, J. E. (1997). The Hubble hypothesis and the developmentalist's dilemma. *Development and Psychopathology*, *9*, 193–229. doi: 10.1017/S0954579497002022

Roberts, B. W., & Del Vecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin*, *126*, 3–25. doi: 10.1037/0033-2909.126.1.3

Rogers, M. J., & Holmbeck, G. N. (1997). Effects of interparental aggression on children's adjustment: The moderating role of cognitive

appraisal and coping. *Journal of Family Psychology*, *11*, 125–130. doi: 10.1037/0893-3200.11.1.125

Roisman, G. I., Collins, W. A., Sroufe, A., & Egeland, B. (2005). Predictors of young adults' representations of and behavior in their current romantic relationship: Prospective tests of the prototype hypothesis. *Attachment & Human Development*, *7*, 105–121. doi: 10.1080/14616730500134928

Rothbart, M. K. (2011). *Becoming who we are: Temperament, personality, and development*. New York, NY: Guilford Press.

Rothbart, M. K., Ellis, L. K., Rueda, R., & Posner, M. I. (2003). Developing mechanisms of temperamental effortful control. *Journal of Personality*, *71*, 1113–1144. doi: 10.1111/1467-6494.7106009

Rothbart, M. K., & Posner, M. I. (2006). Temperament, attention, and developmental psychopathology. In D. Cicchetti (Ed.), *Developmental psychopathology* (2nd ed., Vol. *2*, pp. 465–501). New York, NY: Wiley.

Rothbaum, F., Rosen, K., Ujie, T., & Uchida, N. (2002). Family systems theory, attachment theory, and culture. *Family Processes*, *41*, 328–350. doi: 10.1111/j.1545-5300.2002.41305.x

Rubin, K. H., Bukowski, W. M., & Parker, J. G. (2006). Peer interactions, relationships, and groups. In: N. Eisenberg, W. Damon, & R. M. Lerner (Eds.), *Handbook of child psychology: Vol. .3 Social, emotional, and personality development* (6th ed., pp. 571–645). New York, NY: Wiley.

Rudolph, K. D., & Flynn, M. (2007). Childhood adversity and youth depression: Influence of gender and pubertal status. *Development and Psychopathology*, *19*, 497–521. doi: 10.1017/S0954579407070241

Rutter, M. (1970). Psychological development: Predictions from infancy. *Journal of Child Psychology and Psychiatry*, *11*, 49–62. doi: 10.1111/j.1469-7610.1970.tb01012.x

Sameroff, A. J., & MacKenzie, M. J. (2003). Research strategies for capturing transactional models of development: The limits of the possible. *Development and Psychopathology*, *15*, 613–640. doi: 10.1017/S0954579403000312

Sanders, M. R. (2008). Triple P-positive parenting program as a public health approach to strengthening parenting. *Journal of Family Psychology*, *22*, 506–517. doi: 10.1037/0893-3200.22.3.506

Schermerhorn, A. C., Cummings, E. M., & Davies, P. T. (2005). Children's perceived agency in the context of marital conflict: Relations with marital conflict over time. *Merrill-Palmer Quarterly*, *51*, 121–144. doi: 10.1353/mpq.2005.0012

Schermerhorn, A. C., Cummings, E. M., DeCarlo, C. A., & Davies, P. T. (2007). Children's influence in the marital relationship. *Journal of Family Psychology*, *21*, 259–269. doi: 10.1037/0893-3200.21.2.259

Schulz, M. S., & Kerig, P. K. (2012). Looking beyond adolescence: Translating basic research into clinical practice. In: P. K. Kerig, M. S. Schulz, & S. T. Hauser (Eds.), *Adolescence and beyond: Family processes and development* (pp. 304–314). New York, NY: Oxford University Press.

Schwarz, B., Stutz, M., & Ledermann, T. (2012). Perceived interparental conflict and early adolescents' friendships: The role of attachment security and emotion regulation. *Journal of Youth and Adolescence*, *41*, 1240–1252. doi: 10.1007/s10964-012-9769-4

Shaver P. R., Mikulincer M., & Shemesh-Iron M. (2010). A behavioural-systems perspective on prosocial behaviour. In M. Mikulincer & P. R. Shaver (Eds.), *Prosocial motives, emotions, and behavior: The better angels of our nature* (pp. 72–91). Washington, DC: American Psychological Association.

Shelton, K. H., & Harold, G. T. (2008). Interparental conflict, negative parenting, and children's adjustment: Bridging links between parents' depression and children's psychological distress. *Journal of Family Psychology*, *22*, 712–724. doi: 10.1037/a0013515

Shelton, K. H., Harold, G. T., Goeke-Morey, M. C., & Cummings, E. M. (2006). Children's coping with marital conflict: The role of conflict expression and gender. *Social Development*, *15*, 232–247. doi: 10.1046/j.1467-9507.2006.00338.x

Shields, A. M., Cicchetti, D., & Ryan, R. M. (1994). The development of emotional and behavioral self-regulation and social competence among maltreated school-age children. *Development and Psychopathology*, *6*, 57–57. doi: 10.1017/S0954579400005885

Shifflett, K., & Cummings, E. M. (1999). A program for educating parents about the effects of divorce and conflict on children: An initial evaluation. *Family Relations*, *48*, 79–89. doi: 10.2307/585685

Sih, A., Bell, A., & Johnson, J. C. (2004). Behavioral syndromes: An ecological and evolutionary overview. *Trends in Ecology & Evolution*, *19*, 372–378. doi: 10.1016/j.tree.2004.04.009

Slep, A. M. S., & O'Leary, S. G.(2005). Parent and partner violence in families with young children: Rates, patterns, and connections. *Journal of Consulting and Clinical Psychology*, *73*(3), 435–444. doi: 10.1037/0022-006X.73.3.435

Sloman, L., Atkinson, L., Milligan, K., & Liotti, G. (2002). Attachment, social rank, and affect regulation: Speculations on an ethological approach to family interaction. *Family Process*, *41*, 313–327. doi: 10.1111/j.1545-5300.2002.41304.x

Sloman, L., & Farvolden, P., Gilbert, P., & Price, J. (2006). The interactive functioning of anxiety and depression in agonistic encounters and reconiliation. *Journal of Affective Disorders*, *90*, 93–99. doi:10.1016/j.jad.2005.12.001

Snyder, J. R. (1998). Marital conflict and child adjustment: What about gender? *Developmental Review*, *18*, 390–420. doi: 10.1006/drev.1998.0469

Sroufe, L. A. (1985). Attachment classification from the perspective of infant-caregiver relationships and infant temperament. *Child Development*, *5*, 1–14.

Sroufe, L. A. (1997). Psychopathology as an outcome of development. *Development and Psychopathology*, *9*, 251–268. doi: 10.1017/S0954579497002046

Sroufe, L. A., Egeland, B., & Carlson, E. A. (1999). One social world: The integrated development of parent–child and peer relationships. In W. A. Collins & B. Laursen (Eds.), *Minnesota symposia on child psychology: Vol. 29. Relationships as developmental contexts* (pp. 241–261). Mahwah, NJ: Erlbaum.

Sroufe, L. A., Egeland, B., & Kreutzer, T. (1990). The face of early experience following developmental change: Longitudinal approaches to individual adaptation in childhood. *Child Development*, *61*, 1363–1373. doi: 10.1111/j.1467-8624.1990.tb02867.x

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, *55*, 17–29. doi: 10.2307/1129832

Stocker, C. M., & Youngblade, L. (1999). Marital conflict and parental hostility: Links with children's sibling and peer relationships. *Journal of Family Psychology*, *13*, 598–609. doi: 10.1037/0893-3200.13.4.598

Straus, M. A. (2001). Physical aggression in the family. In M Martinez (Ed.), *Prevention and control of aggression and the impact on its victims* (pp. 181–200). New York, NY: Springer.

Sturge-Apple, M. L., Davies, P. T., & Cummings, E. M. (2006a). Hostility and withdrawal in marital conflict: Effects on parental emotional unavailability and inconsistent discipline. *Journal of Family Psychology*, *20*, 227–238. doi: 10.1037/0893-3200.20.2.227

Sturge-Apple, M. L., Davies, P. T., & Cummings, E. M. (2006b). Impact of hostility and withdrawal in interparental conflict on parental emotional unavailability and children's adjustment difficulties. *Child Development*, *77*, 1623–1641. doi: 10.1111/j.1467-8624.2006.00963.x

Sturge-Apple, M. L., Davies, P. T., Winter, M. A., Cummings, E. M., & Schermerhorn, A. (2008). Interparental conflict and children's school

adjustment: The explanatory role of children's internal representations of interparental and parent–child relationships. *Developmental Psychology*, *44*, 1678–1690. doi: 10.1037/a0013857

Stutzman, S. V., Bean, R. A., Miller, R. B., Day, R. D., Feinauer, L. L., Porter, C. L., & Moore, A. (2011). Marital conflict and adolescent outcomes: A cross-ethnic group comparison of Latino and European American youth. *Children and Youth Services Review*, *33*, 663–668. doi: 10.1016/j.childyouth.2010.11.009

Swain, J. E., Konrath, S., Broan, S L., Finegood, E. D., Akce, L. B., Dayton, C. J., & Ho, S. S. (2012). Parenting and beyond: Common neurocircuits underlying parental and altruistic caregiving. *Parenting: Science and Practice*, *12*, 115–123. doi: 10.1080/15295192.2012.680409

Su, X., Simons, R. L., & Simons, L. G. (2011). Interparental aggression and antisocial behavior among African American youth: A simultaneous test of competing explanations. *Journal of Youth and Adolescence*, *40*, 1489–1502. doi: 10.1007/s10964-011-9673-3

Susman, E. J. (2006). Psychobiology of persistent antisocial behavior: Stress, early vulnerabilities, and the attenuation hypothesis. *Neuroscience and Biobehavioral Reviews*, *30*, 376–389. doi: 10.1016/j.neubiorev.2005.08.002

Taylor, C. T., Bomyea, J., & Amir, N. (2010). Attentional bias away from positive social information mediates the link between social anxiety and anxiety vulnerability to a social stressor. *Journal of Anxiety Disorders*, *24*, 403–408. doi: 10.1016/j.janxdis.2010.02.004

Taylor, Z. E., Larsen-Rife, D., Conger, R. D., & Widaman, K. F. (2012). Familism, interparental conflict, and parenting in Mexican-origin families: A cultural–contextual framework. *Journal of Marriage and Family*, *74*, 312–327. doi: 10.1111/j.1741-3737.2012.00958.x

Taylor, R. D., Seaton, E., & Dominguez, A. (2008). Kinship support, family relations, and psychological adjustment among low-income African American mothers and adolescents. *Journal of Research on Adolescence*, *18*, 1–22. doi: 10.1111/j.1532-7795.2008.00548.x

Taylor, K. A., Sullivan, T. N., & Kliewer, W. (2013). A longitudinal path analysis of peer victimization, threat appraisals to the self, and aggression, anxiety, and depression among urban African American adolescents. *Journal of Youth and Adolescence*, *42*, 178–189. doi: 10.1007/s10964-012-9821-4

Thompson, R. A. (2000). The legacy of early attachments. *Child Development*, *71*, 145–152. doi: 10.1111/1467-8624.00128

Trower, P., Gilbert, P., & Sherling, G. (1990). Social anxiety, evolution, and self-presentation. In H. Leitenberg (Ed.), *Handbook of social evaluation and anxiety*. New York, NY: Plenum Press.

Tschann, J. M., Flores, E., Pasch, L. A., & Marin, B. V. (1999). Assessing interparental conflict: Reports of parents and adolescents in European American and Mexican American families. *Journal of Marriage and the Family*, *61*, 269–283.

Turnbull, A. V., & Rivier, C. L. (1999). Regulation of the hypothalamic-pituitary-adrenal axis by cytokines: Actions and mechanisms of action. *Physiological Reviews*, *79*, 1–71.

Ursache, A., Blair, C., & Raver, C. C. (2012). The promotion of self-regulation as a means of enhancing school readiness and early achievement in children at risk for school failure. *Child Development Perspectives*, *6*, 122–128. doi: 10.1111/j.1750-8606.2011.00209.x

U.S. Census Bureau (2012). *2012 National population projections*. Retrieved June, 30, 2013, from http://www.census.gov/population/projections/data/national/2012.html

Van Der Meer, E., Beyer, R., Horn, J., Foth, M., Bornemann, B., Ries, J., . . . Wartenburger, I. (2010). Resource allocation and fluid intelligence: Insights from pupillometry. *Psychophysiology*, *47*, 158–169. doi: 10.1111/j.1469-8986.2009.00884.x

Volling, B. L., & Blandon, A. Y. (2005). Positive indicators of sibling relationship quality: The sibling inventory of behavior. In K. A. Moore & L. H. Lippman (Eds.), *What do children need to flourish? Conceptualizing and measuring indicators of positive development* (pp. 203–219). New York, NY: Springer Science & Business Media.

Wadsworth, M. E., & Compas, B. E. (2002). Coping with family conflict and economic strain: The adolescent perspective. *Journal of Research on Adolescence*, *12*, 243–274. doi: 10.1111/1532-7795.00033

Wasserstein, S. B., & La Greca, A. M. (1996). Can peer support buffer against behavioral consequences of parental discord? *Journal of Clinical Child Psychology*, *25*, 177–182. doi: 10.1207/s15374424jccp2502_6

Wilson, M. N. (1989). Child development in the context of the Black extended family. *American Psychologist*, *44*, 380–385. doi: 10.1037/0003-066X.44.2.380

Xin, Z., Chi, L., & Yu, G. (2009). The relationship between interparental conflict and adolescents' affective well-being: Mediation of cognitive appraisals and moderation of peer status. *International Journal of Behavioural Development*, *33*, 421–429. doi: 10.1177/0165025409338442

Zeanah, C. H., Boris, N. W., & Larrieu, J. A. (1997). Infant development and developmental risk: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 165–178. doi: 10.1097/00004583-199702000-00007

Zimet, D. M., & Jacob, T. (2001). Influences of marital conflict on child adjustment: Review of theory and research. *Clinical Child and Family Psychology Review*, *4*, 319–335. doi: 10.1023/a:1013595304718

Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model*. Washington, DC: American Psychological Association.

CHAPTER 7

Emotion and the Development of Psychopathology

PAMELA M. COLE

INTRODUCTION 265
THE NATURE OF EMOTION 266
EMOTION AND PSYCHOPATHOLOGY 268
EMOTIONAL COMPETENCE 269
DEVELOPMENT OF EMOTIONAL COMPETENCE 270
The Prenatal Period: Typical Development 270
The Development of the Components of Emotional Competence 273
Emotion Understanding 282
Typical Development of Emotion Regulation 289
CONCLUSION AND FUTURE DIRECTIONS 299
Personal Relevance 300
Dynamics 301
Context 302
Translational Implications 303
REFERENCES 305

INTRODUCTION

The capacity to be emotional is the heart of human development. This capacity can be thought of as a rapid radar-and-response system that has evolved to enable continuous appraisal of the fit between a person's circumstances and goals for well-being and readiness to act in particular ways in relation to the circumstances to achieve, preserve or regain well-being. The capacity to be emotional has its evolutionary origins in a primitive function, common to even simple organisms, to detect and respond to conditions to avoid harm and to reproduce (Darwin, 1872; LeDoux, 2012). The capacity evolved in humans to support the development of social groups, developing a communicative function that organizes social interaction. Its essential importance to being human is seen throughout the centuries; philosophies, religions, and science have contemplated and debated the nature of *emotion*, including grappling with the seeming paradox of why an arguably adaptive capacity is so often a salient feature of *maladaptive* psychological functioning.

This chapter addresses the role of the capacity to be emotional in the development of emotional competence and psychopathology. It uses two conceptual models to discuss the role of emotion in healthy development, the vulnerabilities that emerge along the path to competent emotional functioning, and the challenges encountered that compromise the development of emotional competence and contribute to the development of psychopathology. The first conceptual model is the developmental psychopathology framework (Cicchetti & Cohen, 1995). The principles of this framework include the importance of understanding atypical development in the context of typical development (and vice versa). They include the principle that challenges encountered at one point in life may strengthen psychological functioning by fostering and exercising newly emerging skills but that they may also pose risk for healthy development. The framework appreciates the complexity of ontogenetic pathways; early experience that involves significant risk provides a foundation from which development can cascade toward a particular pathological outcome, and yet developing systems begin with malleability that permit resilience despite risk conditions. The framework appreciates that there are multiple pathways to a single outcome (e.g., a symptom of depression or conduct disorder) as well as commonalities in pathways associated with different outcomes (e.g., a history of child sexual abuse is associated with any number of psychiatric disorders as well as healthy adjustment). In focusing on the capacity to be emotional from a developmental psychopathology framework, this chapter examines how emotional competence develops and how, at different points of development, emotional competence is threatened and can be diverted toward psychopathology.

The second framework that organizes the chapter is the functional perspective on *emotional development* (Barrett & Campos, 1987; Witherington, Campos, & Hertenstein, 2001) and its application to the concept of emotional

competence (Denham et al., 2011; Saarni, 1998). This perspective emphasizes the *relational, contextual, dynamic* nature of emotional functioning. Emotional competence, a desired outcome of emotional development, entails successfully navigating a complex interpersonal and intrapersonal world in a flexible, socioculturally appropriate, and effective manner. Emotional dysfunction, a prominent feature of psychopathology, can be conceptualized as compromised development of emotional competence.

To begin, the nature of the capacity to be emotional from the functional perspective is elaborated. Next the chapter summarizes what is known about the development of emotional competence in childhood and adolescence, organized around three interrelated domains of emotional competence—emotional expressiveness, emotion understanding, and emotion regulation. Because of the emphasis on a functionalist view of emotion, priority is given to evidence that furthers our ability to understand emotion in context as a relational, dynamic, process. Finally, the chapter considers how the development of emotional competencies involves skills that not only enable flexible, appropriate, effective emotional functioning but also occasion new ways that an individual is vulnerable to emotional dysfunction and the risks posed for the development of psychopathology.

THE NATURE OF EMOTION

Scholarly inquiry about healthy and unhealthy aspects of the capacity to be emotional has echoed through the centuries in major world religions, in classic philosophies, in many disciplines of modern science, in contemporary emotion research and theory, including in subfields focused on typical and atypical human development. In all of these efforts, scholars have recognized that emotion is essential for survival, for being fully human, for being a member of a social world. Yet, at the same time, emotion is consistently portrayed as irrational, as interfering with human functioning, or as needing control. In contemporary psychological science, the most influential theories all addressed emotion—Freud, of course, in his many volumes, James notably—but it was also addressed by scholars who did not emphasize emotion, notably Piaget (*Intelligence and Affectivity*, 1981) and even Skinner (*Walden Two*, 1948). Yet the scientific investigation of emotion has not always been in vogue. The ascendancy of behaviorism in the early twentieth century with its sole focus on observable behavior and environmental contingencies shifted attention away from cognition and emotion. This was followed by the post–World War II cognitive revolution and its emphasis on information processing and computer modeling. These periods muted scientific progress in the study of emotion for many years. By the 1980s, however, the study of emotion suddenly surged. Behaviorism and cognitive science had not provided complete accounts of human functioning. Since 2000, there has been tremendous growth in the study of emotion, most recently spurred by major advances in our ability to study genetics and neuroscience. What has been learned has deepened interest in emotion but also furthered debates about its fundamental nature, the degree to which emotions are biologically determined or psychologically specified, and about the conceptual boundaries of the concept of emotion and related concepts such as emotion regulation. The functional perspective, which organizes this chapter, emphasizes the continuous, relational, dynamic, and contextual nature of what we refer to as emotion. It is selected as an overarching framework because of its potential to enlighten our understanding of typical and atypical development, guiding a view of atypical emotional development as a problem of the relation between person and environment. This perspective has a lineage in theoretical positions asserting that the capacity to be emotional evolved as critical for adaptation to the environment (e.g., Arnold, 1960; Izard, 1977; Lazarus, 1999; Plutchik, 1980). Its critical value was that it enables very rapid appraisal of the significance of situations for well-being and very rapid organismic preparation to act to achieve, maintain, or regain well-being.

The functionalist view of emotion has roots in the germinal work of Magda Arnold (1960), who took a distinctly neurobiological approach to understanding emotion at a time when behaviorism was at its peak and the cognitive revolution was underway. In her effort to produce a comprehensive emotion theory, Arnold included discussion of the evolution of emotion, the role of learning in emotional functioning, and the contributions of emotion to personality development, linking her work to leading models of the time. Most importantly, Arnold shifted the scientific focus from subjective feelings and unconscious anxiety, which were the prevalent perspectives in psychoanalytic scholarship, to neurophysiological patterns that supported mental processing of environmental information and organized action. Arnold defined emotion in terms of neurophysiological processes that enable (1) implicit (not necessarily conscious) judgments about the environment that motivate action, or appraisal, and (2) organismic changes that prepare the individual to move toward or away from the environment or action readiness. Nico Frijda (1986) extended this perspective, further specifying

appraisal/action readiness patterns that are uniquely associated with different emotions—joy, anger, fear, sadness, shame, and others. For example, fear and sadness both involve physiological changes that ready the individual to withdraw from rather than approach the environment. But fear and sadness nonetheless differ in important ways. Fear is defined by the appraisal that circumstances endanger well-being and preparedness to act in ways that minimize or escape threat; these require the mobilization of resources (energy) for action. Sadness, by contrast, is defined by the appraisal that a goal for well-being cannot be attained and neurophysiological readiness to relinquish effort, which reduces the demand for energy to act and conserves energy.

Arnold's work is a precursor of Lazarus's (1991) appraisal theory of emotion. Lazarus focused on the mental processes: appraisal as determining whether a situation challenged well-being (stress) and assessing possible actions (coping). The emphasis on appraisal tended to focus on explicit, reflective processes more than on the intuitive, automatic processes that Arnold defined. Lazarus' model does not dwell on neurophysiological changes, such as readiness to act, that were part of Arnold's definition of emotion. For a developmental perspective on the role of emotion in psychopathology, appraisal theory presents two limitations. First, the emphasis on the conscious content of thought turns emotions into mental objects—discrete, periodic mental states that result from interpretive processes. Emotion becomes a result of appraisal, rather than appraisal (and action readiness) being active, ongoing processes that define emotion. The functional perspective intends to dispel the view of emotions as discrete objects or events. This definitional shift is challenging to grasp, in part because the lexicon we use to communicate about emotion relies on nouns, which tend to reify emotion. In this chapter, we emphasize the capacity *to be emotional*, because the functional perspective emphasizes conceptualizing emotion as dynamic relating to the world. The second limitation of a strict appraisal theory is that most of its associated research has not been informed by a developmental perspective. As will be discussed, the behavior of infants and very young *children* offers unique views on the nature and role of the capacity to be emotional. It is from infant research that the functional perspective on emotional development emerged (Barrett & Campos, 1987).

As we can see, the functional perspective challenges traditional conceptualizations of emotion. Its main point of divergence is its assertion that emotion is *relational* in nature; that is, it is a process defined by the ongoing relation of the perceiver to the environment. This perspective recognizes that humans have the mental capacity to reflect on this relation process, creating objects of conscious thought. But the functional perspective on emotional development has its origins in infant research and is influenced by the fact that infants, who cannot consciously reflect on how they are relating to the environment, nonetheless have the capacity to be emotional. In the first year of life, infants communicate emotionally to signal their needs, detect and interpret communicative signals of others' emotions, and use this information to guide their own behavior. Thus, the functionalist perspective conceptualizes emotion as situated in the transactional space between individual needs or goals for well-being and the environment. Emotions are not psychological objects residing within a person but are psychological processes that enable individuals to relate to the environment in ways that serve attaining and preserving well-being (Barrett & Campos, 1987; Campos, Dahl, & He, 2010).

Arnold's (1960) neurobiological view was prescient, forecasting contemporary affective neuroscience, which also conceptualizes the neurophysiology of emotion in terms of processes by which organisms perceive and orient to changing environments. Conscious perceptions of emotions, in the self or in others, create the impression of static events because these conscious percepts are *categorical judgments*. The terms we use for the judgments objectify glimpses of a psychological process that depends on a self-organizing nervous system and its connections to and from peripheral sensory and skeletal-muscular systems, systems that enable us to sense, perceive, process, and act on information. But contemporary neuroscience also shifts from an historic view of emotions as discrete and occasional events and to emotional processing as dynamic and relational (Davidson, 2000). At the physical level, neuronal activity is ongoing and reorganizes as circumstances change. At times, changes in brain circuitry specialized for discerning circumstances of significance for well-being, probably in association with associative networks help to construct meaning and classify experience. These processes occasionally achieve a magnitude of such adaptive importance that they reach conscious awareness (feelings). Similarly, when we observe changes in others' nonverbal signals in the context of situational cues, we are likely to make categorical judgments about their emotions. Whether observing self or others, we classify and conceptualize processes as discrete events. Because most of our knowledge has been founded on analysis of subjective feelings and observations of others, we have conceptualized emotions as categories, discrete

events. However, like many processes that are fundamental to human functioning (seeing, digesting, breathing, or walking), appraising and action readying operate largely outside of conscious mentation (Bargh & Morsella, 2008; LeDoux, 1996; Ohman & Soares, 1994; Panskepp, 1998; Pessoa, 2005). In addition to emphasizing the dynamic and relational nature of emotion, the functional perspective also emphasizes the highly context-sensitive nature of emotion. An example is a mother who frowns and says she is angry when viewing a video of her distressed infant; when she explains that she's angry with herself for letting a research assistant frustrate her infant, her anger is different than if she revealed that she was angry at the infant for getting distressed (Killeen, 2010). In sum, the capacity to be emotional is best conceptualized as continuous appraising of changing circumstances in terms of their significance for well-being and associated changes in readiness to act to maintain or regain states of well-being.

The dynamic, contextualized, relational nature of emotion poses a challenge to scientific endeavors, requiring inferences based on changes in sets of tangible, measurable phenomena in specific situational contexts. Emotional changes are inferred on the basis of patterns of physiology, self-reported experience, or observed actions, including nonverbal and verbal behavior, in the circumstances being processed (Cole, Martin, & Dennis, 2004). There are promising new directions in the study of emotion that will base inferences on examination of the dynamic, temporal flow of tangible indices of latent constructs, evaluated in context (e.g., Butner, Diamond, & Hicks, 2007; Chow, Haltigan, & Messinger, 2010; Steele & Ferrer, 2011). A functional perspective on emotion also requires methods that carefully assess environmental conditions and predictions about end-states of goals and how goals can be achieved (Campos et al., 2010; Campos, Frankel, & Camras, 2004). Finally, it requires that we study emotion in contexts that are personally meaningful to participants and that we use methods that help us understand those personal meanings (Campos et al., 2010).

EMOTION AND PSYCHOPATHOLOGY

Despite its clear adaptive advantages, our capacity to be emotional often gets us into trouble. When emotion compromises the development of competent functioning, we refer to emotional dysfunction. Dysfunction connotes that the emotional processes are functioning in the interest of individual goals for well-being but at a cost to healthy development. Emotional dysfunction has been highlighted as a major correlate and potential precursor of many adult and childhood psychological disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Cicchetti, Ackerman, & Izard, 1995; Cole, Michel, & Teti, 1994; Gross & Muñoz, 1995; Keenan, 2000; Kring & Bachorowski, 1999; Southam-Gerow & Kendall, 2002; Vine & Aldao, 2014). In addition, its broad relevance as a general feature of psychopathology, emotional dysfunction has been conceptualized as a central feature of many specific disorders, including schizophrenia spectrum disorders (Johnson & Fulford, 2009; Kring & Moran, 2008); eating disorders (Greeno, Wing, & Shiffman, 2000; Haynos & Fruzzetti, 2011); mood disorders (Joormann & Gotlib, 2010; Luby, 2009; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008); anxiety disorders (Behar, DiMarco, Hekler, Mohlman, & Staples, 2009; Mennin & Fresco, 2010); and various impulse control and aggression-related disorders, including conduct disorder (Cole, Hall, & Radzioch, 2009; Frick, Ray, Thornton, & Kahn, 2013) and personality disorders (Cyders & Smith, 2008; Gratz & Gunderson, 2006; Linehan, 1993; Tragesser, Solhan, Schwartz-Mette, & Trull, 2007). This range of work has suggested that emotional difficulties are a core feature of all psychological disturbances (Bradley, 2000).

If being emotional is an essential, ubiquitous process motivating action, then it logically follows that emotional processes are intricately involved in both maladaptive and competent functioning. At first flush, a large and wide-ranging body of research has suggested that the differences in emotional functioning between individuals with and without diagnosed disorders involve differences in three related areas: greater intensity, duration, or frequency of negative emotion; biases in attending to or classifying others' emotions; and a lack of certain emotion regulatory strategies. Added to these common themes are aspects of emotional dysfunction that arise from clinical observations, including emotional numbing and blunted emotional expression, limited self-awareness of emotions (poor emotional clarity), insensitivity or hypersensitivity to emotion, and the ineffective strategies or ineffective use of typically effective strategies for coping with negative emotions or problem situations. In sum, the research literature suggests a range of ways in which the emotional functioning of individuals with psychiatric disorders deviates from typical functioning. This literature often compares one group of individuals with a *DSM*-defined diagnostic condition with healthy controls, but this research has not yielded evidence that any single form of emotional dysfunction is associated with any single disorder. Rather, emotional dysfunction is widely implicated in psychopathology.

The realization that emotional dysfunction is pervasive in psychopathology is consistent with the push for a transdiagnostic approach to clinical problem identification and intervention (Cuthbert & Insel, 2013). The integration of a functional perspective on emotional development with a developmental psychopathology framework is compatible with the contemporary trend toward transdiagnostic approaches to psychopathology and intervention (Harvey, Watkins, Mansell, & Shafran, 2004; Nolen-Hoeksema & Watkins, 2011). The transdiagnostic approach contends that psychological processes that have gone awry can be shared across different disorders and that targeted intervention need not depend on diagnostic classification. As one example from the adult psychopathology literature, blunted affect in schizophrenia and anhedonia in depression potentially share a common core. Adults with schizophrenia process positive emotional stimuli similarly to healthy controls in some respects, but they differ in that they do not anticipate or sustain attention to positively valenced stimuli (Gard, Kring, Gard, Horan, & Green, 2007; Horan, Wynn, Kring, Simons, & Green, 2010). Impairments in anticipating pleasure and maintaining positive emotions are also associated with major depression in both adults (e.g., Sherdell, Waugh, & Gotlib, 2012) and have been investigated as a risk factor for depression in young children (e.g., Durbin, Klein, Hayden, Buckley, & Moerk, 2005; Olino et al., 2012).

From the functional perspective, then, there is a need to understand the developmental processes that foster the ability to anticipate positive outcomes, even in the face of blocked or lost goals, and how this capacity may be compromised. In one of our studies, a typically developing 36-month-old cannot get a locked box open to retrieve a desired gift. As she continues to try she becomes increasingly frustrated and begins to mutter, "I can't do it. I can't do it." But then she shifts from the appraisal/action readiness pattern of persisting with effort to overcome the blocked goal to a pattern of relinquishing the goal. That is, she flops back in her chair, discouraged, and her attention wanders. But in a moment she returns her attention to the locked box and says with a lilting voice, "Maybe this time I can do it. I know I can!" Her capacity to anticipate positive outcomes appears to modulate a very reasonable appraisal that she may have encountered an unsolvable problem. Her negative emotion, of which she had a bit, is not the best way to understand her. Rather it is the changing, unfolding emotional relations she has with the locked box that distinguish her response from the children who give up or tantrum. A developmental perspective on the functional nature of emotion, especially work that places emotional dynamics in situational context, has significant promise for identifying features of emotional competence and early detection of risk for atypical emotional development. Resulting findings can inform interventions that remediate atypical conditions and prevent psychological disorders. The functional perspective on emotional development allows us to conceptualize the nature of emotional competence, how it develops, and how it may go awry. To understand atypical emotional development, we must first understand emotional competence.

EMOTIONAL COMPETENCE

If psychopathology involves maladaptive functioning in a person's relation to the environment, then psychopathology must always involve emotion processing and often involves emotional dysfunction. Therefore we can use the construct of emotional competence as a hallmark of healthy emotional functioning against which to contrast emotional dysfunction. Emotional competence has been identified as a critical component of healthy functioning throughout childhood and adolescence in terms of success in both academic and interpersonal functioning (Cicchetti, Ackerman, & Izard, 1995; Denham, 2006; Raver, 2003; Roeser, Eccles, & Sameroff, 1998). The construct is similar to one used in clinical and personality research with adults, emotional intelligence, which is also associated with well-being and success in relationships, education, and work (Brackett, Rivers, & Salovey, 2011; Nelis et al., 2011). From a developmental psychopathology viewpoint, then, it is important to understand and study the ontogeny of emotional competence and how in its course dysfunction arises such that functioning is impaired to the degree that it is regarded as psychopathology. The functionalist perspective on emotional development has informed conceptualizations of child emotional competence (Denham, 2006; Halberstadt, Denham, & Dunsmore, 2001; Saarni, 1999). Consistent with the central premises of the functionalist perspective, these models of emotional competence assert that emotional functioning cannot be conceptualized apart from context, particularly social meaning. In general, emotional competence entails being responsive to one's circumstances, including access to the full range of emotions, the ability to engage emotionally flexibly such that one is effective in accomplishing goals for well-being while behaving appropriately as defined by situational and sociocultural realities (Saarni, 1999). These models subdivide this complex construct into three domains of competence: (1) emotion expressiveness; (2) emotion awareness and understanding; and (3) emotion regulation.

The capacity to express emotion—both nonverbally and verbally—is the earliest developing emotional competency. It is critical to survival because survival depends on emotional communication and the formation and maintenance of early relationships. It includes the use of emotional expression to initiate, maintain, and coordinate social interaction. Building on this essential communicative function, emotional competence comes to include the ability to read and understand the emotions of others and to understand one's own emotional experiences and associated actions. Accurately interpreting the emotion signals of others in terms of their needs for well-being and coordinating their needs with one's own well-being is an essential aspect of emotional competence. As individuals develop across the life span, their accumulating experience and growing emotion knowledge enables them to make increasingly sophisticated judgments and to find solutions to complex intra- or interpersonal problems. Finally, although the capacity to be emotional equips us to respond instantly, intensely, and persistently in our own best interests, competence reflects learning to balance this capacity with the constraints posed by situations and relationships. Thus the third main component of emotional competence is emotion regulation. This last component involves the ability to anticipate, modulate, and reorient emotional responses for them to serve both self-focused and social goals. These three components, which can be distinguished conceptually and are often studied in isolation, are not mutually exclusive; in fact they are deeply intertwined. Nonetheless, they develop on different time courses. In the next sections, we review knowledge regarding the development of the capacity to be emotional and the constituents of emotional competence, beginning with the origins of the capacity to be emotional. We prioritize evidence that examines emotional functioning in situational context and that considers longitudinal relationships that advance our ability to hypothesize the role of emotion in developmental trajectories toward emotional competence and emotional dysfunction. In the discussion, we consider the ways that the emergence of competence in given domains equips the child for productive, effective behavior but also introduces vulnerabilities that set the stage for the development of emotional dysfunction and psychopathology.

DEVELOPMENT OF EMOTIONAL COMPETENCE

The Prenatal Period: Typical Development

For a long time, it was assumed that the behaviors and abilities observed in neonates were innate and inherited, attributable to genetic factors. This view has been altered by remarkable advances in our understanding of genetics and gene–environment interplay. In addition, changing views of the role of genetics in human functioning are shaped by growing knowledge about the development of physiological systems and behavior during the prenatal period. The basic capacity to be emotional depends to a substantial degree on fetal development.

Advances in the field of genetics have revealed the exquisite complexity of the human genome, the limits of its contributions to psychological development, and its intricate dependency on environmental input. It is no longer the case that we can assume that the genes inherited from each parent are simple replicates that explain complex developmental outcomes such as personality traits or psychiatric disorders. Genetic variations influence the formation of physiological systems, by which psychological processes emerge, and contribute to the capacity of the developing organism to sense, perceive, process, and respond to environmental conditions. Moreover, current knowledge indicates that there are over 20,000 genes in humans, thousands more noncoding RNA genes that regulate DNA genes, (perhaps) 80–100 gene mutations between each generation (some of which affect the individual and others of which affect future generations), epigenetic processes that determine whether a gene is expressed, and, complex multigene interactions that most likely underlie the developmental outcomes we are so eager to explain, such as mental processes, behavioral traits, and psychological disorders. A thorough discussion of the field of genetics is beyond the scope of this chapter, but several general points can be derived from current research on genetics to inform the role of emotion in the development of psychopathology:

1. Characteristics of each individual human genome contribute to the physiological structures and functions that enable the capacity to be emotional and to individual differences in this capacity.
2. Genetic variations influence outcomes such as emotional competence or psychopathology through their influences on components along the pathway from genotype to psychopathology, including the development of physiological structures and processes such as neural structures, neural transmission, and neural networks (Bevilacqua & Goldman, 2011; Gottesman & Gould, 2003). These neurophysiological elements are steps in pathways from the genome to emotional functioning, pathways that involve many steps.

3. Despite considerable research on candidate genes, it is generally assumed that the influence of any single gene depends on both epigenetic factors (Gottlieb, 2007; Harper, 2005; Meaney, 2010) and gene-by-gene interactions (Hariri & Holmes, 2006; Stranger, Stahl, & Raj, 2011). Thus, for a gene like the serotonin transporter gene (5-HTT) to influence any aspect of emotional functioning depends on the expression of other genes and environmental conditions.
4. Genetic influences on neurophysiological processes are rarely inherently pathogenic (Cicchetti & Rogosch, 2012; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011; Homberg & Lesch, 2011). A phenotype such as sensitivity to negative emotion in human faces may be an emotional competency, a risk factor, or both. For example, the well-being of marital partners depends on each partner's sensitivity to the other's emotions; heightened perceptual sensitivity to negative emotions in faces, often regarded as a risk factor for psychiatric conditions such as anxiety disorder or borderline personality disorder, is an asset in the partner of a person suffering Parkinson's disease, a condition that impairs the clarity of emotion expression (Petrican, Moscovitch, & Grady, 2014).
5. Finally, genetics research underscores the critical importance of the environment on which much genetic influence depends. Consider again heightened perceptual sensitivity to negative emotion. In the context of emotionally supportive families, children who are predisposed to react strongly to others' negative emotion may be fussy and unhappy infants but some may become highly sensitive, productive, satisfied adults, such as various types of artists who skillfully depict human emotion in the face or professionals like therapists or other care providers who are highly attuned to the unspoken needs of their clients. If, however, the environment surrounding reactive children is emotionally adverse (e.g., unmitigated harsh parenting and the absence of adult supports for coping with adversity), their sensitivity to others' emotions may contribute to self-doubt, compromised self-esteem, and persistent interpersonal difficulties.

In sum, nature and nurture "mutually constitute each other" (Sameroff, 2010, p. 9). The neurophysiological undergirding of appraisal, action readiness, and emotion-relevant behavioral processes depend on genetic contributions but these account for only part of the variance in any given form of psychopathology, even among the more heritable conditions (Duncan, Pollastri, & Smoller, 2014; Bevilacqua & Goldman, 2011). More sophisticated research incorporating the best genetics methods and detailed understanding of the environment, including the prenatal environment, will contribute significantly to our understanding of the development of the capacity to be emotional. This will advance our knowledge beyond the simplistic view of a candidate gene interacting with an adverse environment to produce a psychiatric disorder, and help us study and document the pathway of endophenotypes that link genetic influences to outcomes of relevance for emotional functioning and dysfunction. Thus, the basic tools that undergird the capacity to be emotional depend on genetic processes, and their expression depends on environmental conditions; to survive, it is critical for these aspects of development to occur before the infant is born. Thus, another important step in the emotional pathway to competence includes the influences of the prenatal environment.

In the prenatal period, the nervous system develops rapidly—neurons develop, proliferate, migrate, and begin to form connections with other neurons, allowing sensory, cardiovascular, and cardiopulmonary systems to begin to organize (Anderson & Thomason, 2013; Glynn & Sandman, 2011). Fetal processing of the environment begins, and by the third trimester activity that presages infant behavior is detectable. By the twenty-fifth gestational week, the fetus is responsive to stimuli, such as stimulus-induced heart rate changes and motor activity including facial activity (Gingras, Mitchell, & Grattan, 2005; Kisilevsky, Muir, & Low, 1992). By the thirty-sixth week, the fetus responds to loud sounds, including the mother's voice, with changes in fetal heart rate, head turning, and eye movement (DeCasper, Lecanuet, Busnel, Granier-Deferre, & Maugeais, 1994), which may account for neonates' apparent recognition of their mothers' voices (DeCasper & Fifer, 1980). Notably, a single case study indicated that fetal temporal region activity (assessed by fetal function magnetic resonance imaging [fMRI]) is specific to the changes in the mother's voice (Jardri et al., 2012). In sum, the development of physiological systems that enable psychological functions occurs in utero and may depend on qualities of the fetal environment. Stimuli that affect the structure or function of fetal neurophysiological systems include acoustic signals, nutrients, hormones, and movement (Glover, 2011; Mayes, 1999; Raznahan, Greenstein, Lee, Clasen, & Giedd, 2012). Development in the prenatal period appears to involve neurophysiological changes that ready the infant for entry into the caregiver–infant relationship, in which emotional communication is critical.

Risk for Emotional Dysfunction Associated with Prenatal Period

Appreciation that the prenatal environment influences fetal development has heightened awareness of potential effects of prenatal risk exposure. Much of this research has had to depend on animal models that permit experimental manipulation of hypothesized mechanisms (for reviews see Boksa, 2010, on prenatal infection; Field, Diego, & Hernandez-Reif, 2006, on maternal prenatal depression; Gräff, Kim, Dobbin, & Tsai, 2011, on multiple risk factors; Meyer & Feldon, 2010 on conditions posing risk for schizophrenia). Due to the complexity of human neurophysiological systems, it is not yet possible to extrapolate findings from animal model research to the emotional development of the human child, and this fact has motivated studies of the human prenatal environment. When the effects of the prenatal environment are studied in humans, the main research design has been to contrast infants of mothers with an identified risk factor during the prenatal period—for example, substance use (antidepressants, smoking, cocaine), emotional problems (stress, depression, anxiety), or malnutrition or disease (anemia, infection)—with infants of mothers without such conditions. This work suggests that certain biochemical and environmental conditions can stress, foster, and compromise fetal development.

Research on stress and the human prenatal environment initially focused on fetal physical development, such as gestational age and birth weight. There is, however, considerable interest in the effects of maternal prenatal stress on the infant or child's later psychological functioning, including emotional functioning (Glover, 2011; O'Donnell, O'Connor, & Glover, 2009). Maternal stress, defined by maternal anxiety about the pregnancy or by clinical symptoms of depression and anxiety, affects blood flow to the placenta and distributes glucocorticoids through the placenta, a mechanism by which maternal stress may influence fetal brain development (often referred to as fetal programming). Much of this work suggests that maternal prenatal stress has deleterious effects on infant emotional functioning. Specifically, effects have been reported for greater infant fear and anger reactivity (Davis et al., 2004; DiPietro, Ghera, & Costigan, 2008), cry reactivity (Werner et al., 2007), and stress reactivity (Glover, 2011) and poorer attention control, which could affect regulation of emotional reactions (Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2002).

These findings, however, are often limited by study design. For example, they often focus on only one of various types of stresses a pregnant woman can face (e.g., pregnancy-related anxiety, daily hassles, marital strife, symptoms of psychopathology, physical illness) or on only one of several potential infant outcomes that may be affected. In studies that measured multiple infant outcomes, effects emerge for some but not all outcomes (e.g., attention regulation but not emotion regulation; Glover, 2011). Furthermore, because prenatal stress is typically assessed by maternal self-report, studies are often limited in their ability to parse out the degree to which infant or child outcomes are influenced by maternal trait stability (an anxious pregnant woman may also be a chronically anxious mother), heritable stress responding (shared genetic variance), and prenatal environmental conditions (Federenko et al., 2006; Kendler & Prescott, 2006). In addition, most studies overlook the fact that the fetus *contributes* to the prenatal environment. The fetus generates the placenta, a source of corticotrophin-releasing hormone (CRH), and the fetus also begins to engage in behavior (e.g., motor activity; DiPietro, 2012). A study that attempted to circumvent some of these constraints compared infants who were conceived through in vitro fertilization by related or unrelated parents (Rice et al., 2010). Relying on maternal retrospective reports of stress, the study concluded that lower infant birth weight, earlier gestational age, and childhood misconduct were related to late pregnancy prenatal stress for both related and unrelated parents, suggesting influences of the fetal environment rather than genetic contributions. In contrast, it reported that child attention-deficit/hyperactivity disorder (ADHD) symptoms appeared only for offspring of related parentage and these were thus attributed to genetic factors. Finally, a link between prenatal stress and child anxiety symptoms was mediated by concurrent maternal anxiety and depressive symptoms.

Clearly, critically needed research on the prenatal environment is in its early stages. As limitations of study designs are met with solutions, this line of research is likely to help us understand the conditions that foster or compromise the neurophysiological components that support the capacity to be emotional. It is unknown what will be learned but it is reasonable to anticipate that not all effects of prenatal stress may be deleterious. Pregnancy-specific psychological stress, rather than symptoms of clinical anxiety, was associated with increased fetal motor activity, accelerated neurological maturation as indexed by neonate auditory ERP, and neonate irritability (DiPietro et al., 2010) as well as by higher scores on the Bayley Scales of Mental Development at age 2 years (DiPietro, Novak, Costigan, Atella, & Reusing, 2006). In life, stress exposure creates demands to

adapt and adaptations can facilitate development as well as create risk.

In sum, it is likely that risks associated with prenatal development, both in terms of genetic unfolding and environmental factors, can compromise the capacity to be emotional and the most fundamental building blocks of emotional competence. That is, the consequences of fetal programming of the nervous system and fetal adaptations to the prenatal environment must be considered in the pathway to emotional functioning. Relatively mild stress may facilitate aspects of fetal neurodevelopment in ways that enhance later emotional functioning (e.g., through fostering attention or emotional skills), whereas exposure to more severe stress (e.g., maternal clinical anxiety) may compromise the most basic neurophysiological processes on which the development of emotional competence is founded.

The Development of the Components of Emotional Competence

Emotional Expressiveness, Understanding, and Regulation

The prenatal period equips the infant with the basic neurophysiological systems that enable information processing, including experience–expectant neurophysiological networks that will further develop as the infant interacts with its new environment (Greenough & Black, 1992). The neonate enters an emotionally rich environment, including the critical interpersonal environment. The interactions of infants and caregivers depend on the capacity to be emotional and these interactions influence the development of component skills that will lead to the development of an emotional competent person.

Emotional competence can be analyzed in terms of three main domains of emotional functioning: emotional expressiveness, emotion understanding, and emotion regulation (Denham, 2006; Halberstadt et al., 2001; Saarni, 1999). Each of these domains is discussed separately, with an emphasis on the individual child's development. However, the larger view from the functional perspective is that each domain of emotional competence is inextricably related to the other. Moreover, each domain must be understood in terms of actual situations that are personally relevant to the child, particularly interpersonal contexts. Furthermore, the emotionally competent person draws from each domain to engage the world in an effective way to achieve personal goals for well-being in the context of social interactions, relationships, and rules of conduct. When possible, the review emphasizes studies in which emotional competence has been studied in real or realistic circumstances. Next, each domain of emotional competence and the evidence for its developmental progression described. Each is introduced in terms of how it supports healthy psychological functioning and then considered in terms of how it contributes to vulnerabilities that lead to the development of emotionally dysfunctional appraisal/action readiness patterns and increase the chances of developing psychopathology.

Typical Development of Emotional Expressiveness

Emotional competence involving emotional expressiveness is defined by the ability to respond to circumstances with a full range of emotions and to communicate emotion effectively (in terms of achieving goals, which includes both personal and social goals), appropriately (in terms of sociocultural standards), and flexibly (in terms of situational appropriateness) (Cole, Hall, & Hajal, 2013; Denham, 2006; Halberstadt et al., 2001; Saarni, 1999). The emotionally competent child experiences and conveys emotion in a spontaneous and authentic manner. That is, the child is emotionally responsive to the variety of situations and interactions—familiar and novel—and conveys that emotional responsiveness in age appropriate, situationally relevant, socially acceptable ways. To do so requires the ability to modulate emotional expressiveness in ways that accomplish personal goals but also conform to situational demands and sociocultural values. The importance of emotional expressiveness in early childhood is revealed in detailed observations of preschool-age children at school (Fabes et al., 1999). In this natural setting, children's patterns of emotional expressiveness, both positive and negative, as they interact with peers and teachers are associated with teacher ratings of child social competence, even at such a young age.

The capacity to express emotion begins to develop in utero. In the first year of life, typically developing infants express emotions through nonverbal modes of communication, their facial expressions, the prosody of their vocalizations, and often their whole bodies. Within the first year, children begin to gesture to communicate personal goals (e.g., shaking the head to indicate no) and in the second year, soon after children begin to use words, they begin to refer to simple, basic emotions. By the third birthday, typically developing children show signs that they can engage in spontaneous expressive control. The ability to engage in emotional expressiveness is critical to the development of the other domains of emotional competence—emotion understanding and emotion regulation—and each of these contributes to emotional expressiveness over the course of childhood and adolescence.

Nonverbal Emotional Expressiveness

The neuromuscular systems that will enable emotionally meaningful communicative behavior with caregivers develop in utero for most infants. The motor branch of the facial nerve develops as early as the third week of gestation, and by 12 weeks synapses have begun to form, such that tongue and lip movement are observed (Molina, Reyes, Joshi, Barrios, & Hernandez, 2010). To engage in behavior like smiling, for example, the neural signal takes a somewhat arduous path from the brain through the temporal bone to the facial muscles. Early in the third trimester, fetuses engage in facial activity in which can be discerned behaviors that resemble frowning, smiling, crying, and laughing (Gingras et al., 2005; Kurjak et al., 2003; Reissland, Francis, Mason, & Lincoln, 2011; Yan et al., 2006). Thus, fetal development prepares the infant for interpretable emotional expressiveness, ensuring that the infant will be able to signal information about well-being to caregivers who can read those signals and provide for the child's needs. This process forms the foundation of the deep emotional bonds that will later mature into attachment security.

Interactions between infants and caregivers are emotion-rich exchanges that enable caregivers to interpret and meet the needs of the dependent infant and provide experiences that help infants form expectations about their emotional lives and relationships (Bowlby, 1969; Sroufe, 1997). Infants communicate about their well-being—whether their needs are met or not—through nonverbal behavior, mainly facial and vocal activity, often accompanied by gross motor activity. Initially, infant nonverbal signals are not highly specified in terms of basic categorical emotions. Crying is the earliest emerging signal of distress; even undifferentiated vocal distress conveys information about infant well-being (Izard et al., 1995; Oster, Hegley, & Nagel, 1992). Most caregivers are eager to and intent on interpreting infant nonverbal signals, adjusting their actions in efforts to stimulate and extend infant contentment and to alleviate infant distress. Caregiver reliable, sensitive responsiveness to these signals are the basis of the development of the child's sense of security, internal representations that distress can and will be alleviated. These attachment representations have long-lasting implications for close relationships throughout life (Isabella & Belsky, 1991; Izard, Haynes, Chisholm, & Baak, 1991; Simpson, Collins, Tran, & Haydon, 2007).

There is considerable debate regarding the presence and ontogeny of facial expressions of discrete emotions. In large part, the debate hinges on a conceptual issue regarding the degree to which human infants are biologically prepared to engage in motoric activities, such as facial configurations, that represent discrete emotions, and a methodological issue regarding the degree to which infant facial activity in emotion-relevant situations are discrete and specific. One view contends that infants do not communicate a set of basic, discrete emotions in the first year of life and that emotions are something more than hard-wired programming that unfolds in infancy (Camras et al., 2007; Camras & Shutter, 2010). An alternative view is that there is a core of first-order emotions (e.g., interest, joy, sadness, anger) for which infants are biologically prepared and that as early as the second month infants begin to engage in the first discrete expressions (Izard, 2011; Izard et al., 1995; Malatesta, Culver, Tesman, & Shepard, 1989). One way to integrate the information gleaned from both perspectives is to hypothesize that (1) human infants are biologically prepared for certain forms of appraisal/action-readiness patterns, which include nonverbal communication, that are socially interpreted as basic emotions; (2) that the facial musculature may not be capable of producing discrete expressions in early infancy but that infants may engage in varied and seemingly random facial activities that are a form of facial babbling; (3) that regardless of the clarity and specificity of infant nonverbal communications, most caregivers work diligently to interpret these nonverbal behaviors as signals about the infant's well-being; and (4) that during infant–caregiver interactions psychological meaning is ascribed to these expressions, contributing to the development of discrete expressions (Cole & Moore, 2015). Our understanding of infant expressive development will benefit from a view that includes consideration of both biological preparedness to engage in particular appraisal/action readiness patterns as well as appreciation of the influence of the interpersonal context in which communicative behavior develops and in which meaning is made. In sum, emotional expressiveness develops in the first year of life, contributing to the development of relationships, importantly the attachment relationships.

In the first months of life, infants engage in at least two forms of facial and vocal activity that are interpreted as meaningful by caregivers: crying, which is interpreted as indicating infant distress, and smiling, which is interpreted as indicating infant pleasure. Movements observed in the third trimester through the use of high-resolution sonograms suggest that fetuses engage in cry- and smile-like activities in utero, suggesting biological preparation for communicative signals that will be critical for survival in the first days of life (Gingras et al., 2005; Reissland et al., 2011). Crying and smiling behavior has been observed in neonates (e.g., Barr, Hopkins, & Green, 2000; Bertin

& Striano, 2006; Cecchini, Baroni, Di Vito, & Lai, 2011; Messinger et al., 2002; Zeskind, 2013). Although it is not yet known when infant facial and vocal activity reflects infant social understanding, it is clear that these nonverbal expressive behaviors have meaning for caregivers; typically, caregivers respond both physiologically and behaviorally, organizing to respond contingently, which serves to elicit infant responses, such as infant mirroring of an adult's smile (e.g., Del Vecchio, Walter, & O'Leary, 2009; Lavelli & Fogel, 2005). Moreover, although there are cultural differences in caregiver responsiveness to infant crying and smiling, it appears that all typically developing infants not only cry but also begin to engage in social smiles around age 2 months (Keller, 2013; Wörtmann, Holodysnki, Kärtner, & Keller, 2012).

Risk for Emotional Dysfunction Associated With Nonverbal Emotional Expressiveness. Because of the importance of nonverbal emotional expressiveness for an infant's survival and for the formation of a secure attachment, this capacity is sensitive to conditions that compromise effective emotional expressiveness. First, there are rare conditions that involve paralysis or very poor muscle tone, such as facial palsy, congenital myotonic dystrophy, Willi-Prader syndrome, and Möbius syndrome that interfere with expressiveness, including emotional expressiveness, and introduce significant consequences for social interaction and relationships, including the all-important caregiver–infant relationships on which attachment is founded. Möbius syndrome, for example, causes facial paralysis in infants. Impairments in facially expressive behavior were observed in all 25 children with Möbius syndrome in one of the rare studies of these infants (Sjogreen, Andersson-Norinder, & Jacobsson, 2001). This study, however, did not evaluate infant emotion expressiveness or emotional functioning. A single case study of an infant with Möbius syndrome (Szajnberg, 1994) noted that the child's separation behavior at age 14 months was consistent with an insecure (resistant) attachment, despite apparently normal cognitive development. This was interpreted in terms of the caregiver's difficulty knowing the infant's needs, which affected the ability to respond sensitively, leading to an insecure attachment.

Other neurodevelopmental conditions that are present early in life may also interfere with competent expressive behavior. There is a dearth of research on the emotional expressiveness of infants and young children with these conditions, limiting our ability to trace the contribution of atypical emotional expressiveness to the development of emotional dysfunction (Adams & Oliver, 2011). Down syndrome has varied presentations but children with this condition often present with hypotonia (muscle laxity) and delayed motor coordination, which can affect facial activity. Unlike infants with Möbius syndrome, infants with Down syndrome spontaneously engage in emotional expressions but, relative to typically developing infants, their expressive behavior develops more slowly and can be difficult to interpret (Carvajal & Iglesias, 2006; Cicchetti & Sroufe, 1976), which may contribute to higher rates of insecure attachment in these children by toddler age (Ganiban, Barnett, & Cicchetti, 2000). Due to their difficulties with social communication, there is more research on emotional expressiveness in children with autism than children with other neurodevelopmental disorders, although there is no known neuromuscular cause of expressive impairment. When compared with age-matched (chronological or mental age) children, young children with autism express emotion, both facially and vocally. However, they tend to display more negative and less positive spontaneous emotion, produce less articulated or more awkward expressions both in spontaneous social interaction and when asked to produce or mimic emotional expressions, have more monotonic prosody, and express emotion spontaneously more often during self-absorbed activity than during social interaction (Capps, Kasari, Yirmiya, & Sigman, 1993; Grossman, Edelson, & Tager-Flusberg, 2013; Hubbard & Trauner, 2007; Lord et al., 2000; Loveland et al., 1994; Snow, Hertzig, & Shapiro, 1987). For children with autism, atypical emotional expressiveness may be related to their difficulty engaging in face-to-face social interaction than in an impaired capacity to be expressive (McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006). A tendency to avoid social interaction, including avoiding looking at others' faces, could interfere with the types of affect mirroring and observational learning that is part of the coconstruction of meaning that occurs in typical infant–caregiver interactions. Discomfort or difficulty processing face-to-face communication then could affect how often and how well children will communicate nonverbally, which would then compromise their ability to convey unmet needs, which could increase their frustration, as well as create risk for emotion understanding and emotion regulation.

In sum, early childhood neuroatypical conditions may affect a child's ability to produce clear emotional expressions that others can use to interpret the goals and needs of the child. To the degree that children have such difficulty, their emotional competence in all domains is placed at risk.

There is little work that examines the pathway from neurodevelopmental risk to quality of emotional expression, but this represents one potential explanation for why these children have a higher incidence of mood and behavior problems (Eisenhower, Baker, & Blacher, 2005; Ross & Oliver, 2003). Early infant–caregiver emotional exchanges depend on infant non-verbal emotional expressiveness and compromised infant expressiveness is a risk factor for the development of a secure attachment, which is associated with lifelong challenges to healthy psychological functioning (e.g., Cicchetti & Roisman, 2011; Mikulincer & Shaver, 2012).

Being able to communicate whether needs are met or unmet, however, does not necessarily avoid risk for emotional dysfunction. It is essential that caregivers have the resources to understand those infant expressions and that accurately perceived infant expressions do not overwhelm the capacity of caregivers to attend to infant needs. First, infants who competently convey emotion depend on caregiver responsiveness to further develop their emotional competence. The quality of caregiver responsiveness to infant distress signals appears particularly important for later emotional competence. More than responsiveness to infant contentment and joy, caregivers' emotional responses to infant distress and the promptness and sensitivity of their actions appear to influence infant attachment status at age 16 months and child emotional functioning at 36 months (Leerkes, Blankson, & O'Brien, 2009; Leerkes, Parade, & Gudmundson, 2011). Second, some infants who are able to express emotion do so in ways that interfere with infant–caregiver interaction quality. It has long been understood that there are individual differences in very young infants, which has come to be understood as temperament. That is those individual differences reflect constitutional, neurobiologically based predispositions affecting infant reactivity to the environment and infant readiness and capacity for regulation (Rothbart & Derryberry, 1981). Some infants are characteristically quick and intense in their reactions to changes in the environment and not readily soothed or able to self-soothe (Rothbart & Bates, 2006). Their intense, enduring distress challenges caregiver–infant interactions (Kim & Kochanska, 2012; Leerkes et al., 2011). In this case, emotional expression of distress is not hard for caregivers to perceive, but the source of distress and what will alleviate the distress may be difficult to understand. Thus, the infant's ability to express emotion and the caregiver's ability to read infant emotion expressions are interpersonal microprocesses that are critical to relationship formation and quality, which are in turn critical for healthy development of the components of emotional competence. Atypicalities in infant–caregiver emotional expression interaction pose risk to relationship development, learning about emotion, and developing a repertoire of regulatory strategies. Thus this constitutes a potential path to emotional dysfunction that can consolidate in the form of symptoms of psychopathology, including co-occurring mood and behavior problems in infants with neuroatypical conditions.

Beyond infancy, caregivers continue to address children's needs but they also make more attempts to socialize child emotion expression. Often implicitly, and sometimes explicitly, socialization processes acculturate child emotional expressiveness (Cole & Tan, 2015). That is, in the course of caregiver–child interaction, children's emotions are socialized even caregivers are focused in children's conduct more than their expressiveness. As young children walk and broaden their repertoire for communication, caregivers strive to get toddlers to coordinate their emerging autonomy efforts with the constraints of their settings and to do in terms of caregivers' sociocultural expectations for acceptable, safe behavior. In addition to implicit socialization of emotion, caregivers may also explicitly teach or coach very young children as a response to child emotional expressiveness (Brophy-Herb et al., 2011; Lunkenheimer, Shields, & Cortina, 2007). In the course of these social exchanges, the typically developing child acquires a sense of the categorical, separate self, which matures into a formal theory of mind in the fourth year (Edwards & Liu, 2002; Wellman, 2011). The development of a sense of both self and other then contributes to the expansion of the emotional expressiveness repertoire, seen in toddlers' expressions of pride, embarrassment, guilt, and empathy (Davidov, Zahn-Waxler, Roth-Hanania, & Knafo, 2013; Lewis & Sullivan, 2005; Stipek, 1983).

This discussion of risk for emotional dysfunction has thus far emphasized nonverbal expression in the first years of life. Moreover, it has relied on studies that tend to focus on facial expressions. There is much more limited knowledge about typical and atypical development in other expressive channels, such as the voice, gesture, and body movement, which may have different developmental courses (Malatesta-Magai, Leak, Tesman, & Shepard, 1994). Nonetheless, we can conclude that, by the end of the second year of life, most typically developing children have a sizeable repertoire of nonverbal emotionally communicative behavior. To the degree that children's nonverbal emotional expressiveness is difficult for caregivers to understand, there is risk for the child–caregiver relationship, which is central to all aspects of the development of emotional competence. In the second year, however, an

important developmental milestone in typically developing children is the ability to communicate about goals and needs in a new way, through language. Verbal emotional expressiveness refers to the semantic content of speech rather than nonverbal activity.

Typical Development of Verbal Emotional Expressiveness

Very young children have remarkable growth in expressive language between infancy and school entry (Hart & Risley, 1995). Theoretically, the ability to verbalize needs and desires should reduce reliance on nonverbal communication of emotion and improve the specificity with which needs and goals are communicated in addition to contributing to emotion understanding and emotion regulation (Cole, Armstrong, & Pemberton, 2010). Studies show that children with language delays are more likely than the general population to have behavior problems, and children with behavior problems are more likely than the general population to have language delays. There are however very few studies that explain how and why language development contributes to any aspect of emotional competence (Cole et al., 2010).

Two longitudinal studies build on cross sectional evidence of associations between language development indices and aspects of emotional competence. Each found that toddler language skills contributed significantly to children's self-regulation during the preschool-age years (Roben, Cole, & Armstrong, 2013; Vallotton & Ayoub, 2011). For example, language skill level (relative to others) and the growth of language development (relative to self) in toddlers accelerated the normative decline in anger reactivity (in the context of having to wait to open a gift) between toddlerhood and preschool age (Roben et al., 2013). This effect appeared to involve children's development of putative regulatory strategies, including calmly verbalizing to the mother about the frustrating situation. Yet there is no reason to assume that the relation between expressive language and anger reactivity is unidirectional or that it is always an inverse relation. Intense emotional reactivity during the period of language acquisition may interfere with early language learning. Bloom (1995) contended that intense emotional reactions interfere with first word acquisition and vocabulary growth because the resources required for regulating the intense emotion compete for resources need for language learning. Longitudinal studies provide partial support of this contention, but only in the toddler years (Nozadi et al., 2013; Roben et al., 2012). On the other hand, being better able to verbalize needs appears not to automatically foster self-regulation as in the case of highly verbal, highly argumentative children. It is possible to draw on language skills to further self-determined goals for well-being that conflict with the goals of others. Thus, there is much to learn about the relations among child language development and its many facets and the development of emotional competence. Perhaps the most substantive research relating verbal expression of emotion and competence comes from research on children's ability to use terms for emotions.

Most of the research on children's emotion-related speech involves internal state language and specifically "emotion talk" (Bretherton, Fritz, Zahn-Waxler, & Ridgeway, 1986). As soon as children acquire expressive language, they begin to use terms for basic emotions, such as happy or mad (Bretherton et al., 1986). The methods used to study emotion talk often involve observations of parent and child interaction around hypothetical situations (e.g., talking about storybook characters) or reminiscing about past events involving the child. Parental use of emotion talk fosters young children's use of emotion terms, which in turn contributes to the development of theory of mind and social competence (Brownell, Svetlova, Anderson, Nichols, & Drummond, 2013; Taumoepeau & Ruffman, 2008). From the functionalist perspective, a particular interest is observing how children use language and nonverbal emotion expressiveness in situations that activate personally defined goals and not just contexts in which children are encouraged to talk about others' emotions or their own past emotions. These methods are used, however, because emotion talk occurs less often in naturalistic contexts, such as unconstrained home observations, and then child emotion talk appears to depend on parental use of emotion terms (Dunn, Brown, & Beardsall, 1991; Howe, Rinaldi, & Recchia, 2010). Naturalistic observations in the preschool setting recorded over two months yielded an average of 29 emotion references per child, with large individual differences (Fabes, Eisenberg, Hanish, & Spinrad, 2001). More often, preschoolers' internal state references were largely present tense, first-person references to their own likes and dislikes. Specific emotion terms were significantly less likely to occur: happy (10%), afraid (7%) sad (5%), and angry (3%). An interesting additional finding was that preschoolers' references to anger and sadness, although infrequent, usually entailed explanations about others' emotions rather than references to children's own experiences. Moreover, children who used emotion terms in explaining others' emotions were rated as more likable by their classmates. Thus, children's spontaneous references to emotion are infrequent, must

be taken in context, and are associated with competent social behavior.

Because observations of spontaneous speech are time-consuming and labor-intensive, researchers have used other strategies to place emotion talk in realistic context, such as examining references to emotion when parents and children discuss actual past events (Fivush, Hazzard, Sales, Sarfati, & Brown, 2003). Between ages 9 and 18 years, internal state language increases in narrative accounts of situations children and youth experience (Styers & Baker-Ward, 2013). The relevance of this method for understanding the role of verbal emotion expression may depend on the personal relevance of the recalled events for each partner as well as the timing of the conversations relative to when they happened. Immediately after a devastating tornado, mothers and their children did not refer to internal states any more than they did when discussing non-traumatic situations (Bauer, Stark, Lukowksi, Rademacher, & van Abbema, 2005). However, several months after the tornado, older children (~9 years old) made more references to their internal states than they had at immediately afterward, they did so more than younger children (~3 through 7 years), and their internal state language also increased in its specificity. Moreover, the history of the parent-child interactions may have been a factor. The effects were noted if mothers referred to positive emotions in discussing the tornado immediately afterward, suggesting that reminiscing about actual negative events may have been enhanced by the emotional tone of initial discussions. This work also suggests the value of repeated assessments when observing how children and their parents or peers verbally process the emotions associated with significant experiences.

Despite assumptions that children draw on their verbal abilities to cope with challenges to their goals, few studies have observed children's spontaneous verbalizations in such situations. However, there is research on young children's private speech, which is regarded as indicative of a transition from the sole function of speech for social communication to an internalization of social experience that guides action and problem solving (Berk & Winsler, 1995; Vygotsky, 1986). Day and Smith (2013) recorded preschool-age children's nonverbal emotion expressions and verbalizations in a quasi-naturalistic context: trying to attain a desired toy from a transparent locked box that would not open (mothers were present but asked not to engage in conversation). In this context in which children's expressions appeared authentic and spontaneous, nonverbal sadness was associated with mother-directed verbalizations, which can be interpreted as the use of both modalities to communicate their need for assistance in solving the problem. Their nonverbal anger expressions were associated with speech content that expressed frustration or hopelessness and fewer constructive self-instructions, which can be interpreted in the spirit of increasing effort to overcome the blocked goal. This study's findings raise interesting questions about the temporal dynamics of children's emotion expressiveness, verbalizations about emotion, and strategic efforts. That is, do outcomes depend on what a child verbalizes or the order in which verbalization, emotion expressions, and action occur? If frustration builds, a child verbalizes hopelessness, and then rebounds to try again, as was the case of the little girl described earlier in this chapter, the verbalization of hopelessness has a different context than if the child gives up trying and begins to cry after such a verbalization. There is much to learn about verbal communication related to emotion in typical development.

Risk for Emotional Dysfunction Associated with Verbal Emotional Expressiveness. Problematic verbal interactions are a hallmark of caregiver–child interactions among children or parents with elevated symptoms or diagnosed conditions. Many studies ask participants to discuss family conflicts that allow observations of caregivers and their children (preschoolers through adolescents) talking about situations that are personally relevant. These studies often do not differentiate verbal and nonverbal emotional expressions and if they do code them separately they rarely analyze them separately. Speech content may have a very different meaning depending on the affective prosody and facial expressions with which it is communicated (Cole, Teti, & Zahn-Waxler, 2003; O'Kearney & Dadds, 2005). In general, this research demonstrates that the problem discussions of caregivers and children who are symptomatic involve more negative emotion than asymptomatic control groups. For example, caregiver and adolescent anger during an interaction in which families were asked to resolve conflicts found that adolescents with depression communicated more anger and had higher heart rates (Jackson, Kuppens, Sheeber, & Allen, 2011). In conflictual interactions, partners, including caregivers and children, tend to mirror each other's emotions (e.g., Cole, Teti, & Zahn-Waxler, 2003; Dumas, LaFreniere, & Serketich, 1995). Many of the communications in these studies involve situations in which each partner is blocking the other's goal. However, if a partner expresses sadness, is it reciprocated? Typically, we expect sympathetic responses to sadness. For instance, children report expecting caregiver support when they express sadness in contrast to their

expectations about caregiver responses to anger, which involve sanctions (Zeman & Garber, 1996). Perhaps the more serious risk is caregiver dismissiveness of children's negative emotions, including their sadness and anxiety (Lunkenheimer et al., 2007). Too often however research on verbal emotional expressiveness focuses on general caregiver reactions to child emotion (e.g., encouraging emotion expression) and not responses to different emotion expressions in specific situations (but see Morris et al., 2011; Zeman & Shipman, 1997). We should also be cautious about interpreting the angry conflicts of caregivers and children with symptoms or disorders as indicating that mutual anger necessarily represents risk. Anger, the appraisal/action readiness that a goal is blocked and a person is ready to respond with effort to attain it, is adaptive because it helps personal goal achievement particularly when the partners validate each other's anger and work toward resolution. A person diagnosed with depression who communicates anger is conveying something other than hopelessness or dysphoria. In the context of family interaction, we should consider that a depressed child or youth who is angry is still striving to communicate and achieve personal goals, even if those goals conflict with caregivers' goals. An important clinical question, then, is what distinguishes healthy angry exchanges from those that reflect dysfunctional interaction? Might anger buffer the depressed child or youth against despair and suicidality (Cole, Hall, & Radzioch, 2009)? A functional perspective requires a deeper analysis, which also requires analysis of the temporal, dynamic flow of interaction. Notably, in one prospective study adolescents who had been classified as secure as infants expressed more, rather than less anger, toward their mothers in the context of a frustrating game compared with adolescents who had been classified as insecure as infants (Spangler & Zimmermann, 2014); in other words, anger expression in this context was not associated with risk and may reflect implicit understanding that one can express authentic anger in the safety of a trusted relationship. In sum, how partners verbally communicate about emotions is as central to pathways toward dysfunction as nonverbal expressions. Intense nonverbal expressiveness of negative emotion however is likely to elicit strong reactions from partners that may interfere with the clarity of messages being communicated verbally.

Another clinically relevant issue regarding children's verbal expression of emotion and spontaneous talk in or about personally meaningful circumstances is the fact that many children with clinically elevated symptoms or diagnosed disorders do *not* talk about how they feel. There is little normative research to address why children come to this point, and clinical research dwells more on emotion understanding and regulation than it does on emotional expressiveness, with the exception of work on alexithymia, the condition in which individuals appear unable to label emotions. The ability to talk authentically and spontaneously about actual circumstances is an aim of most therapeutic approaches. There are several hypotheses that can be generated to consider the development of this impairment in an emotional competency. Some children and youth may lack emotional awareness. Others may be acutely aware of their emotions but avoid talking about intense feelings as a method of regulating them. Still others may be aware and willing to talk but expect that others will be punitive, misunderstand, or be indifferent to their needs. Parents and their 8- to 13-year-old children, with and without diagnosed anxiety disorders, were asked to discuss past occasions in which the child had been anxious, angry, and happy (Suveg et al., 2008). Mothers and fathers, particularly those whose children were diagnosed with anxiety disorder, expressed more negative emotion when recalling their children's anger (relative to anxiety and happiness) but this emotion specificity was not observed in the children. How does this pattern of interaction develop and where does it lead? Does parental anger reflect frustration with a challenging child? Is it a cause or a result of clinical levels of child anxiety? A similar reminiscing task was used to observe family interaction with 3- to 9-years-olds who were diagnosed with conduct disorder diagnoses; the children were divided into high and low categories on callous-unemotional (CU) traits (Pasalich et al., 2014); notably, children who were higher in CU traits communicated more negative emotion, but these were accounted for by references to sadness and fear (e.g., "sometimes you shout at me and I get *scared*"; p. 308). These verbalizations, which were contrary to predictions, were independently judged as appropriate and genuine communications, yielding findings that call into question the prevailing view of callous children being emotionally indifferent. They are also interesting in relation to a finding that preschool-age children who were emotionally inexpressive, relative to highly and modulated expressive children, were more likely than either of those other groups of children to report depressive symptoms two years later (Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996).

Research on verbal communication about emotion in school-age children and youth has not yet documented developmental pathways involving authentic verbal expression of emotion. The available literature suggests that the use of words to communicate about emotion begins as early as the second year of life and that the quality of

parenting influences whether children's verbal expression becomes an aspect of competent emotion expressiveness or an additional aspect of symptomatic behavior, such as being argumentative. From a functional perspective, research is needed that examines children's interactions with others about personally meaningful circumstances and that considers these interactions from multiple temporal perspectives: (1) the immediate temporal dynamics of emotion exchanges that involve personally relevant goals, including conflicting goals, and how each partner relates to the other leading to either effective problem solving or to prolonged or increased interpersonal conflict (see Dagne & Snyder, 2011, for an illustrative approach to time series of emotional exchanges), (2) consideration of whether and in which conditions it may become easier to discuss important topics with the passage of time (Bauer et al., 2005), and (3) prospective analysis of how communication about emotion contributes to stability and change of emotional dysfunction over time (Cole et al., 2003; Denham et al., 2000; Schwartz et al., 2012). In addition to incorporating a dynamic view, the functional perspective advocated for research to appreciate that negative emotions are not inherently problematic (anger reflects an effort to effect change) and that positive emotions are not inherently valuable (enjoying another's distress is callous). Emotional competence is defined by authentic, socially appropriate emotional expression, such that expressive control—being expressive but modulating expressiveness is key. The ability to verbalize about emotion, or at least to label emotions, may serve to dampen or modulate emotional reactivity, a regulatory function of language that has been observed in experimental (nonnaturalistic) studies with adults but which has not yet been studied in children (Lieberman, Inagaki, Tabibnia, & Crockett, 2011; Matejka et al., 2013). Competent emotional expressiveness, nonverbal and verbal, involves effective, appropriate, flexible communication and this entails the ability to modulate expressiveness, an aspect of emotion regulation that we discuss in the section on emotional expressiveness.

Typical Development of Expressive Control

The ability to modulate emotional expressiveness is consistently linked to parent- and teacher-rated child socioemotional competence in the preschool, elementary, and high school years (e.g., Blair, 2002; Eisenberg et al., 2001; Feldman, Tomasian, & Coats, 1999; McDowell & Parke, 2005). The earliest development of expressive control occurs during the third year of life, when young children make autonomous, self-initiated efforts to control their expression of negative emotion and distress in the absence of adult directions to do so (Kopp, 1989). Early work on expressive control entailed conscious masking of emotion and intentional deception with a focus on the face (e.g., Feldman, Jenkins, & Popoola, 1979; Lewis, Sullivan, & Vasen, 1987). Children were instructed to pose or deceive, which informs the development of voluntary expressive control in situations that may not have had little personal relevance for children. An exception was a paradigm in which children were told not to peek at a tempting object and then were asked if they had, a more naturalistic analogue of the circumstances in which children lie (Lewis, Stanger, & Sullivan, 1989). A study of 3- through 7-year-olds found that a substantial majority peeked (70%), that lying about peeking increased between age 3 (50% confessed) and age 6, when more children lied (~60%), and that adults (parents and undergraduates) could determine who lied only on the basis of verbal and not nonverbal expressiveness (Talwar & Lee, 2002). This skill is well established by elementary school age (Halberstadt, Grotjohn, Johnson, Furth, & Greig, 1992). From the functional perspective there is a need for more research on involuntary as well as voluntary expressive control in real-life situations. Indeed, school-age children report feeling and expressing more emotion in contrived or hypothetical situations than they express in actual situations, such as being treated unfairly or experiencing pain (Larochette, Chambers, & Craig, 2006; Parker et al., 2001).

In more naturalistic contexts involving negative emotions, typically developing children as young as age 3 years show spontaneous expressive control, although this depends on social context. That is, typically developing preschool-age children try to control disappointment in their faces when disappointed by an unfamiliar person (a research assistant) but not when alone (Cole, 1986). At this young age, children are not skillful dissemblers, but they spontaneously try to control their nonverbal expressions of anger, sadness, or disgust. For example, a girl who had turned three on the previous day smiled broadly as a matter-of-fact research assistant handed her a broken toy, her least desired prize; however, as soon as the assistant left, the little girl frowned, wrinkled up her nose, raised her upper lip, and pushed the prize away. Why she controlled this expression in the presence of the assistant is not known but her behavior was spontaneous control of emotional expression. The expressive control may have served to regulate her own emotions, to avoid a punitive reaction by the assistant, or internalization of rules of polite emotional behavior. Such expressive control is less likely to occur in interactions with trusted caregivers.

Skill at expressive control improves over the course of middle childhood (Garrett-Peters & Fox, 2007; Saarni, 1979), a result of experience and socialization influences. Parental warmth, sensitivity, positive expressiveness, sensitive control, and use of child emotion as a teaching opportunity have all been correlated with child emotional competence, including expressive control (Eisenberg, Cumberland, & Spinrad, 1998; Garner & Power, 1996; Gottman, Katz, & Hooven, 1997; McDowell & Parke, 2005; Morris, Silk, Steinberg, Myers, & Robinson, 2007). However, to inform the development of *psychopathology*, much more needs to be known. For example, many parents react to inappropriate emotional expression in their children by countering with their own negative emotion while others are dismissive. In typical development, parents' warning tone of voice or frowning faces may help children inhibit undesirable behavior. In the context of warm, supportive relationships typical parental anger may enhance rather than compromise children's ability to inhibit behavior and contribute to children's learning of family and sociocultural rules about the contexts in which one does and does not communicate different appraisal/action readiness patterns (see Morris et al., 2007 for review).

Child characteristics that develop in early childhood are thought to contribute to expressive control. Effortful control, defined by processes that modulate emotional reactivity, and general skill at emotion knowledge have both been associated with children's expressive control (Hudson & Jacques, 2014; Kieras, Tobin, Graziano, & Rothbart, 2005; Kochanska, Murray, & Harlan, 2000; Zeman & Shipman, 1998). The inference that children's abilities to recruit executive processes and to access or to apply emotion knowledge to control emotional expressiveness is based on correlational research. Effortful control may aid expressive control because it involves activation of networks that allow for top-down regulatory influence over action readiness or prepotent response tendencies. Functional near infrared spectroscopy (fNIRS) with 3- to 5-year-olds revealed greater lateral prefrontal cortex in the context of a blocked reward, an ecologically meaningful experimental situation, and that greater activation, which is associated with executive attentional control, was associated with better child frustration tolerance as reported by parents (Perlman, Luna, Hein, & Huppert, 2014). Emotion understanding has also generally been associated with younger children's expressive control (Garner & Power, 1996; Hudson & Jacques, 2014) but findings may not extend to all contexts (Ceschi & Scherer, 2003). Thus, the study of evidence for putative sources of expressive control—child characteristics such as effortful control and emotion understanding and parenting characteristics such as warmth, sensitivity, and control—need to be addressed in terms of specific emotions in specific contexts, particularly when participants are engaging in natural or reasonably simulated natural situations. The inclusion of neurobiological measures may also advance understanding of the development of expressive control. Finally, intervention research that distinguishes among treatment components may help to disentangle relations between expressiveness, expressive control, effortful control, and emotion knowledge (Raver, Garner, & Smith-Donald, 2007).

Risk for Emotional Dysfunction Associated with Expressive Control. When children do not engage in the expressive control typical of children their age, they are often regarded as odd or problematic by others. Naturalistic observations of children's emotional expressions indicate that between ages of 4 through 12 years most children's expressions are brief and moderate, even in challenging situations, and the assumption is that they modulate expressiveness (Buss, Davidson, Kalin, & Goldsmith, 2004; Fabes et al., 1999; Sears, Repetti, Reyonds, & Sperling, 2014). There is a number of ways in which the expressive control of at risk and symptomatic children differs from that of typically developing children. Some express emotions judged to be disproportionate and reactive (quick, rapidly intensifying expressions involving multiple elements in the face, voice and possible other motor behavior such as gestures), some express emotions in situations in which their emotional expressions are unexpected or atypical (context inappropriate or context insensitive), and some are inexpressive when expressiveness is typical and expected (Buss, 2011; Cole et al., 2013; Cole, Zahn-Waxler, & Smith, 1994; Cole et al., 1996; Dix, Meunier, Lusk, & Perfect, 2012; Locke, Davidson, Kalin, & Goldsmith, 2009). There are multiple potential factors leading to atypical expressive control. As noted previously, there are conditions that interfere with neurophysiological control of facial muscles, such as facial palsy, Parkinson's disease, and involuntary emotional expression disorder (Bogart, Tickle-Degnen, & Ambady, 2012; Lauterbach, Cummings, & Kuppuswamy, 2013); with intervention, many individuals with these conditions learn compensatory methods of expressing themselves (Bogart et al., 2012). If they do not, their social interactions are impaired, which can cause a cascade of negative effects on their social interactions, relationships and self-worth.

Among children with the physiological capability to engage in expressive control, impaired expressive control

is usually a consequence of other risk factors and has negative consequences on children's self and social development. Most often, negative emotion that is expressed very quickly and intensely is interpreted as reflecting poor expressive control. For example, there is ample evidence that children and adolescents with externalizing behavior problems express more intense and persistent anger and sadness in frustrating or disappointing situations, with parents or with unfamiliar adults, then asymptomatic children who appear to engage in expressive control (e.g., Cole et al., 1994, 1996; Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). Another concern is the expressive control that underlies some children's reluctance to express emotions that are authentic, reasonable, and acceptable to express. Chaplin (2006) used a quasi-naturalistic procedure, a difficult task that required cooperative behavior between friends, to investigate individual differences in expressive control as a function of depressive symptoms. Older adolescents with depressive symptoms reported more anger but displayed less anger, and reported less happiness but displayed more happiness, than the older adolescents without elevation in depressive symptoms. How might this develop? One cross sectional study of 9-year-old girls found that both inhibited and disinhibited emotion expression during a family interaction was associated with depressive symptoms in children whose symptoms had not yet meet criteria for a diagnosis of a depressive disorder (Keenan, Hipwell, Hinze, & Babinski, 2009). A longitudinal study indicated that 13- to 14-year-olds' depressive symptoms predicted a decline in parental support over the course of a year, which mediated a relation between earlier depressive symptoms and increases in inhibited expressive control over two years (Larsen et al., 2012). Finally, preschool-age girls with symptoms of oppositional defiant disorder engaged in more expressive control than typically developing girls and boys, and boys with symptoms, even in situations in which other children conveyed their anger and sadness (Cole et al., 1994). These studies indicate the highly transactional nature of expressive control and the interpersonal climate in which children and youth develop and suggest that suppression of authentic emotions is as much a clinical concern as intense emotional reactions.

In sum, the capacity for authentic emotional expressiveness develops prenatally and emotional expressions become acculturated and articulated early in life. Early nonverbal expressiveness and caregiver responsiveness, particularly to infant distress, is essential in the development of a secure attachment relationship. Throughout life, the capacity to communicate clear, authentic emotion nonverbally is important for the formation and maintenance of all relationships and for skillful social interaction. With this capacity comes risk for emotional dysfunction related to conditions that compromise nonverbal expressiveness. Risk conditions include atypical development of nonverbal expressiveness as well as poor quality caregiver responsiveness to infant nonverbal expression. In addition to nonverbal behavior, emotional expressiveness develops in two further ways over the course of childhood and adolescence. By the second year of life, children begin to express goals, needs, and emotions with words. By the fourth year, they begin to show the capacity to spontaneously modulate what they communicate—expressive control. Difficulty verbalizing about one's emotions, having difficulty modulating emotional expressions, and inhibition of authentic emotion (verbal or nonverbal) are all associated with being less socially skilled and pose risk for the development of emotional dysfunction. When challenging circumstances arise with which children have limited experience (e.g., loss of a grandparent, exposure to community violence, bullying), the emotionally competent child is able to discuss difficult material with a trusted, sensitive caregiver. When the caregiver is poorly equipped to engage in such communication with the child, when the child does not trust caregivers with difficult emotional material, and when the challenging circumstances are presented by the caregiver, the child's ability to effectively express emotion can be compromised. This can lead to the blunting of expressiveness or to unpredictable, disproportionate, or context-inappropriate expressiveness. Furthermore, inability to communicate emotion authentically and appropriately compromises the interpersonal exchanges from which competence in emotion understanding and emotion regulation develops; these skills further refine an individual's emotional expressiveness.

Emotion Understanding

Competence in *emotion understanding* comprises a variety of component skills all of which involve knowledge about one's own and others' emotions. These skills include being able to accurately recognize and discriminate among different emotions, knowing about potential causes and consequences of different emotions, and knowing how emotions can be regulated. Competence in this domain includes related concepts such as emotional awareness or clarity (being able to experience and recognize one's own emotions clearly), emotion perception (being able to accurately decode facial activity and vocal qualities of different emotions expressed by others), and display rule knowledge

(being aware of cultural rules about how emotion is communicated and being aware that expressive behavior may not reflect subjective feeling). Competent emotion understanding also involves the development of theory of mind (being able to understand that another person may have a different emotional reaction than your own) and empathy (understanding another's emotion and being emotionally responsive on behalf of the other's needs). In total, competent emotion understanding entails effective and flexible application of these different skills to actual situations, particularly those in which this knowledge contributes to coping with problematic situations (Saarni, 1999).

As with other domains of emotional competence, emotion understanding is closely linked to the other components—emotion expression and emotion regulation—and is best understood in the context of social interactions and relationships. The reciprocal, interacting components of emotional competence begin to develop within caregiver–infant interactions and extend through the companionships of elders. For example, just as the infant begins to engage in emotional expressiveness, the infant quickly learns to read emotional expressions, such that competence at expressiveness may contribute to competence in emotion perception. Moreover, intense negative emotion may influence the degree to which emotion understanding can be successfully recruited or effectively applied (Cummings & Rennels, 2014; Fizke, Barthel, Peters, & Rakoczy, 2014; Prigatano, 2013). Much of the research on the development of emotion understanding has relied on methods using schematic or hypothetical stimuli but emotional competence requires understanding emotion in the real-life or realistic simulations of circumstances that infants, children, and youth encounter. Although there is a relative dearth of work derived from naturalistic studies, there is, nonetheless, ample evidence that the ability to recognize, conceptualize, and label emotions is associated with children's social competence (Denham, 1998; Dunn, 2004). Moreover, there is considerable research indicating that children and youth with various forms of psychopathology often show less skill in emotion understanding (e.g., Southam-Gerow & Kendall, 2002).

Typical Development of Emotion Understanding

The most basic component of emotion understanding is the ability to perceive and recognize emotional signals in others' nonverbal behavior, notably facial configurations and vocal prosody. This basic component of emotion understanding is a complex psychological process, involving a number of cognitive components whose successful execution depends on the coordinated effort of different regions of the brain (Adolphs, 2002). The readiness to perceive others' emotionally expressive behavior appears to have a very early origin; the development of perception is known to involve the converting of sensory information into percepts (and eventually concepts) and this process depends on exposure to stimulation. Little is known about prenatal visual development. In utero, infants do not observe faces, although by midterm the suprachiasmatic nuclei, which are responsive to light and implicated in circadian rhythm, have developed (Rivkees, 2003). In the first months infants can only see 8–12 inches before them; the development of the ability to discern facial activity details develops during the first year; experience with visual stimulation fosters the perceptual discrimination of shapes, patterns, and motion, spurred in large part by the visual array of the caregiver's face (Graven & Browne, 2008). Auditory processing, on the other hand, develops earlier. The fetus is exposed to auditory stimulation, particularly the mother's voice. Fetal auditory cortical and heart rate changes are evidenced by at least the third trimester, including selective processing of the mother's voice (Jardri et al., 2012; Kisilevsky & Hains, 2011), which may explain why neonates recognize their mother's voice (DeCasper & Fifer, 1980).

The ability to recognize nonverbal emotional information in faces and voices then depends on experience. It does not appear to depend, however, on the ability to produce facial expressions and affective prosody. For example, a case study of three adults with congenital Möbius syndrome indicated they were able to recognize emotions in facial and vocal stimuli (Calder, Keane, Cole, Campbell, & Young, 2000). Moreover, only some aspects of emotion understanding may depend on experience with sensing stimuli. Severely visually impaired children have difficulty identifying emotion in vocal stimuli, but perform as well as children without any sensory impairment (and better than hearing-impaired children) in understanding the meaning of emotion words and in reasoning about the causes of emotion (Dyck, Farrugia, Shoceht, & Holmes-Brown, 2004). Among typically developing infants, the ability to discriminate among different types of affective prosody is evident by the middle of the first year, as inferred from behavioral and neuroimaging studies (Fernald, 1993; Nelson & de Haan, 1997). Similarly, infants discriminate among basic facial expressions of emotion (Grossmann, Striano, & Friederici, 2006; Kobiella, Grossmann, Reid, & Striano, 2008). By the time they reach their first birthday, before they have acquired emotion labels and concepts,

very young children not only differentiate among nonverbal emotional signals; they use the information they have gleaned from others' nonverbal signals to guide their action as indexed by approach behaviors and attribute emotional signals to the expressive person and not to other stimulus conditions (Klinnert, Emde, Butterfield, & Campos, 1986; Repacholi & Meltzoff, 2007; Repacholi, Meltzoff, & Olsen, 2008).

As children enter toddlerhood, they soon begin to develop an emotion lexicon of simple terms, such as happy, scared, and mad (Bretherton & Beeghly, 1982; Ridgeway, Waters, & Kuczaj, 1985), usually referring to their own experience. Between ages 2 and 3, children can associate these terms with simple schematic representations of basic facial expressions (Borke, 1971; Denham, 1986; Reilly, McIntire, & Bellugi, 1990). Children first identify happiness reliably and over the first years continue to develop skill at differentiating among negative emotions (Camras & Allison, 1985; Widen & Russell, 2003). Emotion vocabulary appears to double in size each year from ages 4 to 11 years and then may plateau during adolescence (Baron-Cohen, Golan, Wheelwright, Granader, & Hill, 2010). By preschool-age children can apply their emotion vocabulary to concepts about the nature of emotion. That is, they not only demonstrate a basic understanding of the causes and consequences of the principal emotions but have also developed an understanding that others' emotions may differ from their own, that others' thoughts about their behaviors influence their emotional reactions in the present and over time, that another person's distress may or may not be justified, that one can pretend to feel emotions, and that certain emotions are more acceptable to display than others (Bassett, Denham, Mincic, & Graling, 2012; Hepach, Vaish, & Tomasello, 2013; Lagattuta, 2007, 2008; Sidera, Amadó, & Serrat, 2013). Hence, preschool-age children understand a considerable amount about emotion, which is a contributing factor to their social competence.

In sum, infants navigate a communicative world in which they perceive and interpret emotional signals that are converted into conceptual representations by the time they reach preschool age. There are, however, significant limitations on their ability to understand the complex emotional events they perceive. Thus, perceptual sensitivity and initial attribution of meaning to those percepts is both an accomplishment and potential vulnerability. For example, typically developing infants and toddlers exposed to very brief angry exchanges between two adults orient toward action as evidenced by cardiovascular changes, appear distressed and often become behaviorally disruptive (Cummings, Zahn-Waxler, & Radke-Yarrow, 1981; Cummings et al., 1987; Moore, 2009). Very young children are therefore sensitive to emotional information but also lack the cognitive skills to reason about emotional information in the more sophisticated ways that develop throughout middle childhood and adolescence.

There is less research, especially longitudinal research, on the development of emotion understanding beyond early childhood (Zeman, Klimes-Dougan, Cassano, & Adrian, 2007). Between ages 6 and 13 years, emotion understanding becomes more complex, more nuanced, and more abstract (Lagattuta, 2014). Over this period of time, children become better able to infer emotions on the basis of situational information without the advantage of nonverbal expressive cues, to generate alternative views about how a person might feel in a given situation, and to appreciate that a person can experience multiple emotions, even if those different appraisal/action readiness patterns appear to be contradict each other (Pons & Harris, 2005; Hoffner & Badzinski, 1989; Harter & Whitesell, 1989). In addition, during the middle childhood years, children become more fluent in describing their own emotions and styles of emotional responding, in describing and reasoning about different strategies for regulating emotion, in appreciating that regulatory strategy effectiveness depends on the specific emotion, its intensity, and the situational context (e.g., who is present), and appreciating that how one thinks about a situation affects emotional valence (Bamford & Lagattuta, 2012; Davis, Levine, Lench, & Quas, 2010: Dennis & Kelemen, 2009; Larsen, To, & Fireman, 2007; Penza-Clyde & Zeman, 2002; Reijntjes et al., 2006; Waters & Thompson, 2014; Zeman & Shipman, 1997). The normative emotion understanding development associated with middle childhood that is absent for most preschool-age children is why clinicians are able to move from activity- and play-based to conversation-based interventions around age 9.

By adolescence, experience and cognitive development continue to permit increasingly sophisticated ways of understanding emotion, including integrating knowledge of emotions into ever more complex understanding of human physiology, social relations, and cultural values (e.g., Fischer, Shaver, & Carnochan, 1990). These ways of understanding include advances in metacognition, that is, reflection on cognitive processes and how one uses them, including appraisal/action readiness patterns, self-monitoring, cognitive flexibility, such as the capacity to evaluate the risks and benefits of acting on one's appraisal/action readiness, and more abstract reasoning about emotion relevant circumstances, such as principles like fairness and justice (Arsenio, Gold, & Adams, 2006;

Crone & Dahl, 2012; Flavell, 2000; Steinberg, 2005). Adolescents' emotion understanding skills broaden the capacity to infer emotions (and emotions are inferred processes), enhance coping with stress, including coping with stressful events that have not been encountered previously and for which one lacks experience (Compas, Malcarne, & Fondacaro, 1988; Eisenberg et al., 1997; Williams & McGillicuddy-De Lisi, 1999).

Research ranging from early childhood through adolescence often assumes that emotion understanding is a prerequisite for emotion regulation. Although the various components of emotion understanding are consistently correlated with parent-, teacher-, or peer-reported social competence or behavior problems, effect sizes are modest. Among the components of emotional competence, emotion regulation has larger effect sizes as a mediator of relations between predictors and outcomes than emotion understanding (Garner & Hinton, 2010; Trentacosta & Fine, 2010). One concern about the status of knowledge on the development of emotion understanding and its relation to emotional competence involves the methods used to assess emotion understanding. With participants in middle childhood, many studies have relied on decontextualized, experimental stimuli and hypothetical situations. Research that is informed by clinical concerns and takes a functional perspective will investigate children's understanding in realistic or actual situations, and yield information that is more pertinent to the unique role of emotion understanding in the development of emotional dysfunction and in pathways to psychopathology.

Risk for Emotional Dysfunction Associated with Emotion Understanding

There is ample evidence that children, youth, and adults with elevated symptoms or clinical disorders are less accurate at recognizing emotion, particularly in faces, and are less mature in their reasoning about emotions (Southam-Gerow & Kendall, 2002; Trentacosta & Fine, 2010). Neuroimaging studies indicate that these differences are reflected in temporal and spatial aspects of neural activity (e.g., Blair, 2003; Herba & Phillips, 2004). In considering risks associated with emotion understanding, it is useful to return to a fundamental principle of the functional perspective on emotion. That is, the capacity to be emotional is designed by evolution to enable adaptation to changes in the environment. Most children are biologically prepared to be sensitive from a very early age to the emotional information in the environment. They perceive changes, appraise their meaning and orient to act in relation to them, and in this way adapt to regular patterns in their emotional environments. In typical development, adaptation to environmental challenges can provide opportunities for psychological growth, including experiential learning that cultivates emotional understanding (and all aspects of emotional competence), and is supported by caregivers who help to relieve child distress, model emotional behavior, help children understand emotions and their circumstances, and teach them how to cope with emotions and situations (Eisenberg, Cumberland, & Spinrad, 1998; Morris et al., 2007). However, early competence in emotion understanding also contributes to children's vulnerability. Historically, popular culture believed that adversities like marital distress did not negatively influence very young children because they could not understand; in other words, the very young were oblivious to distress. We now know that infants and young children not only perceive but are sensitive to emotional information, and that limitations on young children's ability to further understand what they perceive creates vulnerability. When environmental conditions are perceived but stress children who lack the cognitive resources to resolve the distress, and most especially when they also lack the social resources because the stress involves caregivers' behavior, children will adapt. But, in doing so, their long-term emotional development may be compromised.

Because young children perceive emotional information, they can be stressed by it. Moreover, depending on their age and experience, children are prone to misunderstand what they perceive, to misattribute the causes of emotional events, and to even assume they play a central role in these events if they have not developed the ability to decenter, that is, mentally separate themselves from events; Shantz, 1993). Moreover, in early childhood when neurophysiological systems that enable appraisal/action readiness are still forming and are plastic, open systems, emotional understanding means children are not impervious to adverse conditions. The resulting stress can have enduring effects on the biological systems that support appraisal and action readiness.

Early adversity is defined by various circumstances, including potentially traumatic interpersonal situations, such as domestic violence, child abuse and neglect, as well as non-interpersonal situations such as injury, malnutrition, and natural disaster. In children ages 24 to 48 months, it is estimated that exposure to potentially traumatic experiences is as high as 26% (Briggs-Gowan, Ford, Fraleigh, McCarthy, & Carter, 2010; U.S. Department of Health and Human Services, 2009). Early and persistent exposure to adversity influences gene expression (Meaney et al., 1996), stress physiology (McEwen, 2000), and neural connectivity

(Burghy et al., 2012; Fan et al., 2014; Graham, Fisher, & Pfeifer, 2013). In these various ways, early adversity has profound implications for all domains of developing emotional competence, including psychopathology (Green et al., 2010). A substantial majority of research, however, has concentrated on emotion understanding.

One form of early adversity occurs when young children are deprived of adequate social interaction, a condition that has pervasive negative consequences. Child neglect and institutionalization represent this form and are known to have consequences for emotion understanding. Child neglect is associated with a range of problem outcomes affecting both cognitive and emotional functioning (Erickson & Egeland, 1996; Heim, Shugart, Craighead, & Nemeroff, 2010). Preschool- and school-age children who experienced neglect are less skilled in detecting, labeling, and understanding emotion, even when child IQ and harsh parenting are taken into account (Pollak, Cicchetti, Hornung, & Reed, 2000; Shipman, Edwards, Brown, Swisher, & Jennings, 2005; Sullivan, Carmody, & Lewis, 2010). Institutionalized children, compared with those placed in foster care or never institutionalized, also are less skilled at recognizing emotion in faces (Nelson, Westerlund, McDermott, Zeanah, & Fox, 2013; Moulson, Fox, Zeanah, & Nelson, 2009; Slopen, McLaughlin, Fox, Zeanah, & Nelson, 2012). Adults with a documented history of childhood emotional neglect, but not physical or sexual abuse, had reduced resting state connectivity in pathways associated with the social brain, that is, between the right amygdala and (1) the precuneus and lateral occipital cortex; (2) the left orbitofrontal cortex, insula, hippocampus, and putamen; and (3) the dorsal anterior cingulate cortex (all active in socio-emotional information processing and emotion regulation) and they have enhanced amygdala reactivity to emotion faces (van der Werff P et al., 2013; van Harmelen et al., 2010). These findings suggest that insufficient opportunity to interact with others' emotions compromises emotion understanding. Fortunately, the plasticity of early development may make it possible for the introduction of adequate caregiving and social interaction to remediate the early effects of neglect and deprivation, although it may be critical for such intervention to occur before age 2 (Moulson et al., 2009; Tarullo, Bruce, & Gunnar, 2007). That is, the costs to emotion processing of insufficient or unresponsive caregiving, through parental neglect or institutionalization, may be ameliorated by healthy emotional interactions, particularly during the most formative period.

While the deleterious effects of insufficient human interaction have consequences for children's development of emotional competence, many children experience interactions that threaten their well-being, notably child abuse (physical and sexual) and exposure to interadult conflict. Of course, these different forms of early adversity often co-occur. From the viewpoint of emotional development, if what is most salient to children are threatening interactions, these should have somewhat different consequences than those of consistent diminished interactions in terms of children's adaptation to those environments. Abused children provide further evidence that significant adversity attunes and shapes neural processing. Physical abuse is linked to *better* rather than less skillful emotion perception, specifically for angry expressions. Physically abused children are quicker, more prepared to detect anger as an adaptive response to unpredictable threat in their environment, which improves their ability to avoid danger although it also causes them sustained anxiety (Pollak, Messner, Kistler, & Cohn, 2009; Pollak & Sinha, 2002; Shackman & Pollak, 2005; Shackman, Shackman, & Pollak, 2007). It is particularly interesting to note that in the case of abusive mothers, their angry nonverbal expressions may not be as clearcut as those of non-abusive mothers (Shackman et al., 2010). The advantage of such quick perception of potential threat, however, does not extend to other aspects of emotion understanding; physically abused children do more poorly than other children in understanding the causes of emotion (Luke & Banerjee, 2013). Thus, perceptual sensitivity may heighten stress when children are exposed to environmental threats, without the presence of supportive caregiving, altering both autonomic and central nervous systems that process emotional information (e.g., Cicchetti, Rogosch, Gunnar, & Toth, 2010; McCrory et al., 2013). However, these conditions do not foster children's ability to develop other aspects of emotional understanding, in part because uncontrollable stress may require them to turn their attention away from emotional experience.

Another well-researched form of adversity encountered by many children in which emotion understanding plays an important role is interpartner conflict. The way that children interpret interpartner conflict influences their adjustment (Grych & Fincham, 1990). Interparental conflict is a fact of life for a large swath of children in the United States, although precise estimates are difficult given the variations in the structure of modern family life. Most adults marry and most couples have children. After rising rapidly, American divorce rates have begun to level off. According to a Pew Research Foundation report (Krogstad, 2014) about 14% of couples, most with children, are separating or divorcing at any given time. In addition, about 50% of children live with a step-sibling,

and a growing number of children are reared by a single parent. In each of these different situations, children may experience interpartner conflict or violence. Exposure to interpartner conflict, notably unresolved conflicts, is believed to confer risk by threatening the emotional security the child has achieved (Cummings & Davies, 2011). Nearly 16 million children live in households experiencing intimate partner violence (McDonald, Jouriles, Ramisetty-Mikler, Caetano, & Green, 2006).

Appraisal and action readiness in the context of conflicted close relationships is influenced both by children's sensitivity to emotional stimuli and the extent of their emotion understanding skills. Infants and toddlers are sensitive to interadult anger, which elicits stress responses in them, as inferred from changes in their behavior, their cardiovascular activity, and their neural activity (Cummings, Zahn-Waxler, & Radke-Yarrow, 1981; Graham, Fisher, & Pfeifer, 2013; Moore, 2009; O'Hearn, Margolin, & John, 1997). But infants and toddlers are quite limited in being able to understand this source of stress. With prolonged exposure to conflict and without compensatory caregiving, very young children's capacity to be responsive can be altered permanently as it is for young children exposed to other adversities.

Children whose first encounters with interpartner conflict occur after early childhood have more emotional understanding tools to draw on, but still have limits on that understanding depending on their phase of emotional development. Their understanding could make them more acutely aware of the problems they witness and lead them to engage in reasoning that is not a match of the problems. Preschool-age children exposed to parental shouting, yelling, screaming, swearing, and cursing had lower self-esteem and more conduct problems *if* they had better rather than poorer emotion knowledge skill (Berzenski & Yates, 2013). Older children (8- to 12-year-olds) are able to describe their strong emotional reactions to interpartner conflict, describing anger, sadness, fear, and self-blame when they witness interpartner conflict (DeBoard-Lucas, Fosco, Raynor, & Grych, 2010; Davis, Hops, Alpert, & Sheeber, 1998; Shelton & Harold, 2007). Yet they are less inclined to express these negative emotions and more inclined to try to intervene (Cummings, Pellegrini, Notarius, & Cummings, 1989; Cummings, Zahn, Waxler, & Radke-Yarrow, 1984). The way that they interpret and reason about the conflict they perceive mediates the effects of exposure on the child's emotional functioning and psychological adjustment (Fosco & Grych, 2007; Grych & Fincham, 1990). If they feel to blame, this influences their own self-evaluation, diminishing their sense of efficacy and worth. If they feel more fearful in situations involving unresolved conflict between their caregivers, more inclined to act if they feel anger, and more avoidant if they feel sad and helpless (Koss et al., 2011).

By adolescence, the effects of exposure to interpartner conflict are more mixed. Some but not all youth feel personally threatened by the conflict, they feel more devalued in their own eyes, and they also report self-blame (Siffert, Schwarz, & Stutz, 2012). However, older children and youth are better able to accept and adjust to conflict, particularly if the conflict does not spill over into unsupportive caregiver-child relations (O'Donnell, Moreau, Cardemil, & Pollastri, 2010). As noted, there is considerable research linking poorer emotion understanding in adolescents with elevated symptoms or psychiatric diagnoses. The leading view is that these youth have strong emotional reactions that have interfered with their ability to regulate emotion, which then interferes with processing emotion information; their strong emotional reactions could be related to combined influences of adversity exposure, including the new challenges faced in adolescence, as well as dispositional vulnerabilities that may be emerging. The view that these youth have strong reactions that are difficult to regulate that compromise their emotion processing is supported by neuroimaging research that links poor emotion recognition to heightened amygdala activity, diminished prefrontal region activity, or both.

Yet more recent work suggests caution, indicating much more needs to be known about the complex ways the brain functions and changes during adolescence (e.g., Crone & Dahl, 2012; Perlman et al., 2013). For instance, Perlman and colleagues recently found that brain regions in addition to the amygdala may be involved; they showed that youth with bipolar spectrum disorder show less activation in the fusiform gyrus, which is involved in general face processing, when processing facial emotion compared with asymptomatic youth (Perlman et al., 2013). In terms of adolescent development, Crone and Dahl (2012) contend that the view that immature prefrontal activity or connectivity may underlie emotional impulsiveness and risk-taking behavior in adolescents may be simplistic; they propose that there is variability, or flexibility, in the degree to which adolescents *apply* emotion understanding when making decisions and that this depends on their motivational stance. That is, the decisions about behavior depend on which appraisal/action readiness tendencies are active in a particular situation. Thus, the adolescent's gains in cognitive flexibility enable them to switch priorities more readily than younger children, which has advantages (increased creativity) and potential risk (poor, impulsive

choices such as risky behaviors). As Crone and Dahl further note, many studies linking adolescent cognitive development, including importantly emotion understanding, are limited by the fact that they do not assess emotion understanding in realistic, personally meaningful contexts (but see Guyer et al., 2008) and rarely address how differences in adolescence *develop*.

In considering risk for emotional dysfunction in the context of emotion understanding, we must consider one additional aspect: emotional awareness. There is very little normative research on the development of emotional awareness but it is a prominent clinical concern (e.g., Flynn & Rudolph, 2010). Many evidence-based interventions for addressing emotional problems, such as anger and anxiety management protocols, require clients to identify initial activation of those emotions and to engage in remedial actions or thoughts early in the emotion generation process. Yet many children and youth lament or protest that they do not recognize their reactions until they are intense. Recently, a client bemoaned the fact that he had no yellow zone, referring to the aid of a traffic light metaphor for assessing his anger intensity.

Emotional awareness involves recognition of bodily changes, bodily action, and different types and blends of subjective feelings (Lane & Schwartz, 1987). From the functional perspective, this awareness occurs when appraisal/action readiness patterns reach conscious awareness and are experienced as subjective feelings that a person may also be able to describe. Its early origins are thought to be cultivated by social interaction with partners who attend to, mirror, and comment on a young child's emotions as described earlier (Bigelow et al., 2010). Over time, emotional exchanges foster the ability to represent emotions in childhood and increasingly apply emotion understanding in self-reflective fashion as children transition from childhood to adolescence (Fonagy, 2002; Gergely & Watson, 1999; Izard & Harris, 1995).

Only one study assessed emotional awareness in young children (Warren & Stifter, 2008), investigating concordance between preschool-age children's verbal descriptions of their reactions and their nonverbal expressions during a long delay to receive a reward and relating these to mother–child communication about a different problem. Children reported being significantly more angry than they appeared but with concordance for sadness and happiness, consistent with previous normative research. A study of 8-year-olds losing a game used latent profile analysis to create groups based on children's self-reported anger, observed anger, and skin conductance (Smith, Hubbard, & Laurenceau, 2011). Of the five resulting groups, the modal profile was reporting high levels of anger but displaying low levels and modest physiological reactivity. A small group was defined by children who reported little anger but who displayed moderate anger and arousal. Each of these studies provides quasi-naturalistic situations that may be useful in studying the early development of emotion awareness.

More often, emotional awareness is studied in adolescence. Emotional awareness plays a role in adolescent friendship formation and maintenance, in increasing sense of well-being over time, and in better mental health outcomes (Ciarrocchi, Kashdan, Leeson, Heaven, & Jordan, 2011; Hamilton, Hamlat, Stange, Abramson, & Alloy, 2014; Roswell, Ciarrocchi, Heaven, & Deane, 2014). In this approach, however, emotional awareness is largely conceptualized as a stable trait. If emotion is best conceptualized as a dynamic process then it is likely that there is intra-individual variability in emotional awareness as situations vary and unfold. For example, does an individual's emotional awareness fluctuate based on the level of significance of circumstances? An approach that might reveal intraindividual variability as well as take into account the context specificity of appraisal/action readiness patterns in naturally occurring situations is ecological momentary assessment (EMA), which has been used to study children's and adolescents' emotions (Dunton et al., 2014; Silk, Steinberg, & Morris, 2003).

In sum, the development of emotional awareness and increasingly sophisticated emotion understanding emerge from neurological maturation, cognitive development, increasing opportunities and variety of social experiences, and accumulating emotional experiences. At each point in the development of emotion understanding there are attendant vulnerabilities, first in terms of perceptual sensitivities and limited ways to cope with what is perceived. Consider the plight of a 4-year-old experiencing sexual abuse by a parent; the child is likely to be flooded with multiple emotions, not just during sexual acts but also the strain of wondering when the next threat will come. Given the child's lack of options for coping, it may be adaptive for perceptual sensitivity to be keen but for further processing to be truncated, which may be why sexually abused children are more likely to avoid emotional experience (Shenk, Putnam, Rausch, Peugh, & Noll, 2014) and to engage in dissociation (Cole & Putnam, 1992). Clearly, the young child's ability to be sensitive to and begin to understand the meanings of the emotional environment are a competency but also a risk factor (Pechtel & Pizzagalli, 2011; Tronick & Beeghly, 2011).

By middle childhood, children can apply greater emotion understanding to the adversities they face but they may nonetheless feel responsible, experiencing self-blame, as well as feeling unable to resolve problems despite feeling they should. By adolescence, a time when youth experience increases in emotional reactivity, develop more complex, abstract, stable and stylized emotion reasoning, and yet face myriad new challenges, including changing academic and social demands and pubertal changes (Abela & Hankin, 2008; Pons & Harris, 2005; Steinberg, 2005), they are better equipped to deal with adversity. However, the advantages of their cognitive growth are coupled with risks, specifically the emergence and stabilization of thought patterns that can increase and sustain emotional reactions without leading to effective problem solving. These include development of capacities for excessive worry, perfectionism, rumination, and hopelessness, hallmarks of psychopathology, particular clinical anxiety and depression (e.g., Abela & Hankin, 2011; Burwell & Shirk, 2007; Damian, Stoeber, Negru, & Băban, 2013; Fowler & Szabó, 2013). These are regarded as ineffective ways to regulate emotion.

Typical Development of Emotion Regulation

The third and final domain of emotional competence is *emotion regulation*; a large corpus of work implicates emotion regulation in the development of children's social and academic competence and their mental health as well as in a wide range of health problems, ranging from situational noncompliance with medical procedures to obesity and psychopathology. The importance of being able to manage one's emotions for each of the outcomes seems self-evident, yet from a scientific perspective emotion regulation is often studied without being defined, using diverse methods, and inferred solely on the degree to which research participants appear or describe themselves as emotionally negative, without assessment of regulatory processes and their relation to that emotion (Cole et al., 2004). The wide variety of approaches and findings reinforces how important effective emotion regulation is, but the imprecision becomes increasingly problematic, preventing progress from the historical view of emotions as discrete events and toward unifying process models.

From the functional perspective, emotion and emotion regulation are part and parcel of the same fundamental process—appraisal/action readiness tendencies that define emotion are inherently regulatory (Campos et al., 2004). Changes in appraisal/action readiness (i.e., emotions) reflect changes in a person's perception of the environment in terms of the person's well-being and attendant orienting of cognitive and behavioral systems, such as narrowing or broadening attention or motivating action. Moreover, the functional perspective on emotional *development*, which emerged in the context of infancy research, defines emotion as relational, drawing attention to the dynamic, context-defined, interpersonal processes involved in infant emotion. As reviewed previously in the section on emotion expressiveness, infancy research clearly demonstrates that (1) partners influence each other's emotions in communicative exchanges, (2) infants perceive and use emotional information to organize their action, and (3) infants initiate behaviors that have the potential to alter their relation to the environment (i.e., emotion). If emotion comprises change in appraisal/action readiness, then emotion regulation involves anticipation, modification, or terminations of those changes (Cole et al., 2004). The dynamic nature of emotion processing must be conceptualized in terms of change and not static events.

A second source of confusion arises when discussions equate different levels of analysis. As neuroscience has begun to document the functioning of the central nervous system, it becomes evident that the brain is a regulatory system, sets of neural networks that coordinate in different ways to afford emergent psychological capacities (Bullmore & Sporns, 2012). This characterization of the brain is consistent with a dynamic, systemic view of emotional functioning but at the same time neural network functioning should not be equated with behavioral processes. Emotion regulation as an aspect of competence functioning cannot be reduced to physiological activity. The nature of neural circuitry in personally meaningful contexts remains to be discovered. Nonetheless, this level of analysis is distinctly different from inferences drawn on the basis of observations of infant or child interactions, the basis of judgments regarding emotional competence.

In terms of developmental pathways to emotional competence, two dominant views of emotion regulation—self-regulation and coregulation—involve *behavioral* outcomes of emotional processes. The functional perspective helps us to interpret appraisal/action readiness patterns as indicative of a person's relation to circumstances in terms of their goals for well-being and to infer changes in those motivational stances from behaviors. At the behavioral level, competent emotion regulation is defined by self-regulation of emotional reactions, both in interpersonal and solitary situations, and coregulation in interactions, in ways that are effective (achieve goals in the specific context) and appropriate (conform to the immediate situational constraints and to

sociocultural standards) (Denham, 2006; Halberstadt et al., 2001; Saarni, 1999). Furthermore, competent self-regulation of emotion entails self-initiated efforts to anticipate to anticipate appraisal/action readiness patterns, to modify appraisal/action readiness patterns (intrapersonal processes, related to emotion-focused coping), and to modify potentiated action readiness, that is, actions motivated by appraisal/action readiness (Gross & Thompson, 2007; Thompson, 1994). The development of emotional competence requires learning to use either intrapersonal or interpersonal mechanisms to engage in effective, flexible, appropriate (age-, sociocultural- and situational-) emotional behavior that achieves, maintains, or regains goals for well-being. For the purposes of this chapter the focus is on the typical development of intrapersonal and interpersonal strategies for emotion regulation and the conditions under which they are associated with emotional dysfunction.

The role of the prenatal period in the development of emotion regulation is largely unknown. Yet, as noted, the available facts support the assumption that genetically influenced neurophysiological systems that are needed for the capacity to be emotional develop in utero and in the context of interaction with the prenatal environment. These systems are so fundamental that they must play a role in the development of emotion regulation as they do with all aspects of emotional competence. The available data, reviewed earlier, further suggest that risk in the prenatal period may compromise the later development of emotion regulation. Specifically, maternal stress and anxiety, probably via the release of cortisol from maternal hypothalamic-pituitary-adrenal (HPA) axis activity, is related to more intense infant sensitivity to some or all stimuli, often observed as infant fussiness, reactivity, or negativity (Sandman & Davis, 2010). Such sensitivity is not in itself evidence of ineffective emotion regulation, but the view is that this sensitivity may hinder infants' rudimentary abilities to recover from distressed states, such that prenatal stress is also associated with newborns being less likely to regain calmness on their own and being less easily consoled (Davis, Glynn, Waffarn, & Sandman, 2011) and 6-month-olds have fewer periods of self-quieting (Bolten et al., 2013).

These findings can be incorporated into the vertical-integrative neurobiological model offered by Geva and Feldman (2008) to characterize the complex origins of emotion regulation. The ability to self-initiate executive control of appraisal/action readiness changes in effective, appropriate ways requires the child to develop neural networks that connect prefrontal cortical regions, which are assumed to support executive or top-down regulatory processes, to limbic regions that support appraisal/action readiness. Geva and Feldman assert that this neural circuitry is predicated on earlier developing neural networks. During the prenatal period, brainstem circuitry develops and forms the basis for neonatal state regulation, including regulation of capacities like breathing and temperature and then vagal tone and circadian rhythms. With this achieved, the neonate is in a better position to have periods of alertness during which the environment can be engaged. In this way, early in infancy, brainstem-limbic system circuitry develops that supports appraisal/action readiness and attention systems. In this model then, risk posed to fetal development of brainstem-regulated systems can have a cascading effect, compromising later developing systems that evolve from earlier developing circuitry and that underlie self-regulation of emotion. Yet the plasticity of developing neurophysiological systems makes it possible for early risk to be mitigated by responsive caregiving and well-coordinated caregiver–infant interaction. Such interactions can enhance newborns' ability to achieve basic neurophysiological regulation, helping them develop patterns of feeding and sleep–alertness cycles. These early elements of neonate regulation appear to have both genetic and non-shared environmental influences (Wang & Saudino, 2013). Although much remains to be learned about the contributions of the prenatal period to the development of emotion regulation, the prenatal period may contribute to basic elements of both emotion processing and state regulation in neonates.

Individual differences in infants are conceptualized as temperament, that is, constitutional, neurobiologically based predispositions involve reactivity to the environment and self-regulation (Rothbart & Derryberry, 1981). There are many approaches to conceptualizing temperament in terms of its constituent dimensions but all include emotional reactivity (Goldsmith et al., 1987; Shiner et al., 2012). Emotional reactivity, usually referred to as negative emotionality, involves an automatic, prepotent, consistent tendency to react with strong negative responses to stimuli, and can be further distinguished in terms of reactivity to frustration (anger proneness) and reactivity to novelty (fear proneness; Rothbart & Bates, 2006). The prevailing view is that moderate emotional reactivity aids the development of emotion regulation (Calkins, 1994). Microanalyses of infant–caregiver interactions reveal (1) periods in which dyads engage in mutually coordinated emotion exchanges (e.g., cooing and smiling), which elicit and sustain each partner's calm, pleasant demeanor; and (2) periods during which dyads are not coordinated but in which each often

acts to gain the other partner's attention and in which the caregiver acts to repair infant distress (Fogel, 2000; Tronick & Cohn, 1989). Over the first months, typically developing infants and their caregivers become increasingly responsive to each other's specific behaviors, resulting in more effective mutual regulation (Beeghly, Fuertes, Liu, Delonis, & Tronick, 2011; Crockenberg & Smith, 2002; Evans & Porter, 2009; Isabella, Belsky, & von Eye, 1989). These periods provide a basis for developing expectations of sustained, mutually coordinated, emotionally positive exchanges, and expectations of exchanges in which unmet needs can and will be addressed. Establishment of predictable emotional exchanges contributes to the development of the attachment relationship (Beebe et al., 2010; Cohn & Tronick, 1987; deWolff & van IJzendoorn, 1997; Sroufe, 1997).

The development of these effective, patterned exchanges is easier to achieve for dyads in which infants are more readily consolable and caregivers have psychological and social resources that enable them to be reliably attuned to and able to address infant needs. Their establishment is more difficult if infants are highly reactive and harder to soothe or disengaged or unresponsive, or if caregivers lack adequate psychological or social resources for handling caregiving challenges (Costa & Figueirdo, 2012; Lyons-Ruth, Bronfman, & Parsons, 1999; Spangler et al., 1994; van den Boom & Hoeksma, 1994). However, for infants who are reactive or unresponsive, there may be a greater need for predictable, effective caregiver skill, a form of differential susceptibility (Ellis, Boyce, Belsky, Bakermans-Kranenberg, & van IJzendoorn, 2011; Geva & Feldman, 2008). The ability of the infant–caregiver dyad to form a stable, predictable pattern of coregulatory exchanges is widely believed to contribute to the development of children's self-regulation of emotion (Cassidy, 1994; Kim & Kochanska, 2012; Martinez-Torteya et al., 2014).

Although regulation of infant needs is largely achieved through dyadic interactions with caregivers, infants appear to have some limited self-regulatory capacity. That is, they initiate behaviors that briefly interrupt or reduce the intensity of their distress, which may sometimes resolve distress. Specifically, distressed infants engage in behaviors that can have a calming effect, such as sucking on their hands or averting their gaze from the source of distress; they also engage in these behaviors when they are not distressed, and it may be that through conditioning they learn to use them in a self-regulatory manner (Stifter & Braungart, 1995; Rothbart, Ziaie, & O'Boyle, 1992). In addition to these self-directed behaviors, infant emotional expressiveness is regulatory in that it typically captures, sustains, and guides caregiver attention and behavior (Braungart-Rieker, Garwood, Powers, & Notaro, 1998; Cohn & Tronick, 1983; Mangelsdorf et al., 1995; Tronick, 1989). Thus, emotional communication (often referred to as support-seeking), self-soothing, and gaze aversion are the primary self-regulatory behaviors observed in the first, year, with gaze aversion increasing as self-soothing declines (Rothbart et al., 1992).

Infant self-regulation of emotion is often observed using the Still Face Procedure, which capitalizes on established dyadic exchanges (Tronick, Als, Adamson, Wise, & Brazelton, 1978). By age three months, infants perceive changes in caregiver responsiveness and are typically distressed by caregiver unresponsiveness. During the period of the procedure during which caregiver demeanor is unresponsive, infant behaviors are interpreted as regulatory, both those that strive to regain caregiver responsiveness and those that potentially regulate infants' own distress. Between ages 3 through 5 months, during the still period, typically developing infants' behavior changes (Ekas, Lickenbrock, & Braungart-Rieker, 2013). Specifically, although infant distress is stable across this age period, infants' looks at the unresponsive caregiver decrease and their looking away and vigorous movement increases. These short-term longitudinal data suggest that infants have learned to shift attention from a source of distress. Their vigorous movement may reflect appraisal/action readiness to regain the thwarted goal (caregiver responsiveness). During this age period, looking away is usually followed by momentary decreases in distress, suggesting that the frequency of looking away increases but its effectiveness does not. Thus, as they reach the midpoint of the first year, typically developing infants expect caregiver responsiveness, are distressed when it stops, and may learn to use a spontaneous tendency to shift attention from the source of distress in a limited, self-regulatory manner.

More often, in distress circumstances, infant behavior is communicative and coregulatory. Emotion coregulation is observed throughout the life span (Butler & Randall, 2013; Coan, Schaefer, & Davidson, 2006) but unlike adults, infants and very young children have few and limited alternatives for regaining well-being other relying on caregivers. Infant–caregiver interactions not only regulate infant appraisal/action readiness tendencies, but also begin to scaffold infant behaviors that contribute to the later development of child self-regulation of emotion (Kopp & Neufeld, 2003; Sroufe, 1997). The scaffolding of young children's self-regulatory efforts begins in earnest in the toddler years, when children engage in more autonomous behavior and caregivers communicate

standards of behavior. Toddlers have limited ability to modulate their prepotent responses, such as inhibiting approach behavior (happy or angry) or approaching a task they do not wish to do (angry or afraid). Their spontaneous self-regulatory efforts are not regarded as reflecting conscious deliberation and they are of limited effectiveness. For example, gaze aversion is a well-practiced behavior but it still has only temporary immediate effect on anger and no effect on fear during quasi-naturalistic laboratory tasks (Buss & Goldsmith, 1998). Toddlers' self-regulatory attempts are also context-dependent. The degree to which toddlers express anger and engage in regulatory efforts, as well as the relation between emotion and regulatory efforts, depends on the situation, e.g., arm restraint versus snack delay (Calkins & Johnson, 1998; Feldman, Dollberg, & Nadam, 2011).

The spontaneous, brief efforts of very young children to regulate negative emotions are soon strengthened by neurophysiological change and learning from thousands of experiences with emotional events. As Kopp (1989) posited, during the transition from toddlerhood to preschool-age children's self-regulation of distress and negative emotion transforms. Children begin to direct and modulate their behavior in accord with adult standards when adults are present, a form of compliance, with increasing understanding and acceptance of those standards (Feldman & Klein, 2003; Kochanska, Coy, & Murray, 2001). During the third year, they begin to deliberately initiate efforts that alter their distress without adult instruction or even presence, suggesting internalization of rules and self-regulation (Kochanska et al., 2001; Kopp, 1982). Typically developing children as young as three and four years engage in spontaneous expressive control (Cole, 1986; Garner & Power, 1996) in the absence of familiar caregivers. Specifically, when a research assistant gives typically developing preschool-age children an undesirable prize for their work, 3- and 4-year-olds modulate nonverbal expressive indications of disappointment (sadness, anger, disgust). Toddler gaze aversion when reward goals are blocked (e.g., Hill, Degnan, Calkins, & Keane, 2006) appears to develop into preschoolers' deliberate effort to distract themselves in such situations (Rothbart, Sheese, Rueda, & Posner, 2011). It is not clear how aware such young children are of their self-regulatory efforts. Their efforts may be more automatic, unconscious strategies, which would be consistent with evidence that they do not articulate strategies although they recognize effective strategies (Cole, 1986; Cole, Dennis, Smith-Simon, & Cohen, 2009). Spontaneous infant and toddler behaviors that serve regulatory functions may begin as non-deliberate, non-conscious behaviors that may later become available to self-awareness although with repeated practice they may eventually become automatic and non-conscious (Bargh & Williams, 2007).

When trusted caregivers are available, preschool-age children seek support when confronted with blocked or lost goals (e.g., Cole, Tan, Hall, Zhang, Crnic, Blair, & Li, 2011; Eisenberg, Fabes, Bernzweig, Karbon, Poulin, & Hanish, 1993). Initiating support-seeking is a form of self-regulation, aimed at achieving a goal by soliciting another's help. Between toddler and preschool age, angry support-seeking declines and calm support-seeking increases (Cole et al., 2011). Support-seeking reflects the representation that caregivers can be helpful, an interpretation consistent with the fact that children of this age engage in less support seeking when their parents are rated as angry and coercive (Eisenberg et al., 2003). During this age period, when self-regulation is just emerging, caregivers often initiate support and they do so more if they perceive children as more emotionally reactive and less able to employ effective self-regulatory strategies (Perez & Gauvain, 2005).

Most studies of young children's autonomous emotion regulation employ quasi-naturalistic laboratory tasks to evoke anger or fear, and in these contexts the most commonly observed self-regulatory strategy is distraction (Buss & Goldsmith, 1998; Calkins, Gill, Johnson, & Smith, 1999; Calkins & Johnson, 1998; Cole et al., 2011; Day & Smith, 2013; Grolnick, Bridges, & Connell, 1996). As noted, distraction is defined by deliberately shifting attention away from the stimulus to which a child has an emotional response (a threat or a blocked goal) and toward an alternative activity. Individual differences in the use of distraction may reflect individual differences in child dispositional negativity, prior regulatory capacity and parenting practices or style (Hastings & De, 2008; Morris et al., 2011; Perry, Calkins, Nelson, Leerkes, & Marcovitch, 2011; Rothbart, Posner, & Keiras, 2006).

As predicted by Kopp's (1989) developmental framework for self-regulation, and Posner and Rothbart's (2000) developmental framework for executive attention, between 24 and 36 months of age, there is a change in young children's self-initiated distraction. This change appears due to the maturation of an executive attention system, a product of neurological development and experience (Rueda, Rothbart, & Posner, 2005). At 24 months children initiate few distractions, the distractions are brief and, if children had expressed negative emotion, the distraction has a momentary, limited effect on reducing emotion intensity (Buss & Goldsmith, 1998; Cole et al. 2011).

By 36 months, however, children initiate distractions more readily, the distractions are sustained somewhat longer, and they often occur *prior* to anger expression (Cole et al., 2011; Supplee, Skuban, Trentacosta, Shaw, & Stoltz, 2011). By 36 months, children are better able to become absorbed in alternative activities once they distract themselves, which appears to help them tolerate a potential frustration and forestalls getting openly angry (Cole et al., 2011). Although there are still limits on preschool-age children's strategy effectiveness, by age 4 and 5 years, distraction has become a more practiced regulatory strategy and typically developing children use it successfully in contrast to same-aged children with behavior problems (Feng, Shaw, & Moilanen, 2011; Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002; Jahromi, Meek, & Ober-Reynolds, 2012; Silk, Shaw, Skuban, Oland, & Kovacs, 2006). Thus, by the time children reach kindergarten age, they can tolerate commonplace experiences with novelty, frustration and disappointment by forestalling and modulating emotionally negative reactions (Blair, 2002; Denham, Bassett, Sirotkin, & Zinsser, 2013; Eisenberg, Valiente, & Eggum, 2010).

There is far less research on the development of emotion regulation in middle childhood (Zeman, Klimes-Dougan, Cassano, & Adrian, 2007). There appear to be two main reasons for this neglect. First, there is considerable interest in adolescence due to its being a period of rapid developmental changes in which youth experience significant new demands in their academic and social lives, emotional intensity increases, and there appears to be significant reorganization at the neural level (Casey, Getz, & Galvan, 2008; Casey, Jones, & Somerville, 2011; Dahl, 2004; Larson, Moneta, Richards, & Wilson, 2002; Peper & Dahl, 2013). Second, research on emotion regulation in school-age children (and adolescents) tends to be dominated by contrasts between individuals with and without symptoms or risk factors, reflecting perhaps the expectations that typically developing children should be fairly well-regulated by school age.

Nonetheless, there is growth in emotion regulation in middle childhood in the form of increased and more varied experiences in which emotion regulation is needed, in their greater autonomy and reduced need for close adult supervision, more opportunities to learn about and use strategies, and an increased repertoire of regulatory strategies. Between 8 to 12 years, children continue to seek support and employ distraction but they are also have more opportunity and are better equipped to engage in direct problem solving, acceptance of problem situations, and altering appraisals (cognitive restructuring), as well as intentionally avoiding or withdrawing from situations (Garber, Braafladt, & Zeman, 1991; Garber, Braafladt, & Weiss, 1995; Tan et al., 2012; Zimmer-Gembeck & Skinner, 2011; Zimmermann & Iwanski, 2014).

During adolescence youth appear to develop more efficient cortical functioning in regions associated with regulatory processes (Lamm & Lewis, 2010; Perlman & Pelphrey, 2010), which may explain their increasing ability to intentionally inhibit prepotent responses such as emotional reactions (Eisenberg et al., 2005). They probably also gain greater flexibility and effectiveness of their strategies and more autonomy and confidence in using them. Cognitive advances in symbolic, abstract reasoning and self-reflection are thought to contribute to the skill with which adolescents select among possible strategies, choosing those that are better suited to situations and switching strategies when one is ineffective (Flynn & Rudolph, 2010; Holodynski & Friedlmeier, 2006; Zimmer-Gembeck, Skinner, Morris, & Thomas, 2012).

These conclusions are tempered by a limitation of research on emotion regulation in middle childhood and adolescence, specifically the heavy reliance on self-report data. Self-report instruments inform us of the regulatory efforts of which participants are aware and which they are willing to endorse. These reports have three important limitations, especially for research guided by a functional perspective on emotion: (1) they tend to treat emotion regulation as a trait; (2) they tend to ignore the varied contexts in which emotions, even the same emotions, occur; and (3) they tend to overlook the dynamic, unfolding nature of emotion and emotion regulation. Moreover, when applied to the development of psychopathology, there will be individual differences in the reliability of self-reports of emotion regulation. The reliance on self-report is due in part because it is difficult to observe older children's and adolescents' emotion regulation in naturalistic contexts; they can generate and use intrapersonal strategies, such as reappraisal, as well as more automatic or potentially problematic strategies, such as denial, rumination, wishful thinking, and emotional numbing (Langrock, Compas, Keller, Merchant, & Copeland, 2002; Wadsworth, Raviv, Compas, & Connor-Smith, 2005; Zeman et al., 2007; Zimmermann & Iwanski, 2014). Clearly, a thorough documentation of the development of emotion regulation in middle childhood and adolescence requires a careful, sophisticated approach. Several studies have employed multiple measures to increase inferences about emotion regulation—including self-report, observations or psychophysiological assessment. Moreover, several of these have been successful at increasing the ecological validity

of the data gathered, assessing children's and adolescents' emotion regulation in actual or quasi-naturalistic relational contexts. These include parents and children discussing actual problems or planning an activity, children and adolescents giving a speech or having actual or computer-based (simulated social media) peer interactions, including some that lend themselves to use in the MRI environment (Guyer, McClure-Tone, Shiffrin, Pine, & Nelson, 2009; Klimes-Dougan & Zeman, 2007; Reijntjes et al., 2006; Yap, Schwartz, Bryne, Simmons, & Allen, 2010; Zalewski, Lengua, Wilson, Trancik, & Bazinet, 2011). Focus groups on important topics and experience sampling methods, such as daily diaries and ecological momentary assessment have also been employed to capture children and adolescents' emotions in real-life situations (Dunton et al., 2014; Larson, Raffaelli, Richards, Ham, & Jewell, 1990; Reeves, Nicholl, & Mackenna, 2011; Silk et al., 2003; Tan et al., 2012; Tenenbaum, Varjas, Meyers, & Parris, 2011). Finally, these types of procedures can generate data that can be used to capture individual differences in emotional dynamics (Dagne & Snyder, 2011; Sheeber, Kuppens, Shortt, Katz, & Davis, 2012).

Risk for Emotional Dysfunction Associated with Emotion Regulation

Many studies have associated deficits in emotion regulation to psychopathology in children, adolescents and adults, including a number showing that emotion regulation mediates relations between risk factors and psychopathology outcomes (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; Boden, et al., 2013, on posttraumatic stress disorder; Cavanagh, Quinn, Duncan, Graham, & Balbuena, 2014, on oppositional defiant disorder; Cisler, Olatunji, Feldner, & Forsyth, 2010, on anxiety disorders; Harrison, Sullivan, Tchanturia, & Treasure, 2010, on eating disorders; Joorman & Gotlib, 2010, on depression; Martel, 2009, on attention-deficit disorder; Beauchaine, Gatzke-Kopp, & Mead, 2007, on conduct disorder; Salsman & Linehan, 2012, on borderline personality disorder). Although these studies are quite limited in informing the dynamic, context specific emotion processing in personally meaningful situations relates to psychopathology. Moreover, few have used longitudinal designs that come closer to addressing the question of whether difficulties with emotion regulation are a correlate, causal factor, or consequence of psychopathology. Another challenge to answering these questions is that emotion regulation changes rapidly in early development but most *DSM*-defined diagnoses require clusters of stable symptoms that are rarely observed before middle childhood.

Early childhood longitudinal studies indicate that infants and toddlers who do not yet engage in age-level emotion regulation tend to have stable or worsening symptoms in the preschool years, particularly in the presence of adversities, such as economic disadvantage or caregiving inadequacies (Calkins & Keane, 2009; Crockenberg, Leerkes, & Bárrig, 2008; Gilliom et al., 2002; Halligan et al., 2013; Kochanska & Knaack, 2003). A small number of additional longitudinal studies extend these findings into middle childhood (Blandon, Calkins, Grimm, Keane, & O'Brien, 2010; Eisenberg et al., 1996; Kim & Cicchetti, 2010; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011; Trentacosta & Shaw, 2009).

Generally, the conditions that pose risk to the development of competent emotion regulation include those already discussed: genetically based influences on the development of physiological systems that support emotion processing and stress responding; atypical patterns in caregiver-child contributions to emotional exchanges, which includes the conditions that lead to insecure attachment; and factors that overly tax the self-regulatory capacities of the child or the capacity of caregivers to foster children's development of effective emotion regulation. Here we organize the discussion into two main categories of risk for emotion regulation: child characteristics, namely temperament and attachment security, and environmental characteristics, including inadequacies in caregiving qualities and exposure to adversity, which often includes inadequate caregiving.

Child temperament is generally shown to be a risk factor for the development of emotion regulation. The research has focused largely on the dimension of negative emotionality, although the degree to which it poses risk depends on the other major dimension of temperament, effortful control. Again, negative emotionality refers to automatic, prepotent, and stable tendencies to react with strong negative responses to stimuli, including to perceived obstacles to well-being (reactivity to frustration) and perceived threats to well-being (reactivity to novelty; Rothbart & Bates, 2006). Such non-conscious, spontaneous and intense reactivity reflects neurobiologically based dispositions that are shaped by prenatal and subsequent experience (Davis et al., 2011; Wang & Saudino, 2013). As noted earlier, emotional reactivity poses a challenge for the development of effective emotion regulation because management of strong negative emotions may outpace children's self-regulatory resources as well as those of caregivers trying to aid and teach them (Calkins, 1994). But negative emotionality may not have longer-term negative consequences if children also have a temperamental

disposition toward effortful control (e.g., Eisenberg et al., 2009). Effortful control involves readiness for attentional flexibility and control that can be deployed to interrupt or inhibit prepotent responses such as emotional reactivity (Rothbart & Bates, 2006). That is, very young reactive children who are readily distracted by caregivers when they are highly distressed, and who later in childhood can more exert cognitive resources to shift attention or inhibit action despite their strong appraisal/action readiness should be less at risk for compromised emotion regulation. Indeed, there is considerable overlap in the literature between the concepts of effortful control and emotion regulation. For the purposes of this chapter, temperament is conceptualized as a largely neurobiological, relatively stable set of predispositions and emotion regulation is largely learned, acquired over time, and influenced by multiple factors, including temperamental dispositions.

Emotionally reactive children are less successful at using regulatory strategies (e.g., Morris & Silk, 2001), but their reactivity does not lead to inappropriate actions if they engage in appropriate regulatory strategies (Calkins, Gill, Johnson, & Smith, 1999; Santucci et al., 2008). However, negative emotionality and effortful control are better understood in the context of interpersonal processes (e.g., Raby et al., 2012). The degree to which young children exert effortful control depends on the situation, including who is interacting with them and the ability of those others to encourage child effortful control (Braungart-Rieker, Garwood, & Stifter, 1997; Spinrad et al, 2012; Volling, McElwain, Notaro, & Herera, 2002). Emotionally reactive infants who are less disposed toward effortful control may be especially dependent on caregiving qualities to develop effective emotion regulation (Ellis et al., 2011; Klein Velderman, Bakermans-Kranenburg, Juffer, & van IJzendoorn, 2006).

One pathway by which high negative emotionality and low effortful control pose risk for the development of effective emotion regulation is through the demands placed on infant–caregiver interactions, including contributions to the establishment of relationship patterns, including the attachment relationship (Lorber & Egeland, 2011; van IJzendoorn & Bakermans-Kranenburg, 2012). If infants and caregivers have difficulty establishing predictable emotion exchanges in which infant distress is reliably addressed by sensitive caregiving, it is more difficult for infants to achieve secure attachment (Beebe et al., 2011; Leerkes et al., 2011). Individual differences in attachment status are defined by different styles of emotional organization when children's need to regain a sense of well-being overtakes their interest in exploring the world, and attachment status establishes a working model of distress relief that contributes to the development of emotion regulation (Cassidy, 1994). Insecure attachment is associated with both reduced spontaneous, authentic support seeking (insecure-avoidant style) and with intense, exaggerated emotional (insecure-resistant style; Cassidy, 1994). It is important to recall that attachment security, which is established in the first year, influences the developing organization of biobehavioral stress systems that affect appraisal/action readiness and regulation (Crockett, Holmes, Granger, & Lyons-Ruth, 2013; Feldman, 2012; Hill-Soderlund et al., 2008; Lujik et al., 2010; Spangler & Grossmann, 1993) and their neural concomitants (DeWall et al., 2012; Eisenberger et al., 2011; Karremans, Heslenfeld, van Dillen, & Van Lange, 2011).

Evidence supports the view that attachment status influences behavioral self-regulation of emotion. In delay and compliance tasks used to assess emotion regulation, insecure-resistant infants communicate more angry distress and engage in fewer self-regulatory attempts than secure infants, and their mothers appear to interfere more with infant self-regulatory attempts (Berlin & Cassidy, 2003; Diener, Mangelsdorf, McHale, & Frosch, 2002; Leerkes & Wong, 2012; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Kok et al., 2013; Vondra, Shaw, Swearingen, Cohen, & Owens, 2001). Resistant attachment style also predicts preschool-age behavior problems, many of which involve poorly regulated anger (Crittenden, 1992; Vondra et al., 2001). Insecure-avoidant and secure infants appear less overtly dissimilar. As Cassidy (1994) suggested, avoidant infants can appear emotionally self-contained despite physiological indices of distress, and engage in more self-soothing and toy exploration, which is understandable given their mothers are less engaged (Braungart & Stifter, 1991; Leerkes & Wong, 2012; Nachmias et al., 1996; Spangler & Grossmann, 1993). Whereas resistant and avoidant attachment represent risk, they are nonetheless regarded as organized adaptations to inadequate caregiving; disorganized attachment is not by definition (Hesse & Main, 1999). Disorganized attachment appears to reflect conditions that would lead infants to have difficulty adopting a patterned appraisal/action readiness stance. Their emotional confusion results from starkly mixed presentations by their mothers who can appear both frightening to or frightened by their infants (Hesse & Main, 1999), a pattern that is more emotionally abusive than inadequate. Thus, disorganized infants are not emotionally spontaneous, intense, or self-contained, but appear dazed, anxious, afraid, and confused and without effective regulatory strategies. These patterns may underlie the

tendencies of individuals with abuse histories to dissociate from emotional experience and to act in highly controlling ways (Hertsgaard, Gunnar, Erickson, & Nachmias, 1995; Liotti, 1999; Main & Solomon, 1990; Solomon, George, & De Jong, 1995; Spangler & Grossmann, 1993).

The relative and conjoint contributions of temperament and attachment status to risk for ineffective emotion regulation have not been studied sufficiently (Lickenbrock et al., 2013). Ideally, studies examining these child characteristics would also include other contextual factors that would predict pathways to emotion regulation. One study employed a multiple domain model to predict different types of symptoms in preschool-age children with oppositional defiant disorder (Lavigne, Gouze, Hopkins, Bryant, & LeBailly, 2012). The findings are limited by the fact that all predictors and outcomes were measured concurrently, mainly with questionnaires, for a single age group; nonetheless, the evidence suggested complex pathways in which child effortful control, negative emotionality, and attachment insecurity intersect with contextual factors such as parental psychopathology and stress.

Attachment status appears to be relatively stable from infancy to adulthood, which is attributed to the early establishment of the internal working model (Fraley, 2002; Hamilton, 2000; van IJzendoorn, 1995; Waters, Merrick, Treboux, Crowell, & Albersheim, 2000; Weinfield, Sroufe, & Egeland, 2000; Zimmermann, 1999). Individuals with a history of insecure-resistant attachment are less able to regulate anger in interactions with persons other than their infant caregivers, as seen in studies of preschool-age children interacting with their younger siblings or foster parents and of adolescents and adults interacting with parents, friends and romantic partners (Allen & Miga, 2010; Crowell et al., 2014; Dozier, 2003; Kobak, Cole, Ferenz-Gillies, Fleming, & Gamble, 1993; Pietromonaco, Uchino, & Schetter, 2013; Volling, 2010; Zimmerman, Maier, Winter, & Grossmann, 2001). Although insecure attachment is a risk factor for the development of psychopathology, it is not in and of itself psychopathology. The question has been raised why insecure attachment is so common if it is places a person at risk and the question posed whether a diversity of attachment styles in a social group may advantage group survival better than a group composed only of securely attached individuals (Ein-Dor, Mikulincer, Doron, & Shaver, 2010). In other words, insecure attachment, an adaptation to inconsistent, unavailable, or unpredictable primary relationships may have advantages in the face of adversity. Moreover, it is likely that attachment status poses risk for emotional dysfunction in the presence of other risk factors.

When discontinuity in attachment status occurs, it is almost always associated with a specific form of adversity, that is, interpersonal problems directed at or occurring in the presence of children, such as family instability, interpartner conflict, or child abuse (Belsky, Campbell, Cohn, & Moore, 1996; Schneider-Rosen, Braunwals, Carlson, & Cicchetti, 1985; Waters, Weinfield, & Hamilton, 2000). As noted, child characteristics affecting emotion regulation must be understood in the context of multiple developmental influences, including the challenges with which child and family are dealing. The full range of environmental factors that can challenge children's capacity to regulate emotion is wide. They can include factors that stress and strain family resources (e.g., poverty) and interact with characteristics of caregivers and their relationships that bear down on parenting. When studies consider multiple influences, it generally appears that the effects of adversity, broadly defined, on child emotion regulation depend on caregiving qualities.

For example, poverty is a condition that captures a variety of circumstances that pose risk to children and their caregivers. Growing up in poverty is associated with less skillful child self-regulation, as indexed by both biological and behavioral indices, some of which include aspects of emotion regulation (Evans & English, 2002; Evans, Gonnella, Macynyszyn, Gentile, & Salpekar, 2005; Evans & Rosenbaum, 2008; Keenan, Shaw, Walsh, Delliquadri, & Giovanelli, 1997; West, Sweeting, Young, & Kelly, 2010; Vernon-Feagans, 2013). The effects of poverty may be most deleterious when present in infancy (e.g., Mistry, Benner, Biesanz, Clark, & Howes, 2010), but the evidence of these effects on behavioral indices of emotion regulation may take time to appear. For example, there are more significant effects on children's spontaneous self-initiated regulatory efforts after age 30 months, an age when self-regulation typically emerges (Caughy, Mills, Owen, & Hurst, 2013). However, poverty can be associated with many factors that can compromise the development of effective emotion regulation, including exposure to discrimination and racism, community violence, exposure to uncontrollable noise, and inadequate nutrition (Aber, Jones, & Cohen, 2000; Evans & Kim, 2013; Gibbons et al., 2012; Lozoff et al., 2008). In the presence of warm, supportive parenting, poor children and adolescents appear to develop effective regulation of anger and sadness (Brody et al., 2014; Burchinal, Roberts, Hooper, & Zeisel, 2000; Garner, Mahatmya, Moses, & Bolt, 2014; Garner & Spears, 2000; McCoy & Raver, 2011).

Thus, the caregiving circumstances that can interfere with the development of effective emotion regulation

warrant more detailed attention. These are proximal relationships that are likely to be potent influences on children's emotion regulation development, particularly in early childhood, and that can moderate the effects of other risk factors. As noted, caregiving quality may be particularly critical for children who bring their own vulnerabilities to the interactions (Ellis et al., 2011). The caregiving circumstances that are known to be associated with children's difficulties with emotion regulation include abusive and neglectful caregiving, parental psychopathology, and impaired coparenting relationships (e.g., due to marital conflict) that children experience or spills over to parenting (Goodman et al., 2011; Kim-Spoon, Cicchetti, & Rogosch, 2013; McCrory, DeBrito, & Viding, 2012; Schudlich, White, Fleischhauer, & Fitzgerald, 2011; Sturge-Apple, Davies, Cicchetti, & Fittoria, 2014; Suveg, Shaffer, Morelen, & Thomassin, 2011). Caregiving problems can confer risk on emotion regulation in three interrelated ways that have been identified as mechanisms of emotion socialization: as emotional partners, as models of emotion regulation, and as scaffolds for the development of self-regulation skills (Eisenberg et al., 1998; Morris et al., 2011).

When caregivers interact with their children, they implicitly convey information in all of these ways, and on some occasions they explicitly attempt to model and guide child emotion regulation. As emotional partners, caregivers communicate emotion and communicate about emotion. In many of the caregiving contexts associated with children's emotional dysregulation, caregivers communicate negative emotions. These often are processed by children as aversive stimuli to which children react and with which they must cope. Studies frequently report that mothers with different forms of psychopathology, such as mood, anxiety, or personality disorders react to their infants' and children's negative emotions with autonomic, neural, and behavioral responses, that is, appraisal/action readiness tendencies, indicative their own personal goals for well-being have been threatened or blocked, and that these caregivers do not readily recover from such reactions (Buckholdt, Parra, & Jobe-Shields, 2014; Burkhouse, Uhrlass, Stone, Knopik, & Gibb, 2012; Connell, Hughes-Scalise, Klostermann, & Azem, 2011; Creswell, Apetroaia, Murray, & Cooper, 2013; Hobson, Patrick, Crandell, Garcia-Perez, & Lee, 2005; Hudson, Comer, & Kendall, 2008; Kiel, Gratz, Moore, Latzman, & Tull, 2011; Oppenheimer, Measelle, Laurent, & Ablow, 2013; Silk et al., 2011; Shipman et al., 2007). These immediate emotional reactions suggest that the caregivers' appraisal/action readiness stances are oriented toward the caregivers' own well-being rather than focused on child needs for well-being. That is, the negative emotions on are on behalf of the caregiver, not the child. If caregivers cannot recover readily, these appraisal/action readiness stances are likely to interfere with their ability to sensitively respond to children's needs (Leerkes, 2010). Although caregivers' negative emotions may ready them to withdraw from caregiving, this is difficult to carry out when caring for young children; mothers who are emotionally competent manage to regulate this type of action readiness and to engage effectively with their children (Hajal, 2014). In situations in which children are challenging, caregivers may initially or subsequently appraise the situation as blocking their goals and be ready to approach; in these moments, caregivers are angry. This may be an explanation for why irritability is a common feature of emotional exchanges between stressed parents and their children.

Because emotions are a rapid radar and response system for meeting one's goals for well-being, it takes skill to be aware of such rapid reactions. Caregivers who are a source of adversity exposure to their children are often less emotionally aware, and they are therefore less mindful of their behavior with their children and less skilled at using emotional episodes as opportunities to learn about emotion regulation. Stressed caregivers are more likely to ignore or dismiss children's negative emotions, missing the messages children's emotions communicate and missing opportunities to validate the child's perception of need and to teach the child alternative coping (Katz, Maliken, & Stettler, 2012; Yap, Allen, & Ladouceur, 2008). Although stressed caregivers can be dismissive and invalidating of children's emotions, they can also be overly reactive or focused on children's negative emotions, and these reactions may model or suggest ineffective emotion regulation. Lower emotional awareness among mothers with depressive symptoms was associated with mothers suggesting cognitive or behavioral avoidance as ways to cope with negative emotion rather than direct problem solving (Monti, Rudolph, & Abaied, 2014). A longitudinal study found that mothers' dwelling on their adolescent children's negative emotions in response to a frustrating task contributed subsequently to youths' tendency to ruminate (Cox, Mezulis, & Hyde, 2010).

Recalling that appraisal/action readiness patterns often unfold rapidly in micro-momentary fashion, they influence interactions with children without caregiver awareness. Yet children, particularly very young children, are sensitive to caregivers' emotions. The evidence indicates that when caregivers communicate anger (irritability, hostility, criticism), it is often perceived as threatening to children, which

requires children to cope with and adapt to caregivers as aversive stimuli. Neurobiological and behavioral evidence suggests that children's initial responses to parental anger reflect appraisal/action readiness patterns involving fear, anxiety, or sadness (Davies, Cicchetti, & Martin, 2012; Shackman et al., 2007; Snyder, Brockman, & Stoolmiller, 2012), which again often occur outside of children's awareness. Caregiver emotional intensity and difficulty regulating emotion may lead some children to blunt their emotional experiences, which are not effective in eliciting adequate care, and which may contribute to problems with emotional awareness or emotional avoidance in later years (Gratz et al., 2014).

When these negative emotions are not well-regulated, caregivers and children may subsequently shift appraisal/action readiness, specifically to the appraisal that goals are blocked and renewed or intensified effort to regain the goal. Such emotional unfolding is often missed in studies of dyadic interaction but can be captured by dynamic analysis of the temporal flow of biological and behavioral indices of appraisal/action readiness (Dagne & Snyder, 2011; Sheeber et al., 2012; Snyder et al., 2012). Throughout the temporal sequences of interactions, emotional partners can shift appraisal/action readiness stances. The reactions described likely occur among typical caregivers but they are able to regulate these reactions (Hajal, 2014). However, this is more challenging with children whose behavior is disruptive or unresponsive or who are more emotionally reactive but less able to regulate themselves; these children are also more likely to be quite dependent on caregiving quality to regain emotional control and learn emotion regulation skills in the course of interactions (Cartwright et al., 2011; Cole et al., 2003; Gibb, Uhrlass, Grassia, Benas, & McGeary, 2009). The ability of caregivers to maintain or recover sensitive, supportive responsiveness with children provides opportunity to help children learn emotion regulation emotion even in the presence of family adversity (Howell, Graham-Bermann, Czyz, & Lilly, 2010). The availability of alternative adequate caregiving early in life, e.g., foster or adoptive parents, appears to mitigate pervasive, deleterious biobehavioral consequences of early exposure to adversity on regulatory processes (e.g., Almas et al., 2012; Dozier, Zeanah, & Bernard, 2013; Lipscomb, Leve, Shaw, Neiderhiser, Scaramella, Ge et al., 2012; Oosterman, DeSchipper, Fisher, Dozier, & Schuengel, 2010). As noted, this is critically important as circumstances in which caregiver behavior taxes very young children's ability to regulate emotion are likely to have pervasive, deleterious effects at all levels of functioning, including the emotion neurophysiology (e.g., Graham et al., 2013; Tottenham et al., 2010). For typically developing children whose caregivers' problems emerge later in the children's development, children's existing ability to engage in effective emotion regulation may buffer the links between adversity exposure and risk for psychopathology (e.g., Kim & Cicchetti, 2010). By early school age, children have more autonomy in regulating their emotional reactions, which may help them cope effectively, but they are still sensitive to and depend on caregiver guidance to deal with situations that challenge their self-regulation.

In sum, infant temperament, particularly effortful control, and attachment security appear to play a role but the robustness of their contributions will only be fully known when these child characteristics are studied in a multidomain longitudinal design. Exposure to adversity, particularly in the absence of adequately responsive caregiving, appears to have profound, enduring deleterious effects on the development of the neurobiological systems that support emotion regulation and on behavioral skill development. As the capacity for effective emotion regulation is established and flourishes in its range, flexibility and sophistication, older children and youth nonetheless face many new challenges; their bodies are changing in ways that are not always anticipated and valued, academic demands increase, peer pressures become more powerful, moral dilemmas that accompany greater autonomy increase and unpleasant and abusive interactions (e.g., bullying, sexual affronts) occur with more frequency, and decisions about the future begin to bear down. As they transition from middle school to high school age, there are increased expectations that youth should handle their autonomy and opportunities in well-regulated ways, although this period is associated with increased emotional intensity (e.g., Larson et al., 1990; Steinberg, 2005). During this same extended period of development, as noted above, the prevalence of psychopathology increases. Youth are increasingly exposed to stressed peers and to risky emotion regulation strategies. Adolescents engage in emotion regulatory strategies that they perceive as providing short-term relief but that increase their risk, including disordered eating behaviors, self-harm behaviors, and alcohol and drug use, strategies often used by youth who are overwhelmed by or disconnected from their emotions and who are unaware of or feel unable to engage in direct problem-solving strategies (Adrian, Zeman, Erdley, Lisa, & Sim, 2011; Goldstein, Vilhena-Churchill, Stewart, & Wekerle, 2012; McMahon et al., 2013; Wong et al., 2013).

Furthermore, in addition to being prepared to express emotion, which serves to engage caregivers in infant

emotion regulation, infants engage in rudimentary, limited self-initiated behaviors—self-soothing and gaze aversion—that serve as temporary regulators of infant state. Infant temperament, notably higher emotional reactivity and lower soothability, influence how easily infants and caregivers achieve regulate distress. When dyadic regulation of infant emotion falters in establishing a consistent, predictable pattern of distress relief, an insecure infant attachment is more likely. Attachment status involves mental representations about distress relief that organize infant behavior in the context of separation from caregivers and that appear to constrain the development of self-regulation of emotion. Of course, all of these developmental steps take place in interpersonal context. Exposure to adversity in early childhood compromises biological systems for stress regulation as well as behavioral skill development in self-regulation. These can have profound effects, particularly if that adversity involves deficiencies in caregiving. Quality caregiving can mitigate risk factors, capitalizing on the plasticity of early developing self-regulatory processes, but when caregiving is unresponsive or threatening, the risks in early childhood are considerable. For children who have navigated early childhood without significant risk, later emerging exposure to adversity may nonetheless overtax their self-regulatory capacities. Over the course of middle childhood and adolescence, there is likely growth in the range, flexibility, and sophistication but also the possibility of developing ineffective regulatory strategies such as experiential avoidance, passive aggressiveness, and rumination. The rapid and substantial biological, psychological, and social changes of adolescence are associated with greater resources, both intrapersonal and interpersonal, for regulating emotion but also with many new challenges and more frequent and intense emotion. Thus at each stage of development there are new emotion regulation skills and new challenges to be encountered and the development of effective emotion regulation is thought to rely in large part on strong, supportive relationships with at least some individuals, both in the family and in larger social networks. However, we lack multidomain longitudinal models that assess the cascading influences of risks associated with emotion regulation that chart the various developmental pathways to ineffective emotion regulation.

CONCLUSION AND FUTURE DIRECTIONS

The functionalist perspective on emotional development offers unique insights into the nature of emotion and emotional competence, insights that can advance the clinical relevance of research on the role of emotion in the development of psychopathology. The functional perspective conceptualizes emotion as a dynamic, context-sensitive, relational process by which individuals, regardless of their mental health status, appraise and prepare to act on the environment in relation their individual goals for well-being. Unless children have gross neurological impairments, they have the capacity to be emotional and they adapt to their circumstances. Generally then, individual differences do not rob children of the ability to function emotionally. Rather it is in the relational process, in how children and youth adapt to their perceived circumstances that must be examined to understand emotional dysfunction.

A very large and heterogeneous number of the world's children navigate the developmental tasks of infancy, childhood, and adolescence effectively. In terms of emotional development, most children come to access a full range of human emotions and to communicate and understand them. Moreover, they come to regulate emotion, which enables them to function in flexible, culturally appropriate, and effective ways. That is, most children develop the emotional competence that enables them to become productive, interpersonally successful adults. They effectively coordinate their individualistic goals for well-being with the individualistic goals of others as well as relinquish individualistic goals on behalf of relationship goals such as camaraderie, respect, empathy, love, and compassion.

Less often yet far too often children's circumstances challenge their goals for well-being. Although they may not be conscious of the nature of their risky conditions, they may nonetheless perceive at a most basic level that their needs are unmet, their goals unattainable, and their well-being threatened. In these circumstances, children's sensitivity to their emotional environments and their innate ability to adapt to their risky conditions creates the context for atypical emotional development and the potential for compromised emotional competence, which can contribute to a course toward psychopathology. From the functional perspective, individual differences with which children are born that make their development of emotional competence more challenging should be protected by caregiving environments that adjust to their relative difficulties, helping them achieve well-being and develop culturally appropriate skills for achieving their goals flexibly and effectively. Thus, the relational nature of emotion not only creates risk because of the inborn readiness to be sensitive to environmental conditions but also contributes to protection because of the dependence on how the environment relates to children's needs.

Thus, the greatest risk is posed by non-optimal caregiving circumstances in which children must adapt to atypical conditions. Examples discussed in this chapter include the heightened perceptual sensitivity to anger cues of physically abused children and the flattened cortisol function of children exposed to chronic stress. The evidence further suggests that early exposure to adverse circumstances, that is, prior to about age 2 years, constitutes a particular severe risk as it interferes with the very building blocks of emotional competence. These can be mitigated by the introduction of adequate caregiving, as seen in the case of Rumanian orphans placed in foster care prior to age 2. Unfortunately, the adversities to which at risk children adapt often include deficiencies and threats in the caregiving environment with limited access to supportive relationships outside of the caregiving environment. These are contexts in which atypical child-environment relations exist and these increase the chances that the child will have atypical emotional development that will manifest as emotional dysfunction.

This brings us then to discussion of the links between emotional competence and the emotional dysfunction associated with psychopathology. The hypothesis put forward from the functional perspective is that risk factors that interfere with the development of emotional competence will increase risk for the development of emotional dysfunction and for the development of psychopathology. From the functional perspective, this is best considered in terms of how individuals relate to and adapt to their circumstances. Evidence from longitudinal studies indicates that poorer performance in one or more domains of emotional competence usually precedes the emergence of formal symptoms of psychopathology and is often associated with symptom maintenance or exacerbation (Abela et al., 2002; Blandon et al., 2010; Broderick & Korteland, 2004; Cole et al., 2003; Das et al., 2013; Herts, McLaughlin, & Hatzenbuehler, 2012; Hill et al., 2006; Kim-Spoon et al., 2013; McLaughlin et al., 2011; Röll, Koglin, & Petermann, 2012; Stepp et al., 2014; Wirtz, Hofmann, Riper, & Berking, 2013). Only a few of these studies examined bidirectional effects. In general, emotional dysfunction precedes the onset of formal symptoms, although the development of symptoms can also heighten emotional challenges and further compromise emotional competence, as shown in longitudinal studies of adolescence or adults (Larsen et al., 2013; Stepp et al., 2014). In sum, the literature review in this chapter indicates ample research relating emotional competencies with success in life, ample research associating deviations in emotion expression, emotion understanding, and emotion regulation to psychopathology, and a growing body of research suggesting that those deviations precede and likely contribute to the development of psychopathology.

The merging of the developmental psychopathology framework and the functionalist perspective on emotional development offers new perspectives on the role of emotion in the development of psychopathology. These two models share appreciation of context, of the importance of change over time, both in terms of moments and of years, and of the complexities of emotional development. They also point to methods that offer significant promise for overcoming the limitations of the current knowledge base, limitations that temper conclusions and hinder the translation of science to the prevention of and intervention with child and adolescent psychopathology. Together, the models help to move the study of the development of emotional dysfunction away from outcomes defined by *DSM* diagnostic categories or overly general symptom checklists and point us toward examination of emotion processes that develop in complex contexts, that can lead to varied outcomes, and that under certain conditions can lead to the development of psychopathology. The emphasis on emotions as relating to and guiding adaptive action helps us understand how children, faced with adversity but without adequate caregiving support, adapt to less than ideal conditions and how the biological and psychological systems involved in that adaptation can be affected in ways that lead to maladaptive functioning in other contexts. A process-oriented developmental model that can document pathways from the earliest neurophysiological building blocks of the capacity to be emotional through its lifelong development, considering how each period affords new levels of competence and yet new vulnerabilities will help us understand how an adaptive function becomes a factor in psychopathology. In their shared focus on emergent processes involving adaptation to environments, these models suggest certain priorities for identifying turning points that promote or interfere with emotional expressiveness, understanding, and regulation, aiming children and youth toward emotional competence and clinically relevant emotional dysfunction. They offer the following research guideposts:

Personal Relevance

The capacity to be emotional is how the personal relevance of circumstances is evaluated and adaptation is directed. Typically, the biological equipment that enables this capacity reaches sufficient maturation to permit the newborn to

communicate information about well-being that is interpretable to caregivers. What constitutes personal relevance in the complex interpersonal world develops with experience, maturation, and socialization. At every point along the way, as the capacity grows, there are challenges that can catalyze competence or compromise it. Evidence suggests that personally relevant challenges can occur at any point, and that those that neglect the personal needs of or pose threat to the personal well-being of the developing child create the most risk. They create the most risk because they affect the neurophysiological building blocks of the capacity for emotional competence, rather than challenge more established competencies.

There is a dearth of evidence that is based on situations or stimuli that capture personal relevance. This is especially the case for research with children with known disorders, and very much the case in studies employing neuroimaging methods. It is critical that we study emotion processing in situations that create or closely approximate circumstances that we know are personally relevant for participants. There are significant limitations to what we can learn about clinically important individual differences in emotional processing from research using static emotion expressions on unfamiliar faces, inquiring about purported feelings, actions and interpretations in hypothetical situations, or making broad generalizations from questionnaires. However, there is work that seems to have overcome some of the important obstacles that lead to over-reliance on these methods. There is promise in observing neural and behavioral activity when children, including those who have been exposed to threatening anger, process their mothers' angry faces or voices (e.g., Shackman et al., 2007); observing nonverbal expressiveness and verbalizations when children and caregivers discuss actual evocative experiences (e.g., Bauer et al., 2005); observing neural activity and behavior in youth anticipating peer evaluation in a simulated social media context (e.g., Guyer, McClure-Tone, Shiffrin, Pine, & Nelson, 2009; Silk et al., 2012); observing children's nonverbal and verbal behavior in laboratory tasks that mimic circumstances that threaten, disappoint, or frustrate young children (e.g., Buss & Goldsmith, 1998; Cole et al., 1994; Cole et al., 2011; Leerkes & Wong, 2013; Moore, 2009); and observing interactions between caregivers and children or youth involving current or past real-life issues and events that are defined as significant by participants (Kuppens, Allen, & Sheeber, 2010; Pasalich, Waschbusch, Dadds, & Hawes, 2014; Sheeber et al., 2012). In addition, experience sampling methods, such as ecological momentary assessment, electronically activated recording and other technology-assisted methods (e.g., Repetti & Wood, 1997; Silk et al., 2003; Slatcher & Trentacosta, 2012) increase the chances of studying emotion processes in circumstances or with stimuli that come closer to capturing real-life emotion processing.

Dynamics

As detailed in this chapter, the processes that fall under the rubric emotion are better thought of as verbs than nouns. It is part of survival to constantly be processing the significance of circumstances for one's well-being, it is highly adaptive that most of this processing occurs outside of awareness, and it is highly adaptive to be able to re-orient physiologically and be prepared to act if circumstances suddenly jeopardize well-being. And it is clinically important to understand the *dynamics* of emotion processes even if we study them by inferential methods and not direct observation. Our argument has been that emotions ebb and flow over the course of daily functioning, occasionally reaching awareness to which we attribute feelings. The importance of the sequence of events in participants' relation to circumstances, and of the characteristics of the pace and variability in the dynamic unfolding of those moments that are personally relevant, cannot be overstated from the clinical perspective.

The time course of emotions, sometimes referred to as emotion dynamics (e.g., Thompson, 1994), is a critical matter for understanding the role of emotion in the development of psychopathology. As is often said, it is not being angry or sad or afraid that marks dysfunction; these are adaptive responses to perceived circumstances and palpable indices of them communicate to the self or others an appraisal/action readiness. But the moment that an emotion is generated (a particular appraisal/action readiness change occurs), the emotion itself, in addition to the circumstances, is a stimulus. Clinicians deal with emotions about emotions all the time (Cole et al., 2013). An adolescent whose parents have moved to terminate their parental rights may appraise this situation as a significant loss; but sadness may be intolerable for that adolescent and the reaction to sadness is intense anger. A child is enraged with a sibling and then is overwhelmed with shame about being so angry or furious with a parent and then afraid of his own anger. Moreover, changes in emotion are likely to activate associative networks that can intensity or modify the emotion in an iterative process (e.g., see Cunningham, Dunfield, & Stillman, 2013).

Sequential analyses can reveal emotional dysfunction is in the extended order of physiological, self-reported, or observed emotions and other behaviors in individuals or

partners. The earlier example of a child who is frustrated and persisting at a blocked goal (a toy in a clear, locked box), then becomes sad and momentarily withdraws from the task, and then briefly brightens, expressing optimism, before she furrows her brown and mutters as she returns to trying to open the box. Her anger appears to motivate appropriate task persistence (see Dennis et al., 2009), and her brief sadness perhaps gives her time to reflect and re-appraise the situation. This emotional sequence might indicate emotional competence in a 3-year-old, and lead to conclusions that are quite different than the child who is briefly happy to try to open the box, then gets frustrated and angry, and then sadly gives up and says I don't want to come here anymore. Each sequence tells us something quite different than the total number of seconds of anger, sadness, and joy would. These two children have the same total number of seconds of each emotion expression but the sequence is different and those differences may be clinically relevant; 4-month-olds who are frustrated by a blocked goal but become primarily sad have higher cortisol production than those who just stay angry, indicating different stress responses (Lewis, Ramsay, & Sullivan, 2006). Several studies used small time intervals (e.g., 1s or 10s) to examine dyadic or individual influences on changes in emotion using sequences between pairs of variables, such as emotion and child strategy or parent strategy (Buss & Goldsmith, 1998; Crockenberg & Leerkes, 2004; Morris et al., 2011; Silk et al., 2011).

These approaches, however, do not capitalize on the full extent of information in a time series. Lorber and Slep (2005), for example, found support for the hypothesis that the degree to which the emotions of mothers of young children are predicted by the mothers' previous emotions distinguishes patterned, predictable caregiving from more erratic behavior; time series analyses indicated that this form of predictability of maternal emotion was associated with organized versus ineffective parenting behavior. This study demonstrates a principle of functionalist perspective that changes in emotion organize changes in behavior. Creative applications of time series analyses have been applied to behavioral, neural, and autonomic response data in emotion-relevant ways (Bach, Daunizeau, Kuelzow, Friston, & Dolan, 2011; Bulteel et al., 2014; Dagne & Snyder, 2011; Fortunato, Gatzke-Kopp, & Ram, 2013; Hamilton, Chen, Thomason, Schwartz, & Gotlib, 2010).

In addition, the concept of emotion variability, which is integral to emotional competence that is defined by emotional responsiveness and flexibility (e.g., Saarni, 1999), can be studied in time series. We know little about normal variability in the emotional lives of children and youth, but evidence suggests that being emotionally rigid (Kuppens et al., 2012) or labile (Silk et al., 2003; Trull et al., 2008) are hallmarks of psychopathology. These conclusions are drawn from time series data, with time extended over minutes or days.

As seen, there are few but a growing number of studies examining emotional functioning as a process rather than as a trait or event. Consider the risks of ignoring this temporality. A recent unpublished finding from our lab illustrates: 48-month-olds who engage in overt verbal self-instruction during a challenging task (the transparent locked box) have *more* rather than fewer anger displays during the task. If verbal self-instruction aids emotion regulation, as would be predicted, then why would children who use this verbal strategy be angry more often? It appears that mounting frustration precedes and probably motivates verbal self-instruction, which then leads to persistence at trying to solve the dilemma appropriately. This illustrates the value of understanding behavior in the time series. Anger served appropriate problem solving and did not indicate dysregulation. Research that considers the dynamic nature of emotional processing may help us to understand clinically relevant phenomena like debilitating shame that follows anger or anger that covers up deep disappointment (Cole et al., 2013) as well as healthy emotional processes such as righteous anger that mobilizes a person after momentary sadness and hopelessness.

Context

Third, emotion research needs to take context into account to advance our understanding of the role of emotion in the development of psychopathology. Context is a broad term that can be applied to the study of emotional development in many ways. Consider as an example the clinical implications of the same emotion expression in different contexts. A toddler is having a full blown tantrum and her mother is expressing anger. The mother could be angry at her daughter for making her late for work or the mother could be angry with herself because she should have anticipated and avoided the circumstances that led to her daughter's tantrum. Self-focused versus child-focused emotions have different implications for maternal behavior (Leerkes, 2010). Alternatively, consider variations in happiness; the joy of a child who watches another child win a prize is different from the joy of a child who has just snatched that prize from the child's hand.

Another clinically relevant application of context to emotional functioning concerns emotion regulation. A person feels many strong emotions—fearful, angry, and

disgusted—by another person. The person does not leave the situation, does not tell the person to stop, and does not seek another person's assistance. Instead, she begins to dissociate, a maladaptive response. But is it? Let us imagine that the persons are a 4-year-old girl whose father is again using her for his sexual gratification; dissociation is thought to be one of the few emotion regulation strategies available to such a small child in such an impossible situation (Cole & Putnam, 1992). In the immediate context, this can be considered adaptive. In other contexts, such as in early adulthood when the young woman is approached by a potential romantic partner, it may not be. A person's history and associations are part of the context in which emotions can be understood. There are also different social contexts (better to be angry with a trusted parent than with a new teacher), cultural contexts (different consequences of expressing shame in with one's Nepalese relatives than with one's American relatives), and so on.

In addition to the need for emotional development research that is context-sensitive, process-oriented, and personally relevant for participants, there are research design issues derived from principles of developmental psychopathology that are needed to understand the role of emotion in the development of psychopathology. First, a priority is longitudinal research that addresses pathways to emotional competence and emotional dysfunction, and that carefully focuses in on proximal sequences in the developmental pathway. Links between early childhood characteristics, for example, temperament or attachment status, and later outcomes such as adolescent psychopathology have been documented. For example, if infants are highly reactive and hard to soothe, and caregivers have difficulty adjusting to the infant's special needs, developing a style of responding insensitively and intrusively, what are the near consequences for emotional functioning that pose risk for emotional dysfunction—an insecure-avoidant attachment? But there are likely advantages to that attachment style in certain unforgiving situations. So perhaps something more is needed to pave the path toward emotional dysfunction and psychopathology? What are the mitigating and exacerbating factors? Longitudinal research that more carefully documents the series of steps between points is needed and a focus on the domains of emotional competence should be useful in that regard. This will help to address the question of whether and when emotional dysfunction is a precursor of psychopathology, rather than a correlated symptom or consequence of another impaired process.

Second, emotion processes must be inferred; they cannot be studied directly. Therefore, there is a need for careful and clever use of multiple measures of emotion processing that heighten the ability that incorporates multiple measures of emotion, and selection of equally valid and reliable assessment of all predictors, correlates, and outcomes. Designs that include carefully selected variations in context (circumstances, stimuli) and that relate changes in indices of emotion processes with changes in context would be very useful. These can also incorporate the emphasis on process as a time series. We know of no study that has examined developmental changes in emotion processes on multiple time scales, for example, emotion regulation as a moment-to-moment time series that changes over the course of years.

Third, longitudinal studies using sophisticated and sensitive assessment of emotional processes need to include predictors and outcomes that represent multiple domains. There is no single path from a child or an environmental characteristic that will predict outcomes as complex as emotional competence and dysfunction. Nor is there a single child–environmental characteristic pairing that will sufficiently explain pathways from risk to clinically meaningful outcomes. As Sameroff (2009) argued, children and contexts shape each other. Emotion processes are central to the transactional nature of human development. Research that will advance knowledge on the role of emotion in the development of psychopathology will incorporate this model.

Translational Implications

Progress in the understanding of the nature of emotion, in emotional development, and in the role of emotion in the development of psychopathology not only has implications for future research directions but also for applications to clinical practice. Research founded on a functional approach to emotional development has potential to add to how children and youth are evaluated, how prevention of emotional problems is implemented, and how therapeutic interventions are aimed.

Emotional dysfunction is generally assessed through structured diagnostic interviews (PAPA, K-SADS; Egger et al., 2006) and by questionnaires, both self-report and caregiver report, such as Child Behavior Checklist (http://www.aseba.org), the Child Depression Inventory (http://www.mhs.com/product.aspx?gr=edu&id=overview&prod=cdi2), the MASC (http://www.mhs.com/product.aspx?gr=cli&id=overview&prod=masc2), and the Behavior Assessment Scale for Children (http://www.pearsonclinical.com/education/products/100000658/behavior-assessment-system-for-children-second-edition-basc-2.html) among

others. These tools assess emotion-related symptoms using a normative approach, yielding scores that allow practitioners to discern if children's symptoms reach clinical thresholds. Research instruments with potential to help us understand the function of emotional symptoms are less readily available. Consider, for example, two of the most commonly studied correlates or precursors of emotional dysfunction: attachment security and temperament. As yet, there is no widespread use of developmentally sensitive measures of these constructs used as clinical tools, in part because there has been limited efforts to translate these constructs into clinically practical, useful tools and the best measures are focused on very early childhood (e.g., Crittenden, Kozlowska, & Landini, 2010; Putnam, Helbig, Gartstein, Rothbart, & Leerkes, 2013).

Yet from the functional perspective, the nature of an emotion-related symptom can be informative. In a recent intake meeting in our clinic, we discussed information gleaned about a 15-year-old whose mother was concerned about her angry hostility and defiant behavior. The family had a history of significant relationship problems that had not been resolved effectively, and the 15-year-old had little insight into how these affected her or why she was angry other than wanting more freedom. Our team of developmental psychopathology-oriented faculty and student clinicians discussed the youth's angry presentation in terms of its immediate functional message and raised questions about her temperament and attachment history. Sharing a functional perspective, the discussion focused on initial hypotheses about the immediate function of anger for this youth, questions about the precise nature of what she was protesting, consideration that her anger may indicate that she had not yet perceived her goals as unattainable and that anger may be protecting against full-blown depression. The intake report suggested the clinician conduct a functional analysis of her anger and other emotions (anxiety, joy, sadness, shame, guilt), including exploring whether she would use a mood diary so that we could gain more detailed perspective on her experience. The continuing assessment was intended to understand her relation to her circumstances and to potentially generate information that would help her better understand, regulate, and express her needs. Moreover, a comprehensive life history interview, which is standard, would address the points in her life that family instability and conflict might have had implications for her attachment security and whether her temperament, as best it can be inferred retrospectively, might point toward the most sensitive tailoring of approaching behavior change.

Emotional competence is regarded as a central focus of preventive intervention and as a protective factor that helps children be resilient in the face of adversity (Gest & Davidson, 2011; Kellam, Koretz, & Mościcki, 1999). Approaches such as Providing Alternative THinking Strategies (PATHS, http://www.pathseducation.com) are based on developmental research on the components of social emotional learning that promote competence and prevent psychopathology (Domitrovich, Cortes, & Greenberg, 2007; Greenberg, Kusché, Cook, & Quamma, 1995) and have been empirically evaluated and shown to be effective (e.g., Bierman et al., 2008; Conduct Problems Prevention Research Group, 2010). PATHS and related social-emotional learning programs include components that teach parents and teachers to understand and affirm the emotions children are communicating and help them derive better solutions to their perceived needs. The Emotions Course (Izard et al., 2008) is founded on the view that emotions reflect information about children's perceived needs and the promotion of emotion understanding helps children more effectively use and regulate emotion (Izard, 2002; Izard, Stark, Trentacosta, & Schultz, 2008; Izard et al., *Emotion Review*).

A functional perspective on emotional competence should also inform therapeutic intervention (Cole et al., 1994; Suveg, Southam-Gerow, Goodman, & Kendall, 2007). At one level, all approaches to therapeutic intervention are designed to improve emotional functioning, whether directly or indirectly. However, few take a functional perspective of emotion in children and youth experience or a developmental psychopathology framework that guides hypotheses about how children or youth functioning deviates in terms of emotional competencies or emotional developmental factors in the etiology or maintenance of symptoms. There are many considerations that a functional perspective on emotional development offers. Cognitive behavioral approaches to therapy have proven effective, although they are often difficult to implement with family members whose functioning is not defined by a sufficient level of emotional competence. A functional perspective on emotion can help. Combat veterans, for example, benefit from sensitive application of anger management skill training; sensitive approaches appreciate the circumstances to which veterans were prepared to adapt, and their heightened sensitivity to environmental cues of potential threat (including posttraumatic stress responses) and to the skills they developed to adapt effectively (Mackintosh, Morland, Frueh, Greene, & Rosen, 2014). Parent management training is known to be effective for child and youth behavior problems. However, many referred families are unable to implement this otherwise effective approach; this method may

benefit from heightened awareness of (1) the function of emotions that motivate behavior problems and (2) the parental emotions that are involved in anticipating or responding to those behaviors (Maliken & Katz, 2013; Mazursky-Horowitz et al., 2014). Finally, there are lessons to be derived from Emotion-Focused Therapy (Greenberg, 2004). This approach views emotions as the organizing core of the self, a view that is consistent with the functional perspective, and argues that a process-experiential approach to emotion is necessary for meaningful symptom change. That is, it is insufficient to talk about emotion; unresolved emotions continue to motivate symptomatic behavior and that effective resolution requires experiencing the emotions in the presence of a supportive other such as a therapist. Clients are gently encouraged to re-experience painful emotions and through the therapeutic relationship to reappraise them as a means of resolving them. This model has not been applied to therapeutic interventions with children and youth but its relational emphasis, its appreciation that past emotional events can continue to exert pressure on perception and behavior, and its guide for reexperiencing and reappraising them has potential to improve therapeutic approaches with children and youth. Perhaps the functional perspective must first guide clinically relevant developmentally informed research to have a greater impact on clinical assessment and intervention.

REFERENCES

Abela, J. R., & Hankin, B. L. (2011). Rumination as a vulnerability factor to depression during the transition from early to middle adolescence: A multiwave longitudinal study. *Journal of Abnormal Psychology*, *120*, 259–271. doi: 10.1037/a0022796

Abela, J. R., & Hankin, B. L. (Eds.). (2008). *Handbook of depression in children and adolescents*. New York, NY: Guilford Press.

Abela, J. R., Hankin, B. L., Sheshko, D. M., Fishman, M. B., & Stolow, D. (2012). Multi-wave prospective examination of the stress–reactivity extension of response styles theory of depression in high-risk children and early adolescents. *Journal of Abnormal Child Psychology*, *40*, 277–287. doi: 10.1007/s10802-011-9563-x

Adams, D., & Oliver, C. (2011). The expression and assessment of emotions and internal states in individuals with severe or profound intellectual disabilities. *Clinical Psychology Review*, *31*, 293–306. doi: 10.1016/j.cpr.2011.01.003

Aber, J. L., Jones, S., & Cohen, J. (2000). The impact of poverty on the mental health and development of very young children. In J. Cohen & C. Zeanah (Eds.), *Handbook of infant mental health* (pp. 113–128). New York, NY: Guilford Press.

Adolphs, R. (2002). Neural systems for recognizing emotion. *Current Opinion in Neurobiology*, *12*, 169–177. doi: 10.1016/S0959-4388(02)00301-X

Adrian, M., Zeman, J., Erdley, C., Lisa, L., & Sim, L. (2011). Emotional dysregulation and interpersonal difficulties as risk factors for nonsuicidal self-injury in adolescent girls. *Journal of Abnormal Child Psychology*, *39*, 389–400. doi: 10.1007/s10802-010-9465-3

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, *30*, 217–237. doi: 10.1016/j.cpr.2009.11.004

Allen, J. P., & Miga, E. M. (2010). Attachment in adolescence: A move to the level of emotion regulation. *Journal of Social and Personal Relationships*, *27*, 181–190. doi: 10.1177/0265407509360898

Almas, A. N., Degnan, K. A., Radulescu, A., Nelson, C. A., Zeanah, C. H., & Fox, N. A. (2012). Effects of early intervention and the moderating effects of brain activity on institutionalized children's social skills at age 8. *Proceedings of the National Academy of Sciences*, *109*, 17228–17231. doi: 10.1073/pnas.1121256109

Anderson, A. L., & Thomason, M. E. (2013). Functional plasticity before the cradle: A review of functional imaging in the human fetus. *Neuroscience and Biobehavioral Reviews*, *37*, 2220–2232. doi: 10.1016/j.neubiorev.2013.03.013.

Arnold, M. (1960). *Emotion and personality*. New York, NY: Columbia University Press.

Arsenio, W. F., Gold, J., & Adams, E. (2006). Children's conceptions and displays of moral emotions. In M. Killen & J. Smetana (Eds.), *Handbook of moral development* (pp. 581–610). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Bach, D. R., Daunizeau, J., Kuelzow, N., Friston, K. J., & Dolan, R. J. (2011). Dynamic causal modeling of spontaneous fluctuations in skin conductance. *Psychophysiology*, *48*, 252–257. doi: 10.1111/j.1469-8986.2010.01052.x

Bamford, C., & Lagattuta, K. H. (2012). Looking on the bright side: Children's knowledge about the benefits of positive versus negative thinking. *Child Development*, *83*, 667–682. doi: 10.1111/j.1467-8624.2011.01706.x

Bargh, J. A., & Morsella, E. (2008). The unconscious mind. *Perspectives on Psychological Science*, *3*, 73–79. doi: 10.1111/j.1745-6916.2008.00064.x

Bargh, J. A., & Williams, L. E. (2007). The nonconscious regulation of emotion. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 429–445). New York, NY: Guilford Press. Retrieved from http://search.proquest.com/docview/621619855?accountid=13158

Baron-Cohen, S., Golan, O., Wheelwright, S., Granader, Y., & Hill, J. (2010). Emotion word comprehension from 4 to 16 years old: A developmental survey. *Frontiers in Evolutionary Neuroscience*, *2*, 109. doi: 10.3389/fnevo.2010.00109

Barr, R. G., Hopkins, B., & Green, J. A. (2000). Crying as a sign, a symptom and a signal: Evolving concepts of crying behavior. In R. G. Barr, B. Hopkins, & J. A. Green (Eds.), *Crying as a sign, a symptom, and a signal: Clinical, emotional and developmental aspects of infant and toddler crying* (pp. 1–7). London, UK: Mac Keith Press.

Barrett, K. C., & Campos, J. J. (1987). Perspectives on emotional development II: A functionalist approach to emotions. In J. D. Osofsky (Ed.), *Handbook of infant development* (pp. 555–578). New York, NY: Guilford Publications, Inc.

Bassett, H. H., Denham, S., Mincic, M., & Graling, K. (2012). The structure of preschoolers' emotion knowledge: Model equivalence and validity using a structural equation modeling approach. *Early Education & Development*, *23*, 259–279. doi: 10.1080/10409289.2012.630825

Bauer, P. J., Stark, E. N., Lukowski, A. F., Rademacher, J., Van Abbema, D. L., & Ackil, J. K. (2005). Working together to make sense of the past: Mothers' and children's use of internal states language in conversations about traumatic and nontraumatic events. *Journal of Cognition and Development*, *6*, 463–488. doi: 10.1207/s15327647jcd0604_2

Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal theory and developmental psychopathology: Emotion dysregulation and

conduct problems from preschool to adolescence. *Biological Psychology*, *74*, 174–184. doi: 10.1016/j.biopsycho.2005.08.008

Beebe, B., Jaffe, J., Markese, S., Buck, K., Chen, H., Cohen, P… & Feldstein, S. (2010). The origins of 12-month attachment: A microanalysis of 4-month mother–infant interaction. *Attachment & Human Development*, *12*, 3–141. doi: 10.1080/14616730903338985

Beeghly, M., Fuertes, M., Liu, C. H., Delonis, M. S., & Tronick, E. (2011). Maternal sensitivity in dyadic context: Mutual regulation, meaning-making, and reparation. In D. W. Davis & M. C. Logsdon (Eds.), *Maternal sensitivity: A scientific foundation for practice* (pp. 45–69.) Hauppauge, NY: Nova Science Publishers.

Behar, E., DiMarco, I., Hekler, E. B., Mohlman, J., & Staples, A. M. (2009). Current theoretical models of generalized anxiety disorder (GAD): Conceptual review and treatment implications. *Journal of Anxiety Disorders*, *23*, 1011–1023. doi: 10.1016/j.janxdis.2009.07.006

Belsky, J., & Beaver, K. M. (2011). Cumulative-genetic plasticity, parenting and adolescent self-regulation. *Journal of Child Psychology and Psychiatry*, *52*, 619–626. doi: 10.1111/j.1469-7610.2010.02327.x

Belsky, J., Campbell, S. B., Cohn, J. F., & Moore, G. (1996). Instability of infant–parent attachment security. *Developmental Psychology*, *32*, 921–924. doi: 10.1037/0012-1649.32.5.921

Berenbaum, H., Raghavan, C., Le, H. N., Vernon, L. L., & Gomez, J. J. (2003). A taxonomy of emotional disturbances. *Clinical Psychology: Science and Practice*, *10*, 206–226. doi: 10.1093/clipsy.bpg011

Berk, L. E., & Winsler, A. (1995). *Scaffolding children's learning: Vygotsky and early childhood education*. Washington, DC: National Association for Young Children.

Berlin, L. J., & Cassidy, J. (2003). Mothers' self-reported control of their preschool children's emotional expressiveness: A longitudinal study of associations with infant–mother attachment and children's emotion regulation. *Social Development*, *12*, 477–495. doi: 10.1111/1467-9507.00244

Bertin, E., & Striano, T. (2006). The still-face response in newborn, 1.5-, and 3-month-old infants. *Infant Behavior and Development*, *29*, 294–297. doi: 10.1016/j.infbeh.2005.12.003

Berzenski, S. R., & Yates, T. M. (2013). Preschoolers' emotion knowledge and the differential effects of harsh punishment. *Journal of Family Psychology*, *27*, 463–472. doi: 10.1037/a0032910

Bevilacqua, L., & Goldman, D. (2011). Genetics of emotion. *Trends in Cognitive Science*, *15*, 401–408. doi: 10.1016/j.tics.2011.07.009

Bierman, K. L., Domitrovich, C. E., Nix, R. L., Gest, S. D., Welsh, J. A., Greenberg, M. T., …, Gill, S. (2008). Promoting academic and social-emotional school readiness: The Head Start REDI program. *Child Development*, *179*, 1802–1817. doi: 10.1111/j.1467-8624.2008.01227.x

Bigelow, A. E., MacLean, K., Proctor, J., Myatt, T., Gillis, R., & Power, M. (2010). Maternal sensitivity throughout infancy: Continuity and relation to attachment security. *Infant Behavior and Development*, *33*, 50–60. doi: 10.1016/j.infbeh.2009.10.009

Blair, C. (2002). School readiness: Integrating cognition and emotion in a neurobiological conceptualization of children's functioning at school entry. *American Psychologist*, *57*, 111–127. doi: 10.1037/0003-066X.57.2.111

Blair, R. J. R. (2003). Neurobiological basis of psychopathy. *British Journal of Psychiatry*, *182*, 5–7. doi: 10.1192/bjp.182.1.5

Blandon, A. Y., Calkins, S. D., Grimm, K. J., Keane, S. P., & O'Brien, M. (2010). Testing a developmental cascade model of emotional and social competence and early peer acceptance. *Development and Psychopathology*, *22*, 737–748. doi: 10.1017/S0954579410000428

Bloom, P. (Ed.). (1993). *Language acquisition: Core readings*. Loughborough, UK: Harvester Wheatsheaf.

Boden, M. T., Westermann, S., McRae, K., Kuo, J., Alvarez, J., Kulkarni, M. R., … & Bonn-Miller, M. O. (2013). Emotion regulation and posttraumatic stress disorder: A prospective investigation. *Journal of Social and Clinical Psychology*, *32*, 296–314. doi: 10.1521/jscp.2013.32.3.296

Bogart, K. R., Tickle-Degnen, L., & Ambady, N. (2012). Compensatory expressive behavior for facial paralysis: Adaptation to congenital or acquired disability. *Rehabilitation Psychology*, *57*, 43–51. doi: 10.1037/a0026904

Boksa, P. (2010). Effects of prenatal infection on brain development and behavior: A review. *Brain, Behavior, and Immunity*, *24*, 881–897. doi: 10.1016/j.bbi.2010.03.005

Bolten, M., Nast, I., Skrundz, M., Stadler, C., Hellhammer, D. H., & Meinlschmidt, G. (2013). Prenatal programming of emotion regulation: Neonatal reactivity as a differential susceptibility factor moderating the outcome of prenatal cortisol levels. *Journal of Psychosomatic Research*, *75*, 351–357. doi: 10.1016/j.jpsychores.2013.04.014

Borke, H. (1971). Interpersonal perception of young children: Egocentrism or empathy? *Developmental Psychology*, *5*, 263–269. doi: 10.1037/h0031267

Bowlby, J. (1969). *Attachment and loss, volume i: Attachment*. New York, NY: Basic Books

Brackett, M. A., Rivers, S. E., & Salovey, P. (2011). Emotional intelligence: Implications for personal, social, academic, and workplace success. *Social and Personality Psychology Compass 5*, 88–103. doi: 10.1111/j.1751-9004.2010.00334.x

Bradley, M. (2000). Emotion and motivation. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 602– 642). New York, NY: Cambridge University Press.

Braungart, J. M., & Stifter, C. A. (1991). Regulation of negative reactivity during the Strange Situation: Temperament and attachment in 12-month-old infants. *Infant Behavior and Development*, *14*, 349–364. doi: 10.1016/0163-6383(91)90027-P

Braungart-Rieker, J., Garwood, M. M., Powers, B. P., & Notaro, P. C. (1998). Infant affect and affect regulation during the still-face paradigm with mothers and fathers: The role of infant characteristics and parental sensitivity. *Developmental Psychology*, *34*, 1428–1437. doi: 10.1037/0012-1649.34.6.1428

Braungart-Rieker, J., Garwood, M. M., & Stifter, C. A. (1997). Compliance and noncompliance: The roles of maternal control and child temperament. *Journal of Applied Developmental Psychology*, *18*, 411–428. doi: 10.1016/S0193-3973(97)80008-1

Bretherton, I., & Beeghly, M. (1982). Talking about internal states: The acquisition of an explicit theory of mind. *Developmental Psychology*, *18*, 906–921. doi: 10.1037/0012-1649.18.6.906

Bretherton, I., Fritz, J., Zahn-Waxler, C., & Ridgeway, D. (1986). Learning to talk about emotions: A functionalist perspective. *Child Development*, 529–548. doi: 10.2307/1130334

Briggs-Gowan, M. J., Ford, J. D., Fraleigh, L., McCarthy, K., & Carter, A. S. (2010). Prevalence of exposure to potentially traumatic events in a healthy birth cohort of very young children in the northeastern United States. *Journal of Traumatic Stress*, *23*, 725–733. doi: 10.1002/jts.20593

Broderick, P. C., & Korteland, C. (2004). A prospective study of rumination and depression in early adolescence. *Clinical Child Psychology and Psychiatry*, *9*, 383–394. doi: 10.1177/1359104504043920

Brody, G. H., Yu, T., Beach, S. R., Kogan, S. M., Windle, M., & Philibert, R. A. (2014). Harsh parenting and adolescent health: A longitudinal analysis with genetic moderation. *Health Psychology*, *33*, 401–409. doi: 10.1037/a0032686

Brophy-Herb, H. E.; Schiffman, R. F.; Bocknek, E. L., Dupuis, S. B. Fitzgerald, H. E.; Horodynski, M. … & Hillaker, B. (2011). Toddlers'

social-emotional competence in the contexts of maternal emotion socialization and contingent responsiveness in a low-income sample. *Social Development*, *20*, 73–92. doi: 10.1111/j.1467-9507.2009.00570.x

Brownell, C. A., Svetlova, M., Anderson, R., Nichols, S. R., & Drummond, J. (2013). Socialization of early prosocial behavior: Parents' talk about emotions is associated with sharing and helping in toddlers. *Infancy*, *18*, 91–119. doi: 10.1111/j.1532-7078.2012.00125.x

Buckholdt, K. E., Parra, G. R., & Jobe-Shields, L. (2014). Intergenerational transmission of emotion dysregulation through parental invalidation of emotions: Implications for adolescent internalizing and externalizing behaviors. *Journal of Child and Family Studies*, *23*, 324–332. doi: 10.1007/s10826-013-9768-4

Bullmore, E., & Sporns, O. (2012). The economy of brain network organization. *Nature Reviews Neuroscience*, *13*, 336–349. doi: 10.1038/nrn3214

Bulteel, K., Ceulemans, E., Thompson, R. J., Waugh, C. E., Gotlib, I. H., Tuerlinckx, F., & Kuppens, P. (2014). DeCon: A tool to detect emotional concordance in multivariate time series data of emotional responding. *Biological Psychology*, *98*, 29–42. doi: 10.1016/j.biopsycho.2013.10.011

Burchinal, M. R., Roberts, J. E., Hooper, S., & Zeisel, S. A. (2000). Cumulative risk and early cognitive development: a comparison of statistical risk models. *Developmental Psychology*, *36*, 793–807. doi: 10.1037/0012-1649.36.6.793

Burghy, C. A., Stodola, D. E., Ruttle, P. L., Molloy, E. K., Armstrong, J. M., Oler, J. A., … & Birn, R. M. (2012). Developmental pathways to amygdala-prefrontal function and internalizing symptoms in adolescence. *Nature Neuroscience*, *15*, 1736–1741. doi: 10.1038/nn.3257

Burkhouse, K. L., Uhrlass, D. J., Stone, L. B., Knopik, V. S., & Gibb, B. E. (2012). Expressed emotion-criticism and risk of depression onset in children. *Journal of Clinical Child & Adolescent Psychology*, *41*, 771–777. doi: 10.1080/15374416.2012.703122

Burwell, R. A., & Shirk, S. R. (2007). Subtypes of rumination in adolescence: Associations between brooding, reflection, depressive symptoms, and coping. *Journal of Clinical Child and Adolescent Psychology*, *36*, 56–65. doi: 10.1080/15374410709336568

Buss, K. A. (2011). Which fearful toddlers should we worry about? Context, fear regulation, and anxiety risk. *Developmental Psychology*, *47*, 804–819. doi: 10.1037/a0023227

Buss, K. A., Davidson, R. J., Kalin, N. H., & Goldsmith, H. H. (2004). Context-specific freezing and associated physiological reactivity as a dysregulated fear response. *Developmental Psychology*, *40*, 583–594. doi: 10.1037/0012-1649.40.4.583

Buss, K. A., & Goldsmith, H. H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development*, *69*, 359–374. doi: 10.1111/j.1467-8624.1998.tb06195.x

Butler, E. A., & Randall, A. K. (2013). Emotional coregulation in close relationships. *Emotion Review*, *5*, 202–210. doi: 10.1177/1754073912451630

Butner, J., Diamond, L. M., & Hicks, A. M. (2007). Attachment style and two forms of affect coregulation between romantic partners. *Personal Relationships*, *14*, 431–455. doi: 10.1111/j.1475-6811.2007.00164.x

Calder, A. J., Keane, J., Cole, J., Campbell, R., & Young, A. W. (2000). Facial expression recognition by people with Möbius syndrome. *Cognitive Neuropsychology*, *17*, 73–87. doi: 10.1080/02643290038049

Calkins, S. D. (1994). Origins and outcomes of individual differences in emotion regulation. *Monographs of the Society for Research in Child Development*, *59*, 53–72. doi: 10.1111/j.1540-5834.1994.tb01277.x

Calkins, S. D., Gill, K. L., Johnson, M. C., & Smith, C. L. (1999). Emotional reactivity and emotional regulation strategies as predictors of social behavior with peers during toddlerhood. *Social Development*, *8*, 310–334. doi: 10.1111/1467-9507.00098

Calkins, S. D., & Johnson, M. C. (1998). Toddler regulation of distress to frustrating events: Temperamental and maternal correlates. *Infant Behavior and Development*, *21*, 379–395. doi: 10.1016/S0163-6383(98)90015-7

Calkins, S. D., & Keane, S. P. (2009). Developmental origins of early antisocial behavior. *Development and Psychopathology*, *21*, 1095–1109. doi: 10.1017/S095457940999006X

Campos, J. J., Dahl, A., & He, M. (2010). Beyond breaches and battles: Clarifying important misconceptions about emotion. *Emotion Review*, *2*, 100–104. doi: 10.1177/1754073909355013

Campos, J. J., Frankel, C. B., & Camras, L. (2004). On the nature of emotion regulation. *Child Development*, *75*, 377–394. doi: 10.1111/j.1467-8624.2004.00681.x

Camras, L. A., & Allison, K. (1985). Children's understanding of emotional facial expressions and verbal labels. *Journal of Nonverbal Behavior*, *9*, 84–94. doi: 10.1007/BF00987140

Camras, L. A., Oster, H., Bakeman, R., Meng, Z., Ujiie, T., & Campos, J. (2007). Do infants show distinct negative facial expressions for fear and anger? Emotional expression in 11-month-old European-American, Chinese, and Japanese infants. *Infancy*, *11*, 131–155. doi: 10.1080/15250000709336636

Camras, L. A., & Shutter, J. (2010). Emotional facial expressions in infancy. *Emotion Review*, *2*, 120–129. doi: 10.1177/1754073909352529

Capps, L., Kasari, C., Yirmiya, N., & Sigman, M. (1993). Parental perception of emotional expressiveness in children with autism. *Journal of Consulting and Clinical Psychology*, *61*, 475–484. doi: 10.1037/0022-006X.61.3.475

Cartwright, K. L., Bitsakou, P., Daley, D., Gramzow, R. H., Psychogiou, L., Simonoff, E., … & Sonuga-Barke, E. J. (2011). Disentangling child and family influences on maternal expressed emotion toward children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *50*, 1042–1053. doi: 10.1016/j.jaac.2011.07.006

Carvajal, F., & Iglesias, J. (2006). Judgements of facial and vocal signs of emotion in infants with Down syndrome. *Developmental Psychobiology*, *48*, 644–652. doi: 10.1002/dev.20173

Casey, B. J., Getz, S., & Galvan, A. (2008). The adolescent brain. *Developmental Review*, *28*, 62–77. doi: 10.1016/j.dr.2007.08.003

Casey, B. J., Jones, R. M., & Somerville, L. H. (2011). Braking and accelerating of the adolescent brain. *Journal of Research on Adolescence*, *21*, 21–33. doi: 10.1111/j.1532-7795.2010.00712.x

Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. *Monographs of the Society for Research in Child Development*, *59*, 228–249. doi: 10.1111/j.1540-5834.1994.tb01287.x

Caughy, M. O. B., Mills, B., Owen, M. T., & Hurst, J. R. (2013). Emergent self-regulation skills among very young ethnic minority children: A confirmatory factor model. *Journal of Experimental Child Psychology*, *116*, 839–855. doi: 10.1016/j.jecp.2013.07.017

Cavanagh, M., Quinn, D., Duncan, D., Graham, T., & Balbuena, L. (2014). Oppositional defiant disorder is better conceptualized as a disorder of emotional regulation. *Journal of Attention Disorders*, 1–9. doi: 10.1177/1087054713520221

Cecchini, M., Baroni, E., Di Vito, C., & Lai, C. (2011). Smiling in newborns during communicative wake and active sleep. *Infant Behavior and Development*, *34*, 417–423. doi: 10.1016/j.infbeh.2011.04.001

Ceschi, G., & Scherer, K. (2003). Children's ability to control the facial expression of laughter and smiling: Knowledge and behaviour. *Cognition & Emotion*, *17*, 385–411. doi: 10.1080/02699930143000725

Chaplin, T. M. (2006). Anger, happiness, and sadness: Associations with depressive symptoms in late adolescence. *Journal of Youth and Adolescence*, *35*, 977–986. doi: 10.1007/s10964-006-9033-x

Chow, S. M., Haltigan, J. D., & Messinger, D. S. (2010). Dynamic infant–parent affect coupling during the face-to-face/still-face. *Emotion*, *10*, 101–114. doi: 10.1037/a0017824

Ciarrochi, J., Kashdan, T. B., Leeson, P., Heaven, P., & Jordan, C. (2011). On being aware and accepting: A one-year longitudinal study into adolescent well-being. *Journal of Adolescence*, *34*, 695–703. doi: 10.1002/dev.20173

Cicchetti, D., Ackerman, B. P., & Izard, C. E. (1995). Emotions and emotion regulation in developmental psychopathology. *Development and Psychopathology*, *7*, 1–10. doi: 10.1017/S0954579400006301

Cicchetti, D., & Cohen, D. (Eds.). (1995). *Developmental psychopathology: Vol 1. Theory and methods*. New York, NY: Wiley.

Cicchetti, D., & Rogosch, F. A. (2012). Gene × environment interaction and resilience: Effects of child maltreatment and serotonin, corticotropin releasing hormone, dopamine, and oxytocin genes. *Development and Psychopathology*, *24*, 411–427. doi: 10.1017/S0954579412000077

Cicchetti, D., Rogosch, F. A., Gunnar, M. R., & Toth, S. L. (2010). The differential impacts of early physical and sexual abuse and internalizing problems on daytime cortisol rhythm in school-aged children. *Child Development*, *81*, 252–269. doi: 10.1111/j.1467-8624.2009.01393.x

Cicchetti, D., & Roisman, G. I. (2011). *Minnesota symposium on child psychology: The origins and organization of adaptation and maladaptation*, *6*. New York, NY: Wiley.

Cicchetti, D., & Sroufe, L. A. (1976). The relationship between affective and cognitive development in Down's syndrome infants. *Child Development*, *47*, 920–929. doi: 10.2307/1128427

Cisler, J. M., Olatunji, B. O., Feldner, M. T., & Forsyth, J. P. (2010). Emotion regulation and the anxiety disorders: An integrative review. *Journal of Psychopathology and Behavioral Assessment*, *32*, 68–82. doi: 10.1007/s10862-009-9161-1

Coan, J. A., Schaefer, H. S., & Davidson, R. J. (2006). Lending a hand social regulation of the neural response to threat. *Psychological Science*, *17*, 1032–1039. doi: 10.1111/j.1467-9280.2006.01832.x

Cohn, J. F., & Tronick, E. Z. (1987). Mother–infant face-to-face interaction: The sequence of dyadic states at 3, 6, and 9 months. *Developmental Psychology*, *23*, 68–77. doi: 10.1037/0012-1649.23.1.68

Cohn, J. F., & Tronick, E. Z. (1983). Three-month-old infants' reaction to simulated maternal depression. *Child Development*, 185–193. doi: 10.2307/1129876

Cole, P. M. (1986). Children's spontaneous control of facial expression. *Child Development*, *1309–1321*. doi: 10.2307/1130411

Cole, P. M., Armstrong, L. M., & Pemberton, C. K. (2010). The role of language in the development of emotion regulation. In S. Calkins & M. A. Bell (Eds.), *Child development at the intersection of emotion and cognition. Human brain development* (pp. 59–77). Washington, DC: American Psychological Association. doi: 10.1037/12059-004

Cole, P. M., Dennis, T. A., Smith-Simon, K. E., & Cohen, L. H. (2009a). Preschoolers' emotion regulation strategy understanding: Relations with emotion socialization and child self-regulation. *Social Development*, *18*, 324–352. doi: 10.1111/j.1467-9507.2008.00503.x

Cole, P. M., Hall, S. E., & Hajal, N. J. (2013). Emotion dysregulation as a risk factor for psychopathology. In T. P. Beauchaine, & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (2nd ed., pp. 341–376). Hoboken, New Jersey: Wiley.

Cole, P. M., Hall, S. E., & Radzioch, A. M. (2009). Emotional dysregulation and the development of serious misconduct. In S. L. Olson & A. J. Sameroff (Eds.), *Biopsychosocial regulatory processes in the development of childhood behavioral problems* (pp. 186–211). New York, NY: Cambridge University Press.

Cole, P. M., LeDonne, E. N., & Tan, P. Z. (2013). A longitudinal examination of maternal emotions in relation to young children's developing self-regulation. *Parenting*, *13*, 113–132. doi: 10.1080/15295192.2012.709152

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development*, *75*, 317–333. doi: 10.1111/j.1467-8624.2004.00673.x

Cole, P. M., Michel, M. K., & Teti, L. O. D. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research in Child Development*, *59*, 73–102. doi: 10.1111/j.1540-5834.1994.tb01278.x

Cole, P. M., & Moore, G. A. (2015). About face! Infant facial expression of emotion. *Emotion Review*.

Cole, P. M., & Putnam, F. W. (1992). Effect of incest on self and social functioning: A developmental psychopathology perspective. *Journal of Consulting and Clinical Psychology*, *60*, 174–184. doi: 10.1037/0022-006X.60.2.174

Cole, P. M., & Tan, P. Z. (2015). Emotion socialization from a cultural perspective. *Handbook of socialization: Theory and research* (2nd ed., 499–519). New York, NY: Guilford Press.

Cole, P. M., Tan, P. Z., Hall, S. E., Zhang, Y., Crnic, K. A., Blair, C. B., & Li, R. (2011). Developmental changes in anger expression and attention focus: Learning to wait. *Developmental Psychology*, *47*, 1078–1089. doi: 10.1037/a0023813

Cole, P. M., Teti, L. O., & Zahn-Waxler, C. (2003). Mutual emotion regulation and the stability of conduct problems between preschool and early school age. *Development and Psychopathology*, *15*, 1–18. doi: 10.1017.S0954579403000014

Cole, P. M., Zahn-Waxler, C., Fox, N. A., Usher, B. A., & Welsh, J. D. (1996). Individual differences in emotion regulation and behavior problems in preschool children. *Journal of Abnormal Psychology*, *105*, 518–529. doi: 10.1037/0021-843X.105.4.518

Cole, P. M., Zahn-Waxler, C., & Smith, K. D. (1994). Expressive control during a disappointment: Variations related to preschoolers' behavior problems. *Developmental Psychology*, *30*, 835–846. doi: 10.1037/0012-1649.30.6.835

Compas, B. E., Malcarne, V. L., & Fondacaro, K. M. (1988). Coping with stressful events in older children and young adolescents. *Journal of Consulting and Clinical Psychology*, *56*, 405–411. doi: 10.1037/0022-006X.56.3.405

Conduct Problems Prevention Research Group. (2010). Fast Track intervention effects on youth arrests and delinquency. *Journal of Experimental Criminology*, *6*, 131–157. doi: 10.1007/s11292-010-9091-7

Connell, A. M., Hughes-Scalise, A., Klostermann, S., & Azem, T. (2011). Maternal depression and the heart of parenting: Respiratory sinus arrhythmia and affective dynamics during parent–adolescent interactions. *Journal of Family Psychology*, *25*, 653–662. doi: 10.1037/a0025225

Costa, R., & Figueiredo, B. (2012). Infants' behavioral and physiological profile and mother–infant interaction. *International Journal of Behavioral Development*, *36*, 205–214. doi: 10.1177/0165025411428248

Cox, S. J., Mezulis, A. H., & Hyde, J. S. (2010). The influence of child gender role and maternal feedback to child stress on the emergence of the gender difference in depressive rumination in adolescence. *Developmental Psychology*, *46*, 842–852. doi: 10.1037/a0019813

Creswell, C., Apetroaia, A., Murray, L., & Cooper, P. (2013). Cognitive, affective, and behavioral characteristics of mothers with anxiety

disorders in the context of child anxiety disorder. *Journal of Abnormal Psychology*, *122*, 26–38. doi: 10.1037/a0029516

Crittenden, P. M. (1992). Treatment of anxious attachment in infancy and early childhood. *Development and Psychopathology*, *4*, 575–602. doi: 10.1017/S0954579400004880

Crittenden, P., Kozlowska, K., & Landini, A. (2010). Assessing attachment in school-age children. *Clinical Child Psychology and Psychiatry*, *15*, 185–208. doi: 10.1177/1359104509356741

Crockenberg, S. B., & Smith, P. (2002). Antecedents of mother–infant interaction and infant irritability in the first 3 months of life. *Infant Behavior and Development*, *25*, 2–15. doi: 10.1016/S0163-6383(02)00088-7

Crockenberg, S. C., & Leerkes, E. M. (2004). Infant and maternal behaviors regulate infant reactivity to novelty at 6 months. *Developmental Psychology*, *40*, 1123–1132. doi: 10.1037/0012-1649.40.6.1123

Crockenberg, S. C., Leerkes, E. M., & BÁrrig JÓ, P. S. (2008). Predicting aggressive behavior in the third year from infant reactivity and regulation as moderated by maternal behavior. *Development and Psychopathology*, *20*, 37–54. doi: 10.1017/S0954579408000023

Crockett, E. E., Holmes, B. M., Granger, D. A., & Lyons-Ruth, K. (2013). Maternal disrupted communication during face-to-face interaction at 4 months: Relation to maternal and infant cortisol among at-risk families. *Infancy*, *18*, 1111–1134. doi: 10.1111/infa.12015

Crone, E. A., & Dahl, R. E. (2012). Understanding adolescence as a period of social–affective engagement and goal flexibility. *Nature Reviews Neuroscience*, *13*, 636–650. doi: 10.1038/nrn3313

Crowell, S. E., Baucom, B. R., Yaptangco, M., Bride, D., Hsiao, R., McCauley, E., & Beauchaine, T. P. (2014). Emotion dysregulation and dyadic conflict in depressed and typical adolescents: Evaluating concordance across psychophysiological and observational measures. *Biological Psychology*, *98*, 50–58. doi: 10.1016/j.biopsycho.2014.02.009

Cummings, A. J., & Rennels, J. L. (2014). How mood and task complexity affect children's recognition of others' emotions. *Social Development*, *23*, 80–99. doi: 10.1111/sode.12038

Cummings, E. M., & Davies, P. T. (2011). *Marital conflict and children: An emotional security perspective*. New York, NY: Guilford Press.

Cummings, E. M., Zahn, Waxler, C., & Radke, Yarrow, M. (1984). Developmental changes in children's reactions to anger in the home. *Journal of Child Psychology and Psychiatry*, *25*, 63–74. doi: 10.1111/j.1469-7610.1984.tb01719.x

Cummings, E. M., Zahn-Waxler, C., & Radke-Yarrow, M. (1981). Young children's responses to expressions of anger and affection by others in the family. *Child Development*, 1274–1282. doi: 10.2307/1129516

Cummings, J. S., Pellegrini, D. S., Notarius, C. I., & Cummings, E. M. (1989). Children's responses to angry adult behavior as a function of marital distress and history of interparent hostility. *Child Development*, 1035–1043. doi: 10.2307/1130777

Cunningham, W. A., Dunfield, K. A., & Stillman, P. E. (2013). Emotional states from affective dynamics. *Emotion Review*, *5*, 344–355. doi: 10.1177/1754073913489749

Cuthbert, B. N., & Insel, T. R. (2013). Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC medicine*, *11*(1), 126. PMCID: PMC3653747

Cyders, M. A., & Smith, G. T. (2008). Emotion-based dispositions to rash action: Positive and negative urgency. *Psychological Bulletin*, *134*, 807–828. doi: 10.1037/a0013341

Dagne, G. A., & Snyder, J. (2011). Relationship of maternal negative moods to child emotion regulation during family interaction. *Development and Psychopathology*, *23*, 211–223. doi: 10.1017/S095457941000074X

Dahl, R. E. (2004). Adolescent brain development: a period of vulnerabilities and opportunities. Keynote address. *Annals of the New York Academy of Sciences*, *1021*, 1–22. doi: 10.1196/annals.1308.001

Damian, L. E., Stoeber, J., Negru, O., & Băban, A. (2013). On the development of perfectionism in adolescence: Perceived parental expectations predict longitudinal increases in socially prescribed perfectionism. *Personality and Individual Differences*, *55*, 688–693. doi: 10.1016/j.paid.2013.05.021

Darwin, C. (1872). *Emotions in man and animals*. New York, NY: D. Appleton and Company.

Das, P., Coulston, C. M., Bargh, D. M., Tanious, M., Phan, K. L., Calhoun, V. D., & Malhi, G. S. (2013). Neural antecedents of emotional disorders: A functional magnetic resonance imaging study of subsyndromal emotional symptoms in adolescent girls. *Biological Psychiatry*, *74*(4), 265–272. doi: 10.1016/j.biopsych.2013.01.030

Davidov, M., Zahn-Waxler, C., Roth-Hanania, R., & Knafo, A. (2013). Concern for others in the first year of life: theory, evidence, and avenues for research. *Child Development Perspectives*, *7*, 126–131. doi: 10.1111/cdep.12028

Davidson, R. J. (2000). Affective style, psychopathology, and resilience: Brain mechanisms and plasticity. *American Psychologist*, *55*, 1196–1214. doi: 10.1037/0003-066X.55.11.1196

Davies, P. T., Cicchetti, D., & Martin, M. J. (2012). Toward greater specificity in identifying associations among interparental aggression, child emotional reactivity to conflict, and child problems. *Child Development*, *83*, 1789–1804. doi: 10.1111/j.1467-8624.2012.01804.x

Davis, B. T., Hops, H., Alpert, A., & Sheeber, L. (1998). Child responses to parental conflict and their effect on adjustment: A study of triadic relations. *Journal of Family Psychology*, *12*, 163–177. doi: 10.1037/0893-3200.12.2.163

Davis, E. L., Levine, L. J., Lench, H. C., & Quas, J. A. (2010). Metacognitive emotion regulation: children's awareness that changing thoughts and goals can alleviate negative emotions. *Emotion*, *10*, 498–510. doi: 10.1037/a0018428

Davis, E. P., Glynn, L. M., Waffarn, F., & Sandman, C. A. (2011). Prenatal maternal stress programs infant stress regulation. *Journal of Child Psychology and Psychiatry*, *52*, 119–129. doi: 10.111/j.1469-7610.2010.02314.x

Davis, E. P., Snidman, N., Wadhwa, P. D., Glynn, L. M., Schetter, C. D., & Sandman, C. A. (2004). Prenatal maternal anxiety and depression predict negative behavioral reactivity in infancy. *Infancy*, *6*, 319–331. doi: 10.1207/s15327078in0603_1

Day, K. L., & Smith, C. L. (2013). Understanding the role of private speech in children's emotion regulation. *Early Childhood Research Quarterly*, *28*, 405–414. doi: 10.1016/j.ecresq.2012.10.003

DeBoard-Lucas, R. L., Fosco, G. M., Raynor, S. R., & Grych, J. H. (2010). Interparental conflict in context: Exploring relations between parenting processes and children's conflict appraisals. *Journal of Clinical Child & Adolescent Psychology*, *39*, 163–175. doi: 10.1080/15374410903532593

DeCasper, A. J., & Fifer, W. P. (1980). Of human bonding: Newborns prefer their mothers' voices. *Science*, *208*, 1174–1176. doi: 10.1126/science.7375928

DeCasper, A. J., Lecanuet, J., Busnel, M., Granier-Deferre, C., & Maugeais, R. (1994). Fetal reactions to recurrent maternal speech. *Infant Behavior and Development*, *17*, 159–164. doi: 10.1016/0163-6383(94)90051-5

Del Vecchio, T., Walter, A., & O'Leary, S. G. (2009). Affective and physiological factors predicting maternal response to infant crying. *Infant Behavior & Development*, *32*, 117–122. doi: 10.1016/j.infbeh.2008.10.005

Denham, S. A. (1998). *Emotional development in young children*. New York, NY: Guilford Press.

Denham, S. A. (1986). Social cognition, prosocial behavior, and emotion in preschoolers: Contextual validation. *Child Development*, 194–201. doi: 10.2307/1130651

Denham, S. A. (2006). Social-emotional competence as support for school readiness: What is it and how do we assess it? *Early Education and Development*, *17*, 57–89. doi: 10.1207/s15566935eed1701_4

Denham, S. A., Bassett, H. H., Sirotkin, Y. S., & Zinsser, K. (2013). Head start preschoolers' emotional positivity and emotion regulation predict their social-emotion behavior, classroom adjustment, and early school success. *NHSA Dialog*, *16,* https://journals.uncc.edu/dialog/article/view/92.

Denham, S. A., Workman, E., Cole, P. M., Weissbrod, C., Kendziora, K. T., & Zahn-Waxler, C. (2000). Prediction of externalizing behavior problems from early to middle childhood: The role of parental socialization and emotion expression. *Development and Psychopathology*, *12*, 23–45. doi: 10.1017/S0954579400001024

Dennis, T. A., & Kelemen, D. A. (2009). Preschool children's views on emotion regulation: Functional associations and implications for social-emotional adjustment. *International Journal of Behavioral Development*, *33*, 243–252. doi: 10.1177/0165025408098024

DeWall, C. N., Masten, C. L., Powell, C., Combs, D., Schurtz, D. R., & Eisenberger, N. I. (2012). Do neural responses to rejection depend on attachment style? An fMRI study. *Social Cognitive and Affective Neuroscience*, *7*, 184–192. doi: 10.1093/scan/nsq107

Diener, M. L., Mangelsdorf, S. C., McHale, J. L., & Frosch, C. A. (2002). Infants' behavioral strategies for emotion regulation with fathers and mothers: Associations with emotional expressions and attachment quality. *Infancy*, *3*, 153–174. doi: 10.1207/S15327078IN0302_3

DiPietro, J. A. (2012). Maternal stress in pregnancy: considerations for fetal development. *Journal of Adolescent Health*, *51*, S3–S8. doi: 10.1016/j.jadohealth.2012.04.008

DiPietro, J. A., Ghera, M. M., & Costigan, K. A. (2008). Prenatal origins of temperamental reactivity in early infancy. *Early Human Development*, *84*, 569–575. doi: 10.1016/j.earlhumdev.2008.01.004

DiPietro, J. A., Kivlighan, K. T., Costigan, K. A., Rubin, S. E., Shiffler, D. E., Henderson, J. L., & Pillion, J. P. (2010). Prenatal antecedents of newborn neurological maturation. *Child Development*, *81*, 115–130. doi: 10.1111/j.1467-8624.2009.01384.x

DiPietro, J. A., Novak, M. F., Costigan, K. A., Atella, L. D., & Reusing, S. P. (2006). Maternal psychological distress during pregnancy in relation to child development at age two. *Child Development*, *77*, 573–587. doi: 10.1111/j.1467-8624.2006.00891.x

Dix, T., Meunier, L. N., Lusk, K., & Perfect, M. (2012). Mothers' depressive symptoms and children's facial emotions: Examining the depression-inhibition hypothesis. *Development and Psychopathology*, *24*, 195–210. doi: 10.1017/S0954579411000770

Domitrovich, C. E., Cortes, R. C., & Greenberg, M. T. (2007). Improving young children's social and emotional competence: A randomized trial of the preschool "PATHS" curriculum. *Journal of Primary Prevention*, *28*, 67–91. doi: 10.1007/s10935-007-0081-0

Dozier, M. (2003). Attachment-based treatment for vulnerable children. *Attachment & Human Development*, *5*, 253–257. doi: 10.1080/1461673031000159615 1

Dozier, M., Zeanah, C. H., & Bernard, K. (2013). Infants and toddlers in foster care. *Child Development Perspectives*, *7*, 166–171. doi: 10.1111/cdep.12033

Dumas, J. E., LaFreniere, P. J., & Serketich, W. J. (1995). "Balance of power": A transactional analysis of control in mother–child dyads involving socially competent, aggressive, and anxious children. *Journal of Abnormal Psychology*, *104*, 104–113. doi: 10.1037/0021-843X.104.1.104

Duncan, L. E., Pollastri, A. R., & Smoller, J. W. (2014). Mind the gap: Why many geneticists and psychological scientists have discrepant views about gene–environment interaction (GxE) research. *American Psychologist*, *69*, 249–268. doi: 10.1037/a0036320

Dunn, J. (2004). The development of individual differences in understanding emotion and mind: Antecedents and sequelae. In A. S. R. Manstead, N. Frijda, & A. Fischer (Eds.), *Feelings and emotions: The Amsterdam symposium. Studies in emotion and social interaction* (pp. 303–320). New York, NY: Cambridge University Press.

Dunn, J., Brown, J., & Beardsall, L. (1991). Family talk about feeling states and children's later understanding of others' emotions. *Developmental Psychology*, *27*, 448–455. doi: 10.1037/0012-1649.27.3.448

Dunton, G. F., Huh, J., Leventhal, A. M., Riggs, N., Hedeker, D., Spruijt-Metz, D., & Pentz, M. A. (2014). Momentary assessment of affect, physical feeling states, and physical activity in children. *Health Psychology*, *33*, 255–263. doi: 10.1037/a0032640

Durbin, C. E., Klein, D. N., Hayden, E. P., Buckley, M. E., & Moerk, K. C. (2005). Temperamental emotionality in preschoolers and parental mood disorders. *Journal of Abnormal Psychology*, *114*, 28–37. doi: 10.1037/0021-843X.114.1.28

Dyck, M. J., Farrugia, C., Shochet, I. M., & Holmes-Brown, M. (2004). Emotion recognition/understanding ability in hearing or vision-impaired children: do sounds, sights, or words make the difference? *Journal of Child Psychology and Psychiatry*, *45*, 789–800. doi: 10.1111/j.1469-7610.2004.00272.x

Edwards, C. P., & Liu, W. (2002). Parenting toddlers. In M. H. Bornstein (Ed.), *Handbook of parenting*, *1* (pp. 45–71). Mahwah, NJ: Lawrence Erlbaum Associates.

Egger, H. L., & Angold, A. (2006). Common emotional and behavioral disorders in preschool children: presentation, nosology, and epidemiology. *Journal of Child Psychology and Psychiatry*, *47*, 313–337. doi: 10.1111/j.1469-7610.2006.01618.x

Ein-Dor, T., Mikulincer, M., Doron, G., & Shaver, P. R. (2010). The Attachment paradox how can so many of us (the insecure ones) have no adaptive advantages? *Perspectives on Psychological Science*, *5*, 123–141. doi: 10.1177/1745691610362349

Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry*, *9*, 241–273. doi: 10.1207/s15327965pli0904_1

Eisenberg, N., Fabes, R. A., Bernzweig, J., Karbon, M., Poulin, R., & Hanish, L. (1993). The relations of emotionality and regulation to preschoolers' social skills and sociometric status. *Child Development*, *64*, 1418–1438. doi: 10.1111/j.1467-8624.1993.tb02961.x

Eisenberg, N., Fabes, R. A., Guthrie, I. K. (1997). Coping with stress: The roles of regulation and development. In S. A. Wolchik & I. N. Sandler (Eds.), *Handbook of children's coping: Linking theory and intervention. Issues in clinical child psychology* (pp. 41–70). New York, NY: Plenum Press.

Eisenberg, N., Fabes, R. A., Guthrie, I. K., Murphy, B. C., Maszk, P., Holmgren, R., & Suh, K. (1996). The relations of regulation and emotionality to problem behavior in elementary school children. *Development and Psychopathology*, *8*, 141–162. doi: 10.1017/S095457940000701X

Eisenberg, N., Gershoff, E. T., Fabes, R. A., Shepard, S. A., Cumberland, A. J., Losoya, S. H., … & Murphy, B. C. (2001). Mother's emotional expressivity and children's behavior problems and social competence: Mediation through children's regulation. *Developmental Psychology*, *37*, 475–490. doi: 10.1037/0012-1649.37.4.475

Eisenberg, N., Valiente, C., & Eggum, N. D. (2010). Self-regulation and school readiness. *Early Education and Development*, *21*, 681–698. doi: 10.1080/10409289.2010.497451

Eisenberg, N., Valiente, C., Morris, A. S., Fabes, R. A., Cumberland, A., Reiser, M., … & Losoya, S. (2003). Longitudinal relations among parental emotional expressivity, children's regulation, and quality of socioemotional functioning. *Developmental Psychology*, *39*, 3–19. doi: 10.1037/0012-1649.39.1.3

Eisenberg, N., Valiente, C., Spinrad, T. L., Cumberland, A., Liew, J., Reiser, M., … & Losoya, S. H. (2009). Longitudinal relations of children's effortful control, impulsivity, and negative emotionality to their externalizing, internalizing, and co-occurring behavior problems. *Developmental Psychology*, *45*, 988–1008. doi: 10.1037/a0016213

Eisenberg, N., Zhou, Q., Spinrad, T. L., Valiente, C., Fabes, R. A., & Liew, J. (2005). Relations among positive parenting, children's effortful control, and externalizing problems: A three-wave longitudinal study. *Child Development*, *76*, 1055–1071. doi: 10.1111/j.1467-8624.2005.00897.x

Eisenberger, N. I., Master, S. L., Inagaki, T. K., Taylor, S. E., Shirinyan, D., Lieberman, M. D., & Naliboff, B. D. (2011). Attachment figures activate a safety signal-related neural region and reduce pain experience. *Proceedings of the National Academy of Sciences*, *108*, 11721–11726. doi: 10.1073/pnas.1108239108

Eisenhower, A. S., Baker, B. L., & Blacher, J. (2005). Preschool children with intellectual disability: syndrome specificity, behavior problems, and maternal well-being. *Journal of Intellectual Disabilities Research*, *49*, 657–671. doi: 10.1111/j.1365-2788.2005.00699.x

Ekas, N. V., Lickenbrock, D. M., & Braungart-Rieker, J. M. (2013). Developmental trajectories of emotion regulation across infancy: Do age and the social partner influence temporal patterns. *Infancy*, *18*, 729–754. doi: 10.1111/infa.12003

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & Van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology*, *23*, 7–28. doi: 10.1017/S0954579410000611

Erickson, M. F., Egeland, B. (2002) Child neglect In J. E. B. Myers, L. Berliner, J. Briere, C. T. Hendriz, C. Jenny, & T. A. Reid (Eds.), *The APSAC Handbook on child maltreatment* (2nd ed.), Thousand Oaks, CA: Sage Publications.

Evans, C. A., & Porter, C. L. (2009). The emergence of mother–infant co-regulation during the first year: Links to infants' developmental status and attachment. *Infant Behavior and Development*, *32*, 147–158. doi: 10.1016/j.infbeh.2008.12.005

Evans, G. W., & English, K. (2002). The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development*, *73*, 1238–1248. doi: 10.1111/1467-8624.00469

Evans, G. W., Gonnella, C., Marcynyszyn, L. A., Gentile, L., & Salpekar, N. (2005). The role of chaos in poverty and children's socioemotional adjustment. *Psychological Science*, *16*, 560–565. doi: 10.1111/j.0956-7976.2005.01575.x

Evans, G. W., & Kim, P. (2013). Childhood poverty, chronic stress, self-regulation, and coping. *Child Development Perspectives*, *7*, 43–48. doi: 10.1111/cdep.12013

Evans, G. W., & Rosenbaum, J. (2008). Self-regulation and the income-achievement gap. *Early Childhood Research Quarterly*, *23*, 504–514. doi: 10.1016/j.ecresq.2008.07.002

Fabes, R. A., Eisenberg, N., Hanish, L. D., & Spinrad, T. L. (2001). Preschoolers' spontaneous emotion vocabulary: Relations to likability. *Early Education and Development*, *12*, 11–27. doi: 10.1207/s15566935eed1201_2

Fabes, R. A., Eisenberg, N., Jones, S., Smith, M., Guthrie, I., Poulin, R., … & Friedman, J. (1999). Regulation, emotionality, and preschoolers' socially competent peer interactions. *Child Development*, *70*, 432–442. doi: 10.1111/1467-8624.00031

Fan, Y., Herrera-Melendez, A. L., Pestke, K., Feeser, M., Aust, S., Otte, C., … & Grimm, S. (2014). Early life stress modulates amygdala-prefrontal functional connectivity: Implications for oxytocin effects. *Human Brain Mapping*. 1–12. doi: 10.1002/hbm.22553

Federenko, I. S., Schlotz, W., Kirschbaum, C., Bartels, M., Hellhammer, D. H., & Wüst, S. (2006). The heritability of perceived stress. *Psychological Medicine*, *36*, 375–385. doi: 10.1017/S0033291705006616

Feldman, R. (2012). Parent–infant synchrony: A biobehavioral model of mutual influences in the formation of affiliative bonds. *Monographs of the Society for Research in Child Development*, *77*, 42–51. doi: 10.1111/j.1540-5834.2011.00660.x

Feldman, R. S., Dollberg, D., & Nadam, R. (2011). The expression and regulation of anger in toddlers: Relations to maternal behavior and mental representations. *Infant Behavior and Development*, *34*, 310–320. doi: 10.1016/j.infbeh.2011.02.001

Feldman, R. S., Jenkins, L., & Popoola, O. (1979). Detection of deception in adults and children via facial expressions. *Child Development*, 350–355. doi: 10.2307/1129409

Feldman, R., & Klein, P. S. (2003). Toddlers' self-regulated compliance to mothers, caregivers, and fathers: implications for theories of socialization. *Developmental Psychology*, *39*, 680–692. doi: 10.1037/0012-1649.39.4.680

Feldman, R. S., Tomasian, J. C., & Coats, E. J. (1999). Nonverbal deception abilities and adolescents' social competence: Adolescents with higher social skills are better liars. *Journal of Nonverbal Behavior*, *23*, 237–249. doi: 10.1023/A:1021369327584

Feng, X., Shaw, D. S., & Moilanen, K. L. (2011). Parental negative control moderates the shyness–emotion regulation pathway to school-age internalizing symptoms. *Journal of Abnormal Child Psychology*, *39*, 425–436. doi: 10.1007/s10802-010-9469-z

Fernald, A. (1993). Approval and disapproval: Infant responsiveness to vocal affect in familiar and unfamiliar languages. *Child Development*, *64*, 657–674. doi: 10.1111/j.1467-8624.1993.tb02934.x

Field, T., Diego, M., & Hernandez-Reif, M. (2006). Prenatal depression effects on the fetus and newborn: A review. *Infant Behavior and Development*, *29*, 445–455. doi: 10.1016/j.infbeh.2006.03.003

Fischer, K. W., Shaver, P. R., & Carnochan, P. (1990). How emotions develop and how they organise development. *Cognition and Emotion*, *4*, 81–127. doi: 10.1080/02699939008407142

Fivush, R., Hazzard, A., McDermott Sales, J., Sarfati, D., & Brown, T. (2003). Creating coherence out of chaos? Children's narratives of emotionally positive and negative events. *Applied Cognitive Psychology*, *17*, 1–19. doi: 10.1002/acp.854

Fizke, E., Barthel, D., Peters, T., & Rakoczy, H. (2014). Executive function plays a role in coordinating different perspectives, particularly when one's own perspective is involved. *Cognition*, *130*, 315–334. doi: 10.1016/j.cognition.2013.11.017

Flavell, J. H. (2000). Development of children's knowledge about the mental world. *International Journal of Behavioral Development*, *24*, 15–23. doi: 10.1080/016502500383421

Flynn, M., & Rudolph, K. D. (2010). The contribution of deficits in emotional clarity to stress responses and depression. *Journal of Applied Developmental Psychology*, *31*, 291–297. doi: 10.1016/j.appdev.2010.04.004

Fogel, A. (2000). Systems, attachment, and relationships. *Human Development*, *43*, 314–320. doi: 10.1159/000022692

Fonagy, P. (2002). *Understanding of mental states, mother–infant interaction and the development of the self*. Washington, DC: American Psychiatric Publishing.

Fortunato, C. K., Gatzke-Kopp, L. M., & Ram, N. (2013). Associations between respiratory sinus arrhythmia reactivity and internalizing and

externalizing symptoms are emotion specific. *Cognitive, Affective, & Behavioral Neuroscience*, *13*, 238–251. doi: 10.3758/s13415-012-0136-4

Fosco, G. M., & Grych, J. H. (2007). Emotional expression in the family as a context for children's appraisals of interparental conflict. *Journal of Family Psychology*, *21*, 248–258. doi: 10.1037/0893-3200.21.2.248

Fowler, S., & Szabó, M. (2013). The Emotional experience associated with worrying in adolescents. *Journal of Psychopathology and Behavioral Assessment*, *35*, 65–75. doi: 10.1007/s10862-012-9316-3

Fraley, R. C. (2002). Attachment stability from infancy to adulthood: Meta-analysis and dynamic modeling of developmental mechanisms. *Personality and Social Psychology Review*, *6*, 123–151. doi: 10.1207/S15327957PSPR0602_03

Frick, P. J., Ray, J. V., Thornton, L. C., & Kahn, R. E. (2013). Annual research review: A developmental psychopathology approach to understanding callous-unemotional traits in children and adolescents with serious conduct problems. *Journal of Child Psychology and Psychiatry.* 532–548. doi: 10.1111/jcpp.12152

Frijda, N. (1986). *The emotions*. New York, NY: Cambridge University Press.

Ganiban, J., Barnett, D., & Cicchetti, D. (2000). Negative reactivity and attachment: Down syndrome's contribution to the attachment-temperament debate. *Development and Psychopathology*, *12*, 1–21. doi: 10.1017/S0954579400001012

Garber, J., Braafladt, N., & Weiss, B. (1995). Affect regulation in depressed and nondepressed children and young adolescents. *Development and Psychopathology*, *7*, 93–115. doi: 10.1017/S0954579400006362

Garber, J., Braafladt, N., & Zeman, J. (1991). The regulation of sad affect: An information-processing perspective. In J. Garber & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation. Cambridge studies in social and emotional development*. (pp. 208–240). New York, NY: Cambridge University Press. doi: 10.1017/CBO9780511663963.011

Gard, D. E., Kring, A. M., Gard, M. G., Horan, W. P., & Green, M. F. (2007). Anhedonia in schizophrenia: Distinctions between anticipatory and consummatory pleasure. *Schizophrenia Research*, *93*, 253–260. doi: 10.1016/j.schres.2007.03.008

Garner, P. W., & Hinton, T. S. (2010). Emotional display rules and emotion self-regulation: Associations with bullying and victimization in community-based after school programs. *Journal of Community & Applied Social Psychology*, *20*, 480–496. doi: 10.1002/casp.1057

Garner, P. W., Mahatmya, D., Moses, L. K., & Bolt, E. N. (2014). Associations of preschool type and teacher–child relational quality with young children's social-emotional competence. *Early Education and Development*, *25*, 399–420. doi: 10.1080/10409289.2013.801706

Garner, P. W., & Power, T. G. (1996). Preschoolers' emotional control in the disappointment paradigm and its relation to temperament, emotional knowledge, and family expressiveness. *Child Development*, *67*, 1406–1419. doi: 10.1111/1467-8624.ep9704041011

Garner, P. W., & Spears, F. M. (2000). Emotion regulation in low-income preschoolers. *Social Development*, *9*, 246–264. doi: 10.1111/1467-9507.00122

Garrett-Peters, P. T., & Fox, N. A. (2007). Cross-cultural differences in children's emotional reactions to a disappointing situation. *International Journal of Behavioral Development*, *31*, 161–169. doi: 10.1177/0165025407074627

Gergely, G., & Watson, J. S. (1999). Early socio-emotional development: Contingency perception and the social-biofeedback model. In P. Rochat (Ed.), *Early social cognition: Understanding others in the first months of life*, (pp. 101–136). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Gest, S. D., & Davidson, A. J. (2011). A developmental perspective on risk, resilience, and prevention. In M. K. Underwood & L. H. Rosen (Eds.), *Social development: Relationships in infancy, childhood, and adolescence* (pp. 427–454). New York, NY: Guilford Press.

Geva, R., & Feldman, R. (2008). A neurobiological model for the effects of early brainstem functioning on the development of behavior and emotion regulation in infants: Implications for prenatal and perinatal risk. *Journal of Child Psychology and Psychiatry*, *49*, 1031–1041. doi: 10.1111/j.1469-7610.2008.01918.x

Gibb, B. E., Uhrlass, D. J., Grassia, M., Benas, J. S., & McGeary, J. (2009). Children's inferential styles, 5-HTTLPR genotype, and maternal expressed emotion-criticism: An integrated model for the intergenerational transmission of depression. *Journal of Abnormal Psychology*, *118*, 734–745. doi: 10.1037/a0016765

Gibbons, F. X., O'Hara, R. E., Stock, M. L., Gerrard, M., Weng, C. Y., & Wills, T. A. (2012). The erosive effects of racism: reduced self-control mediates the relation between perceived racial discrimination and substance use in African American adolescents. *Journal of Personality and Social Psychology*, *102*, 1089–1104. doi: 10.1037/a0027404

Gilliom, M., Shaw, D. S., Beck, J. E., Schonberg, M. A., & Lukon, J. L. (2002). Anger regulation in disadvantaged preschool boys: strategies, antecedents, and the development of self-control. *Developmental Psychology*, *38*, 222–235. doi: 10.1037/0012-1649.38.2.222

Gingras, J. L., Mitchell, E. A., & Grattan, K. E. (2005). Fetal homologue of infant crying. *Archives of Childhood Disease*, *90*, 415–518. doi: 10.1136/adc.2004.062257

Glover, V. (2011). Annual research review: Prenatal stress and the origins of psychopathology: an evolutionary perspective. *Journal of Child Psychology and Psychiatry*, *52*, 356–367. doi: 10.1111/j.1469-7610.2011.02371.x

Glynn, L. M., & Sandman, C. A. (2011). Prenatal origins of neurological development: A critical period for fetus and mother. *Current Directions in Psychological Science*, *20*, 384–389. doi: 10.1177/0963721411422056

Goldsmith, H. H., Buss, A. H., Plomin, R., Rothbart, M. K., Thomas, A., Chess, S., … & McCall, R. B. (1987). Roundtable: What is temperament? Four approaches. *Child Development*, *58*, 505–529. doi: 10.2307/1130527

Goldstein, A. L., Vilhena-Churchill, N., Stewart, S. H., & Wekerle, C. (2012). Coping motives as moderators of the relationship between emotional distress and alcohol problems in a sample of adolescents involved with child welfare. *Advances in Mental Health*, *11*, 67–75. doi: 10.5172/jamh.2012.11.1.67

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: a meta-analytic review. *Clinical Child and Family Psychology Review*, *14*, 1–27. doi 10.1007/s10567-010-0080-1

Gottesman, I., & Gould, T. D. (2003). The endophenotypes concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry*, *160*, 636–645. doi: 10.1176/appi.ajp.160.4.636

Gottlieb, G. (2007). Probabilistic epigenesist. *Developmental Science*, *10*, 1–11. doi: 10.1111/j.1467-7687.2007.00556.x

Gottman, J. M., Katz, L. F., & Hooven, C. (1997). *Meta-emotion: How families communicate emotionally*. Mahwah, NJ: Psychology Press.

Gräff, J., Kim, D., Dobbin, M. M., & Tsai, L. (2011). Epigenetic regulation of gene expression in physiological and pathological brain processes. *Physiological Reviews*, *91*, 603–649. doi: 10.1152/physrev.00012.2010

Graham, A. M.; Fisher, P. A., & Pfeifer, J. H. (2013). What sleeping babies hear: A functional MRI study of interparental conflict and infant's emotion processing. *Psychological Science*, *24*, 782–789. doi: 10.1177/0956797612458803

Gratz, K. L., & Gunderson, J. G. (2006). Preliminary data on an acceptance-based emotion regulation group intervention for deliberate self-harm among women with borderline personality disorder. *Behavior Therapy*, *37*, 25–35. doi: 10.1016/j.beth.2005.03.002

Gratz, K. L., Kiel, E. J., Latzman, R. D., Elkin, T. D., Moore, S. A., & Tull, M. T. (2014). Emotion: Empirical contribution: maternal borderline personality pathology and infant emotion regulation: Examining the influence of maternal emotion-related difficulties and infant attachment. *Journal of Personality Disorders*, *28*, 52–69. doi: 10.1521/pedi.2014.28.1.52

Graven, S. N., & Browne, J. V. (2008). Sensory development in the fetus, neonate, and infant: introduction and overview. *Newborn and Infant Nursing Reviews*, *8*, 169–172. doi: 10.1053/j.nainr.2008.10.007

Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication I: Associations with first onset of *DSM-IV* disorders. *Archives of General Psychiatry*, *67*, 113–123. doi: 10.1001/archgenpsychiatry.2009.186.

Greenberg, L. S. (2004). Emotion-focused therapy. *Clinical Psychology & Psychotherapy*, *11*, 3–16. doi: 10.1002/cpp.388

Greenberg, M. T., Kusche, C. A., Cook, E. T., & Quamma, J. P. (1995). Promoting emotional competence in school-aged children: The effects of the PATHS curriculum. *Development and Psychopathology*, *7*, 117–136. doi: 10.1017/S0954579400006374

Greeno, C., Wing, R., & Shiffman, S. (2000). Binge antecedents in obese women with and without binge eating disorder. *Journal of Consulting and Clinical Psychology*, *68*, 95–102. doi: 10.1037/0022-006X.68.1.95

Greenough, W. T., & Black, J. E. (1992). Induction of brain structure by experience: Substrates for cognitive development. In M. Gunnar & C. Nelson (Eds.), *Developmental behavioral neuroscience: The Minnesota symposia on child psychology* (Vol. 24, pp. 155–200). Hillsdale, NJ: Erlbaum.

Grolnick, W. S., Bridges, L. J., & Connell, J. P. (1996). Emotion regulation in two-year-olds: strategies and emotional expression in four contexts. *Child Development*, *67*, 928–941. doi: 10.1111/j.1467-8624.1996.tb01774.x

Gross, J. J., & Muñoz, R. F. (1995). Emotion regulation and mental health. *Clinical Psychology: Science and Practice*, *2*, 151–164. doi: 10.1111/j.1468-2850.1995.tb00036.x

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation*, *3* (pp. 3–24). New York, NY: Guilford Press.

Grossman, R. B., Edelson, L. R., & Tager-Flusberg, H. (2013). Emotional facial and vocal expressions during story retelling by children and adolescents with high-functioning autism. *Journal of Speech, Language, and Hearing Research*, *56*, 1035–1044. doi: 10.1044/1092-4388

Grossmann, T., Striano, T., & Friederici, A. D. (2006). Crossmodal integration of emotional information from face and voice in the infant brain. *Developmental Science*, *9*, 309–315. doi: 10.1111/j.1467-7687.2006.00494.x

Grych, J. H., & Fincham, F. D. (1990). Marital conflict and children's adjustment: a cognitive-contextual framework. *Psychological Bulletin*, *108*, 267–290. doi: 10.1037/0033-2909.108.2.267

Guyer, A. E., Lau, J. Y., McClure-Tone, E. B., Parrish, J., Shiffrin, N. D., Reynolds, R. C., … & Nelson, E. E. (2008). Amygdala and ventrolateral prefrontal cortex function during anticipated peer evaluation in pediatric social anxiety. *Archives of General Psychiatry*, *65*, 1303–1312. doi: 10.1001/archpsyc.65.11.1303.

Guyer, A. E., McClure-Tone, E. B., Shiffrin, N. D., Pine, D. S., & Nelson, E. E. (2009). Probing the neural correlates of anticipated peer evaluation in adolescence. *Child Development*, *80*, 1000–1015. doi: 10.1111/j.1467-8624.2009.01313.x

Hajal, N. J. (2014). *Neurophysiological and behavioral dynamics of emotion in mothers of young children*. Unpublished doctoral dissertation, The Pennsylvania State University.

Halberstadt, A. G., Denham, S. A., & Dunsmore, J. C. (2001). Affective social competence. *Social Development*, *10*, 79–119. doi: 10.1111/1467-9507.00150

Halberstadt, A. G., Grotjohn, D. K., Johnson, C. A., Furth, M. S., & Greig, M. M. (1992). Children's abilities and strategies in managing the facial display of affect. *Journal of Nonverbal Behavior*, *16*, 215–230. doi: 10.1007/BF01462003

Halligan, S. L., Cooper, P. J., Fearon, P., Wheeler, S. L., Crosby, M., & Murray, L. (2013). The longitudinal development of emotion regulation capacities in children at risk for externalizing disorders. *Development and Psychopathology*, *25*, 391–406. doi: 10.1017/S0954579412001137

Hamilton, C. E. (2000). Continuity and discontinuity of attachment from infancy through adolescence. *Child Development*, *71*, 690–694. doi: 10.1111/1467-8624.00177

Hamilton, J. P., Chen, G., Thomason, M. E., Schwartz, M. E., & Gotlib, I. H. (2010). Investigating neural primacy in major depressive disorder: Multivariate Granger causality analysis of resting-state fMRI time-series data. *Molecular Psychiatry*, *16*, 763–772. doi: 10.1038/mp.2010.46

Hamilton, J. L., Hamlat, E. J., Stange, J. P., Abramson, L. Y., & Alloy, L. B. (2014). Pubertal timing and vulnerabilities to depression in early adolescence: Differential pathways to depressive symptoms by sex. *Journal of Adolescence*, *37*, 165–174. doi: 10.1016/j.adolescence.2013.11.010

Hariri, A. R., & Holmes, A. (2006). Genetics of emotion regulation: The role of the serotonin transporter in neural function. *Trends in Cognitive Science*, *10*, 182–191. doi: 10.1016/j.tics.2006.02.011

Harper, L. V. (2005). Epigenetic inheritance and the intergenerational transfer of experience. *Psychological Bulletin*, *131*, 340–360. doi: 10.1037/0033-2909.131.3.340

Harrison, A., Sullivan, S., Tchanturia, K., & Treasure, J. (2010). Emotional functioning in eating disorders: attentional bias, emotion recognition and emotion regulation. *Psychological Medicine*, *40*, 1887–1897. doi: 10.1017/S0033291710000036

Hart, B., & Risley, T. R. (1995). *Meaningful differences in the everyday experience of young American children*. Baltimore, MD: Paul H. Brookes Publishing.

Harter, S., & Whitesell, N. R. (1989). Developmental changes in children's understanding of single, multiple, and blended emotion concepts. In C. Saarni & P. Harris (Eds.), *Children's understanding of emotion* (pp. 81–116). New York, NY: Cambridge University Press.

Harvey, A., Watkins, E., Mansell, W., & Shafran, R. (2004). *Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment*. New York, NY: Oxford University Press.

Hastings, P. D., & De, I. (2008). Parasympathetic regulation and parental socialization of emotion: Biopsychosocial processes of adjustment in preschoolers. *Social Development*, *17*, 211–238. doi: 10.1111/j.1467-9507.2007.00422.x

Haynos, A. F., & Fruzzetti, A. E. (2011). Anorexia nervosa as a disorder of emotion dysregulation: Evidence and treatment implications. *Clinical Psychology: Science and Practice*, *18*, 183–202. doi: 10.1111/j.1468-2850.2011.01250.x

Heim, C., Shugart, M., Craighead, W. E., & Nemeroff, C. B. (2010). Neurobiological and psychiatric consequences of child abuse and neglect. *Developmental Psychobiology*, *52*, 671–690. doi: 10.1002/dev.20494

Hepach, R., Vaish, A., & Tomasello, M. (2013). Young children sympathize less in response to unjustified emotional distress. *Developmental Psychology*, *49*, 1132–1138. doi: 10.1037/a0029501

Herba, C., & Phillips, M. (2004). Annotation: Development of facial expression recognition from childhood to adolescence: Behavioural and neurological perspectives. *Journal of Child Psychology and Psychiatry*, *45*, 1185–1198. doi: 10.1111/j.1469-7610.2004.00316.x

Herts, K. L., McLaughlin, K. A., & Hatzenbuehler, M. L. (2012). Emotion dysregulation as a mechanism linking stress exposure to adolescent aggressive behavior. *Journal of Abnormal Child Psychology*, *40*, 1111–1122. doi: 10.1007/s10802-012-9629-4

Hertsgaard, L., Gunnar, M., Erickson, M. F., & Nachmias, M. (1995). Adrenocortical responses to the Strange Situation in infants with disorganized/disoriented attachment relationships. *Child Development*, *66*, 1100–1106. doi: 10.1111/j.1467-8624.1995.tb00925.x

Hesse, E., & Main, M. (1999). Second-generation effects of unresolved trauma in nonmaltreating parents: Dissociated, frightened, and threatening parental behavior. *Psychoanalytic Inquiry*, *19*, 481–540. doi: 10.1080/07351699909534265

Hill, A. L., Degnan, K. A., Calkins, S. D., & Keane, S. P. (2006). Profiles of externalizing behavior problems for boys and girls across preschool: The roles of emotion regulation and inattention. *Developmental Psychology*, *42*, 913–928. doi: 10.1037/0012-1649.42.5.913

Hill-Soderlund, A. L., Mills-Koonce, W. R., Propper, C., Calkins, S. D., Granger, D. A., Moore, G. A., … & Cox, M. J. (2008). Parasympathetic and sympathetic responses to the Strange Situation in infants and mothers from avoidant and securely attached dyads. *Developmental Psychobiology*, *50*, 361–376. doi: 10.1002/dev.20302

Hobson, R. P., Patrick, M., Crandell, L., Garcia-Perez, R. O. S. A., & Lee, A. (2005). Personal relatedness and attachment in infants of mothers with borderline personality disorder. *Development and Psychopathology*, *17*, 329–347. doi: 10.1017/S0954579405050169

Hoffner, C., & Badzinski, D. M. (1989). Children's integration of facial and situational cues to emotion. *Child Development*, 411–422. doi: 10.2307/1130986

Holodynski, M., & Friedlmeier, W. (2006). *Development of emotions and emotion regulation*. New York, NY: Springer Science+Business Media.

Homberg, J. R., & Lesch, K. P. (2011). Looking on the bright side of serotonin transporter gene variation. *Biological Psychiatry*, *69*, 513–519. doi: 10.1016/j.biopsych.2010.09.024

Horan, W. P., Wynn, J. K., Kring, A. M., Simons, R. F., & Green, M. F. (2010). Electrophysiological correlates of emotional responding in schizophrenia. *Journal of Abnormal Psychology*, *119*, 18–30. doi: 10.1037/a0017510

Howe, N., Rinaldi, C. M., & Recchia, H. E. (2010). Patterns in mother–child internal state discourse across four contexts. *Merrill-Palmer Quarterly*, *56*, 1–20. doi: 10.1353/mpq.0.0042

Howell, K. H., Graham-Bermann, S. A., Czyz, E., & Lilly, M. (2010). Assessing resilience in preschool children exposed to intimate partner violence. *Violence and Victims*, *25*, 150–164. doi: 10.1891/0886-6708.25.2.150

Hubbard, K., & Trauner, D. A. (2007). Intonation and emotion in autistic spectrum disorders. *Journal of Psycholinguistic Research*, *36*, 159–173. doi: 10.1007/s10936-006-9037-4

Hudson, A., & Jacques, S. (2014). Put on a happy face! Inhibitory control and socioemotional knowledge predict emotion regulation in 5-to 7-year-olds. *Journal of Experimental Child Psychology*, *123*, 36–52. doi: 10.1016/j.jecp.2014.01.012

Hudson, J. L., Comer, J. S., & Kendall, P. C. (2008). Parental responses to positive and negative emotions in anxious and nonanxious children. *Journal of Clinical Child & Adolescent Psychology*, *37*, 303–313. doi: 10.1080/15374410801955839

Huizink, A. C., Robles de Medina, P. G., Mulder, E. J., Visser, G. H., & Buitelaar, J. K. (2002). Psychological measures of prenatal stress as predictors of infant temperament. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*, 1078–1085. doi: 10.1097/00004583-200209000-00008

Isabella, R. A., & Belsky, J. (1991). Interactional synchrony and the origins of infant-mother attachment: A replication study. *Child Development*, *62*, 373–384. doi: 10.1111/j.1467-8624.1991.tb01538.x

Isabella, R. A., Belsky, J., & von Eye, A. (1989). Origins of infant–mother attachment: An examination of interactional synchrony during the infant's first year. *Developmental Psychology*, *25*, 12–21. doi: 10.1037/0012-1649.25.1.12

Izard, C. E. (2011). Forms and functions of emotions: Matters of emotion–cognition interactions. *Emotion Review*, *3*, 371–378. doi: 10.1177/1754073911410737

Izard, C. E. (1977). *Human emotions*. New York, NY: Plenum.

Izard, C. E. (1991). *The psychology of emotions*. New York, NY: Plenum Press.

Izard, C. E. (2002). Translating emotion theory and research into preventive interventions. *Psychological Bulletin*, *128*, 796–824. doi: 10.1037/0033-2909.128.5.796

Izard, C. E., Haynes, O. M., Chisholm, G., & Baak, K. (1991). Emotional determinants of infant-mother attachment. *Child Development*, *62*, 906–917. doi: 10.1111/j.1467-8624.1991.tb01579.x

Izard, C. E., Fantauzzo, C. A., Castle, J. M., Haynes, O. M., Rayias, M. F., & Putnam, P. H. (1995). The ontogeny and significance of infants' facial expressions in the first 9 months of life. *Developmental Psychology*, *31*, 997–1013. doi: 10.1037/0012-1649.31.6.997

Izard, C. E., & Harris, P. (1995). Emotional development and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, 1* (pp. 467–503). Oxford, UK: Wiley

Izard, C. E., King, K. A., Trentacosta, C. J., Laurenceau, J. P., Morgan, J. K., Krauthamer-Ewing, E. S., & Finlon, K. J. (2008). Accelerating the development of emotion competence in Head Start children. *Development and Psychopathology*, *20*, 369–397. doi: 10.1017/S0954579408000175

Izard, C., Stark, K., Trentacosta, C., & Schultz, D. (2008). Beyond emotion regulation: Emotion utilization and adaptive functioning. *Child Development Perspectives*, *2*, 156–163. doi: 10.1111/j.1750-8606.2008.00058.x

Jackson, J., Kuppens, P., Sheeber, L. B., & Allen, N. B. (2011). Expression of anger in depressed adolescents: The role of the family environment. *Journal of Abnormal Child Psychology*, *39*, 463–474. doi: 10.1007/s10802-010-9473-3

Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry*, *53*, 1250–1258. doi: 10.1111/j.1469-7610.2012.02560.x

Jardri, R., Houfflin-DeBarge, V., Delion, P., Pruvo, J. P., Thomas, P., & Pins, D. (2012). Assessing fetal response to maternal speech using a noninvasive functional brain imaging technique. *International Journal of Developmental Neuroscience*, *30*, 159–161. doi: 10.1016/j.ijdevneu.2011.11.002

Johnson, S. L., & Fulford, D. (2009). Preventing mania: A preliminary examination of the goals program. *Behavior Therapy*, *40*, 103–113. doi: 10.1016/j.beth.2008.03.002

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion*, *24*, 281–298. doi: 10.1080/02699930903407948

Karremans, J. C., Heslenfeld, D. J., Van Dillen, L. F., & Van Lange, P. A. (2011). Secure attachment partners attenuate neural responses to

social exclusion: An fMRI investigation. *International Journal of Psychophysiology*, *81*, 44–50. doi: 10.1016/j.ijpsycho.2011.04.003

Katz, L. F., Maliken, A. C., & Stettler, N. M. (2012). Parental meta-emotion philosophy: A Review of research and theoretical framework. *Child Development Perspectives*, *6*, 417–422. doi: 10.1111/j.1750-8606.2012.00244.x

Keenan, K. (2000). Emotion dysregulation as a risk factor for child psychopathology. *Clinical Psychology: Science and Practice*, *7*, 418–434. doi: 10.1093/clipsy.7.4.418

Keenan, K., Hipwell, A., Hinze, A., & Babinski, D. (2009). Equanimity to excess: Inhibiting the expression of negative emotion is associated with depression symptoms in girls. *Journal of Abnormal Child Psychology*, *37*, 739–747. doi: 10.1007/s10802-009-9301-9

Keenan, K., Shaw, D. S., Walsh, B., Delliquadri, E., & Giovannelli, J. (1997). Disorders in preschool children from low-income families. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 620–627. doi: 10.1097/00004583-199705000-00012

Kellam, S. G., Koretz, D., & Mościcki, E. K. (1999). Core elements of developmental epidemiologically based prevention research. *American Journal of Community Psychology*, *27*(4), 463–482.

Keller, H. (2013). *Cultures of infancy*. Mahwah, NJ: Psychology Press.

Kendler, K. S., & Prescott, C. A. (2006). *Genes, environment, and psychopathology: Understanding the causes of psychiatric and substance use disorders*. London, UK: Guilford.

Kiel, E. J., Gratz, K. L., Moore, S. A., Latzman, R. D., & Tull, M. T. (2011). The impact of borderline personality pathology on mothers' responses to infant distress. *Journal of Family Psychology*, *25*, 907–918. doi: 10.1037/a0025474

Kieras, J. E., Tobin, R. M., Graziano, W. G., & Rothbart, M. K. (2005). You can't always get what you want effortful control and children's responses to undesirable gifts. *Psychological Science*, *16*, 391–396. doi: 10.1111/j.0956-7976.2005.01546.x

Killeen, L. A. (2010). *Understanding parenting as a process: Frontal EEG alpha asymmetry as a measure of "online" maternal responsiveness to infant cues*. Doctoral dissertation, The Pennsylvania State University. Retrieved from http://gradworks.umi.com/34/36/3436079.html

Kim, J., & Cicchetti, D. (2010). Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology. *Journal of Child Psychology and Psychiatry*, *51*, 706–716. doi: 10.1111/j.1469-7610.2009.02202.x

Kim, S., & Kochanska, G. (2012). Child temperament moderates effects of parent–child mutuality on self-regulation: A relationship-based path for emotionally negative infants. *Child Development*, *83*, 1275–1289. doi: 10.1111/j.1467-8624.2012.01778.x

Kim-Spoon, J., Cicchetti, D., & Rogosch, F. A. (2013). A longitudinal study of emotion regulation, emotion lability-negativity, and internalizing symptomatology in maltreated and nonmaltreated children. *Child Development*, *84*, 512–527. doi: 10.1111/j.1467-8624.2012.01857.x

Kisilevsky, B. S., & Hains, S. M. (2011). Onset and maturation of fetal heart rate response to the mother's voice over late gestation. *Developmental Science*, *14*, 214–223. doi: 10.1111/j.1467-7687.2010.00970.x

Kisilevsky, B. S., Muir, D. W., & Low, J. A. (1992). Maturation of human fetal responses to vibroacoustic stimulation. *Child Development*, *63*, 1497–1508. doi: 10.1111/j.1467-8624.1992.tb01710.x

Klein Velderman, M., Bakermans-Kranenburg, M. J., Juffer, F., & van IJzendoorn, M. H. (2006). Effects of attachment-based interventions on maternal sensitivity and infant attachment: differential susceptibility of highly reactive infants. *Journal of Family Psychology*, *20*, 266–274. doi: 10.1037/0893-3200.20.2.266

Klimes-Dougan, B., & Zeman, J. (2007). Emotion socialization in childhood and adolescence. *Social Development*, *16*, 203–209. doi: 10.1111/j.1467-9507.2007.00380.x

Klinnert, M. D., Emde, R. N., Butterfield, P., & Campos, J. J. (1986). Social referencing: The infant's use of emotional signals from a friendly adult with mother present. *Developmental Psychology*, *22*, 427–432. doi: 10.1037/0012-1649.22.4.427

Kobak, R. R., Cole, H. E., Ferenz-Gillies, R., Fleming, W. S., & Gamble, W. (1993). Attachment and emotion regulation during mother-teen problem solving: A control theory analysis. *Child Development*, *64*, 231–245. doi: 10.1111/j.1467-8624.1993.tb02906.x

Kobiella, A., Grossmann, T., Reid, V. M., & Striano, T. (2008). The discrimination of angry and fearful facial expressions in 7-month-old infants: An event-related potential study. *Cognition and Emotion*, *22*, 134–146. doi: 10.1080/02699930701394256

Kochanska, G., Coy, K. C., & Murray, K. T. (2001). The development of self-regulation in the first four years of life. *Child Development*, *72*, 1091–1111. doi: 10.1111/1467-8624.00336

Kochanska, G., & Knaack, A. (2003). Effortful control as a personality characteristic of young children: Antecedents, correlates, and consequences. *Journal of Personality*, *71*, 1087–1112. doi: 10.1111/1467-6494.7106008

Kochanska, G., Murray, K. T., & Harlan, E. T. (2000). Effortful control in early childhood: continuity and change, antecedents, and implications for social development. *Developmental Psychology*, *36*, 220–232. doi: 10.1037/0012-1649.36.2.220

Kochanska, G., Philibert, R. A., & Barry, R. A. (2009). Interplay of genes and early mother–child relationship in the development of self-regulation from toddler to preschool age. *Journal of Child Psychology and Psychiatry*, *50*, 1331–1338. doi: 10.1111/j.1469-7610.2008.02050.x

Kok, R., van IJzendoorn, M. H., Linting, M., Bakermans-Kranenburg, M. J., Tharner, A., Luijk, M. P. C. M., … Tiemeier, H. (2013). Attachment insecurity predicts child active resistance to parental requests in a compliance task. *Child: Care, Health and Development*, *39*, 277–287. doi: 10.1111/j.1365-2214.2012.01374.x

Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology*, *18*, 199–214. doi: 10.1037/0012-1649.18.2.199

Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, *25*, 343–354. doi: 10.1037/0012-1649.25.3.343

Kopp, C. B., & Neufeld, S. J. (2003). Emotional development during infancy. In R. Davidson, K.R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 347–374). Oxford, UK: Oxford University Press.

Koss, K. J., George, M. R., Bergman, K. N., Cummings, E. M., Davies, P. T., & Cicchetti, D. (2011). Understanding children's emotional processes and behavioral strategies in the context of marital conflict. *Journal of Experimental Child Psychology*, *109*, 336–352. doi: 10.1016/j.jecp.2011.02.007

Kring, A. M., & Bachorowski, J. A. (1999). Emotions and psychopathology. *Cognition & Emotion*, *13*, 575–599. doi: 10.1080/026999399379195

Kring, A. M., & Moran, E. K. (2008). Emotional response deficits in schizophrenia: Insights from affective science. *Schizophrenia Bulletin*, *3*, 819–834. doi: 10.1093/schbul/sbn071

Krogstad, J. M. (2014). *5 Facts about the modern American family*. Retrieved from Pew Research Center Website: http://www.pewresearch.org/fact-tank/2014/04/30/5-facts-about-the-modern-american-family/

Kuppens, P., Allen, N. B., & Sheeber, L. B. (2010). Emotional inertia and psychological maladjustment. *Psychological Science*, *21*, 984–991. doi: 10.1177/0956797610372634

Kuppens, P., Sheeber, L. B., Yap, M. B., Whittle, S., Simmons, J. G., & Allen, N. B. (2012). Emotional inertia prospectively predicts the onset of depressive disorder in adolescence. *Emotion*, *12*, 283–289. doi: 10.1037/a0025046

Kurjak, A., Azumendi, G., Veček, N., Kupešic, S., Solak, M., Varga, D., & Chervenak, F. (2003). Fetal hand movements and facial expression in normal pregnancy studied by four-dimensional sonography. *Journal of Perinatal Medicine*, *31*, 496–508. doi: 10.1515/JPM.2003.076

Lagattuta, K. H. (2014) *Children and emotion: New insights into developmental affective science*. Basel, Switzerland: Karger.

Lagattuta, K. H. (2007). Thinking about the future because of the past: Young children's knowledge about the causes of worry and preventative decisions. *Child Development*, *78*, 1492–1509. doi: 10.1111/j.1467-8624.2007.01079.x

Lagattuta, K. H. (2008). Young children's knowledge about the influence of thoughts on emotions in rule situations. *Developmental Science*, *11*, 809–818. doi: 10.1111/j.1467-7687.2008.00727.x

Lagattuta, K. H., & Wellman, H. M. (2002). Differences in early parent–child conversations about negative versus positive emotions: Implications for the development of psychological understanding. *Developmental Psychology*, *38*, 564–580. doi: 10.1037/0012-1649.38.4.564

Lamm, C., & Lewis, M. D. (2010). Developmental change in the neurophysiological correlates of self-regulation in high-and low-emotion conditions. *Developmental Neuropsychology*, *35*, 156–176. doi: 10.1080/87565640903526512

Lane, R. D., & Schwartz, G. E. (1987). Levels of emotional awareness: A cognitive-developmental theory and its application to psychopathology. *American Journal of Psychiatry*, *144*, 133–143. Retrieved from: http://psycnet.apa.org/psycinfo/1987-18836-001

Langrock, A. M., Compas, B. E., Keller, G., Merchant, M., & Copeland, M. (2002). Coping with the stress of parental depression: Parents' reports of children's coping, emotional, and behavioral problems. *Journal of Clinical Child & Adolescent Psychology*, *31*, 312–324. doi: 10.1207/S15374424JCCP3103_03

Larochette, A. C., Chambers, C. T., & Craig, K. D. (2006). Genuine, suppressed and faked facial expressions of pain in children. *Pain*, *126*, 64–71. doi: 10.1016/j.pain.2006.06.013

Larsen, J. K., Vermulst, A. A., Eisinga, R., English, T., Gross, J. J., Hofman, E., … & Engels, R. C. (2012). Social coping by masking? Parental support and peer victimization as mediators of the relationship between depressive symptoms and expressive suppression in adolescents. *Journal of Youth and Adolescence*, *41*, 1628–1642. doi: 10.1007/s10964-012-9782-7

Larsen, J. K., Vermulst, A. A., Geenen, R., van Middendorp, H., English, T., Gross, J. J., … Engels, R. C. M. E. (2013). Emotion regulation in adolescence: A prospective study of expressive suppression and depressive symptoms. *Journal of Early Adolescence*, *33*, 184–200. doi: 10.1177/0272431611432712

Larsen, J. T., To, Y. M., & Fireman, G. (2007). Children's understanding and experience of mixed emotions. *Psychological Science*, *18*, 186–191. doi: 10.1111/j.1467-9280.2007.01870.x

Larson, R. W., Moneta, G., Richards, M. H., & Wilson, S. (2002). Continuity, stability, and change in daily emotional experience across adolescence. *Child Development*, *73*, 1151–1165. doi: 10.1111/1467-8624.00464

Larson, R., Raffaelli, M., Richards, M. H., Ham, M., & Jewell, L. (1990). Ecology of depression in late childhood and early adolescence: A profile of daily states and activities. *Journal of Abnormal Psychology*, *99*, 92–102. doi: 10.1037/0021-843X.99.1.92

Lauterbach, E. C., Cummings, J. L., & Kuppuswamy, P. S. (2013). Toward a more precise, clinically—informed pathophysiology of pathological laughing and crying. *Neuroscience & Biobehavioral Reviews*, *37*, 1893–1916. doi: 10.1016/j.neubiorev.2013.03.002

Lavelli, M., & Fogel, A. (2005). Developmental changes in the relationship between the infant's attention and emotion during early face-to-face communication: the 2-month transition. *Developmental Psychology*, *41*, 265–280. doi: 10.1037/0012-1649.41.1.265

Lavigne, J. V., Gouze, K. R., Hopkins, J., Bryant, F. B., & LeBailly, S. A. (2012). A multi-domain model of risk factors for ODD symptoms in a community sample of 4-year-olds. *Journal of Abnormal Child Psychology*, *40*, 741–757. doi: 10.1007/s10802-011-9603-6

Lazarus, R. S. (1991). *Emotion and adaptation*. New York, NY: Oxford University Press.

Lazarus, R. S. (1999). Hope: An emotion and a vital coping resource against despair. *Social Research*, 653–678. Retrieved from: http://www.jstor.org/stable/40971343

LeDoux, J. E. (1996). *The emotional brain*. New York, NY: Simon & Shuster.

LeDoux, J. E. (2012). Evolution of human emotion: A view through fear. *Progress in Brain Research*, *195*, 431–442. doi: 10.1016/B978-0-444-53860-4.00021-0

LeDoux, J. (2012). Rethinking the emotional brain. *Neuron*, *73*, 653–676. doi: 10.1016/j.neuron.2012.02.004

Leerkes, E. M. (2010). Predictors of maternal sensitivity to infant distress. *Parenting: Science and Practice*, *10*, 219–239. doi: 10.1080/15295190903290840

Leerkes, E. M., Blankson, A. N., & O'Brien, M. (2009). Differential effects of maternal sensitivity to infant distress and non-distress on social-emotional functioning. *Child Development*, *80*, 762–775. doi: 10.1111/j.1467-8624.2009.01296.x

Leerkes, E. M., Parade, S. H., & Gudmundson, J. A. (2011). Mothers' emotional reactions to crying pose risk for subsequent attachment insecurity. *Journal of Family Psychology*, *25*, 635–643. doi: 10.1037/a0023654

Leerkes, E. M., & Wong, M. S. (2012). Infant distress and regulatory behaviors vary as a function of attachment security regardless of emotion context and maternal involvement. *Infancy*, *17*, 455–478. doi: 10.1111/j.1532-7078.2011.00099.x

Lewis, M., Ramsay, D. S., & Sullivan, M. W. (2006). The relation of ANS and HPA activation to infant anger and sadness response to goal blockage. *Developmental Psychobiology*, *48*, 397–405. doi: 10.1002/dev.20151

Lewis, M., Stanger, C., & Sullivan, M. W. (1989). Deception in 3-year-olds. *Developmental Psychology*, *25*, 439–443. doi: 10.1037/0012-1649.25.3.439

Lewis, M., & Sullivan, M. W. (2005). The development of self-conscious emotions. In A. J. Elliot & C. S. Dweck (Eds.), *Handbook of competence and motivation* (pp. 185–201). New York, NY: Guilford Press.

Lewis, M., Sullivan, M. W., & Vasen, A. (1987). Making faces: Age and emotion differences in the posing of emotional expressions. *Developmental Psychology*, *23*, 690–697. doi: 10.1037/0012-1649.23.5.690

Lickenbrock, D. M., Braungart-Rieker, J. M., Ekas, N. V., Zentall, S. R., Oshio, T., & Planalp, E. M. (2013). Early temperament and attachment security with mothers and fathers as predictors of toddler compliance and noncompliance. *Infant and Child Development*, *22*, 580–602. doi: 10.1002/icd.1808

Lieberman, M. D., Inagaki, T. K., Tabibnia, G., & Crockett, M. J. (2011). Subjective responses to emotional stimuli during labeling, reappraisal, and distraction. *Emotion*, *11*, 468–480. doi: 10.1037/a0023503

Linehan, M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY: Guilford Press.

Liotti, G. (1999). *Disorganization of attachment as a model for understanding dissociative psychopathology*. New York, NY: Guilford Press.

Lipscomb, S. T., Leve, L. D., Shaw, D. S., Neiderhiser, J. M., Scaramella, L. V., Ge, X., … & Reiss, D. (2012). Negative emotionality and externalizing problems in toddlerhood: Overreactive parenting as a moderator of genetic influences. *Development and Psychopathology*, *24*, 167–179. doi: 10.1017/S0954579411000757

Locke, R. L., Davidson, R. J., Kalin, N. H., & Goldsmith, H. H. (2009). Children's context inappropriate anger and salivary cortisol. *Developmental Psychology*, *45*, 1284–1297. doi: 10.1037/a0015975

Lorber, M. F., & Egeland, B. (2011). Parenting and infant difficulty: Testing a mutual exacerbation hypothesis to predict early onset conduct problems. *Child Development*, *82*, 2006–2020. doi: 10.1111/j.1467-8624.2011.01652.x

Lorber, M. F., & Smith Slep, A. M. (2005). Mothers' emotion dynamics and their relations with harsh and lax discipline: Microsocial time series analyses. *Journal of Clinical Child and Adolescent Psychology*, *34*(3), 559–568. doi: 10.1037/0022-006X.73.5.972

Lord, C., Risi, S., Lambrecht, L., Cook Jr., E. H., Leventhal, B. L., DiLavore, P. C., … & Rutter, M. (2000). The Autism Diagnostic Observation Schedule—Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, *30*, 205–223. doi: 10.1023/A:1005592401947

Loveland, K. A., Tunali-Kotoski, B., Pearson, D. A., Brelsford, K. A., Ortegon, J., & Chen, R. (1994). Imitation and expression of facial affect in autism. *Development and Psychopathology*, *6*, 433–444. doi: 10.1017/S0954579400006039

Lozoff, B., Clark, K. M., Jing, Y., Armony-Sivan, R., Angelilli, M. L., & Jacobson, S. W. (2008). Dose-response relationships between iron deficiency with or without anemia and infant social-emotional behavior. *Journal of Pediatrics*, *152*, 696–702. doi: 10.1016/j.jpeds.2007.09.048

Luby, J. L. (Ed.). (2009). *Handbook of preschool mental health: Development, disorders, and treatment*. New York, NY: Guilford Press.

Lujik, M. P., Saridjan, N., Tharner, A., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Jaddoe, V. W., … & Tiemeier, H. (2010). Attachment, depression, and cortisol: Deviant patterns in insecure-resistant and disorganized infants. *Developmental Psychobiology*, *52*(5), 441–452. doi: 10.1002/dev.20446

Luke, N., & Banerjee, R. (2013). Differentiated associations between child maltreatment experiences and social understanding: A meta-analysis and systematic review. *Developmental Review*, *33*, 1–28. doi: 10.1016/j.dr.2012.10.001

Lunkenheimer, E. S., Shields, A. M., & Cortina, K. S. (2007). Parental emotion coaching and dismissing in family interaction. *Social Development*, *16*, 232–248. doi: 10.1111/j.1467-9507.2007.00382.x

Lyons-Ruth, K., Bronfman, E., & Parsons, E. (1999). Maternal frightened, frightening, or atypical behavior and disorganized infant attachment patterns. *Monographs of the Society for Research in Child Development*, 67–96. doi: 10.1111/1540-5834.00034

Mackintosh, M. A., Morland, L. A., Frueh, B. C., Greene, C. J., & Rosen, C. S. (2014). Peeking into the Black Box: Mechanisms of action for anger management treatment. *Journal of Anxiety Disorders*. doi: 10.1016/j.janxdis.2014.07.001

Main, M., & Solomon, J. (1990). Procedures for identifying infants as disorganized/disoriented during the Ainsworth Strange Situation. In M. T. Greenberg, D. Cicchetti, & E. M. Cummings (Eds.), *Attachment in the preschool years: Theory, research, and intervention* (Vol. 1, pp. 121–160). Chicago, IL: University of Chicago Press.

Malatesta, C. Z., Culver, C., Tesman, J. R., Shepard, B., Fogel, A., Reimers, M., & Zivin, G. (1989). The development of emotion expression during the first two years of life. *Monographs of the Society for Research in Child Development*, 1–136. doi: 10.2307/1166153

Malatesta-Magai, C., Leak, S., Tesman, J., & Shepard, B. (1994). Profiles of emotional development: Individual differences in facial and vocal expression of emotion during the second and third years of life. *International Journal of Behavioral Development*, *17*, 239–269. doi: 10.1177/016502549401700202

Maliken, A. C., & Katz, L. F. (2013). Exploring the impact of parental psychopathology and emotion regulation on evidence-based parenting interventions: A transdiagnostic approach to improving treatment effectiveness. *Clinical Child and Family Psychology Review*, *16*, 173–186. doi: 10.1007/s10567-013-0132-4

Mangelsdorf, S. C., Shapiro, J. R., & Marzolf, D. (1995). Developmental and temperamental differences in emotion regulation in infancy. *Child Development*, *66*, 1817–1828. doi: 10.1111/j.1467-8624.1995.tb00967.x

Martel, M. M. (2009). Research review: A new perspective on attention-deficit/hyperactivity disorder: emotion dysregulation and trait models. *Journal of Child Psychology and Psychiatry*, *50*, 1042–1051. doi: 10.1111/j.1469-7610.2009.02105.x

Martinez-Torteya, C., Dayton, C. J., Beeghly, M., Seng, J. S., McGinnis, E., Broderick, A., … & Muzik, M. (2014). Maternal parenting predicts infant biobehavioral regulation among women with a history of childhood maltreatment. *Development and Psychopathology*, *26*, 379–392. doi: 10.1017/S0954579414000017

Matejka, M., Kazzer, P., Seehausen, M. Bajbouj, M., Klann-Delius, G., Menninghaus, W., … & Prehn, K. (2013). Talking about emotion: Prosody and skin conductance indicate emotion regulation. *Frontiers in Psychology*, *4*, 260. doi: 10.3389/fpsyg.2013.00260

Mayes, L. C. (1999). Developing brain and in utero cocaine exposure: effects on neural ontogeny. *Development and Psychopathology*, *11*, 685–714. doi: 10.1017/S0954579499002278

Mazursky-Horowitz, H., Felton, J. W., MacPherson, L., Ehrlich, K. B., Cassidy, J., Lejuez, C. W., & Chronis-Tuscano, A. (2014). Maternal emotion regulation mediates the association between adult attention-deficit/hyperactivity disorder symptoms and parenting. *Journal of Abnormal Child Psychology*, 1–11. doi: 10.1007/s10802-014-9894-5

McCoy, D. C., & Raver, C. C. (2011). Caregiver emotional expressiveness, child emotion regulation, and child behavior problems among head start families. *Social Development*, *20*, 741–761. doi: 10.1111/j.1467-9507.2011.00608.x

McCrory, E. J., De Brito, S. A., Kelly, P. A., Bird, G., Sebastian, C. L., Mechelli, A., … & Viding, E. (2013). Amygdala activation in maltreated children during pre-attentive emotional processing. *British Journal of Psychiatry*, *202*, 269–276. doi: 10.1192/bjp.bp.112.116624

McCrory, E., De Brito, S. A., & Viding, E. (2012). The link between child abuse and psychopathology: a review of neurobiological and genetic research. *Journal of the Royal Society of Medicine*, *105*, 151–156. doi: 10.1258/jrsm.2011.110222

McDonald, R., Jouriles, E. N., Ramisetty-Mikler, S., Caetano, R., & Green, C. E. (2006). Estimating the number of American children living in partner-violent families. *Journal of Family Psychology*, *20*, 137–142. doi: 10.1037/0893-3200.20.1.137

McDowell, D. J., & Parke, R. D. (2005). Parental control and affect as predictors of children's display rule use and social competence with peers. *Social Development*, *14*, 440–457. doi: 10.1111/j.1467-9507.2005.00310.x

McEwen, B. S. (2000). The neurobiology of stress: from serendipity to clinical relevance. *Brain Research*, *886*, 172–189. doi: 10.1016/S0006-8993(00)02950-4

McIntosh, D. N., Reichmann-Decker, A., Winkielman, P., & Wilbarger, J. L. (2006). When the social mirror breaks: Deficits in

automatic, but not voluntary, mimicry of emotional facial expressions in autism. *Developmental Science*, *9*, 295–302. doi: 10.1111/j.1467-7687.2006.00492.x

McLaughlin, K. A., Hatzenbuehler, M. L., Mennin, D. S., & Nolen-Hoeksema, S. (2011). Emotion dysregulation and adolescent psychopathology: A prospective study. *Behaviour Research and Therapy*, *49*, 544–554. doi: 10.1016/j.brat.2011.06.003

McMahon, E. M., Corcoran, P., McAuliffe, C., Keeley, H., Perry, I. J., & Arensman, E. (2013). Mediating effects of coping style on associations between mental health factors and self-harm among adolescents. *Crisis: Journal of Crisis Intervention and Suicide Prevention*, *34*, 242–250. doi: 10.1027/0227-5910/a000188

Meaney, M. J. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, *81*, 41–79. doi: 10.1111/j.1467-9624.2009.01281.x

Meaney, M. J., Diorio, J., Francis, D., Widdowson, J., LaPlante, P., Caldji, C., … & Plotsky, P. M. (1996). Early environmental regulation of forebrain glucocorticoid receptor gene expression: implications for adrenocortical responses to stress. *Developmental Neuroscience*, *18*, 61–72. doi: 10.1159/000111396

Mennin, D. S., & Fresco, D. M. (2010). Emotion regulation as a framework for understanding and treating anxiety pathology. In A. M. Kring & D. M. Sloan (Eds.), *Emotion regulation in psychopathology* (pp. 356–379). New York, NY: Guilford Press.

Messinger, D., Dondi, M., Christina Nelson-Goens, G., Beghi, A., Fogel, A., & Simion, F. (2002). How sleeping neonates smile. *Developmental Science*, *5*, 48–54. doi: 10.1111/1467-7687.00208

Meyer, U., & Feldon, J. (2010). Epidemiology-driven neurodevelopmental animal models of schizophrenia. *Progress in Neurobiology*, *90*, 285–326. doi: 10.1016/j.pneurobio.2009.10.018

Mikulincer, M., & Shaver, P. R. (2012). An attachment perspective on psychopathology. *World Psychiatry*, *11*, 11–15. doi: 10.1016/j.wpsyc.2012.01.003

Mikulincer, M., Solomon, Z., Shaver, P. R., & Ein-Dor, T. (2014). Attachment-related consequences of war captivity and trajectories of posttraumatic stress disorder: A 17-year longitudinal study. *Journal of Social and Clinical Psychology*, *33*, 207–228. doi: 10.1521/jscp.2014.33.3.207

Mistry, R. S., Benner, A. D., Biesanz, J. C., Clark, S. L., & Howes, C. (2010). Family and social risk, and parental investments during the early childhood years as predictors of low-income children's school readiness outcomes. *Early Childhood Research Quarterly*, *25*, 432–449. doi: 10.1016/j.ecresq.2010.01.002

Molina, W., Reyes, E., Joshi, N., Barrios, A., & Hernandez, L. (2010). Maturation of the neuromuscular junction in masseters of human fetus. *Romanian Journal of Morphology and Embryology*, *51*, 537–541. Retrieved from: http://www.rjme.ro/RJME/resources/files/510310537541.pdf

Monti, J. D., Rudolph, K. D., & Abaied, J. L. (2014). Contributions of maternal emotional functioning to socialization of coping. *Journal of Social and Personal Relationships*, *31*, 247–269. doi: 10.1177/0265407513492304

Moore, G. A. (2009). Infants' and mothers' vagal reactivity in response to anger. *Journal of Child Psychology and Psychiatry*, *50*, 1392–1400. doi: 10.1111/j.1469-7610.2009.02171.x

Morris, A. S., & Silk, J. S. (2001, April). *Parental influences on children's regulation of anger and sadness*. Paper presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

Morris, A. S., Silk, J. S. Morris, M. D. S., Steinberg, L., Aucoin, K. J., & Keyes, A. W. (2011). The influence of mother–child emotion regulation strategies on children's expression of anger and sadness. *Developmental Psychology*, *47*, 213–225. doi: 10.1037/a0021021

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development*, *16*, 361–388. doi: 10.1111/j.1467-9507.2007.00389.x

Moulson, M. C., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2009). Early adverse experiences and the neurobiology of facial emotion processing. *Developmental Psychology*, *45*, 17. doi: 10.1037/a0014035

Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R. H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development*, *67*, 508–522. doi: 10.1111/j.1467-8624.1996.tb01748.x

Nelis, D., Kotsou, I., Quoidbach, J., Hansenne, M., Weytens, F., Dupuis, P., & Mikolajczak, M. (2011). Increasing emotional competence improves psychological and physical well-being, social relationships, and employability. *Emotion*, *11*, 354–366. doi: 10.1037/a0021554

Nelson, C. A., & de Haan, M. (1997). A neurobehavioral approach to the recognition of facial expressions in infancy. In J. A. Russel & J. M. Fernandez-Dols, *The psychology of facial expression* (pp. 176–204). New York, NY: Cambridge University Press.

Nelson, C. A., Westerlund, A., McDermott, J. M., Zeanah, C. H., & Fox, N. A. (2013). Emotion recognition following early psychosocial deprivation. *Development and Psychopathology*, *25*, 517–525. doi: 10.1017/S0954579412001216.

Nolen-Hoeksema, S., & Watkins, E. R. (2011). A heuristic for developing transdiagnostic models of psychopathology explaining multifinality and divergent trajectories. *Perspectives on Psychological Science*, *6*, 589–609. doi: 10.1177/1745691611419672

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomisky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, *3*, 400–424. doi: 10.1111/j.1745-6924.2008.00088.x

Nozadi, S. S., Spinrad, T. L., Eisenberg, N., Bolnick, R., Eggum-Wilkens, N. D., Smith, C. L., … & Sallquist, J. (2013). Prediction of toddlers' expressive language from maternal sensitivity and toddlers' anger expressions: A developmental perspective. *Infant Behavior and Development*, *36*, 650–661. doi: 10.1016/j.infbeh.2013.06.002

O'Donnell, E. H., Moreau, M., Cardemil, E. V., & Pollastri, A. (2010). Interparental conflict, parenting, and childhood depression in a diverse urban population: The role of general cognitive style. *Journal of Youth and Adolescence*, *39*, 12–22. doi: 10.1007/s10964-008-9357-9

O'Donnell, K., O'Connor, T. G., & Glover, V. (2009). Prenatal stress and neurodevelopment of the child: Focus on the HPA axis and the role of the placenta. *Developmental Neuroscience*, *31*, 285–292. doi: 10.1159/000216539

O'Hearn, H. G., Margolin, G., & John, R. S. (1997). Mothers' and fathers' reports of children's reactions to naturalistic marital conflict. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 1366–1373. doi: 10.1097/00004583-199710000-00018

Öhman, A., & Soares, J. J. (1994). "Unconscious anxiety": Phobic responses to masked stimuli. *Journal of Abnormal Psychology*, *103*, 231–240. doi: 10.1037/0021-843X.103.2.231

O'Kearney, R., & Dadds, M. R. (2005). Language for emotions in adolescents with externalizing and internalizing disorders. *Development and Psychopathology*, *17*, 529–548. doi: 10.1017/S095457940505025X

Olino, T. M., Lopez-Duran, N. L., Kovacs, M., George, C. J., Gentzler, A. L., & Shaw, D. S. (2012). Developmental trajectories of positive and negative affect in children at high and low familial risk for depressive disorder. *Journal of Child Psychology and Psychiatry*, *52*, 792–799. doi: 10.1111/j.1469-7610.2010.02331.x

Oosterman, M., De Schipper, J. C., Fisher, P., Dozier, M., & Schuengel, C. (2010). Autonomic reactivity in relation to attachment and early adversity among foster children. *Development and Psychopathology*, *22*, 109–118. doi: 10.1017/S0954579409990290

Oppenheimer, J. E., Measelle, J. R., Laurent, H. K., & Ablow, J. C. (2013). Mothers' vagal regulation during the still-face paradigm: normative reactivity and impact of depression symptoms. *Infant Behavior and Development*, *36*, 255–267. doi: 10.1016/j.infbeh.2013.01.003

Oster, H., Hegley, D., & Nagel, L. (1992). Adult judgments and fine-grained analysis of infant facial expressions: Testing the validity of a priori coding formulas. *Developmental Psychology*, *28*, 1115–1131. doi: 10.1037/0012-1649.28.6.1115

Panskepp, J. (1998). *Affective neuroscience*. New York: Oxford University Press.

Parker, E. H., Hubbard, J. A., Ramsden, S. R., Relyea, N., Dearing, K. F., Smithmyer, C. M., & Schimmel, K. D. (2001). Children's use and knowledge of display rules for anger following hypothetical vignettes versus following live peer interaction. *Social Development*, *10*, 528–557. doi: 10.1111/1467-9507.00179

Pasalich, D. S., Waschbusch, D. A., Dadds, M. R., & Hawes, D. J. (2014). Emotion socialization style in parents of children with callous–unemotional traits. *Child Psychiatry and Human Development*, *45*, 229–242. doi: 10.1007/s10578-013-0395-5

Pechtel, P., & Pizzagalli, D. A. (2011). Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology*, *214*, 55–70. doi: 10.1007/s00213-010-2009-2

Penza-Clyve, S., & Zeman, J. (2002). Initial validation of the Emotion Expression Scale for Children. *Journal of Clinical Child and Adolescent Psychology*, *31*, 540–547. doi: 10.1207/S15374424JCCP3104_12

Peper, J. S., & Dahl, R. E. (2013). The teenage brain: Surging hormones—brain–behavior interactions during puberty. *Current Directions in Psychological Science*, *22*, 134–139. doi: 10.1177/0963721412473755

Perez, S. M., & Gauvain, M. (2005). The role of child emotionality in child behavior and maternal instruction on planning tasks. *Social Development*, *14*, 250–272. doi: 10.1111/j.1467-9507.2005.00301.x

Perlman, S. B., Fournier, J. C., Bebko, G., Bertocci, M. A., Hinze, A. K., Bonar, L., … & Phillips, M. L. (2013). Emotional face processing in pediatric bipolar disorder: Evidence for functional impairments in the fusiform gyrus. *Journal of the American Academy of Child & Adolescent Psychiatry*, *52*, 1314–1325. doi: 10.1016/j.jaac.2013.09.004

Perlman, S. B., Luna, B., Hein, T. C., & Huppert, T. J. (2014). fNIRS evidence of prefrontal regulation of frustration in early childhood. *Neuroimage*, *85*, 326–334. doi: 10.1016/j.neuroimage.2013.04.057

Perlman, S. B., & Pelphrey, K. A. (2010). Regulatory brain development: balancing emotion and cognition. *Social Neuroscience*, *5*, 533–542. doi: 10.1080/17470911003683219

Perry, N. B., Calkins, S. D., Nelson, J. A., Leerkes, E. M., & Marcovitch, S. (2012). Mothers' responses to children's negative emotions and child emotion regulation: The moderating role of vagal suppression. *Developmental Psychobiology*, *54*, 503–513. doi: 10.1002/dev.20608

Pessoa, L. (2005). To what extent are emotional visual stimuli processed without attention and awareness?. *Current Opinion in Neurobiology*, *15*, 188–196. doi: 10.1016/j.conb.2005.03.002

Petrican, R., Moscovitch, M., & Grady, C. (2014). Proficiency in positive vs. negative emotion identification and subjective well-being among long-term married elderly couples. *Frontiers in Psychology*, *5*, 338–363. doi: 10.3389/fpsyg.2014.00338

Piaget, J. (1981). *Intelligence and affectivity: Their relationship during child development*. (Trans & Ed. T. A. Brown & C. E. Kaegi). Oxford, UK: Annual Reviews.

Pietromonaco, P. R., Uchino, B., & Dunkel Schetter, C. (2013). Close relationship processes and health: Implications of attachment theory for health and disease. *Health Psychology*, *32*, 499–513. doi: 10.1037/a0029349

Plutchik, R. (1980). *Emotion: A psychoevolutionary synthesis*. New York, NY: Harper & Row.

Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: developmental effects of child abuse and neglect. *Developmental Psychology*, *36*, 679–688. doi: 10.1037/0012-1649.36.5.679

Pollak, S. D., Messner, M., Kistler, D. J., & Cohn, J. F. (2009). Development of perceptual expertise in emotion recognition. *Cognition*, *110*, 242–247. doi: 10.1016/j.cognition.2008.10.010

Pollak, S. D., & Sinha, P. (2002). Effects of early experience on children's recognition of facial displays of emotion. *Developmental Psychology*, *38*, 784–791. doi: 10.1037/0012-1649.38.5.784

Pons, F., & Harris, P. L. (2005). Longitudinal change and longitudinal stability of individual differences in children's emotion understanding. *Cognition and Emotion*, *19*, 1158–1174. doi: 10.1080/02699930500282108

Posner, M. I., & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology*, *12*, 427–441. Retrieved from http://search.proquest.com/docview/619455347?accountid=13158

Prigatano, G. P. (2013). Denial, anosodiaphoria, and emotional reactivity in anosognosia. *Cognitive Neuroscience*, *4*, 201–202. doi: 10.1080/17588928.2013.854758

Putnam, S. P., Helbig, A. L., Gartstein, M. A., Rothbart, M. K., & Leerkes, E. (2013). Development and assessment of short and very short forms of the Infant Behavior Questionnaire–Revised. *Journal of Personality Assessment*, *96*, 445–458. doi: 10.1080/00223891.2013.841171

Raby, K. L., Cicchetti, D., Carlson, E. A., Cutuli, J. J., Englund, M. M., & Egeland, B. (2012). Genetic and caregiving-based contributions to infant attachment unique associations with distress reactivity and attachment security. *Psychological Science*, *23*, 1016–1023. doi: 10.1177/0956797612438265

Raver, C. (2003). Young children's emotional development and school readiness. *Social Policy Report*, *16*, 3–19. Retrieved from: http://ecap.crc.illinois.edu/eecearchive/digests/2003/raver03.pdf

Raver, C. C., Garner, P. W., & Smith-Donald, R. (2007). The roles of emotion regulation and emotion knowledge for children's academic readiness: Are the links causal? In R. C. Pianta, M. J. Cox, & K. L. Snow (Eds.), *School readiness and the transition to kindergarten in the era of accountability* (pp. 121–147). Baltimore, MD: Brookes.

Raznahan, A., Greenstein, D., Lee, N. R., Clasen, L. S., & Giedd, J. N. (2012). Prenatal growth in humans and postnatal brain maturation into late adolescence. *Proceedings of the National Academy of Sciences*, *109*, 11366–11371. doi: 10.1073/pnas.1203350109

Reeves, C. W., Nicholl, A. R., & Mackenna, J. (2011). Longitudinal analyses of stressors, perceived control, coping, and coping effectiveness among early and middle adolescent soccer players. *International Journal of Sport Psychology*, *42*, 186–203. Retrieved from http://www.cabdirect.org/abstracts/20113203335.html;jsessionid=7AE0E53EAA0159E99879A2AA9BFC379C

Reijntjes, A., Stegge, H., Terwogt, M. M., Kamphuis, J. H., & Telch, M. J. (2006). Emotion regulation and its effects on mood improvement in response to an in vivo peer rejection challenge. *Emotion*, *6*, 543–552. doi: 10.1037/1528-3542.6.4.543

Reilly, J. S., McIntire, M. L., & Bellugi, U. (1990). Faces: The relationship between language and affect. In V. Volterra (Ed.), *From gesture to language in hearing and deaf children* (pp. 128–141). Berlin, Germany: Springer. doi: 10.1007/978-3-642-74859-2_11

Reissland, N., Francis, B., Mason, J., & Lincoln, K. (2011). Do facial expressions develop before birth? *PLoS One*, *6*, e24081. doi: 10.1371/journal.pone.0024081

Renninger, K. A., & Cocking, R. R. (1993). Psychological distance and behavioral paradigms. In R. Cocking & K. Renninger (Eds.), *The development and meaning of psychological distance* (pp. 19–35). Mahwah, NJ: Lawrence Erlbaum.

Repacholi, B. M., & Meltzoff, A. N. (2007). Emotional eavesdropping: Infants selectively respond to indirect emotional signals. *Child Development*, *78*, 503–521. doi: 10.1111/j.1467-8624.2007.01012.x

Repacholi, B. M., Meltzoff, A. N., & Olsen, B. (2008). Infants' understanding of the link between visual perception and emotion: "If she can't see me doing it, she won't get angry." *Developmental Psychology*, *44*, 561–574. doi: 10.1037/0012-1649.44.2.561

Repetti, R. L., & Wood, J. (1997). Effects of daily stress at work on mothers' interactions with preschoolers. *Journal of Family Psychology*, *11*, 90–108. doi: 10.1037/0893-3200.11.1.90

Rice, F., Harold, G. T., Boivin, J., Van den Bree, M., Hay, D. F., & Thapar, A. (2010). The links between prenatal stress and offspring development and psychopathology: disentangling environmental and inherited influences. *Psychological Medicine*, *40*, 335–345. doi: 10.1017/S0033291709005911

Ridgeway, D., Waters, E., & Kuczaj, S. A. (1985). Acquisition of emotion-descriptive language: Receptive and productive vocabulary norms for ages 18 months to 6 years. *Developmental Psychology*, *21*, 901–908. doi: 10.1037/0012-1649.21.5.901

Rivkees, S. A. (2003). Developing circadian rhythmicity in infants. *Pediatrics*, *112*, 373–381. doi: 10.1542/peds.112.2.373

Roben, C. K., Cole, P. M., & Armstrong, L. M. (2013). Longitudinal relations among language skills, anger expression, and regulatory strategies in early childhood. *Child Development*, *84*, 891–905. doi: 10.1111/cdev.12027

Roeser, R. W., Eccles, J. S., & Sameroff, A. J. (1998). Academic and emotional functioning in early adolescence: Longitudinal relations, patterns, and prediction by experience in middle school. *Development and Psychopathology*, *10*, 321–352. doi: 10.1017/S0954579498001631

Röll, J., Koglin, U., & Petermann, F. (2012). Emotion regulation and childhood aggression: Longitudinal associations. *Child Psychiatry & Human Development*, *43*, 909–923. doi: 10.1007/s10578-012-0303-4

Ross, E., & Oliver, C. (2003). The assessment of mood in adults who have severe or profound mental retardation. *Clinical Psychology Review*, *23*, 225–245. doi: 10.1016/S0272-7358(02)00202-7

Roswell, H. C., Ciarrochi, J., Heaven, P. C. L., & Deane, F. P. (2014). The role of emotion identification skill in the formation of male and female friendships: A longitudinal study. *Journal of Adolescence*, *37*, 103–111. doi: 10.1016/j.adolescence.2013.11.005

Rothbart, M. K., & Bates, J. E. (2006). Temperament. In N. Eisenberg (Ed.), *Handbook of child psychology: Vol. 3 Social, emotional and personality development* (Vol. 6, pp. 99–166). New York, NY: Wiley.

Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. L. Brown (Eds.), *Advances in developmental psychology*. (pp. 37–86). Hillsdale, NJ: Erlbaum.

Rothbart, M. K., Posner, M. I., & Kieras, J. (2006). Temperament, attention, and the development of self-regulation. In K. McCartney & D. Phillips (Eds.), *Blackwell handbook of early childhood development. Blackwell handbooks of developmental psychology* (pp. 338–357). Malden, MA: Blackwell Publishing. doi: 10.1002/9780470757703.ch17

Rothbart, M. K., Sheese, B. E., Rueda, M. R., & Posner, M. I. (2011). Developing mechanisms of self-regulation in early life. *Emotion Review*, *3*, 207–213. doi: 10.1177/1754073910387943

Rothbart, M. K., Ziaie, H., & O'Boyle, C. G. (1992). Self-regulation and emotion in infancy. *New Directions for Child Development*, *55*, 7–23. doi: 10.1002/cd.23219925503

Rueda, M. R., Posner, M. I., & Rothbart, M. K. (2005). The development of executive attention: Contributions to the emergence of self-regulation. *Developmental Neuropsychology*, *28*, 573–594. doi: 10.1207/s15326942dn2802_2

Saarni, C. (1979). Children's understanding of display rules for expressive behavior. *Developmental Psychology*, *15*, 424–429. doi: 10.1037/0012-1649.15.4.424

Saarni, C. (1999). *The development of emotional competence*. New York, NY: Guilford Press.

Saarni, C. (1998). Issues of cultural meaningfulness in emotional development. *Developmental Psychology*, *34*, 647–652. doi: 10.1037/0012-1649.34.4.647

Salsman, N. L., & Linehan, M. M. (2012). An investigation of the relationships among negative affect, difficulties in emotion regulation, and features of borderline personality disorder. *Journal of Psychopathology and Behavioral Assessment*, *34*, 260–267. doi: 10.1007/s10862-012-9275-8

Sameroff, A. (2010). A unified theory of development: A dialectic integration of nature and nurture. *Child Development*, *81*, 6–22. doi: 10.1111/j.1467-8624.2009.01378.x

Sameroff, A. E. (Ed). (2009). *The transactional model of development: How children and contexts shape each other*. Washington, DC: American Psychological Association.

Sandman, C. A., & Davis, E. P. (2010). Gestational stress influences cognition and behavior. *Future Neurology*, *5*, 675–690. doi: 10.2217/fnl.10.35

Santucci, A. K., Silk, J. S., Shaw, D. S., Gentzler, A., Fox, N. A., & Kovacs, M. (2008). Vagal tone and temperament as predictors of emotion regulation strategies in young children. *Developmental Psychobiology*, *50*, 205–216. doi: 10.1002/dev.20283

Schneider-Rosen, K., Braunwald, K. G., Carlson, V., & Cicchetti, D. (1985). Current perspectives in attachment theory: Illustration from the study of maltreated infants. *Monographs of the Society for Research in Child Development*, 194–210. doi: 10.2307/3333833

Schudlich, T. D. D. R., White, C. R., Fleischhauer, E. A., & Fitzgerald, K. A. (2011). Observed infant reactions during live interparental conflict. *Journal of Marriage and Family*, *73*, 221–235. doi: 10.1111/j.1741-3737.2010.00800.x

Schwartz, O. S., Dudgeon, P., Sheeber, L. B., Yap, M. B. H., Simmons, J. G., & Allen, N. B. (2012). Parental behaviors during family interactions predict changes in depression and anxiety symptoms during adolescence. *Journal of Abnormal Child Psychology*, *40*, 59–71. doi: 10.1007/s10802-011-9542-2

Sears, M. S., Repetti, R. L., Reynolds, B. M., & Sperling, J. B. (2014). A naturalistic observational study of children's expression of anger in the family context. *Emotion*, *14*, 272–283. doi: 10.1037/a0034753

Shackman, J. E., Fatani, S., Camras, L. A., Berkowitz, M. J., Bachorowski, J. A., & Pollak, S. D. (2010). Emotion expression among abusive mothers is associated with their children's emotion processing and problem behaviors. *Cognition and Emotion*, *24*, 1421–1430.

Shackman, J. E., & Pollak, S. D. (2005). Experiential influences on multimodal perception of emotion. *Child Development*, *76*, 1116–1126. doi: 10.1111/j.1467-8624.2005.00901.x

Shackman, J. E., Shackman, A. J., & Pollak, S. D. (2007). Physical abuse amplifies attention to threat and increases anxiety in children. *Emotion*, *7*, 838–852. doi: 10.1037/1528-3542.7.4.838

Shantz, C. U. (1993). Children's conflicts: Representations and lessons learned. In R. R. Cocking & K. A. Renniger (Eds.), *The development and meaning of psychological distance* (pp. 185–202). Hillsdale, NJ: Erlbaum.

Sheeber, L., Allen, N., Davis, B., & Sorensen, E. (2000). Regulation of negative affect during mother–child problem-solving interactions: Adolescent depressive status and family processes. *Journal of Abnormal Child Psychology*, *28*, 467–479. doi: 10.1023/A:1005135706799

Sheeber, L. B., Kuppens, P., Shortt, J. W., Katz, L. F., & Davis, B. (2012). Depression is associated with the escalation of adolescents' dysphoric behavior during interactions with parents. *Emotion*, *12*, 913–918. doi: 10.1037/a0025784

Shelton, K. H., & Harold, G. T. (2007). Marital conflict and children's adjustment: The mediating and moderating role of children's coping strategies. *Social Development*, *16*, 497–512. doi: 10.1111/j.1467-9507.2007.00400.x

Shenk, C. E., Putnam, F. W., Rausch, J. R., Peugh, J. L., & Noll, J. G. (2014). A longitudinal study of several potential mediators of the relationship between child maltreatment and posttraumatic stress disorder symptoms. *Development and Psychopathology*, *26*, 81–91. doi: 10.1017/S0954579413000916

Sherdell, L., Waugh, C. E., & Gotlib, I. H. (2012). Anticipatory pleasure predicts motivation for reward in major depression. *Journal of Abnormal Psychology*, *121*, 51–60. doi: 10.1037/a0024945

Shiner, R. L., Buss, K. A., McClowry, S. G., Putnam, S. P., Saudino, K. J., & Zentner, M. (2012). What is temperament now? Assessing progress in temperament research on the twenty-fifth anniversary of Goldsmith. *Child Development Perspectives*, *6*, 436–444. doi: 10.1111/j.1750-8606.2012.00254.x

Shipman, K. L., Schneider, R., Fitzgerald, M. M., Sims, C., Swisher, L., & Edwards, A. (2007). Maternal emotion socialization in maltreating and non-maltreating families: Implications for children's emotion regulation. *Social Development*, *16*, 268–285. doi: 10.1111/j.1467-9507.2007.00384.x

Shipman, K., Edwards, A., Brown, A., Swisher, L., & Jennings, E. (2005). Managing emotion in a maltreating context: A pilot study examining child neglect. *Child Abuse & Neglect*, *29*, 1015–1029. doi: 10.1016/j.chiabu.2005.01.00

Sidera, F., Amadó, A., & Serrat, E. (2013). Are you really happy? Children's understanding of real vs. pretend emotions. *Current Psychology: A Journal for Diverse Perspectives on Diverse Psychological Issues*, *32*, 18–31. doi: 10.1007/s12144-012-9159-9

Siffert, A., Schwarz, B., & Stutz, M. (2012). Marital conflict and early adolescents' self-evaluation: The role of parenting quality and early adolescents' appraisals. *Journal of Youth and Adolescence*, *41*, 749–763. doi: 10.1007/s10964-011-9703-1

Silk, J. S., Shaw, D. S., Prout, J. T., O'Rourke, F., Lane, T. J., & Kovacs, M. (2011). Socialization of emotion and offspring internalizing symptoms in mothers with childhood-onset depression. *Journal of Applied Developmental Psychology*, *32*, 127–136. doi: 10.1016/j.appdev.2011.02.001

Silk, J. S., Shaw, D. S., Skuban, E. M., Oland, A. A., & Kovacs, M. (2006). Emotion regulation strategies in offspring of childhood-onset depressed mothers. *Journal of Child Psychology and Psychiatry*, *47*, 69–78. doi: 10.1111/j.1469-7610.2005.01440.x

Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents' emotion regulation in daily life: Links to depressive symptoms and problem behavior. *Child Development*, *74*, 1869–1880. doi: 10.1046/j.1467-8624.2003.00643.x

Silk, J. S., Stroud, L. R., Siegle, G. J., Dahl, R. E., Lee, K. H., & Nelson, E. E. (2012). Peer acceptance and rejection through the eyes of youth: pupillary, eyetracking and ecological data from the Chatroom Interact task. *Social Cognitive and Affective Neuroscience*, *7*, 93–105. doi: 10.1093/scan/nsr044

Simpson, J. A., Collins, W. A., Tran, S., & Haydon, K. C. (2007). Attachment and the experience and expression of emotions in romantic relationships: A developmental perspective. *Journal of Personality and Social Psychology*, *92*, 355–367. doi: 10.1037/0022-3514.92.2.355

Sjogreen, L., Andersson-Norinder, J., & Jacobsson, C. (2001). Development of speech, feeding, eating, and facial expression in Mobius sequence. *International Journal of Pediatric Otorhinolaryngology*, *60*, 197–204. doi: 10.1016/S0165-5876(01)00532-8

Skinner, B. F. (1948). 'Superstition' in the pigeon. *Journal of Experimental Psychology*, *38*, 168–172. doi: 10.1037/h0055873

Skinner, B. F., & Hayes, J. (1976). *Walden two*. New York, NY: Macmillan.

Slatcher, R. B., & Trentacosta, C. J. (2012). Influences of parent and child negative emotionality on young children's everyday behaviors. *Emotion*, *12*, 932–942. doi: 10.1037/a0027148

Slopen, N., McLaughlin, K. A., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2012). Alterations in neural processing and psychopathology in children raised in institutions. *Archives of General Psychiatry*, *69*, 1022–1030. doi: 10.1001/archgenpsychiatry.2012.444.

Smith, M., Hubbard, J. A., & Laurenceau, J. P. (2011). Profiles of anger control in second-grade children: Examination of self-report, observational, and physiological components. *Journal of Experimental Child Psychology*, *110*, 213–226. doi: 10.1016/j.jecp.2011.02.006

Snow, M. E., Hertzig, M. E., & Shapiro, T. (1987). Expression of emotion in young autistic children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *26*, 836–838. doi: 10.1097/00004583-198726060-00006

Snyder, J., Brockman, C., & Stoolmiller, M. (2012). A nonlinear dynamical systems analysis of child emotion displays in relation to family context and child adjustment: a cox hazard approach. *Nonlinear Dynamics, Psychology, and Life Sciences*, *16*, 313–330. Retrieved from: http://europepmc.org/abstract/MED/22695151

Solomon, J., George, C., & De Jong, A. (1995). Children classified as controlling at age six: Evidence of disorganized representational strategies and aggression at home and at school. *Development and Psychopathology*, *7*, 447–463. doi: 10.1017/S0954579400006623

Southam-Gerow, M., & Kendall, P. C. (2002). Emotion regulation and understanding: Implications for child psychopathology and therapy. *Clinical Psychology Review*, *22*, 189–222. doi: 10.1016/S0272-7358(01)00087-3

Spangler, G., & Grossmann, K. E. (1993). Biobehavioral organization in securely and insecurely attached infants. *Child Development*, *64*, 1439–1450. doi: 10.1111/j.1467-8624.1993.tb02962.x

Spangler, G., Schieche, M., Ilg, U., Maier, U., & Ackermann, C. (1994). Maternal sensitivity as an external organizer for biobehavioral regulation in infancy. *Developmental Psychobiology*, *27*, 425–437. doi: 10.1002/dev.420270702

Spangler, G., & Zimmermann, P. (2014). Emotional and adrenocortical regulation in early adolescence Prediction by attachment security and disorganization in infancy. *International Journal of Behavioral Development*, *38*, 142–154. doi: 10.1177/0165025414520808

Spinrad, T. L., Eisenberg, N., Silva, K. M., Eggum, N. D., Reiser, M., Edwards, A., … & Gaertner, B. M. (2012). Longitudinal relations among maternal behaviors, effortful control and young children's committed compliance. *Developmental Psychology*, *48*, 552. doi: 10.1037/a0025898

Sroufe, L. A. (1997). *Emotional development: The organization of emotional life in the early years*. New York: Cambridge University Press.

Steele, J. S., & Ferrer, E. (2011). Latent differential equation modeling of self-regulatory and coregulatory affective processes. *Multivariate Behavioral Research*, *46*, 956–984. doi: 10.1080/00273171.2011.625305

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences*, *9*, 69–74. doi: 10.1016/j.tics.2004.12.005

Stepp, S. D., Scott, L. N., Morse, J. Q., Nolf, K. A., Hallquist, M. N., & Pilkonis, P. A. (2014). Emotion dysregulation as a maintenance factor

of borderline personality disorder features. *Comprehensive Psychiatry*, *55*, 657–666. doi: 10.1016/j.comppsych.2013.11.006

Stifter, C. A., & Braungart, J. M. (1995). The regulation of negative reactivity in infancy: Function and development. *Developmental Psychology*, *31*, 448–455. doi: 10.1037/0012-1649.31.3.448

Stipek, D. J. (1983). A developmental analysis of pride and shame. *Human Development*, *26*, 42–54. doi: 10.1159/000272869

Stranger, B. E., Stahl, E. A., & Raj, T. (2011). Progress and promise of genome-wide association studies for human complex trait genetics. *Genetics*, *187*, 367–383. doi: 10.1534/genetics.110.120907

Sturge-Apple, M. L., Davies, P. T., Cicchetti, D., & Fittoria, M. G. (2014). A typology of interpartner conflict and maternal parenting practices in high-risk families: Examining spillover and compensatory models and implications for child adjustment. *Development and Psychopathology*, 1–16. doi: 10.1017/S0954579414000509

Styers, M. K., & Baker-Ward, L. (2013). Finding the light at the end of the tunnel: Age differences in the relation between internal states terms and coping with potential threats to self. *Memory*, *21*, 27–43. doi: 10.1080/09658211.2012.705849

Sullivan, M. W., Carmody, D. P., & Lewis, M. (2010). How neglect and punitiveness influence emotion knowledge. *Child Psychiatry and Human Development*, *41*, 285–298. doi: 10.1007/s10578-009-0168-3

Supplee, L. H., Skuban, E. M., Trentacosta, C. J., Shaw, D. S., & Stoltz, E. (2011). Preschool boys' development of emotional self-regulation strategies in a sample at risk for behavior problems. *The Journal of Genetic Psychology*, *172*, 95–120. doi: 10.1080/00221325.2010.510545

Suveg, C., Shaffer, A., Morelen, D., & Thomassin, K. (2011). Links between maternal and child psychopathology symptoms: mediation through child emotion regulation and moderation through maternal behavior. *Child Psychiatry & Human Development*, *42*, 507–520. doi: 10.1007/s10578-011-0223-8

Suveg, C., Sood, E., Barmish, A., Tiwari, S., Hudson, J. L., & Kendall, P. C. (2008). "I'd rather not talk about it": Emotion parenting in families of children with an anxiety disorder. *Journal of Family Psychology*, *22*, 875–884. doi: 10.1037/a0012861

Suveg, C., Southam-Gerow, M. A., Goodman, K. L., & Kendall, P. C. (2007). The role of emotion theory and research in child therapy development. *Clinical Psychology: Science and Practice*, *14*, 358–371. doi: 10.1111/j.1468-2850.2007.00096.x

Szajnberg, N. M. (1994). Mobius syndrome: Alternatives in affective communication. *Developmental Medicine and Child Neurology*, *36*, 459–462. doi: 10.1111/j.1469-8749.1994.tb11872.x

Talwar, V., & Lee, K. (2002). Development of lying to conceal a transgression: Children's control of expressive behaviour during verbal deception. *International Journal of Behavioral Development*, *26*, 436–444. doi: 10.1080/01650250143000373

Tan, P. Z., Forbes, E. E., Dahl, R. E., Ryan, N. D., Siegle, G., Ladouceur, C. D., & Silk, J. S. (2012). Emotional reactivity and regulation in anxious and nonanxious youth: A cell phone ecological momentary assessment study. *Journal of Child Psychology and Psychiatry*, *53*, 197–206. doi: 10.1111/j.1469-7610.2011.02469.x

Tarullo, A. R., Bruce, J., & Gunnar, M. R. (2007). False belief and emotion understanding in post-institutionalized children. *Social Development*, *16*, 57–78. doi: 10.1111/j.1467-9507.2007.00372.x

Taumoepeau, M., & Ruffman, T. (2008). Stepping stones to others' minds: Maternal talk relates to child mental state language and emotion understanding at 15, 24, and 33 months. *Child Development 79*, 284–302. doi: 10.1111/j.1467-8624.2007.01126.x

Tenenbaum, L. S., Varjas, K., Meyers, J., & Parris, L. (2011). Coping strategies and perceived effectiveness in fourth through eighth grade victims of bullying. *School Psychology International*, *32*, 263–287. doi: 10.1177/0143034311402309

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, *59*, 25–52. doi: 10.1111/j.1540-5834.1994.tb01276.x

Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., … & Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and difficulties in emotion regulation. *Developmental Science*, *13*, 46–61. doi: 10.1111/j.1467-7687.2009.00852.x

Tragesser, S. L., Solhan, M., Schwartz-Mette, R., & Trull, T. J. (2007). The role of affective instability and impulsivity in predicting future BPD features. *Journal of Personality Disorders*, *21*, 603–614. doi: 10.1521/pedi.2007.21.6.603

Trentacosta, C. J., & Fine, S. E. (2010). Emotion knowledge, social competence, and behavior problems in childhood and adolescence: A meta-analytic review. *Social Development*, *19*, 1–29. doi: 10.1111/j.1467-9507.2009.00543.x

Trentacosta, C. J., & Shaw, D. S. (2009). Emotional self-regulation, peer rejection, and antisocial behavior: Developmental associations from early childhood to early adolescence. *Journal of Applied Developmental Psychology*, *30*, 356–365. doi: 10.1016/j.appdev.2008.12.016

Tronick, E. Z. (1989). Emotions and emotional communication in infants. *American Psychologist*, *44*, 112–119. doi: 10.1037/0003-066X.44.2.112

Tronick, E. Z., Als, H., Adamson, L. B., Wise, S., & Brazelton, T. B. (1978). The infant's response to entrapment between contradictory messages in face-to-face interaction. *Journal of the American Academy of Child Psychiatry*, *17*, 1–13. doi: 10.1016/S0002-7138(09)62273-1

Tronick, E., & Beeghly, M. (2011). Infants' meaning-making and the development of mental health problems. *American Psychologist*, *66*, 107. doi: 10.1037/a0021631

Tronick, E. Z., & Cohn, J. F. (1989). Infant–mother face-to-face interaction: Age and gender differences in coordination and the occurrence of miscoordination. *Child Development*, *60*, 85–92. doi: 10.2307/1131074

Trull, T. J., Solhan, M. B., Tragesser, S. L., Jahng, S., Wood, P. K., Piasecki, T. M., & Watson, D. (2008). Affective instability: Measuring a core feature of borderline personality disorder with ecological momentary assessment. *Journal of Abnormal Psychology*, *117*, 647–661. doi: 10.1037/a0012532

U.S. Department of Health and Human Services, Administration on children, youth and families. (2009). *Child Maltreatment 2007* Washington, DC: U.S. Government Printing Office. Retrieved from: http://archive.acf.hhs.gov/programs/cb/pubs/cm07/cm07.pdf

Vallotton, C., & Ayoub, C. (2011). Use your words: The role of language in the development of toddlers' self-regulation. *Early Childhood Research Quarterly*, *26*, 169–181. doi: 10.1016/j.ecresq.2010.09.002

van den Boom, D. C., & Hoeksma, J. B. (1994). The effect of infant irritability on mother–infant interaction: A growth-curve analysis. *Developmental Psychology*, *30*, 581–590. doi: 10.1037/0012-1649.30.4.581

van der Werff, S. J. A., Pannekoek, J. N., Veer, I. M., van Tol, M. J., Aleman, A., Veltman, D. J., … & van der Wee, N. J. A. (2013). Resting-state functional connectivity in adults with childhood emotional maltreatment. *Psychological Medicine*, *43*, 1825–1836. doi: 10.1017/S0033291712002942

van Harmelen, A. L., van Tol, M. J., van der Wee, N. J., Veltman, D. J., Aleman, A., Spinhoven, P., … & Elzinga, B. M. (2010). Reduced medial prefrontal cortex volume in adults reporting childhood emotional maltreatment. *Biological Psychiatry*, *68*, 832–838. doi: 10.1016/j.biopsych.2010.06.011

van IJzendoorn, M. (1995). Adult attachment representations, parental responsiveness, and infant attachment: A meta-analysis on the predictive validity of the Adult Attachment Interview. *Psychological Bulletin*, *117*, 387–403. doi: 10.1037/0033-2909.117.3.387

van IJzendoorn, M. H., & Bakermans-Kranenburg, M. J. (2012). A sniff of trust: meta-analysis of the effects of intranasal oxytocin administration on face recognition, trust to in-group, and trust to out-group. *Psychoneuroendocrinology*, *37*, 438–443. doi: 10.1016/j.psyneuen.2011.07.008

Vernon-Feagans, L. (2013). Discussion and implications for children living in rural poverty. *Monographs of the Society for Research in Child Development*, *78*, 92–108. doi: 10.1111/mono.12052

Vine, V., & Aldao, A. (2014). Impaired emotional clarity and psychopathology: A transdiagnostic deficit with symptom-specific pathways through emotion regulation. *Journal of Social and Clinical Psychology*, *33*, 319–342. doi: 10.1521/jscp.2014.33.4.319

Volling, B. L. (2010). Early attachment relationships as predictors of preschool children's emotion regulation with a distressed sibling. *Early Education and Development*, *12*, 185–207. doi: 10.1207/s15566935eed1202_2

Volling, B. L., McElwain, N. L., Notaro, P. C., & Herrera, C. (2002). Parents' emotional availability and infant emotional competence: Predictors of parent–infant attachment and emerging self-regulation. *Journal of Family Psychology*, *16*, 447–465. doi: 10.1037/0893-3200.16.4.447

Vondra, J. I., Shaw, D. S., Swearingen, L., Cohen, M., & Owens, E. B. (2001). Attachment stability and emotional and behavioral regulation from infancy to preschool age. *Development and Psychopathology*, *13*, 13–33. doi: 10.1017/S095457940100102X

Vygotsky, L. S. (1986). *Thought and language*. Cambridge MA: MIT Press.

Wadsworth, M. E., Raviv, T., Compas, B. E., & Connor-Smith, J. K. (2005). Parent and adolescent responses to poverty-related stress: Tests of mediated and moderated coping models. *Journal of Child and Family Studies*, *14*, 283–298. doi: 10.1007/s10826-005-5056-2

Wang, M., & Saudino, J. K. (2013). Genetic and environmental influences on individual differences in emotion regulation and its relation to working memory in toddlerhood. *Emotion*, *13*, 1055–1067. doi: 10.1037/a0033784

Warren, H. K., & Stifter, C. A. (2008). Maternal emotion-related socialization and preschoolers' developing emotion self-awareness. *Social Development*, *17*, 239–258. doi: 10.1111/j.1467-9507.2007.00423.x

Waters, E., Merrick, S., Treboux, D., Crowell, J., & Albersheim, L. (2000a). Attachment security in infancy and early adulthood: a twenty-year longitudinal study. *Child Development*, *71*, 684–689. doi: 10.1111/1467-8624.00176

Waters, E., Weinfield, N. S., & Hamilton, C. E. (2000b). The stability of attachment security from infancy to adolescence and early adulthood: General discussion. *Child Development*, *71*, 703–706. doi: 10.1111/1467-8624.00179

Waters, S. F., & Thompson, R. A. (2014). Children's perceptions of the effectiveness of strategies for regulating anger and sadness. *International Journal of Behavioral Development*, *38*, 174–181. doi: 10.1177/0165025413515410

Weinfield, N. S., Sroufe, L. A., & Egeland, B. (2000). Attachment from infancy to early adulthood in a high-risk sample: Continuity, discontinuity, and their correlates. *Child Development*, *71*, 695–702. doi: 10.1111/1467-8624.00178

Wellman, H. M. (2011). *The child's theory of mind*. Cambridge MA: MIT Press.

Werner, E. A., Myers, M. M., Fifer, W. P., Cheng, B., Fang, Y., Allen, R., & Monk, C. (2007). Prenatal predictors of infant temperament. *Developmental Psychobiology*, *49*, 474–484. doi: 10.1002/dev.20232

West, P., Sweeting, H., Young, R., & Kelly, S. (2010). The relative importance of family socioeconomic status and school-based peer hierarchies for morning cortisol in youth: An exploratory study. *Social science & Medicine*, *70*, 1246–1253. doi: 10.1016/j.socscimed.2009.12.006

Widen, S. C., & Russell, J. A. (2003). A closer look at preschoolers' freely produced labels for facial expressions. *Developmental Psychology*, *39*, 114–128. doi: 10.1037/0012-1649.39.1.114

Williams, K.,& McGillicuddy DeLisi, A. V. (1999). Coping strategies in adolescents. *Journal of Applied Developmental Psychology*, *20*, 537–519. doi: 10.1016/S0193-3973(99)00025-8

Wirtz, C. M., Hofmann, S. G., Riper, H., & Berking, M. (2013). Emotion regulation predicts anxiety over a five-year interval: A cross-lagged panel analysis. *Depression and Anxiety*, *31*, 87–95. doi: 10.1002/da.22198

Witherington, D. C., Campos, J. J., & Hertenstein, M. J. (2001). Principles of emotion and its development in infancy. In J. Bremner & A. Fogel (Eds.), *Blackwell handbook of infant development* (pp. 427–464). New York, NY: Blackwell Publishing.

de Wolff, M. S., & van Ijzendoorn, M. H. (1997). Sensitivity and attachment: A meta-analysis on parental antecedents of infant attachment. *Child Development*, *68*, 571–591. doi: 10.1111/j.1467-8624.1997.tb04218.x

Wong, C. F., Silva, K., Kecojevic, A., Schrager, S. M., Bloom, J. J., Iverson, E., & Lankenau, S. E. (2013). Coping and emotion regulation profiles as predictors of nonmedical prescription drug and illicit drug use among high-risk young adults. *Drug and Alcohol Dependence*, *132*, 165–171. doi: 10.1016/j.drugalcdep.2013.01.024

Wörmann, V., Holodynski, M., Kärtner, J., & Keller, H. (2012). A cross-cultural comparison of the development of the social smile: A longitudinal study of maternal and infant imitation in 6-and 12-week-old infants. *Infant Behavior and Development*, *35*, 335–347. doi: 10.1016/j.infbeh.2012.03.002

Yan, F., Dai, S. Y., Akther, N., Kuno, A., Yanagihara, T., & Hata, T. (2006). Four-dimensional sonographic assessment of fetal facial expression early in the third trimester. *International Journal of Gynecology & Obstetrics*, *94*, 108–113. doi: 10.1016/j.ijgo.2006.05.004

Yap, M. B., Allen, N. B., & Ladouceur, C. D. (2008). Maternal socialization of positive affect: The impact of invalidation on adolescent emotion regulation and depressive symptomatology. *Child Development*, *79*, 1415–1431. doi: 10.1111/j.1467-8624.2008.01196.x

Yap, M. B., Schwartz, O. S., Byrne, M. L., Simmons, J. G., & Allen, N. B. (2010). Maternal positive and negative interaction behaviors and early adolescents' depressive symptoms: Adolescent emotion regulation as a mediator. *Journal of Research on Adolescence*, *20*, 1014–1043. doi: 10.1111/j.1532-7795.2010.00665.x

Zalewski, M., Lengua, L. J., Wilson, A. C., Trancik, A., & Bazinet, A. (2011). Associations of coping and appraisal styles with emotion regulation during preadolescence. *Journal of Experimental Child Psychology*, *110*, 141–158. doi: 10.1016/j.jecp.2011.03.001

Zeman, J., & Garber, J. (1996). Display rules for anger, sadness, and pain: It depends on who is watching. *Child Development*, *67*, 957–973. doi: 10.1111/j.1467-8624.1996.tb01776.x

Zeman, J., Klimes-Dougan, B., Cassano, M., & Adrian, M. (2007). Measurement issues in emotion research with children and adolescents. *Clinical Psychology: Science and Practice*, *14*, 377–401. doi: 10.1111/j.1468-2850.2007.00098.x

Zeman, J., & Shipman, K. (1998). Influence of social context on children's affect regulation: A functionalist perspective. *Journal of Nonverbal Behavior*, *22*, 141–165. doi: 10.1023/A:1022900704563

Zeman, J., & Shipman, K. (1997). Social-contextual influences on expectancies for managing anger and sadness: the transition from middle childhood to adolescence. *Developmental Psychology*, *33*, 917–924. doi: 10.1037/0012-1649.33.6.917

Zeskind, P. S. (2013). Infant crying and the synchrony of arousal. In E. Altenmüller, S. Schmidt, & E. Zimmermann (Eds.), *The evolution of emotional communication: from sounds in nonhuman mammals to speech and music in man* (pp. 155–176). Oxford, UK: Oxford University Press.

Zimmer-Gembeck, M. J., & Skinner, E. A. (2011). Review: The development of coping across childhood and adolescence: An integrative review and critique of research. *International Journal of Behavioral Development*, *35*, 1–17. doi: 10.1177/0165025410384923

Zimmer-Gembeck, M. J., Skinner, E. A., Morris, H., & Thomas, R. (2012). Anticipated coping with interpersonal stressors: Links with the emotional reactions of sadness, anger, and fear. *Journal of Early Adolescence*, *33*, 684–709. doi: 10.1177/0272431612466175

Zimmerman, B. (1999). Commentary: toward a cyclically interactive view of self-regulated learning. *Educational Research*, *31*, 545–551. doi: 10.1016/S0883-0355(99)00021-X

Zimmermann, P., & Iwanski, A. (2014). Emotion regulation from early adolescence to emerging adulthood and middle adulthood age differences, gender differences, and emotion-specific developmental variations. *International Journal of Behavioral Development*, *38*, 182–194. doi: 10.1177/0165025413515405

Zimmermann, P., Maier, M. A., Winter, M., & Grossmann, K. E. (2001). Attachment and adolescents' emotion regulation during a joint problem-solving task with a friend. *International Journal of Behavioral Development*, *25*, 331–343. doi: 10.1080/01650250143000157

CHAPTER 8

Attachment and Developmental Psychopathology

R. M. PASCO FEARON, ASHLEY M. GROH, MARIAN J. BAKERMANS-KRANENBURG, MARINUS H. VAN IJZENDOORN, and GLENN I. ROISMAN

OVERVIEW 325
HISTORICAL OVERVIEW OF ATTACHMENT THEORY 326
INDIVIDUAL DIFFERENCES IN ATTACHMENT 328
INTERNAL WORKING MODELS OF ATTACHMENT 331
MEASUREMENT OF INDIVIDUAL DIFFERENCES IN ATTACHMENT 332
Childhood Attachment Assessments 332
Adult Attachment Assessments 334
THE DETERMINANTS OF INDIVIDUAL DIFFERENCES IN ATTACHMENT 335
Intergenerational Transmission 335
Parental Behavior 336
Genes, Environments, and Temperament 340
CONTINUITY AND CHANGE IN ATTACHMENT SECURITY ACROSS THE LIFE COURSE 346
CHILDREN'S ATTACHMENT SECURITY AND PSYCHOPATHOLOGY 349
THE SEARCH FOR MEDIATORS 354
Physiological Mediators 354
Cognitive, Social-Cognitive, and Affective Mediators 357
Emotional Reactivity and Self-regulation 359
ADULT ATTACHMENT AND PSYCHOPATHOLOGY 360
MECHANISMS IN ADULT ATTACHMENT AND PSYCHOPATHOLOGY 363
ATTACHMENT AND INTERVENTION 364
Parenting Interventions 364
Holding and Trauma Therapies 367
Adult Attachment and Reflective Functioning in Interventions 367
Attachment Fit Between Client and Therapist 368
CONCLUSIONS 370
REFERENCES 370

OVERVIEW

Attachment theory is arguably one of the most important theoretical frameworks for understanding normal and abnormal development to have emerged in the field of developmental psychology in the last 60 years. By characterizing the interconnections and mechanisms that mediate between early experiences in primary attachment relationships and long-term emotional adjustment across the life span, attachment theory typifies, and indeed has made major contributions to, the developmental *psychopathology* approach (Cicchetti & Cohen, 1995; Rutter & Sroufe, 2000). The field of attachment—although not without a host of outstanding questions, conundrums, and fruitful seams of new research—could reasonably be described as a mature discipline, having tested, confirmed or clarified many of its major theoretical assertions. The theory remains a highly influential and important way of understanding the nature of a child's tie to primary caregivers, which coherently integrates data and theory from social-developmental psychology, cognitive psychology, life span developmental psychology, evolutionary psychology, and ethology. Its relevance for understanding emotional and behavioral maladjustment, and its value as a framework for *intervention* and prevention, seem undeniable.

In this chapter, our goal is to provide a comprehensive overview of attachment theory and research from its earliest origins to the very latest research findings, focusing on topics of most direct relevance for understanding the development of psychopathology. We begin with an overview

This work was supported in part by a postdoctoral fellowship provided by the National Institute of Child Health and Human Development (T32-HD07376) through the Center for Developmental Science, University of North Carolina at Chapel Hill, to Ashley M. Groh. Marian Bakermans-Kranenburg and Marinus van IJzendoorn were supported by research awards from the Netherlands Organization for Scientific Research (MHvIJ: SPINOZA prize; MJBK: VICI grant no. 453-09-003), and by the Gravitation program of the Dutch Ministry of Education, Culture, and Science and the Netherlands Organization for Scientific Research (NWO grant number 024.001.003).

of the historical beginnings of attachment theory and then outline the fundamental tenets of the theory. Next, we review the empirical literature on the developmental antecedents and consequences of attachment security and insecurity, exploring what is known about mediating mechanisms in the evolution of maladjustment. Next, we turn to work on adolescent and adult attachment. We conclude the chapter with a review of the current state of the art regarding attachment-based interventions and discuss directions for future research.

HISTORICAL OVERVIEW OF ATTACHMENT THEORY

With its primary focus on the nature of the child's tie to caregivers and the significance of these relationships for developmental adaptation across the life course, attachment theory builds on a long intellectual tradition. Indeed, questions concerning the legacy of early interpersonal experiences with primary caregivers predate the origins of formal developmental science and have their roots in the work of Sigmund Freud. Nevertheless, it is widely acknowledged that when John Bowlby began his thinking on the subject of attachment he was beginning a process, involving an intellectually rich and eclectic group of scientists and clinicians, that would lead to the development of a more or less revolutionary theory, radically different to those of the past. Fundamental to this sea change in thinking was his use of evolutionary theory as the basis for his theoretical approach, which was greatly inspired by Harlow's work on the Nature of Love, separating attachment from food and sex (see van der Horst & van der Veer, 2010).

Bowlby was particularly influenced by his experiences as a teacher caring for young children before World War II, which were to have a profound impact on him and his thinking (Bretherton, 1992; van Dijken, 1998). He was to work with children whose remote, or anxious and dependent personalities Bowlby viewed as having been caused by highly disturbed early primary relationships (Bretherton, 1992). He followed up this line of thinking in detailed case studies of 44 delinquent boys, of whom more than half had experienced significant periods of maternal separation during the first five years of life (Bowlby, 1944). After the war, at a time when displacement, loss, and separation were major preoccupations for all concerned with the welfare of children, Bowlby established a research unit dedicated to understanding the effects of separation on children's development. During this period, James Robertson, a psychiatric social worker, had joined Bowlby's team, bringing with him a keen interest in naturalistic observation—a method that, as a consequence particularly of Mary Ainsworth's later work, was to become fundamental to contemporary attachment research. Through extensive observational work, Bowlby and Robertson began to document children's profound response to separation from the mother, which began with protest, followed by despair, and then finally detachment (Bowlby, Robertson, & Rosenbluth, 1952). Such clinical observations raised important questions concerning why children respond so dramatically and predictably to separation from their mothers and what accounted for the intense affective tie between the child and mother. In addressing such questions, Bowlby viewed as inadequate Freud's (e.g., Freud, 1938) drive reduction account of the parent–child relationship in which it was argued that the infant's affective tie to the mother is the secondary result of the mother satisfying the infant's more primary drive for nourishment.

To a significant extent a consequence of this work, in 1949 Bowlby was commissioned by the World Health Organization (WHO) to write a report on the mental health of homeless children. In the course of his research for this report, he immersed himself in what was then known, scientifically and clinically, about separation, foster care, and child psychiatry more broadly, and consulted widely with colleagues in Europe and the United States. He found striking parallels between his own research and observations and those of others in Europe, notably that of his Dutch colleagues Tibout and De Leeuw (see van Dijken, 1998). Regarding the findings of their work, he stated:

> Frequent changes in foster home almost always had very adverse effects, leading the child to become withdrawn and apathetic. This was sometimes accompanied by a superficial sociability and, later, promiscuity. Some young children managed to weather a single change, but others could not stand even this and developed symptoms such as anxiety, depression, excessive clinging, and bedwetting. (pp. 146–147).

The report, once completed, sold 400,000 copies and was translated into 16 different languages, and famously concluded that for optimal development "the infant and young child should experience a warm, intimate, and continuous relationship with his mother figure (or permanent mother substitute) in which both find satisfaction and enjoyment" (Bowlby, 1951, p. 13). In his writing around this time, the first clear evidence also appears that Bowlby had begun to consider the evolutionary origins of attachment very seriously. He was strongly influenced by the earlier work of Konrad Lorenz on imprinting in birds and by Harry Harlow's work on rearing in rhesus monkeys

(Harlow & Zimmermann, 1959). These experiments appeared to demonstrate convincingly that evolutionary forces had favored the evolution of a parental bond to promote survival of young animals and that the child's tendency to cling to a parental figure is a primary instinct and not secondary to some other form of reinforcement such as nutrition.

Thus, drawing on evidence from his own research, and that of others, as well as insights from ethology, cognitive psychology, evolution, and biological systems theory, Bowlby (Bowlby, 1969, 1973, 1980) proposed an alternative account to explain the nature of the child's tie to the mother. According to Bowlby, human infants develop attachments to caregivers because it would have been evolutionarily adaptive to have done so. The infant's intense affective tie to caregivers is not the by-product of a more fundamental drive or motivation. Instead, according to Bowlby, attachment represents a primary, biologically based motivational system, selected through evolution to promote survival and inclusive fitness through maintenance of proximity to a primary caregiver(s) (the neo-Darwinian term inclusive fitness was incorporated into later editions of Bowlby's Attachment and Loss trilogy (see Fraley, Brumbaugh, & Marks, 2005; Simpson & Belsky, 2008).

Drawing on systems theory, Bowlby claimed that attachment behaviors are organized into an attachment behavioral system, in which the specific attachment behaviors are less important than the internal organization of such behaviors toward the goal of proximity. Bowlby adopted a control systems approach in which the attachment behavioral system becomes activated by both internal and external cues that signal potential threat. Once activated, the attachment system recruits a flexible set of behaviors (e.g., crying) organized around the goal of establishing contact with an attachment figure. Once the child's desired proximity to the caregiver is achieved, and a reduction in the perceived degree of threat occurs, there is a decrease in the activation of the attachment system, resulting in the termination of attachment behaviors. Bowlby theorized that the child's desired degree of proximity to the caregiver might depend on a number of factors, including (1) child conditions (e.g., child health), (2) mother conditions (e.g., quality of caregiving), and (3) environmental conditions (e.g., familiarity with the environment).

Bowlby (1969) also provided an account of the normative development of the parent–child attachment relationship, which highlights a hallmark of Bowlby's attachment theory—*universality*. According to attachment theory, essentially all infants are born with the building blocks that develop into the attachment behavioral system. All that is necessary for the infant to develop an attachment to a caregiver is that the caregiver has been interactively present over an extended period of time.

As described by Bowlby (1969), the normative developmental course of this relationship is believed to unfold through the series of four phases (see Marvin & Britner, 2008). Although Bowlby characterized attachment during these periods as undergoing distinctive shifts, he emphasized that the boundary between one phase and the next is blurred. The first phase, *orientation and signals with limited discrimination of figure* (birth to 2–3 months), is characterized by the infant being responsive to all social stimuli. According to Bowlby, infants respond to social stimuli in ways that promote continued social interaction, and attachment behaviors, in particular, are crucial in eliciting caregiving behavior from adults. During this phase, infants' signals are not directed toward any particular person, a feature Bowlby viewed as adaptive, as it would have promoted infant survival, even if the infant's biological caregiver were not available (e.g., if mother died after giving birth). During the second phase, *orientation and signals directed toward a discriminated figure* (2–3 months to 7 months), the infant begins to discriminate specific caregivers from strangers, attachment behaviors are activated and terminated by these specific caregivers, and preference for a particular caregiver begins to develop. The third phase, *maintenance of proximity to a discriminated figure* (7 months to 3 years), is characterized by the emergence of active attachment behaviors that the infant is able to more flexibly alter to achieve the goal of proximity. Goal-corrected behavior begins to be evident, as the infant begins to use the caregiver as a secure base from which to explore the environment and as a safe haven to retreat to in times of threat or challenge. It is early in this phase that the infant is said to become attached to the caregiver in a clear-cut, qualitative sense (and when stranger anxiety emerges). Finally, the fourth phase in the development of the attachment relationship is the *formation of a goal-corrected partnership* (beginning at around 3 years) between the child and caregiver. With the development of language and other cognitive skills, the child becomes able to consider the mother's perspective and consider her wishes in guiding the child's behavior. Thus, the relationship becomes more like a partnership, whereby the child weighs both the parent's and his or her own goals in planning behavior, and attachment needs can increasingly be negotiated collaboratively between parent and child.

Inherent in this view of development is the idea that personality becomes increasingly complex at every stage

of development and that, with development, the child becomes a stronger force in determining developmental pathways (Bowlby, 1969). Thus, Bowlby believed that early social experiences have important implications for subsequent development, with initial adaptations within relationships with primary caregivers serving to lay the foundations for subsequent development. In addition, Bowlby proposed that early experiences color encounters with later experiences through the action of working models, or cognitively embedded abstractions of direct experiences with caregivers. As will be described in more detail, such working models are carried with individuals into novel situations, informing expectations and behavior in interpersonal contexts.

Thus, it is fair to say that in its initial formulations, Bowby's attachment theory was primarily concerned with the fundamental nature of attachment, its evolutionary and biological dimensions, its development, and the impact on personality development of major disruptions to the integrity of the attachment bond, such as permanent or lengthy separation, or bereavement. It was through his collaboration with Mary Ainsworth that the theory broadened to incorporate variations in the quality, or security, of attachment in circumstances where no major disruptions to the integrity of the bond had occurred. This represents a critical turning point for attachment research in two fundamental respects. First, with this new perspective and the now-famous tools that Ainsworth and others developed for rigorously assessing individual differences in the organization or quality of attachment there opened up a highly tractable set of scientific possibilities that were to generate a vast wealth of important findings that has given the field much of its momentum ever since. Second, it represented a shift of focus to a different set of populations—initially children experiencing major separation or privation of attachment experiences, then subsequently to children having experienced a continuous and unbroken bond with their stable caregiver (in most cases their biological parent), but who vary in how their attachment relationship and their own attachment behavior is organized. Crucially, this shift moved the field to focus on fundamentally different aspects of attachment. Bowlby's early thinking was focused on the mechanisms by which a bond is established and what effects occur when it is disrupted. The later work on attachment tended not to address this, and instead examined how attachment behavior toward a consistent caregiver is shaped by that caregiver's behavior, particularly sensitivity.

We can think of these two different lines of inquiry as being fundamentally concerned with *attachment formation* on the one hand, *and attachment organization* on the other. Although research on attachment organization has blossomed, much less is known about the processes that shape attachment formation. Nevertheless, this latter topic is of great significance for attachment researchers and developmental psychopathologists, and has seen a relatively recent resurgence, as scientists have begun to rigorously study the impact of institutional rearing on children's growth and development and interest in the so-called attachment disorders has grown dramatically (e.g., see van IJzendoorn et al., 2011). It is also interesting to note that much, though not all, animal research on attachment has continued in the early tradition—examining the biobehavioral effects of separation and the role of critical periods in early development (Hofer, 1994). Bringing together our understanding of these different aspects of attachment and integrating the methods, findings and insights arising from the study of them is an important long-term goal for the field and a key priority if we are to advance attachment theory's capacity to inform understanding and treatment of psychological disorders. A proper treatment of the impact of institutional rearing, of severe privation more generally, and of the so-called reactive attachment disorders is beyond the scope of this chapter. However, the interested reader is encouraged to study the monographs of Kumsta et al. (2010), McCall, van IJzendoorn, Juffer, Groark, and Groza (2012) and Nelson, Fox & Zeanah (2014), as well as the review paper by Rutter, Kreppner, and Sonuga-Barke (2009).

INDIVIDUAL DIFFERENCES IN ATTACHMENT

As emphasized in the preceding section, Bowlby's major contribution to attachment theory was in the formulation of the basic tenets of the theory. Notwithstanding her important role in refining and elaborating that theory, Ainsworth's key contribution was the development of the methodology for testing it. Her seminal theorizing and groundbreaking research was vital for the field, by making possible the systematic assessment of individual differences in the quality of parent–child attachment relationships (Ainsworth, Blehar, Waters, & Wall, 1978a), and by richly documenting the interactive processes taking place between parent and child that offered a plausible account of the origins of such individual differences (Ainsworth, 1967; Ainsworth et al., 1978a).

Ainsworth's key work, which built upon her earlier observational research in Uganda and drew inspiration from her collaboration with Bowlby and her training in

security theory (Blatz, 1966), was to become a template for how to study the development of parent-infant relationships in naturalistic settings. Critically, it led to the development of what has now become the most widely used procedure for assessing individual differences in the infant–caregiver attachment relationship, the Strange Situation Procedure (Ainsworth & Wittig, 1969; Ainsworth et al., 1978a). This 20-minute procedure originally developed for 12- to 18-month-olds was designed to examine the balance between the infant's attachment and exploratory behavioral systems. Importantly, it capitalizes on several key anxieties that peak in infants at this age, including stranger anxiety and separation anxiety, to observe the infant's use of the caregiver as a secure base and safe haven. Over the course of the Strange Situation, the infant goes through a series of mildly stressful experiences designed to activate the attachment biobehavioral system. The Strange Situation begins with the infant and caregiver being introduced to a novel laboratory setting, with toys for the infant placed in the center of the room and seating for the mother on the perimeter. After 3 minutes, in which the infant is encouraged to play with the toys while the mother is seated at a distance, a stranger enters the room. While the stranger interacts with the infant, the mother departs from the room, leaving the infant in the room with the stranger. After 3 minutes (or less if the infant becomes distressed), the mother returns and the stranger leaves. Shortly thereafter, a second, more stressful separation occurs when the mother leaves the infant alone in the room. After a maximum of 3 minutes alone, the stranger returns, followed thereafter by the mother.

Individual differences in infant attachment in this procedure are revealed in the infants' flexible transition between using the caregiver as a secure base to explore the environment and as a safe haven in times of uncertainty. Based on the patterning of infants' secure base and safe haven behavior—particularly during the two reunion episodes—Ainsworth and her colleagues (1969, 1978a) established that the majority of infants demonstrated what became referred to as a *secure* attachment pattern. Such infants are able to use their caregiver as a secure base of exploration, evidenced in their exploration of toys at a distance from the caregiver in the novel laboratory environment. Although they may check back with caregivers, especially when the stranger enters, they appear confident to explore the environment. When separated from caregivers, infants classified as secure may become distressed, and, if they do, they use the caregiver as a source of effective comfort. Prototypically, the distressed secure infant seeks the caregiver upon reunion, is comforted by his or her presence, and eventually returns to exploration. Even if not overtly distressed by the separation, infants classified as secure tend to acknowledge the return of the caregiver, greeting the caregiver and initiating interaction. Although the term 'secure infant' is often used, the term 'security' more precisely refers to the child's attachment behavior to a particular caregiver and is thus better thought of as a characteristic of their relationship than of the infant per se.

In contrast to secure infants, insecure infants appear unable (or less able) to effectively use the caregiver as a secure base or safe haven. Based on variations in these behavioral themes, Ainsworth and her colleagues identified two types of attachment insecurity—avoidance and resistance. A large minority of insecure infants are classified as avoidant. *Avoidant* infants may explore the environment by engaging the toys before the separation. However, upon reunion, instead of acknowledging the caregiver's return, avoidant infants ignore or actively avoid their caregiver. A small minority of insecure infants are classified as resistant. *Resistant* infants tend to stay close to the caregiver and typically do not engage in play even prior to the first separation episode. When separated from the caregiver, resistant infants often become quite distressed, yet seem unable to use their caregiver effectively as a way of gaining comfort upon reunion. Such infants exhibit a kind of behavioral ambivalence, simultaneously signaling their desire for proximity, yet appear angry and resist contact when it is achieved.

Even though Ainsworth's pioneering study comprised only a small cohort of children, in many respects, this early work—and subsequent research conducted with Everett Waters' Attachment Q-Set (AQS, Waters & Deane, 1985) for characterizing children's secure base behavior in naturalistic settings—has scaled up quite well. Remarkably consistent evidence across all cultures in which individual differences in security have been examined provide evidence that the majority of infants demonstrate secure attachment patterns. In addition, such work has provided evidence that avoidant and resistant patterns of attachment behavior can be reliably identified in a minority of infants across cultures (although the distribution of these classifications has been found to vary both within and between cultures; van IJzendoorn & Sagi-Schwartz, 2008).

Another key contribution of this work is that Ainsworth demonstrated via extensive home observations that the individual differences observed in the laboratory Strange Situation mapped onto individual differences in the parenting children experienced in more naturalistic environments (Ainsworth et al., 1978a). Ainsworth and her colleagues developed behavioral rating scales for systematically

assessing the quality of mother caregiving behavior, which were to become extremely important for subsequent observational studies and for inspiring attachment-focused interventions. Analyses linking such caregiving behavior to infant attachment revealed that mothers who were more sensitive in their interactions with their infant, characterized by their awareness of, and prompt responsiveness to, infant attachment signals, were more likely to have a securely attached infant. Such evidence greatly contributed to theorizing regarding the origins of infant attachment variation. Indeed, virtually all hypotheses regarding the antecedents of individual differences in attachment, at least within attachment scholarship, emphasize environmentally mediated, and in particular, *interpersonal* processes that give rise to secure versus insecure attachment, with the dominant theoretical account being that secure parent–child attachment relationships largely result from sensitive exchanges between caregivers and children. Regarding the origins of the specific patterns of insecurity, it has been argued further that particular types of insensitive parenting give rise to avoidant versus resistant attachment patterns. Specifically, rejecting caregiving is thought to lead to the development of avoidant attachment, as a history of having attachment signals rebuffed by caregivers might understandably lead infants to develop a strategy of minimizing attachment behavior. On the other hand, inconsistently responsive caregiving is thought to promote the development of resistant attachment because such caregiving might lead the infant to develop a strategy of emphasizing or heightening the expression of attachment behavior (see Belsky & Fearon, 2008).

Shortly after Ainsworth and her colleagues introduced the Strange Situation to the field, evidence was presented that attachment behaviors were not highly intercorrelated, exhibited only weak stability over time, and varied across contexts (e.g., Coates, Anderson, & Hartup, 1972; Maccoby & Feldman, 1972), leading some to question not only findings from Ainsworth's work, but also the usefulness of the construct of attachment itself (Cairns, 1972; Gewirtz, 1972a, 1972b; Masters & Wellman, 1974; Rosenthal, 1973; Weinraub, Brooks, & Lewis, 1977). However, these critiques were misguided, focusing on the wrong level of analysis of attachment, at least from the point of view of attachment theorists (Sroufe & Waters, 1977).

The attachment biobehavioral system, according to Bowlby, Ainsworth and others, operates at the level of the goal-directed organization of attachment behavior within the individual, and in a given context. The emphasis thus is not on specific attachment behaviors, but on the patterning of such behaviors and what ends they are organized to achieve. For example, under conditions of threat, the infant may try any number of attachment behaviors (e.g., crying, crawling) to gain proximity to the caregiver, depending in large part on the context and the child's capabilities. Further, the behaviors enlisted by the attachment system are expected to change over the course of development, with, for example distance interaction (including language) becoming more significant in toddlerhood than in infancy (Ainsworth, 1972; Bowlby, 1969; Sroufe & Waters, 1977).

As best articulated by Sroufe and Waters (1977), this distinction between attachment behaviors and the attachment behavioral system is fundamental to the operationalization of individual differences in attachment security. When viewed as an organizational construct, evidence of weak associations among specific attachment behaviors and their weak stability over time and across contexts becomes largely irrelevant to the theory. Instead, what matters is the organization of such behavior around the caregiver within specific contexts (Ainsworth & Bell, 1974). The stability of patterns of attachment as measured at an organizational level is, however, a critical question; one that we return to in a later section.

The standard conceptualization of individual differences in attachment as originally formulated by Ainsworth and her colleagues has also been met with some ultimately generative challenges over the years. As more researchers began to use the Strange Situation, it became increasingly apparent that not all children could be classified as either secure, avoidant, or resistant using the traditional classification system. As first noted by Mary Main and her colleagues (Main & Hesse, 1990; Main & Solomon, 1986, 1990), a subset of infants (especially those living under conditions of high stress or abusive conditions), in addition to exhibiting these organized patterns of attachment behavior in the Strange Situation, also displayed momentary, yet striking, anomalous behaviors suggestive of a "breakdown" or *disorganization* of their attachment-relevant strategy. For example, throughout the course of the Strange Situation, such infants might exhibit stilling or freezing behaviors (i.e., frozen postures believed to represent indication of an approach–avoidance conflict), stereotypies, or marked contradictory behavior (e.g., strong approach followed by sudden avoidance). Or such infants may hit the parent in apparently positive mood or exhibit direct fear of the parent without apparent reason at reunion. In short, disorganization may manifest itself in a variety of ways, yet the common theme is that such displays of conflicted, contradictory, or disoriented behaviors are

indicative of a profoundly disturbed relationship with the attachment figure and of being unable to maintain an organized behavioral strategy when distressed.

Moreover, unlike the organized patterns of attachment (secure, avoidant, resistant) that are thought to result from variation in the sensitivity of caregiving, disorganized attachment is thought to result from frightening (e.g., abusive) or frightened (e.g., caregiver exhibits behavior indicative of apprehension of infant) caregiving. Such caregiving is believed to present disorganized children with an irresolvable dilemma in which the very individuals they are evolutionarily predisposed to seek out under conditions of threat are, in fact, a source of fear for the infant. According to the most influential model of disorganized attachment proposed by Main and Hesse (1990), fear, with the parent as its source, creates for the child an approach–avoidance conflict, in which the mutually opposing action tendencies of proximity seeking and avoidance vie for control of behavior. These opposing tendencies are believed to give rise to the oscillatory, fragmentary or contradictory behaviors characteristic of disorganized attachment.

A second challenge to the validity of the standard view of individual differences in infant attachment security has emerged as a result of more recent taxometric and factor analytic work conducted by Chris Fraley and Susan Spieker (2003) based on the NICHD Study of Early Child Care and Youth Development (SECCYD), one of the largest studies of the Strange Situation conducted to date (original $N = 1{,}364$). Fraley and Spieker's (2003) study focused on the distributional properties and latent structure of individual differences in infant attachment, and findings from their taxometric analyses have provided some evidence that instead of being categorically distributed, variation in attachment security may be compatible with a model in which individual differences in attachment are continuously distributed (see also Kroonenberg & van IJzendoorn, 1987; Richters, Waters, & Vaughn, 1988).

Fraley and Spieker (2003) also presented results from exploratory factor analyses demonstrating that attachment-related individual differences vary along two weakly correlated axes—one of attachment-related avoidance and one of attachment-related resistance (the latter a combination of resistance and disorganization indicators). Such findings challenge the current conceptualization of variation in attachment, in which it is implied that avoidance and resistance are mutually exclusive insecure organizations of attachment behavior. More specifically, such evidence raises the possibility that resistance and disorganization may not be empirically distinct and that an infant can be simultaneously high or low on avoidance and resistance or high on one dimension and low on the other. As discussed further, combined with similar evidence that has emerged from taxometric and factor analytic work (e.g., Fraley & Roisman, 2014; Haltigan, Roisman, & Haydon, 2014; Roisman, Fraley, & Belsky, 2007) on the most widely used developmental measure of adult attachment, the Adult Attachment Interview (AAI; Main, Kaplan, & Cassidy, 1985), we caution that some of the conclusions of prior research might need to be reevaluated in light of such emerging evidence that individual differences in attachment might be best represented by two weakly correlated dimensions reflecting attachment-related avoidance and anxiety. Indeed, one contributing factor to some of the mixed evidence regarding the distinctive antecedents and consequences of attachment variation might be the imperfect relationship between existing measures and their underlying latent structure. This possibility requires further exploration. One key limitation of existing taxometric studies is the reliance on rating scales whose individual validity and sensitivity have received relatively little attention. One particularly critical issue is whether the overlap currently observed in the dimensional framework between resistance and disorganization should be taken as an indication of their common underlying meaning, or a limitation in the way the two constructs are measured. Further work directly contrasting these differing measurement approaches in terms of their distinct or common outcomes or antecedents is an important area for future research (van IJzendoorn & Bakermans-Kranenburg, 2014).

INTERNAL WORKING MODELS OF ATTACHMENT

In contemporary attachment theory, and in keeping with Bowlby's original proposals, attachment behaviors in infancy are thought to be guided by underlying cognitive structures. These internal working models are thought to consist of representations of the parent, the self, and the attachment relationship that are derived from day-to-day experiences with the caregiver. Through repeated interactions the infant is thought to derive a set of interactional schemas or models of the attachment relationship which include sensorimotor, cognitive, and affective components. These representations are seen as a set of expectations regarding the future behavior of the attachment figure that allow the infant to act rapidly and adaptively in the event of threat in accordance with the demands of the

immediate situation and the likely actions of the parent. The Strange Situation, and other attachment assessments, are considered windows into the infant's internal working model of the attachment relationship.

Since Bowlby's early writings on internal working models several researchers have made efforts to recast his ideas in terms derived from modern theories of infant cognition and event representation. Most notable amongst these attempts has been the work of Inge Bretherton (Bretherton, 1990, 1995), who suggested that Bowlby's view of internal working models of attachment is highly compatible with Schank and Abelson's notion of scripts (Schank & Abelson, 1988). In Schank and Abelson's model infants are thought to extract constancies and generalizations from stored memories of repeated events. These invariant aspects of interactions come to form the basis of higher order representations or schemas of the relational structure of interactions. Through a continual process of integration and cross-referencing of new information with existing schema the infant develops a set of horizontally and vertically integrated representations that encode the spatiotemporal structure of common events (Bretherton, 1995). Event-schemas include information that is event-near (specific interaction type) as well as higher level representations that involve generalizations across many event classes (such as general representations of parental accessibility).

The conceptualization of attachment behavior as the functioning of internal working models of attachment has had a major impact on thinking in attachment theory and research and has played a vital role in the understanding of the organization of attachment behavior. Internal working models have been especially important in extending attachment theory beyond infancy into later childhood and adulthood (Greenberg, Cicchetti, & Cummings, 1990; Main, 1991; Main et al., 1985). The most important contribution to this development was made by Main and her colleagues (Main, Goldwyn, & Hesse, 2003; Main et al., 1985). Main and colleagues reformulated the concept of an internal working model from something predominantly behavioral and affective in nature to a representational construct, controlling behavior, thinking, feeling, cognition, and language. More specifically, according to Main et al. (1985, pp. 66–67):

> We define the internal working model of attachment as a set of conscious and/or unconscious rules for the organization of information relevant to attachment and for obtaining or limiting access to that information, that is, to information regarding attachment-related experiences, feelings and ideations.

This crucial reformulation opened up the possibility of investigating the organization and development of internal working models across the life span. The so-called move to the level of representation focused attention on narrative patterns as the expression of the organization and functioning of internal working models of attachment at a cognitive level and has become fundamental for assessing attachment beyond the preschool years.

MEASUREMENT OF INDIVIDUAL DIFFERENCES IN ATTACHMENT

Bowlby (1969) proposed that attachment relationships continue to be relevant across the life course. Initial efforts to assess individual differences in attachment were focused on children, but as interest in attachment across the life span grew, assessments of attachment in adulthood were developed. More recently, there has also been increasing interest in children's attachment representations, leading to a number of representational assessments of attachment being developed for use in early and middle childhood. In this section, we provide an overview of these attachment assessments, specifically focusing on those assessments that are most commonly used within the field of developmental psychology.

Childhood Attachment Assessments

Behavioral Assessments of Attachment

The most commonly used measure of attachment in infancy is the Strange Situation Procedure (Ainsworth & Wittig, 1969; Ainsworth et al., 1978a). As we have already extensively discussed this assessment, we turn our attention here to other behavioral assessments of attachment used in early childhood. In addition to providing an overview of these assessments, we provide evidence regarding the association between individual differences in attachment security as assessed with these attachment measures and as assessed by the Strange Situation. A more extensive discussion of the stability of attachment security across the life course is provided later in this chapter. However, because the Strange Situation generally serves as the standard by which all other attachment assessments are evaluated, we briefly discuss such evidence here when relevant data are available.

Several procedures designed to assess variation in attachment security beyond infancy have been developed that involve modifications to the standard Strange Situation procedure, including the Cassidy-Marvin assessment

of attachment for preschoolers (Cassidy, Marvin, & The MacArthur Working Group on Attachment, 1992) and the Main-Cassidy assessment of attachment for kindergarten-age children (Main & Cassidy, 1988). These modified Strange Situation procedures are similar to the Strange Situation, except that the caregiver and child are separated for a longer duration, a manipulation that is believed to elevate the level of stress in the child. As with the original procedure, in these modified Strange Situations, children's separation and reunion behavior are generally coded using similar categories to those used in the Strange Situation (secure, insecure-avoidant, insecure-resistant, disorganized) based on the child's ability to use the caregiver as a secure base and safe haven. Several longitudinal studies have included measures of both the Strange Situation and these modified Strange Situations, allowing for examination of the concordance between individual differences in attachment as assessed with these measures. Many of these studies have demonstrated stability in attachment security (vs. insecurity), and in studies in which discontinuity was found, it was lawful, in that shifts in security were explained by corresponding changes in mother–child interaction or changes in other attachment-relevant contextual variables (e.g., marital distress and separation, negative life events; Ammaniti, Speranza, & Candelori, 1996; Howes & Hamilton, 1992; Jacobsen, Huss, Fendrich, Kruesi, & Ziegenhain, 1997; Main et al., 1985; Wartner, Grossmann, Fremmer-Bombik, & Suess, 1994).

In contrast to the Strange Situation and modified Strange Situation, in the Attachment Q-Set (AQS, Waters & Deane, 1985), observations are conducted in the home for 2-3 hours or longer. This assessment was developed as an alternative to Ainsworth's extensive home observation narratives collected in the context of her Baltimore study that were used to validate the Strange Situation. Based on home observations, 90 cards reflecting aspects of secure base behavior are sorted into nine piles based on the extent to which the cards describe behavior characteristic of the child's observed behavior. The sort is then compared with a prototypical sort to assess attachment security continuously. A meta-analysis of over 100 investigations comprising over 13,000 children using the AQS conducted by van IJzendoorn and colleagues (van IJzendoorn, Vereijken, Bakermans-Kranenburg, & Riksen-Walraven, 2004) provided important information regarding the validity of this attachment assessment. Findings from this meta-analysis indicated a modest association between security as measured by the Strange Situation and the AQS ($r = .23$), and adequate predictive validity with parental sensitivity (averaging $r = .39$ across studies). The meta-analysis also indicated that the mother-reported AQS is not as psychometrically valid or reliable as the observer-reported AQS. Mother-reported AQS security exhibited a relatively weak association with security as assessed by the Strange Situation ($r = .14$) and a moderate association with temperamental reactivity ($r = .35$).

Representational Assessments of Attachment

As noted already, internal working models are believed to be encoded primarily in sensorimotor representations, making the aforementioned behavioral assessments of attachment particularly appropriate. Indeed, the quality of the attachment relationship is readily observable in the organization of the child's behavior around the caregiver in infancy and early childhood. With increased cognitive capacity, however, fewer stimuli elicit such strong manifestations of attachment behavior, making it increasingly difficult to assess individual differences in the quality of attachment relationships on the basis of the organization of manifest attachment behavior as the child matures. In addition, beginning in the preschool years, children begin to use symbolic forms of representation and to organize knowledge conceptually (Bretherton, 2005), allowing for the use of representational assessments of individual differences in attachment. Accordingly, there is a move in the assessment of individual differences in attachment from the level of behavior in infancy to the level of representation in childhood and adulthood.

A number of representational assessments of attachment have been developed for use in childhood. Such measures largely fall into three categories: (1) picture-response procedures; (2) doll-play scenarios; and (3) family drawings. There are far too many assessments of children's attachment representations to comprehensively review here (but see Solomon & George, 2008); thus, we focus our discussion on a general overview of these three categories of representational assessments. Picture-response procedures involve a series of pictures depicting a range of mildly to moderately stressful attachment-relevant scenarios (e.g., child watches parent leave), to which the child is asked to describe how the child in the scene feels and what the child will do (Klagsbrun & Bowlby, 1976). Both a classification system (Kaplan, 1987) that parallels the one used in the Strange Situation and attachment-relevant rating scales (Slough & Greenberg, 1990) have been developed to code children's responses on this task. Several prompted story-telling procedures using doll play have also been developed (e.g., MacArthur Story

Stem Battery—Bretherton & Oppenheim, 2003; Attachment Story Completion Task—Bretherton, Ridgeway, & Cassidy, 1990; Manchester Attachment Story Task—Goldwyn, Stanley, Smith, & Green, 2000; Attachment Doll-Play Interview—Oppenheim, 1997), and these procedures generally involve the narration and enactment of a series of attachment-relevant stories. Each story contains an attachment-relevant problem that occurs (e.g., child is hurt, parent–child separation). This problem is introduced by the adult, after which the child is prompted to enact the rest of the story. As with the picture response procedures, both classification systems and attachment-relevant scales have been developed to code children's narratives. Finally, in family-drawing procedures, children are asked to draw a picture of their family, and such drawings are evaluated using a classification system that parallels the one developed by Ainsworth for the Strange Situation (Kaplan & Main, 1986).

The diverse approaches to assessing attachment representations in childhood have been beneficial in that multiple measures allow researchers to thoroughly test the construct in a variety of ways. However, there is currently no dominant conceptual or methodological approach, and because researchers have primarily focused on assessment creation, information regarding the reliability and validity of the assessments is limited to only a handful of studies for each measure without independent tests (see Kerns, 2008; Solomon & George, 2008). Although this area of attachment research has produced many potentially useful measures of children's attachment representations, more research focused on the (discriminant) validity of the measures is needed, for example taking careful account of verbal abilities (which otherwise may lead to spurious findings and artificial gender differences).

Adult Attachment Assessments

In adolescence and adulthood, narrative-based measures are the most common way that researchers have approached the assessment of individual differences in attachment. As noted earlier, Mary Main and her colleagues developed the first of such assessments, the Adult Attachment Interview (AAI, George, Kaplan, & Main, 1985), which is a semistructured interview in which adults are asked to describe their early experiences with attachment figures and to provide autobiographical memories to support their evaluations of such experiences. Narratives produced within the context of the AAI are evaluated based on coherence, or the extent to which narratives are internally consistent without becoming emotionally overwrought. Paralleling the Strange Situation, individuals are typically classified by AAI coders into one of three categories. The majority of adults, classified as *secure-autonomous,* produce narratives characterized by the ability to freely and flexibly evaluate childhood experiences, regardless of whether they are described in retrospect as supportive or difficult in nature. A smaller, although substantial, number of adults defensively distance themselves from the emotional content of the interview by normalizing harsh experiences, claiming an inability to recall past experiences or idealizing caregivers and are classified as *insecure-dismissing*. Finally, a small minority of adults are classified as *insecure-preoccupied* and are unable to discuss early attachment-relevant experiences without becoming emotionally overwhelmed, as reflected in the production of narratives characterized by becoming either angrily or passively enmeshed in prior experiences with caregivers. In addition to classifying individuals into one of these mutually exclusive groups, individuals may also be classified as unresolved if their discourse becomes disorganized in the context of discussing the loss of a close loved one or experiences of abuse (see Bakermans-Kranenburg & van IJzendoorn, 2009; Hesse, 2008).

The AAI was developed with the aim of identifying individual differences in parents' representations of early attachment-relevant experiences according to individual differences in the quality of the attachment relationship infants developed with their parent. Accordingly, individual differences in parents' attachment representations as assessed by the AAI would be expected to map onto individual differences in their infant's attachment security. Indeed, a meta-analysis of 18 studies comprising 854 parent–child dyads indicated strong intergenerational continuity in attachment security (vs. insecurity; $r = .47$; van IJzendoorn, 1995).

Recently, Harriet Waters and her colleagues introduced the Attachment Script Assessment (ASA; Waters & Rodrigues-Doolabh, 2004), another narrative-based assessment of adult attachment in which adults are asked to develop attachment-relevant narratives using a set of words provided to them. Narratives produced within the context of the ASA are evaluated for adults' access to a secure-base script, as reflected in adults' ability to develop coherent, nonautobiographical, attachment-relevant narratives. More specifically, narratives are rated on a 7-point scale of secure base script knowledge for the extent to which they are organized around a secure base script. Narratives receive high scores if they contain a clear secure-base structure in which attachment-relevant threats are recognized, competent help from an attachment figure

is offered, such help is effective in resolving the problem, and the situation returns to normal. Narratives receive low scores if they lack this secure-base structure or include bizarre details (e.g., one of the members of the attachment relationship dies; see Waters & Waters, 2006).

In both attachment assessments, the narratives that adults produce, whether in relation to autobiographical attachment-relevant life stories in the case of the AAI or in relation to generic attachment-relevant stories in the case of the ASA, are implicitly evaluated by trained coders for adults' access to a secure attachment-relevant script—or an understanding that effective attachment relationships serve the complementary functions of the provision of a secure base of exploration and a safe haven in times of uncertainty (see Roisman, 2007). Given the similarity between the AAI and the ASA, it is not surprising that, at least in preliminary studies based on relatively modest samples, adults' coherence as measured by the AAI is highly correlated with adults' secure base script knowledge as assessed by the ASA (*r*'s range from .50–.60; H. S. Waters & Waters, 2006). In addition to the conceptual convergence of the AAI and ASA, there is increasing evidence for the empirical convergence of these assessments. Specifically, individual differences in adult attachment as assessed by both the AAI and ASA have been found to predict infant attachment security (Bost et al., 2006; van IJzendoorn, 1995; Verissimo & Salvaterra, 2006); maternal sensitivity (Coppola, Vaughn, Cassibba, & Costantini, 2006; De Wolff & van IJzendoorn, 1997); and physiological reactivity when confronted with attachment-relevant challenges (Beijersbergen, Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2008; Dozier & Kobak, 1992; Groh & Roisman, 2009; Holland & Roisman, 2010; Roisman, Tsai, & Chiang, 2004). Adolescent security (in the AAI) has also been found to correlate with the ASA at age 23 years in a sample of adoptees (Schoenmaker, Juffer, van IJzendoorn, Linting, van der Voort & Bakermans-Kranenburg, 2015). However, more work—in particular, focused on the developmental antecedents of variation in individual differences in attachment as assessed by the ASA—is needed to determine whether the ASA serves as a rough proxy for the AAI, which is a more costly measure in terms of both time and monetary expense (e.g., training, transcription, and coding).

THE DETERMINANTS OF INDIVIDUAL DIFFERENCES IN ATTACHMENT

Since the outset, the causal determinants of variations in attachment behavior have been a central concern of attachment researchers. As noted already, the preeminent causal influences on the development of individual differences in young children's attachments are believed to be located within the patterning or quality of parent–child interactions. Furthermore, contemporary theorizing about the origins of individual differences in attachment place great emphasis on the intergenerational transmission of patterns of attachment from parent to child, driven in large part by the adult's internal working model, or state of mind with respect to attachment (Main et al., 1985; van IJzendoorn, 1995). In the following sections, we review the evidence in support of this, and examine the evidence for alternative explanations including temperament, genetics, and other domains of parental behavior.

Intergenerational Transmission

As noted previously, one of the most robust and well replicated predictors of individual differences in attachment security and insecurity is the parent's state of mind with respect to attachment, as assessed using the Adult Attachment Interview (van IJzendoorn, 1995). van IJzendoorn's (1995) meta-analysis consisted of 18 studies that involved assessments of both parental AAI (either mother or father) and infant security of attachment measured either by the Strange Situation or the AQS. Overall, the net association across studies conducted at that time was very substantial, representing a correlation of .47 (which was homogeneous across studies). Indeed, only two studies in the meta-analysis failed to find cross-generational correspondence between the AAI and the Strange Situation (see van IJzendoorn, 1995). Overall, the three-way match between infant and parental attachment status was 70% (n = 661). The only significant moderator of the effect of overall parental security on infant security was the child's age at the time of the attachment assessment, with smaller effect sizes for groups of older children. The effect of dismissing adult attachment status on infant avoidance was also substantial with an effect size $r = .45$, although there was also significant between-study heterogeneity. Analyses of potential moderators also revealed some interesting differences between studies. Generally speaking maternal dismissing status was more strongly related to infant avoidance than was paternal dismissing classification and, as before, samples of older children produced smaller effect sizes. For parental preoccupation, a marginally smaller although nonetheless highly significant association emerged with infant resistance ($d = .91$, $r = .42$). There was again significant heterogeneity in effect sizes across studies. Two study characteristics appeared to explain some of this

between-study variability: samples of older adults generated smaller effect sizes, as did samples from non-American populations. Finally, there was significant correspondence between parental lack of resolution of mourning and infant disorganized attachment status with a combined effect size of .65, equivalent to a correlation of .31. An analysis based on four-way classifications of the AAI showed similar results as the aforementioned three-way results with the notable exception of the association between parental preoccupation and infant resistance, which was considerably weaker once parental U status and infant disorganization were taken into account ($d = .39$, $r = .19$).

In the same paper, van IJzendoorn (1995) was able to examine the association between parental state of mind with respect to attachment and their observed caregiving behavior, which also revealed quite large effects (meta-analytic average $r = .34$). By the application of the tracing rules of path analysis, van IJzendoorn highlighted an important possible paradox. The direct effect of adult attachment on infant attachment ($r = .47$) was substantially larger than the estimated mediating pathway between adult attachment status to child security via the parent's caregiving behavior (.34 [AAI to caregiving] x .32 [caregiving to attachment, as estimated from a previous meta-analysis] = .11). This led to the coining of the term the *transmission gap*, which refers to the fact that observational measures of parental care seemed unable to account for all of the (very large) association between the AAI and infant attachment security. How to make sense of this transmission gap has been, and continues to be, a key question for attachment research. In the next section, we examine in greater detail the evidence concerning the role of *parental sensitivity* in the development of attachment as it currently stands, and explore possible explanations for the so-called transmission gap. Before doing so, we should also note that it seems prudent, given the amount of time that has elapsed since this meta-analytic work, to assume that the effect size for the AAI–SSP link will have declined somewhat (Verhage et al., 2015).

Parental Behavior

Since Ainsworth's seminal work, the sensitivity of parental care has long been considered the primary determinant of variations in child attachment security. In Ainsworth's ground-breaking study (Ainsworth et al., 1978) she carefully documented, in great detail, and from lengthy observations in the home, the key features of parent–child interactions that appeared to distinguish secure from insecure attachments. A critical insight of Ainsworth's, which paralleled Bowlby's insight regarding the organizational nature of attachment, was that specific caregiving behaviors were less important than the way in which parental responses to the child's signals were appropriately matched to the particular needs and characteristics of the child as well as the situational and temporal context. In that sense, Ainsworth's conception of sensitivity is clearly an organizational construct, pertaining to the caregiving system rather than the attachment system (see George & Solomon, 1999). Ainsworth defined sensitivity as the parent's ability to be appropriately responsive to the child's attachment cues, and distinguished four aspects:

1. Awareness of the child's cues
2. Appropriate interpretation of the child's cues
3. Timeliness of the parent's response
4. Appropriateness of the parent's response

According to Ainsworth, a parent scoring high on sensitivity is continually aware of the child's whereabouts, state, focus of attention, activities and feelings and so is able to detect quite subtle attachment cues, understand the context in which they occur, and notice the incipient signs of distress so that prompt and in some cases preemptive action can be taken. This awareness is no doubt partly what allows a sensitive parent to come to appropriate interpretations of what the attachment signals mean. On the other hand, insensitive parents, according to Ainsworth, may be aware of the child's cues but interpret them inaccurately and negatively, perhaps as a result of their own past experiences, biases, or defenses (Ainsworth et al., 1978). The kinds of interpretations Ainsworth had in mind here included those regarding the infant's subjective experience or mental state. Indeed, the parent's ability to think about and hold in mind the child's experience and to take that appropriately into account when attending to the child's needs was central to Ainsworth, Bell and Stayton's (1974) thinking on sensitivity (pp. 127–128):

> Interpretation has three main components (a) her awareness, . . . , (b) her freedom from distortion, and (c) her empathy . . . . . The mother must be able to empathize with her baby's feelings and wishes before she can respond with sensitivity.

The elaboration of the sensitivity construct to encompass the parent's mind-mindedness, insightfulness, or reflective function is therefore a very natural evolution of Ainsworth's original thinking.

Even if a parent has noticed the infant's attachment cue, and correctly interpreted it, the parental response

needs to be timely so that, from the infant's point of view, it is experienced as contingent upon his or her signal. According to Ainsworth the infant needs to be able to connect, in time, his signal with the mother's response so he feels a sense of efficacy in having his needs met. Finally, the parental response itself needs to be appropriate and provide the infant with what it seems he needs. According to Ainsworth, these aspects of sensitivity collectively form steps in a chain of events that allows a parent to respond optimally to the child's attachment-related signals and communications. A thorough understanding of Ainsworth's definition of sensitivity is important when considering some of the complexities of the literature concerning the link between sensitivity and attachment. It is also essential when considering how attachment concepts can be translated into effective interventions aimed at promoting the quality of parental care.

Since Ainsworth's early observations, which led to a grounded hypothesis of a strong connection between attachment and sensitivity, an impressive database of findings has emerged, broadly confirming her original work (De Wolff & van IJzendoorn, 1997). As the field developed and differentiated, a wide range of ways of measuring sensitivity arose, and the constructs that were chosen as foci for investigating the antecedents of attachment broadened. Although the outcome of this work was not entirely uniform, it has become clear that secure attachment is associated with higher parental sensitivity, a finding replicated across numerous countries and social contexts (Belsky & Fearon, 2008). Summarizing the extant findings of the time, and following-up findings of previous work by Goldsmith and Alansky (1987), De Wolff and van IJzendoorn's (1997) meta-analysis of studies of sensitivity and attachment came to several key conclusions. First, across all studies included in the meta-analysis (k = 30, N = 1,666) the average correlation between sensitivity and attachment security was .22, which was highly significant, but also highly heterogeneous (i.e., the effect size varied substantially across studies to a degree greater than expected by chance). The broad trend across studies was thus entirely in keeping with Ainsworth's hypothesis, although for some scholars the effect size was interpreted to be smaller than expected. A second important finding to emerge from this meta-analysis was that when different characterizations of the sensitivity construct were analyzed separately, those studies that used Ainsworth's original definition (and rating scales) produced somewhat larger effect sizes (r = .24) than those more distantly related to the original. Notably, this focused set of studies was also homogeneous, meaning that across studies the variability in effect sizes was not greater than would be expected as a result of chance fluctuations, and no study characteristics were identified that moderated the effect size. Within the broader set of sensitivity studies however, a number of important moderators did arise. First, studies with older infants tended to produce larger effect sizes (older than age 1, r = .27) than those with younger infants (r = .20). Similarly, studies that measured attachment at later ages tended to obtain larger associations (r = .25) than those with younger age groups (r = .19), as did studies with shorter time intervals between the assessment of sensitivity and attachment. These results might suggest that the attachment relationship, as indicated by parental sensitivity on the mother's side, and attachment security on the child's, coheres progressively over time (De Wolff & van IJzendoorn, 1997). As we will describe further, the long-term predictive significance of attachment also appears to strengthen as attachment is measured later in development, which could also be taken to imply a gradual consolidating process taking place sometime after the first or second year. One notable further development in the field is an increase in interest in the role of paternal sensitivity in the development of father–child attachment. A recent meta-analysis of eight studies found a significant overall association (r = .13), although the effect size was clearly smaller than the meta-analytic average for mothers (Lucassen et al., 2011). A recent paper by Brown, Mangelsdorf, and Neff (2012) found more robust associations between attachment and paternal sensitivity at age 3 (β = .53) than in infancy (β = .33), which mirrors the pattern referred to previously regarding mothers (De Wolff & van IJzendoorn, 1997). Whether this pattern is more marked in fathers is worthy of exploration, but currently we do not have direct evidence of this.

Despite the fact that meta-analytic work has identified study-level variables that seem to be associated with larger effect sizes, it is clear that at best the association between sensitivity and attachment is modest, and there may be several explanations for this (Belsky & Fearon, 2008). First, the modest association may result from a *technological gap*, resulting from limitations in the quality, intensity, or context of measurement of sensitivity. There is no doubt that measurements of sensitivity (and indeed attachment) are noisy, and the extent of noise in any measure sets an upper limit on its capacity to correlate with a criterion (Shrout & Fleiss, 1979). In that regard, work by Dallaire and Weinraub (2005) is notable. They analyzed data on maternal sensitivity across the first six years of life, using the NICHD Study of Early Child Care and Youth Development data set and found that although correlations

between specific sensitivity indicators at one time point and the next were consistently in the $r = .30–.40$ range, latent variables representing common variance among several indicators correlated at around $r = .60$. This latent variable approach removes some, though not all, of the measurement error in sensitivity assessments, and suggests that sensitivity may be a highly stable construct once error is partialed out. Furthermore, it indirectly suggests that the modest association between sensitivity and attachment might be substantially higher than is commonly assumed. If one assumes that only 70% of the variance in measurements of attachment and sensitivity is reliable (which may be generous given that reported interrater reliabilities are often close to this figure and this is only one source of unreliability), then a correlation of .24 rises to .34 after correction for attenuation. Higher levels of measurement error would of course lead to larger increases in the association after correction for attenuation. Measurement error is a (sometimes underappreciated) key issue when interpreting results from observational studies (see also De Wolff & van IJzendoorn, 1997). These considerations also underline the importance of continuing to refine measurement protocols for attachment related constructs, so that more stable and reliable measurements can be obtained. A particularly important study in that regard was recently conducted by Lindheim, Bernard and Dozier (2011), who undertook 10 repeated home observations of sensitivity in a small ($N = 25$), high-risk sample of mothers and babies. Critically, they found insecure/nonautonomous mothers, as assessed with the AAI, showed greater variability in their sensitivity than secure/autonomous mothers, and that the lowest, but not the highest, level of sensitivity observed during the repeated assessments discriminated nonautonomous from autonomous mothers. The same was true with regard to the discrimination of infants who were removed from the home due to child protection concerns. Lindheim et al. (2011) also plotted the degree of association between sensitivity and maternal state of mind as a function of the number of observation episodes, and found that it rose in a curvilinear fashion (increasing rapidly initially, then plateauing), from $r = .37$ for a single observation to $r = .54$ after ten observations. The steepest increment in predictive validity was observed between one and two observations, and inspection of the curve would suggest that three observations may represent the optimal balance between predictive validity and researcher burden. Though only a single study and in a small and high-risk sample, this study powerfully demonstrates the importance of studying the measurement properties of sensitivity assessments to minimize error. The same logic applies, of course, to measurements of attachment. In that context it may be significant that van IJzendoorn and colleagues (van IJzendoorn et al., 2004) found the correlation between security derived from the AQS and the SSP to be stronger for studies that used longer observations when conducting the AQS ($r = .23$ for <1.5 hours, $r = .42$ for > 1.5 hours). Further work of this nature is vital for scientific studies aiming to reveal the etiological mechanisms driving the development of attachment and for practitioners who need to be able to reliably assess attachment and sensitivity as part of their routine work.

Aside from measurement error, other technological limitations may hamper efforts to find strong and consistent associations between sensitivity and attachment. One critical factor has been highlighted by two recent studies, which both indicate that sensitivity-attachment associations are stronger when the measurement context affords opportunity to observe the parent's sensitivity to the child's distress (Gaensbauer, Connell, & Schulz, 1983; McElwain & Booth-LaForce, 2006). In the first (McElwain & Booth-LaForce, 2006), maternal responsiveness to distress at 6 months cues was found to be a somewhat stronger predictor of attachment than responsiveness to nondistress cues. However, as noted by Leerkes (2011), a substantial proportion of the sample did not become distressed during the observation and were therefore excluded from the analysis. To address this limitation, Leerkes (2011) observed mothers in two different tasks, one free play, the other involving two brief distress-eliciting tasks (each 4 minutes in duration, one fear-evoking, the other frustration-evoking). As predicted, sensitivity during the distress tasks was more robustly associated with attachment security than sensitivity in the free play context. Along similar lines, several studies have suggested that sensitivity may be a stronger predictor when the observational context places competing demands on the parent (Smith & Pederson, 1988). Such contexts may increase the frequency of distress cues emitted by the child, as well as possibly stretching parents' capacities in ways that may mimic common attachment and sensitivity experiences outside of the laboratory, and hence increasing ecological validity. Because of the extensive observations in the natural setting, the Maternal Behavior Q-set (MBQS; see Pederson et al., 1990) might nicely combine the two facets that seem important for valid sensitivity measurements: observation of infant distress episodes in combination with competing demands on the parent. This may in turn explain the apparently larger associations between attachment and sensitivity produced by studies using the MBQS. Interestingly though, when Atkinson and

colleagues (2005) examined the contributions of sensitivity, as measured using the MBQS, and maternal security derived from the AAI, they did find strong associations between each of these and infant security, but they did not find strong evidence of mediation. In other words, despite the stronger correlation between sensitivity and attachment when measured this way, sensitivity and adult attachment security were partially, if not largely, independent. As a consequence, despite the substantial variance in security being accounted for overall, a transmission gap nevertheless remained. As a rejoinder to this, the authors also noted that in both the samples they analyzed (one using the MBQS, and another using an event-coding of a short lab observation), there was some indication that sensitivity and parental state of mind might interact to predict attachment security. In particular, sensitive parenting tended to block the transmission of insecure patterns from parent to child (Atkinson et al., 2005).

Another relatively clear-cut piece of evidence seeming to implicate methodological factors in the modest association between attachment and sensitivity is the notable stronger association between attachment and sensitivity when attachment is measured using the Attachment Q-Set (average $r = .39$, see van IJzendoorn et al., 2004). The AQS differs in several respects to the Strange Situation which may be relevant in interpreting this stronger association. First, it is based on a longer period of observation of behavior (typically 1.5–2 hours), and is conducted in the home environment. The AQS may therefore capture a larger sample of representative attachment behavior than the Strange Situation. However, another important consideration is the age at which the AQS is usually conducted, which is generally between the ages of 2 and 3. As noted already, there is reason to believe that the association between attachment and sensitivity becomes more robust with age. Currently, it is unclear which of these interpretations is the more accurate, although of course they are not mutually exclusive.

The second general class of explanations for the modest association between sensitivity and attachment could be termed the *domain gap* (Belsky & Fearon, 2008). What is intended here is consideration of ways novel aspects of parental behavior, partially but not fully overlapping with the sensitivity construct, might more closely match the causal inputs to the development of security and insecurity and therefore reveal stronger concurrent or predictive associations in observational studies. Although one could imagine a potentially wide ranging search for such candidates, there has been particular interest in recent years in operationalizations of sensitivity related to the parent's tendency to imagine and make sense of their child's mental states, such as their thoughts, feelings, or desires. For example, Slade and colleagues (Slade, Grienenberger, Bernbach, Levy, & Locker, 2005) found that parental reflective functioning as coded from verbatim transcripts of the Parent Development Interview at 10 months predicted 14-month attachment security, as well as the mother's parenting (Kelly, Grienenberger, & Slade, 2005). Focusing on a related construct in two separate studies, Oppenheim, Koren-Karie and colleagues found maternal insightfulness (e.g., the sophistication of thinking regarding the child's thoughts and feelings, the degree of insight into the child's motives) to predict the infant's later attachment security (Koren-Karie, Oppenheim, Dolev, Sher, & Etzion-Carasso, 2002; Oppenheim, Koren-Karie, & Sagi, 2001). Meins and colleagues have conducted perhaps the most comprehensive series of studies on the role of parental *mind-mindedness* and attachment, by coding spontaneous mind-related statements that parents make during their interactions. Four separate studies (Laranjo, Bernier, & Meins, 2008; Lundy, 2003; Meins, Fernyhough, Fradley, & Tuckey, 2001; Meins et al., 2012) have found maternal mind-mindedness to be predictive of attachment security. In all four of these studies, the typical effects sizes were consistently and substantially higher than the meta-analytic average reported in De Wolff & van IJzendoorn's original article (Laranjo et al.: $r = .41$; Lundy: $r = .58$ and .60 for mothers and fathers, respectively; Meins et al., 2001: $r = .45$; Meins et al., 2012: approx., $r = .54$), although such work was, of course, based on small or moderate sample sizes.

These studies are promising in pointing toward domains of parental care that may more robustly predict infant attachment security. However, by focusing on patterns of speech (during interactions or during semistructured interviews) these studies beg the question of the immediate, proximal, behavioral mediators. Rather than consider these studies as describing a novel domain of parenting—conceptualized at the same level of analysis as sensitivity—it is arguably more appropriate to consider them as social-cognitive processes residing in the parent, whose behavioral correlates, as they affect attachment, remain to be determined.

A third explanation of the modest association between attachment and sensitivity is that the connection is moderated by other third variables (the *moderator gap*; Belsky & Fearon, 2008). Some examples of course were already discussed in the context of the De Wolff and van IJzendoorn meta-analysis. In that meta-analysis, evidence emerged that sensitivity may be more strongly associated with attachment in middle-class samples ($r = .28$) than in lower

socioeconomic (SES) samples ($r = .15$), a finding echoed in at least one more recent study (Meins et al., 2012). As noted already, genetic factors have been suggested as possible moderators of the association between sensitivity and attachment, but the robustness of these findings may be limited (Luijk et al., 2011). A recent study nevertheless found some evidence in a relatively large sample that the sensitivity–attachment association may be moderated by genetic variability in the mineralocorticoid receptor gene (Luijk et al., 2011). Furthermore, there is reason to believe that sensitivity-focused interventions may be more successful in enhancing security for temperamentally reactive infants (see especially Cassidy, Woodhouse, Sherman, Stupica, & Lejuez, 2011), but whether the same is true in typical development is currently unclear. We referred to another example of moderation previously, in the study by Atkinson et al. (2005), but in this case the moderation was by sensitivity itself: high-levels of sensitivity (presumably arising for reasons partially unrelated to parental security) attenuated the association between insecure parental attachment status and the security of the parent-infant attachment relationship.

The clearest example of a domain gap arises in the context of disorganized attachment, where studies have consistently revealed a weak association with traditionally conceived sensitivity (meta-analytic average, $r = .10$, van IJzendoorn, Schuengel, & Bakermans-Kranenburg, 1999). There is quite consistent evidence that disorganized attachment is markedly more common among infants and children who have experienced maltreatment (Cyr, Euser, Bakermans-Kranenburg, & van IJzendoorn, 2010; van IJzendoorn et al., 1999). However, research just as clearly shows that other factors also play a role in disorganized attachment, particularly anomalous patterns of parental care. The evidence implicating anomalous parental behavior in disorganization is relatively clear, with the basic association having been found consistently in at least 10 independent studies (Abrams, Rifkin, & Hesse, 2006; Goldberg, Benoit, Blokland, & Madigan, 2003; Jacobvitz, Leon, & Hazen, 2006; Kelly et al., 2005; Lyons-Ruth, Bronfman, & Parsons, 1999; Madigan, Benoit, & Boucher, 2011; Madigan, Moran, & Pederson, 2006; Schuengel, Bakermans-Kranenburg, & van IJzendoorn, 1999; Tomlinson, Cooper, & Murray, 2005; True, Pisani, & Oumar, 2001). A meta-analytic review of the evidence published in 2006 (Madigan et al., 2006) found the overall association between anomalous parental behavior and disorganized attachment to be equivalent to a correlation of $r = .34$. It is worth bearing in the mind that while the original theorizing regarding disorganized attachment focused on fear, and particularly parental frightening or frightened behavior (FR), the broader term *anomalous parenting* includes a variety of disturbances in the affective qualities of parental behavior beyond the FR construct, including parental dissociative behavior, disturbances in affective communication, role-reversal, and more broadly defined extreme insensitivity (see Out, Bakermans-Kranenburg, & van IJzendoorn, 2009). Thus far, few studies have been conducted that have simultaneously measured all of these to determine their relative importance or to test whether they are independently predictive of disorganization. In one recent study, Out and colleagues (2009) found that only the constellation of parenting behaviors falling under the category of frightened, frightening or dissociative behavior was predictive of disorganization and not extreme insensitivity (e.g., harsh, highly intrusive parenting). This finding is broadly consistent with a tendency for dissociative behavior to be the numerically stronger predictor of disorganization in some studies (e.g., Abrams et al., 2006; Madigan, Moran et al., 2006; Schuengel et al., 1999). In addition to anomalous parental behavior, it is also notable that a recent study found that parental mind-mindedness, particularly nonattuned mind-related comments, was quite strongly predictive of attachment disorganization (Meins et al., 2012).

There is thus rather strong evidence regarding the primary determinants of disorganized attachment, although the causal status of the association between disorganization and anomalous parental behavior cannot be inferred from these data alone. Reasons to be optimistic that the association might be a causal one are provided by results of a longitudinal study by Forbes and colleagues (Forbes, Evans, Moran, & Pederson, 2007), which examined cross-lagged associations between anomalous parental behavior and disorganization from 12 to 24 months. The critical finding in this investigation was that earlier anomalous parental behavior predicted subsequent disorganization, even after prior disorganization was statistically controlled for. However, causal status is most powerfully tested using experimental studies. Thus far, intervention studies have shown that rates of disorganized can be improved through therapeutic intervention (as we will review), but whether the mechanisms of action involve changes in parental frightening, frightened and dissociative behavior remains to be definitively established.

Genes, Environments, and Temperament

Attachment theory provides a clear and coherent model of the mechanisms by which variations in the quality of

parental care influence the child's attachment organization, and subsequently biases the child's socioemotional development in systematic ways. In light of the findings of contemporary behavioral genetics research, which has documented strong and pervasive genetic influences on emotion, personality, and cognition right across the life span (Plomin, DeFries, Knopik, & Neiderhiser, 2013), attachment theory makes rather bold predictions about the environmental causation of individual differences in attachment security and insecurity (O'Connor, Croft, & Steele, 2000).

Questions about the environmental or genetic causes of attachment security and insecurity originally centered on the role of temperament (for a review, see Vaughn, Bost, & van IJzendoorn, 2008). In particular, temperament researchers argued that differences in secure-base behavior observed in the strange situation are in fact constitutionally based differences in proneness to distress. Associations with maternal sensitivity, according to this view, simply represent the noncausal parenting correlates of infant temperament (e.g., Calkins & Fox, 1992). Goldsmith and Alansky (1987) carried out an early meta-analysis of 18 studies that had assessed infant temperament and infant-parent attachment and found that temperament predicted resistant behavior in the Strange Situation. However, the effect size was small and no association was found for overall attachment classification. Hints of associations between attachment and measures of temperament have come and gone in more recent research. For example, Kagan (1994) subsequently found resistant infants to be overrepresented in groups of infants who showed high levels of behavioral inhibition in early infancy. In Van den Boom's study (van den Boom, 1990; 1994), highly irritable infants were more likely to be classified as avoidant at 12 months than less irritable infants. Belsky (1996) on the other hand reported that only one association out of eight comparisons showed a relationship between temperament and attachment with father and none for attachment with mother. Mangesldorf et al. (Mangelsdorf, Gunnar, Kestenbaum, Lang, & Andreas, 1990) found no direct links with temperament but one out of four possible interactions between temperament and maternal personality. Difficult infant temperament, in conjunction with high maternal constraint, seemed to lead to insecure infant-mother attachment at 12 months. In fact there is some suggestion that difficult temperament may represent a risk factor for the development of insecure attachments. Crockenberg (1981), for example, found that temperament was associated with attachment classification only in the context of low maternal social support. The data regarding temperamental influences on attachment are certainly mixed. The interpretation of any associations, however qualified or moderated, also needs to be tempered by a recognition that temperament itself is influenced by both genetic and environmental factors, and hence cannot be straightforwardly attributed to endogenous causes (van IJzendoorn & Bakermans-Kranenburg, 2012).

Thus, broadly speaking, the debate regarding the role of temperament led to an accumulation of evidence that quite clearly underscored the rather distinctive nature of attachment, and the limited associations between attachment and temperament. However, such studies do not directly address the question of genetic or environmental causes of variation in security and insecurity. Only behavioral-genetic studies are capable of doing so. The first twin study of attachment using a standardized assessment tool was conducted by O'Connor and Croft (2001). This study used the Cassidy and Marvin (Cassidy et al., 1992) modified Strange Situation Procedure in 125 3.5 year-old twin pairs, and found substantial correspondence between twins in attachment security (67% for security). When twin similarity was assessed separately for monozygotic (MZ; identical) and dizygotic (DZ; fraternal) twins using a continuous measure of security, the correlation was .48 for MZ twins, and .38 for DZ twins. Quantitative genetic analysis of these patterns of twin-twin similarity led to estimates of 32% for the shared environment, 53% for the nonshared environment 53%, and 14% for genetic influences. In the latter case, the genetic effect was not significantly different to zero. Given the more limited validation (in relative terms) of the preschool attachment assessment compared with what in some respects might be considered the gold standard attachment assessment (12-month Strange Situation), as well as the modest sample size, these findings, while highly consistent with attachment theory, were open to question, and certainly required replication.

Bokhorst and colleagues (Bokhorst et al., 2003) conducted a somewhat larger twin study ($N = 157$ twin pairs) using the standard 12-month Strange Situation in two parallel groups seen in labs in Leiden and London. Overall similarity between twins for attachment security was 56% for MZ twins and 60% for DZ twins. Quantitative genetic modelling estimated that 52% of the variance was attributable to the shared environment and 48% to the nonshared environment (plus measurement error), leaving the estimate of heritability at zero. The findings were thus quite consistent with O'Connor and Croft's study (2001), but indicated even more strongly the role of the environment, and particularly the shared environment. Subsequently, a third twin study was completed

by Roisman and Fraley (2008), using a modified version of the Attachment Q-sort (conducted in the home) in a substantially larger sample of twins aged 2 years (485 twin pairs). The heritability estimate of the continuous security score from the modified AQS was .17 and nonsignificant. The shared environment, by contrast, was estimated to account for 53% of the variance in security, and 36% was attributable to nonshared environment and measurement error.

Despite the differing ages and methodologies, the three studies are remarkably consistent in documenting strong evidence of the primacy of environmental influences in the development of individual differences in attachment security, and collectively reflect a rather resounding confirmation of a key assumption of attachment theory. It is important to also note that the levels of twin correspondence in these studies converges remarkably well with studies of nontwin siblings (Teti & Ablard, 1989; van IJzendoorn et al., 2000; Ward, Vaughn, & Robb, 1988), which goes some considerable way to addressing concerns that the twin design might in some way lack validity or generalizability in this context. Furthermore, each of these studies used rigorous observational measures with strict blindness protocols, so that coders knew nothing about the co-twin's attachment security or twin zygosity.

Of course, attachment theory also makes specific predictions regarding the nature of the environmental influences on attachment security, and so to be fully consistent with theory the environmental influences on attachment security, particularly the shared environmental influences, should be traceable to variation in the sensitivity of care. This key corollary of the theory was first examined by Fearon and colleagues (Fearon et al., 2006). Using the same twin sample referred to already, Fearon et al. (2006) coded home observations of maternal sensitivity obtained at 10 months, and related these to infant attachment security in the Strange Situation at 12 months. Maternal sensitivity in this study was found to be highly consistent between twins regardless of their zygosity, indicating sensitivity to be a highly shared environmental variable. As predicted, when the shared environmental variance in sensitivity was isolated from the nonshared variance (there was no genetic variance in sensitivity), it correlated significantly with the shared environmental component of attachment security. In other words, a significant portion of the shared environmental influences on attachment was attributable to similarities in how sensitive, or insensitive, mothers were to both of their infants. Roisman and Fraley (2008) subsequently replicated this finding using home observations of parenting from the Early Childhood Longitudinal Study-Birth cohort.

Behavioral genetic methods have proved to be key tools for testing the critical environmental hypotheses of attachment theory, and have produced remarkably consistent, and cumulatively persuasive, evidence that these basic assumptions are robust. Several further issues regarding the etiology of attachment warrant consideration. First, as indicated before, the nonshared environment includes measurement error, which is likely to be quite significant for assessments of attachment, and hence estimates of the shared environment are almost certainly *underestimates*. Second, standard genetic modelling of twin data does not take account of gene–environment interaction. When ignored, gene × shared environment interactions are estimated as genetic effects in the twin design, and gene × nonshared environment interactions are estimated as nonshared variance (Johnson, 2010). This is important for two reasons. First, although genetic effects (e.g., observed in other domains of behavior) could, and probably do, mask gene-by-shared environment interactions, shared environmental influences, such as those observed to influence attachment, represent pure environmental effects. Second, in the only study that attempted to estimate the heritability of disorganized attachment (Bokhorst et al., 2003), marked evidence of nonshared environmental influence was found. Although this could be taken to indicate twin or sibling-specific environmental influences (such as parental differential treatment), it could also reflect interactions between genes and the nonshared environment. When these considerations are set alongside the fact that very limited numbers of disorganized cases have been subjected to quantitative genetic analysis, it is appropriate to conclude that uncertainty still remains about the genetic and environmental influences on disorganized attachment.

This latter caveat takes on particular significance in light of the fact that some studies have reported associations between disorganized attachment and directly measured genetic polymorphisms. The first of these was conducted by Lakatos and colleagues (Lakatos et al., 2000), who, in a community sample of 90 infants, examined the association between disorganized attachment and a polymorphism of the dopamine D4 receptor gene. Of 17 infants classified as disorganized, 12 (71%) had a variant of this gene containing 7 or more repeats of a 48 base pair sequence in the third coding region of the gene, which is thought to alter dopamine signalling. A follow up paper (Lakatos et al., 2002) reported that a single nucleotide polymorphism in the promoter region of the same gene (a C>T substitution) amplified the effect of the

DRD4 gene on disorganization. Neither of these genes, nor their interaction, was found to relate to attachment disorganization in two independent samples studied by Bakermans-Kranenburg and van IJzendoorn (Bakermans-Kranenburg & van IJzendoorn, 2004; van IJzendoorn & Bakermans-Kranenburg, 2006). Numerous studies followed also attempting to replicate the association, but none of them did so (see Bakermans-Kranenburg & van IJzendoorn, 2007; Frigerio et al., 2009; Gottfried Spangler, Johann, Ronai, & Zimmermann, 2009; Luijk et al., 2011). A meta-analytic average of available samples in 2007, including the Lakatos sample, was $d = .05$ ($r = .02$), $p = .66$, $N = 542$ (Bakermans-Kranenburg & van IJzendoorn, 2007). In the largest replication study, which combined two relatively large cohort studies from the United States and the Netherlands (Luijk et al., 2011), involving 478 and 543 participants respectively, the associations between the DRD4 7+ VNTR and attachment disorganization were nonsignificant $r = .03$ and $r = .04$. The conclusion is unavoidable that the evidence does not indicate that the DRD4 polymorphism is involved in the development of attachment disorganization. Having said that, as we will elaborate, contemporary genetics is strongly pointing to the fact that main effects of single genes, where they exist, are typically of extremely small magnitude, and hence that past studies have lacked power by several orders of magnitude. It is therefore conceivable that single genes, such as the DRD4, might play a very small role in disorganized attachment but we have so far lacked power to detect them reliably. Nevertheless, the current evidence does not lend support to the DRD4 gene being a more promising gene candidate than polymorphisms in any other region of the genome.

Another line of work that has emerged in this area is the investigation of gene-by-environment interactions. For example, evidence of gene-by-environment interaction was found by Bakermans-Kranenburg and van IJzendoorn (2006) in a sample of 85 mothers who were selected on the basis of having experienced an important loss. These authors found that the DRD4 gene interacted with maternal unresolved loss, as assessed using the Adult Attachment Interview. Infants that had the 7+ allele of the DRD4 gene and whose mothers were unresolved with respect to loss were more likely to be disorganized. Put another way, it appeared that the effect of maternal unresolved loss was stronger for infants with the DRD4 7+ allele. Or, put the other way around, the findings indicated that carriers of the 7+ repeat were the least disorganized when their mothers were not unresolved, and in that sense this study was an early example of evidence indicating genetic "differential susceptibility" (Bakermans-Kranenburg & van IJzendoorn, 2007). Spangler and colleagues (2009) found no DRD4-by-parental care interactions in relation to attachment disorganization in a sample of 106 infants from the Regensburg longitudinal study. They did however find a GxE effect of parental care on disorganization in relation to a different gene—the 5HTTLPR polymorphism of the serotonin transporter gene (with the short form being associated with disorganized attachment, but only when parental responsiveness was low). Notably, Barry, Kochanska and Philibert (N = 88, Barry, Kochanska, & Philibert, 2008) found the short form of the 5HTTLPR gene to be associated with attachment *insecurity* overall (they did not report effects specifically on disorganization), and that the short-form predicted insecurity more strongly (indeed, only) when maternal responsiveness was low. The form of this effect, though not the precise nature of the outcome (insecurity, rather than disorganization) mirrors that found by Spangler and colleagues quite closely. However, a recent report by Raby and colleagues (Raby et al., 2012) failed to replicate this 5HTTLPR × parental responsiveness interaction.

The picture concerning gene environment interactions in the development of disorganized (or indeed insecure) attachment is thus quite mixed, and currently it is unclear whether replicable associations exist. The recent, larger scale investigation by Luijk and colleagues (Luijk et al., 2011) favors the view that gene–environment interactions, in addition to the genetic main effects referred to earlier, may not be reliable. In both US and Dutch samples, data were available on several other genes in addition to DRD4 (5HTTLPR, DRD2, COMT, OXTR) and on observed maternal sensitivity. After taking account of multiple hypothesis testing, no reliable genetic main effects, or gene x sensitivity interactions were apparent in relation to security or disorganization. However, although this is the most statistically powerful test of molecular genetic main effects and interactions conducted thus far, it did not involve measures of the most promising candidate environmental factor with respect to disorganization: parental anomalous, or frightening/frightened parenting. We await large-scale tests of such GxE predictions.

Broadly speaking then, the extant work on the molecular genetics of attachment in infancy, in contrast to the behavior genetic studies, is marked by inconsistency and failures to replicate. Overall, the net implication of both the twin and candidate gene studies is that, in keeping with the predictions of attachment theory, attachment in early life is strongly influenced by the environment and shows little evidence of being determined by genes.

Should we therefore conclude that further genetic research is not warranted? In our view, this would be a premature conclusion to draw. Some useful lessons can be drawn from the larger field of psychiatric genetics. As Kendler (2013) notes, quantitative behavioral genetics convincingly demonstrated that all major forms of psychiatric disorder show marked genetic influence, but early studies selecting candidate genes involved in neurotransmitter function (the so-called usual suspects in candidate gene studies, see Ebstein, Israel, Chew, Zhong, & Knafo, 2010) largely failed to find replicable associations. As larger-scale studies emerged, and researchers increasingly recognized the importance of several methodological issues (particularly the need for strict controls on type I error), initial findings tended not to be replicated. Genome-wide association studies (GWAS), which genotype very large numbers of polymorphisms (often > 500,000 single nucleotide polymorphisms [SNPs]), came to subsequently dominate the field, with an emphasis on blind identification of genes without any prior selection based on biological plausibility. Initially, these studies, which required very large samples, also failed to find replicable effects. However, with even larger samples (often *N*s greater than 20,000) what has emerged is quite clear evidence of replicable gene associations for several major conditions (e.g., schizophrenia, bipolar disorder, see Kendler, 2013). Thus, GWAS appear to indicate quite convincingly that the majority of genes associated with liability to psychiatric disorder have reliable but extremely small main effects, and require very large samples to detect. Second, GWAS studies tend to suggest that the replicable genetic associations are not those that would have been chosen on the basis of, in hindsight, a perhaps simple-minded model of psychiatric disorder. Instead, replicable associations for several psychiatric disorders have involved genes with diverse and largely unanticipated functions, such as inflammatory processes, calcium channel signaling, cell adhesion, and myelination. The meaningfulness of these findings is supported not only by the statistical data, but also by the fact that the identified genes are consistently found to be expressed in the brain and that a substantial proportion of the heritability of psychiatric conditions can be "recovered" from an analysis of variability in common SNPs across the genome (Kendler, 2013).

In light of the fact that, unlike most psychiatric disorders, the overall heritability of attachment security appears low (at least in infancy/toddlerhood), it seems fair to say that the search for genetic main effects on attachment faces considerable hurdles. However, GWAS studies, for the most part, focus on genetic main effects. Somewhat stronger effects have been found for studies of candidate gene × environment interactions, although even here the picture is complex. Arguably the most promising gene–environment interaction—the interaction between the short form of the 5HTTLPR gene and life stress in determining depression (Caspi et al., 2003)—has been fraught with nonreplications and remains controversial to this day. For example, meta-analytic studies have drawn widely divergent conclusions regarding the reliability of the effect, in part as a result of their differing inclusion criteria (Uher & McGuffin, 2010). The most recent meta-analysis by Karg et al. (Karg, Burmeister, Shedden, & Sen, 2011) did, however, find strong overall evidence of G×E interaction in depression, based on over 40,000 individuals, but the results were notably more robust for childhood maltreatment than for life stress/life events. A review by Uher and McGuffin (2010) concluded that the evidence in favor of G×E in depression is quite strong, and that the quality of environmental measures is a key determining factor in the strength and reliability of findings. In particular, objective measures of the environment produced more clear-cut evidence of interaction than subjective self-reports. Thus, we may conclude that gene–environment interaction studies hold more promise for understanding the development of attachment than studies of genetic main effects, but that high-quality measures of the environment, and of the phenotype, and large-scale epidemiologically rigorous samples will likely be necessary to detect them. More, and larger scale, quantitative genetic studies (e.g., twin and adoption studies) are also needed to guide molecular genetic studies to the most promising aspects of attachment (i.e., those with the highest heritability) and to the right environmental moderators.

Thus far, we have only addressed the genetic and environmental determinants of attachment in the first three years of life, but of course attachment is considered an important aspect of human functioning across the life span. Given the modest stability in measures of attachment over time, it is not appropriate to assume that the genetic and environmental determinants of childhood, adolescent or adult attachment are the same as those that obtain in infancy or toddlerhood. Furthermore, it is important to note that to the extent that behavior genetic studies have found evidence of shared environmental effects on other aspects of development, this has tended to be early in the life span (infants and preschoolers). The shared environment may thus represent a set of causal influences that are more dominant in early life and that may diminish with time (Plomin et al., 2013). Another important

consideration is that attachment status in childhood, adolescence, and adulthood is measured and conceptualized very differently to the separation-reunion or secure base tools used in the early years, a difference that may alter the balance of genetic and environmental determinants one observes. Indeed, Main (1996) argued that the ability to reflect on and integrate what might be difficult early experiences, which is central to adult/adolescence measures of attachment, may place greater weight on individual characteristics that are partly heritable (Main, 1996).

Several studies have examined the role of genes and environments in attachment styles (as indicated by self-reports, such as the Experiences in Close Relationships Scale; Fraley, Waller, & Brennan, 2000) in adults. In a sample of 239 adult twin pairs, Brussoni and colleagues (Brussoni, Jang, Livesley, & MacBeth, 2000) estimated that 37% of the variance in attachment anxiety was due to genetic differences and 60% to nonshared environment. Attachment avoidance on the other hand was not heritable, and 29% of the variance was attributable to shared environment. These results were replicated by Crawford and colleagues (Crawford et al., 2007), finding 40% heritability for attachment anxiety and no genetic influence on avoidance. However, Picardi and colleagues (Picardi, Fagnani, Nistico, & Stazi, 2011), using a relatively large sample of young adult twins ($n = 677$ twin pairs), replicated the findings for attachment anxiety (45% heritability; 55% nonshared environment), but not those for avoidance (finding 36% heritability, and 64% nonshared environment). In a recent large-scale study of middle age men, Franz et al. (2011) found that genetic differences accounted for 38% of the variance in attachment anxiety and 27% of the variance in attachment avoidance. Notably, they found that the genes involved in attachment anxiety and avoidance only partly overlapped and that attachment avoidance in particular showed distinct genetic influences that were independent of measures of depression and personality (see also Donnellan, Burt, Levendosky, & Klump, 2008). There is thus quite consistent evidence that self-reported attachment anxiety is influenced by genetic factors, and larger twin studies also tend to indicate genetic influence on attachment avoidance. Inconsistencies between studies may also reflect the different measures used, as the two studies that found shared environmental effects on avoidance used the Relationship Scales Questionnaire (Griffin & Bartholomew, 1994), while those that did not used the ECR or the Adult Attachment Scale (Collins & Read, 1990).

These findings provide a partial picture of the genetic and environmental determinants of attachment in adulthood, as measured using self-reports. However, one cannot infer from this the pattern of heritability of attachment as measured using representational/interview measures, because numerous studies show these two measurement approaches to be essentially orthogonal (Roisman et al., 2007). Recent studies with adult or adolescent siblings using the AAI provide some useful data in that regard. Two independent studies have found effectively no correspondence between siblings in their overall security of attachment as assessed in the AAI (Fortuna, Roisman, Haydon, Groh, & Holland, 2011; Kiang & Furman, 2007). Although these data do not bear directly on the role of genes, they do suggest that the shared environment may not play as strong a role in adult attachment as that observed in infants (cf. van IJzendoorn et al., 2000). Currently, the strongest available direct evidence regarding the role of genes and environments in representational measures of attachment comes from a recent relatively large-scale twin study by Fearon and colleagues (Fearon, Shmueli-Goetz, Viding, Fonagy, & Plomin, 2014). Fearon et al. used the Child Attachment Interview (Shmueli-Goetz, Target, Fonagy, & Datta, 2008) to study the attachment representations of 551 adolescent twin pairs (aged 15 years), drawn from the Twins Early Development birth cohort study (TEDS, see Trouton, Spinath, & Plomin, 2002). The results differed quite dramatically from the findings of the earlier twin studies of infant/toddler attachment. First, no evidence of shared environmental influence was found; DZ twins, much like the previously described siblings, showed no significant correspondence in their attachment classifications, and only weak correlations for the continuous security scale ($r = .20$, $N = 261$ twin pairs). Second, strong evidence of genetic effects emerged. In contrast to the DZ twins, MZ twins showed marked resemblance for security and coherence, with the correlation between twins for coherence being double that observed for DZ twins ($r = .40$). Standard genetic analysis showed that after controlling for gender and age, 38% of the variance in coherence, and 35% of the variance in binary security was attributable to genetic factors. The remaining variance was attributable to the nonshared environment and measurement error. When one considers that measurement error attenuates the genetic effect, these heritability figures are likely to be underestimates. The same considerations of course would caution against ruling out a modest shared environmental effect, as attenuation could have occurred to such a degree that it could not be reliably detected in this sample.

These findings are striking in their marked contrast to those identified in very young children. We cannot at this stage ascertain whether they reflect a distinct

feature of the adolescent period (where we know normative increases in dismissing speech can be observed, see Bakermans-Kranenburg & van IJzendoorn, 2009; Waters, Weinfield, & Hamilton, 2000; Weinfield, Sroufe, & Egeland, 2000), or are reflective of a broader unfolding process of developmental change taking place between early childhood and adolescence. They certainly draw attention to the possibility that the genetic and environmental determinants of attachment may not be constant across development, and raise the possibility that genes may begin to shape the organization of attachment, at least at the representational level, in adolescence, and possibly beyond. If these findings prove to be correct, a key question to be addressed by future research is how such changes in the genetic profile of attachment between infancy and adolescence come about. We can imagine mechanisms operating at two distinct levels: it may be that these genetic effects occur via gene–environment interplay, such that heritable traits in the child begin to affect parent–child interactions (e.g., evoking hostility/negativity, see Avinun & Knafo, 2013; Narusyte et al., 2008), which in turn impact on the adolescent's attachment organization. Such a mechanism is an example of *gene–environment correlation*. Another possibility is that these effects arise at the level of the individual's response to attachment-related experiences. For example, genetically influenced traits may affect adolescents' emotion-regulation skills, bias their interpretations of attachment experiences, or influence their ability to integrate them coherently within an internal working model. All of these processes might feed into adolescents' states of mind with respect to attachment as they shift with time developmentally. It would be wrong of course to emphasize only the genetic findings regarding adolescent attachment. In keeping with studies of infants and toddlers, attachment in adolescence clearly demonstrates marked nonshared environmental influence and these findings compel the field to investigate the environmental mechanisms leading to differences in security between children raised in the same family. Finally, we note that an urgent research question is whether genetic influences also characterize attachment into adulthood as assessed in the Adult Attachment Interview. In that context, there is of course already existing evidence that representational assessments of adult attachment are associated with earlier caregiving experiences (e.g., Beijersbergen, Juffer, Bakermans-Kranenburg, & van IJzendoorn, 2012). Furthermore, even were one to find genetic influences on adult attachment, this would not necessarily imply that the intergenerational transmission of attachment from parent to child is mediated by genes (D'Onofrio et al., 2007). In that regard, evidence of intergenerational continuity from parent to child among children in foster care (Dozier, Stovall, Albus, & Bates, 2001), where caregivers and their children are not biologically related, would seem to indicate environmental transmission.

CONTINUITY AND CHANGE IN ATTACHMENT SECURITY ACROSS THE LIFE COURSE

The nature of internal working models contributes to an implicit assumption in attachment theory that individual differences in attachment security are relatively stable over time. To be sure, according to attachment theory (Bowlby, 1969), relative stability in internal working models is thought to be ensured through the increasing automatization of parent–child interactions, ultimately decreasing attentional demands and conscious revision of working models. Such habitual interaction patterns also lead to negative or positive perceptual biases that color expectations and interpretations of subsequent experiences. Moreover, because both the caregiver and child hold working models of the relationship, if one partner attempts to change routinized relationship patterns, the other is likely to resist and attempt to maintain the established pattern. Bowlby also acknowledged, however, that updates to internal working models are expected under certain environmental conditions, including changes in parent–child relationship quality or changes in life circumstances that improve or undermine the caregiver's perceived or actual ability to sensitively respond to the child's attachment needs. Thus, although attachment security is expected to remain relatively stable across the life course, it is also recognized that lawful change in security is possible given attachment-relevant changes in life circumstances (see Bretherton & Munholland, 2008).

Questions concerning stability and lawful change in attachment security have garnered a great deal of interest and have been the focus of much empirical inquiry. Initial investigations in this area focused on short-term stability in the organization of attachment behavior during childhood. In the first investigation of this kind, Waters (1978) provided evidence for remarkably strong stability in infants' organized attachment patterns in relation to their mother as measured by the Strange Situation from 12 to 18 months (see also Goossens, van IJzendoorn, Tavecchio, & Kroonenberg, 1986). However, subsequent investigations of the stability of mother–child attachment security from the Strange Situation to another standardized observational assessment of attachment during early childhood (e.g., Strange Situation, AQS, modified Strange Situation)

provided mixed evidence. Although some studies found significant stability (Howes & Hamilton, 1992; Main et al., 1985; Main & Weston, 1981; Owen, Easterbrooks, Chase-Lansdale, & Goldberg, 1984; Vaughn, Egeland, Sroufe, & Waters, 1979; Wartner et al., 1994), others did not (Belsky, Campbell, Cohn, & Moore, 1996; Egeland & Farber, 1984; Thompson, Lamb, & Estes, 1982).

An understandably more challenging question to address concerns the stability of attachment security from infancy to adulthood, as empirically addressing such questions necessarily requires prospective, longitudinal data (for an overview of such studies, see Grossmann, Grossmann, & Waters, 2005; Roisman & Haydon, 2011). As such, evidence regarding stability and change in attachment security from infancy into the years of maturity emerged only relatively recently, dating to 2000 when a handful of studies were published simultaneously in the journal of *Child Development* (Hamilton, 2000; Lewis, Feiring, & Rosenthal, 2000; Weinfield et al., 2000). As with the literature on short-term stability in attachment security, the extant longitudinal studies have provided mixed evidence, with some studies finding stability in attachment security from infancy, as assessed with the mother in the Strange Situation, to adulthood, as assessed with the AAI (e.g., Hamilton, 2000; Main, 2000, 2001; Main, Hesse, & Kaplan, 2005; Waters, Merrick, Treboux, Crowell, & Albersheim, 2000), and other not (Beijersbergen et al., 2012; Lewis et al., 2000; Weinfield et al., 2000; Zimmermann, Fremmer-Bombik, Spangler, & Grossmann, 1997).

Although the apparent discrepancy among these findings regarding attachment stability across the life course can be explained in a number of ways, one interpretation that has emerged in the literature in light of evidence that studies focused on middle-class, relatively low-risk families have generally provided evidence for stability in attachment security (e.g., Main et al., 2005; E. Waters, 1978) and studies focused on at-risk samples have not (e.g., Egeland & Farber, 1984; Weinfield et al., 2000) is that attachment security is generally stable, except in populations that are at higher risk for experiencing life stress and, thus, more profound changes in the caregiving environment (e.g., Waters, Weinfield et al., 2000). To test such claims and provide a more accurate estimate of the stability of attachment security across the early life course, Fraley (2002) conducted a meta-analysis of this literature, specifically including studies that reported on the association between attachment security as assessed by the Strange Situation and another behavioral measure of attachment (Strange Situation, AQS, modified Strange Situation) or the AAI. Findings from this meta-analysis provided evidence that attachment security is moderately stable from infancy to adulthood ($r[\Phi] = .39$), and consistent with prior conclusions, attachment security was found to be less stable in samples considered to be at higher risk ($r = .27$) than those at lower risk ($r = .48$).

Pinquart, Feusner, and Ahnert (2013) provided an update of this effort in light of more recently published work in their meta-analysis on the stability of attachment security. Similar to Fraley's (2002) findings, Pinquart and his colleagues (2013) provided evidence for moderate stability in attachment security across early childhood (r's range from .24 to .46 depending on the time interval between assessments) and that the effect was significantly weaker in at-risk samples ($r = .21$) than in not at-risk samples ($r = .36$). In contrast to findings from Fraley (2002), however, Pinquart and colleagues (2013) found that attachment security was not significantly stable from infancy to adulthood ($r = .14$). Unfortunately, because Pinquart and colleagues (2013) included a range of adult attachment assessments, whereas Fraley (2002) focused on studies employing the AAI as the assessment of adult attachment, it is difficult to determine whether this discrepancy is due to the emergence of new evidence of the relative instability in attachment security published in the interim between Fraley's (2002) and Pinquart et al.'s (2013) meta-analyses or due to differences in the scope of the two meta-analyses.

It is even more important to note that, due to the small sample size of many of the investigations of attachment stability across the early life course, the total number of individuals studied from infancy to young adulthood in the extant literature is, by most standards, quite modest, resulting in a relatively limited corpus on which to estimate the stability of attachment security. In fact, only about 785 participants have been studied across all investigations to date (Roisman & Haydon, 2011). In the most recent wave of the NICHD Study of Early Child Care and Youth Development (SECCYD)—a longitudinal study following over 1,000 low-risk participants from infancy to adulthood and comprising several early observational assessments of mother–child attachment security (Strange Situation administered at 15 mo., AQS administered at 24 mo., modified Strange Situation administered at 36 mo.)—857 participants were administered the AAI at age 18, making it the largest, most comprehensive single investigation of the stability of attachment security to date. Helping to reconcile prior mixed evidence in the literature, findings from this study indicate that although attachment security is significantly stable over the early life course, the magnitude

of such stability is quite weak by conventional standards (r = .12; Groh et al., 2014).

As with prior investigations of attachment stability, caregiving and contextual sources of variation that might contribute to stability and lawful change in attachment security were also examined in the SECCYD (Booth-LaForce et al., 2014). Prior investigations have identified several theoretically relevant aspects of the caregiving environment that might contribute to stability and change in attachment security (e.g., Beijersbergen et al., 2012; Egeland & Farber, 1984; Frodi, Grolnick, & Bridges, 1985; Hamilton, 2000; Lewis et al., 2000; Main et al., 2005; NICHD Early Child Care Research Network, 2001, 2006; van Ryzin, Carlson, & Sroufe, 2011; Waters, Weinfield, et al., 2000; Weinfield et al., 2000; Weinfield, Whaley, & Egeland, 2004). However, studies have varied in terms of the contextual factors investigated and findings across studies have not always converged, making it difficult to draw firm conclusions regarding whether instability in attachment security is lawful and which environmental factors contribute to positive and negative deflections from infancy to adulthood.

The large longitudinal SECCYD sample includes assessments over time of important potential sources of change and continuity in attachment security; thus, it is well positioned to address questions concerning environmental factors associated with stability and change in attachment. Consistent with expectations that internal working models are reinforced by stability in parent–child relationship quality over time, evidence from this study indicated that stability in attachment security (vs. insecurity) is associated with higher levels of maternal sensitivity, higher levels paternal physical presence in the home, and lower levels of paternal psychopathology over the course of development from infancy to adulthood. Overall, findings from this investigation were also consistent with claims that instability in attachment-relevant variation across the early life course is lawful, in that change in attachment security can be explained by comparable changes in attachment-relevant life circumstances. Specifically, individuals who experienced high quality relationships in infancy but came to develop insecure attachment representations in adulthood experienced higher levels of paternal physical absence from the home, larger increases in negative life events, and larger declines in the quality of maternal sensitivity over the course of development. In contrast, individuals who overcame early insecurity and developed secure attachment representations in adulthood were found to experience higher levels of maternal sensitivity over the early life course (Booth-LaForce et al., 2014).

Taken together, findings from across the literature on continuity and change in attachment security provide evidence that attachment security is not especially stable across the life course, but that when discontinuity exists, it can be explained by attachment-relevant changes in the caregiving environment. That said, several limitations of this literature should be noted and addressed in future research. The literature on attachment stability is largely a literature of *mother–child* attachment. Pinquart and colleagues (2013) have provided evidence in their meta-analysis that the stability of father–child attachment security (r = .27) is comparable to that of mother–child attachment security. However, such evidence is based on a relatively limited corpus of data. Indeed, only two studies based on relatively small samples have investigated the concordance between father–child attachment security in infancy and the security of attachment representations in adulthood (Steele & Steele, 2005; Zimmermann et al., 1997). As it may be the case that attachment security with both the mother and father, rather than security with the mother alone, is more predictive of states of mind with respect to attachment in adulthood, there is a clear need for more comprehensive investigations of the stability of attachment security that include assessments of attachment with both mothers and fathers.

In addition, two diverging perspectives of attachment stability have emerged in the literature. According to what has been referred to as the *revisionist* perspective, working models are revised and updated in light of ongoing experiences, and thus, it is not necessarily expected that working models in adulthood will correspond with those developed in infancy. In contrast, according to the *prototype* perspective, while updating of working models in light of new experiences is acknowledged, it is also argued that models developed in infancy remain unchanged and continue to anchor interpersonal experiences throughout the life course (see Fraley, 2002). Because evaluating such claims requires attachment security to be assessed repeatedly over developmental periods, longitudinal investigations of attachment stability that only include two assessments of attachment security, which is typical in the extant literature, are unable to distinguish between these two perspectives. Fraley (2002) meta-analytically evaluated the empirical support for such claims in light of available evidence at the time, providing evidence consistent with the prototype perspective. However, in light of evidence from Pinquart and colleagues' (2013) meta-analysis and findings from the SECCYD (Groh et al., 2014) of relatively weak stability in attachment security from infancy to adulthood, such

claims might have to be reevaluated in light of this more recent evidence.

Inherent in the study of attachment stability is the assumption that stability and lawful change in attachment security is environmentally mediated. However, nearly all studies on the stability of attachment security have relied on samples in which parents and children are genetically related (but see Beijersbergen et al., 2012), leaving open the possibility that stability and lawful change in attachment security might be attributed to the genetic relatedness between the parent and child. Initial evidence from a study of adopted children and their mothers indicates that greater levels of maternal sensitivity in infancy and adolescence are associated with stability in attachment security and that positive changes in maternal sensitivity from infancy to adolescence are associated with positive deflections in attachment security (Beijersbergen et al., 2012), supporting an environmentally mediated pathway (although such findings could also be explained by gene–environment correlation). Given the centrality of this issue to attachment theory, future work should further employ genetically informed research designs (including studies of monozygotic and dizygotic twins) to thoroughly address issues concerning environmental or genetic mediation of stability and lawful change.

Finally, the literature on the stability of attachment security is predicated on the assumption that the key test of stability in security should involve the prediction from infant assessments of attachment security (e.g., Strange Situation) to adult assessments (e.g., AAI). Such a focus might be an error of emphasis for at least two reasons. First, given that Bowlby (1973) theorized that internal working models are only tolerably accurate representations of prior experiences, such a focus may be an overly conservative test of attachment stability. For example, when examining the contribution of attachment-relevant experiences with caregivers over the course of childhood to adults' states of mind with respect to attachment as measured at age 18 with the AAI in the context of the SECCYD, 20% of the variance in dismissing states of mind and 11% of the variance in preoccupied states of mind were accounted for by prior experiences with mothers and fathers (Haydon, Roisman, Owen, Booth-LaForce, & Cox, 2014). Second, there is a very clear need for studies that examine stability and change in security *across adolescence and into adulthood* (see Allen, McElhaney, Kuperminc, & Jodl, 2004; Crowell, Treboux, & Waters, 2002), not incidentally a period of development when it is possible to use a common assessment of security over assessments.

CHILDREN'S ATTACHMENT SECURITY AND PSYCHOPATHOLOGY

Bowlby was particularly interested in the developmental origins of children's emotional disturbance, and believed that early relational experiences were of great importance in the ontogeny of mental health and illness. Yet, the majority of initial work on the developmental significance of individual differences in early attachment security primarily focused on children's normative development, and in particular on the contribution of early attachment relationships to subsequent interpersonal competence (Berlin, Cassidy, & Appleyard, 2008; Groh et al., 2014; Schneider, Atkinson, & Tardif, 2001). However, as the field of developmental psychopathology took shape (see Cicchetti, 1984) and early attachment insecurity was further conceptualized as a risk factor for the development of behavioral problems (Sroufe, 1988), there was renewed interest in the significance of early parent–child attachment relationships for the development of psychopathology (see DeKlyen & Greenberg, 2008). In the past few decades, a sizable literature on the links between early attachment and psychopathology has emerged, establishing attachment theory as a dominant theoretical framework guiding research on the role of early experiences in the development of psychopathology. In this section, we provide an overview of this now vast literature and offer new insights in light of recent meta-analytic evidence (Fearon, Bakermans-Kranenburg, van IJzendoorn, Lapsley, & Roisman, 2010; Groh, Roisman, van IJzendoorn, Bakermans-Kranenburg, & Fearon, 2012; Madigan, Atkinson, Laurin, & Benoit, 2013). Following the focus of research in this area, we primarily discuss investigations of the links between early mother–child attachment security and externalizing (e.g., aggression) and internalizing (e.g., anxiety, depression) symptomatology.

A number of (nonmutually exclusive) mechanisms have been proposed to explain why early attachment experiences might be associated with psychopathology. The dominant explanation concerns the internal working models (IWM) construct (Bowlby, 1973, 1980; Bretherton & Munholland, 2008). As described previously in this chapter, based on caregivers responsiveness to attachment signals, infants develop cognitive-affective representations, or IWMs, that encompass views of the caregiver as (un)responsive, and in turn, views of the self as (un)worthy of love and care that over time become generalized into views of the self, others, and the nature of relationships. As a result of the care they receive, insecure infants are believed to develop IWMs characterized by anger, mistrust, anxiety, or fear,

coloring their subsequent social experiences and ultimately heightening their risk for developing psychopathology, whereas secure children are believed to develop IWMs characterized by inner confidence, efficacy, and self-worth, helping to buffer them against developing mental illness.

Because both theory and research have established the importance of emotion regulation in the etiology and maintenance of internalizing and externalizing problems (Chaplin & Cole, 2005; Izard, Youngstrom, Fine, Mostow, & Trentacosta, 2006), another powerful explanatory framework concerns the contribution of early attachment relationships to children's developing capacity to tolerate and manage affect. More specifically, it has been argued that within the parent–child attachment relationship, children develop either adaptive (in the case of secure children) or maladaptive (in the case of insecure children) emotion regulation strategies (Cassidy, 1994; Isabella, 1993; Sroufe, 1979, 1996) that serve, respectively, as either protective or risk factors for later psychopathology (e.g., Carlson, 1998; Guttmann-Steinmetz & Crowell, 2006).

Another potential avenue through which early attachment experiences might contribute to the development of psychopathology occurs at the level of behavior. Through the history of care they receive, children develop specific behavioral strategies for interacting with their caregivers that might be considered adaptive organizations of behavior given the constraints of the quality of caregiving the children have experienced. For example, on the assumption that resistant infants experience a history of inconsistently responsive care, it would be considered adaptive (at least in the narrow, proximal sense) to exaggerate attachment behavior to increase the likelihood that the caregiver will respond to the infant's needs (Simpson & Belsky, 2008). However, such behavioral strategies might be considered maladaptive *outside the context of the parent–child relationship*, placing insecure children at heightened risk for social exclusion and developing psychopathology (Greenberg, Speltz, & DeKlyen, 1993; Weinfield, Sroufe, Egeland, & Carlson, 2008). Other potential mechanisms that have been proposed to buffer secure children from the development of internalizing and externalizing symptoms include (1) developing a sense of self-confidence stemming from experiences of support and comfort and through effective exploration of the environment (Goldberg, 1997), (2) generalized positive social expectations (e.g., Dodge & Coie, 1987), (3) socialization of moral emotions and values (Kochanska, 1997; van IJzendoorn, 1997), (4) continuity in ongoing supportive caregiving (Lamb, Thompson, Gardner, Charnov, & Estes, 1984), and (5) social regulation of biological systems mediating effective stress and arousal regulation (e.g., Suomi, 2003; Weaver et al., 2004).

Although Bowlby's ideas concerning the significance of children's early experiences with primary caregivers for subsequent developmental (mal)adaptation were highly influential, the development of the Strange Situation procedure greatly facilitated the empirical investigation of the correlates and consequences of the quality of early parent–child attachment relationships. One of the earliest and most influential longitudinal studies of the developmental sequelae of early attachment variation was launched in Minnesota by Byron Egeland and Alan Sroufe (see Sroufe, Egeland, Carlson, & Collins, 2005a). It was in the context of this landmark longitudinal study that the conceptual links between attachment and psychopathology were elaborated on, providing the field with more specific hypotheses regarding the legacy of early attachment experiences for the development of psychopathology.

Specifically, this relatively large, high-risk cohort has been followed for over 35 years beginning at birth, and results from extensive assessment of adaptive and maladaptive behavior have provided consistent evidence that individuals who developed an insecure attachment relationship with their mother in infancy are at heightened risk for developing psychopathology across the life course (Sroufe et al., 2005a). Results have also revealed that specific types of attachment insecurity serve as distinct diatheses for the development of externalizing versus internalizing symptomatology, with insecure-avoidant infants found to be at heightened risk for developing aggression (believed to result from feelings of anger derived from early rejection by caregivers) and resistant infants found to be at heightened risk for developing anxiety (believed to result from feelings of worry derived from early inconsistent or unresponsive care; Sroufe, 2003). Such evidence has contributed to the hypothesis that early avoidance increases risk for developing externalizing symptoms, whereas early resistance increases risk for developing internalizing symptoms. In addition, with the discovery of attachment disorganization, it was further hypothesized that because disorganized infants show contradictory and fragmentary behaviors—indicative of a breakdown of an organized emotion regulation strategy in the face of stress—they might be at heightened risk for developing both internalizing and externalizing symptoms (Carlson, 1998).

A second hypothesis to emerge from the work at Minnesota is that early attachment experiences play an enduring, if probabilistic, role in shaping developmental (mal)adaptation. Consistent with Bowlby's (1973)

view that current adaptation is a function of both prior and current circumstances, Sroufe and his colleagues (Sroufe, Egeland, & Kreutzer, 1990) claimed that current experiences have the power to transform adaptation without erasing the history of prior adaptation. Thus, early attachment-relevant experiences are thought to contribute in enduring ways to adaptation across the life course.

Although consistent evidence from the Minnesota study has been presented in support of these hypotheses (Sroufe et al., 2005a; Sroufe, Egeland, Carlson, & Collins, 2005b), other investigations have not always found a significant link between early attachment insecurity and disorganization and symptoms of psychopathology. Indeed, in the same monograph in which the first evidence was presented from the Minnesota study of a significant link between attachment insecurity and behavior problems (e.g., hostility, withdrawal; Erickson, Sroufe, & Egeland, 1985), evidence from another longitudinal study of 120 middle-class families was presented in which no association between individual differences in mother–child attachment security and behavioral problems was found (Bates, Maslin, & Frankel, 1985). This lack of consistent evidence across studies might be explained in a number of ways, but because investigations of middle-class, relatively low-risk families have tended to not find a main effect of attachment insecurity on psychopathology (e.g., Bates et al., 1985; Fagot & Kavanagh, 1990; Lewis, Feiring, McGuffog, & Jaskir, 1984), whereas studies focused on more at-risk samples have (e.g., Easterbrooks, Davidson, & Chazan, 1993; Goldberg, Gotowiec, & Simmons, 1995; Lyons-Ruth, Easterbrooks, & Cibelli, 1997; Shaw, Keenan, Vondra, Delliquadri, & Giovannelli, 1997; Shaw & Vondra, 1995), one explanation that has emerged in this literature to explain such divergent findings is that attachment insecurity and disorganization should be thought of as interactive risk factors that are more predictive when considered with other potential risk factors (e.g., socioeconomic disadvantage, parent psychopathology; Belsky & Fearon, 2002; DeKlyen & Greenberg, 2008; Fearon & Belsky, 2011; Kobak, Cassidy, Lyons-Ruth, & Zir, 2005). That said, it is important to note that some studies have found risk status not to heighten insecure children's risk for developing symptoms of psychopathology (e.g., Cicchetti, Rogosch, & Toth, 1998), and main effects of attachment insecurity on symptoms of psychopathology have emerged in low-risk samples (e.g., Bohlin, Hagekull, & Rydell, 2000).

Given the sheer volume and diversity of studies that have examined the contribution of early attachment in security to the development of psychopathology and the complex pattern of findings that have emerged from such investigations, it has become virtually impossible to provide a clear narrative review of this literature. Accordingly, we recently conducted a series of meta-analyses to quantitatively evaluate the extant empirical evidence in light of key hypotheses regarding the significance of early attachment insecurity and disorganization (using observational assessments of mother–child attachment administered between 1 and 6 years) for the development of externalizing (Fearon et al., 2010) and internalizing (Groh et al., 2012) symptomatology. The need for such quantitative reviews of this literature to draw clearer conclusions regarding links between early attachment variation and psychopathology is evident by the fact that shortly after the publication of our meta-analysis on early attachment and internalizing symptoms, a second meta-analysis on the same topic was published (Madigan et al., 2013). As the results of this meta-analysis largely converged with our own, we focus our discussion on the results from our own work.

Overall, these meta-analyses provide support for the hypothesis that early attachment insecurity heightens children's risk for developing externalizing and internalizing symptoms (Fearon et al., 2010; Groh et al., 2012; Madigan et al., 2013). More importantly, however, as the meta-analysis on early mother–child attachment variation and externalizing symptoms includes nearly 70 independent samples comprising nearly 6,000 children and the meta-analysis on early mother–child attachment variation and internalizing symptoms includes over 40 independent samples comprising over 4,000 children, these meta-analyses provide a more precise estimate of the magnitude of such effects. Specifically, findings from these meta-analyses indicate that the effect of early mother–child insecurity on externalizing symptoms is modest ($d = 0.31$; Fearon et al., 2010) and significantly stronger than the surprisingly weak association between early mother–child insecurity and internalizing symptoms ($d = 0.15$; Groh et al., 2012).

Consistent with perhaps one of the most provocative hypotheses that has emerged in this literature that early attachment-relevant experiences have enduring implications for developmental (mal)adaptation (Sroufe et al., 1990), the magnitude of the effects of early insecurity we documented in our meta-analytic work were not found to vary with the age at which internalizing and externalizing symptoms were assessed (Fearon et al., 2010; Groh et al., 2012), indicating that the association between early mother–child attachment insecurity and symptoms of psychopathology does not wane over the course of development from infancy to early adolescence. In light

of such evidence, there is an urgent need for increased, theory-driven investigation of the mediating processes accounting for such effects. Indeed, questions remain regarding whether long-term continuities in the effects of attachment are due to the ongoing supportive function provided by attachment relationships or the early effects of attachment experiences on the construction of stable psychological structures, such as internal working models. Currently, there is evidence in the literature to support both processes. For example, Belsky and Fearon's (2002) analysis of the NICHD Study of Early Child Care and Youth Development data at 1–3 years suggested that the effect of attachment tended to persist primarily when there was continuity in the quality of maternal care. An analysis of the same data set by Haltigan, Roisman, and Fraley (2013) revealed that early maternal sensitivity, a robust predictor of early security (Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2003; De Wolff & van IJzendoorn, 1997), had an enduring effect on children's psychopathology as rated by teachers through age 15, even after accounting for concurrent effects of maternal sensitivity. Given the importance of understanding how variation in the early mother–child attachment relationship exerts an enduring force on children's mental health, we return to this topic in the next section of this chapter.

Regarding the contribution of specific types of insecurity to the development of specific symptoms of psychopathology, results from these meta-analyses provide scant evidence for claims regarding the differential predictive significance of insecurity sub-types (avoidance, resistance, disorganization) for the development of externalizing versus internalizing symptomatology (Fearon et al., 2010; Groh et al., 2012; Madigan et al., 2013). Specifically, in contrast to claims that early avoidance increases risk for developing externalizing symptoms and early resistance increases risk for developing internalizing symptoms, we found that early avoidance was significantly associated with both internalizing (d = 0.17) and externalizing (d = 0.12) symptoms, and that early resistance was not significantly associated with internalizing (d = 0.03) or externalizing (d = 0.03) symptoms. However, partially supporting claims that early disorganization increases risk for developing both internalizing and externalizing symptoms, early disorganization was found to be significantly associated with externalizing (d = 0.34), but not internalizing (d = 0.08), symptoms (Fearon et al., 2010; Groh et al., 2012). In light of recent factor analytic and taxometric evidence (e.g., Fraley & Roisman, 2014; Fraley & Spieker, 2003; Haltigan, Leerkes, et al., 2014; Haydon et al., 2012; Roisman et al., 2007), a potential reason that such theoretically unanticipated effects emerged in these meta-analyses might be that the traditional categorical coding systems used in developmental assessments of attachment may not completely capture the latent structure and taxonicity of attachment-related variation. In some studies, when empirically derived dimensions of adult dismissing and preoccupied states of mind have been used, distinct and theoretically anticipated correlates of adult attachment variation have been identified (Fraley & Roisman, 2014; Fraley & Spieker, 2003; Haydon, Roisman, Marks, & Fraley, 2011; Whipple, Bernier, & Mageau, 2011). As such, the continuous avoidance and resistance dimensions of early attachment might prove useful in identifying distinctive links between specific patterns of attachment insecurity and the development of internalizing versus externalizing symptomatology beyond what the conventional categories are able to achieve.

Following arguments that the magnitude of the effect of early attachment insecurity might vary according to whether the child experiences other psychosocial stressors (Belsky & Fearon, 2002; DeKlyen & Greenberg, 2008; Kobak et al., 2005), several moderators of the effect of attachment insecurity on the development of psychopathology were also examined in these meta-analyses. Focusing first on contextual risk factors that have been theorized to amplify the effect of attachment insecurity on the development of psychopathology, in contrast to expectations, socioeconomic status was not found to moderate the effect of early insecurity on internalizing or externalizing symptoms (Fearon et al., 2012; Groh et al., 2012; Madigan et al., 2013). Such evidence therefore does not provide support for a diathesis stress model in which the influence of early attachment on maladaptation is theorized to be strongest in economically deprived populations. Regarding psychological risk factors, our meta-analytic work did indicate that insecure children were at especially heightened risk for developing externalizing symptoms (but not internalizing symptoms [Groh et al., 2012; Madigan et al., 2013]) if they were diagnosed with a clinical disorder or had a parent suffering from psychopathology (Fearon et al., 2010). Taken together, findings from these meta-analyses provide limited support for claims that the effects of early insecurity on psychopathology are magnified when children experience other risk factors.

However, it is important to note that relatively few studies have systematically examined the effect of multiple risk factors in combination (e.g., low SES, parental psychopathology, and clinical status). For example, evidence from the NICHD SECCYD indicates that the association

between attachment and psychopathology is amplified under conditions of multiple contextual risk factors (Belsky & Fearon, 2002). Furthermore, in a recent paper, also using data from the SECCYD, Fearon and Belsky (2011) found statistically rather strong evidence that disorganized attachment, in particular, foreshadowed marked increases in teacher-reported externalizing behavior problems across the primary school years for boys reared in low-SES circumstances. Thus, these findings suggest that both gender and low-SES status might be relevant when considering attachment and the development of externalizing problems in childhood. In light of such evidence and theoretical arguments that the experience of multiple risk factors might be especially important for heightening children's risk for psychopathology (Rutter, 1979), further research in this area focused on children experiencing multiple forms of psychosocial risk is needed.

Partially supporting claims that insecurity might heighten the risk for developing externalizing symptoms in boys and internalizing symptoms in girls (DeKlyen & Greenberg, 2008), the association between early mother–child insecurity and externalizing symptoms was found to be stronger for boys than girls (Fearon et al., 2010). In contrast, early mother–child insecurity was not more strongly associated with internalizing symptoms for girls than boys (Groh et al., 2012; Madigan et al., 2013). Instead, Madigan and colleagues (2013) found that insecure boys were also at significantly heightened risk for developing internalizing symptoms. Although Groh and colleagues (2012) also found that the effect of insecurity on internalizing symptoms was larger for boys than girls, the difference was not quite statistically significant ($p = .06$). This almost certainly nonsignificant difference in findings is attributable to the slightly different selection criteria of the meta-analyses. Importantly, however, findings from both meta-analyses clearly demonstrate that insecure girls are not at higher risk than boys for developing internalizing symptoms.

In addition to providing more precise estimates of the magnitude of the effects of early insecurity on internalizing and externalizing symptoms and identifying moderators of such effects, quantitative reviews of the literature on early attachment and psychopathology have also identified key gaps in this literature. For example, there is a clear paucity of research on the significance of children's attachment relationships with fathers for social and emotional development. Because of the relative neglect of father-child relationships in research on early attachment and internalizing (but see Fagot, 1995; Rothbaum, Rosen, Pott, & Beatty, 1995; Suess, Grossmann, & Sroufe, 1992) and externalizing (but see Aviezer, Sagi, Resnick, & Gini, 2002) symptomatology, firm conclusions regarding the significance of such attachment relationships for the development of psychopathology cannot yet be drawn. There is clearly an urgent need for further research on the developmental significance of early father–child attachment security. Such research, in addition to examining the unique effects of father-child attachment on children's development, should also incorporate a broader family systems approach in which the interactive effect of attachment security with both parents is examined. For example, recent evidence suggests that developing a secure attachment relationship with one parent might offset the negative effects of developing an insecure attachment relationship with the other on children's mental health (Kochanska & Kim, 2013; see also Suess et al., 1992; van IJzendoorn, Sagi & Lambermon, 1992).

In addition, the overwhelming majority of investigations of attachment and psychopathology have used the mother-reported Child Behavior Checklist (Achenbach, Edelbrock, & Howell, 1987) or the Teacher Report Form (Achenbach, 1991) of the Achenbach System of Empirically Based Assessment (ASEBA) as measures of children's externalizing and internalizing symptoms. Because parents and teachers might find it more difficult to report on internalizing symptoms than externalizing symptoms due to the more "public" and observable nature of externalizing versus internalizing symptoms, the heavy reliance on such reports in this literature might have contributed to the surprisingly small effect for early mother–child attachment security on internalizing symptoms. Accordingly, future research in this area should make use of trained observers (e.g., clinicians) of internalizing symptoms to determine whether the effect of early insecurity on internalizing symptoms is stronger when such reports are used.

Taken together, evidence from across the literature on the developmental significance of early attachment-relevant experiences for psychopathology suggests that the quality of the early attachment relationship that children develop with their mother has a modest, yet enduring, influence on the evolution of children's mental health over the early life course. Such evidence clearly demonstrates that avoidant or resistant attachment relationships are not inherently pathological. Instead, early attachment security and insecurity are best considered as protective or risk factors, respectively, that should be considered in a broader developmental model of psychopathology. The status of disorganized attachment is more difficult to outline due to a lack of systematic research efforts in clinical populations using adequate outcome parameters. Its strong association

with child maltreatment (Cyr et al., 2010) and with growing up in institutional settings (Bakermans-Kranenburg et al., 2011) might make disorganized attachment a clearer candidate for a direct causative agent in the emergence of psychopathology, perhaps particularly for disorders with a dissociative component (Carlson, 1998; Sroufe et al., 2005b) or for problems related to aggression (Fearon et al., 2010; Lyons-Ruth et al., 1997).

THE SEARCH FOR MEDIATORS

Although an extensive volume of research has examined the basic connection between attachment and children's functioning, considerably less has been done to track the longitudinal mediating processes, residing in the child, that could help account for attachment's beneficial or detrimental effects. However, quite a number of studies have examined cross sectional and longitudinal associations between attachment and a range of theoretically plausible mediating variables. A diverse set of processes has been explored, from the underlying biology of stress regulation, to cognitive and social-cognitive mechanisms involved in social perception and social action. Understanding these mediating factors is extremely important for a full account of the role of attachment in the development of psychopathology and, in principle, in the use of attachment concepts in intervention.

Physiological Mediators

Attachment behavior is assumed to serve as the infant's primary means of regulating stress, and hence a logical mechanism mediating long-term outcomes associated with attachment security and insecurity is via alterations in the functioning of the stress system. Extensive work in humans and animals testifies to the deleterious effects of chronic stress exposure, and of the role of sympathetic, parasympathetic, endocrine, and immune systems in these effects on both emotional and physical health (Juster, McEwen, & Lupien, 2010). Furthermore, recent animal studies suggest that variations in parental care during an early critical period induce a set of more or less permanent epigenetic changes to the stress system (Weaver et al., 2004). While these fascinating findings are difficult to investigate directly in humans, they nevertheless underline the close connections between early care and the neurobiology of stress. Seminal early work by Sroufe and Waters (Sroufe & Waters, 1977) and Spangler and Grossman (G. Spangler & Grossmann, 1993) first studied the relationship between attachment patterns and heart rate during the Strange Situation Procedure. Sroufe and Waters's original paper, based on detailed case studies, appeared to indicate that the heart rates of secure infants, relative to both resistant and avoidant infants, decelerated most quickly upon reunion, and also that avoidant infants, despite their outward demeanor, showed similar elevations in heart rate to the other attachment categories during separation. These initial findings thus provided support for two key predictions of attachment theory: (1) that avoidance is a secondary behavioral strategy for coping with attachment-related stress, and not an indication of a lack of distress; and (2) that secure attachment behavior affords the infant a more efficient means of regulating physiological arousal. It is notable, however, that rather limited replication studies have been conducted to confirm these biobehavioral profiles of the secure and insecure attachment groups in the Strange Situation (see Fox & Hane, 2008) and the limited work done has not consistently found the same effects. Spangler and Grossman's (1993) study did not find differences in heart rate between secure and insecure infants during reunion, nor did Zelenko and colleagues (2005) or Oosterman and Schuengel (2007) in a sample of older children. However, Spangler and Grossman (1993) did find elevated heart rate in insecure infants, particularly disorganized infants, during the final separation episode in the Strange Situation. More recently, Hill-Soderlund and colleagues (2008) found that avoidant (vs. secure) infants exhibited greater decreases from baseline to separation in respiratory sinus arrhythmia (RSA), a marker of parasympathetic activity, suggestive of a greater recruitment of internal resources toward actively coping with stress. In a sample of 4.5-year-olds, Stevenson-Hinde and Marshall (1999) found that secure children who also scored low on behavioral inhibition showed lower heart rate after reunion than other children. This may indicate therefore that temperament and attachment interact in determining the level of physiological stress observed during separation and reunion. This is a highly plausible explanation, as the former relates to children's reactive fearfulness or innate disposition to negative affectivity while the latter concerns how attachment behavior is deployed to regulate fear or negative affect. Similarly, Nachmias, Gunnar, Mangelsdorf, Parntz, and Buss (1996) found that more behaviorally inhibited children had higher poststress cortisol levels if they were also insecure, but not when they were securely attached to their mother. Security of attachment may thus buffer against stress or act as a moderator of the initial physiological disposition (see van IJzendoorn & Bakermans-Kranenburg, 2012).

Rather more work has explored the role of markers in the endocrine axis of the stress system, and in particular the hormone cortisol, which is a key player in the hypothalamic-pituitary-adrenocortical (HPA) axis (see Juster et al., 2010). In a now classic study, Spangler and Grossmann (1993) measured 41 infants' salivary cortisol concentrations before and 15 and 30 minutes after the Strange Situation (in keeping with the relatively slow rise time of the cortisol response). Their results indicated that insecure infants showed greater increases in cortisol from baseline to post–Strange Situation, an effect that was most pronounced in the disorganized group. Nachmias and colleagues (1996) also found elevated cortisol responses to the Strange Situation in insecure infants, although in this case only among those infants who were assessed as temperamentally inhibited. Spangler and Schieche (1998) similarly found higher cortisol responses in the Strange Situation amongst insecure infants who were also highly temperamentally inhibited. Notably, it was the resistant infants in this case that showed the highest cortisol concentrations following the Strange Situation within this group.

A more recent study by Bernard and Dozier (2010) found evidence that disorganized infants ($N = 8$) may in fact show lower basal cortisol levels than infants in the other attachment categories ($N = 24$). Unlike all other studies conducted previously, Bernard and Dozier contrasted cortisol responses to the Strange Situation to a control task (play interaction), and found that disorganized infants showed a greater increase in cortisol specifically in response to the Strange Situation. For secure and organized insecure infants, neither the Strange Situation nor the play task elicited a significant change from baseline in cortisol concentration. In light of the modest sample size, the lack of effect for the insecure organized groups is difficult to interpret. However, the study is very valuable in indicating the specific relevance of an attachment-related context for eliciting heightened stress responses from disorganized infants. Frigerio et al. (2009) failed to find heightened cortisol reactivity to the Strange Situation in a sample of 114 infants. In the largest study to investigate physiological function and attachment to date ($N = 369$), Luijk and colleagues (Luijk, Saridjan, et al., 2010) did not find evidence of greater cortisol reactivity to the Strange Situation among disorganized infants, but instead found that resistant infants showed the largest increases in cortisol from baseline to post-SSP, which was particularly pronounced when their mothers reported high levels of depression. The study also examined diurnal variation in cortisol, and demonstrated that disorganized children showed a more flattened diurnal cortisol pattern compared to nondisorganized children. In a follow-up paper on the same sample, Luijk, Velders, et al. (2010) found that the cortisol reactivity effect of resistant attachment was greater still for infants with the T allele of the FKBP5 gene, which is involved in glucorticoid receptor function (and which showed a significant main effect on cortisol response as well). Related to that, Frigerio et al. (2009) examined several gene polymorphisms that had been previously related to physiological stress sensitivity (5HTTLPR, GABRA-6 [GABA receptor gene], and COMT), and although no attachment-by-gene interactions were found for cortisol, two of these three genes interacted with attachment for another stress marker, alpha amylase. Alpha amylase, which is produced in saliva, is a sensitive marker of sympathetic nervous system activity, and results indicated that insecure children with relevant alleles in the 5HTTLPR (short-form) and GABRA-6 (CC genotype) genes were prone to larger increases in alpha amylase in response to the Strange Situation. Taken together, although the evidence is not uniform, results of psychophysiological studies of attachment suggest that insecure infants show greater physiological arousal to separation or poorer down-regulation of stress during reunion. Several studies suggest that these effects are stronger or more robust when endogenous factors such as fearful or inhibited temperament, or certain gene polymorphisms, are also present.

In addition to the question of physiological function during separation-reunion situations, several studies have addressed the relationship between attachment security and stress sensitivity in contexts outside the Strange Situation, thereby addressing the generalizability of stress-attachment associations and their broader developmental significance. These studies indeed indicate that attachment influences stress responding outside the Strange Situation, particularly when combined with temperamental traits related to negative affect. Thus, in a cross sectional study by Nachmias and colleagues (Nachmias et al., 1996) insecure infants who were also temperamentally inhibited were found to show greater cortisol responses to a challenging/fear provoking task (not involving separation). Furthermore, in the same sample, Gunnar and colleagues found that insecure (as assessed at 18 months) and temperamentally inhibited infants showed greater cortisol responses to an inoculation at 15 months relative to secure, inhibited infants (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996). Schieche and Spangler (2005) also found that infant attachment and temperamental inhibition interacted in their contribution

to toddlers' cortisol responses to a challenging task. Specifically, resistant and disorganized, though not avoidant, infants who were also temperamentally inhibited, showed less of a decrease in cortisol concentration than secure inhibited toddlers and noninhibited toddlers. The implication was that the flattened decline in this group reflected an increase in cortisol output superimposed on the standard diurnal pattern. Van Bakel et al. (2004) failed to find direct evidence of greater cortisol reactivity to a challenging task in insecure 15-month-olds, assessed using the attachment Q-set, but they did find that secure attachment moderated the association between stress responsiveness and cognitive function. Gilissen, Bakermans-Kranenburg, van IJzendoorn, and van der Veer (2008) showed that temperamentally fearful children with less secure relationships showed the highest skin conductance reactivity to a scary film clip, whereas comparable children with more secure relationships showed the lowest skin conductance activity. A particularly important example of work addressing the role of attachment in children's stress responses outside the Strange Situation is the work of Ahnert and colleagues (Ahnert, Gunnar, Lamb, & Barthel, 2004). These authors looked at children's stress response during the transition to childcare. Large increases in cortisol relative to baseline were observed during the transition to care in these toddlers, and differences in this response were found in relation to attachment security during the adaptation phase, when the toddlers' mothers remained present in the childcare setting. As expected, secure toddlers showed lower cortisol levels during this phase than their insecure counterparts. This difference was not apparent at later phases of the transition to childcare, when the mothers were no longer present.

Rather little work has directly tackled the *longitudinal* associations between attachment and physiological function. However, an important study by Burgess, Marshall, Rubin, and Fox (2003) found that avoidant attachment, though not concurrently associated with RSA in infancy (14 months) was predictive of *lower* resting heart rate and RSA at age 4 years. Avoidant infants, particularly those that had also been assessed as temperamentally uninhibited at age 2, also had higher rates of externalizing behavior problems. This set of associations is significant in light of the fact that lower resting heart rate is a consistent correlate of externalizing behavior problems in later childhood (Ortiz & Raine, 2004). It has been argued that this pattern of physiological hypoarousal, in addition to being influenced by genetic and temperamental factors, may result from developmental changes in the stress system in which early chronic stress leads to a subsequent dampening of the stress system (Gunnar & Quevedo, 2007). Thus, one might speculate that one pathway from attachment insecurity to externalizing problems occurs via downward alterations in the stress system brought about by early physiological hyperarousal associated with avoidant attachment. The modest effect size for the association between avoidance and externalizing problems referred to earlier might suggest that this association is contingent on other risk factors, either temperamental in nature, or related to other psychosocial variables.

One key issue that has not been rigorously addressed thus far is the role of parental behavior in accounting for associations between attachment and children's stress functioning. There is ample evidence that the quality of parental care is linked to the effectiveness with which the child's physiological arousal is regulated, and it is unclear whether this can explain the effects of attachment, or whether the child's attachment behavior or pattern makes a unique and distinctive contribution (Gunnar, 2005). This is an important area for future research.

Of particular note, two studies have reported positive effects of attachment-focused interventions on cortisol function in young children. Dozier, Peloso, Lewis, Laurenceau, and Levine (2008) found that their Attachment and Bio-behavioral Catch-up (ABC) intervention, which is focused on promoting security of attachment and in supporting the child's stress regulation through sensitive and responsive care, led to a reduction in basal cortisol in a sample of infants and toddlers in foster care, relative to a randomly assigned control treatment (RCT). Bakermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, and Juffer (2008) also examined changes in cortisol function in an RCT of a sensitivity-focused video-feedback intervention for toddlers at risk of behavioral problems. Although these authors found no main effect of the intervention on cortisol activity, as assessed at home during the course of the day, they found an interaction between the intervention and variation in the dopamine D4 VNTR. Among children with the 7-repeat allele of the dopamine receptor gene, who might be considered at risk of cortisol hyper-reactivity, the intervention appeared to lead to a reduction in basal cortisol. Of course, what cannot be determined is whether changes in security of attachment, sensitivity of care, or both, mediate these physiological outcomes, although the latter study also indicated that children with the 7-repeat allele showed the strongest decrease in externalizing problems when their mothers increased more than average in the use of sensitive discipline strategies (Bakermans-Kranenburg, van IJzendoorn, Pijlman, et al., 2008). These studies provide important evidence

concerning the causal role of parental sensitivity either via attachment or independently, on the child's functioning at the level of biology. Further work along these lines in the future would be highly desirable, particularly studies testing the mediating role of treatment-related changes in physiology for children's subsequent adjustment.

In summary, there is reasonably consistent, albeit mostly correlational, evidence that attachment security and insecurity relate in theoretically predicted ways to variations in young children's stress functioning. There is evidence that this relationship, particularly with respect to cortisol, but possibly other stress markers as well, is moderated by a range of factors, including maternal depression, temperament and genetic factors considered more broadly. In general, the evidence tends to suggest that the disorganized and resistant groups are most prone to increased cortisol responses to separation or other stressors. Further, it remains unclear whether attachment plays a specific role that can be distinguished from the ongoing effects of parental support in these associations. Finally, children's stress physiology appears to be modifiable by attachment-focused interventions, although how generalizable and sustained these effects are remains to be established. Thus far, no study has directly tested the role of stress physiology in mediating the links between attachment and children's developmental outcomes in correlational or treatment studies.

Cognitive, Social-Cognitive, and Affective Mediators

As noted previously, a range of *psychological* mechanisms has been proposed as potential mediators of the effects of attachment insecurity and disorganization on children's adaptation and risk for maladjustment. However, remarkably little work has systematically investigated the psychological or affective mechanisms mediating longitudinal associations between attachment and later outcomes. Nevertheless, studies do suggest that attachment is coherently linked to several cognitive and affective functions, in ways that are broadly in line with predictions from attachment theory. For an excellent in-depth review, the reader is encouraged to read Dykas and Cassidy (2011). In the following sections, we summarize the results of studies testing links between attachment and (1) social information processing, (2) theory of mind, and (3) emotional reactivity and regulation.

Social Information Processing

A number of studies have investigated the natural interpretation arising from attachment theory that internal working models should influence children's encoding and interpretation of social experiences or socially relevant cues in systematic ways. One of the earliest studies to examine this idea was conducted by Cassidy and colleagues (Cassidy, Kirsh, Scolton, & Parke, 1996). In the first of their reported experiments they followed a group of 60 3.5 year-old children who had been observed in the Strange Situation at 15–18 months to assess several aspects of their representations of, or social-information processing about, peers. In the first experiment, children were read several stories involving social interactions in which the participant was the imagined protagonist. Following a social-challenge event (e.g., the child is hit by a ball, a child's toy is broken by a peer), the experimenter asked a number of questions regarding peer intentions, imagined social responses that the child might engage in, and their thoughts about their peers' feelings. Although inferred intentions did not vary by prior attachment status, avoidant children tended to imagine engaging in more negative social actions in response to the challenge (than previously secure and resistant children). In a second experiment, older children's (Grade 1) responses to the same stories were examined in relation to concurrent attachment security as assessed using the Main and Cassidy (1988) procedure. In this older sample ($N = 33$), evidence emerged of insecure children's tendency to infer hostile intentions to peers, to entertain negative social actions in response to peer challenge and to provide less positive responses regarding their peers' feelings after the event had occurred. The findings also indicated that these differences in thinking style were related in turn to the quality of children's peer relations, as assessed via a sociometric instrument. A subsequent study by Kirsh and Cassidy (1997) looked at children's attention to attachment-relevant stimuli (pictures of mother–child interactions of varying valence) using the same cohort as study 1 in Cassidy et al. (1996). In the first task, they showed children three pictures side-by-side, depicting positive, negative, and neutral interactions. Although no differences were found in children's looking time to pictures of differing valence, avoidant children tended to look away from the stimuli more frequently than the other groups in general. Of course, while the content of the stimuli may well have been relevant to this difference, it is also possible that general inattentativeness could also explain such a finding. To test this, they showed children attachment-relevant and attachment-irrelevant images side by side and measured their looking time. Consistent with expectations, the authors found that insecure children spent less time looking at the attachment-relevant images, compared to the attachment-irrelevant images,

than their secure counterparts. In the same sample, Kirsh and Cassidy also found evidence to suggest that recall for attachment-relevant stories may be biased by attachment security, with previously secure children showing better memory for stories involving responsive care than insecure children. More recently, in a sample of 7–8 year-olds, Ziv, Oppenheim, and Sagi-Schwartz (2004) used films depicting negative, neutral or ambiguous peer responses to a bid by the study child to initiate play, to test the association between early attachment and attributional biases. Possibly reflecting Cassidy et al.'s finding in their experiment 1, Ziv et al., did not find differences associated with attachment at the level of attribution, but rather in their evaluations of prosocial responses. In particular, secure children tended to endorse a greater belief in the value (and positive consequences) of competent social responses to the peer challenge than insecure children. Although working with a much older age group, Dykas, Woodhouse, Ehrlich, and Cassidy (2012) recently presented fascinating evidence regarding the connections between social-information processing and adolescent attachment. In this study, adolescents interacted with an unfamiliar peer for 10 minutes, and were then subsequently interviewed regarding their perceptions or recollections of that interaction. Immediately after the interaction adolescents' perceptions of the interaction did not differ according to their attachment status. However, when the same assessment was repeated two weeks later, insecure adolescents' recollection of the interaction shifted in a negative direction more steeply than secure adolescents, viewing the interaction as having been less positive and more hostile (on the part of the unfamiliar peer). The majority of these studies (with the exception of Cassidy et al., 1996; experiment 2) have not directly tested whether these social-cognitive processes might mediate attachment effects on social competence or psychopathology (though see Raikes & Thompson, 2008, for one study not finding mediation).

Theory of Mind

Several authors have suggested that attachment may promote the development of children's Theory of Mind, or mentalizing abilities, and that this might provide an account of the longitudinal benefits associated with secure attachment. At least nine papers have been published reporting on the association between attachment and theory of mind, as assessed by a range of procedures designed to measure false belief understanding or perspective taking. These studies have produced mixed results. One of the first of these was a report by Meins and colleagues (Meins, Fernyhough, Russell, & Clark-Carter, 1998). Using an unexpected transfer task, they found that children who had been secure in infancy showed superior false belief understanding at age 4 (though not age 5). This longitudinal finding from infancy to age 4 was replicated more recently by McElwain and Volling (2004), although not by Meins et al. (2002) or Symons and Clark (the latter relating age 2 security to age 5 false belief understanding; see Symons & Clark, 2000). Laranjo and colleagues (2008) did not find overall associations between attachment, as assessed with the AQS at age 15 months, and perspective taking at age 2 years, although an association was apparent for boys. In an early cross sectional study, Fonagy, Redfern, and Charman (1997) found that attachment, as measured with the Separation Anxiety Test, was associated with belief-desire reasoning in a sample of 3–6-year-olds (see also De Rosnay & Harris, 2002). Other cross sectional studies have also produced positive findings (Arranz, Artamendi, Olabarrieta, & Martin, 2002; Repacholi & Trapolini, 2004; Symons & Clark, 2000) as well as at least one negative finding (Greig & Howe, 2001). Broadly speaking then, there is evidence that attachment may be related to theory of mind performance, but the association is not consistently replicated, particularly in longitudinal studies. Meins and colleagues (2002) argue that maternal mind-mindedness, which as we noted already is correlated with attachment security, is a more robust predictor of children's theory of mind performance than attachment, and may account for its apparent effects.

There is relatively consistent evidence that attachment is associated with children's recognition and understanding of emotions (as opposed to false beliefs). For example, Steele and colleagues (Steele, Steele, & Croft, 2008; Steele, Steele, Croft, & Fonagy, 1999) found that children aged 6 (though not 11) who had previously been insecure with their mothers in infancy were poorer at labeling emotional faces, particularly positive emotions, and understanding mixed emotions, than secure children. In a cross sectional study of 7-year-olds, Colle and Del Guidice (2011) found that insecure children (as assessed with the Manchester Child Attachment Story Task [MCAST]; Goldwyn et al., 2000) performed less accurately in matching pairs of dynamic emotional faces. Laible and Thompson (1998) found that secure children (using mother-completed AQS) between the ages of 2.5 and 6 years performed better on an emotional perspective taking task (after controlling for age), an effect which was mostly apparent in relation to negative emotions. These results were also replicated by Greig and Howe (2001) in a sample of 4.5-year-olds assessed using the Attachment Story Completion Task of Bretherton, Ridgeway and Cassidy (1990) and in two

low-SES samples of preschoolers (Kidwell et al., 2010; Raikes & Thompson, 2006). Notably, in the Raikes and Thompson (2006) study, mother–child conversations in secure dyads tended to involve more frequent reference to emotion terms, which itself was associated with children's emotional perspective taking/understanding (see also Oppenheim, Koren-Karie, & Sagi-Schwartz, 2007; Raikes & Thompson, 2008). Although few studies have examined clinical samples, Barone and Lionetti (2012) found that disorganization in the MCAST was associated with poorer overall emotion comprehension (indicating recognition of emotion, understanding the causes of emotion and emotion regulation strategies) in a sample of late-adopted preschoolers.

Overall then, there is relatively consistent evidence that attachment security is related to better emotion recognition and emotion understanding in preschool and school aged children, although existing studies tend to be based on modest samples, using a wide range of tools for assessing attachment and emotion understanding, and longitudinal data beginning in infancy is limited. Few studies have thus far directly tested the mediating role of emotion understanding or theory of mind in the association between attachment and psychopathology. The broader literature on the developmental significance of social-cognitive skills in children's social competence suggests that this is a hypothesis worthy of further investigation (e.g., see Astington, 2003). As eloquently summarized by Oppenheim (2006), many authors in the field see deep connections between security of attachment, the coherence of internal working models, and the ways parents and children co-construct affectively meaningful narratives of social and relational experiences during interactions. These internalized, co-constructed narratives, scripts or models may then shape future interpersonal and emotional functioning, in ways that may enhance or undermine adaptation and adjustment. The evidence reviewed in the previous sections appears broadly consistent with this way of thinking.

Emotional Reactivity and Self-regulation

The first longitudinal studies of attachment and later child development drew attention to the potentially important mediating role of the child's emerging capacity for self-regulation (Arend, Gove, & Sroufe, 1979). Arend and colleagues (1979) for example found that infants classified as secure at 18 months showed greater ego-resiliency and ego-control as preschoolers, based on both laboratory observations and reports from kindergarten teachers. Similarly, in the well-known Minnesota study (Sroufe et al., 2005b), consistent evidence was found suggesting that insecure children were more dependent on teachers in preschool and preadolescence (Urban, Carlson, Egeland, & Sroufe, 1991). Gilliom, Shaw, Beck, Schonberg, and Lukon (2002) examined emotion-regulation strategies directly in a sample of low-SES 3.5 year-old boys by observing emotion-regulatory behaviors during a task designed to elicit frustration. The authors coded regulatory behaviors, including active distraction, seeking information, passive waiting, and comfort seeking as well as the child's overall emotional response. In addition to showing that the hypothesized regulatory behaviors did reduce children's levels of anger in temporal contingency analyses, they also found that secure boys tended to employ more of these regulatory behaviors during the frustration task than insecure boys. These connections between emotional reactivity and regulation appear to emerge early in development. For example, Leerkes and Wong (2012) found that during a frustration task avoidant infants engaged in greater self-soothing and used fewer mother-directed regulatory behaviors than secure infants, and that resistant infants showed more negative affect and engaged in fewer adaptive regulatory strategies. Similarly, Sherman, Stupica, Dykas, Romas-Marcuse, and Cassidy (2013) found that resistant infants at age 1 showed the greatest emotional reactivity in a frustration task, and avoidant infants the least, with secure infants falling in the middle. Somewhat consistent with this, Martins and colleagues (Martins, Soares, Martins, Tereno, & Osorio, 2012) found that avoidant infants showed less emotional reactivity during object-focused play with the mother than secure or resistant infants. Underscoring the potential importance of emotion-related processes for psychopathology, Murray and colleagues (2011) recently found that the association between maternal postnatal depression and later offspring depression was mediated by a pathway involving insecure (primarily avoidant) attachment at 18 months and low ego resilience in childhood. Finally, Dawson and colleagues (Dawson et al., 2001) have also presented data indicating that insecurely attached infants show differences in EEG spectra—specifically reduced relative left frontal alpha power—than securely attached infants. Such neural effects are interesting, because alpha asymmetries of this nature have been found to be quite stable over time (Vuga, Fox, Cohn, Kovacs, & George, 2008) and are associated with negative emotionality and behavior problems in infancy and toddlerhood (e.g., Dawson et al., 2003).

Berlin and Cassidy (2003) directly addressed the suggestion indicated by several past studies that mothers of insecurely attached infants react in systematic ways to encourage or discourage the infant's affect expressiveness. Mothers were asked a series of questions regarding their attitudes to their child's emotions, and in particular their tendency to control their child's expression of negative affect. Mothers of avoidant children reported exerting greater control, and mothers of resistant children less control, over their child's emotions than mothers of secure children. These findings are quite consistent with theorizing by Cassidy (1994) and Berlin and Cassidy (1994), that parental working models of attachment influence mothers' unconscious responses to the child's affect that in turn serve to regulate or maintain the parent's state of mind with respect to attachment.

In addition to emotion-regulation, some studies have also indicated that security of attachment may influence children's self-regulation skills more broadly. For example, using data from the NICHD SECCYD study, Fearon and Belsky (2004), found that insecure attachment in infancy was associated with later attentional control, as measured objectively using a continuous performance test at age 54 months. The connections between attachment and attentional control in this study also appeared to be amplified by social-contextual risk, particularly for boys. In a more recent paper, Drake, Belsky and Fearon (2013) examined teacher's reports of social self-control between Grades 1 and 5 and found that earlier security, at both 15 months and 3 years, predicted higher levels of social self-control. Social self-control in turn predicted Grade 5 school engagement, and mediated the effects of early attachment on school engagement.

Thus, there is evidence that attachment is associated with a broad range of cognitive, social-cognitive, and emotional competencies that are plausible candidates for mediating mechanism in the effect of attachment security and insecurity on children's adjustment. Relatively little work has systematically examined these processes longitudinally and tested mediation directly, and fewer studies still have examined these various candidate mediators simultaneously, to tease apart their joint and independent effects. The greater capacity for flexible emotion-regulation or self-regulation might plausibly underpin some of the other competencies associated with security, including emotion understanding and empathy (e.g., see Panfile & Laible, 2012), as well as mediate effects of attachment on later emotional and behavioral problems (e.g., see Belsky, Fearon, & Bell, 2007), but large-scale studies designed to test this programmatically have yet to be done.

ADULT ATTACHMENT AND PSYCHOPATHOLOGY

The conceptual links between attachment and psychopathology have been examined for both infant attachment and adult attachment classifications. Past theorizing tended to assume that children with a resistant attachment should show more internalizing problems, and avoidant children would be at risk for externalizing problems, whereas children with disorganized attachment relationships might be at a heightened risk for developing either internalizing or externalizing symptoms (see previous section). As noted earlier in this chapter, meta-analytic results provided only partial support for these hypotheses, in particular the role of resistant attachment appeared to be less clear-cut than expected (see Fearon et al., 2010; Groh et al., 2012).

The expected associations between the organized adult attachment classifications and psychopathology were roughly analogous to those suggested for children: preoccupied attachment representations might be associated primarily with internalizing disorders such as depression or borderline personality disorder, whereas dismissing representations were expected to be associated with externalizing indices of distress, such as eating disorders, conduct disorders, and hard-drug use (Tyrrell & Dozier, 1997; Tyrrell, Dozier, Teague, & Fallot, 1999). Contrary to infant disorganized attachment however, the hypothesis concerning the psychopathological correlates of unresolved attachments were more distinctively suggested to be in the range of disorders with a dissociative component such as posttraumatic stress disorders (Harari et al., 2009; Hesse, 1999; Liotti, 2004; Sroufe et al., 2005b).

From the number of studies involving clinical samples it is evident that from the early years onward the AAI has been considered relevant to psychopathology. Selecting studies that were based on the original coding system with the conventional three-way and four-way classifications of the AAI (Main et al., 2003), Bakermans-Kranenburg and van IJzendoorn showed in a review of the first 10,000 AAIs that 76 out of 218 studies involved clinical samples, whereas "only" 36 studies focused on nonclinical low-risk mothers and 13 on fathers (Bakermans-Kranenburg & van IJzendoorn, 2009).

The nonclinical low-risk North American mothers provided a norm distribution for comparison with classification frequencies in clinical groups. They showed a combined distribution of 23% dismissing, 58% autonomous, and 19% preoccupied classifications ($N = 748$). With the unresolved category included, the norm distribution was 16% dismissing, 56% autonomous, 9% preoccupied, and

18% unresolved classifications (N = 700). These distributions were quite similar to those in the larger group of all nonclinical and not at-risk groups (irrespective of gender, country, age, and parenthood), with a three-way distribution of 29% dismissing, 56% autonomous, and 14% preoccupied (N = 4,392), and a four-way distribution of 24% dismissing, 50% autonomous, 9% preoccupied, and 16% unresolved (N = 4454). Thus, albeit a "clean" and therefore selective group of North American nonclinical low-risk mothers, its usefulness as a norm group is corroborated by its similarity to the larger, more global group of nonclinical samples.

As expected, the combined clinical groups showed an extremely deviating distribution of AAI classifications compared with the norm group. In the combined clinical samples, 37% were classified as dismissing, 27% as autonomous, and 37% as preoccupied. In short, a large majority (73%) of the clinical adults (N = 1956) were classified as insecure. Clinical samples were more often dismissing than the norm group, and in contrast to samples at-risk (e.g., single low-SES mothers, adolescent mothers) they were also more often preoccupied. With the unresolved category included, the combined sample of clinical individuals showed the following distribution: 23% dismissing, 21% autonomous, 13% preoccupied, and 43% unresolved. The unresolved category was strongly overrepresented, and the dismissing and preoccupied categories were overrepresented as well, but to a lesser extent than the unresolved category.

In search for links between specific disorders and one or more of the insecure attachments we compared the combined distributions of clinical samples of a similar kind with those of the norm group. Disorders with an internalizing orientation, in particular borderline personality disorders, have been suggested to be associated with maximizing attachment signals. Indeed, the three-way distribution of suicidal and borderline patients showed a significant overrepresentation of preoccupied attachments, whereas dismissing attachments were not significantly overrepresented. In the four-way distribution unresolved attachments were also overrepresented. Similarly, individuals with abuse experiences or current PTSD were particularly characterized by unresolved loss or trauma. In contrast, depressive symptomatology was not related to higher rates of unresolved attachment, but both in the three-way and four-way distributions dismissing and preoccupied classifications were overrepresented.

The strong association between PTSD and unresolved loss or trauma is perhaps to be expected given the similarity in indicators, notably intrusive symptoms and avoidance (Fearon & Mansell, 2001). Stovall-McClough and Cloitre (2006) found that abused women who were classified as unresolved with regard to trauma were 7.5 times more likely to be diagnosed with PTSD. Examining combat veterans with PTSD, the continuous score for unresolved deployment-related trauma overlapped almost completely with severity of PTSD (r = .80) in one study (Harari et al., 2009). The overlap between unresolved loss and PTSD in the other study was 50%, but in that study combat-related traumatic experiences were not queried in the AAI, decreasing the overlap between PTSD and unresolved state of mind (Nye et al., 2008). Such results could be considered as validation of the unresolved category; however they also show the limits of the coding system in terms of the incremental validity of the classification over existing PTSD measures such as the CAPS (Clinician Administered PTSD Scale; Blake et al., 1998).

Schizophrenia appeared to be related to dismissing attachment, but only one study was available at the time of the meta-analysis (Tyrrell & Dozier, 1997). More recently, one study using the AAI added to that database, confirming the link with dismissing attachment in a sample of 34 patients with a first episode of psychosis (MacBeth, Gumley, Schwannauer, & Fisher, 2011). Self-reported schizotypal symptomatology was related to AAI unresolved trauma and unresolved loss in a psychiatric sample of trauma survivors (Riggs et al., 2007). Other studies used self-reported adult attachment measures and are thus basically incomparable, and the results were inconsistent (versus Ponizovsky, Nechamkin, & Rosca, 2007; e.g., Ponizovsky, Vitenberg, Baumgarten-Katz, & Grinshpoon, 2013).

Externalizing problems, in particular antisocial or conduct disorders, were expected to be associated with dismissing attachments, which was confirmed; however at the same time the preoccupied category was also overrepresented. The four-way distribution demonstrated that externalizing problems were associated with increased rates of unresolved loss or trauma as well. When distinguishing between the various types of violence, that is violence within the family (against partner or child), against the outside world (criminal offenders), or against their own body (eating disorders, drug addiction), we found that family violence was associated with more preoccupied representations, whereas violence against the outside world was strongly associated with dismissing attachment representations, and self-directed violence with both dismissing (three-way) and preoccupied and unresolved (four-way) classifications. The three-way and four-way distributions of samples with self-directed violence were based on

samples that showed hardly any overlap (the majority of these studies presented *only* three-way or four-way distributions). This may explain the pattern of results with an overrepresentation of the dismissing classification in the three-way distribution, and an overrepresentation of the preoccupied classification in the four-way distribution. A more recent study on incarcerated women, most of whom had been convicted for drug-related crimes, showed an underrepresentation of secure classifications and an overrepresentation of preoccupied and unresolved classifications (Borelli, Goshin, Joestl, Clark, & Byrne, 2010).

Given that the AAI was developed in the context of the prediction of infant-parent attachment and child socioemotional development, one might expect an enhanced risk of parental insecurity in samples with child psychopathology. Insecure parental attachment increases the likelihood of insensitive parenting (van IJzendoorn, 1995), biased perceptions of child behavior and intentions, harsh discipline (Verschueren, Dossche, Marcoen, Mahieu, & Bakermans-Kranenburg, 2006), and instability of the caregiving environment (e.g., Crowell, Treboux, & Brockmeyer, 2009). Indeed, the large majority of parents of children with psychopathology were found to be insecure (83%). Both the dismissing and preoccupied classifications were overrepresented. Importantly, only psychological problems appeared associated with more insecure attachment, not physical disability, even if they were severe and with hampering consequences for social interactions, like blindness or deafness. The set of studies with adults with physical handicaps was small, but the finding that their attachment distributions did not significantly deviate from the norm is important in terms of discriminant validity (Bakermans-Kranenburg & van IJzendoorn, 2009).

From a clinical perspective, the Cannot Classify classification might be of particular interest. This category, identified by Hesse (1999, 2008), was intended to capture AAIs that showed a global collapse of coherence across the entire discourse. Contrasting strategies for maximizing and minimizing the expression of attachment within the context of one and the same interview is a hallmark of this classification. Minde and Hesse (1996) presented a case study of a mother with highly conflicted feelings toward her role as a caretaker, where the identification of her attachment representation as Cannot Classify (incoherent and switching between contrasting strategies) helped the therapist in choosing a specific treatment approach. In normal, nonclinical samples hardly any adult is assigned this classification, but in samples of criminal offenders (van IJzendoorn et al., 1997), victims of sexual abuse (Stalker & Davies, 1998), and suicidal adolescents (Adam, Sheldon-Keller, & West, 1996) the category was overrepresented. However, at the time of the meta-analysis an insufficient number of AAI studies with separate Cannot Classify ratings hampered testing the association of this category with psychiatric disorders, and that situation has not changed since.

One study reported on the association between psychopathology and reflective functioning. Reflective functioning in the context of the AAI refers to the adult's capacity to reflect upon memories of the childhood relationship with the parents in mentalizing terms, that is, showing awareness of mental states in others, and the ability to experience one's own and others' emotions in a nondefensive way without becoming overwhelmed (Fonagy, Target, Steele, & Steele, 1998; Slade, 2005). Reflective functioning is closely related to metacognitive monitoring during the AAI, and indeed associated with AAI security (Fonagy, Steele, Steele, & Holder, 1997). During the AAI, female inpatients with depression showed significantly lower capacity for mentalization as assessed with the Reflective Functioning scale than healthy controls (Fischer-Kern et al., 2013).

A literature search with the key words *adult attachment* and *psychopathology* shows that not that many studies with clinical samples have accumulated after our meta-analysis was published in 2009. The majority of the studies on clinical samples used self-report measures of adult attachment, such as the Attachment Style Questionnaire (Feeney, Noller, & Hanrahan, 1994), or the Experiences in Close Relationships questionnaire (Bartholomew & Horowitz, 1991; see also Mikulincer & Shaver, 2007). The preference for an efficient paper-and-pencil or website-based instrument is not difficult to understand, but the results are—notwithstanding some early optimism (e.g., Bernier & Dozier, 2002; Shaver & Mikulincer, 2002)—not directly comparable with those of the AAI. As demonstrated meta-analytically for nonclinical samples, the overlap between the AAI and adult attachment questionnaires is trivial to small (r =.09; Roisman, Holland, et al., 2007). Similarly, in a psychiatric sample of trauma survivors self-report and AAI attachment classifications were not related, and different associations with psychological dysfunction emerged for the two measures (Riggs et al., 2007). In a study on pregnant women from deprived backgrounds, Pianta, Egeland, and Adam (1996) found that preoccupied adult attachment representations appeared to be associated with highest levels of self-reported psychopathology (as assessed with the Minnesota Multiphasic Personality Inventory-2 [MMPI-2]) whereas females with dismissing attachments showed the lowest levels of psychopathology

as they emphasized independence and low anxiety. The secure-autonomous individuals scored in-between the two insecure groups. This suggests that self-reports of attachment, parenting style or psychopathology may suffer from systematic response biases with overly negative and worried reports by individuals with preoccupied attachments, and overly positive and defensive reports by dismissing individuals.

In sum, whereas the original hypotheses suggested that preoccupied attachment representations might be associated primarily with internalizing disorders, dismissing representations with externalizing problems (Tyrrell & Dozier, 1997; Tyrrell et al., 1999), and unresolved attachments would predispose to the development of disorders with a dissociative component such as posttraumatic stress disorder, the emerging evidence confirms only the latter hypothesis consistently: indices for unresolved loss or other trauma are strongly correlated with posttraumatic stress symptoms assessed independently. Externalizing and internalizing disorders both show elevated levels of preoccupied and dismissing attachments but more specific associations have remained equivocal across studies. Firm evidence on the causal direction of the associations is still largely absent because the overwhelming majority of studies in this area are correlational with most of them concurrently measuring both attachment and psychopathology. More experimental studies are therefore badly needed.

MECHANISMS IN ADULT ATTACHMENT AND PSYCHOPATHOLOGY

What are the neural mechanisms underlying attachment-related individual differences in parenting behavior? Adults with insecure internal working models of attachment may process attachment-relevant social information in one of two ways, either defensively excluding the information from further processing, or processing the information in a negatively biased manner, congruent with their negative attachment-related experiences (Dykas & Cassidy, 2011). In the past decade, the neural mechanism underlying the perception of infant attachment signals, in particular infant crying, has been the focus of several functional Magnetic Resonance Imaging (fMRI) studies. These studies have shown that a highly interactive cognitive-affective neural network is involved in the perception of infant crying (Bos, Panksepp, Bluthe, & van Honk, 2012).

The amygdala is an important functional hub within this network. The amygdala is part of the limbic system and plays a role in the detection of threat and the experience of fear and aversion (Davis & Whalen, 2001; Fusar-Poli et al., 2009; Gamer, Zurowski, & Buchel, 2010; Morris et al., 1998). It is activated during exposure to infant crying (Lorberbaum et al., 2002; Riem et al., 2011; Seifritz et al., 2003) and connected with other brain regions that are involved in the perception and evaluation of crying such as the orbitofrontal cortex and the anterior cingulate cortex (Riem et al., 2012). Heightened amygdala activation is an indication of hyperemotionality and has been observed in depression and anxiety disorders (Rauch et al., 2000; Yang et al., 2010) and in intrusive mothers (Atzil, Hendler, & Feldman, 2011).

Several fMRI studies also point to a role of the amygdala in the processing of attachment-related information. For example, Buchheim et al. (2006) found elevated amygdala activation in individuals with unresolved loss during the Adult Attachment Projective (AAP). In 10-year-old children amygdala hyperactivity was found to mediate the association between adverse early attachment experiences (growing up in institutionalized care) and decreased eye-contact during dyadic interaction (Tottenham et al., 2011). This indicates that early adverse attachment experiences affect amygdala activity, possibly because of the vulnerability of the amygdala to environmental exposures in early life (Lupien, McEwen, Gunnar, & Heim, 2009; Sabatini et al., 2007) which in turn influences social behavior. Amygdala hyperactivity might thus be one of the mechanisms underlying the association between adult attachment representation and insensitive parenting.

A number of fMRI studies on the influence of adult attachment have focused on the perception of infant facial expressions. Strathearn et al. (2009), examining neural responses to own infant smiling and sad faces, found that mothers with an insecure attachment representation (as assessed with the Crittenden coding system) showed less activation in dopaminergic reward regions such as the ventral striatum compared with secure mothers. Lenzi et al. (Lenzi et al., 2012) found that individuals with a dismissing AAI classification showed more activation in the limbic and mirror neuron system and greater deactivation in the OFC and ACC in response to infant facial expressions compared with individuals with a secure representation. The authors suggest that hyperactivation of limbic and mirror system areas may reflect emotional dysregulation of infantile experiences of rejection and lack of protection, whereas increased deactivation of fronto-medial areas may be the expression of the inhibition of attachment behaviors, which is a typical aspect of dismissing attachment. Galynker et al. (2012) examined the influence of depression and AAI insecurity on neural responses to

images of the participant's mother, friend, or a stranger. No effects of attachment security on amygdala activation were found, perhaps because of the absence of negative affective stimuli.

The amygdala thus seems to be involved in both adult attachment and the perception of infant signals. Riem et al. (2012) explored whether amygdala hyperactivity played a role in the relation between insecure adult attachment representations and negative emotional and behavioral responses to infant crying. Adults with insecure attachment representations showed heightened amygdala activation when exposed to infant crying compared to individuals with secure attachment representations. In addition, they reported more irritation during infant crying, and they used more excessive force as indicated by grip strength using a hand-grip dynamometer during exposure to infant crying. The relation between attachment representation and emotional or behavioral responses to infant crying was however not mediated by amygdala activation, indicating that feelings of irritation and the use of excessive force in response to infant crying in insecure individuals cannot be solely explained by a hyperactive amygdala. Other brain regions involved in attachment-related influences on the perception of infant crying may be regions important for empathy and emotion understanding, such as the insula and the inferior frontal gyrus (IFG). Interestingly, intranasal administration of oxytocin decreased amygdala responses and increased insula and IFG responses to infant crying (Riem et al., 2011).

Disruptions in amygdala *connectivity* may also play a role in the negative perception of infant crying in insecure individuals. The amygdala is strongly connected with other brain regions within a neural network involved in the perception and evaluation of crying (Riem et al., 2012), and neural disorganization within this network has been associated with anxious parenting (Atzil et al., 2011). Disruptions in amygdala connectivity have been observed in patients with depression and anxiety disorders (Dannlowski et al., 2009; Pillay, Gruber, Rogowska, Simpson, & Yurgelun-Todd, 2006) and might also play a role in insecure attachment representations.

It has been proposed that the attachment system would be located in the orbitofrontal cortex (OFC; Schore, 2001). The OFC is involved in reward processing, emotional regulation, and the perception of infant signals (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Kringelbach, 2005; Kringelbach et al., 2008; Stein et al., 2007; Swain & Ho, 2010). The OFC shows a very rapid and specific response to infant faces and it has been suggested that this might be the brain basis for the "innate releasing mechanism" described by Lorenz (Kringelbach et al., 2008). However, it is by no means the only brain region involved in attachment, as evident from the significance of amygdala activity in attachment-related studies. Coan (2008) suggested that searching for the identification of a single attachment neural construct is like "trying to find the real artichoke by peeling away all its leaves." (pp. 242). The attachment system most likely relies on a comprehensive neural network and no single neural construct is decisive. Indeed, given the emotion, attention, motivation, empathy, decision-making and other thinking processing involved, it has been suggested that networks of hypothalamic–midbrain–limbic–paralimbic– cortical circuits act in concert to support sensitive parenting (Swain, 2011).

ATTACHMENT AND INTERVENTION

The field of attachment, with its empirically well-placed emphasis on the environmental causes of security and insecurity, its clearly specified mechanisms of influence, and its well-developed evidence base regarding the longitudinal outcomes linked to insecurity, provides a coherent framework for intervention. In this final section, we first focus on interventions developed to promote parental sensitivity or secure infant attachment, and then turn to interventions aimed at affecting insecure adult attachment, and the role of adult attachment (of both client and therapist) in clinical process and outcome. Intervention studies in this field serve several crucial functions: first, they help establish the plasticity of attachment to environmental modification. Second, they provide highly relevant information for examining causal questions about the mechanisms driving attachment security and insecurity. Third, and most importantly, they are vital for demonstrating the potential impact and clinical value of attachment as a focus for improving outcomes for children and adults.

Parenting Interventions

In light of the extensive evidence concerning the environmental determinants of early attachment and the consistent association between parenting sensitivity and attachment security, a vital question is whether early preventive interventions are effective in enhancing parental sensitivity and in doing so can they improve rates of infant attachment security. Furthermore, important secondary questions concern the type of intervention is most successful and for whom. A great deal of work has gone into the development and testing of attachment-inspired intervention or

prevention programs. The interventions apparent in the literature vary widely in intensity, duration, and focus, and the relevant studies have used highly divergent outcome measures. Some programs aim at enhancing parental sensitivity, others at affecting adult insecure attachment representations and still others at the parent-infant relationship (infant attachment). The vast majority of intervention studies have aimed to bring about change at the level of parental sensitivity or the level of infant-parent attachment, but a few address adult attachment as an outcome measure. We focus here on attachment-based parenting interventions and we do not include parent behavior-management interventions aimed at helping parents of children with severe behavior problems or conduct disorders (e.g., Scott & O'Connor, 2012; Webster-Stratton, Reid, & Hammond, 2004; Webster-Stratton, Rinaldi, & Reid, 2011). A meta-analysis on the effectiveness of sensitivity and attachment interventions with 81 studies (N = 7,636) on sensitivity and 29 studies (N = 1,503) on infant–parent attachment showed that randomized interventions with a focus on sensitive behavior were most effective in changing insensitive parenting (d = 0.33) as well as infant attachment insecurity (d = 0.20; Bakermans-Kranenburg et al., 2003). The most effective interventions did not include a large number of intervention sessions with the families, and they did not necessarily start before birth or even early in life. Indeed, it was notable that interventions with up to a maximum of 16 sessions, starting 6 months after birth or later, were most effective. Surprisingly, intervention studies were generally effective regardless of the presence or absence of multiple problems in the family. It might be expected that at-risk groups would have different needs, and thus may profit from different types of interventions, particularly more intensive interventions, than lower risk or normative populations. However, in the set of multiproblem samples (30 studies, N = 4,119 families), interventions focusing on sensitivity were more effective (d = 0.48) than all other types of intervention combined (d = 0.25). The most effective interventions consisted of fewer than 16 sessions.

A key finding from this meta-analytic work was that attachment insecurity emerged as being more difficult to change than maternal insensitivity. Nevertheless, the fact that sensitivity-focused interventions led to significant improvements in security, and that those studies that achieved a greater change in sensitivity (ds >.40) tended to yield better outcomes for the child's attachment security (d = .45). Interventions that were less effective in promoting sensitivity did not appear to lead to consistent changes in attachment security. Similar to intervention effects on parenting sensitivity, highly effective interventions (with a behavioral focus, i.e., improving parental sensitivity) were effective in improving attachment regardless of the presence or absence of multiple problems in the family.

For disorganized attachment, the overall effectiveness of parenting interventions was evaluated in two more recent meta-analyses (15 studies, N = 842) and, in marked contrast to findings regarding the impact of interventions on secure attachment, no significant overall impact of the interventions was found for disorganized attachment, d = 0.05 (Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2005, 2008). However, interestingly, the five intervention studies focusing on parental sensitivity were found to be effective in reducing rates of disorganized attachment (d = 0.26), and were significantly more effective than interventions with a different or broader focus (d = –0.08). This finding is intriguing as disorganized attachment has been more strongly and consistently associated with frightening and atypical parenting (e.g., Madigan, Bakermans-Kranenburg, et al., 2006) than parental sensitivity which was the focus of the effective interventions. It is possible that despite the focus on sensitivity, these interventions may also have been successful in reducing frightening or extremely intrusive or unresponsive parental behavior, or in reducing parental dissociation by focusing parents' attention on the child's behavior (Bakermans-Kranenburg et al., 2005). Unfortunately, thus far studies have not explicitly measured parental frightening or anomalous behavior as an outcome to test whether this might account for observed effects on disorganized attachment.

One of the interventions effective in reducing disorganized attachments is video-feedback intervention to promote positive parenting (VIPP; Juffer, Bakermans-Kranenburg, & van IJzendoorn, 2008) which is a short-term, home-based intervention directed at sensitive parenting behavior via video feedback. VIPP has been tested in a series of randomized control trials on a range of nonclinical and clinical samples (children with autism spectrum disorders—Poslawsky et al., 2014; including samples with eating disordered mothers—Stein et al., 2006; children at high risk for externalizing problems—Van Zeijl et al., 2006). The main objective of VIPP is to promote the parent's sensitivity by showing and stimulating mentalization of moments of sensitive parenting of the parent in carefully selected video fragments. In a randomized control trial with early adopted children the VIPP appeared to significantly reduce rates of disorganized attachment, probably because parents had learned to mentalize their interactions with the child, reflect on their own role in the relationship, and to keep the child's

attachment signals more in focus, and thus, were less distracted or dysregulated by other external or internal stimuli (Bakermans-Kranenburg et al., 2005).

Only a few intervention programs have the explicit goal of decreasing disorganized attachments by diminishing threatening behaviors. One of them is the Attachment and Biobehavioral Catch-up (ABC) program (Bernard et al., 2012; Dozier et al., 2008; Dozier et al., 2006; Lewis-Morrarty, Dozier, Bernard, Terracciano, & Moore, 2012), designed as an intervention for parents of young children in foster care and for parents identified by child protective services as being at high risk of maltreating their children. Children in the ABC intervention showed significantly lower rates of disorganized attachment and higher rates of secure attachment compared to the control children (Bernard et al., 2012). Indeed, there is increasing evidence that attachment outcomes can be improved in very high-risk families through the promotion of parental sensitivity. Targeting a group of maltreating families, a Canadian attachment-based home-visiting intervention with a behavioral focus enhanced parental sensitivity, improved children's attachment security, and reduced attachment disorganization (Moss et al., 2011; Tarabulsy et al., 2008). Inspired by the VIPP intervention, this intervention included elements identified as effective in the meta-analyses of attachment-based interventions (Bakermans-Kranenburg et al., 2003). The intervention was brief, including eight home visits. The main focus of the intervention was parental sensitivity. The intervention sessions with video feedback were structured in a fixed order, including videotaping interactive parent–child behavior and video feedback during which the intervener played back and discussed the video fragment (see also Juffer et al., 2008).

Additionally, a major randomized control study by Cicchetti et al. (Cicchetti, Rogosch, & Toth, 2006) has demonstrated the remarkable effectiveness of an attachment-based intervention for maltreated children and their biological parents. After going through 23 sessions of child–parent psychotherapy focusing on enhancing maternal sensitivity through maternal reinterpretation of past attachment experiences, a substantial reduction in infant disorganized attachment, and an increase in attachment security was observed for the intervention group. Interestingly, Cicchetti and his colleagues (2011) also searched for moderators of intervention effectiveness from the perspective of differential susceptibility theory. Differential susceptibility theory suggests that individuals differ in their susceptibility to the influence of the environment, for better and for worse, and that intervention effects might be substantially underestimated when we only look at overall effects across the whole sample (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). Genetic markers of differential susceptibility tested in the Cicchetti et al. (2011) intervention were dopamine- and serotonin-system related genes. However, they did not find significant genetic moderation of the treatment effect. One might speculate that genetic moderation was absent because the effects of the environment on disorganized attachment overrode the influence of any genetic component. It may be that differential susceptibility to the environment operates within certain environmental margins, beyond which they are overwhelmed (e.g., maltreating families, orphanages) or remediated as a result of major transitions to vastly improved environments (e.g., through intervention, foster care, or adoption).

The Bucharest Early Intervention Project (BEIP; Drury et al., 2012), however, seems to disconfirm this latter interpretation. In this unique randomized control trial 2-year-old children growing up in Romanian orphanages were pretested with the Disturbances of Attachment Interview for levels of disorganized and disturbed attachments and indiscriminate friendliness, and subsequently randomly assigned to either care as usual or high-quality foster care. The children were genotyped for the Brain Derived Neurotrophic Factor (BDNF) SNP and the serotonin transporter Variable Number Tandem Repeat (5HTTLPR). The posttest at 54 months showed that children with the short 5HTTLPR alleles or met66 BDNF genotype displayed lowest levels of indiscriminate friendliness at posttest and highest levels at pretest. Their peers without the susceptibility genotypes did not change their indiscriminate friendliness as a consequence of the foster care, a clear demonstration of differential susceptibility (Drury et al., 2012). This study illustrates the importance of considering risk modifiers or differential susceptibility markers in treatment outcome studies at the genetic, biological, and social level.

It is remarkable to note the similarities in the results of the meta-analyses addressing maternal sensitivity, attachment security, and attachment disorganization. They all provide evidence for the "less is more" adage: interventions with a focus on parenting sensitivity turn out to be most successful. A focused approach, centering an intervention on a small set of well-defined targets, is also clearly preferable from a methodological perspective as well (van IJzendoorn et al., 2005). Broadband interventions may be very effective on certain outcome measures but it may remain unclear which ingredients are responsible for

the effects. A piecemeal approach to constructing effective interventions, starting with testing the effectiveness of small building blocks or intervention modules that, after successful evaluations, can be combined into an even more effective overall program can produce more unambiguous insights than beginning with a complex multimodal intervention. Also, the modular approach might suit the differing needs of clients and services more efficiently, because it allows for a stepped-care approach in which a single intervention module is deployed to address the most common and least complex problems, and additional modules are added in more severe cases, or when earlier intervention efforts do not bear fruit.

Taking into account the enormous effort that goes into planning and conducting an intervention study, meta-analysis offers an indispensable tool for identifying generalizable, effective interventions and to facilitate evidence-based decision-making in the public health arena. It is therefore remarkable that the Triple-P program (Sanders, Markie-Dadds, & Turner, 2003) has been used on a whole-population basis as a public health intervention and enjoys adoption at a large scale across the world, with substantial cost implications, notwithstanding a lack of randomized controlled studies showing its effectiveness. A recent meta-analysis showed no between-group differences for studies with an adequate control group, and substantial selective reporting bias (Wilson et al., 2012). Interestingly, 32 of the 33 eligible studies were authored by Triple-P affiliated personnel (Wilson et al., 2012)—meta-analysis uncovers telling details.

Holding and Trauma Therapies

So-called *attachment therapy* and similar approaches such as *holding* or *trauma therapy* have received a great deal of public attention and enjoy considerable popularity on numerous internet sites for parents of troubled children. The American Professional Society on the Abuse of Children (APSAC) Task Force published an important report on the controversial *attachment therapies* provided to maltreated children and their biological, foster, or adoptive parents (Chaffin et al., 2006). In some cases such therapies have already proven to be harmful for children. In holding therapy, children are forced to make physical contact with their parent or other caregivers although they strongly resist these attempts. Physical force may even be used to reach the goal of proximity. The child would thus, according to this way of thinking, be enabled to regress to an earlier stage of bonding, and the reexperience of this crucial stage would stimulate the reconstruction of a secure attachment relationship. Holding therapy, however, has not been proven to be effective, and might even be dangerous (Chaffin et al., 2006; O'Connor & Zeanah, 2003; Sroufe, Erickson, & Friedrich, 2002). In fact, holding therapy is not implied at all by attachment theory. Therapists force the parent or caregiver to be extremely insensitive and to ignore clear signs from the child not wanting physical contact. At the core of sensitive parenting is the careful reading of children's attachment signals and needs, and to respond promptly and adequately to those signals (Ainsworth, Blehar, Waters, & Wall, 1978b). Holding therapy may well encourage the antithesis of sensitive care (Cyr et al., 2010).

Adult Attachment and Reflective Functioning in Interventions

As noted earlier, evidence suggests that it may be more difficult to change insecure adult attachment representations than it is to enhance infant attachment security or parental sensitivity (Korfmacher, Adam, Ogawa, & Egeland, 1997; van IJzendoorn, Juffer, & Duyvesteyn, 1995), particularly in clinical samples. Nevertheless, there is evidence that effective change can be achieved. Stovall-McClough and Cloitre (2003), working with women with PTSD following a history of childhood sexual abuse, with high rates of unresolved trauma, showed that unresolved AAI attachment status was most strongly associated with PTSD avoidant symptoms, and used imaginal exposure therapy to address the avoidant symptoms. The treatment appeared effective, not just for PTSD but also for unresolved loss. They observed a 62% reduction in unresolved attachment and absence of PTSD diagnosis for women in the exposure group. In a randomized trial with 90 Borderline Personality Disorder patients, Levy and colleagues (Levy, Beeney, & Temes, 2011) showed the effectiveness of one of the intervention modalities on changing attachment representations. Patients were assigned to one of three types of therapy, transference-focused psychotherapy (TFP), dialectical behavior therapy (DBT), or a modified psychodynamic supportive psychotherapy (SPT). The primary goal of TFP is to reduce symptomatology and self-destructive behavior through the modification of representations of self and others as they are enacted in the treatment. During the first year of treatment, TFP focuses on the identification of dominant relational patterns as they are experienced and expressed in the transference relationship. Based on the AAI the representation of attachment was classified and Reflective Functioning (RF) was coded. After 12 months of treatment, TFP participants showed a significant increase in secure classifications and

RF that was absent for the other two treatments. However, no changes in resolution of loss or trauma were observed across treatments. Toth and colleagues (Toth, Rogosch, & Cicchetti, 2008; Toth, Rogosch, Manly, & Cicchetti, 2006) administered the AAI before and after parent–child psychotherapy in a sample of mothers with major depressive disorder and their young children. The randomized controlled trial included a depressed control group and a nondepressed control group. After treatment, mothers in the depressed group showed a significant increase in RF in the AAI, as well as more positive relationships with their children.

The social-cognitive and affective concept of mentalization has become increasingly important to theory in clinical treatment in general, and psychoanalysis in particular. RF was first described by Fonagy, Steele, Steele, Moran, and Higgit (1991) as the ability to put oneself in another's shoes and to think about the thoughts, feelings, and intentions that may guide or have guided the other's behavior. The concept of RF arose out of the AAI state of mind scale metacognition, defined as monitoring and correcting one's own speech and thoughts during the interview (Main et al., 2003). RF expresses itself in response to AAI questions that demand reflection by asking "why" questions (Steele, Steele, & Murphy, 2009), such as: "Why do you think your parents behaved as they did during your childhood?" Fonagy and colleagues coined the term mentalization (Fonagy, Gergely, Jurist, & Target, 2002; Fonagy & Target, 1996, 2006) to describe the capacity to interpret or make sense of behavior in oneself and others in terms of intentional mental states such as thoughts, feelings, and beliefs. Thus, mentalization is the capacity to evoke and reflect on one's own experience to make inferences about behavior in oneself and others. RF has been operationalized to evaluate the quality of mentalization in the context of attachment relationships, and has been used as an outcome measure in several randomized controlled trials. It is related to such concepts as insight, intraception, and self-observing capacities of the ego, mindfulness, and mind-mindedness.

Although the concept of reflective functioning is broadly embraced (e.g., it has been argued that in the treatment of narcissism increasing self-reflective functioning is crucial to recovery, Bennett, 2006), it has also been noted that its validity and stability as an outcome measure are uncertain because adequate reports on its test-retest reliability and convergent and discriminant validity are absent (Choi-Kain & Gunderson, 2008). Moreover, it may be that individuals vary in their reflective functioning depending on who he or she is mentalizing about (Choi-Kain & Gunderson, 2008). Indeed, the RF scale has been used with a variety of interviews and that may in part be responsible for the relatively fuzzy boundaries of the concept and difficulties in the assessment. Nevertheless, the introduction of the concepts of mentalizing and reflective functioning was a catalyst for the development of a range of novel and potentially effective treatments, in particular for patients with bipolar disorder.

Attachment Fit Between Client and Therapist

The attachment representations of both patients and clinicians may play a role in the process and outcome of treatment. Dozier (1990) reported that individuals with a more dismissing stance to attachment were less likely to disclose symptomatology, more likely to minimize the interventions of case managers, and less likely to engage with treatment. In an intervention study involving mothers with an insecure attachment representation, dismissing mothers did not profit from discussion about attachment issues, and tended to view four home-based intervention sessions as requiring too much time investment from them (Bakermans-Kranenburg, Juffer, & van IJzendoorn, 1998). They were evaluated as less invested than other mothers in an intervention focusing on parenting sensitivity (Korfmacher et al., 1997). Adult attachment style has also been found to relate to adolescent's approach to treatment, with preoccupied adolescents in a residential treatment program showing the highest levels of truancy, rule breaking, and externalizing behavior problems (Zegers, Schuengel, van IJzendoorn, & Janssens, 2008).

Process research in psychoanalysis and psychoanalytic psychotherapy has indicated various factors that are related to positive treatment outcome. Patient–therapist fit is a factor that is generally indicated as predictive of treatment success (Fonagy et al., 1996; Kantrowitz, 1993; Stolk et al., 2008), but evidently there may be a wide variety of models of what is meant by *fit*. Both interpersonal theory and attachment theory suggest that a central task of the therapist is to resist the tendency to respond to the client's interpersonal or attachment orientation in a complementary manner, so as not to reinforce the client's rigid way of approaching relationships (Bernier & Dozier, 2002). Providing the client with a contrasting relational orientation appears to be beneficial in psychotherapy (Andrews, 1991), case management (Tyrrell et al., 1999), and academic counseling (Larose & Bernier, 2001), and indeed the corrective emotional experience may be a key

factor in therapeutic change. Bowlby (1988) suggested that the therapist should help the client recognize and change insecure working models or maladaptive relational patterns. He proposed that the therapist should challenge the client's beliefs about relationships by flexibly adopting a stance that is in contrast to the client's expectations. It is not far-fetched to argue that for a therapist with a secure representation of attachment it will be easier to respond in a contrasting (noncomplementary) way to the client's relational orientation than for an insecure therapist. In support of this idea Dozier, Cue, and Barnett (1994) have shown that clinicians with an autonomous AAI were more effective at providing noncomplementary responses than were nonautonomous clinicians. That is, autonomous clinicians worked in greater depth with clients presenting dismissing features than with preoccupied clients, whereas nonautonomous clinicians provided complementary responses, working in greater depth with preoccupied clients. This may explain why Gerber, Fonagy and their colleagues (Gerber, Fonagy, Bateman, & Higgitt, 2004), in a quasi-experimental trial of psychoanalysis and psychoanalytic psychotherapy, found that patients who became progressively more insecure on the AAI in the first phase of treatment were significantly more likely to improve in terms of their symptomatology by termination (Gerber et al., 2004): these patients may have been challenged to take a stance contrasting with their previous representation, temporarily leading to more insecurity or even less organization of attachment representation, which led to a better outcome in the end. If this line of reasoning is correct, then therapists' own tendencies to more dismissing or more preoccupied representations within the range of security may play a facilitating or impeding role in treatment, depending on the client's attachment representation. Consistent with this idea, clients and therapists showing dissimilar attachment tendencies on the dismissing–preoccupied dimension were shown to have better alliances and more productive relationships (Tyrrell et al., 1999). In such dyads, the natural style of the therapist appears to make him or her more likely to adopt an interpersonal stance that is contrary to what the client "pulls for," thereby disconfirming the client's rigid perceptions and strategies (Bernier & Dozier, 2002).

It should however be noted that the vast majority of the therapists in the Tyrrell et al. (1999) study were rated as autonomous based on a modified version of the Attachment Q Set (Kobak, 1989). Direct comparison with AAI classifications of the mentors in the adolescents' residential treatment institution of the Zegers et al. (2008) study is thus not possible, but the 55% of autonomous mentors in that study—although similar to the norm distribution—is considerably less than those in the Tyrrell study. At 3 months into treatment, no difference in therapeutic relationships between adolescents and secure versus insecure mentors could be traced, but between 3 and 10 months into treatment, the psychological availability of nonautonomous mentors decreased in the perception of the adolescents, whereas the perceived psychological availability of autonomous mentors increased. Similarly, reliance on autonomous mentors increased between 3 and 10 months, but reliance on nonautonomous mentors decreased over time (Zegers, Schuengel, van IJzendoorn, & Janssens, 2006).

The AAI can also be part of the intake and treatment process. As an interview it goes beyond available screening instruments by tapping into experiences in the family of origin, and it also opens up the perceived state of mind of the individual with respect to these experiences, including experiences of loss and other trauma. When multiple losses or abusive events are presented, it is informative which experiences are reported in a coherent way, and when disorganized speech, indicative of unresolved loss or trauma, takes over (Steele et al., 2009). Who should administer the AAI in the therapeutic context, and when should the interview take place? Steele and colleagues (2009) advise that the AAI be administered early in the treatment by the clinician himself or herself, unless the fragility of the patient would ask for postponement until a therapeutic alliance is in place. They argue that in other cases the AAI is likely to promote alliance. Over time, it is expected that content from the interview will filter through material covered in the course of the therapy (Steele et al., 2009).

In sum, secure adult attachment representations and associated mentalizing competence can be stimulated by therapeutic interventions although the evidence base still is disappointingly small. Psychotherapy is about creating more coherent autobiographical narratives (Bowlby, 1988), which implies a shift from preoccupied or dismissing representations of the past to more balanced, open and coherent discourse from a mentalizing stance. Some of the most promising attachment-related psychotherapy studies show that attachment representations of both client and therapist might help us understand why some client–therapist combinations are more effective than others. It is not the perfect match but a mild mismatch of counterbalancing representational biases that might be most productive in creating positive change.

CONCLUSIONS

Attachment theory and research has had a major impact on thinking regarding the developmental mechanisms contributing to adaptation and risk for psychological disorder. Its contribution to the field has occurred at a number of levels. First, it pioneered, in many respects, the developmental approach to understanding psychopathology, particularly its treatment of the connections between levels of analysis—the evolutionary, biological, cognitive/affective and relational—and their unfolding interactions and transformations across development. Second, the field has revealed a host of rich phenomena relevant to understanding psychopathology, and worthy of serious scientific and clinical investigation: protest, despair, detachment, secure base behavior, avoidance, resistance, disorganization, sensitivity of care, frightened/frightening parenting, coherence of mind, lack of resolution or mourning or trauma, to name some of the most prominent. Third, the field has established a strong evidence base supporting its major tenets, most notably: (1) the environmental origins of individual differences in attachment security and insecurity; (2) the important predictive, and likely causal, role of parental sensitivity; (3) the long-term developmental disadvantages associated with insecure attachment in the domains of internalizing problems, externalizing problems, and social competence; and (4) the intergenerational transmission of patterns of attachment. In the process of establishing this evidence base, attachment research has also highlighted the limits of the attachment construct, at least as currently measured and conceptualized, the role of important moderators in modifying or qualifying its effects, and the potentially complex developmental pathways by which attachment is connected to later outcomes. Finally, and perhaps most importantly, the field has developed a range of approaches to intervention and prevention that are delivering reliable positive outcomes for children and families.

REFERENCES

Abrams, K. Y., Rifkin, A., & Hesse, E. (2006). Examining the role of parental frightened/frightening subtypes in predicting disorganized attachment within a brief observational procedure. *Development and Psychopathology*, *18*(2), 345–361. doi: 10.1017/S0954579406060184

Achenbach, T. M. (1991). *Manual for Teacher's Report Form and 1991 Profile*. Burlington, VT: University of Vermont Department of Psychiatry.

Achenbach, T. M., Edelbrock, C., & Howell, C. T. (1987). Empirically based assessment of the behavioral/emotional problems of 2- and 3-year-old children. *Journal of Abnormal Child Psychology*, *15*(4), 629–650. doi: 10.1007/BF00917246

Adam, K. S., Sheldon-Keller, A. E., & West, M. (1996). Attachment organization and history of suicidal behavior in clinical adolescents. *Journal of Consulting and Clinical Psychology*, *64*(2), 264–272. doi: 10.1037/0022-006X.64.2.264

Ahnert, L., Gunnar, M. R., Lamb, M. E., & Barthel, M. (2004). Transition to child care: Associations with infant–mother attachment, infant negative emotion, and cortisol elevations. *Child Development*, *75*(3), 639–650. doi: 10.1111/j.1467-8624.2004.00698.x

Ainsworth, M. D. S. (1967). *Infancy in Uganda: Infant care and the growth of attachment*. Baltimore, MD: The Johns Hopkins Press.

Ainsworth, M. D. S. (1972). Attachment and dependency: A comparison. In J. L. Gewirtz, *Attachment and dependency* (pp. 92–137). Oxford, UK: V.H. Winston & Sons.

Ainsworth, M. D. S., & Bell, S. M. (1974). Mother-infant interaction and the development of competence. In C. Connolly & J. Bruner (Eds.), *The growth of competence* (pp. 97–118). New York: Academic Press.

Ainsworth, M. D. S., Bell, S. M., & Stayton, D. J. (1974). Infant mother attachment and social development: Socialization as a product of reciprocal responsiveness to signals. In M. P. M. Richards (Ed.), *The integration of the child into a social world* (pp. 99–135). Cambridge, U.K.: Cambridge University Press.

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation* (Vol. xviii). Hillsdale, NJ: Lawrence Erlbaum.

Ainsworth, M. D. S., & Wittig, B. A. (1969). Attachment and exploratory behavior of one-year-olds in a strange situation. In B.M. Foss (Ed.), *Determinants of infant behavior* (pp. 113–136). London, UK: Methuen.

Allen, J. P., McElhaney, K. B., Kuperminc, G. P., & Jodl, K. M. (2004). Stability and Change in Attachment Security Across Adolescence. *Child Development*, *75*(6), 1792–1805. doi: 10.1111/j.1467-8624.2004.00817.x

Ammaniti, M., Speranza, A. M., & Candelori, C. (1996). Stabilita dell'attaccamento infantile e transmissione intergenerazionale dell'attaccamento [Stability of infant attachement and intergenerational transmission of attachment]. *Psichiatria dell'Infanzia e dell'Adolescenza*, *63*, 313–332.

Andrews, J. D. W. (1991). *The active self in psychotherapy: An integration of therapeutic styles*. New York: Gardner.

Arend, R., Gove, F. L., & Sroufe, L. A. (1979). Continuity of individual adaptation from infancy to kindergarten: A predictive study of ego-resiliency and curiosity in preschoolers. *Child Development*, *50*(4), 950–959. doi: 10.2307/1129319

Arranz, E., Artamendi, J., Olabarrieta, F., & Martin, J. (2002). Family context and theory of mind development. *Early Child Development and Care*, *172*(1), 9–22. doi: 10.1080/03004430210880

Astington, J. W. (2003). Sometimes necessary, never sufficient: False-belief understanding and social competence *Individual differences in theory of mind: Implications for typical and atypical development* (pp. 13–38). New York, NY: Psychology Press.

Atkinson, L., Goldberg, S., Raval, V., Pederson, D., Benoit, D., Moran, G., ... Leung, E. (2005). On the relation between maternal state of mind and sensitivity in the prediction of infant attachment security. *Developmental Psychology*, *41*(1), 42–53. doi: 10.1037/0012-1649.41.1.42

Atzil, S., Hendler, T., & Feldman, R. (2011). Specifying the neurobiological basis of human attachment: Brain, hormones, and behavior in synchronous and intrusive mothers. *Neuropsychopharmacology*, *36*(13), 2603–2615. doi: 10.1038/npp.2011.172

Aviezer, O., Sagi, A., Resnick, G., & Gini, M. (2002). School competence in young adolescence: Links to early attachment relationships beyond concurrent self-perceived competence and representations of relationships. *International Journal of Behavioral Development*, *26*(5), 397–409. doi: 10.1080/01650250143000328

Avinun, R., & Knafo, A. (2013). Parenting as a reaction evoked by children's genotype: A meta-analysis of children-as-twins studies. *Personality and Social Psychology Review*, *12*, 1–16. doi: 10.1177/1088868313498308

Bakermans- Kranenburg, M. J., & van IJzendoorn, M. H. (2004). No association of the dopamine D4 receptor (DRD4) and -521 C/T promoter polymorphisms with infant attachment disorganization. *Attachment & Human Development*, *6*(3), 211–218. doi: 10.1080/14616730412331281584

Bakermans-Kranenburg, M. J., Juffer, F., & van IJzendoorn, M. H. (1998). Interventions with video feedback and attachment discussions: Does type of maternal insecurity make a difference? *Infant Mental Health Journal*, *19*(2), 202–219. doi: 10.1002/%28SICI%291097-0355%28199822%2919:2%3C202::AID-IMHJ8%3E3.0.CO;2-P

Bakermans-Kranenburg, M. J., Sonuga-Barke, E. J., Bos, K., Bunkers, K. M., Dobrova-Krol, N. A., Engle, P. L., . . . Grotevant, H. D. (2011). Children without permanent parents: Research, practice, and policy: Abstract. *Monographs of the Society for Research in Child Development*, *76*(4), 1–11. doi: 10.1111/j.1540-5834.2011.00624.x

Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Research review: Genetic vulnerability or differential susceptibility in child development: The case of attachment. *Journal of Child Psychology and Psychiatry*, *48*(12), 1160–1173. doi: 10.1111/j.1469-7610.2007.01801.x

Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2009). The first 10,000 adult attachment interviews: Distributions of adult attachment representations in clinical and non-clinical groups. *Attachment & Human Development*, *11*(3), 223–263. doi: 10.1080/14616730902814762

Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., & Juffer, F. (2003). Less is more: Meta-analyses of sensitivity and attachment interventions in early childhood. *Psychological Bulletin*, *129*(2), 195–215. doi: 10.1037/0033-2909.129.2.195

Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., & Juffer, F. (2005). Disorganized Infant Attachment and Preventive Interventions: A review and meta-analysis. *Infant Mental Health Journal*, *26*(3), 191–216. doi: 10.1002/imhj.20046

Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., & Juffer, F. (2008). Earlier is better: A meta-analysis of 70 years of intervention improving cognitive development in institutionalized children. *Monographs of the Society for Research in Child Development*, *73*(3), 279–293. doi: 10.1111/j.1540-5834.2008.00498.x

Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Pijlman, F. T., Mesman, J., & Juffer, F. (2008). Experimental evidence for differential susceptibility: Dopamine D4 receptor polymorphism (DRD4 VNTR) moderates intervention effects on toddlers' externalizing behavior in a randomized controlled trial. *Developmental Psychology*, *44*(1), 293–300. doi: 10.1037/0012-1649.44.1.293

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. (2007). Amygdala-frontal connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience*, *2*(4), 303–312. doi: 10.1093/scan/nsm029

Barone, L., & Lionetti, F. (2012). Attachment and social competence: A study using MCAST in low-risk Italian preschoolers. *Attachment & Human Development*, *14*(4), 391–403. doi: 10.1080/14616734.2012.691653

Barry, R. A., Kochanska, G., & Philibert, R. A. (2008). GxE interaction in the organization of attachment: Mothers' responsiveness as a moderator of children's genotypes. *Journal of Child Psychology and Psychiatry*, *49*(12), 1313–1320. doi: 10.1111/j.1469-7610.2008.01935.x

Bartholomew, K., & Horowitz, L. M. (1991). Attachment styles among young adults: A test of a four-category model. *Journal of Personality and Social Psychology*, *61*(2), 226–244. doi: 10.1037/0022-3514.61.2.226

Bates, J. E., Maslin, C. A., & Frankel, K. A. (1985). Attachment security, mother–child interaction, and temperament as predictors of behavior-problem ratings at age three years. *Monographs of the Society for Research in Child Development*, *50*(1-2), 167–193. doi: 10.2307/3333832

Beijersbergen, M. D., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., & Juffer, F. (2008). Stress regulation in adolescents: Physiological reactivity during the Adult Attachment Interview and conflict interaction. *Child Development*, *79*(6), 1707–1720. doi: 10.1111/j.1467-8624.2008.01220.x

Beijersbergen, M. D., Juffer, F., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2012). Remaining or becoming secure: Parental sensitive support predicts attachment continuity from infancy to adolescence in a longitudinal adoption study. *Developmental Psychology*, *48*(5), 1277–1282. doi: 10.1037/a0027442

Belsky, J. (1996). Parent, infant, and social-contextual antecedents of father–son attachment security. *Developmental Psychology*, *32*(5), 905–913. doi: 10.1037/0012-1649.32.5.905

Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science*, *16*(6), 300–304. doi: 10.1111/j.1467-8721.2007.00525.x

Belsky, J., Campbell, S. B., Cohn, J. F., & Moore, G. (1996). Instability of infant-parent attachment security. *Developmental Psychology*, *32*(5), 921–924. doi: 10.1037/0012-1649.32.5.921

Belsky, J., & Fearon, R. (2002). Infant-mother attachment security, contextual risk, and early development: A moderational analysis. *Development and Psychopathology*, *14*(2), 293–310. doi: 10.1017/S0954579402002067

Belsky, J., & Fearon, R. M. P. (2002). Early attachment security, subsequent maternal sensitivity, and later child development: does continuity in development depend upon continuity of caregiving? *Attachment & Human Development*, *4*(3), 361–387. doi: 10.1080/14616730210167267

Belsky, J., & Fearon, R. M. P. (2008). Precursors of attachment security *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 295–316). New York, NY: Guilford Press.

Belsky, J., Fearon, R. P., & Bell, B. (2007). Parenting, attention and externalizing problems: Testing mediation longitudinally, repeatedly and reciprocally. *Journal of Child Psychology and Psychiatry*, *48*(12), 1233–1242. doi: 10.1111/j.1469-7610.2007.01807.x

Bennett, C. (2006). Attachment theory and research applied to the conceptualization and treatment of pathological narcissism. *Clinical Social Work Journal*, *34*(1), 45–60. doi: 10.1007/s10615-005-0001-9

Berlin, L. J., & Cassidy, J. (2003). Mothers' self-reported control of their preschool children's emotional expressiveness: A longitudinal study of associations with infant-mother attachment and children's emotion regulation. *Social Development*, *12*(4), 477–495. doi: 10.1111/1467-9507.00244

Berlin, L. J., Cassidy, J., & Appleyard, K. (2008). The influence of early attachments on other relationships. In J. Cassidy & P.R. Shaver (Eds), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 333–347). New York, NY: Guilford Press

Bernard, K., & Dozier, M. (2010). Examining infants' cortisol responses to laboratory tasks among children varying in attachment disorganization: Stress reactivity or return to baseline? *Developmental Psychology*, *46*(6), 1771–1778. doi: 10.1037/a0020660

Bernard, K., Dozier, M., Bick, J., Lewis-Morrarty, E., Lindhiem, O., & Carlson, E. (2012). Enhancing attachment organization among maltreated children: Results of a randomized clinical trial. *Child Development*, *83*(2), 623–636.

Bernier, A., & Dozier, M. (2002). The client-counselor match and the corrective emotional experience: Evidence from interpersonal and attachment research. *Psychotherapy: Theory, Research, Practice, Training*, *39*(1), 32–43. doi: 10.1037/0033-3204.39.1.32

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Klauminser, G., & Charney, D. S. (1998). *Clinician-administered PTSD scale for DSM-IV*. Boston, MA: National Center for Posttraumatic Stress Disorder-Behavioral Science Division.

Blatz, W. E. (1966). *Human security: Some reflections*. Oxford, UK: Toronto Press.

Bohlin, G., Hagekull, B., & Rydell, A.-M. (2000). Attachment and social functioning: A longitudinal study from infancy to middle childhood. *Social Development*, *9*(1), 24–39. doi: 10.1111/1467-9507.00109

Bokhorst, C. L., Bakermans-Kranenburg, M. J., Fearon, R., van IJzendoorn, M. H., Fonagy, P., & Schuengel, C. (2003). The importance of shared environment in mother-infant attachment security: A behavioral genetic study. *Child Development*, *74*(6), 1769–1782. doi: 10.1046/j.1467-8624.2003.00637.x

Booth-LaForce, C., Groh, A. M., Burchinal, M. R., Roisman, G. I., Owen, M. T., & Cox, M. J. (2014). Caregiving and contextual sources of continuity and change in attachment security from infancy to late adolescence. In C. Booth-LaForce & G. I. Roisman (Eds.), The Adult Attachment Interview: Psychometrics, stability and change from infancy, and developmental origins. *Monographs of the Society for Research in Child Development*, *79*, 67–84. Doi: 10.1111/mono.12114

Borelli, J. L., Goshin, L., Joestl, S., Clark, J., & Byrne, M. W. (2010). Attachment organization in a sample of incarcerated mothers: Distribution of classifications and associations with substance abuse history, depressive symptoms, perceptions of parenting competency and social support. *Attachment & Human Development*, *12*(4), 355–374. doi: 10.1080/14616730903416971

Bos, P. A., Panksepp, J., Bluthe, R. M., & van Honk, J. (2012). Acute effects of steroid hormones and neuropeptides on human social-emotional behavior: a review of single administration studies. *Front Neuroendocrinol*, *33*(1), 17–35. doi: 10.1016/j.yfrne.2011.01.002

Bost, K. K., Shin, N., McBride, B. A., Brown, G. L., Vaughn, B. E., Coppola, G., … Korth, B. (2006). Maternal secure base scripts, children's attachment security, and mother–child narrative styles. *Attachment & Human Development*, *8*(3), 241–260. doi: 10.1080/14616730600856131

Bowlby, J. (1944). Fourty-four juvenile theives: Their characters and home life. *International Journal of Psycho-Analysis*, *25*, 19–52.

Bowlby, J. (1969). *Attachment and loss, vol. 1: Attachment*. London, UK: Hogarth Press and the Institute of Psycho-Analysis.

Bowlby, J. (1973). *Attachment and Loss, vol. 2: Separation: Anxiety and Anger*. London, UK: Hogarth Press and Institute of Psycho-Analysis.

Bowlby, J. (1980). *Attachment and Loss, vol. 3: Loss: Sadness and Depression*. London, UK: Hogarth Press and Institute of Psycho-Analysis.

Bowlby, J. (1988). *A secure base: Clinical applications of attachment theory*. London, UK: Routledge.

Bowlby, J., Robertson, J., & Rosenbluth, D. (1952). A two-year-old goes to the hospital. In R. S. Eisler, A. Freud, H. Hartmann, & E. Kris (Eds.), *Psychoanalytic study of the child*. New York, NY: International Universities Press.

Bretherton, I. (1990). Communication patterns, internal working models, and the intergenerational transmission of attachment relationships. *Infant Mental Health Journal*, *11*(3), 237–252. doi: 10.1002/1097-0355%28199023%2911:3%3C237::AID-IMHJ2280110306%3E3.0.CO;2-X

Bretherton, I. (1992). The origins of attachment theory: John Bowlby and Mary Ainsworth. *Developmental Psychology*, *28*(5), 759–775. doi: 10.1037/0012-1649.28.5.759

Bretherton, I. (1995). A communication perspective on attachment relationships and internal working models. *Monographs of the Society for Research in Child Development*, *60*(2-3), 310–329. doi: 10.2307/1166187

Bretherton, I. (2005). In pursuit of the internal working model construct and its relevance to attachment relationships. In J. Cassidy & P.R. Shaver (Eds), *Attachment from infancy to adulthood: The major longitudinal studies* (pp. 13–47). New York, NY: Guilford Publications

Bretherton, I., & Munholland, K. A. (2008). Internal working models in attachment relationships: Elaborating a central construct in attachment theory. In J. Cassidy & P.R. Shaver (Eds), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 102–127). New York, NY: Guilford Press

Bretherton, I., & Oppenheim, D. (2003). The MacArthur Story Stem Battery: Development, directions for administration, reliability, validity and reflections about meaning. In R. N. Emde, D. P. Wolf, & D. Oppenheim (Eds.), *Revealing the inner worlds of young children: The MacArthur Story Stem Battery and parent–child narratives* (pp. 55–80). New York, NY: Oxford University Press.

Bretherton, I., Ridgeway, D., & Cassidy, J. (1990). Assessing internal working models of the attachment relationship: An attachment story completion task for 3-year-olds *Attachment in the preschool years: Theory, research, and intervention* (pp. 273–308). Chicago, IL: University of Chicago Press.

Brown, G. L., Mangelsdorf, S. C., & Neff, C. (2012). Father involvement, paternal sensitivity, and father–child attachment security in the first 3 years. *Journal of Family Psychology*, *26*(3), 421–430. doi: 10.1037/a0027836

Brussoni, M. J., Jang, K. L., Livesley, W., & MacBeth, T. M. (2000). Genetic and environmental influences on adult attachment styles. *Personal Relationships*, *7*(3), 283–289. doi: 10.1111/j.1475-6811.2000.tb00017.x

Buchheim, A., Erk, S., George, C., Kachele, H., Ruchsow, M., Spitzer, M., … Walter, H. (2006). Measuring attachment representation in an fMRI environment: A pilot study. *Psychopathology*, *39*(3), 144–152. doi: 10.1159/000091800

Burgess, K. B., Marshall, P. J., Rubin, K. H., & Fox, N. A. (2003). Infant attachment and temperament as predictors of subsequent externalizing problems and cardiac physiology. *Journal of Child Psychology and Psychiatry*, *44*(6), 819–831. doi: 10.1111/1469-7610.00167

Cairns, R. B. (1972). Attachment and dependency: A psychobiological and social-learning synthesis. In J. L. Gewirtz (Ed.), *Attachment and dependency* (pp. 29–80). Oxford, UK: V.H. Winston & Sons.

Calkins, S. D., & Fox, N. A. (1992). The relations among infant temperament, security of attachment, and behavioral inhibition at twenty-four months. *Child Development*, *63*(6), 1456–1472. doi: 10.2307/1131568

Carlson, E. A. (1998). A prospective longitudinal study of attachment disorganization/disorientation. *Child Development*, *69*(4), 1107–1128. doi: 10.2307/1132365

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., … Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, *301*(5631), 386–389. doi: 10.1126/science.1083968

Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. *Monographs of the Society for Research in Child Development*, *59*(2–3), 228–283. doi: 10.2307/1166148

Cassidy, J., & Berlin, L. J. (1994). The insecure/ambivalent pattern of attachment: Theory and research. *Child Development*, *65*(4), 971–981. doi: 10.2307/1131298

Cassidy, J., Kirsh, S. J., Scolton, K. L., & Parke, R. D. (1996). Attachment and representations of peer relationships. *Developmental Psychology*, *32*(5), 892–904. doi: 10.1037/0012-1649.32.5.892

Cassidy, J., Marvin, R. S., & the MacArthur Working Group on Attachment. (1992). *Attachment organization in preschool children: Coding guidelines* (4th ed.). Unpublished manuscript, University of Virginia.

Cassidy, J., Woodhouse, S. S., Sherman, L. J., Stupica, B., & Lejuez, C. (2011). Enhancing infant attachment security: An examination of treatment efficacy and differential susceptibility. *Development and Psychopathology*, *23*(1), 131–148. doi: 10.1017/S0954579410000696

Chaffin, M., Hanson, R., Saunders, B. E., Nichols, T., Barnett, D., Zeanah, C., … Miller-Perrin, C. (2006). Report of the APSAC task force on attachment therapy, reactive attachment disorder, and attachment problems. *Child Maltreatment*, *11*(1), 76–89. doi: 10.1177/1077559505283699

Chaplin, T. M., & Cole, P. M. (2005). The role of emotion regulation in the development of psychopathology. In *Development of psychopathology: A vulnerability-stress perspective* (pp. 49–74). Thousand Oaks, CA: Sage Publications, Inc

Choi-Kain, L. W., & Gunderson, J. G. (2008). Mentalization: Ontogeny, assessment, and application in the treatment of borderline personality disorder. *American Journal of Psychiatry*, *165*(9), 1127–1135. doi: 10.1176/appi.ajp.2008.07081360

Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development*, *55*(1), 1–7. doi: 10.2307/1129830

Cicchetti, D., & Cohen, D. J. (1995). Perspectives on developmental psychopathology. In D. Cicchetti & D.J. Cohen (Eds), *Developmental psychopathology, vol. 1: Theory and methods* (pp. 3–20). Oxford, UK: John Wiley & Sons.

Cicchetti, D., Rogosch, F. A., & Toth, S. L. (1998). Maternal depressive disorder and contextual risk: Contributions to the development of attachment insecurity and behavior problems in toddlerhood. *Development and Psychopathology*, *10*(2), 283–300. doi: 10.1017/S0954579498001618

Cicchetti, D., Rogosch, F. A., & Toth, S. L. (2006). Fostering secure attachment in infants in maltreating families through preventive interventions. *Development and Psychopathology*, *18*(3), 623–649. doi: 10.1017/S0954579406060329

Cicchetti, D., Rogosch, F. A., & Toth, S. L. (2011). The effects of child maltreatment and polymorphisms of the serotonin transporter and dopamine D4 receptor genes on infant attachment and intervention efficacy. *Development and Psychopathology*, *23*(2), 357–372. doi: 10.1017/S0954579411000113

Coan, J. A. (2008). Toward a neuroscience of attachment. In J. Cassidy & P.R. Shaver, *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 241–265). New York, NY: Guilford Press.

Coates, B., Anderson, E. P., & Hartup, W. W. (1972). Interrelations in the attachment behavior of human infants. *Developmental Psychology*, *6*(2), 218–230. doi: 10.1037/h0032087

Colle, L., & Del Giudice, M. (2011). Patterns of attachment and emotional competence in middle childhood. *Social Development*, *20*(1), 51–72. doi: 10.1111/j.1467-9507.2010.00576.x

Collins, N. L., & Read, S. J. (1990). Adult attachment, working models, and relationship quality in dating couples. *Journal of Personality and Social Psychology*, *58*(4), 644–663. doi: 10.1037/0022-3514.58.4.644

Coppola, G., Vaughn, B. E., Cassibba, R., & Costantini, A. (2006). The attachment script representation procedure in an Italian sample: Associations with Adult Attachment Interview scales and with maternal sensitivity. *Attachment & Human Development*, *8*(3), 209–219. doi: 10.1080/14616730600856065

Crawford, T. N., Livesley, W., Jang, K. L., Shaver, P. R., Cohen, P., & Ganiban, J. (2007). Insecure attachment and personality disorder: A twin study of adults. *European Journal of Personality*, *21*(2), 191–208. doi: 10.1002/per.602

Crockenberg, S. B. (1981). Infant irritability, mother responsiveness, and social support influences on the security of infant–mother attachment. *Child Development*, *52*(3), 857–865. doi: 10.2307/1129087

Crowell, J. A., Treboux, D., & Brockmeyer, S. (2009). Parental divorce and adult children's attachment representations and marital status. *Attachment & Human Development*, *11*(1), 87–101. doi: 10.1080/14616730802500867

Crowell, J. A., Treboux, D., & Waters, E. (2002). Stability of attachment representations: The transition to marriage. *Developmental Psychology*, *38*(4), 467–479. doi: 10.1037/0012-1649.38.4.467

Cyr, C., Euser, E. M., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2010). Attachment security and disorganization in maltreating and high-risk families: A series of meta-analyses. *Development and Psychopathology*, *22*(1), 87–108. doi: 10.1017/S0954579409990289

D'Onofrio, B. M., Turkheimer, E., Emery, R. E., Maes, H. H., Silberg, J., & Eaves, L. J. (2007). A children of twins study of parental divorce and offspring psychopathology. *Journal of Child Psychology and Psychiatry*, *48*(7), 667–675. doi: 10.1111/j.1469-7610.2007.01741.x

Dallaire, D. H., & Weinraub, M. (2005). The stability of parenting behaviors over the first 6 years of life. *Early Childhood Research Quarterly*, *20*(2), 201–219. doi: 10.1016/j.ecresq.2005.04.008

Dannlowski, U., Ohrmann, P., Konrad, C., Domschke, K., Bauer, J., Kugel, H., … Suslow, T. (2009). Reduced amygdala-prefrontal coupling in major depression: Association with MAOA genotype and illness severity. *International Journal of Neuropsychopharmacology*, *12*(1), 11–22. doi: 10.1017/S1461145708008973

Davis, M., & Whalen, P. (2001). The amygdala: Vigilance and emotion. *Molecular Psychiatry*, *6*(1), 13–34. doi: 10.1038/sj.mp.4000812

Dawson, G., Ashman, S. B., Hessl, D., Spieker, S., Frey, K., Panagiotides, H., & Embry, L. (2001). Autonomic and brain electrical activity in securely- and insecurely-attached infants of depressed mothers. *Infant Behavior & Development*, *24*(2), 135–149. doi: Doi 10.1016/S0163-6383(01)00075-3

Dawson, G., Ashman, S. B., Panagiotides, H., Hessl, D., Self, J., Yamada, E., & Embry, L. (2003). Preschool outcomes of children of depressed mothers: Role of maternal behavior, contextual risk, and children's brain activity. *Child Development*, *74*(4), 1158–1175. doi: 10.1111/1467-8624.00599

De Rosnay, M., & Harris, P. L. (2002). Individual differences in children's understanding of emotion: The roles of attachment and language. *Attachment & Human Development*, *4*(1), 39–54. doi: 10.1080/14616730210123139

De Wolff, M., & van IJzendoorn, M. H. (1997). Sensitivity and attachment: A meta-analysis on parental antecedents of infant attachment. *Child Development*, *68*(4), 571–591. doi: 10.2307/1132107

DeKlyen, M., & Greenberg, M. T. (2008). Attachment and psychopathology in childhood. In J. Cassidy & P.R. Shaver, *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 637–665). New York, NY: Guilford Press.

Dodge, K. A., & Coie, J. D. (1987). Social-information-processing factors in reactive and proactive aggression in children's peer groups. *Journal of Personality and Social Psychology*, *53*(6), 1146–1158. doi: 10.1037/0022-3514.53.6.1146

Donnellan, M., Burt, S. A., Levendosky, A. A., & Klump, K. L. (2008). Genes, personality, and attachment in adults: A multivariate

behavioral genetic analysis. *Personality and Social Psychology Bulletin*, *34*(1), 3–16. doi: 10.1177/0146167207309199

Dozier, M. (1990). Attachment organization and treatment use for adults with serious psychopathological disorders. *Development and Psychopathology*, *2*(1), 47–60. doi: 10.1017/S0954579400000584

Dozier, M., Cue, K. L., & Barnett, L. (1994). Clinicians as caregivers: Role of attachment organization in treatment. *Journal of Consulting and Clinical Psychology*, *62*(4), 793–800. doi: 10.1037/0022-006X.62.4.793

Dozier, M., & Kobak, R. (1992). Psychophysiology in attachment interviews: Converging evidence for deactivating strategies. *Child Development*, *63*(6), 1473–1480. doi: 10.2307/1131569

Dozier, M., Peloso, E., Lewis, E., Laurenceau, J.-P., & Levine, S. (2008). Effects of an attachment-based intervention of the cortisol production of infants and toddlers in foster care. *Development and Psychopathology*, *20*(3), 845–859. doi: 10.1017/S0954579408000400

Dozier, M., Peloso, E., Lindhiem, O., Gordon, M., Manni, M., Sepulveda, S., … Levine, S. (2006). Developing evidence-based interventions for foster children: An example of a randomized clinical trial with infants and toddlers. *Journal of Social Issues*, *62*(4), 767–785. doi: 10.1111/j.1540-4560.2006.00486.x

Dozier, M., Stovall, K., Albus, K. E., & Bates, B. (2001). Attachment for infants in foster care: The role of caregiver state of mind. *Child Development*, *72*(5), 1467–1477. doi: 10.1111/1467-8624.00360

Drake, K., Belsky, J., & Fearon, R. (2013). From early attachment to engagement with learning in school: The role of self-regulation and persistence. *Developmental Psychology*, *50*, 1350–1361. doi: 10.1037/a0032779

Drury, S. S., Gleason, M. M., Theall, K. P., Smyke, A. T., Nelson, C. A., Fox, N. A., & Zeanah, C. H. (2012). Genetic sensitivity to the caregiving context: The influence of 5httlpr and BDNF val66met on indiscriminate social behavior. *Physiology & Behavior*, *106*(5), 728–735. doi: 10.1016/j.physbeh.2011.11.014

Dykas, M. J., & Cassidy, J. (2011). Attachment and the processing of social information across the life span: Theory and evidence. *Psychological Bulletin*, *137*(1), 19–46. doi: 10.1037/a0021367

Easterbrooks, M., Davidson, C. E., & Chazan, R. (1993). Psychosocial risk, attachment, and behavior problems among school-aged children. *Development and Psychopathology*, *5*(3), 389–402. doi: 10.1017/S095457940000448X

Ebstein, R. P., Israel, S., Chew, S. H., Zhong, S., & Knafo, A. (2010). Genetics of human social behavior. *Neuron*, *65*(6), 831–844. doi: 10.1016/j.neuron.2010.02.020

Egeland, B., & Farber, E. A. (1984). Infant–mother attachment: Factors related to its development and changes over time. *Child Development*, *55*(3), 753–771. doi: 10.2307/1130127

Ellis, B. J., Boyce, W., Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. *Development and Psychopathology*, *23*(1), 7–28. doi: 10.1017/S0954579410000611

Erickson, M. F., Sroufe, L. A., & Egeland, B. (1985). The relationship between quality of attachment and behavior problems in preschool in a high-risk sample. *Monographs of the Society for Research in Child Development*, *50*(1–2), 147–166. doi: 10.2307/3333831

Fagot, B. I. (1995). Classification of problem behaviors in young children: A comparison of four systems. *Journal of Applied Developmental Psychology*, *16*(1), 95–106. doi: 10.1016/0193-3973%2895%2990018-7

Fagot, B. I., & Kavanagh, K. (1990). The prediction of antisocial behavior from avoidant attachment classification. *Child Development*, *61*(3), 864–873. doi: 10.2307/1130970

Fearon, P., Shmueli-Goetz, Y., Viding, E., Fonagy, P., & Plomin, R. (2014). Genetic and environmental influences on adolescent attachment. *Journal of Child Psychology and Psychiatry*, *55*(9), 1033–1041. doi: 10.1111/jcpp.12171

Fearon, R., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Lapsley, A. M., & Roisman, G. I. (2010). The significance of insecure attachment and disorganization in the development of children s externalizing behavior: A meta-analytic study. *Child Development*, *81*(2), 435–456. doi: 10.1111/j.1467-8624.2009.01405.x

Fearon, R., & Belsky, J. (2004). Attachment and attention: Protection in relation to gender and cumulative social-contextual adversity. *Child Development*, *75*(6), 1677–1693. doi: 10.1111/j.1467-8624.2004.00809.x

Fearon, R., & Mansell, W. (2001). Cognitive perspectives on unresolved loss: Insights from the study of PTSD. *Bulletin of the Menninger Clinic*, *65*(3), 380–396. doi: 10.1521/bumc.65.3.380.19845

Fearon, R., van IJzendoorn, M. H., Fonagy, P., Bakermans-Kranenburg, M. J., Schuengel, C., & Bokhorst, C. L. (2006). In search of shared and nonshared environmental factors in security of attachment: A behavior-genetic study of the association between sensitivity and attachment security. *Developmental Psychology*, *42*(6), 1026–1040. doi: 10.1037/0012-1649.42.6.1026

Fearon, R. M. P., & Belsky, J. (2011). Infant–mother attachment and the growth of externalizing problems across the primary-school years. *Journal of Child Psychology and Psychiatry*, *52*(7), 782–791. doi: 10.1111/j.1469-7610.2010.02350.x

Feeney, J. A., Noller, P., & Hanrahan, M. (1994). Assessing adult attachment. In M.B. Sperling & W.H. Berman (Eds), *Attachment in adults: Clinical and developmental perspectives* (pp. 128–152). New York, NY: Guilford Press.

Fischer-Kern, M., Fonagy, P., Kapusta, N. D., Luyten, P., Boss, S., Naderer, A., … Leithner, K. (2013). Mentalizing in female inpatients with major depressive disorder. *Journal of Nervous and Mental Disease*, *201*(3), 201–207. doi: 10.1097/NMD.0b013e3182845c0a

Fonagy, P., Gergely, G., Jurist, E. L., & Target, M. (2002). *Affect regulation, mentalization, and the development of the self*. New York, NY: Other Press.

Fonagy, P., Leigh, T., Steele, M., Steele, H., Kennedy, R., Mattoon, G., … Gerber, A. (1996). The relation of attachment status, psychiatric classification, and response to psychotherapy. *Journal of Consulting and Clinical Psychology*, *64*(1), 22–31. doi: 10.1037/0022-006X.64.1.22

Fonagy, P., Redfern, S., & Charman, T. (1997). The relationship between belief-desire reasoning and a projective measure of attachment security (SAT). *British Journal of Developmental Psychology*, *15*(Pt 1), 51–61. doi: 10.1111/j.2044-835X.1997.tb00724.x

Fonagy, P., Steele, H., Steele, M., & Holder, J. (1997). Attachment and theory of mind: Overlapping constructs. *Association for Child Psychology and Psychiatry Occasional Papers*, *14*, 31–40.

Fonagy, P., Steele, M., Steele, H., Moran, G. S., & Higgitt, A. C. (1991). The capacity for understanding mental states: The reflective self in parent and child and its significance for security of attachment. *Infant Mental Health Journal*, *12*(3), 201–218. doi: 10.1002/1097-0355%28199123%2912:3%3C201::AID-IMHJ2280120307%3E3.0.CO;2-7

Fonagy, P., & Target, M. (1996). Playing with reality: I. Theory of mind and the normal development of psychic reality. *International Journal of Psychoanalysis*, *77*(2), 217–233.

Fonagy, P., & Target, M. (2006). The menatalization-focused approach to self pathology. *Journal of Personality Disorders*, *20*(6), 544–576. doi: 10.1521/pedi.2006.20.6.544

Fonagy, P., Target, M., Steele, H., & Steele, M. (1998). *Reflective-functioning manual, version 5.0, for application to adult attachment interviews*. London, UK: University College London.

Forbes, L., Evans, E., Moran, G., & Pederson, D. (2007). Change in atypical maternal behavior predicts change in attachment disorganization from 12 to 24 months in a high-risk sample. *Child Development*, *78*(3), 955–971. doi: 10.1111/j.1467-8624.2007.01043.x

Fortuna, K., Roisman, G. I., Haydon, K. C., Groh, A. M., & Holland, A. S. (2011). Attachment states of mind and the quality of young adults' sibling relationships. *Developmental Psychology*, *47*(5), 1366–1373. doi: 10.1037/a0024393

Fox, N. A., & Hane, A. A. (2008). Studying the biology of human attachment. In J. Cassidy & P.R. Shaver (Eds), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 217–240). New York, NY: Guilford Press.

Fraley, R. C. (2002). Attachment stability from infancy to adulthood: Meta-analysis and dynamic modeling of developmental mechanisms. *Personality and Social Psychology Review*, *6*(2), 123–151. doi: 10.1207/S15327957PSPR0602_03

Fraley, R. C., Brumbaugh, C. C., & Marks, M. J. (2005). The evolution and function of adult attachment: A comparative and phylogenetic analysis. *Journal of Personality* and Social Psychology, *89*(5), 731–746. doi: 10.1037/0022-3514.89.5.751

Fraley, R. C., & Roisman, G. I. (2014). Categories or dimensions? A taxometric analysis of the Adult Attachment Interview. In C. Booth-LaForce & G. I. Roisman (Eds.), The Adult Attachment Interview: Psychometrics, stability and change from infancy, and developmental origins. *Monographs of the Society for Research in Child Development*, *79*, 36–50. doi: 10.1111/mono.12111

Fraley, R. C., & Spieker, S. J. (2003). Are infant attachment patterns continuously or categorically distributed? A taxometric analysis of strange situation behavior. *Developmental Psychology*, *39*(3), 387–404. doi: 10.1037/0012-1649.39.3.387

Fraley, R. C., Waller, N. G., & Brennan, K. A. (2000). An item response theory analysis of self-report measures of adult attachment. *Journal of Personality and Social Psychology*, *78*(2), 350–365. doi: 10.1037/0022-3514.78.2.350

Franz, C. E., York, T. P., Eaves, L. J., Prom-Wormley, E., Jacobson, K. C., Lyons, M. J., ... Kremen, W. S. (2011). Adult romantic attachment, negative emotionality, and depressive symptoms in middle aged men: A multivariate genetic analysis. *Behavior Genetics*, *41*(4), 488–498. doi: 10.1007/s10519-010-9428-z

Freud, S. (1938). An outline of psychoanalysis. In J. Strachey (Ed.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 23, pp. 139–208). London, UK: Hogarth.

Frigerio, A., Ceppi, E., Rusconi, M., Giorda, R., Raggi, M. E., & Fearon, P. (2009). The role played by the interaction between genetic factors and attachment in the stress response in infancy. *Journal of Child Psychology and Psychiatry*, *50*(12), 1513–1522. doi: 10.1111/j.1469-7610.2009.02126.x

Frodi, A., Grolnick, W., & Bridges, L. (1985). Maternal correlates of stability and change in infant-mother attachment. *Infant Mental Health Journal*, *6*(2), 60–67. doi: 10.1002/1097-0355%28198522%296:2%3C60::AID-IMHJ2280060203%3E3.0.CO;2-Q

Fusar-Poli, P., Placentino, A., Carletti, F., Landi, P., Allen, P., Surguladze, S., ... Politi, P. (2009). Functional atlas of emotional faces processing: A voxel-based meta-analysis of 105 functional magnetic resonance imaging studies. *Journal of Psychiatry & Neuroscience*, *34*(6), 418–432.

Gaensbauer, T. J., Connell, J. P., & Schulz, L. A. (1983). Emotion and attachment: Interrelationships in a structured laboratory paradigm. *Developmental Psychology*, *19*(6), 815–831. doi: 10.1037/0012-1649.19.6.815

Galynker, I. I., Yaseen, Z. S., Katz, C., Zhang, X., Jennings-Donovan, G., Dashnaw, S., ... Winston, A. (2012). Distinct but overlapping neural networks subserve depression and insecure attachment. *Social Cognitive and Affective Neuroscience*, *7*(8), 896–908. doi: 10.1093/scan/nsr074

Gamer, M., Zurowski, B., & Buchel, C. (2010). Different amygdala subregions mediate valence related and attentional effects of oxytocin in humans. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, *107*(20), 9400–9405. doi: 10.1073/pnas.1000985107

George, C., Kaplan, N., & Main, M. (1985). *Adult Attachment Interview protocol*: Unpublished manuscript, University of California, Berkeley.

George, C., & Solomon, J. (1999). The development of caregiving: A comparison of attachment theory and psychoanalytic approaches to mothering. *Psychoanalytic Inquiry*, *19*(4), 618–646. doi: 10.1080/07351699909534268

Gerber, A., J, Fonagy, P., Bateman, A., & Higgitt, A. (2004). Structural and symptomatic change in psychoanalysis and psychodynamic psychotherapy of young adults: A quantitative study of process and outcome. *Journal of the American Psychoanalytic Association*, *52*(4), 1235–1236.

Gewirtz, J. L. (1972a). Attachment, dependence, and a distinction in terms of stimulus control. In J. L. Gerwitz (Ed.), *Attachment and dependency* (pp. 139–177.). Oxford, UK: V.H. Winston & Sons.

Gewirtz, J. L. (1972b). On the selection and use of attachment and dependence indices. In J. L. Gerwitz (Ed.), *Attachment and dependency* (pp. 179–215). Oxford, UK: V.H. Winston & Sons.

Gilissen, R., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., & van der Veer, R. (2008). Parent–child relationship, temperament, and physiological reactions to fear-inducing film clips: Further evidence for differential susceptibility. *Journal of Experimental Child Psychology*, *99*(3), 182–195. doi: 10.1016/j.jecp.2007.06.004

Gilliom, M., Shaw, D. S., Beck, J. E., Schonberg, M. A., & Lukon, J. L. (2002). Anger regulation in disadvantaged preschool boys: Strategies, antecedents, and the development of self-control. *Developmental Psychology*, *38*(2), 222–235. doi: 10.1037/0012-1649.38.2.222

Goldberg, S. (1997). Attachment and childhood behavior problems in normal, at-risk and clinical samples. In *Attachment and psychopathology* (pp. 171–195). New York, NY: Guilford Press.

Goldberg, S., Benoit, D., Blokland, K., & Madigan, S. (2003). Atypical maternal behavior, maternal representations, and infant disorganized attachment. *Development and Psychopathology*, *15*(2), 239–257. doi: 10.1017/S0954579403000130

Goldberg, S., Gotowiec, A., & Simmons, R. J. (1995). Infant-mother attachment and behavior problems in healthy and chronically ill preschoolers. *Development and Psychopathology*, *7*(2), 267–282. doi: 10.1017/S0954579400006490

Goldsmith, H., & Alansky, J. A. (1987). Maternal and infant temperamental predictors of attachment: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *55*(6), 805–816. doi: 10.1037/0022-006X.55.6.805

Goldwyn, R., Stanley, C., Smith, V., & Green, J. (2000). The Manchester Child Attachment Story Task: Relationship with parental AAI, SAT and child behaviour. *Attachment & Human Development*, *2*(1), 71–84. doi: 10.1080/146167300361327

Goossens, F. A., van IJzendoorn, M. H., Tavecchio, L., & Kroonenberg, P. M. (1986). Stability of attachment across time and context in a Dutch sample. *Psychological Reports*, *58*(1), 23–32. doi: 10.2466/pr0.1986.58.1.23

Greenberg, M. T., Cicchetti, D., & Cummings, E. M. (1990). Attachment in the preschool years: Theory, research, and intervention. *Attachment*

in the preschool years: Theory, research, and intervention. Chicago, IL: University of Chicago Press.

Greenberg, M. T., Speltz, M. L., & DeKlyen, M. (1993). The role of attachment in the early development of disruptive behavior problems. *Development and Psychopathology*, *5*(1–2), 191–213. doi: 10.1017/S095457940000434X

Greig, A., & Howe, D. (2001). Social understanding, attachment security of preschool children and maternal mental health. *British Journal of Developmental Psychology*, *19*(3), 381–393. doi: 10.1348/026151001166164

Griffin, D. W., & Bartholomew, K. (1994). Models of the self and other: Fundamental dimensions underlying measures of adult attachment. *Journal of Personality and Social Psychology*, *67*(3), 430–445. doi: 10.1037/0022-3514.67.3.430

Groh, A. M., Fearon, R. M. P., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Steele, R. D., & Roisman, G. I. (2014). The significance of attachment security for children's social competence with peers: A meta-analytic study. *Attachment & Human Development*, *16*, 103–136. doi: 10.1080/14616734.2014.883636

Groh, A. M., & Roisman, G. I. (2009). Adults' autonomic and subjective emotional responses to infant vocalizations: The role of secure base script knowledge. *Developmental Psychology*, *45*(3), 889–893. doi: 10.1037/a0014943

Groh, A. M., Roisman, G. I., Booth-LaForce, C., Fraley, R. C., Owen, M., Cox, M., & Burchinal, M. (2014). Stability of attachment security from infancy to late adolescence. In C. Booth-LaForce & G. I. Roisman (Eds.), The Adult Attachment Interview: Psychometrics, stability and change from infancy, and developmental origins. *Monographs of the Society for Research in Child Development*, *79*, 51–56. doi: 10.1111/mono.12113

Groh, A. M., Roisman, G. I., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., & Fearon, R. (2012). The significance of insecure and disorganized attachment for children's internalizing symptoms: A meta-analytic study. *Child Development*, *83*(2), 591–610.

Grossmann, K. E., Grossmann, K., & Waters, E. (2005). *Attachment from infancy to adulthood: The major longitudinal studies*. New York, NY: Guilford Publications.

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145–173. doi: 10.1146/annurev.psych.58.110405.085605

Gunnar, M. R. (2005). Attachment and stress in early development: Does attachment add to the potency of social regulators of infant stress? In *Attachment and bonding: A new synthesis* (pp. 245–255). Cambridge, MA: MIT Press.

Gunnar, M. R., Brodersen, L., Nachmias, M., Buss, K. A., & Rigatuso, J. (1996). Stress reactivity and attachment security. *Developmental Psychobiology*, *29*(3), 191–204. doi: 10.1002/%28SICI%291098-2302%28199604%2929:3%3C191::AID-DEV1%3E3.0.CO;2-M

Guttmann-Steinmetz, S., & Crowell, J. A. (2006). Attachment and externalizing disorders: A developmental psychopathology perspective. *Journal of the American Academy of Child & Adolescent Psychiatry*, *45*(4), 440–451. doi: 10.1097/01.chi.0000196422.42599.63

Haltigan, J. D., Roisman, G. I., & Fraley, R. C. (2013). The predictive significance of early caregiving experiences for symptoms of psychopathology through midadolescence: Enduring or transient effects? *Development and Psychopathology*, *25*(1), 209–221. doi: 10.1017/S0954579412000260

Haltigan, J. D., Roisman, G. I., & Haydon, K. C. (2014). The latent structure of the Adult Attachment Interview: Exploratory and confirmatory evidence. In C. Booth-LaForce & G. I. Roisman (Eds.), The Adult Attachment Interview: Psychometrics, stability and change from infancy, and developmental origins. *Monographs of the Society for Research in Child Development*, *79*, 15–35. doi: 10.1111/mono.12111

Hamilton, C. E. (2000). Continuity and discontinuity of attachment from infancy through adolescence. *Child Development*, *71*(3), 690–694. doi: 10.1111/1467-8624.00177

Harari, D., Bakermans-Kranenburg, M. J., de Kloet, C., Geuze, E., Vermetten, E., Westenberg, H., & van IJzendoorn, M. H. (2009). Attachment representations in Dutch veterans with and without deployment-related PTSD. *Attachment & Human Development*, *11*(6), 515–536. doi: 10.1080/14616730903282480

Harlow, H. F., & Zimmermann, R. R. (1959). Affectional responses in the infant monkey. *Science*, *130*, 421–432. doi: 10.1126/science.130.3373.421

Haydon, K. C., Roisman, G., Marks, M. J., & Fraley, R. C. (2011). An empirically derived approach to the latent structure of the Adult Attachment Interview: Additional convergent and discriminant validity evidence. *Attachment & Human Development*, *13*(5), 503–524. doi: 10.1080/14616734.2011.602253

Haydon, K. C., Roisman, G. I., Owen, M. T., Booth-LaForce, C., & Cox, M. (2014). Shared and distinctive antecedents of Adult Attachment Interview state-of-mind and inferred experience dimensions. In C. Booth-LaForce & G. I. Roisman (Eds.), The Adult Attachment Interview: Psychometrics, stability and change from infancy, and developmental origins. *Monographs of the Society for Research in Child Development*, *79*, 108–125. doi: 10.1111/mono.12116

Hesse, E. (1999). The adult attachment interview: Historical and current perspectives. In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 395–433). New York, NY: Guilford Press.

Hesse, E. (2008). The Adult Attachment Interview: Protocol, method of analysis, and empirical studies. In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 552–598). New York, NY: Guilford Press.

Hill-Soderlund, A. L., Mills-Koonce, W., Propper, C., Calkins, S. D., Granger, D. A., Moore, G. A., … Cox, M. J. (2008). Parasympathetic and sympathetic responses to the strange situation in infants and mothers from avoidant and securely attached dyads. *Developmental Psychobiology*, *50*(4), 361–376. doi: 10.1002/dev.20302

Hofer, M. A. (1994). Hidden regulators in attachment, separation, and loss. *Monographs of the Society for Research in Child Development*, *59*(2/3), 192–207. doi: 10.2307/1166146

Holland, A. S., & Roisman, G. I. (2010). Adult attachment security and young adults' dating relationships over time: Self-reported, observational, and physiological evidence. *Developmental Psychology*, *46*(2), 552–577. doi: 10.1037/a0018542

Howes, C., & Hamilton, C. E. (1992). Children's relationships with child care teachers: Stability and concordance with parental attachments. *Child Development*, *63*(4), 867–878. doi: 10.2307/1131239

Isabella, R. A. (1993). Origins of attachment: Maternal interactive behavior across the first year. *Child Development*, *64*(2), 605–621. doi: 10.2307/1131272

Izard, C. E. , Youngstrom, E. A., Fine, S. E., Mostow, A. J., & Trentacosta, C. J. (2006). Emotions and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental Psychology: Theory and method*, 2nd edition (Vol. 1, pp. 244–292). New York: John Wiley & Sons, Inc.

Jacobsen, T., Huss, M., Fendrich, M., Kruesi, M. J., & Ziegenhain, U. (1997). Children's ability to delay gratification: Longitudinal relations to mother–child attachment. *Journal of Genetic Psychology:*

Research and Theory on Human Development, *158*(4), 411–426. doi: 10.1080/00221329709596679

Jacobvitz, D., Leon, K., & Hazen, N. (2006). Does expectant mothers' unresolved trauma predict frightened/frightening maternal behavior? Risk and protective factors. *Development and Psychopathology*, *18*(2), 363–379. doi: 10.1017/S0954579406060196

Johnson, W. (2010). Understanding the genetics of intelligence: Can height help? Can corn oil? *Current Directions in Psychological Science*, *19*(3), 177–182. doi: 10.1177/0963721410370136

Juffer, F., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2008). Promoting positive parenting: An attachment-based intervention. *Promoting positive parenting: An attachment-based intervention*. New York, NY: Taylor & Francis Group/Lawrence Erlbaum Associates.

Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, *35*(1), 2–16. doi: 10.1016/j.neubiorev.2009.10.002

Kagan, J., Snidman, N., Arcus, D., & Reznick, J. S. (1994). *Galen's prophecy: Temperament in human nature*. New York, NY: Basic Books.

Kantrowitz, J., L. (1993). Outcome research in psychoanalysis: Review and reconsiderations. *Journal of the American Psychoanalytic Association*, *41*, 313–329.

Kaplan, N. (1987). *Individual differences in six-year-olds' thoughts about separation: Predicted from attachment to mother at one year of age*: Unpublished doctoral dissertation, University of California at Berkeley.

Kaplan, N., & Main, M. (1986). *A system for the analysis of children's family drawings in terms of attachment*: Unpublished manuscript, University of California at Berkeley.

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, *68*(5), 444–454. doi: 10.1001/archgenpsychiatry.2010.189

Kelly, K., Grienenberger, J., & Slade, A. (2005). Maternal reflective functioning, mother-infant affective communication, and infant attachment: Exploring the link between mental states and observed caregiving behavior in the intergenerational transmission of attachment. *Attachment & Human Development*, *7*(3), 299–311. doi: 10.1080/14616730500245963

Kendler, K. S. (2013). What psychiatric genetics has taught us about the nature of psychiatric illness and what is left to learn. *Molecular Psychiatry*, *18*(10), 1058–1066. doi: 10.1038/mp.2013.50

Kerns, K. A. (2008). Attachment in middle childhood. In J. Cassidy & P.R. Shaver (Eds), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 366–382). New York, NY: Guilford Press.

Kiang, L., & Furman, W. (2007). Representations of attachment to parents in adolescent sibling pairs: Concordant or discordant? *New Directions for Child and Adolescent Development*, *2007*(117), 73–89. doi: 10.1002/cd.195

Kidwell, S. L., Young, M. E., Hinkle, L. D., Ratliff, A. D., Marcum, M. E., & Martin, C. N. (2010). Emotional competence and behavior problems: Differences across preschool assessment of attachment classifications. *Clinical Child Psychology and Psychiatry*, *15*(3), 391–406. doi: 10.1177/1359104510367589

Kirsh, S. J., & Cassidy, J. (1997). Preschoolers' attention to and memory for attachment-relevant information. *Child Development*, *68*(6), 1143–1153. doi: 10.2307/1132297

Klagsbrun, M., & Bowlby, J. (1976). Responses to separation from parents: A clinical test for young children. *British Journal of Projective Psychology and Personality Study*, *21*, 7–26.

Kobak, R. (1989). *The Attachment Interview Q-Set*. Unpublished manuscript.

Kobak, R., Cassidy, J., Lyons-Ruth, K., & Zir, Y. (2005). Attachment, stress and psychopathology: A developmental pathways model. In D. Cicchetti & D. J. Cohen (Eds.), *Handbook of developmental psychopathology* (pp. 333–369). Cambridge, UK: Cambridge University Press.

Kochanska, G. (1997). Mutually responsive orientation between mothers and their young children: Implications for early socialization. *Child Development*, *68*(1), 94–112. doi: 10.2307/1131928

Kochanska, G., & Kim, S. (2013). Early attachment organization with both parents and future behavior problems: From infancy to middle childhood. *Child Development*, *84*(1), 283–296. doi: 10.1111/j.1467-8624.2012.01852.x

Koren-Karie, N., Oppenheim, D., Dolev, S., Sher, E., & Etzion-Carasso, A. (2002). Mothers' insightfulness regarding their infants' internal experience: Relations with maternal sensitivity and infant attachment. *Developmental Psychology*, *38*(4), 534–542. doi: 10.1037/0012-1649.38.4.534

Korfmacher, J., Adam, E., Ogawa, J., & Egeland, B. (1997). Adult attachment: Implications for the therapeutic process in a home visitation intervention. *Applied Developmental Science*, *1*(1), 43–52. doi: 10.1207/s1532480xads0101_5

Kringelbach, M. L. (2005). The human orbitofrontal cortex: Linking reward to hedonic experience. *Nature Reviews Neuroscience*, *6*(9), 691–702. doi: 10.1038/nrn1747

Kringelbach, M. L., Lehtonen, A., Squire, S., Harvey, A. G., Craske, M. G., Holliday, I. E., . . . Stein, A. (2008). A specific and rapid neural signature for parental instinct. *PLoS One*, *3*(2), e1664. doi: 10.1371/journal.pone.0001664

Kroonenberg, P. M., & van IJzendoorn, M. H. (1987). Exploring children's behavior in the Strange Situation. In L. W. C. Tavecchio & M. H. van Ijzendoorn (Eds.), *Attachment in social networks* (Vol. 44, pp. 379–425). Amsterdam: Elsevier.

Kumsta, R., Kreppner, J., Rutter, M., Beckett, C., Castle, J., Stevens, S., & Sonuga-Barke, E. J. (2010). Deprivation-specific psychological patterns. *Monographs of the Society for Research in Child Development*, *75*(1), 48–78. doi: 10.1111/j.1540-5834.2010.00550.x

Laible, D. J., & Thompson, R. A. (1998). Attachment and emotional understanding in preschool children. *Developmental Psychology*, *34*(5), 1038–1045. doi: 10.1037/0012-1649.34.5.1038

Lakatos, K., Nemoda, Z., Toth, I., Ronai, Z., Ney, K., Sasvari- Szekely, M., & Gervai, J. (2002). Further evidence for the role of the dopamine D4 receptor (DRD4) gene in attachment disorganization: Interaction of the exon III 48-bp repeat and the -521 C/T promoter polymorphisms. *Molecular Psychiatry*, *7*(1), 27–31. doi: 10.1038/sj/mp/4000986

Lakatos, K., Toth, I., Nemoda, Z., Ney, K., Sasvari-Szekely, M., & Gervai, J. (2000). Dopamine D4 receptor (DRD4) gene polymorphism is associated with attachment disorganization in infants. *Molecular Psychiatry*, *5*(633–637).

Lamb, M. E., Thompson, R. A., Gardner, W. P., Charnov, E. L., & Estes, D. (1984). Security of infantile attachment as assessed in the "strange situation": Its study and biological interpretation. *Behavioral and Brain Sciences*, *7*(1), 127–171. doi: 10.1017/S0140525X00026522

Laranjo, J., Bernier, A., & Meins, E. (2008). Associations between maternal mind-mindedness and infant attachment security: Investigating the mediating role of maternal sensitivity. *Infant Behavior & Development*, *31*(4), 688–695. doi: 10.1016/j.infbeh.2008.04.008

Larose, S., & Bernier, A. (2001). Social support processes: Mediators of attachment state of mind and adjustment in late adolescence. *Attachment & Human Development*, *3*(1), 96–120. doi: 10.1080/14616730010024762

Leerkes, E. M. (2011). Maternal sensitivity during distressing tasks: A unique predictor of attachment security. *Infant Behavior & Development*, *34*(3), 443–446. doi: 10.1016/j.infbeh.2011.04.006

Leerkes, E. M., & Wong, M. S. (2012). Infant distress and regulatory behaviors vary as a function of attachment security regardless of emotion context and maternal involvement. *Infancy*, *17*(5), 455–478. doi: 10.1111/j.1532-7078.2011.00099.x

Lenzi, D., Trentini, C., Pantano, P., Macaluso, E., Lenzi, G. L., & Ammaniti, M. (2012). Attachment models affect brain responses in areas related to emotions and empathy in nulliparous women. *Human Brain Mapping*, *34*(6), 1399–1414. doi: 10.1002/hbm.21520.

Levy, K. N., Beeney, J. E., & Temes, C. M. (2011). Attachment and its vicissitudes in borderline personality disorder. *Current Psychiatry Reports*, *13*(1), 50–59. doi: 10.1007/s11920-010-0169-8

Lewis, M., Feiring, C., McGuffog, C., & Jaskir, J. (1984). Predicting psychopathology in six-year-olds from early social relations. *Child Development*, *55*(1), 123–136. doi: 10.2307/1129839

Lewis, M., Feiring, C., & Rosenthal, S. (2000). Attachment over time. *Child Development*, *71*(3), 707–720. doi: 10.1111/1467-8624.00180

Lewis-Morrarty, E., Dozier, M., Bernard, K., Terracciano, S. M., & Moore, S. V. (2012). Cognitive flexibility and theory of mind outcomes among foster children: Preschool follow-up results of a randomized clinical trial. *Journal of Adolescent Health*, *51*(2, Suppl), S17–S22. doi: 10.1016/j.jadohealth.2012.05.005

Lindhiem, O., Bernard, K., & Dozier, M. (2011). Maternal sensitivity: Within-person variability and the utility of multiple assessments. *Child Maltreatment*, *16*(1), 41–50. doi: 10.1177/1077559510387662

Liotti, G. (2004). Trauma, dissociation, and disorganized attachment: Three strands of a single braid. *Psychotherapy: Theory, Research, Practice, Training*, *41*(4), 472–486. doi: 10.1037/0033-3204.41.4.472

Lorberbaum, J. P., Newman, J. D., Horwitz, A. R., Dubno, J. R., Lydiard, R., Hamner, M. B., … George, M. S. (2002). A potential role for thalamocingulate circuitry in human maternal behavior. *Biological Psychiatry*, *51*(6), 431–445. doi: 10.1016/S0006-3223%2801%2901284-7

Lucassen, N., Tharner, A., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Volling, B. L., Verhulst, F. C., … Tiemeier, H. (2011). The association between paternal sensitivity and infant–father attachment security: A meta-analysis of three decades of research. *Journal of Family Psychology*, *25*(6), 986–992. doi: 10.1037/a0025855

Luijk, M. P., Roisman, G. I., Haltigan, J. D., Tiemeier, H., Booth-LaForce, C., van IJzendoorn, M. H., … Hofman, A. (2011). Dopaminergic, serotonergic, and oxytonergic candidate genes associated with infant attachment security and disorganization? In search of main and interaction effects. *Journal of Child Psychology and Psychiatry*, *52*(12), 1295–1307.

Luijk, M. P., Saridjan, N., Tharner, A., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Jaddoe, V. W., … Tiemeier, H. (2010). Attachment, depression, and cortisol: Deviant patterns in insecure-resistant and disorganized infants. *Developmental Psychobiology*, *52*(5), 441–452.

Luijk, M. P., Tharner, A., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Jaddoe, V. W., Hofman, A., … Tiemeier, H. (2011). The association between parenting and attachment security is moderated by a polymorphism in the mineralocorticoid receptor gene: evidence for differential susceptibility. *Biological Psychology*, *88*(1), 37–40. doi: 10.1016/j.biopsycho.2011.06.005

Luijk, M. P., Velders, F. P., Tharner, A., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Jaddoe, V. W., … Tiemeier, H. (2010). FKBP5 and resistant attachment predict cortisol reactivity in infants: Gene–environment interaction. *Psychoneuroendocrinology*, *35*(10), 1454–1461.

Lundy, B. L. (2003). Father– and mother–infant face-to-face interactions: Differences in mind-related comments and infant attachment? *Infant Behavior & Development*, *26*(2), 200–212. doi: 10.1016/S0163-6383%2803%2900017-1

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the life span on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*(6), 434–445. doi: 10.1038/nrn2639

Lyons-Ruth, K., Bronfman, E., & Parsons, E. (1999). Maternal frightened, frightening, or atypical behavior and disorganized infant attachment patterns. *Monographs of the Society for Research in Child Development*, *64*(3), 67–96. doi: 10.1111/1540-5834.00034

Lyons-Ruth, K., Easterbrooks, M., & Cibelli, C. D. (1997). Infant attachment strategies, infant mental lag, and maternal depressive symptoms: Predictors of internalizing and externalizing problems at age 7. *Developmental Psychology*, *33*(4), 681–692. doi: 10.1037/0012-1649.33.4.681

MacBeth, A., Gumley, A., Schwannauer, M., & Fisher, R. (2011). Attachment states of mind, mentalization, and their correlates in a first-episode psychosis sample. *Psychology and Psychotherapy: Theory, Research and Practice*, *84*(1), 42–57.

Maccoby, E. E., & Feldman, S. (1972). Mother-attachment and stranger-reactions in the third year of life. *Monographs of the Society for Research in Child Development*, *37*(1), 86. doi: 10.2307/1165693

Madigan, S., Atkinson, L., Laurin, K., & Benoit, D. (2013). Attachment and internalizing behavior in early childhood: A meta-analysis. *Developmental Psychology*, *49*(4), 672–689. doi: 10.1037/a0028793

Madigan, S., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Moran, G., Pederson, D. R., & Benoit, D. (2006). Unresolved states of mind, anomalous parental behavior, and disorganized attachment: A review and meta-analysis of a transmission gap. *Attachment & Human Development*, *8*(2), 89–111. doi: 10.1080/14616730600774458

Madigan, S., Benoit, D., & Boucher, C. (2011). Exploration of the links among fathers' unresolved states of mind with respect to attachment, atypical paternal behavior, and disorganized infant–father attachment. *Infant Mental Health Journal*, *32*(3), 286–304. doi: 10.1002/imhj.20297

Madigan, S., Moran, G., & Pederson, D. R. (2006). Unresolved states of mind, disorganized attachment relationships, and disrupted interactions of adolescent mothers and their infants. *Developmental Psychology*, *42*(2), 293–304. doi: 10.1037/0012-1649.42.2.293

Main, M. (1991). Metacognitive knowledge, metacognitive monitoring, and singular (coherent) vs. multiple (incoherent) model of attachment: Findings and directions for future research. In C. Murray-Parkes, J. Stevenson-Hinde & P. Marris (Eds.), *Attachment across the life cycle* (pp. 127–159). New York, NY: Tavistock/Routledge.

Main, M. (1996). Introduction to the special section on attachment and psychopathology: 2. Overview of the field of attachment. *Journal of Consulting and Clinical Psychology*, *64*(2), 237–243. doi: 10.1037/0022-006X.64.2.237

Main, M. (2000). The Adult Attachment Interview as related to the Ainsworth Strange Situation. *Journal of the American Psychoanalytic Association*, *48*, 1055–1096.

Main, M. (2001). *Attachment to mother and father in infancy as related to the Adult Attachment Interview and to a self-visualization task at age 19*. Poster session presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

Main, M., & Cassidy, J. (1988). Categories of response to reunion with the parent at age 6: Predictable from infant attachment classifications and stable over a 1-month period. *Developmental Psychology*, *24*(3), 415–426. doi: 10.1037/0012-1649.24.3.415

Main, M., Goldwyn, R., & Hesse, E. (2003). *The Adult Attachment Interview: Scoring and Classification System, Version 7.2*. Unpublished manuscript, University of California, Berkeley.

Main, M., & Hesse, E. (1990). Parents' unresolved traumatic experiences are related to infant disorganized attachment status: Is frightened and/or frightening parental behavior the linking mechanism? In M.T. Greenberg, D. Ciccetti & E.M. Cummings (Eds.), *Attachment in the preschool years: Theory, research, and intervention* (pp. 161–182). Chicago, IL: University of Chicago Press.

Main, M., Hesse, E., & Kaplan, N. (2005). Predictability of Attachment Behavior and Representational Processes at 1, 6, and 19 Years of Age: The Berkeley Longitudinal Study. In K.E. Grossmann, K. Grossmann & E. Waters (Eds.), *Attachment from infancy to adulthood: The major longitudinal studies* (pp. 245–304). New York, NY: Guilford Publications.

Main, M., Kaplan, N., & Cassidy, J. (1985). Security in infancy, childhood, and adulthood: A move to the level of representation. *Monographs of the Society for Research in Child Development*, *50*(1–2), 66–104. doi: 10.2307/3333827

Main, M., & Solomon, J. (1986). Discovery of an insecure-disorganized/disoriented attachment pattern. In T.B. Brazelton, & M.W. Yogman *(Eds.), Affective development in infancy* (pp. 95–124). Westport, CT: Ablex Publishing.

Main, M., & Solomon, J. (1990). Procedures for identifying infants as disorganized/disoriented during the Ainsworth Strange Situation. In M.T. Greenberg , D. Cicchetti & E.M. Cummings (Eds.), *Attachment in the preschool years: Theory, research, and intervention* (pp. 121–160). Chicago, IL: University of Chicago Press.

Main, M., & Weston, D. R. (1981). The quality of the toddler's relationship to mother and to father: Related to conflict behavior and the readiness to establish new relationships. *Child Development*, *52*(3), 932–940. doi: 10.2307/1129097

Mangelsdorf, S., Gunnar, M., Kestenbaum, R., Lang, S., & Andreas, D. (1990). Infant proneness-to-distress temperament, maternal personality, and mother–infant attachment: Associations and goodness of fit. *Child Development*, *61*(3), 820–831. doi: 10.2307/1130966

Martins, E. C., Soares, I., Martins, C., Tereno, S., & Osorio, A. (2012). Can we identify emotion over-regulation in infancy? Associations with avoidant attachment, dyadic emotional interaction and temperament. *Infant and Child Development*, *21*(6), 579–595. doi: 10.1002/icd.1760

Marvin, R. S., & Britner, P. A. (2008). Normative development: The ontogeny of attachment. In In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 269–294). New York, NY: Guilford Press.

Masters, J. C., & Wellman, H. M. (1974). The study of human infant attachment: A procedural critique. *Psychological Bulletin*, *81*(4), 218–237. doi: 10.1037/h0036184

McCall, R., van IJzendoorn, M. H., Juffer, F., Groark, C., & Groza, V. (2012). Children without permanent parents: Research, practice, and policy. *Monographs of the Society for Research in Child Development*, *76*(4), 1–318.

McElwain, N. L., & Booth-LaForce, C. (2006). Maternal sensitivity to infant distress and nondistress as predictors of infant–mother attachment security. *Journal of Family Psychology*, *20*(2), 247–255. doi: 10.1037/0893-3200.20.2.247

McElwain, N. L., & Volling, B. L. (2004). Attachment security and parental sensitivity during infancy: Associations with friendship quality and false-belief understanding at age 4. *Journal of Social and Personal Relationships*, *21*(5), 639–667. doi: 10.1177/0265407504045892

Meins, E., Fernyhough, C., de Rosnay, M., Arnott, B., Leekam, S. R., & Turner, M. (2012). Mind-mindedness as a multidimensional construct: Appropriate and nonattuned mind-related comments independently predict infant–mother attachment in a socially diverse sample. *Infancy*, *17*(4), 393–415. doi: 10.1111/j.1532-7078.2011.00087.x

Meins, E., Fernyhough, C., Fradley, E., & Tuckey, M. (2001). Rethinking maternal sensitivity: Mothers' comments on infants' mental processes predict security of attachment at 12 months. *Journal of Child Psychology and Psychiatry*, *42*(5), 637–648. doi: 10.1111/1469-7610.00759

Meins, E., Fernyhough, C., Russell, J., & Clark-Carter, D. (1998). Security of attachment as a predictor of symbolic and mentalising abilities: A longitudinal study. *Social Development*, *7*(1), 1–24. doi: 10.1111/1467-9507.00047

Meins, E., Fernyhough, C., Wainwright, R., Gupta, M. D., Fradley, E., & Tuckey, M. (2002). Maternal mind-mindedness and attachment security as predictors of theory of mind understanding. *Child Development*, *73*(6), 1715–1726. doi: 10.1111/1467-8624.00501

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York, NY: Guilford Press.

Minde, K., & Hesse, E. (1996). The role of the adult attachment interview in parent-infant psychotherapy: A case presentation. *Infant Mental Health Journal*, *17*(2), 115–126. doi: 10.1002/%28SICI%291097-0355%28199622%2917:2%3C115::AID-IMHJ2%3E3.0.CO;2-P

Morris, J., Friston, K., Buchel, C., Frith, C., Young, A., Calder, A., & Dolan, R. (1998). A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain: A Journal of Neurology*, *121*(1), 47–57. doi: 10.1093/brain/121.1.47

Moss, E., Dubois-Comtois, K., Cyr, C., Tarabulsy, G. M., St-Laurent, D., & Bernier, A. (2011). Efficacy of a home-visiting intervention aimed at improving maternal sensitivity, child attachment, and behavioral outcomes for maltreated children: A randomized control trial. *Development and Psychopathology*, *23*(1), 195–210. doi: 10.1017/S0954579410000738

Murray, L., Arteche, A., Fearon, P., Halligan, S., Goodyer, I., & Cooper, P. (2011). Maternal postnatal depression and the development of depression in offspring up to 16 years of age. *Journal of the American Academy of Child & Adolescent Psychiatry*, *50*(5), 460–470. doi: 10.1016/j.jaac.2011.02.001

Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R. H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development*, *67*(2), 508–522. doi: 10.2307/1131829

Narusyte, J., Neiderhiser, J. M., D'Onofrio, B. M., Reiss, D., Spotts, E. L., Ganiban, J., & Lichtenstein, P. (2008). Testing different types of genotype-environment correlation: An extended children-of-twins model. *Developmental Psychology*, *44*(6), 1591–1603. doi: 10.1037/a0013911

Nelson, C.A., Fox, N.A. & Zeanah, C.H. (2014). *Romania's Abandoned Children: Deprivation, Brain Development, and the Struggle for Recovery*. Harvard University Press, Cambridge MA.

NICHD Early Child Care Research Network. (2001). Child-care and family predictors of preschool attachment and stability from infancy. *Developmental Psychology*, *37*(6), 847–862. doi: 10.1037/0012-1649.37.6.847

NICHD Early Child Care Research Network. (2006). Infant–mother attachment classification: Risk and protection in relation to changing maternal caregiving quality. *Developmental Psychology*, *42*(1), 38–58. doi: Doi 10.1037/0012-1649.42.1.38

Nye, E. C., Katzman, J., Bell, J. B., Kilpatrick, J., Brainard, M., & Haaland, K. Y. (2008). Attachment organization in Vietnam combat veterans with posttraumatic stress disorder. *Attachment & Human Development*, *10*(1), 41–57. doi: 10.1080/14616730701868613

O'Connor, T. G., Croft, C., & Steele, H. (2000). The contributions of behavioural genetic studies to attachment theory. *Attachment & Human Development*, *2*(1), 107–122. doi: 10.1080/146167300361345

O'Connor, T. G., & Croft, C. M. (2001). A twin study of attachment in preschool children. *Child Development*, *72*(5), 1501–1511. doi: 10.1111/1467-8624.00362

O'Connor, T. G., & Zeanah, C. H. (2003). Attachment disorders: Assessment strategies and treatment approaches. *Attachment & Human Development*, *5*(3), 223–244. doi: 10.1080/1461673031000159397 4

Oosterman, M., & Schuengel, C. (2007). Physiological effects of separation and reunion in relation to attachment and temperament in young children. *Developmental Psychobiology*, *49*(2), 119–128. doi: 10.1002/dev.20207

Oppenheim, D. (1997). The attachment doll-play interview for preschoolers. *International Journal of Behavioral Development*, *20*(4), 681–697. doi: 10.1080/016502597385126

Oppenheim, D. (2006). Child, parent, and parent–child emotion narratives: Implications for developmental psychopathology. *Development and Psychopathology*, *18*(3), 771–790. doi: 10.1017/S095457940606038X

Oppenheim, D., Koren-Karie, N., & Sagi, A. (2001). Mothers' empathic understanding of their preschoolers' internal experience: Relations with early attachment. *International Journal of Behavioral Development*, *25*(1), 16–26. doi: 10.1080/01650250042000096

Oppenheim, D., Koren-Karie, N., & Sagi-Schwartz, A. (2007). Emotion dialogues between mothers and children at 4.5 and 7.5 years: Relations with children's attachment at 1 year. *Child Development*, *78*(1), 38–52. doi: 10.1111/j.1467-8624.2007.00984.x

Ortiz, J., & Raine, A. (2004). Heart rate level and antisocial behavior in children and adolescents: A meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*(2), 154–162. doi: 10.1097/00004583-200402000-00010

Out, D., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2009). The role of disconnected and extremely insensitive parenting in the development of disorganized attachment: Validation of a new measure. *Attachment & Human Development*, *11*(5), 419–443. doi: 10.1080/14616730903132289

Owen, M. T., Easterbrooks, M., Chase-Lansdale, L., & Goldberg, W. A. (1984). The relation between maternal employment status and the stability of attachments to mother and to father. *Child Development*, *55*(5), 1894–1901. doi: 10.2307/1129936

Panfile, T. M., & Laible, D. J. (2012). Attachment security and child's empathy: The mediating role of emotion regulation. *Merrill-Palmer Quarterly*, *58*(1), 1–21. doi: 10.1353/mpq.2012.0003

Pederson, D. R., Moran, G., Sitko, C., Campbell, K., Ghesquire, K., & Acton, H. (1990). Maternal sensitivity and the security of infant–mother attachment: A Q-sort study. *Child Development*, *61*(6), 1974–1983. doi: 10.2307/1130851

Pianta, R. C., Egeland, B., & Adam, E. K. (1996). Adult attachment classification and self-reported psychiatric symptomatology as assessed by the Minnesota Multiphasic Personality Inventory—2. *Journal of Consulting and Clinical Psychology*, *64*(2), 273–281. doi: 10.1037/0022-006X.64.2.273

Picardi, A., Fagnani, C., Nistico, L., & Stazi, M. A. (2011). A twin study of attachment style in young adults. *Journal of Personality*, *79*(5), 965–991. doi: 10.1111/j.1467-6494.2010.00707.x

Pillay, S. S., Gruber, S. A., Rogowska, J., Simpson, N., & Yurgelun-Todd, D. A. (2006). fMRI of fearful facial affect recognition in panic disorder: The cingulate gyrus-amygdala connection. *Journal of Affective Disorders*, *94*(1–3), 173–181. doi: 10.1016/j.jad.2006.04.007

Pinquart, M., Feusner, C., & Ahnert, L. (2013). Meta-analytic evidence for stability in attachments from infancy to early adulthood. *Attachment & Human Development*, *15*(2), 189–218. doi: 10.1080/14616734.2013.746257

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2013). *Behavioral genetics* (6th ed.). New York, NY: Worth Publishers.

Ponizovsky, A. M., Nechamkin, Y., & Rosca, P. (2007). Attachment patterns are associated with symptomatology and course of schizophrenia in male inpatients. *American Journal of Orthopsychiatry*, *77*(2), 324–331. doi: 10.1037/0002-9432.77.2.324

Ponizovsky, A. M., Vitenberg, E., Baumgarten-Katz, I., & Grinshpoon, A. (2013). Attachment styles and affect regulation among outpatients with schizophrenia: Relationships to symptomatology and emotional distress. *Psychology and Psychotherapy: Theory, Research and Practice*, *86*(2), 164–182. doi: 10.1111/j.2044-8341.2011.02054.x

Poslawsky, I.E., Naber, F.B.A., Bakermans-Kranenburg, M.J., Daalen, E. van, Engeland, H. van & IJzendoorn, M.H. van (2014). Video-feedback intervention to promote Positive Parenting adapted to Autism (VIPP-AUTI): A randomized controlled trial. *Autism*, *1–16*. doi: 10.1177/1362361314537124

Raby, K., Cicchetti, D., Carlson, E. A., Cutuli, J., Englund, M. M., & Egeland, B. (2012). Genetic and caregiving-based contributions to infant attachment: Unique associations with distress reactivity and attachment security. *Psychological Science*, *23*(9), 1016–1023. doi: 10.1177/0956797612438265

Raikes, H., & Thompson, R. A. (2006). Family emotional climate, attachment security and young children's emotion knowledge in a high risk sample. *British Journal of Developmental Psychology*, *24*(1), 89–104. doi: 10.1348/026151005X70427

Raikes, H., & Thompson, R. A. (2008). Attachment security and parenting quality predict children's problem-solving, attributions, and loneliness with peers. *Attachment & Human Development*, *10*(3), 319–344. doi: 10.1080/14616730802113620

Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B., . . . Pitman, R. K. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry*, *47*(9), 769–776. doi: 10.1016/S0006-3223%2800%2900828-3

Repacholi, B., & Trapolini, T. (2004). Attachment and preschool children's understanding of maternal versus non-maternal psychological states. *British Journal of Developmental Psychology*, *22*(3), 395–415. doi: 10.1348/0261510041552693

Richters, J. E., Waters, E., & Vaughn, B. E. (1988). Empirical classification of infant-mother relationships from interactive behavior and crying during reunion. *Child Development*, *59*(2), 512–522. doi: 10.2307/1130329

Riem, M. M., Bakermans-Kranenburg, M. J., Pieper, S., Tops, M., Boksem, M. A., Vermeiren, R. R., . . . Rombouts, S. A. (2011). Oxytocin modulates amygdala, insula, and inferior frontal gyrus responses to infant crying: A randomized controlled trial. *Biological Psychiatry*, *70*(3), 291–297. doi: 10.1016/j.biopsych.2011.02.006

Riem, M. M., van IJzendoorn, M. H., Tops, M., Boksem, M. A., Rombouts, S. A., & Bakermans-Kranenburg, M. J. (2012). No laughing matter: Intranasal oxytocin administration changes functional brain connectivity during exposure to infant laughter. *Neuropsychopharmacology*, *37*(5), 1257–1266. doi: 10.1038/npp.2011.313

Riggs, S. A., Paulson, A., Tunnell, E., Sahl, G., Atkison, H., & Ross, C. A. (2007). Attachment, personality, and psychopathology among adult inpatients: Self-reported romantic attachment style versus Adult Attachment Interview states of mind. *Development and Psychopathology*, *19*(1), 263–291. doi: 10.1017/S0954579407070149

Roisman, G. I. (2007). The psychophysiology of adult attachment relationships: Autonomic reactivity in marital and premarital interactions. *Developmental Psychology*, *43*(1), 39–53. doi: 10.1037/0012-1649.43.1.39

Roisman, G. I., & Fraley, R. C. (2008). A behavior-genetic study of parenting quality, infant attachment security, and their covariation in a nationally representative sample. *Developmental Psychology*, *44*(3), 831–839. doi: 10.1037/0012-1649.44.3.831

Roisman, G. I., Fraley, R. C., & Belsky, J. (2007). A taxometric study of the Adult Attachment Interview. *Developmental Psychology*, *43*(3), 675–686. doi: 10.1037/0012-1649.43.3.675

Roisman, G. I., & Haydon, K. C. (2011). Earned-security in retrospect: Emerging insights from longitudinal, experimental, and taxometric investigations. In D. Cicchetti & G.I. Roisman (Eds), *Minnesota symposium on child psychology (Volume 36): The origins and organization of adaptation and maladaptation* (pp. 109–154). Hoboken, NJ: John Wiley & Sons Inc.

Roisman, G. I., Holland, A., Fortuna, K., Fraley, R. C., Clausell, E., & Clarke, A. (2007). The Adult Attachment Interview and self-reports of attachment style: An empirical rapprochement. *Journal of Personality and Social Psychology*, *92*(4), 678–697. doi: 10.1037/0022-3514.92.4.678

Roisman, G. I., Tsai, J. L., & Chiang, K. H. (2004). The emotional integration of childhood experience: Physiological, facial expressive, and self-reported emotional response during the adult attachment interview. *Developmental Psychology*, *40*(5), 776–789. doi: 10.1037/0012-1649.40.5.776

Rosenthal, M. K. (1973). Attachment and mother–infant interaction: Some research impasse and a suggested change in orientation. *Child Psychology & Psychiatry & Allied Disciplines*, *14*(3), 201–207. doi: 10.1111/j.1469-7610.1973.tb01187.x

Rothbaum, F., Rosen, K. S., Pott, M., & Beatty, M. (1995). Early parent–child relationships and later problem behavior: A longitudinal study. *Merrill-Palmer Quarterly*, *41*(2), 133–151.

Rutter, M. (1979). Protective factors in children's responses to stress and disadvantage. In M. W. Kent & J. E. Rolf (Eds.), *Primary prevention in psychopathology: Social competence in children* (pp. 49–74). Hanover, NH: University Press of New England.

Rutter, M., Kreppner, J., & Sonuga-Barke, E. (2009). Emanuel Miller lecture: Attachment insecurity, disinhibited attachment, and attachment disorders: Where do research findings leave the concepts? *Journal of Child Psychology and Psychiatry*, *50*(5), 529–543. doi: 10.1111/j.1469-7610.2009.02042.x

Rutter, M., & Sroufe, L. A. (2000). Developmental psychopathology: Concepts and challenges. *Development and Psychopathology*, *12*(3), 265–296. doi: 10.1017/S0954579400003023

Sabatini, M. J., Ebert, P., Lewis, D. A., Levitt, P., Cameron, J. L., & Mirnics, K. (2007). Amygdala gene expression correlates of social behavior in monkeys experiencing maternal separation. *Journal of Neuroscience*, *27*(12), 3295–3304. doi: 10.1523/JNEUROSCI.4765-06.2007

Sanders, M. R., Markie-Dadds, C., & Turner, K. M. (2003). *Theoretical, scientific and clinical foundations of the Triple P-Positive Parenting Program: A population approach to the promotion of parenting competence* (Vol. 1). Queensland: Parenting and Family Support Centre, the University of Queensland.

Schank, R. C., & Abelson, R. P. (1988). Scripts, plans, goals, and understanding. In A. Collins & E.E. Smith (Eds.), *Readings in cognitive science: A perspective from psychology and artificial intelligence* (pp. 190–223). San Mateo, CA: Morgan Kaufmann.

Schieche, M., & Spangler, G. (2005). Individual differences in biobehavioral organization during problem-solving in toddlers: The influence of maternal behavior, infant-mother attachment, and behavioral inhibition on the attachment-exploration balance. *Developmental Psychobiology*, *46*(4), 293–306. doi: 10.1002/dev.20065

Schneider, B. H., Atkinson, L., & Tardif, C. (2001). Child–parent attachment and children's peer relations: A quantitative review. *Developmental Psychology*, *37*(1), 86–100. doi: 10.1037/0012-1649.37.1.86

Schoenmaker, C., Juffer, F., van IJzendoorn, M. H., Linting, M., Van der Voort, A., & Bakermans-Kranenburg, M.J. (2015). From maternal sensitivity in infancy to adult attachment representations: A longitudinal adoption study with secure base scripts. *Attachment & Human Development*, doi: 10.1080/14616734.2015.1037315

Schore, A. N. (2001). Effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal*, *22*(1–2), 7–66. doi: 10.1002/1097-0355%28200101/04%2922:1%3C7::AID-IMHJ2%3E3.0.CO;2-N

Schuengel, C., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (1999). Frightening maternal behavior linking unresolved loss and disorganized infant attachment. *Journal of Consulting and Clinical Psychology*, *67*(1), 54–63. doi: 10.1037/0022-006X.67.1.54

Scott, S., & O'Connor, T. G. (2012). An experimental test of differential susceptibility to parenting among emotionally-dysregulated children in a randomized controlled trial for oppositional behavior. *Journal of Child Psychology and Psychiatry*, *53*(11), 1184–1193. doi: 10.1111/j.1469-7610.2012.02586.x

Seifritz, E., Esposito, F., Neuhoff, J. G., Luthi, A., Mustovic, H., Dammann, G., . . . Di Salle, F. (2003). Differential sex-independent amygdala response to infant crying and laughing in parents versus nonparents. *Biological Psychiatry*, *54*(12), 1367–1375. doi: 10.1016/S0006-3223%2803%2900697-8

Shaver, P. R., & Mikulincer, M. (2002). Attachment-related psychodynamics. *Attachment & Human Development*, *4*(2), 133–161. doi: 10.1080/14616730210154171

Shaw, D. S., Keenan, K., Vondra, J. I., Delliquadri, E., & Giovannelli, J. (1997). Antecedents of preschool children's internalizing problems: A longitudinal study of low-income families. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(12), 1760–1767. doi: 10.1097/00004583-199712000-00025

Shaw, D. S., & Vondra, J. I. (1995). Infant attachment security and maternal predictors of early behavior problems: A longitudinal study of low-income families. *Journal of Abnormal Child Psychology*, *23*(3), 335–357. doi: 10.1007/BF01447561

Sherman, L. J., Stupica, B., Dykas, M. J., Ramos-Marcuse, F., & Cassidy, J. (2013). The development of negative reactivity in irritable newborns as a function of attachment. *Infant Behavior & Development*, *36*(1), 139–146. doi: 10.1016/j.infbeh.2012.11.004

Shmueli-Goetz, Y., Target, M., Fonagy, P., & Datta, A. (2008). The Child Attachment Interview: A psychometric study of reliability and discriminant validity. *Developmental Psychology*, *44*(4), 939–956. doi: 10.1037/0012-1649.44.4.939

Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin*, *86*(2), 420–428. doi: 10.1037/0033-2909.86.2.420

Simpson, J. A., & Belsky, J. (2008). Attachment theory within a modern evolutionary framework. In In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 131–157). New York, NY: Guilford Press.

Slade, A. (2005). Parental reflective functioning: An introduction. *Attachment & Human Development*, *7*(3), 269–281. doi: 10.1080/14616730500245906

Slade, A., Grienenberger, J., Bernbach, E., Levy, D., & Locker, A. (2005). Maternal reflective functioning, attachment, and the transmission gap: A preliminary study. *Attachment & Human Development*, *7*(3), 283–298. doi: 10.1080/14616730500245880

Slough, N. M., & Greenberg, M. T. (1990). Five-year-olds' representations of separation from parents: Responses from the perspective of

self and other. *New Directions for Child Development*, *48*, 67–84. doi: 10.1002/cd.23219904806

Smith, P. B., & Pederson, D. R. (1988). Maternal sensitivity and patterns of infant-mother attachment. *Child Development*, *59*(4), 1097–1101. doi: 10.2307/1130276

Solomon, J., & George, C. (2008). The measurement of attachment security and related constructs in infancy and early childhood. In In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 383–416). New York, NY: Guilford Press.

Spangler, G., & Grossmann, K. (1993). Biobehavioral organization in securely and insecurely attached infants. *Child Development*, *64*(5), 1439–1450. doi: 10.2307/1131544

Spangler, G., Johann, M., Ronai, Z., & Zimmermann, P. (2009). Genetic and environmental influence on attachment disorganization. *Journal of Child Psychology and Psychiatry*, *50*(8), 952–961. doi: 10.1111/j.1469-7610.2008.02054.x

Spangler, G., & Schieche, M. (1998). Emotional and adrenocortical responses of infants to the strange situation: The differential function of emotional expression. *International Journal of Behavioral Development*, *22*(4), 681–706. doi: 10.1080/016502598384126

Sroufe, L. A. (1979). The coherence of individual development: Early care, attachment, and subsequent developmental issues. *American Psychologist*, *34*(10), 834–841. doi: 10.1037/0003-066X.34.10.834

Sroufe, L. A. (1988). The role of infant–caregiver attachment in development. In J. Belsky & T. Nezworski (Eds.), *Clinical implications of attachment* (pp. 18–38). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Sroufe, L. A. (1996). *Emotional development: The organization of emotional life in the early years*. New York, NY: Cambridge University Press.

Sroufe, L. A. (2003). Attachment categories as reflections of multiple dimensions: Comment on Fraley and Spieker (2003). *Developmental Psychology*, *39*(3), 413–416. doi: 10.1037/0012-1649.39.3.413

Sroufe, L. A., Egeland, B., Carlson, E., & Collins, W. A. (2005a). Placing early attachment experiences in developmental context: The Minnesota Longitudinal Study. *Attachment from infancy to adulthood: The major longitudinal studies* (pp. 48–70). New York, NY: Guilford Press.

Sroufe, L. A., Egeland, B., Carlson, E. A., & Collins, W. A. (2005b). *The development of the person: The Minnesota study of risk and adaptation from birth to adulthood*. New York, NY: Guilford Press.

Sroufe, L. A., Egeland, B., & Kreutzer, T. (1990). The fate of early experience following developmental change: Longitudinal approaches to individual adaptation in childhood. *Child Development*, *61*(5), 1363–1373. doi: 10.2307/1130748

Sroufe, L. A., Erickson, M. F., & Friedrich, W. N. (2002). Attachment theory and "attachment therapy." *APSAC Advisor* (*14*), 4–6.

Sroufe, L. A., & Waters, E. (1977). Attachment as an organizational construct. *Child Development*, *48*(4), 1184–1199. doi: 10.2307/1128475

Stalker, C. A., & Davies, F. (1998). Working models of attachment and representations of the object in a clinical sample of sexually abused women. *Bulletin of the Menninger Clinic*, *62*(3), 334–335.

Steele, H., & Steele, M. (2005). Understanding and resolving emotional conflict: The London Parent–child Project. In , K.E. Grossmann, K. Grossmann & E. Waters (Eds.), *Attachment from infancy to adulthood: The major longitudinal studies* (pp. 137–164). New York, NY: Guilford Press.

Steele, H., Steele, M., & Croft, C. (2008). Early attachment predicts emotion recognition at 6 and 11 years old. *Attachment & Human Development*, *10*(4), 379–393. doi: 10.1080/14616730802461409

Steele, H., Steele, M., Croft, C., & Fonagy, P. (1999). Infant-mother attachment at one year predicts children's understanding of mixed emotions at six years. *Social Development*, *8*(2), 161–178. doi: 10.1111/1467-9507.00089

Steele, H., Steele, M., & Murphy, A. (2009). Use of the adult attachment interview to measure process and change in psychotherapy. *Psychotherapy Research*, *19*(6), 633–643. doi: 10.1080/10503300802609698

Stein, A., Woolley, H., Senior, R., Hertzmann, L., Lovel, M., Lee, J., . . . Fairburn, C. G. (2006). Treating disturbances in the relationship between mothers with bulimic eating disorders and their infants: A randomized, controlled trial of video feedback. *American Journal of Psychiatry*, *163*(5), 899–906. doi: 10.1176/appi.ajp.163.5.899

Stein, J. L., Wiedholz, L. M., Bassett, D. S., Weinberger, D. R., Zink, C. F., Mattay, V. S., & Meyer-Lindenberg, A. (2007). A validated network of effective amygdala connectivity. *Neuroimage*, *36*(3), 736–745. doi: 10.1016/j.neuroimage.2007.03.022

Stevenson-Hinde, J., & Marshall, P. J. (1999). Behavioral inhibition, heart period, and respiratory sinus arrhythmia: An attachment perspective. *Child Development*, *70*(4), 805–816. doi: 10.1111/1467-8624.00058

Stolk, M. N., Mesman, J., van Zeijl, J., Alink, L. R., Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., . . . Koot, H. M. (2008). Early parenting intervention aimed at maternal sensitivity and discipline: A process evaluation. *Journal of Community Psychology*, *36*(6), 780–797. doi: 10.1002/jcop.20280

Stovall-McClough, K., & Cloitre, M. (2006). Unresolved attachment, PTSD, and dissociation in women with childhood abuse histories. *Journal of Consulting and Clinical Psychology*, *74*(2), 219–228. doi: 10.1037/0022-006X.74.2.219

Stovall-McClough, K. C., & Cloitre, M. (2003). Reorganization of unresolved childhood traumatic memories following exposure therapy. *Annals of the New York Academy of Sciences*, *1008*, 297–299.

Strathearn, L., Fonagy, P., Amico, J., & Montague, P. (2009). Adult attachment predicts maternal brain and oxytocin response to infant cues. *Neuropsychopharmacology*, *34*(13), 2655–2666. doi: 10.1038/npp.2009.103

Suess, G., Grossmann, K., & Sroufe, L. A. (1992). Effects of infant attachment to mother and father on quality of adaptation in preschool: From dyadic to individual organisation of self. *International Journal of Behavioral Development*, *15*(1), 43–65.

Suomi, S. J. (2003). Gene–environment interactions and the neurobiology of social conflict. In *Roots of mental illness in children* (pp. 132–139). New York, NY: New York Academy of Sciences.

Swain, J. E. (2011). The human parental brain: In vivo neuroimaging. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *35*(5), 1242–1254. doi: 10.1016/j.pnpbp.2010.10.017

Swain, J. E., & Ho, S. (2010). Baby smile response circuits of the parental brain. *Behavioral and Brain Sciences*, *33*(6), 460–461. doi: 10.1017/S0140525X10001615

Symons, D. K., & Clark, S. E. (2000). A longitudinal study of mother–child relationships and theory of mind in the preschool period. *Social Development*, *9*(1), 3–23. doi: 10.1111/1467-9507.00108

Tarabulsy, G. M., Pascuzzo, K., Moss, E., St-Laurent, D., Bernier, A., Cyr, C., & Dubois-Comtois, K. (2008). Attachment-based intervention for maltreating families. *American Journal of Orthopsychiatry*, *78*(3), 322–332. doi: 10.1037/a0014070

Teti, D. M., & Ablard, K. E. (1989). Security of attachment and infant-sibling relationships: A laboratory study. *Child Development*, *60*(6), 1519–1528. doi: 10.2307/1130940

Thompson, R. A., Lamb, M. E., & Estes, D. (1982). Stability of infant–mother attachment and its relationship to changing life

circumstances in an unselected middle-class sample. *Child Development*, *53*(1), 144–148. doi: 10.2307/1129646

Tomlinson, M., Cooper, P., & Murray, L. (2005). The Mother-infant relationship and infant attachment in a South African peri-urban settlement. *Child Development*, *76*(5), 1044–1054. doi: 10.1111/j.1467-8624.2005.00896.x

Toth, S. L., Rogosch, F. A., & Cicchetti, D. (2008). Attachment theory-informed intervention and reflection functioning in depressed mothers. In H. Steele & M. Steele (Eds.), *Clinical applications of the Adult Attachment Interview* (pp. 154–172). New York, NY: Guilford Press.

Toth, S. L., Rogosch, F. A., Manly, J. T., & Cicchetti, D. (2006). The efficacy of toddler–parent psychotherapy to reorganize attachment in the young offspring of mothers with major depressive disorder: A randomized preventive trial. *Journal of Consulting and Clinical Psychology*, *74*(6), 1006–1016. doi: 10.1037/0022-006X.74.6.1006

Tottenham, N., Hare, T., Millner, A., Gilhooly, T., Zevin, J., & Casey, B. (2011). Elevated amygdala response to faces following early deprivation. *Developmental Science*, *14*(2), 190–204.

Trouton, A., Spinath, F. M., & Plomin, R. (2002). Twins Early Development Study (TEDS): A multivariate, longitudinal genetic investigation of language, cognition and behavior problems in childhood. *Twin Research*, *5*(5), 444–448. doi: 10.1375/136905202320906255

True, M. M., Pisani, L., & Oumar, F. (2001). Infant–mother attachment among the Dogon of Mali. *Child Development*, *72*(5), 1451–1466. doi: 10.1111/1467-8624.00359

Tyrrell, C., & Dozier, M. (1997). *The role of attachment in the therapeutic process and outcome for adults with serious psychiatric disorders*. Paper presented at the biennial meeting of the Society for Research in Child Development, Washington, DC.

Tyrrell, C. L., Dozier, M., Teague, G. B., & Fallot, R. D. (1999). Effective treatment relationships for persons with serious psychiatric disorders: The importance of attachment states of mind. *Journal of Consulting and Clinical Psychology*, *67*(5), 725–733. doi: 10.1037/0022-006X.67.5.725

Uher, R., & McGuffin, P. (2010). The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. *Molecular Psychiatry*, *15*(1), 18–22. doi: 10.1038/mp.2009.123

Urban, J., Carlson, E., Egeland, B., & Sroufe, L. A. (1991). Patterns of individual adaptation across childhood. *Development and Psychopathology*, *3*(4), 445–460. doi: 10.1017/S0954579400007628

van Bakel, H. J., & Riksen-Walraven, J. (2004). Stress reactivity in 15-month-old infants: Links with infant temperament, cognitive competence, and attachment security. *Developmental Psychobiology*, *44*(3), 157–167. doi: 10.1002/dev.20001

van den Boom, D. (1990). Preventive intervention and the quality of mother–infant interaction and infant exploration in irritable infants. In W. Koops (Ed.), *Developmental psychology behind the dikes* (pp. 249–270). Amsterdam: Eburon.

van den Boom, D. C. (1994). The influence of temperament and mothering on attachment and exploration: An experimental manipulation of sensitive responsiveness among lower-class mothers with irritable infants. *Child Development*, *65*(5), 1457–1477.

van der Horst, F. C., & van der Veer, R. (2010). The ontogeny of an idea: John Bowlby and contemporaries on mother–child separation. *History of Psychology*, *13*(1), 25–45. doi: 10.1037/a0017660

Van Dijken, S. (1998). *John Bowlby, his early life: A biographical journey into the roots of attachment theory*. London: Free Association Books.

van IJzendoorn, M. H. (1995). Adult attachment representations, parental responsiveness, and infant attachment: A meta-analysis on the predictive validity of the Adult Attachment Interview. *Psychological Bulletin*, *117*(3), 387–403. doi: 10.1037/0033-2909.117.3.387

van IJzendoorn, M. H. (1997). Attachment, emergent morality, and aggression: Toward a developmental socioemotional model of antisocial behaviour. *International Journal of Behavioral Development*, *21*(4), 703–727. doi: 10.1080/016502597384631

van IJzendoorn, M. H., & Bakermans-Kranenburg, M. J. (2006). DRD47-repeat polymorphism moderates the association between maternal unresolved loss or trauma and infant disorganization. *Attachment & Human Development*, *8*(4), 291–307. doi: 10.1080/14616730601048159

van IJzendoorn, M. H., & Bakermans-Kranenburg, M. J. (2012). Integrating temperament and attachment: The differential susceptibility paradigm. In M. Zentner & R.L. Shiner (Eds.), *Handbook of temperament* (pp. 403–424). New York, NY: Guilford Press.

van IJzendoorn, M.H., & Bakermans-Kranenburg, M.J., (2014). Confined quest for continuity: The categorical versus continuous nature of attachment. *Monographs of the Society for Research in Child Development. 79*(3):157–167. doi: 10.1111/mono.12120.

van IJzendoorn, M. H., Feldbrugge, J., Derks, F., de Ruiter, C., Verhagen, M., Philipse, M., ... Riksen-Walraven, J. (1997). Attachment representations of personality-disordered criminal offenders. *American Journal of Orthopsychiatry*, *67*(3), 449–459. doi: 10.1037/h0080246

van IJzendoorn, M. H., Juffer, F., & Duyvesteyn, M. G. (1995). Breaking the intergenerational cycle of insecure attachment: A review of the effects of attachment-based interventions on maternal sensitivity and infant security. *Child Psychology & Psychiatry & Allied Disciplines*, *36*(2), 225–248. doi: 10.1111/j.1469-7610.1995.tb01822.x

van IJzendoorn, M. H., Moran, G., Belsky, J., Pederson, D., Bakermans-Kranenburg, M. J., & Kneppers, K. (2000). The similarity of siblings' attachments to their mother. *Child Development*, *71*(4), 1086–1098. doi: 10.1111/1467-8624.00211

van IJzendoorn, M. H., Palacios, J., Sonuga-Barke, E. J., Gunnar, M. R., Vorria, P., McCall, R. B., ... Juffer, F. (2011). Children without permanent parents: Research, practice, and policy: I. Children in institutional care: Delayed development and resilience. *Monographs of the Society for Research in Child Development*, *76*(4), 8–30. doi: 10.1111/j.1540-5834.2011.00626.x

van IJzendoorn, M.H., & Sagi-Schwartz, A. (2008). Cross-Cultural patterns of attachment: Universal and contextual dimensions. In J. Cassidy, & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed.) (pp. 880–905). New York: Guilford Press.

van IJzendoorn, M.H., Sagi, A., & Lambermon, M.W.E. (1992). The multiple caregiver paradox. Some Dutch and Israeli data. In R.C. Pianta (Ed.), Beyond the parent: The role of other adults in children's lives. *New Directions for Child Development*, *57*, 5–25.

van IJzendoorn, M. H., Schuengel, C., & Bakermans-Kranenburg, M. J. (1999). Disorganized attachment in early childhood: Meta-analysis of precursors, concomitants, and sequelae. *Development and Psychopathology*, *11*(2), 225–249. doi: 10.1017/S0954579499002035

van IJzendoorn, M. H., Vereijken, C. M., Bakermans-Kranenburg, M. J., & Riksen-Walraven, J. (2004). Assessing attachment security with the Attachment Q Sort: Meta-analytic evidence for the validity of the Observer AQS. *Child Development*, *75*(4), 1188–1213. doi: 10.1111/j.1467-8624.2004.00733.x

Van Ryzin, M. J., Carlson, E. A., & Sroufe, L. A. (2011). Attachment discontinuity in a high-risk sample. *Attachment & Human Development*, *13*(4), 381–401. doi: 10.1080/14616734.2011.584403

Van Zeijl, J., Mesman, J., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Juffer, F., Stolk, M. N., ... Alink, L. R.

(2006). Attachment-based intervention for enhancing sensitive discipline in mothers of 1- to 3-year-old children at risk for externalizing behavior problems: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *74*(6), 994–1005. doi: 10.1037/0022-006X.74.6.994

Vaughn, B. E., Bost, K. K., & van IJzendoorn, M. H. (2008). Attachment and temperament: Additive and interactive influences on behavior, affect, and cognition during infancy and childhood. In In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 192–216). New York, NY: Guilford Press.

Vaughn, B. E., Egeland, B. R., Sroufe, L. A., & Waters, E. (1979). Individual differences in infant-mother attachment at twelve and eighteen months: Stability and change in families under stress. *Child Development*, *50*(4), 971–975. doi: 10.2307/1129321

Verhage, M. L., Schuengel, C., J., B.-K. M., Oosterman, M., Madigan, S., Fearon, R. M. P., … van IJzendoorn, M. H. (2013). *Intergenerational transmission of attachment: Preliminary meta-analytic results*. Poster presented at the ISED Research Days, Amsterdam. The Netherlands.

Verissimo, M., & Salvaterra, F. (2006). Maternal secure-base scripts and children's attachment security in an adopted sample. *Attachment & Human Development*, *8*(3), 261–273. doi: 10.1080/14616730600856149

Verschueren, K., Dossche, D., Marcoen, A., Mahieu, S., & Bakermans-Kranenburg, M. (2006). Attachment representations and discipline in mothers of young school children: An observation study. *Social Development*, *15*(4), 659–675. doi: 10.1111/j.1467-9507.2006.00363.x

Vuga, M., Fox, N. A., Cohn, J. F., Kovacs, M., & George, C. J. (2008). Long-term stability of electroencephalographic asymmetry and power in 3 to 9 year-old children. *International Journal of Psychophysiology*, *67*(1), 70–77. doi: 10.1016/j.ijpsycho.2007.10.007

Ward, M. J., Vaughn, B. E., & Robb, M. D. (1988). Social-emotional adaptation and infant-mother attachment in siblings: Role of the mother in cross-sibling consistency. *Child Development*, *59*(3), 643–651. doi: 10.2307/1130564

Wartner, U. G., Grossmann, K., Fremmer-Bombik, E., & Suess, G. (1994). Attachment patterns at age six in south Germany: Predictability from infancy and implications for preschool behavior. *Child Development*, *65*(4), 1014–1027. doi: 10.2307/1131301

Waters, E. (1978). The reliability and stability of individual differences in infant–mother attachment. *Child Development*, *49*(2), 483–494. doi: 10.2307/1128714

Waters, E., & Deane, K. E. (1985). Defining and assessing individual differences in attachment relationships: Q-methodology and the organization of behavior in infancy and early childhood. *Monographs of the Society for Research in Child Development*, *50*(1-2), 41–65. doi: 10.2307/3333826

Waters, E., Merrick, S., Treboux, D., Crowell, J., & Albersheim, L. (2000). Attachment security in infancy and early adulthood: A twenty-year longitudinal study. *Child Development*, *71*(3), 684–689. doi: 10.1111/1467-8624.00176

Waters, E., Weinfield, N. S., & Hamilton, C. E. (2000). The stability of attachment security from infancy to adolescence and early adulthood: General discussion. *Child Development*, *71*(3), 703–706. doi: 10.1111/1467-8624.00179

Waters, H. S., & Rodrigues-Doolabh, L. (2004). *Manual for decoding secure base narratives*: Unpublished manuscript, State University of New York at Stony Brook.

Waters, H. S., & Waters, E. (2006). The attachment working models concept: Among other things, we build script-like representations of secure base experiences. *Attachment & Human Development*, *8*(3), 185–197. doi: 10.1080/14616730600856016

Weaver, I. C., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., … Meaney, M. J. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience*, *7*(8), 847–854. doi: 10.1038/nn1276

Webster-Stratton, C., Reid, M., & Hammond, M. (2004). Treating children with early-onset conduct problems: Intervention outcomes for parent, child, and teacher training. *Journal of Clinical Child and Adolescent Psychology*, *33*(1), 105–124. doi: 10.1207/S15374424JCCP3301_11

Webster-Stratton, C., Rinaldi, J., & Reid, J. M. (2011). Long-term outcomes of Incredible Years parenting program: Predictors of adolescent adjustment. *Child and Adolescent Mental Health*, *16*(1), 38–46. doi: 10.1111/j.1475-3588.2010.00576.x

Weinfield, N. S., Sroufe, L. A., & Egeland, B. (2000). Attachment from infancy to early adulthood in a high-risk sample: Continuity, discontinuity, and their correlates. *Child Development*, *71*(3), 695–702. doi: 10.1111/1467-8624.00178

Weinfield, N. S., Sroufe, L. A., Egeland, B., & Carlson, E. (2008). Individual differences in infant–caregiver attachment: Conceptual and empirical aspects of security. In J. Cassidy & P.R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 78–101). New York, NY: Guilford Press.

Weinfield, N. S., Whaley, G. J., & Egeland, B. (2004). Continuity, discontinuity, and coherence in attachment from infancy to late adolescence: Sequelae of organization and disorganization. *Attachment & Human Development*, *6*(1), 73–97. doi: 10.1080/14616730310001659566

Weinraub, M., Brooks, J., & Lewis, M. (1977). The social network: A reconsideration of the concept of attachment. *Human Development*, *20*(1), 31–47. doi: 10.1159/000271546

Whipple, N., Bernier, A., & Mageau, G. A. (2011). A dimensional approach to maternal attachment state of mind: Relations to maternal sensitivity and maternal autonomy support. *Developmental Psychology*, *47*(2), 396–403. doi: 10.1037/a0021310

Wilson, P., Rush, R., Hussey, S., Puckering, C., Sim, F., Allely, C. S., … Gillberg, C. (2012). How evidence-based is an "evidence-based parenting program"? A PRISMA systematic review and meta-analysis of Triple P. *BMC Medicine*, *10*, 130. doi: 10.1186/1741-7015-10-130

Yang, T. T., Simmons, A. N., Matthews, S. C., Tapert, S. F., Frank, G. K., Max, J. E., … Paulus, M. P. (2010). Adolescents with major depression demonstrate increased amygdala activation. *Journal of the American Academy of Child & Adolescent Psychiatry*, *49*(1), 42–51. doi: 10.1097/00004583-201001000-00008

Zegers, M. A., Schuengel, C., van IJzendoorn, M. H., & Janssens, J. M. (2006). Attachment representations of institutionalized adolescents and their professional caregivers: Predicting the development of therapeutic relationships. *American Journal of Orthopsychiatry*, *76*(3), 325–334. doi: 10.1037/0002-9432.76.3.325

Zegers, M. A., Schuengel, C., van IJzendoorn, M. H., & Janssens, J. M. (2008). Attachment and problem behavior of adolescents during residential treatment. *Attachment & Human Development*, *10*(1), 91–103. doi: 10.1080/14616730701868621

Zelenko, M., Kraemer, H., Huffman, L., Gschwendt, M., Pageler, N., & Steiner, H. (2005). Heart rate correlates of attachment status in young mothers and their infants. *Journal of the American Academy of Child & Adolescent Psychiatry*, *44*(5), 470–476. doi: 10.1097/01.chi.0000157325.10232.b1

Zimmermann, P., Fremmer-Bombik, E., Spangler, G., & Grossmann, K. E. (1997). Attachment in adolescence: A longitudinal perspective. In W. Koops, J. B. Hoeksma, & D. C. van den Boom (Eds.), *Development of interaction and attachment: Traditional and non-traditional approaches* (pp. 281–292). North-Holland, Amsterdam: Koninklijke Nederlandse Akademie van Wetenschappen Verhandelingen, Afd.

CHAPTER 9

Autonomy and Autonomy Disturbances in Self-Development and Psychopathology: Research on Motivation, Attachment, and Clinical Process

RICHARD M. RYAN, EDWARD L. DECI, and MAARTEN VANSTEENKISTE

INTRODUCTION 385
Autonomy, Growth, and Psychopathology 385
Chapter Overview 387
AUTONOMOUS REGULATION AND FACILITATIVE ENVIRONMENTS 387
Autonomous Regulation of Behavior 387
Distinguishing Autonomy from Independence 389
Facilitating Environments: The Key Role of Need-Supportive Socialization 390
AUTONOMY AND AUTONOMY SUPPORT IN MAJOR DEVELOPMENTAL PROCESSES: ATTACHMENT, INTRINSIC MOTIVATION, INTERNALIZATION, EMOTION REGULATION, AND IDENTITY FORMATION 396
Autonomy and Attachment Security 396
Intrinsic Motivation: A Spontaneous Expression of Human Autonomy 399
Internalization: Assimilating Social Regulations and Values 403
Emotion Regulation 406
Identity Formation 410
AUTONOMY DISTURBANCES IN DEVELOPMENT AND PSYCHOPATHOLOGY 413
Need Frustration and Psychopathology 413
Disorders Involving Introjection and Internally Controlling States 414
Impairments of Internalization in Externalizing Disorders 420
Severe Need Thwarting in Dissociative Identity and Borderline Personality Disorders 422
Translational Implications: Autonomy and Autonomy Support in Psychotherapy and Intervention Programs 425
SDT AND THE STUDY OF DEVELOPMENT AND PSYCHOPATHOLOGY: CONCLUSIONS AND IMPLICATIONS 426
Future Directions 427
REFERENCES 429

INTRODUCTION

Autonomy, Growth, and Psychopathology

Autonomy, defined as self-regulation and integration in acting, is central to healthy psychological development and functioning. Classic organismic and dynamic theories have long considered the movement toward greater autonomy and integration to be a hallmark of optimal development (e.g., Hartmann, 1947/1964; Jahoda, 1958; Loevinger, 1976; Piaget, 1971; Werner, 1948), a focus that continues in developmental psychologies today (see Sokol, Grouzet, & Muller, 2013). Because of the importance of children developing capacities for autonomy and integrated behavioral regulation, support for autonomy is recognized as a central aspect of optimal parenting by many theorists (e.g., Bretherton, 1987; Grolnick, 2002; Grusec & Goodnow, 1994; Hmel & Pincus, 2002).

Issues of autonomy and integration are in fact frequently implicated in the development of psychopathology (Ryan, Deci, Grolnick, & La Guardia, 2006). Historically, clinical literatures have highlighted that many forms of behavioral and mental disorders are characterized by autonomy disturbances (e.g., Shapiro, 1981; Winnicott, 1965). In many disorders, people's behaviors, emotions, and thoughts are experienced as pressured, compelled, or controlled, or alternatively uncontrollable (Ryan et al., 2006). For example, people can feel unable to regulate specific behaviors (e.g., some addictive and impulsive disorders). In other pathologies there is highly controlled, intentional behavior (e.g., restrictive eating, obsessive rituals) that is not well integrated or reflectively self-endorsed, whereas in other forms of psychopathology, usually involving considerable trauma at the hands of others, behaviors are enacted without being intentionally mediated by the self (e.g., dissociative disorders). In still others the motivation

for pursuing intentions and goals is absent or depleted (e.g., some forms of depression). In short, compromised autonomy is entailed in a wide range of pathologies, from those involving a lack of behavior regulation to those that entail rigidly enforced self-controls and compulsive behaviors. Corresponding to this, caregivers' obstruction of children's autonomy in conjunction with genetic vulnerabilities has been shown to foster a broad range of negative effects on development and is thought to contribute to the onset of varied disorders (e.g., Bruch, 1973; Depue & Lenzenweger, 2001; Ryan, 2005).

Given the role played by autonomy (or its absence) in both optimal development and developmental psychopathology, it is not surprising that autonomy support is emerging as an important, indeed critical, element in successful treatments for psychopathology (e.g., Mansour et al., 2012; Zuroff et al., 2007). In fact, most current behavioral and psychological intervention approaches attempt to support autonomy, albeit through different means, and with more or less explicit foci (Ryan, Lynch, Vansteenkiste, & Deci, 2011). In some treatment approaches, support for autonomy is seen as important in its own right for building essential inner resources that can buffer vulnerabilities and enhance adjustment (e.g., Ryan & Deci, 2008). Yet even when capacities for autonomy are not a focus, enhancing autonomous motivation for treatment enhances clinical success and engagement in behavior change over time (e.g., see Ng et al., 2012; Savard, Joussemet, Pelletier, & Mageau, 2013; Zuroff, Koestner, & Moskowitz, 2012).

Recognition of the importance of autonomy for healthy development and wellness has contributed to a fast-rising number of empirical studies on the topic of autonomy and its developmental dynamics. New research is coordinating phenomenological, functional, and mechanistic viewpoints in understanding autonomy and what influences it. Salient within this research is the power of social contexts (e.g., parents, teachers, coaches, peers) to facilitate the development and expression of autonomy and capacities for self-regulation in children. Salient as well is how the thwarting of autonomy, through either excessive control, conditional regard, or lack of empathy, disrupts development, and especially in vulnerable individuals, leads to dysregualtion and ill-being. Indeed, autonomy thwarting plays an important etiological role in many forms of psychopathology.

In this chapter we review the growing body of research on autonomy and autonomy disturbances primarily through the lens of Self-determination theory (SDT; Deci & Ryan 2000; Vansteenkiste, Niemiec, & Soenens, 2010). SDT is an empirically grounded theory informed by philosophical, clinical, and cross-cultural analyses of autonomy. SDT argues that among the predominant factors that foster resilience in development are opportunities to satisfy people's *basic* psychological needs, namely those for autonomy, competence, and relatedness (Ryan, 1995). Briefly, the need for autonomy refers to the necessity of experiencing a sense of volition; competence refers to the experience of effectiveness; and relatedness refers to the experience of warmth and reciprocal care. Support for the satisfaction of these needs is said to promote growth and well-being. Indeed, when social conditions support these psychological need satisfactions, individuals develop inner resources that allow them to better cope with adversity and flourish in their adult development. Interestingly, even individuals who do not explicitly value or desire these need satisfactions benefit from their fulfillment (Chen et al., in press). In contrast, the thwarting and frustration of these basic psychological needs is conducive to maladjustment and psychopathology (Ryan & Deci, 2000; Vansteenkiste & Ryan, 2013).

As an organismic theory, the principles informing SDT are fully consistent with a developmental psychopathology perspective (e.g., Cicchetti, 2006; Cicchetti & Toth, 2009). In particular, SDT focuses on nonclinical, subclinical, and clinical populations, connecting the conditions conducive to optimal psychological development with depriving and thwarting conditions that redirect developmental energies into self-protection, defense, and maladaptive coping. SDT sees the pervasive experience of having basic psychological needs frustrated during development as setting the stage for later difficulties and deficits in one's capacity for self-regulation of action and experience, often through cascading negative effects (Vansteenkiste & Ryan, 2013). In short, SDT suggests that the very same mechanisms, namely the satisfaction and frustration of people's basic psychological needs, accounts for substantial variance both in situational motivation and wellness and in developmental trajectories in self-regulation, well-being, and psychopathology.

In addition, and again in line with both developmental psychopathology (Cicchetti & Toth, 20009) and organismic perspectives more generally, SDT sees multiple pathways to both health and illness. By identifying the resources associated with integrative tendencies and psychological resilience, SDT allows for considerable flexibility and equifinality in development (Ryan, 1993), and conversely identifies many pathways of harm leading to diverse outcomes. Conditions affecting basic psychological need satisfaction versus frustration range from biologic to cultural, and the consequences of need supports are manifest at every level of analysis, from physiological to phenomenological. SDT additionally seeks to coordinate

these varied levels of analysis within its empirically driven framework, an aspiration that also characterizes the field of developmental psychopathology (e.g., Cicchetti, 2006; Cicchetti & Toth, 2009).

Chapter Overview

This chapter consists of three major sections. First we define autonomy as it is classically understood and currently applied within the SDT perspective. In doing so, we not only specify what autonomy entails, but also distinguish it, both theoretically and operationally, from other developmental constructs with which autonomy is often confounded. These include the constructs of independence (Silk, Morris, Kanaya, & Steinberg, 2003; Smetana, Campione-Barr, & Dadis, 2004); individualism (e.g., Iyenger & Devoe, 2003); individuation (e.g., Blos, 1979); and emotional autonomy (e.g., Steinberg & Silverberg, 1986). Next, we elaborate on the type of social environments that are conducive to autonomous regulation via the satisfaction of individuals' psychological needs and those that have been found to obstruct and undermine autonomous regulation by eliciting need frustration.

Then we discuss the different healthy manifestations of autonomy, thereby reviewing in greater detail its developmental origins. Using research from both SDT and attachment theory, we begin by highlighting that secure attachments in childhood are developed through autonomy supportive and involved caregivers, who not only hold and protect (i.e., provide security) but also stimulate the budding initiative and self-expression of the infant (i.e., support autonomy), setting the stage for developing further capacities for mature self-regulation (e.g., Miklikowska, Duriez, & Soenens, 2011; Whipple, Bernier, & Mageau, 2009). Apart from being conducive to secure attachments, autonomy supportive relationships support the integrative functions of the self that allow for greater internal psychological coherence and more effective behavioral regulation. Exemplifying this, we then turn to evidence concerning how autonomy supportive contexts facilitate developmentally relevant integrative processes including: (1) intrinsic motivation; (2) internalization and integration of social regulations; (3) the development of emotion regulation; and (4) the formation of identity. We pay particular attention to how socializers, especially parents, either facilitate or thwart the developing child's autonomy, thereby supporting or hindering the individual's inner resources for successfully negotiating each of these developmental lines.

These reviews of autonomy in psychological development, and of parental nurturance versus thwarting of autonomy, provide the foundation for examining autonomy disturbances in varied types and presentations of psychopathology. The next section focusing on psychopathology is twofold: we consider autonomy depriving and thwarting environments as an *etiological factor* in psychopathology; and we highlight how autonomy frustrations are often a *symptomatic factor* in various disorders and sometimes a by-product of other cascading effects. Moreover, we identify three general types of psychopathology in which autonomy is differently implicated. These are *internally controlling disorders*, in which there is excessive self-control or internal compulsion; *impairments of internalization*, reflecting both impoverished development of self-regulation capacities or lack of value for social regulations; and finally *fragmented self-functioning,* which we relate to chronic or traumatic need thwarting. We conclude this section by considering implications not only for clinical interventions, but also for the design of social institutions such as schools and workplaces that can support persons vulnerable to psychopathology. Finally, we provide a summarizing overview and discuss a number of critical future research directions.

AUTONOMOUS REGULATION AND FACILITATIVE ENVIRONMENTS

Autonomous Regulation of Behavior

Before proceeding to the intricacies of autonomy in development we begin with some basic definitions and distinctions. The term autonomy is derived from the Greek words *autos* (self) and *nomos* (rule). Autonomous actions are thus those that are self-ruled or governed by the self. The opposite of autonomy is heteronomy, when one's behavior is regulated or ruled by forces experienced as *heteron* or alien to one's sense of self.

Phenomenological analyses suggest that, when people act autonomously, they feel a sense of ownership of their actions (Pfander, 1967/1908). When autonomous, people are willingly engaged in activities. Accordingly, their actions are characterized by an *internal perceived locus of causality* (de Charms, 1968; Ryan & Connell, 1989). This quality of self-endorsement indicates that autonomous actions are informed by, and reflective of, individuals' abiding preferences and values and are congruent rather than discordant with their sense of self (Ryan Legate, Niemiec, & Deci, 2012; Shapiro, 1981; Weinstein, Prybylski, & Ryan, 2013). In contrast, when behaviors are heteronomous they are experienced as *controlled*. The actor experiences a sense of external or internal control, compulsion, or pressure to enact the behavior (Deci & Ryan, 2000). In attribution terms, people have an *external*

perceived locus of causality with respect to controlled behaviors. Interestingly, the functional effects of autonomy can be detected from early development and a sense of initiative and ownership of action is apparent from early infancy (Stern, 1985).

Although the concept of autonomy refers to integrated regulation by the self, autonomy is nonetheless a relative concept insofar as actions vary in their degree of autonomy. Autonomous functioning reflects the extent to which ongoing behaviors, thoughts, and feelings match with one's interests and values. Since most actions are both multiply motivated and more or less congruent, autonomy is inevitably a dimensional concept, and differentially associated with different types of motives. It is therefore basic to our view that autonomy is a *quality* of behavioral regulation, which is characterized by and requires an open processing of possibilities such that ongoing behaviors and underlying experiences are maximally integrated and volitional. This quality of behavioral regulation is relevant across the life span, even as the contents and focus of activities and values change with development.

Phenomenological analysis also points to the relationship between autonomy and integration in functioning. When autonomous, people are wholeheartedly behind their actions, and this is reflected in the coherence and lack of internal conflict involved in those actions. Yet to varying degrees behavioral regulations can represent compartmentalized or defended motives, or can be enacted because of external or internal pressures without reflective ascent or full endorsement. Thus the experience of autonomy is directly related to the relative integration of one's motivations and valuing (Ryan & Deci, 2008).

Although SDT's definition and understanding of autonomy is relatively unique within empirical psychology, it is strongly informed by philosophical treatments of the topic (e.g., Dworkin, 1968; Friedman, 2003). It can also be related to psychodynamic (e.g., Schafer, 1968; Loevinger, 1976; Shapiro, 1981) and humanistic (e.g., Rogers, 1963) traditions that have considered autonomy and integration to be highly important. Whether addressed in terms of self-endorsement, congruence, self-actualization, authenticity, will, or operating from the true self (see Ryan et al., 2006) many of these prior thinkers were addressing a shared idea, which is termed autonomy within SDT.

In addition, both the underlying sensibilities (e.g., recognition of values or interests in acting) and abilities (e.g., reflective capacities, goal selection, self-regulation skills) entailed in autonomy must develop, and accordingly there are individual differences in autonomous functioning (Deci & Ryan, 1985a; Weinstein, Przybylski, & Ryan, 2012). Yet autonomy is also situational, in part because social contexts differ in the extent to which they are autonomy supportive, controlling, or amotivating. This is true at all levels of development as parents, teachers, coaches, managers, and spouses, for example, can all facilitate or stifle autonomy in an individual, with significant mental health consequences. Yet whether due to dispositional or proximal supports, people evidence fuller functioning and more positive behavioral and psychological outcomes when more autonomous, including more sustained persistence, enhanced creativity, and higher quality performance (Vansteenkiste & Ryan, 2013).

Although phenomenologically autonomy is characterized by the experience of actions as being self-organized and volitionally supported, in terms of the quality of behavior, autonomy is manifest at varied levels of analysis. For example, evidence is emerging concerning the distinct neural basis of autonomous functioning relative to controlled, nonautonomous, motivational processes (e.g., Lee & Reeve, 2013; Murayama et al., 2014). In general terms, autonomy depends on the coordination between prefrontal cortical regions and subcortical striatal-thalamic areas that promote or inhibit motivation as well as on inputs from the hippocampus and amygdala that can provide contextual and affective information (e.g., Bradley, 2000; Chambers, Taylor, & Potenza, 2003). Autonomy requires supervisory and selective functions that are fully informed by affective and memory related processes (Di Domenico, Fournier, Ayaz, & Ruocco, 2013; Ryan, Kuhl, & Deci, 1997). Impairment in the development of prefrontal areas and interconnections with limbic structures produces vulnerability to autonomy disturbance (e.g., Bechara, Tranel, Damasio, & Damasio, 1996).

Interestingly, under autonomy-supportive conditions people appear more capable to recruit and use self-regulation strategies associated with executive functioning, a fact with important implications for development. For example, Bernier, Carlson, and Whipple (2010) examined whether maternal sensitivity and autonomy support were related to toddlers' capacity for executive control, which was assessed via a set of cognitive indicators involving flexible goal setting, impulse control, and planning. After controlling for children's cognitive capacity and maternal education, both sensitivity and maternal autonomy support, assessed when children were 12–15 months old, related to executive control at 18 and 26 months, with maternal autonomy support emerging as the most consistent predictor of enhanced executive control.

In fact, exposure to particular social contexts shapes the use of neurological resources required for autonomous functioning. For example, Di Domenico et al. (2013) argued, based on SDT, that people's abilities to develop

and act from a coherent sense of self are facilitated by satisfaction of the basic psychological needs. Using functional near-infrared spectroscopy (fNIRS), they examined the effect of need satisfaction on activity in the medial prefrontal cortex (MPFC), an important brain region for integrative processing of information about the self (Ryan et al., 1997). Participants engaged in a task involving a series of forced choices expressing personal preferences. Decisional conflict (i.e., choice difficulty) between the response options was manipulated using participants' previously collected preference ratings. Findings revealed that need satisfaction predicted elevated MPFC activity during high-conflict situations, suggesting that need satisfaction may facilitate access to and utilization of self-knowledge.

To summarize, autonomy is integrated self-regulation, and it is accompanied by a phenomenological experience of self-organization and volition, which is described in attribution terms by an internal perceived locus of causality (Deci & Ryan, 2000). Autonomy thus involves an experience of regulation by the self, and it is contrasted with actions experienced as pressured, controlled, or caused by sources external to the self. These later experiences are central to many forms of psychopathology, and can increasingly be mapped to neurological underpinnings.

Distinguishing Autonomy from Independence

An important distinction, particularly relevant to development, is that between *autonomy* and *independence*. Although autonomy and independence have been clearly distinguished in philosophical analyses, theorists in developmental psychology have often equated or conflated these concepts (e.g., Silk et al., 2003). *Dependence* is reliance on others for resources or guidance, so independence is not relying on others. Autonomy, by contrast, connotes willingness or volition. Given this, in the SDT view individuals can be autonomously dependent or autonomously independent, depending on circumstances. They can also sometimes be controlled or pressured into dependence, as when they feel forced to rely on others because of illness or financial hardship, or they can be heteronomously independent, as when pushed away from relying on others, who are unavailable or unwilling to provide assistance.

SDT suggests that autonomous forms of dependence (for example, when a teenager willingly turns to parents for guidance) are often quite beneficial, a view that stands in contrast to some developmental perspectives that have equated autonomy with separation or independence (Ryan & Lynch, 1989). For instance, Blos (1979) portrayed normative development in terms of an increasing relinquishing of both dependency on, and attachment to, parents. He viewed moving away from parental supports as indicative of increasing *individuation*. Based on Blos, Steinberg, and Silverberg (1986) developed a construct labeled *emotional autonomy* that was intended to be a marker of individuation. Scale items assessing emotional autonomy made reference to "not using parents as resources," "disagreeing with parents," and "being different when not with parents."

Working from the SDT lens, Ryan and Lynch (1989) reexamined the emotional autonomy scale, suggesting that it captured a detached relationship with parents rather than autonomy and they argued that such detachment or separation from parents is neither a necessary nor ideal pathway of development. They also suggested that such detachment might be more likely in adolescents who did not have autonomy-supportive parents. They therefore predicted that emotional autonomy as thus measured, rather than being indicative of positive maturation, would predict poorer adjustment. Consistent with this reasoning, findings indicated that adolescents high in emotional autonomy were those feeling less accepted by their parents and less lovable. These teens also evidenced poorer wellness outcomes overall. Ryan and Lynch (1989) interpreted these findings as highlighting the need to differentiate independence or nonreliance from autonomy.

Extending SDT's distinction between autonomy and independence, Van Petegem, Beyers, Vansteenkiste, and Soenens (2012) directly examined adolescents' level of independence, as well as their motives for both independent and dependent functioning. They found that autonomous (i.e., volitional) and controlled (i.e., pressured) motives for both independence and dependence played a more significant role in predicting adolescents' psychosocial functioning than did their level of independence as such. More autonomous forms of motivation for either dependence or independence were strongly associated with greater well-being and less problem behavior, whereas the level of independence was less predictive of these outcomes.

Because of the widespread interest within the developmental literature in assessments of autonomy and independence, Van Petegem, Vansteenkiste, and Beyers (2013) undertook a comprehensive study of 15 prevailing measures in adolescent psychology relevant to these constructs. Using higher order factor analyses, Van Petegem et al. (2013) showed that two basic dimensions provided the most parsimonious summary of the existing heterogeneity across available measures. A first dimension denoted the degree of *distance versus proximity* in the parent–child relation; a second dimension represented *volition versus pressure* in the relationship. The crossing of these two dimensions gives support for SDT's distinctions between autonomy and independence. For example, the

emotional autonomy scale (Steinberg & Silverberg, 1986), characterized by Ryan and Lynch (1989) as reflecting more detachment than autonomy, fell in the quadrant tapping pressured distance. In contrast, a scale of *emotional reliance* (Ryan, La Guardia, Solky-Butzel, Chirkov, & Kim, 2005), tapping adolescents' willing reliance on parents, fell in the volitional proximity-seeking quadrant. Van Petegem et al. (2013) further showed that the dimension of distance (versus proximity) was positively related to both avoidance and anxiety-based forms of attachment, whereas the dimension of volition (versus pressure) was negatively related to these insecure attachment styles. Such studies thus support the SDT literature in strongly distinguishing, both theoretically and empirically, autonomy from independence.

The distinction between autonomy and independence is critical to developmental psychology not only in childhood and adolescence, but also across the life span. For example, with advanced age people inevitably become more reliant or dependent on others. Nonetheless, there are important differences in wellness outcomes as a function of whether an older person's reliance on others feels autonomous and volitional or forced upon them (e.g., V. Kasser & Ryan, 1999; Vallerand, O'Conner, & Hamel, 1995). When elderly individuals can feel volitionally dependent and still experience a sense of choice in everyday living they remain more vital and resilient.

This distinction is also critical to cross-cultural psychologies. Various scholars have suggested that, because collectivist cultures emphasize duty and obligation to the group, autonomy is not very important for their members (e.g., Iyengar & Devoe, 2003; Markus, Kitayama, & Heiman, 1996). Yet this is a misinterpretation of the concept of autonomy, as it assumes people cannot autonomously engage in a duty, willingly comply with their parents' choices, or, with full volition, adhere to collectivistic cultural norms. In a direct examination of the distinction between autonomy and independence in the cross-cultural area, Chen, Vansteenkiste, Beyers, Soenens, and Van Petegem (2013) demonstrated that both Chinese adolescents' volitional reliance on parents as well as their volitional independent functioning yielded unique well-being benefits. Overall then, considerable data suggest that across cultural contexts, support for autonomy matters in both the internalization of ambient practices and in fostering individuals' overall wellbeing (Chirkov, Ryan, & Sheldon, 2010).

Finally, it should be noted that the concepts of independence and autonomy yield a different relation to individuals' development of relatedness and intimacy. Excessive strivings for independence often come at the cost of the development of trustful and warm relations, suggesting that, for individuals to develop most optimally, they need to balance their strivings for independence and relatedness. Yet, from the SDT perspective, the experience of volition and autonomy does not necessarily stand in conflict with the development of warm relations, because as one volitionally relies on another, a sense of intimacy can be maintained without giving up one's autonomy. Thus, instead of autonomy development extracting a price in terms of increased relational distance or tension, in SDT's view autonomy and relatedness optimally stand in a mutually reinforcing relation to each other (Hodgins, Koestner, & Duncan, 1996; LaGuardia, Ryan, Couchman, & Deci, 2000). Indeed, parental support for children's autonomy enhances parent-child relatedness, a pattern that persists across the age-related changes in types and foci of dependencies.

Facilitating Environments: The Key Role of Need-Supportive Socialization

As an organismic theory, SDT endorses a transactional-ecological conceptualization of development (e.g., Cicchetti & Valentino, 2006), in which growth and competencies, as well as impairments and liabilities at earlier stages of development impact the direction and quality subsequent development. Within such an organismic model a critical issue concerns the necessary nutrients for optimal development, which for SDT are represented by the satisfaction of the needs for autonomy, competence, and relatedness (Ryan, 1995). That is, individuals are prepared to optimally develop psychologically and socially when their caregiving environments support these basic needs. When these needs are thwarted, people are prepared with other protective, even defensive, responses, and both the healthier and the more defensive processes can have cascading developmental consequences (Vansteenkiste & Ryan, 2013).

Already in the earliest SDT parenting research (Grolnick & Ryan, 1989), three distinct parenting dimensions were identified as being critical for satisfying children's basic psychological needs and thus fostering more integrative development and wellness (Grolnick & Pomerantz, 2009; Soenens & Vansteenkiste, 2010). Specifically, these dimensions are parental *autonomy support*, parental *structure*, and parental *involvement*. Environments that supply these three core nutrients for basic psychological need satisfactions energize healthy self-development. SDT's focus on the satisfaction of children's psychological needs and facilitation of intrinsic growth tendencies is relatively unique in the prevailing parenting literature because few models are clear about the exact processes underlying

children's growth. At the same time, SDT maintains that the very blocking and direct thwarting of these needs not only prevents children from growing but also can even elicit maladjustment and psychopathology. Children's psychological needs will get more readily frustrated when parents are *controlling, chaotic,* and *neglectful*. In what follows we discuss the SDT model of parenting in detail, comparing it with alternative perspectives and contrasting constructs.

Parental Autonomy Support and Control

Being autonomy supportive literally means that one person is supporting the other's self-management or self-rule. Research has identified a number of elements in interpersonal interactions that enhance experiences of autonomy. Most important, in being autonomy supportive a parent (or other caregiver) attempts to understand and acknowledge the child's perspective. Critical to taking the children's frame of reference is an authentic curiosity from the side of the caregiver, who is sincerely interested in what is going on for the child. When this occurs, one is in a position to be responsive to whatever issues are salient. In addition, autonomy support entails using minimal controls to foster behavior, and instead providing a meaningful rationale for behavioral requests and limits where possible to help anchor internalized motivation. Autonomy support requires respecting the children's pace of development, so demands can be volitionally and effectively engaged, and providing opportunities for choice and voice when possible, all facilitate autonomy. In contrast, being controlling involves parents pressuring the children to do what they want or value, emphasizing obedience and compliance (Grolnick, 2002; Soenens & Vansteenkiste, 2010) and, in more extreme contexts, using punishments and physical threats. Dozens of studies have convincingly shown that autonomy-supportive parenting yields various benefits, including greater teacher-rated competence and grades (e.g., Grolnick & Ryan, 1989), more personal well-being (e.g., Chirkov & Ryan, 2001; Lekes, Gingras, Philippe, Koestner, & Fang, 2010), and better social adjustment (e.g., Soenens & Vansteenkiste, 2005).

Importantly, much like the experience of autonomy cannot be equated with independent functioning, the *support of autonomy* in SDT does not denote the *promotion of independence*. This was demonstrated in four studies by Soenens and colleagues (2007), who showed that parental promotion of independence and parental autonomy support were only moderately positively correlated. Moreover, while both independence promotion and autonomy support were positively associated with psychosocial functioning at the correlational level, when the two competed for variance in predicting these outcomes, only autonomy support was a significant predictor. In other words, the aspect of parenting accounting for better outcomes was autonomy support rather than the promotion of nonreliance. Soenens, Vansteenkiste, and Sierens (2009) subsequently showed that adolescents who perceived parents as allowing volitional dependence reported better psychosocial functioning than those who felt parental pressure toward either independence or dependence.

Rather than working from their children's perspectives, controlling socializing agents alter, minimize, or deny the children's perspectives, thereby projecting their own wishes and standards upon the children and pushing the children in the parent-desired directions. SDT's formulation of controlling parenting can be further differentiated into two categories (Soenens & Vansteenkiste, 2010). Parents can be controlling using *externally controlling* methods, such as the reliance on controlling reward or punishment contingencies. Externally controlling methods can be explicitly stated, but often the controlling contingencies are implicit and learned over time. A second subtype of controlling parental practices is *internally controlling* practices. This style of parenting has some overlap with *psychological control* as discussed in the frameworks of Schaefer (1959), Barber (1996), and Steinberg (2005). In this case, parents attempt to instill in the child positive and negative self-related feelings contingent upon compliance with parental standards and goals.

Internally controlling parental practices have been a concerted focus of research within SDT, especially through work on parental conditional regard. *Parental conditional regard* (PCR) describes parents or other caregivers using their attention, affection, and love to control their children's behavior (Assor, Roth, & Deci, 2004). As a socializing method, PCR is a widely used and sometimes advocated parenting practice (e.g., Sears, Maccoby & Levin, 1957; Fu & Markus, 2014). Yet from the SDT perspective PCR is theorized to be controlling and therefore to have both subtle and more manifest detrimental effects on self-functioning. Indeed, parents who use conditional regard are essentially pitting the children's needs for relatedness and autonomy against each other. The message conveyed is essentially "to get my love you need to give up your autonomy." To maintain positive relatedness, the children have to comply, irrespective of the fit of the parental demands with their own developing interests, skills, or values. Yet, even when complying, children who experience PCR do not feel accepted but instead evidence resentment, because what is conveyed is that they are not being accepted for who they are but instead for whether they live up to their parents' demands. As such, the use

of PCR with children serves to thwart the needs for both relatedness and autonomy, with resulting decrements in adjustment and well-being (e.g., Assor, et al., 2004; Roth & Assor, 2012).

Parental Structure and Parental Chaos

Apart from parental autonomy support, SDT also highlights the critical role of parental *structure*, which concerns the degree to which parents or other caregivers create the necessary conditions for children to begin developing a sense of mastery and provide ongoing assistance to optimize competence development (Farkas & Grolnick, 2010; Grolnick & Pomerantz, 2009; Reeve & Halusic, 2009). Structure entails the provision of an environment that makes optimal demands on children's expanding skills. Structuring parents provide guidance and scaffolding upon which the unfolding processes of development can climb. Specifically, provision of structure by parents includes: helping to adjust environments so that they can be mastered by the children without excessive anxiety; conveying consistent and clear expectations; providing guidance about the contingencies between behaviors and outcomes; offering meaningful informational feedback as the children engage in activities; and other elements of support focused on promoting the children's feeling of effectance in acting. In the research by Grolnick and Ryan (1989), parental structure predicted children's understanding the sources of control over outcomes in their lives, enhancing both the children's sense of predictability, and of competence or efficacy. In the educational domain, Mouratidis, Michou, Vansteenkiste, and Lens (2013) showed that competence need satisfaction largely accounted for the relation between perceived teacher structure and children's wellness and use of self-regulation strategies.

Although structure aids the building of competence, this process might be more or less facilitated depending on whether an autonomy-supportive or controlling style is used when introducing and maintaining the structure (Soenens & Vansteenkiste, 2010). Specifically, the competence-enhancing impact of structure is likely strengthened when the structure is communicated in an autonomy-supportive way and diminished when done in a controlling way.

The differential impact of providing structure in autonomy supportive versus controlling ways was demonstrated early within SDT research, most notably in an experimental study by Koestner, Ryan, Bernieri, and Holt (1984). In a school setting first- and second-grade children came individually to an art room to do a painting project. An experimenter set limits on their behaviors, advocating neatness and organization, either in a controlling or autonomy-supportive way. The autonomy-supportive method was based on SDT and included a rationale, an expression of empathy, and a clear limit. The controlling method simply directed the child to follow the limit. It was found that the controlling limits undermined the enjoyment of painting and creativity relative to autonomy-supportive limits.

Overall then, structure is in no way antithetical to support for autonomy; on the contrary, the two often go hand in hand (Stipek & Seal, 2001). Yet this conceptual point can easily be lost if parental structure is described as, or equated with, parental control. That is, if parental structure involves parents exerting pressure and control over children's functioning, that parental structure would indeed stand in contradiction to parents' encouragement of children's volitional functioning. Similarly, if parents are permissive, granting unmitigated freedom rather than specifically supporting autonomy, this would be contrary to providing structure. Parental autonomy support, in fact, can and optimally does involve parents introducing and maintaining structure, and does not entail avoiding all guidance and scaffolding (Grolnick & Pomerantz, 2009; Soenens & Vansteenkiste, 2010). SDT also expects that structure can be imposed in controlling ways, with less beneficial outcomes. Thus, the concepts of autonomy support and structure allow for a clearer differentiation between critical dimensions needed in parenting.

Consistent with this reasoning, Jang, Reeve, and Deci (2010) showed that structure and autonomy support were modestly positively (rather than negatively) correlated and independently contributed to positive educational outcomes. Along similar lines, examining a broad set of components of parental structure, Farkas and Grolnick (2010) found parental structure and autonomy support to yield unique positive associations with seventh- and eighth-grade children's perceived competence and actual grades. Overall, then, past research has found structure and autonomy support to be positively related to each other rather than antagonistic, and often each has yielded unique associations with a host of different outcomes. Moreover, by distinguishing the two concepts, one can assess the meaningful combinations between them, with expectations that controlling structure will have negative effects on development, whereas autonomy-supportive structure will be more positive. In fact, a number of studies in the domains of education (e.g., Vansteenkiste et al., 2012) and sports (e.g., Curran, Hill, & Niemiec, 2013) indicate that

the most optimal outcomes are attained under conditions of high autonomy support and high structure.

Parental Involvement and Parental Neglect

SDT also suggests that more than structure and autonomy support are needed to facilitate development. In addition, *involvement,* especially autonomy-supportive involvement is also helpful. Involvement refers to a parent's dedication of attention and caring, which both supports the child directly and conveys relatedness and connection as he or she faces developmental challenges (Grolnick & Ryan, 1989). Parental involvement has long been found to have positive effects on children's behavioral regulation and well-being (e.g., Baldwin, 1955). In the Grolnick and Ryan (1989) study, assessment of involvement included the time parents spent with their children in interactive ways, their knowledge of their children's day-to-day life, and the warm tone of interpersonal contact. Parental involvement and in particular the mothers' involvement predicted children's achievement and grades in school, and was negatively correlated with teachers' ratings of children's acting out behaviors. Subsequently, Grolnick, Ryan, and Deci (1991) found that parental involvement, along with autonomy support, predicted children having greater inner resources for school engagement, especially greater feelings of autonomy and competence. Further, Cheung and Pomerantz (2011) found that in both U.S. and Chinese samples, involvement predicted engagement and achievement in schools. In both nations, when such involvement was accompanied by autonomy support children also had greater perceptions of competence and better emotional functioning.

More recently, Grolnick (in press) looked at parents' motivation for their involvement with children. This is a particularly interesting area of investigation because parents can be involved both out of controlling motivations such as introjection or external pressures or because of value and interest in their children. Grolnick found that the more autonomous the parental motivation, the higher the level of involvement. Moreover, identified regulation was especially predictive of child outcomes one would expect from such willing involvement, such as more positive academic self-concept and better school grades.

These and related findings speak to the ways parents are involved, which can vary considerably. For example, much like the dynamic involved in conditional regard, parents can make their involvement (i.e., their devotion of time and resources to the child) dependent on obedient behavior, which SDT suggests will come with a cost. Consistent with this idea, Weiss and Grolnick (1991) studied the relations of parental involvement and autonomy support to adolescents' internalizing and externalizing symptoms. Adolescents rated their mothers and fathers on dimensions of involvement and autonomy support and completed a self-report profile of symptoms. Analyses indicated that parents who were perceived to be both highly involved and autonomy supportive had children who reported very low levels of either internalizing or externalizing symptoms. Yet there were significant interactions between perceived parental involvement and autonomy support on both types of symptoms, indicating that the combination of high involvement and low autonomy support yielded a high level of symptoms.

This general pattern of findings has been observed in other studies. Wouters, Doumen, Germeijs, Colpin, and Verschueren (2013) found that high parental responsiveness strengthened the relations between psychologically controlling parenting and contingent self-esteem in university students. Aunola and Nurmi (2004) found in a three-year longitudinal study among 5–6-year-old children that parents' exertion of controlling practices slowed children's progress in mathematics, a trend that was exacerbated in cases where parents displayed high affection. Interestingly, the combination of high parental control and responsiveness appeared not only to yield a personal well-being cost but also to generalize to young adults' empathic responses. In this context, Kanat-Maymon and Assor (2010) showed that parental control related positively to the child perceived distress when their romantic partners were in need, whereas parental control was negatively related to empathic responding as reported by the partners, both effects being strengthened when parents were experienced as highly responsive.

It thus appears that feeling close to very controlling parents can be detrimental to a developing child's personal and social well-being, presumably because these children's psychological needs for autonomy and relatedness are insufficiently met. That is due to the high yet controlling involvement of parents, which may leave the children feeling as if they have no other choice than to connect with their parents by complying with their demands. Similar to the dynamics characterized by enmeshed families, however, the children's connections are likely to be fueled with feelings of internal conflict and pressured loyalty, thereby causing heightened emotionality and self-preoccupation, which may interfere with their capacities to respond empathically to others (Kanat-Maymon & Assor, 2010). In short, the combination of high parental control and high

responsiveness may create confusion because parents are sending conflicting messages to the their children (Aunola & Nurmi, 2005).

Parent's Psychological Needs: Contextual and Reciprocal Influences

Thus far, our focus has been on how parents affect children's development and problem behavior through the nurturance or undermining of children's psychological needs. Yet parents themselves are subject to influences that can either support or thwart their capacities to support their children's development. That is, to the extent that parents engage in need-satisfying activities in their own lives, they are likely to have more energy available to be involved with their children, thereby being better attuned to the children's perspectives and, hence, being more capable of supporting children's unfolding interests, values, and skill development. To the extent that parents' psychological needs get thwarted, their energy levels would be more readily eroded so they would become more self-centered rather than open and receptive for their children's concerns and interests. Such self-centered preoccupation may either manifest through a direct withdrawal and involvement from the children or, to the extent that they are involved, parents would be more easily enforcing their own viewpoint or be unpredictable and inconsequent. This reasoning is graphically displayed in Figure 9.1, in which parents' own need satisfaction and need frustration are part of the proximal determinants of their approach to their children. In an examination of this dynamic, De Haan, Soenens, Dekovic, and Prinzie (2013) found need satisfaction as reported by the parents in the parent–child relationships relate negatively to the adolescents' reports of negative parental styles (e.g., overreactive or controlling).

At the same time, a variety of more distal factors, either pressuring or facilitative, will impact on parents' experiences of need satisfaction and need frustration. Pressures on parents can come from their children's behaviors in the interactions of the parents and children. That is, there are both *reciprocal influences* from the children and proximal and distal social and economic pressures on parents from other sources that impact their resources (Deci & Ryan, 2012). For example, research on child temperament suggests that children with difficult temperaments may make it harder for parents to refrain from controlling behaviors. Within the SDT literature, Grolnick, Weiss, McKenzie, and Wrightman (1996) found that parents who perceived their adolescents to be more difficult in temperament were also more controlling. In addition, it has been found that when children evidence poorer performance or get poorer grades in schools, parents often become more controlling (Aunola & Nurmi, 2005; Grolnick, Gurland, DeCourcey, & Jacob, 2002). In a particularly interesting study, Pomerantz and Eaton (2001) found that a child's low achievement was

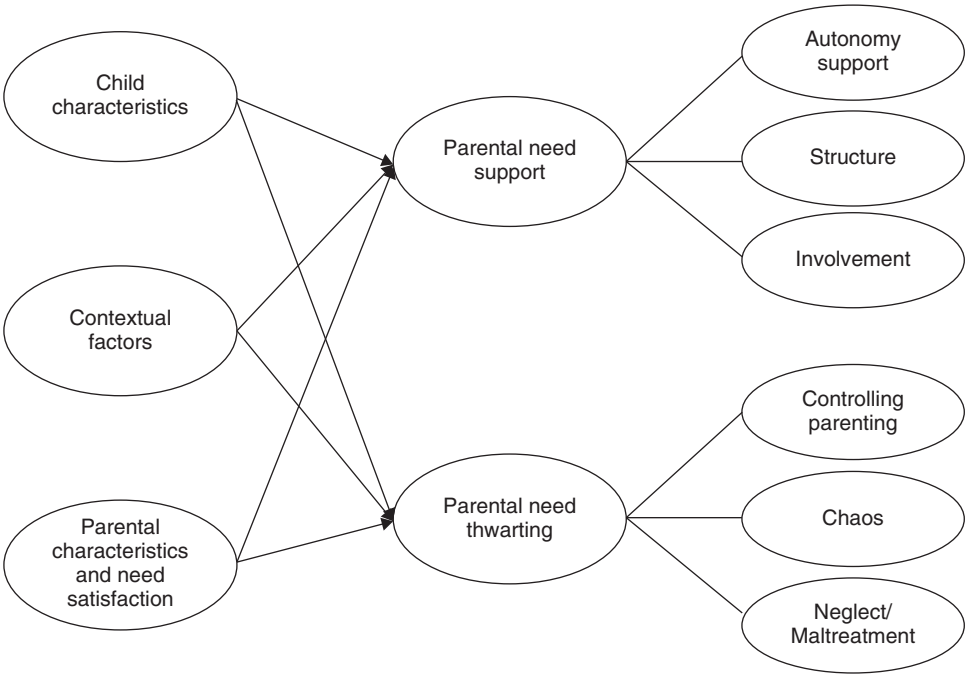


Figure 9.1 Graphic overview of the role of antecedents of parental need support and need thwarting.

associated with increased maternal worry, and worry predicted more controlling behavior, which in turn negatively affected the child's achievement. Similarly, adolescents' defiance against parents' moral rules elicited a more controlling parental approach over time (Vansteenkiste, Soenens, Van Petegem, & Duriez, 2014).

The parents' own supports can also bear on their capacity to nurture their children. For instance, Landry et al. (2008) showed that mothers who experienced greater social and partner supports were more able to trust the developmental process and thus support their children's autonomy. Similarly, Bouchard, Lee, Asgary, and Pelletier (2007) found that fathers' involvement and satisfaction in parenting was related, not only to their own perceived competence, but also to their perceptions that their partners placed confidence in their parenting abilities. In addition, Assor et al. (2004) found that many parents display the same parenting approaches (e.g., conditional regard) they were exposed to as children, thus exhibiting the intergenerational transmission of parenting styles.

Also parents often face a host of contextual pressures, which lead them to adopt a more controlling approach toward their children. Economic strain (Kasser, Ryan, Zax, & Sameroff, 1995), stressful life events (Grolnick, Bridges, & Connell, 1996), and experimentally induced stress (Grolnick et al., 2002) have all been associated with controlling parenting behavior. In one illustrative experimental study, Grolnick, Price, Beiswenger, and Sauck (2007) examined the effects of situational pressures on mothers' autonomy support, relative to control, in the social domain. The authors had 60 mothers work on a laboratory task with their fourth-grade children, presumably in preparation for meeting new children. Mothers in an evaluation condition were told that their children would be evaluated by other children and that based on this, the researchers would determine how much each child was liked and accepted by their peers. In the no-evaluation condition, mothers received no mention of peer evaluation. Results revealed that mothers in the evaluation condition spent more time giving answers to their children, especially mothers with more controlling parenting attitudes, and they also exhibited more controlling behaviors. In addition, mothers with high social contingent-self-worth who were in the evaluation condition exhibited the most controlling behaviors.

Apart from contextual pressures, internal forms of pressure, such as anxieties and worry, have also been associated with more controlling parent behaviors, especially when the worries concern their children. Gurland and Grolnick (2005), for example, examined mothers' perceptions of environmental threat regarding such issues as future economic instability and resource scarcity. Perceptions of future threat were positively associated with more controlling behaviors exhibited during parent–child tasks. In turn, these controlling behaviors were negatively associated with children's volitional motivation. Moreover, the relation between perceived threat and children's motivation was mediated by controlling parenting.

As can be noticed in Figure 9.1, parents' own personality functioning may also lead them to rely on certain parenting strategies more than others. Critical in this respect is parents' organismic trust in the development of their children (Landry et al., 2008), with parents high in organismic trust being more relaxed in their expectations regarding milestones and engaging in fewer social comparisons. Landry and colleagues showed that such organismic trust led mothers to rely on different parenting strategies during a play session where they and their 12–13-month-old infants were given problems to solve together. Mothers' autonomy support and structure were assessed using ratings of the extent to which the mothers took the children's perspectives, followed the children's pace, and structured the situation to be fitting with the children's skills. Autonomy thwarting behaviors were also measured, including tallying empathic failures and interfering in the children's pace, among other indicators. Results revealed that organismic trust, even when controlling for parental income and education and children's temperament and level of development, was significantly associated with greater autonomy support as opposed to autonomy thwarting.

Other personality factors within parents are also relevant. For example, Deci and Ryan (1985a) early on reported that persons with a stronger control orientation were less prone to support autonomy in others. The reason that parents with a stronger controlled orientation are more likely to use controlling techniques is because they more easily hinge their own self-worth upon their children's achievements. Thus, control oriented parents extend their ego-involved function to their offspring, thereby measuring their own success and worth versus failure and shame as parents through their children's accomplishments (Wuyts, Vansteenkiste, Soenens, & Assor, in press). Further, Soenens, Vansteenkiste, Duriez, and Goossens (2006) hypothesized that parents who have high separation anxiety may perceive expressions of their children's autonomy as threatening, and may be more likely to restrain autonomous behavior through controlling techniques. In addition, they argued that self-critical perfectionism in parents would lead them to project higher demands

on their children, thereby pressuring toward specified outcomes. Their results supported these hypotheses.

Together such findings underscore that parenting behaviors are themselves influenced by a variety of factors both external and internal—from the socioeconomic pressures on parents that stretch their resources and lead to insecurities, to internalized social pressures to make their children achieve. Pressures on parents in the form of threats thus interrupt organismic trust and capacities for nurturance. On the other hand, it is important to highlight that most parents, because of their inherent attachment and love for their children, naturally seek to provide support and guidance so that even their controlling behaviors are often, despite their negative consequences, expressions of their parental caring (e.g., see Chua, Wong, & Koestner, 2014).

Summary

SDT is primarily focused on nurturing, facilitating environments that support the growth of the self. The theory specifies three operationally distinctive nutritive dimensions, namely, autonomy support, structure, and involvement. These parenting dimensions are systematically related to the satisfaction of children's basic psychological needs and allow for greater specification of optimal parenting relative to mixed constructs such as Baumrind's (1996, 2012) construct of authoritative parenting and confrontive power (see Grolnick, 2002, 2012). Further, in discussing the need-thwarting role of parents, we especially emphasized the role of controlling parenting, with conditional regard being the most studied controlling practice within SDT. Finally we suggested that parent behaviors do not occur in a vacuum. Parental stress, sense of threat, and ego involvement can all lead to controlling behaviors and can detract from positive involvement and capacities for autonomy support.

AUTONOMY AND AUTONOMY SUPPORT IN MAJOR DEVELOPMENTAL PROCESSES: ATTACHMENT, INTRINSIC MOTIVATION, INTERNALIZATION, EMOTION REGULATION, AND IDENTITY FORMATION

Having elucidated the critical parenting dimensions fostering growth, in this section we elaborate the role of autonomy in development and personality functioning. First, we focus on the relations between autonomy and *attachment security* (Bowlby, 1969). SDT suggests that autonomy support is an underemphasized yet critical predictor of secure attachment experiences (e.g., Whipple et al., 2009). Even more, we argue that security provision is insufficient in accounting for healthy self-development, which requires the more active notion of autonomy support. We then turn to a review of research on four critical developmental processes in which autonomy figures heavily, namely, intrinsic motivation, internalization, emotion regulation, and identity formation, and we explicate the role of autonomy and the impact of contextual supports for it.

Autonomy and Attachment Security

Connections between autonomy and relatedness have been hotly debated in both developmental and social psychologies. Some authors see autonomy and relatedness as largely orthogonal if not antithetical constructs (e.g., Blos, 1979; Ingeyar & DeVoe, 2003; Jordan, 1992). As noted already, SDT differs, viewing autonomy and relatedness as being interrelated and, indeed, integral to one another. This is so from the earliest phases of life, in which the strength and security of attachment is, according to SDT, in very large part a function of the autonomy support afforded by caregivers.

Attachment and Autonomy in Infancy

It is particularly difficult to think about autonomy without its foundations within relatedness during the child's first years of life and vice versa. During early development, establishing a core self is a primary agenda for the infant (Stern, 1985). Although the self that is consolidated within the first six months does not take the form of a conscious representation, it can manifest as coherence and volition. Yet because infants are highly dependent on caregivers—not only for biological necessities such as food and temperature regulation but also for interpersonal necessities such as love, contact, and comfort—these earliest experiences of the self as initiator of action typically occur within the context of close parent–child relationships.

Bowlby (1969) and later theorists elaborating his attachment theory framework (e.g., Mikulincer & Shaver, 2007) have argued that early social relations with primary caregivers shape both the security of attachment and the corresponding working models of self and others that will organize future social relations. Perhaps the most emphasized feature within attachment theory that contributes to security of attachment is caregiver responsiveness or *sensitivity* (Ainsworth, Blehar, Waters, & Wall, 1978). Sensitivity is defined as the provision of contingent, appropriate, and consistent responses to the child's signals and needs (Lamb & Easterbrooks, 1981). Sensitivity thus conveys that caregivers are responsive to the child's initiations

in ways that are empathic and appropriate to the child's needs. In our view, sensitivity is part of autonomy support, though by no means encompasses that construct.

Within SDT it has been suggested that when one unpacks the kind of parenting that fosters growth and security, chief among them will be autonomy support (e.g., Ryan, 1993; Ryan, Brown & Creswell, 2007). As noted, in autonomy support what one is being sensitive to are the child's initiations, strivings, and the needs of the developing self (rather than the caregiver's agendas or projected needs). Indeed, Bretherton's (1987) description of sensitivity as "maternal respect for the child's autonomy" (p. 1075) captures the essence of such active responsiveness. The sensitive caretaker responds to the initiations, cues, concerns, and needs that emerge from the child, and this contingent responsiveness gradually strengthens the child's inner sense of agency and coherence, and thus the meaningfulness of the child's self-initiated action and expression. When responded to, the child experiences a sense of safety and interconnection that is lacking for one whose biddings and expressions have been neglected.

Autonomy support is thus a critical component of early parent-child relationships, for it facilitates not only the solidity of attachment but also self-development more generally. Numerous past studies support this perspective by linking sensitivity or autonomy support to more curiosity, effectance, initiative, and adjustment (e.g., Stevenson & Lamb, 1981; Waters, Wippman, & Sroufe, 1979; Yarrow, Rubenstein, & Pederson, 1975). Specifically applying SDT, Grolnick, Frodi, and Bridges (1984) found that mothers rated as autonomy supportive had 1-year-old infants who both explored more and were more persistent at a play task than infants of mothers rated as controlling. Further, Frodi, Bridges, and Grolnick (1985) reported similar results for the persistence and competence at 20 months of age. Such studies suggest that in early stages of development, parental attunement and support for autonomy are enhancing the child's inner resources for self-regulation.

We suggest, in fact, that autonomy support is an aspect of caregiving that is far too underemphasized in attachment theory's foundational focus on security, protection, and responsiveness. Underscoring this argument, Whipple, Benier, and Mageau (2011) drew from SDT in using observational assessments of maternal autonomy support during infant (15 months) exploration. They also assessed maternal sensitivity with a 90-item measure of the quality of maternal behaviors during a home visit and security of attachment using an observational Q-sort method. Results showed that not only sensitivity but also autonomy support explained independent variance in attachment security. This shows how autonomy support is not encompassed by traditional conceptions of sensitivity.

This interrelation between autonomy support, attachment, and self-development is nonetheless consistent with some of Bowlby's (1969) speculations concerning infant exploration and mastery motivation. Bowlby postulated that to the extent that a child experiences security (i.e., caregiver accessibility and responsiveness in times of need), then the child will feel enabled to venture forth to explore with interest. Yet SDT would suggest, beyond Bowlby, that security is much more than protection and comfort; it is in fact responsiveness *to the self* of the child that fosters secure relationships, not simply reliable protection (Ryan, Brown, & Crewell, 2007).

It is worth noting in this regard that for Bowlby, attachment was first and foremost about safety and protection rather than facilitating growth and development. As Kobak, Cassidy, and Zir (2004) stated, attachment is a "behavioral system activated by appraisals of danger and accompanying signals of fear" (p. 388). Adult attachment theorists concur, arguing that "the goal of the system is a sense of protection and security" (Mikulincer & Shaver, 2007, p. 5). Without doubting the importance of protection and the value of reducing anxiety in moments of danger, one can question whether this is a sufficient foundation for a full theory of self-development (see Ryan et al., 2007). Although moments of dangers and strangers can be salient, they are episodic, and comfort following them may be a necessary, but not a sufficient, condition for the interpersonal support for self-development. Even among the object-relations theorists in Bowlby's time, figures such Winnicott (1965) more strongly pointed to the importance of ongoing positive supports for the nascent self to not only feel safe, but to develop and flourish. Winnicott highlighted not only a safe haven but also active empathy and having spontaneous initiations amplified through mirroring and responsive care. Like Winnicott, SDT argues that support and nurturing relationships in infancy extend well beyond security and protection to the *active facilitation* of self-functioning. Indeed, security and protection are likely key to prevent experiencing of need frustration, yet, for children to experience autonomy, competence, and relatedness, more active support of these needs is required. In fact, as we turn to other developmental processes such as intrinsic motivation, internalization, emotion regulation, and identity formation, the role of caregivers' support for the child's psychological needs, including autonomy, emerges as a critical facilitator of self-development.

This brief analysis of the role of autonomy and relatedness in infancy thus suggests that autonomy support is

a critical component of caregiving in infancy that shapes both the experienced quality of relatedness (as reflected in the security of attachment) and the consolidation and vitality of the self (as reflected in both well-being and mastery motivation). Caregiving that is low in autonomy support, by contrast, will set the stage for attachment disturbances and impoverished agency.

More recent studies have begun to examine the independent role of parental autonomy support and attachment security per se in young children's development. For instance, Verschueren, Doumen, Vervoort, and Vansteenkiste (2013) showed in a longitudinal sample of preschool children that both a secure attachment and maternal autonomy support, as observed during a problem solving task, related to higher achievement on a standardized language and mathematics test taken at the end of the first elementary school year, through partially distinct pathways. Attachment security in preschool was related to greater closeness in the teacher–child relation in the first school year, whereas maternal autonomy support related to both greater teacher–child closeness and more learning based in interest and enjoyment (i.e., intrinsic motivation), which in turn predicted greater actual achievement. These findings suggest that a secure and autonomy-supportive child–parent relationship promotes a more successful transition from preschool to elementary school, with need-supportive interactions generalizing to new socialization figures.

Attachment and Autonomy in Adolescence and Beyond

There is increasing evidence that at whatever age attachment security is assessed, the provision of autonomy support from attachment figures will be a critical predictor of attachment security. Adolescence, for example, is a critical period of life in which teens are rapidly developing capacities for independence, while still needing considerable support and nurturance. As noted previously, in SDT's view, rather than individuating *from* parents, healthy adolescent development is characterized by continued attachment and dependence along with autonomy. In fact, evidence suggests that security of attachment and reliance on parents in this age is quite important for well-being and that these are facilitated by caregivers' support for autonomy.

First, several studies support the view that adolescents continue to benefit from secure attachments to parents rather than by the detachment that Blos (1979) advocated. Ryan, Stiller, and Lynch (1994) investigated the contributions of felt security with parents, teachers, and friends to the motivation, adjustment, and self-esteem of early adolescents. They found that felt security with parents and teachers, as well as utilization (reliance on) these figures, predicted adjustment. Further, they found evidence that felt security with parents predicted security with both teachers and peers, and appeared to have priority among working models. Similarly, Bober and Grolnick (1995) assessed adolescents' felt security with and emulation of parents. Adolescents who displayed internalizing symptomatology reported low levels of felt security with parents yet emulated them. Adolescents with externalizing symptomatology reported low levels of felt security and low emulation of parents.

Ryan and Kuczkowski (1994) examined felt security with parents as it relates to the emergence of *private audiences* in the cognitive development of adolescents. Following Elkind (1967), they argued that the private-audience phenomenon initially represents a heteronomous influence on behavior for most adolescents, who become more self-conscious and more prone to conformity. Yet with self-development the salience of private audiences has been hypothesized to lessen. Ryan and Kuczkowski (1994) predicted and found that whereas felt security with parents was unrelated to the strength of private audiences in early adolescence (when it is a normative aspect of development), it was more strongly related in later adolescents. Adolescents experiencing insecurity with parents remained more preoccupied with private audiences, showing one pathway through which the growth of autonomy is hampered.

Ryan and Lynch (1989) examined the issues of autonomy and attachment in early, middle, and late adolescent samples. They argued that autonomy is typically *facilitated* by secure attachment to, and dependence on, parents rather than by detachment and nonreliance on them. They further suggested that among the primary reasons that adolescents are insecure with or detached from parents is that parents are overly controlling. Their studies confirmed that adolescents with more secure attachment to parents were, in fact, better adjusted, and had parents who were more autonomy supportive. These findings have been well replicated, including recent studies of both referred and nonreferred youth and their mothers, in which parental autonomy support related to lower anxiety-based attachment, while parental responsiveness and sensitivity related to lower avoidance-based attachment (Brenning, Soenens, Braet, & Bal, 2012; Karavasilis, Doyle, & Markiewicz, 2003).

The importance of autonomy supports for secure attachments extends to early adulthood. La Guardia, Ryan, Couchman, and Deci (2000) studied the attachment experiences of college students with multiple social partners such

as their mothers, fathers, best friends, and romantic partners. They found, as classical attachment theory predicts, that individual differences in security of attachment were a moderately significant predictor of patterns of security of attachment across social partners. Yet they also hypothesized and found significant variability in students' security of attachment with these different social partners as a function of the different qualities of interpersonal supports provided within each relationship. In three studies, La Guardia and colleagues applied multilevel modeling to reveal that within-person variability in attachment security was systematically explained by relationship-specific autonomy support. In fact autonomy support was an especially consistent predictor of felt security, relationship satisfaction, and willingness to rely on that person.

In a more recent study of adult attachments, autonomy support, and emotional reliance, Lynch (2013) collected daily data on autonomy support and security of attachment to predict patterns of *emotional reliance*—that is, the individual's willingness to turn to another person for emotional support (see Ryan et al., 2005). It has been assumed by attachment theorists that secure attachments would be associated with positive abilities to recruit support from others when distressed (e.g., Belsky, 2002). Yet Ryan et al. (2005) argued that autonomy support was also particularly crucial in facilitating such emotional reliance, as people are less prone to rely on others who are controlling. Lynch (2013) therefore allowed measures of both attachment security and autonomy to compete at both trait and state levels in predicting emotional reliance. At the trait level both security of attachment and trait autonomy predicted greater tendencies to emotionally rely on others. At the within-person level, results showed, as predicted by SDT, that people were specifically more willing to turn to those people whom they perceived to be autonomy supportive. Noteworthy is that at both within- and between-person levels of analysis, emotional reliance was more strongly linked with autonomy than with attachment security.

Summary

SDT and attachment theory thus concur that attachment security is associated with higher well-being and ability to rely on others. SDT additionally maintains that autonomy support is critical to experiencing attachment security, including primary attachment figures. Moreover, through the more active nurturance of explorative behaviors, emerging interests and personal values, autonomy-supportive agents do more than provide comfort; they promote children's healthy development.

Intrinsic Motivation: A Spontaneous Expression of Human Autonomy

Intrinsic Motivation: Definition

Perhaps the most direct catalyst of empirical research on autonomy within both social and developmental psychologies has been interest in the phenomenon of intrinsic motivation. *Intrinsic motivation* is defined as motivation derived from the inherent satisfactions of an activity—it applies when actions are experienced as interesting or enjoyable for their own sake (Deci & Ryan, 1985b).

Harlow (1950) was, to our knowledge, the first scholar to employ the term intrinsic motivation. He used it in reference to the persistence of primates, curiously working at mechanical puzzles without reinforcements or rewards. In fact, he was an early observer of an "undermining effect" of rewards, noting that attempts to enhance curious behaviors with reinforcements disrupted such intrinsic motivation. Subsequently, White (1959), drawing on both comparative psychology and developmental studies, argued that this type of interest-driven behavior underlies much of human social and cognitive development. Indeed, this idea of an innate, natural propensity toward the exercise and growth of capacities has been an important postulate in the study of developmental and learning processes (Deci & Ryan, 2013; Flavell, Miller, & Miller, 2002; Koestner & McClelland, 1990).

Intrinsic motivation reflects a natural assimilative tendency, which is evident in the active exploration, manipulation, curiosity, and playful exercise of skills we observe in healthy children. Intrinsically motivated play and activity foster learning and the growth of competencies, as developmental theorists have long argued (Harter, 2012). Also studied as *mastery motivation,* intrinsic motivation is clearly evident in infancy (e.g., Yarrow et al., 1975) and differentiates into more specific interests as children grow (Deci & Ryan, 2013). Numerous studies have linked intrinsic motivation to deeper learning (e.g., Grolnick & Ryan, 1987; Yamauchi & Tanaka, 1997), creativity (e.g., Amabile, 1983), and other indicators of high-quality assimilation.

Social Contexts and Intrinsic Motivation

Although intrinsic motivation is posited to be natural, its unfolding and development is nonetheless viewed within SDT as being dependent on specific social and contextual nutrients (Deci & Ryan, 1985b; Ryan, 1993). A substantial amount of research has therefore explored the effects of various social contexts on intrinsic motivation, and this

body of research has been described within a specific mini-theory of SDT called *cognitive evaluation theory* (CET; Deci & Ryan, 1985b). In brief, CET argues that intrinsic motivation is dependent on experiences of both autonomy and competence. Social contexts that are either controlling or autonomy inhibiting (Grolnick & Ryan, 1989) or that lack challenges or feedback that supports felt competence (e.g., Abuhamdeh & Csikszentmihalyi, 2012; Mouratids et al., 2013) are expected within CET to diminish intrinsic motivation. In contrast, environments that support autonomy (e.g., provide choice, are responsive to initiatives) and competence (are well structured, provide positive feedback) are expected to maintain or enhance intrinsic motivation. Finally, in contradistinction to social-cognitive approaches (e.g., Bandura, 1989), CET holds that that intrinsic motivation requires autonomy satisfactions; self-efficacy alone will not sustain this spontaneous form of activity.

Studies testing CET have been done with toddlers to adults, allowing us to draw inferences about some fundamental, age-invariant aspects of intrinsic motivation. One type of evidence comes from experimental studies. In the typical paradigm children or adults work on an activity under different conditions that vary in terms of contextual factors affecting perceived autonomy, perceived competence, or both. Subsequently, intrinsic motivation is assessed by a free-choice behavioral measure. Participants are given a postexperimental period without demands, in which the target activity is available along with alternative activities (e.g., reading a magazine). The time participants spend spontaneously engaging the target activity is recorded and used as the *free-choice measure of intrinsic motivation*. Ratings of how interesting and enjoyable participants find the task are a complementary measure of intrinsic motivation.

One interesting factor affecting intrinsic motivation is external rewards. CET postulates that because rewarded participants are more likely to see the impetus or cause of their activity as stemming from external sources, when they are offered rewards for acting they are vulnerable to losing an internal perceived locus of causality and sense of autonomy. When originally proposed (Deci, 1971), this CET hypothesis was provocative because many psychologists then held a view, based in operant psychology (Skinner, 1953), that all behavior was controlled by external contingencies of reward and punishment. CET not only assumes this is not the case, but moreover suggests that some types of rewards that are very effective in shaping behavior can at the same time yield detrimental effects on intrinsic motivation, precisely because of their negative effect on autonomy. In contrast when rewards are not controlling, but rather provide more *informational* (or competence relevant) feedback, they can be more motivationally positive (Ryan, Mims, & Koestner, 1983).

Danner and Lonky (1981), combining Piagetian and CET frameworks, presented a classic study illustrating some of the developmental implications of this undermining effect of rewards on intrinsic motivation. Children were classified with regard to their level of cognitive development using a Piagetian assessment. They then worked on additional cognitive tasks either explicitly for, or without, externally offered rewards. Subsequently, children were given a free choice period in which they were allowed to select activities during a play opportunity. Children who did not receive external rewards gravitated to tasks that were optimally challenging—those stretching their assessed cognitive capacities. In contrast, children who had been externally rewarded sought easier tasks and avoided challenges during free play. These results illustrate how attempts to enhance development through controlling rewards can backfire.

Another example comes from studies of young children's intrinsic interest in helping others, which many argue is a deeply evolved human propensity (see Ryan & Hawley, in press). To demonstrate that early altruism constitutes an intrinsic or a natural inclination Warneken and Tomasello (2008) created situations where 20-month-old toddlers could help adults (e.g., pick up dropped objects). In one condition they gave children a concrete reward every time they helped, whereas children in another condition did not get rewards. In a second phase, they found that children previously rewarded for helping were less helpful than those never rewarded. Presumably, the reward undermined children's spontaneous, intrinsically motivated tendency to be helpful, a finding paralleling adult findings (e.g., Weinstein & Ryan, 2010). This experiment is notable in finding the effect in children before they can cognitively weight or discount the motives underlying their behavior, ruling out so-called overjustification explanations of this effect (e.g., Lepper, Greene, & Nisbett, 1973).

Despite a plethora of studies documenting undermining effects, debate about the undermining effect was resparked by Eisenberger and Cameron (1996). who presented meta-analyses of reward effects that seemingly contradicted previously published summaries and meta-analyses on the topic. In their analysis they surprisingly found no evidence for an undermining effect by rewards, and they labeled the undermining effect a myth. Their null findings were immediately heralded by many behaviorists. Yet detecting serious methodological problems, Deci, Koestner, and

Ryan (1999) reanalyzed these experimental data, taking care to include all studies summarized by Eisenberger and Cameron (1996). The reanalysis, presented in *Psychological Bulletin,* not only confirmed this well-known undermining of intrinsic motivation by rewards, but more importantly, supported the differentiated pattern of reward and feedback effects on intrinsic motivation predicted by CET. Data indicated that controlling reward contingencies undermined intrinsic motivation, whereas noncontrolling and informational rewards did not.

Many studies continue to document this important effect. One study of particular interest focused on neurological supports for intrinsic motivation (Murayama, Matsumoto, Izuma, & Matsumoto, 2010), providing a new way of demonstrating the undermining effect of rewards on intrinsic motivation. These researchers had participants engage in an interesting task under reward and no-reward conditions, tracking activations with fMRI. The no-reward group found the task interesting and cognitively engaging, and these inherent satisfactions were associated with meaningful striatal and lateral prefrontal cortical activity. For the reward group, Murayama et al. (2010) provided a performance-contingent monetary reward, and as predicted both striatal and lateral prefrontal cortical activity increased significantly, suggesting that the reward added to task motivation and engagement. Yet in a second phase of the study, participants repeated the task, but in this phase no rewards were offered. The researchers again examined the striatal and lateral prefrontal cortical activity the task generated. For participants in the prior no-reward condition, striatal and lateral prefrontal cortical activation were essentially stable; the task remained just as rewarding and engaging as before. Yet for those in the reward condition, striatal and lateral prefrontal cortical activity declined—the task now failed to generate pleasure (striatum) or cognitive engagement (lateral prefrontal cortical), as predicted by CET.

SDT scholars do not question the power of rewards to control behavior. However, SDT emphasizes that, at the same time, the very power of externally administered rewards can also lead individuals away from their intrinsic inclinations. Powerful extrinsic rewards, whether they be tangible rewards or conditional love, can lead people to forego autonomy, forget their values and preferences, and neglect their interests and relationships. That is precisely what unhealthy regulation and alienation is all about (Ryan & Deci, 2000a). Not only can controlling rewards undermine intrinsic motivation, so can motivational tactics such as controlling praise (e.g., Ryan, 1982); threats of punishment (e.g., Deci & Cascio, 1972); salient surveillance (e.g., Enzle & Anderson, 1993; Plant & Ryan, 1985); controlling-directive language (e.g., Hooyman, Wulf, & Lewthwaite, 2014; Reeve & Jang, 2006); and grades (e.g., Pulfrey, Buch, & Butera, 2012). These and other frequently used methods of controlling behavior can have a psychological meaning, or *functional significance*, of being controlling, which in turn negatively affects people's experience of autonomy and engagement. To the extent that adults use salient prods, prompts, or pressures to induce children to perform interesting activities, they are likely to disrupt intrinsic motivation and autonomous functioning (Assor et al., 2004).

Effects Across Developmental Periods

Although many experimental studies of intrinsic motivation have been in school and college settings, the effects of autonomy support versus control on intrinsic motivation are robust across developmental epochs and interpersonal settings. We review some examples.

Earlier we cited studies by Grolnick and colleagues (1984; 1985) in which infants and toddlers whose mothers were more controlling evidenced less mastery motivation and persistence than those whose mothers more actively supported their initiations and autonomous play. As another illustration, Grolnick et al. (2002) observed mothers and their third-grade children working together on an assigned task of problem solving and constructing poems. Following the interaction, children were asked to complete similar tasks on their own, without their mothers being present. Children whose mothers interacted with them in a more controlling manner had performed just as well as other children when with their mothers, but, when completing tasks on their own, they were less accurate in problem solving, and wrote less creative poems.

Deci, Driver, Hotchkiss, Robbins, and Wilson (1993) looked at maternal autonomy support and control with five- to seven-year-olds. They recorded mothers' vocalizations during an interactive-play period, and had raters code the interactions for autonomy supportive and controlling contents. After sessions in which mothers were rated, children's intrinsic motivation was assessed using persistence observations during solo play. Children whose mothers were rated as more autonomy supportive were more intrinsically motivated.

The significance for development of this general effect concerning autonomy support and control on intrinsic motivation cannot be overstated. Children's exploration, challenge seeking, and curiosity are inherent and natural resources for cognitive and emotional development, and a wellspring from which specific competencies derive.

Also, when led by curiosity and exploration, children gradually learn to discover their interests, a first step toward pursuing their interests and gradually building their skills. Yet this resource is curtailed by controlling environments. Intrinsic motivational tendencies that support growth can be stifled by even well meaning caretakers as they attempt to elicit development through reinforcement or control.

The significance of intrinsic motivation, and the impact of social contexts on it, has been documented in various domains, including education, exercise, and work. For example, within schools Deci, Schwartz, Sheinman, and Ryan (1981) collected teacher's reports about their classroom motivational strategies before they met their students for the year. Several weeks into the school year Deci et al. (1981) returned to assess student motivation and well-being. They found that students assigned to teachers who were oriented toward supporting autonomy displayed higher levels of curiosity, perceived cognitive competence, and self-esteem than those assigned to teachers espousing more controlling orientations. Ryan and Grolnick (1986) reported parallel results using elementary school children's perceptions of their teachers. Notably, even in an "elective" domain such as sport, O'Rorke, Smith, Small, and Cumming (2012) found that young athletes' autonomy for playing sport was related to the motivational climate created by parents. Parents focused on mastery motivation with little ego-involvement had children with higher levels of autonomy and intrinsic motivation for sport. A prospective analysis over the course of a sport season showed, in fact, that ego-involved parents had children whose autonomy for playing decreased over the season.

Indeed there is now an extensive literature that continues to document the beneficial effects of autonomy-supportive socialization, including their generalizability across cultures. For example, Chirkov and Ryan (2001), studied Russian and U.S. high school students, finding that perceived autonomy support from parents and teachers was associated with more internalized academic motivation and better mental health in both samples, to comparable degrees. Jang, Reeve, Ryan, and Kim (2009), in a prospective study of South Korean high school students found that teacher autonomy support predicted greater intrinsic motivation in school. Jang, Kim, and Reeve (2012) extended these findings in a multiwave longitudinal study of more than 500 South Korean students, testing a complex path model. As expected, teacher autonomy support enhanced student need satisfaction and engagement, resulting in better achievement. But reciprocal effects were also apparent, such that when students' need satisfaction was high, teachers were influenced to be less controlling. This kind of longitudinal approach reveals the complex dynamic effects between autonomy support and autonomous motivation, which are truly interpersonal and in which influences can be reciprocal.

Intrinsic motivation is quite contextually sensitive, fluctuating over time with perceived autonomy support. For example, Tsai, Kunter, Ludtke, Trautwein, and Ryan (2008) showed that German public school students, followed over multiple days in three different required courses, showed considerable lesson-to-lesson variability in interest. Yet the fluctuations were systematic: they were directly associated with variability in teacher autonomy support. On days when the teacher was more controlling students' interest waned. These findings were extended by Mouratidis, Vansteenkiste, Lens, and Sideridis (2011). Rather than following daily variations in perceived teaching behavior, they instructed teachers to randomly build in autonomy supportive segments within their more typical teaching approaches. In classes where the strategies were inserted adolescents reported enjoying the class more and feeling more vital and energized. Moreover, these effects from inserting autonomy-supportive strategies were specifically accounted for by students' reports of autonomy need satisfaction.

Factors that support a person's autonomy can, of course, enhance interest and intrinsic motivation. For example, a meta-analysis by Patall, Cooper, and Robinson (2008) showed that in general the affordance of meaningful choices to people has an enhancing effect on intrinsic motivation (see also Katz & Assor, 2007). Autonomy support is also conveyed by taking the person's *internal frame of reference*, or understanding their point of view (Deci & Ryan, 1985b; Koestner, et al., 1984). In fact, Patall, Dent, Oyer, and Wynn (2012) found that teachers' perspective taking and provision of choice were both autonomy-enhancing factors for high school students. Such studies indicate that providing choice and acknowledging perspectives can enhance intrinsic motivation, as these events leave individuals feeling more autonomous.

Summary

Experiments and field studies in diverse domains, including parenting, teaching, and coaching, suggest that social contexts that are experienced as *controlling* (i.e., as pressure to think, feel, or behave in specific ways) undermine autonomy, resulting in diminished intrinsic motivation and more negative self-related emotions. In contrast, contexts that are *autonomy supportive* (i.e., that consider

the individual's frame of reference, provide choice, and encourage self-initiation) have positive effects on intrinsic motivation and persistence.

Internalization: Assimilating Social Regulations and Values

The Internalization Continuum

Intrinsic motivation is a prototype of autonomous motivation that occurs spontaneously, as an expression of our active, assimilative human nature. Many behaviors, however are not intrinsically interesting, but parents, school systems, or other authorities believe them to be in children's best interests and therefore important for them to acquire. Although parents and teachers, as primary socializing agents, attempt to motivate children to perform such activities, the methods they use vary. Some may be controlling and authoritarian, forcing the child to conform to norms or expectations; others may be autonomy supportive, influencing through reasoning, modeling, and guidance of choices. SDT predicts that whereas controlling methods foster at best short-term compliance, autonomy support, structure, and involvement in conjunction enhance the *internalization* and *integration* of behavioral regulations.

Ryan, Connell, and Deci (1985) defined internalization within SDT as the means through which individuals can become more autonomous in performing an activity that was initially externally prompted. In this view internalization is an active process of selecting and transforming ambient regulations into personally endorsed values or motivational propensities. Through internalization, people acquire values and behaviors that allow them to be effective, connect with others, and experience volition—that is, to get their psychological needs met. When functioning optimally, the internalization process results in values and regulations that are more self-endorsed, that is, are fully integrated within the self.

SDT further hypothesizes that supports for autonomy, competence, and relatedness are necessary for such optimal, integrated internalization (Deci & Ryan, 2000; Ryan, 1993). Unfortunately, these ideal contextual conditions are not widely prevalent, and full integration of regulations is not often attained. Instead, when socializing agents are controlling or uninvolved, internalization results in values and regulations that are only poorly internalized. SDT therefore places importance on distinguishing between types of internalization for predicting the nature and quality of behavioral and psychological functioning. Specifically, SDT specifies a continuum of regulatory styles that spans from low to high autonomy for acting.

At the low end is *external* regulation, which describes extrinsically motivated behavior that is initiated and maintained by external rewards or punishment avoidance. A child who does chores to avoid parental nagging would be externally regulated, as would one who does them to get tangibly rewarded. External regulation is characterized by a lack of internalization, and although external rewards and punishments can be powerful motivators, externally regulated behavior is poorly maintained in the absence of direct control.

Somewhat more autonomous is regulation based on *introjection*. Introjected regulation is motivated by feelings of self-worth and loveability for acting as one should, and guilt or self-disparagement for failing to meet the standards or goals. Introjected regulation is thus often rigid and accompanied by feelings of internal pressure and tension. A child who does chores because she feels pressured to avoid feeling guilty and to feel like a "good girl" is displaying introjection. Indeed, introjection supplies the developmental basis for *internally controlling* regulation, as exemplified in ego involvement (Ryan, 1982), contingent self-worth (Deci & Ryan, 1995; Kernis, 2003), and self-critical perfectionism (Frost et al., 1993; Soenens, Vansteenkiste, Duriez, Luyten, & Goossens, 2005). Accordingly, research has related introjection to rigidity, anxiety, self-derogation, and other maladaptive patterns (e.g., Assor, Vansteenkiste, & Kaplan, 2009; Weinstein, Deci, & Ryan, 2011).

Still somewhat higher on this continuum of autonomy is *identified regulation* of behavior. In this type of regulation the person consciously identifies with a value or behavioral goal—accepting it as his or her own. It is therefore enacted with less resistance and conflict, and often with persistence, diligence, and efforts based in valuing. Children who clean their rooms because they understand that a clean room allows them to more easily find what they need have concurred with the importance of cleaning, that is, they have more fully internalized the value and reason for cleaning. Finally, *integrated* regulation represents the most autonomous form of internalized regulation, as it indicates that an identified value and its accompanying regulatory process have been reciprocally assimilated with other values and important personal goals. Various identified values may be more or less challenging to integrate, but if integrated, are wholeheartedly endorsed and performed.

These four modes of extrinsic regulation lie along a continuum of relative internalization and autonomy, where external regulation represents the least internalized and autonomous mode and integrated regulation represents the most internalized and autonomous mode of extrinsically

motivated behavior. Intrinsic motivation is also on the high autonomy end of this dimension, yet does not require internalization of regulation as the behavior already spontaneously manifests when people act out of enjoyment and interest. Considerable evidence backs up the continuum nature of this array. Ryan and Connell (1989) provided the initial studies assessing children's external, introjected, identified and intrinsic regulatory styles, and their data confirmed that these were reliably ordered along a continuum of increasing autonomy. Moreover, the more autonomous forms of regulation (identified and intrinsic) were positively correlated with positive affect and proactive coping, whereas the less autonomous forms (external and introjected) were correlated with negative affect and maladaptive coping. Introjection, in particular, was associated with anxiety and anxiety amplification following failure, highlighting the inner stress and tension associated with this internally controlling form of regulation.

Enhancing internalization and integration is a valuable goal in its own right, yet when students have more autonomous forms of motivation they also tend to more deeply process the learning material (Sobral 2004; Yamauchi & Tanaka, 1998), to be more fully absorbed in the task at hand (e.g., Abuhamdeh & Csikszentmihalyi, 2012), and to perform better. For example, De Naeghel, Van Keer, Vansteenkiste, and Rosseel (2012) examined elementary students' motives for reading, both in school and in leisure time. They found that more internalized, autonomous motives not only related positively to teacher-rated engagement in reading, but also to children's scores on a standardized reading comprehension test. At the other end of educational development, Kusurkar, TenCate, Vos, Westers, and Croiset (2013) found that autonomous motivation is positively associated with the use of a deeper study strategy and better GPA in medical students. In fact, a recent meta-analysis by Cerasoli, Nicklin, and Ford (2014) supports this pattern of findings, with intrinsic/autonomous motivation predicting both better quality and quantity performance, while incentives related only to better quantity performance.

The relations of internalization and autonomy to higher quality behavior are not limited to learning and achievement domains. For example, Ryan and Connell (1989) found that more autonomous regulation in the prosocial domain was associated with greater empathy, more mature moral reasoning, and more relatedness. Blais, Sabourin, Boucher, and Vallerand (1990) assessed adults' reasons for maintaining their primary relationship, finding that regulatory styles representing lower autonomy were negatively associated with dyadic adjustment and relationship satisfaction. Ryan, Rigby, and King (1993) examined the internalization of religious values, finding that introjected regulation of religious behaviors was positively associated with symptoms of depression, anxiety, and low self-esteem, whereas identified regulation was negatively associated with these indicators of ill-being. Neyrinck, Vansteenkiste, Lens, Hutsebaurt, and Duriez (2006) found that greater internalization of religious activities was associated with a more open and flexible interpretation of doctrines, stronger adherence to beliefs, and greater well-being. In the health care domain, Williams, Rodin, Ryan, Grolnick, and Deci (1998) showed that patients who had more strongly internalized the self-importance of taking their medication were more likely to effectively take them. Finally, although parents constitute primary socialization figures in the transmission of values, only the autonomous agreement with parental values relates positively to well-being, even after for controlling for the degree of parent-child agreement as such (Knafo & Assor, 2007). Across these domains and others, findings consistently show that the more autonomous people's motives, the more effective their functioning and psychological experiences.

Social Contexts and Internalization

Given these benefits of fuller internalization, the developmental conditions that foster or facilitate internalization and autonomous regulation are an important focus of research. Although like intrinsic motivation, internalization is hypothesized within SDT to be a natural process (Ryan & Hawley, in press), it too requires specific nutrients and supports from the social context. In fact, all three needs are critical to foster the full integration of socially prescribed norms and values. That is, a sense of competence is an important yet not sufficient condition for fuller internalization of an activity, as one can feel fairly competent, yet externally pressured to do the activity. Similarly, a sense of relatedness is conducive to taking in the behavioral regulation, yet a sense of internal conflict can still be underlying the behavior if it is emitted to avoid feelings of disloyalty and guilt vis-à-vis the socialization figure. For full internalization to occur, a sense of autonomy is critical. Consistent with this idea, Markland and Tobin (2010) showed that the satisfaction of the three needs maps in theoretically meaningful ways to the different types of regulation for exercising. Whereas relatedness satisfaction yielded a unique positive association with introjected regulation, it is only when all three needs were fulfilled, that the exercisers identified with the importance of exercising.

To promote autonomy need satisfaction and subsequent internalization, the use of control has been found

to be harmful, whereas provision of autonomy support has been shown to be critical, a finding that generalizes across cultures (e.g., Ahmad, Vansteenkiste, & Soenens, 2013; Barber, Stolz, & Olsen, 2005; Chirkov et al., 2011; Jang et al., 2009). There are many elements to autonomy support, but they all start from an empathic grasping of an individual's perspective so as to better understand why the person refuses to engage in the noninteresting activity. By taking the frame of reference of that person, the socializing agents can provide a more relevant and meaningful rationale, provide additional competence-enhancing information, if needed, and offer choices concerning how and when an activity is carried out. Along with support for relatedness, provision of such autonomy support and structure deepens internalization and contributes to social integration and wellness.

The critical role of need-supportive socialization for children's internalization has been documented in dozens of studies. For instance, in the study by Grolnick and Ryan (1989), parents who were rated as more autonomy supportive from interviews had children whose motivation for schoolwork was more fully internalized. Notably, many parents who were rated as controlling were clearly involved in their children's lives, and many were well intended, some highly invested in their children's success (Wuyts et al., in press). Yet in attempting to control the children to ensure that they attained certain outcomes, they often failed to promote their children's ownership of these goals or the children's personal investment in achievement, instead creating a reliance on external regulation.

As noted, the practice of *parental conditional regard* (PCR), or the degree to which a parent shows more attention and affection after compliance or success, and less affection and attention after noncompliance or failure, has received increasing recent attention. For instance, Assor et al. (2004), studying four domains (i.e., academics, sports, prosocial behavior, emotion regulation), showed that student perceptions of PCR, assessed separately for mothers and fathers, predicted compliant behavior that was largely based in introjection and inner compulsion. PCR appeared also negatively associated with identification and feeling a sense of choice about the activities, whereas PCR was positively related to contingent self-esteem, shame, guilt after failure, and short-lived satisfaction after success—all variables one would expect to be associated with introjection. Most tellingly, students who experienced more PCR also reported more anger and resentment toward their parents.

Interestingly, the negative repercussion of PCR may not only manifest at the personal, but also at the interpersonal level. Moller, Roth, Niemiec, and Deci (2014) found that late adolescents who experienced parents as being higher on PCR not only reported less secure attachments with them; they also reported more conditional regard and less secure attachments with their current romantic partners and best friends. The experience of being conditionally regarded thus appears to generalize from experiences of parents to other close relationships. Along similar lines, Soenens, Vansteenkiste, Goossens, Duriez, and Niemiec (2008) found that parental psychological control, which compromises conditional regard as one of its central components, was associated with more physical aggression and poorer peer relationships

Conditional regard, as a form of internally controlling parenting, can be differently valenced—it can take a more seductive form of *positive parental conditional regard,* involving the provision of more attention and affection than usual in case of desirable child behavior, and a more punitive form of *negative conditional regard,* involving the withholding or even withdrawal of attention and affection in case of undesirable child behavior. Although most theorists might expect negative PCR to be problematic, some approaches (e.g., many social learning and cognitive approaches) might argue that positive PCR is an effective form of parenting. Indeed, some theorists appear to justify and even advocate tiger mom tactics that involve the display of PCR (e.g., Fu & Markus, 2014). Unfortunately, it appears that also positive PCR comes with a cost. For instance, Roth, Assor, Niemiec, Ryan, and Deci (2009) showed that parents using positive PCR to promote academic success had adolescents who reported internal compulsion to do school work rather than a sense of autonomy, and their teachers reported them to be grade-focused. In addition, extending Assor et al.'s (2004) findings, these adolescents reported greater resentment toward parents. Negative PCR was also related to resentment of parents, and was related negatively with school engagement. Thus negative PCR does not promote internalization (not even introjection) although years earlier Sears et al. (1957) had proposed that it would. Most likely the parents who used love withdrawal in their samples also used positive conditional regard, and it may have been the positive PCR that led to their proposal. In a second study by Roth et al. (2009), positive PCR was compared to autonomy support as parenting methods for children's school achievement. Results showed that both mothers' and fathers' positive PCR were positively related to the child exhibiting grade-focused engagement, and negatively to interest-focused engagement. Further, these relations were mediated by the experience of internal compulsion

(i.e., introjection), as predicted. In contrast, autonomy support from each parent predicted interest-focused engagement, a relation that was mediated by a sense of choice.

While internally controlling strategies, such as conditional regard, may elicit partial internalization of parental demands through introjection, parents' reliance on external controls is likely to merely elicit external regulation, representing a complete lack of internalization of the behavioral regulation. Even more, in response to external controls, children may be noncompliant or even reactive. In an illustrative observational study among Mexican children, Brunstein (1994) showed that parents' engagement in externally controlling practices (e.g., orders, reprimands, and threats) related positively to both children's observed inattentive obedience and their passive and rather blunt resistance to comply with parents' requests. More recently, Talwar and Lee (2011) conducted a field experiment comparing two West African schools, which strongly varied in terms of their use of physical punishment as a discipline practice. Specifically, 3–4-year-olds of the one school, who were very frequently exposed to the physical punishment, were 12 times more likely to lie during a toy game and to conceal their lies.

Thus, for socializing agents to promote internalization and ownership of regulations, a controlling approach would best be avoided. Yet the absence of control does not imply the absence of guidance, as would be the case with a laissez-faire climate. What is critical for children to begin to internalize societal expectations is that prescribed behaviors and limits need to be clearly communicated; in other words, sufficient structure and guidance need to be provided. Furthermore, in communicating and monitoring these limits, an autonomy-supportive style is essential.

Consistent with this, Vansteenkiste, et al. (2014) showed that the style of introducing prohibitions to adolescents affects their acceptance of (i.e., internalization) versus oppositional defiance against the prohibitions. Specifically, their one-year longitudinal study revealed that a more autonomy-supportive style of introducing and monitoring prohibitions elicited greater identification with and ownership of the limits, while a perceived controlling style predicted either increases in externally enforced compliance with the prohibitions or oppositional defiance against them. Notably, these effects were found to be reciprocal such that a virtuous positive cycle unfolded when autonomy support was salient, and a vicious negative cycle was instigated in the case of a controlling style. These effects occurred regardless of whether the prohibitions had a more personal character (e.g., friendships) or were moral in nature.

Summary

A crucial task for socializing agents is to transmit norms, values, and regulations, such that children and adolescents will not stick to these norms out of strict, slavish obedience but rather out of volitional commitment, presumably because they have come to fully own (i.e., internalize) the norms. Whereas controlling socialization practices, such as positive PCR from parents, can produce compliant behavior, it is often at the cost of a sense of volition to behave. Negative PCR and more externally controlling practices may even be more counterproductive, eliciting at best externally driven obedience, and, at worst, anger and resentment as well as blunt defiance against requested norms. What is needed to foster full internalization of norms is an autonomy-supportive approach in combination with clear communication of expectations (structure) and involved guidance.

Emotion Regulation

Distinguishing Different Styles of Emotion Regulation

Similar to the process of internalization, the development of emotional self-regulation can be conceptualized as movement from reliance on outside sources to identify, modulate, and regulate affect, toward a growing capacity for autonomous, flexible, and adaptive regulation (Cicchetti, Ganiban, & Barnett, 1991; Grolnick et al., 1996). Deci and Ryan (1985b) early on drew from Greenspan (1979) in referring to the development of regulatory processes for autonomously managing emotions as *integration at the internal boundary*. This was contrasted with the development of regulatory processes for engaging in behaviors deemed important by the social world as *integration at the external boundary*. Although these two developmental functions, referred to, respectively, as the development of emotion regulation and internalization, are distinct, they are similar in many respects. Both involve gaining the capacities to regulate oneself with respect to behavior that is not intrinsically motivated and both entail a developmental progression in which the child gradually relies less on cues and structures from the social context and more on internal cues and structures. Equally clear, deficits in the regulation of emotion and the ability to use emotions to appropriately inform or guide behavior are cardinal features of most forms of psychopathology. Accordingly, we turn now to this critical issue of emotional regulation and its healthy development.

We begin with acknowledging the complexity of emotions, and the multiple dimensions that impact upon their regulation and expression (see Cacioppo, Bernston,

Sheridan, & McClintock, 2000). In particular, there are salient individual differences in people's tendencies to experience emotions (e.g., Gable, Reis, & Elliot, 2000) based on both genetic and temperament-based factors and their interactions with early environmental events. Individual difference factors make the problem of emotional regulation and integration much more formidable for some persons, whose sensitivity or lack thereof make the task of accessing, modulating, expressing, and using emotions much more challenging (Assor et al., 2004). Nonetheless, any reasonable summary of that complex literature makes clear that even given such individual differences or tendencies, social environments play a significant and interactive role in fostering healthy emotion regulation (Vansteenkiste & Ryan, 2013). To more concretely understand what it is about social environments that can facilitate versus debilitate a growing person's capacities for emotional awareness and regulation first requires a clear conception of what healthy emotion regulation involves.

SDT's conceptualization of healthy emotional regulation begins with the assumption that emotions are *informational* inputs essential to guiding action and growth (Deci & Ryan, 1985b). In this view, specific emotions are neither good nor bad; they simply provide organismically valuable guidance (Deci & Ryan, 2000; Parrott, 1993). A similar view is expressed by Levenson (1999), who suggested that emotions provide organisms with a built-in feedback system that signal them to inspect, explore and evaluate their environment and act in accord with that evaluation.

SDT views wellness in terms of a person being fully functioning—being able to exercise potentials, connect with others, find meaning, and experience vitality. This *eudaimonic* view (Ryan, Curren, & Deci, 2013) suggests that rich access to both positive and negative feelings, a capacity to express them, and the ability to integrate and use them to inform one's behaviors and goals is optimal. This in turn supports personal growth, self-acceptance, and abilities to intimately connect with others.

To capture this quality of emotional regulation we distinguish *emotional integration* as an autonomous form of emotional regulation from notions of *emotional control* and *emotional dysregulation*. Specifically, emotional integration involves a differentiated awareness of one's emotional states and the capacity to use this sensitivity and awareness in the volitional regulation of action. Autonomous behavioral functioning is dependent on emotional integration, because emotions, when openly received, supply essential information that guides one in setting goals and adjusting them toward the fulfillment of psychological needs (Ryan et. al 2006). Healthy emotional integration is thus characterized by an allowing of and interest in inner experience, rather than the control or stifling of emotions (Deci, Ryan, Schultz, & Niemiec, in press).

Yet emotions can be experienced as more than just information; they can be experienced as evaluative and controlling, as when affect overwhelms self-regulation or automatically drives action. Even the etymology of the term emotion conveys this idea of a force that propels motion. Alternatively, emotions can be so overcontrolled such that people lose access to them, and in such cases the emotions can fail to move the individuals when they should. Controlled emotional regulation is manifest through the ignorance, suppression, or denial of emotions and, as we will see later, is implicated in specific forms of psychopathology and ill-being. Accordingly any conception of healthy or integrated emotional regulation is based neither on emotions controlling people nor on people controlling (e.g., suppressing) their emotions. When the self can access feelings without being controlled by them, the opportunity for autonomous self-regulation is maximized. This of course is a capacity that only develops over time and that requires contextual supports, in the form of guidance and need satisfaction.

Indeed, the process of increasingly integrating one's emotions and regulatory capacities is a continuation of the work a child does in gaining regulatory capacities with respect to emotions, but it represents a mature version of that process. Whereas emotional regulation is concerned with the modulation of emotional experience and expression, emotional integration is concerned with the flexibility and choice people feel in the regulation of emotions and emotion-related actions. Individuals high in emotional integration are more likely to be receptive to emotional signals, to take interest in them, and to experience them as valuable inputs to actions. In contrast, if regulatory processes are rigidly introjected and thus in conflict with the emotions, people are likely to suppress the feelings and thus ignore their personal meaning.

Consistent with the idea that integrative emotional regulation allows for a more open and nondefensive processing of emotion-related stimuli, Roth et al. (in press) showed that, after having seen a fear-eliciting movie, individuals high in emotional integration were more likely to make use of self-referenced words, to make use of causal language and to write in the past tense, all indicative of a more open and full processing of the emotional event. In contrast, individuals high in suppressive regulation made use of fewer self-referenced and past tense wording, while at the same time using less negative emotional words, all indicative of the avoidance and lack of processing of the negative event.

This capacity for integrative emotion regulation of one's fear and sadness has also been found to promote a greater ability to support a romantic partner's difficulties in handling negative emotions (Roth & Assor, 2012). In contrast, suppressive regulation or dysregulation of fear and sadness was found to compromise individuals' capacity for intimacy. Individuals who tended to suppress fears and sadness reported more difficulties in disclosing negative emotions vis-à-vis their romantic partner, as well as feeling less capable of providing emotional support to their partner. Individuals who tended toward emotion dysregulation equally reported being less able to support their partner emotionally, presumably because they were so overwhelmed by their own negative emotions. Brenning, Soenens, Van Petegem, and Vansteenkiste (in press) further showed that self-esteem is affected by emotion regulation. They reported that increases in integrative regulation covaried with increases in self-esteem among young adolescents over a one-year interval. In contrast, increased use of suppressive regulation predicted increases in depressive symptoms.

Autonomy as an Antecedent of Emotional Integration

A number of studies have examined whether greater autonomy would be associated with more effective emotion regulation, and better long-term coping with negative events. For instance, Weinstein and Hodgins (2009) had participants view an emotionally distressing movie twice, at least a day apart. The film was a graphic Hiroshima–Nagasaki documentary that was challenging to assimilate. In an initial study they showed that participants with greater trait autonomy, when given an opportunity to express emotional reactions, later had more positive well-being, energy, and memory after the second viewing. In a second study they created situational motivation by priming autonomy and control. Results showed that dispositionally controlled individuals received the same benefits as autonomous individuals, but only when primed with autonomy and encouraged to express emotions. Weinstein and Hodgins (2009) coded the contents of the emotion expressions and found that the positive outcomes associated with autonomy were mediated by nondefensive processing, as reflected in varied indices such as more self-referencing, more words reflecting internal cognitive processes, and lower use of concrete words. In short, their results suggest that autonomy facilitates ownership and expression of feelings, enhancing emotional integration over time.

Koestner, Bernieri, and Zuckerman (1992) explored the relation of autonomy to the integration and synchrony of emotions, cognitions, and behavior. They separated participants into a more autonomy-oriented group and a more control-oriented one (Deci & Ryan, 1985a) and then explored the consistency of behaviors, attitudes, and traits within each of these two groups. In the first of two experiments, they found high correlations between behavioral persistence and self-reports of intrinsic interest within the autonomy-oriented group, but no significant correlations within the control-oriented group, suggesting less congruence in the latter. In a second study, Koestner et al. (1992) had participants complete a measure of conscientiousness, and then gave them an opportunity to behave conscientiously in succeeding days. Participants' self-reported conscientiousness and their actual conscientious behaviors were significantly more highly correlated within the autonomous group than the controlled group. Self-ratings and peer ratings of conscientiousness were also more highly correlated for the autonomy oriented. Together these studies provide support for the proposition that autonomy is associated with greater congruence among traits, behaviors, and feelings, reflecting greater integration in personality functioning.

Related to the issue of emotion regulation and integration is that of people's *open* versus *defensive* functioning. Openness versus defense can be manifest in various ways, but in social interactions it entails being honest, transparent, trusting and receptive, whereas defense in contrast entails concealment, distrust, and often self-distortion of experience (Hodgins & Knee, 2002). Investigating openness versus defensiveness using a diary methodology, Hodgins et al. (1996) found that people with a more autonomous orientation evidenced more disclosure, trust in others, and honesty in their daily interpersonal interactions, whereas those with a controlled orientation showed lower honesty and openness.

Another indicator of openness versus defensiveness concerns the alignment or *congruence* between implicit and explicit attitudes and motives. Thrash and Elliot (2002) found that persons higher in trait autonomy had lower discrepancies between implicit (projective) and explicit (self-report) assessments of achievement motivation. Hodgins, Brown, and Carver (2007) found that control-primed individuals evidenced greater discrepancies between explicit and implicit self-esteem. Weinstein, Ryan, and colleagues (2012) found that children of controlling parents showed a greater discrepancy between their implicitly assessed sexual attractions to same sex others and their self-reports of sexual orientation. This discrepancy, in turn, related to greater hostility toward gay targets, indicative of *reaction formation* (Freud, 1966). In other words,

parental autonomy thwarting can foster incongruence, and the defensive behaviors it spawns. Indeed, the mere priming of a controlled orientation has been sufficient to elicit defensive responding, as indexed by the use of hostile humor (Weinstein, Hodgins, & Ostvik-White, 2011), the avoidance of negative experiences (Hodgins, Yacko, & Gottlieb, 2006), or avoidance of emotionally distressing information (Weinstein, Deci, et al., 2011; Weinstein & Hodgins, 2009). Across these studies of emotional integration and congruence it seems clear that controlled people show less integrity, whereas autonomy is conducive to more integration in both attitudes and less defensive and more open functioning.

Social Contexts: Developing Emotional Integration

The development of capacities to regulate and integrate emotions in flexible and autonomous ways is assumed within SDT to be supported by responsive, need supportive caregivers. Grolnick, Bridges, and Connell (1996) explored this with 12- to 32-month-old children. They observed toddlers in two stressful situations: waiting to play with an attractive toy (or to eat a food) and having a brief separation from mother. The children's emotionality (upset) and strategies to regulate this upset were coded. Results supported an autonomy continuum in that the more autonomous strategies were associated with the least upset, whereas the more controlled or passive strategies were associated with the most upset. Further, there were increases with age in the use of autonomous, proactive strategies, indicating development of emotion regulation through these toddler years.

As noted, the regulation of emotions and impulses can be done in a fairly controlling way. Some children, when in a negatively arousing situation such as being asked to delay gratification, exert great effort, forcing themselves to push emotions out of their minds. But controlling themselves in this way requires attention and energy, which can diminish the capacity for adaptive engagement with the environment. When used as a chronic way to deal with distress, it leads children to experience being controlled by their own harsh thoughts and to display nonadaptive engagement. In contrast, other children in the same stressful settings may be more flexible (i.e., less rigid and pressured) in dealing with the situation, for example by doing alternative activities or expressing their disappointment or frustration, signifying that they have developed more adaptive capacities for both accessing and regulating emotion.

This developing capacity for emotional regulation is not just a function of time and maturation. In fact, it is very much an *interpersonal* process. It is in the interpersonal realm, within which caregivers regulate children's expressed emotions more or less responsively and effectively, that children learn to regulate their own emotions. Autonomy-supportive interpersonal contexts are theorized within SDT to afford access to and expression of emotional experience, and to facilitate more reflective engagement and executive guidance of subsequent behavioral regulation (Grolnick et al., 1996; Gross & John, 2002). Calkins and Johnson (1998) found evidence for this in demonstrating that mothers' use of positive guidance (akin to an autonomy-support approach) was associated with greater use of distraction and constructive coping in emotion inducing situations. On the other hand, children of mothers who used high levels of negative control during free play spent more time orienting to a desired but forbidden stimulus. They used less self-distraction and were less physiologically well regulated during a waiting situation relative to those of mothers who used less negative control.

Grolnick, Kurowski, McMenamy, Rivkin, and Bridges (1998) also examined strategies mothers use to help their young children regulate distress. Children of mothers who maintained their active assistance beyond what the children needed were less able to regulate their distress when on their own. Thus, while responsiveness to distress was important, parents who took responsibility for regulating children's distress and did not allow the children opportunities to self-regulate appeared to undermine children's self-regulatory capacities. Because the capacity to effectively modulate distress is so integral to mental health and the prevention of psychopathology, an environment supporting this capacity is of crucial importance.

More recently, Roth, Asssor, et al. (2009) showed in a sample of ninth-grade adolescents that parents who made use of conditional regard when adolescents expressed negative emotions (e.g., showing less acceptance and positive regard) had children who more frequently engaged in *suppressive regulation* of emotions, reported greater dysregulation of emotions and more resentment vis-à-vis the parents. In contrast, both mother and father autonomy support related to children showing greater *integrative regulation* of negative emotions. These adolescents allowed their emotions to be more fully experienced and could then, with a greater sense of choice, decide whether and how to express them. Along similar lines, Roth, Ron, and Benita (2009) showed that parents' use of conditional regard related to reduced disclosure of difficulties at school among 14–16-year-old teenagers. Presumably, with contingently regarding parents, children tend to keep the encountered problems for themselves rather than opening up, as they anticipate critical reactions from their

mothers. A longitudinal study of adolescents by Brenning et al. (in press) further supported these associations. They found that parental autonomy support predicted increases in emotional integration and decreases in suppressive emotion regulation, whereas higher levels of emotional dysregulation were related to lower perception of parental autonomy support.

Further, Roth and Assor (2010) showed that the costs of a controlling parenting approach are apparent by the ages of five or six. Parents reported the degree to which they used both conditional regard with their children to socialize them regarding regulation of sad emotions. These parent reports were then related to various assessments of their children's emotional skills. Results indicated that parental use of conditional regard was related to children being less aware of sad feelings in themselves, less able to recognize sad feelings in other children, and less empathic in responding to other children who felt sad.

Although parents' display of contingent regard for showing negative emotions may lead them to suppress their negative emotions, it is also often the case that some parents may pressure their children to express (rather than withhold) negative emotions (Roth & Assor, 2012). Although such a practice may seem benign at first sight because negative emotions get shared, the pressure to share negative emotions to gain parental appreciation may come at the cost of adequate emotion regulation. Consistent with this, late adolescents experiencing conditional regard to express negative emotions reported more emotional dysregulation, which hampered their capacity for intimate relations with romantic partners. In contrast, an autonomy-supportive approach was related to more integrative emotion regulation.

Summary

Developing the capacities for respecting others and delaying gratification represents a central agenda from toddlerhood through adulthood; it involves learning when and how it is reasonable and appropriate to express one's feelings and being able to use that information in a way that enhances the autonomous regulation of behavior. Thus, emotional regulation entails children internalizing caregiver-provided values and regulatory structures to manage their internal urges—a process that, similar to the internalization of behavioral regulations for uninteresting tasks, is facilitated by supports for the children's basic psychological needs. Although differences in autonomous functioning contribute to a greater congruence between felt emotions, held thoughts, and enacted behaviors, differences in contextual need support also played a role. Previous studies suggest that controlling parental practices regarding emotions, whether involving pressure to keep negative emotions to oneself or to express them before one is ready to do so, come with costs. In contrast, need-supportive parents respect the children's rhythm and pace of expressing emotions, displaying an active interest and concern with what is emotionally salient for them. They also set appropriate limits on behavior while allowing adequate expression of feelings. This facilitates the children's attunement to the social world, and acceptance of self in the process of adapting to it.

Identity Formation

An SDT View of Identity and Self

Identity formation is among the most formidable of developmental challenges (Kroger & Marcia, 2011; Soenens & Vansteenkiste, 2011). This is especially so in modern cultures within which apparent options for identities are greater than ever before and in which there are fewer of the explicit vertical constraints that have historically channeled people into preordained roles and identities (Ryan & Deci, 2011). For many scholars this fluidity and opportunity for choice represents a potential for persons to be better matched in their life roles with their interests and sensibilities (see Appiah, 2005), whereas other scholars emphasize the psychological burdens that such latitude and choice present to developing individuals (e.g., Schwartz, 2000). Both positions have substantial evidence in their favor.

Identities are self-representations—they are what and who people understand and describe themselves to be. Once adopted, maintaining and defending identities can be a strong motivator of behaviors, and can orient people's learning, behaviors, and friendships (Vallerand, 2010). Different types of identities can be distinguished (Vignoles, Schwartz, & Luyckx, 2011), with some being more personal (e.g., personal interests), others being social in nature (e.g., roles), and still others being collective in nature, thereby reflecting the groups and institutions with which we are affiliated, and supplying varied venues through which people can be recognized and valued.

Particularly during adolescent and young adult years, a challenging task is to form a meaningful identity that will serve to organize and guide an adult life. This is a period when people are particularly vulnerable to external forces. With the emergence of adolescent egocentrism and self-consciousness (Elkind, 1967), teens tend to see themselves in ways they imagine others see them, and they show a greater tendency to conform in an effort to be accepted by others. Thus, as they are attempting to formulate a sense

of their own identity, they are also struggling with real or imagined social pressures from many sources including, not only peers but also parents, teachers, role models, and advertisers. As adolescents develop they must work to explore, try on, and accept or reject possible identities, sometimes painfully. During this process, pressures from parents and other socializers can detract the young people from smooth identity formation insofar as the offered identities are incongruent with their developing and abiding interests and sensibilities. Indeed, all people, but perhaps especially adolescents, are today exposed to the wide reach of media, which attempts to orient them toward particular types of identities and affiliations (Dittmar, 2011; Kasser, 2002). Advertisers work to have people define themselves in terms of the brands they purchase and the media cultures they engage.

Central to the process of forming identities is people's inherent desire to experience solid connectedness to individuals, groups, or cultures. By accepting the values, mores, missions, attitudes, and behaviors of others, people do not feel alone. Rather, they feel part of a group, experiencing a sense of relatedness to others and a place in the social order (Baumeister & Leary, 1995; Ryan, 1993). Further, identities can also support the need for competence. Often people orient toward identities—perhaps being a painter or physicist, for example—that require skill acquisition, offer optimal challenges, and allow them to feel effective. Finally, the formation of identities can fulfill people's need for autonomy if they engage the relevant activities with a sense of choice and as an expression of their own values and interests. In short, people tend to gravitate toward those identities that allow maximal satisfaction of their basic psychological needs (Luyckx, Vansteenkiste, Duriez, & Goossens, 2009).

Need satisfaction then serves as the energetic basis for anchoring identities within one's sense of self. Thus, although the terms *identity* and *self* are often used interchangeably in the identity literature, they get clearly differentiated within the SDT perspective. That is, although we have multiple identities, each is more or less internalized and integrated within the self (Soenens & Vansteenkiste, 2011; Ryan & Deci, 2011).

Anchoring Identities Within the Self

SDT suggests that there are two interrelated means through which identities form and become integrated to different degrees within people's self-functioning: (1) through the discovery and differentiation of intrinsic interests and talents; and (2) through the internalization of values and self-representations.

People manifest individual differences in their competencies, interests, and inclinations. Some children seem inclined toward music, others toward physical activity. The forms that such inclinations take in people's lives is a function of their interactions with the social world, but having the inclinations—for music, or sports, for example—does increase the likelihood that related activities will become important parts of identity (Deci & Ryan, 1985b). Their inclinations lead them to explore different options and, over time, to gravitate toward others with similar interests, such that the activities, values, and relationships associated with those interests become central aspects of their lives and come to define who they are. They also are likely to experience greater competence, further enhancing their interests in the activities. In other words, people are fortunate when they adopt identities that engage intrinsic motivation (Krapp, 2002).

Still, for identities to develop out of intrinsic interests often requires both enduring affordances and social supports. A child who is naturally inclined to be physically active but who has no access or opportunities to play sports might not develop an identity as an athlete. In short, the development of an identity depends to some extent on the cultural affordances available to the person (Erikson, 1994). Further, to form an identity as an athlete will require the experience of need supports in relation to the engagement of sport activities. Supports for autonomy, competence development, and relatedness in sport will be critical in facilitating identity formation as an athlete. This is the same for identities in every domain, whether it be academic, religious, family, or career related.

Few identities are sustained solely out of intrinsic interests. Instead most are taken on or elaborated through processes of internalization. Importantly, however, much like behavioral regulations, identities can differ in the degree to which they are integrated with the self. When identities are adopted within the context of pressure and control, it is likely that they will be introjected. That is, people will take them on as "shoulds," or as identities they must enact, which essentially means that they will be identity deficits. Often in college settings, for example, there are individuals who identify as pre-med because they have always been told they will be a doctor, regardless of their interests or predilections. In fact, the students' sense of worth and esteem may depend heavily on the enactment of such an introjected identity, leading to a defensive adherence to identity and inner conflict when failing to engage in identity-consistent behaviors. Nonetheless, for identities to be not just adopted, but also autonomously engaged, the individuals will need to integrate the identities with

their other available identities, values, beliefs, behaviors, and interests, which will allow them to be both authentic and vital in their pursuit.

Soenens, Berzonsky, Dunkel, Papini, and Vansteenkiste (2011) assessed late adolescents' identity commitments, and their autonomous and controlled motives for committing to particular identities. Even after controlling for the strength of identity commitments per se, those commitments that were autonomously motivated were associated with more positive adjustment, whereas controlled identity commitments were predictive of poorer adjustment. Thus, although adolescents could commit themselves to particular self-representations (e.g., musician, basketball player), such commitments must be accepted and integrated to ensure adjustment.

Fostering Identity Development: The Role of Need Support

Given that identities may involve intrinsic motivation, and may be anchored by either introjected or more integrated regulations, it becomes clear how a need-supportive environment is important in the process of identity formation (Soenens & Vansteenkiste, 2011). As Assor (2012) pointed out, parents vary in the extent to which they foster inner valuing and allow adolescents to explore different interests and values, thereby nurturing self-discovery. To the extent such need supportive practices are provided, children and teenagers are more likely to develop an *inner compass* when making critical identity-related decisions. In contrast, finding life roles and self-definitions that are congruent with interests and integrated values is more difficult in controlling contexts. Consistent with this, Luyckx, Soenens, Vansteenkiste, Goossens, and Berzonsky (2007) showed that college students who perceived their parents as using psychologically controlling strategies were more inhibited in making identity-related decisions and in truly endorsing and identifying with the identity decisions being made.

Importantly, the development of an integrated set of regulations is presumed to be critical for subsequent need satisfaction, such that a positive spiral would develop. Evidence for such a model was obtained in longitudinal research on high school and college students by Luyckx et al. (2009), who focused on the extent of exploration, commitment, and ruminative thought these students displayed with regard to the directions and identities they were adopting. Results showed that satisfaction of SDT's basic psychological needs was in general positively related to making and identifying with particular identities, and to enhanced breadth and depth of identity explorations. Those with less consolidated identity commitments and those associated with more rumination had lower basic need satisfaction. Moreover, cross-lagged analyses provided evidence for reciprocal relations—need satisfaction both facilitated more positive identity formation, and more positive identity formation facilitated greater need satisfaction.

Although there are clear advantages to integrating identities, some identities are socially stigmatized, and some may conflict with other identities or roles to which a person aspires. Such identities often remain poorly integrated. These identifications can be resisted, or remain relatively isolated or compartmentalized, as opposed to being integrated with other identities, needs, and value attachments of the self (Ryan & Deci, 2011). For instance, suppose in his political considerations a man identifies with an ideology that is oppressive to certain minorities, and yet in his religious considerations he endorses being a generous and loving human being. Both might be values that he deems important, but their inconsistency requires that he keep them compartmentalized. Advocating harm to others might catalyze guilt or uncertainty when he is in his more religious identity. Yet integrating these might require change in one or both identities, and the psychological threat or loss of relatedness that might accompany such change.

The more autonomy supportive one's social context, the more easily the person can integrate identities and self-characteristics. For example Weinstein, Deci, et al. (2011) examined how autonomous versus controlled motivation, operationalized through either individual differences or the semantic priming of autonomy versus control, facilitates or inhibits the integration of positive and negative past identities. Results of their studies showed that more autonomously motivated participants felt closer to, and were more accepting of, both negative and positive past characteristics and identities, whereas more control-motivated participants were closer to and more accepting of only their positive, but not negative identities and characteristics. Moreover, defensive processes, as reflected in the use of nonpersonal pronouns and stronger escape motives, mediated these effects, indicating that lower defensiveness allowed fuller integration of negative identities. Finally, results demonstrated that when participants did more fully integrate both positive and negative past identities, they exhibited greater vitality and sense of meaning.

Toward Qualitatively Different Types of Identities

Finding identities that are need satisfying is often a struggle, as many social contexts pressure individuals to adopt identities that may or may not befit their interests or talents. Social contexts also can reject or demean other identities and roles. Thus, identities can be adopted for

controlled reasons, with the predicted negative outcomes for both well-being and persistence.

Identities can also be motivated as a response to need thwarting. SDT argues that, when needs are strongly thwarted in development, persons may adopt need substitutes referred to as *extrinsic aspirations*, such as those involving the pursuit of materialism, an appealing outer image, or ideas of fame and popularity (Kasser & Ryan, 1996). These identity pursuits often serve a defensive function, blocking feelings of vulnerability, and often are pursued to compensate for a lack of support and acceptance from others during development. In line with this, Kasser, Ryan, Sameroff, and Zax (1996) predicted that identities focused on materialistic life goals might be associated with (and compensating for) experiences of autonomy and relatedness thwarting from caregivers. Using a longitudinal dataset, they showed that adolescents whose mothers were less warm and autonomy supportive when the children were 14 years old were more materialistic at age 18.

Adolescents who experience need thwarting may be especially vulnerable to extrinsic ideals promoted within the media and advertising, which often suggest that the pursuit of extrinsic ideals will yield the confidence, love, or self-esteem such teens do not feel. For example, women high in autonomy frustration may be more susceptible to ambient extrinsic ideals for thinness and self-objectification. Mask and Blanchard (2011) examined this by having individuals differing in general levels of autonomy watch and react to a video depicting the thin ideal. After watching the video, women low in autonomy reported greater pressure to be thin, more dissatisfaction with their bodies, and increased concern with the quantity of food they ate. In contrast, women high in autonomy became more concerned with the quality of the food they ate, but they did not feel increased pressure or self-dissatisfaction (see also Pelletier, Dion, & Levesque, 2004). Thus, to the extent adolescents have built up a stronger inner foundation, they can rely on it to more autonomously decide how to handle identity pressures, which suggests that autonomy is an important factor in resilience.

Summary

We have discussed how the formation of identities and self-representations that are well internalized and integrated represents a major challenge for contemporary adolescents. Research suggests that without sufficient contextual need support, adolescents' development of a strong inner compass (Assor, 2012), and the adoption of identities truly grounded in their emerging interests and personally held values, may be forestalled.

AUTONOMY DISTURBANCES IN DEVELOPMENT AND PSYCHOPATHOLOGY

Need Frustration and Psychopathology

The development of the self proceeds most optimally in social contexts where children experience ongoing supports for their basic psychological needs. When needs are supported in domains and activities appropriate to their developmental level children display increasing amounts of autonomy. The situational and cumulative effects of support for psychological needs are expected to yield inner resources in the developing persons that further contribute to subsequent wellness and adaptation. Yet often development does not proceed optimally, especially when social contexts thwart the children's basic needs and in doing so amplify vulnerabilities and potentiate psychopathology.

Many forms of psychopathology entail *impairments of autonomy,* as failures in organismic integration and in the development of self contribute to a range of dysfunctions. From the perspective of SDT, explicating autonomy impairments involves a consideration of the interplay between the psychological needs for autonomy, competence, and relatedness that support the developmental processes of attachment, intrinsic motivation, internalization, emotional regulation, and identity formation, and the social contexts, such as parent or institutional behaviors, that either support or thwart the satisfaction of these basic psychological needs. In what follows we focus on specific types of disorders, both in terms of a theoretical account of autonomy impairments and a review of relevant research. Specifically we discuss three general categories of psychopathology, all of which involve serious autonomy disturbances but each of which has particular forms of autonomy impairments, leading to one of the categories of pathology, as is graphically illustrated in Figure 9.2.

First, we consider disorders in which *internally controlling regulation* of behavior predominates. These are disorders that, despite their many differences, are characterized by the salience of introjection or rigid internalizations and the associated feelings of anxiety, self-criticism, and threat. We especially focus on obsessive-compulsive disorders, paranoid personality, self-critical depression, and controlled eating disorders (i.e., anorexia and bulimia).

A second type of autonomy disturbance concerns *impairments in internalization* of social values and regulations, characterized by difficulties in the self-regulation of behavior. To illustrate these types of autonomy disturbance, we discuss problems of impulsivity and aggression, including oppositional defiance, conduct, and antisocial

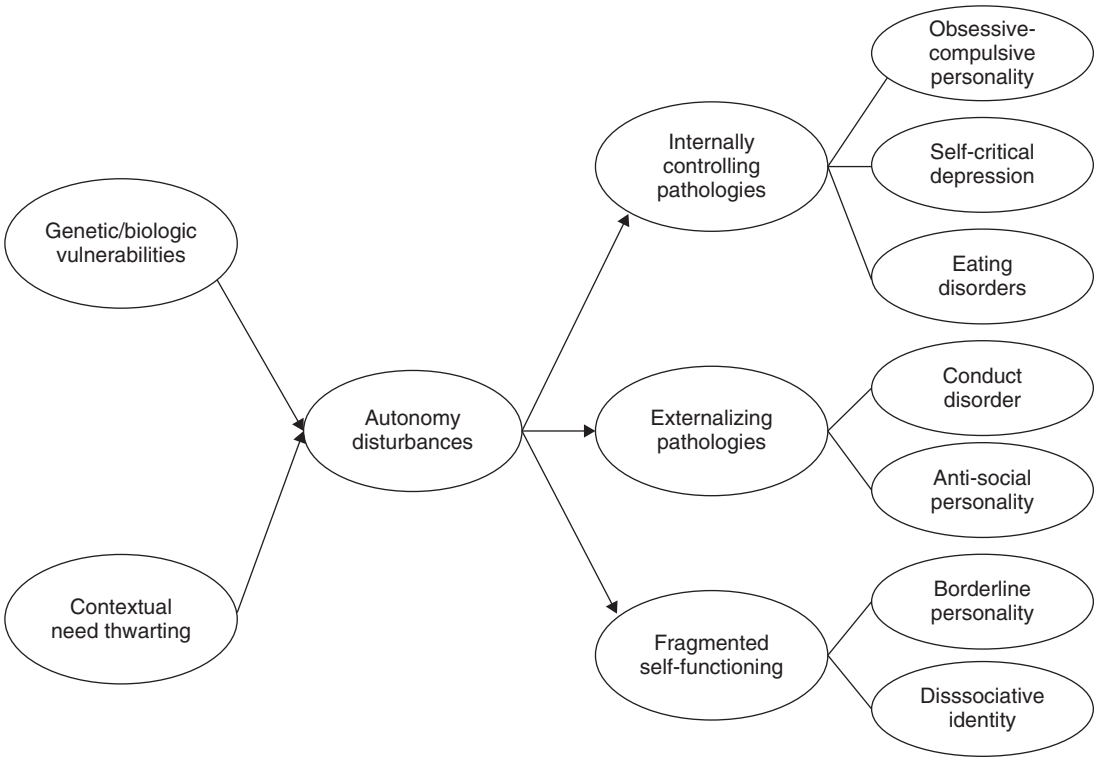


Figure 9.2 Graphic overview of selected psychopathologies reflecting autonomy disturbances.

personality disorders. A third category of autonomy disturbances we discuss are disorders associated with more severe thwarting of psychological needs in development, namely disorders associated with *fragmented self-functioning*. Here we focus especially on persons with borderline personality disorder and dissociative identity disorders, and on the integrative difficulties posed by controlling, inconsistent and intrusive others who thwart basic psychological needs and damage self-development.

In discussing each of these types of autonomy disturbance we address the experience of autonomy within each, and discuss factors relating to the social contexts of development that may contribute to impairments in autonomy and integration. Although it is the case that any factor that disrupts the organizational tendency, whether biological, interpersonal, or cultural in nature, can potentiate disturbances of autonomy, in many cases the sources of disrupted autonomy can be strongly related to deficiencies in the social environment—that is, to its failure to provide appropriate autonomy support, structure, or involvement (Ryan & Deci, 2000b; Vansteenkiste & Ryan, 2013). Yet even in those cases where the causes of the disorder do not lie in need thwarting contexts, the symptoms associated with such impairments can themselves interfere with autonomous functioning, and are often experienced as not under volitional control. In short, we discuss autonomy issues both as both an etiological factor and as a symptomatic factor, in varied forms of psychopathology.

Disorders Involving Introjection and Internally Controlling States

There are rich and variegated literatures concerning internalizing disorders, in which a person is beset with internal pressures, judgments, compulsions, and other self-directed mandates to act. These disorders, associated with both anxiety and depression, centrally involve a lack of autonomy and volition, as often motivations to act are highly controlled and even coercive. This literature also indicates that caregiver styles characterized by a lack of autonomy support and the presence of controlling or authoritarian strategies contribute to *internalizing disorders*, especially in vulnerable children (e.g., Bayer, Sanson, & Hemphill, 2006; Muhtadie, Zhou, Eisenberg, & Wang, 2013). What SDT's perspective adds to this literature is a rich body of findings concerning how controlling environments foster the development of internally controlling regulations—that is, how such conditions lead to regulation through *introjects* (Deci & Ryan, 1995; Ryan & Deci, 2008).

Introjects are understood within SDT as motivations characterized by internal pressure, enforced by contingent self-worth. Introjection involves an ongoing salience of strict internalized demands. Such demands and standards are introjected in the sense that one's motivation is based largely on internalized self-related emotions and appraisals. In introjection people adopt external standards as their own and determine self-worth based on whether they meet them, without making the standards a part of their integrated sense of self. Often the process of introjection entails a type of *self-deception* in which people think they want what the authority originally prescribed (e.g., Weinstein, Ryan, et al., 2012). This is akin to what Winnicott (1960/1965) referred to as *false self*, in which cognitive functions gradually lose their grounding in organismic processes. Yet heteronomy is phenomenally manifest in the pressured, have-to nature of experience, backed by threat of anxiety and self-criticism that drives much of the person's behavior. Introjects can indeed be highly motivating, and represent the core driving forces in people's lives. Thus, even when not formally diagnosed, the dynamics of introjection can catalyze significant inner distress. In addition, when rigid, punitive, or overly pervasive, they can be highly dysfunctional. This is particularly true for persons who are diagnosed as having an internalizing disorder.

Although, as we shall stress, the etiology, presentation, and course of internalizing disorders are complex and multidetermined, evidence that caregiver styles can contribute to such problems is also relatively clear. For example, consider that factors such as loss of parents (and thus of attachment supports), depression in parents (and thus low involvement or autonomy support), and excessive parental control (thus thwarting autonomy needs) have all been implicated in the development of childhood depression and self-critical styles (Ryan et al., 2006). SDT more specifically relates the formation of introjects to the thwarting of autonomy and relatedness needs, a process central to a variety of clinical presentations (Ryan et al., 2006). Thus, we look at parental thwarting of autonomy and relatedness as factors in internally controlling disorders.

The Obsessive-Compulsive Disorders

Two separable diagnoses carry the rubric of obsession and compulsion: obsessive-compulsive disorder (OCD) and obsessive-compulsive personality (OCP). These distinct entities share some common features in the dynamics of behavior regulation, but they also differ in meaningful ways.

OCD is characterized by the experience of intrusive thoughts and demands that typically can be alleviated only by engaging in ritualistic, rigid behavior. The thoughts are often ego-dystonic, in that they are unwelcome and anxiety provoking, and thus such thoughts are experienced as having an origin outside the self. Indeed, these thoughts often are inconsistent with the person's conscious values and ideals. An example is an OCD-diagnosed woman who reported continual intrusive thoughts concerning hurting her baby whenever she was near certain objects. These intrusive thoughts raised considerable anxiety precisely because they ran against her strongly held desire to keep her child safe. To alleviate this anxiety she was compelled to check continuously for dangerous objects in her vicinity. OCD is classified as an anxiety disorder precisely because obsessions and rituals often have the function of regulating anxiety, albeit usually with only temporary success.

An important feature of the dissonant thoughts often found in OCD is that they are persistent, unwanted, and difficult to control. From the perspective of the individual the unwanted thoughts befall him or her and are not autonomous or volitional. The person then feels coerced or compelled into ritualistic behaviors; he or she must do them or face dreadful anxiety. The actions that alleviate obsessional thoughts therefore have an external perceived locus of causality. As an aspect of this external causality, compulsive behavior patterns typically are performed under strict constraints—there is an inner demand to engage in actions in rigidly prescribed ways. These orders are experienced as heteronomous, albeit within the person. The cost of failure is guilt, anxiety, and self-disparagement, and in other cases a sense of panic and fragmentation of the self. The regulatory process of compulsive acts is thus accompanied by a sense of inner pressure that we would describe as internally controlling. That is OCD has the regulatory form of introjection, even though it may not always have social roots.

Like OCD, OCP is characterized by living in a very structured and orderly way, which can have both positive and negative consequences depending on how severe it is. In less severe cases, the regulation of behavior is still introjected and thus has the negative psychological consequences associated with introjection. Nonetheless, the order that is manifest from mild OCP can have adaptive aspects, for example in getting homework done or organizing possessions in orderly ways. Yet OCP is often more severe and compromising, with debilitating pressure and anxiety, and behaviors that are rigid and extreme. For example, people's collecting which is likely to be based in mild OCP is likely to become hoarding when the pathology becomes more serious, and their organizing may become

intensely rigid with threats of self-disparagement and self-punishment hanging over their heads to keep them in line.

Unlike OCD, there appears to be a less robust biological contribution to OCP, even though there are some common symptom dynamics. Supporting the differential role of biology, results by Jenike as early as 1991 found responsiveness among OCD patients to pharmacological interventions, but not among those with OCP. Also OCP, in contrast to OCD, is more a lifestyle than a symptom, and in many ways OCP can be viewed as a continuum from a somewhat rigidly focused set of attitudes to a disorder that interferes with ongoing functioning. Accordingly, many features of this controlled lifestyle are ego-syntonic rather than disturbing (Sperry, 2003). For example, individuals with OCP who organize their possessions and become distraught when things get moved or misplaced might go into treatment not because they view the orderliness as problematic, but because their pattern of living, which seems fine to them is causing interpersonal or vocational difficulties. In addition, as reported by Othmer and Othmer (2002), persons with OCP often display their characteristic drivenness, compulsiveness, and inflexibility when events involving authority, intimacy, or lack of structure trigger them. Nonetheless, the surface structure of these behaviors share similarities with the controlled behaviors so often manifest in OCD.

The internally controlling nature of regulation in OCP can, in some instances, be highly stable and self-sustaining, in part because it can come with ongoing external approval and derivative internal gratifications. For example, Assor and Tal (2012) examined adolescents' perceptions of their mother's use of parental conditional positive regard (PCPR) to motivate them to academically achieve. Those teens whose mothers applied contingent positive regard evidenced more self-aggrandizement following successes yet greater self-devaluation and shame following failures. This hypersensitivity to outcomes in turn predicted compulsive over-investment and maladaptive self-feelings, even when controlling for the effects of conditional negative regard. Thus, it appears that the experience of maternal conditional positive regard to promote school achievement leads to self-esteem dynamics in which the adolescents vacillate between feelings of grandiosity following success and self-derogation and shame following failure, representing significant emotional costs. These general population findings are reminiscent of the distress many OCP patients vividly describe.

It is particularly clear with OCP that the pathology of obsessive-compulsive actions is not always defined by the behavior itself, because as noted the behavior can be effective and functional. Rather, it is the rigid regulatory processes underlying behavior that are dysfunctional. Persons with OCP, while not acutely afflicted with intrusive thoughts sometimes befalling those with OCD, are often inordinately concerned with carrying out the actual or presumed demands of authority. For example, a person with this personality style can be quite industrious and fastidious in complying with introjected demands, and this may yield high activity and even productivity. However, rigidly controlled workaholics, who are likely to have OCP, tend to evidence a range of ill-being consequences (Van den Broeck et al., 2011). Clearly, the industriousness and rigidity of people with OCP is quite different from the vitality and spontaneity that characterizes autonomously functioning individuals, even ones who work excessively. Ryan and Deci (2008) argued that vitality concerns the free energy a person has at his or her disposal. The internally controlled regulation of an individual with OCP is often experienced as energy draining and without joy. As Sperry (2003) noted, the demeanor of someone with OCP can be "grim and cheerless" (p. 178). Moreover, resistance to inner dictates, when possible, may feel like a gargantuan effort.

There is an interesting point about OCP that highlights an important aspect of its disturbed autonomy. Although obsessive-compulsive individuals often act with determination in carrying out introjected dictates, they also sometimes display confusion and indecision. The problem especially arises when there is no dictate, no established course to follow—that is, when there is uncertainty and one needs to fall back on inner guidance. At such times, these people may be rocked with indecisiveness and ambivalence. This illustrates clearly how dependence on controlling regulations, being unintegrated, leaves one without self-direction. The anxiety of indecision highlights how the seeming determination behind many obsessive-compulsive acts is not autonomous; there is no inner compass to rely on when external direction is lacking.

Although the obsessive-compulsive disorders represent clear instances of psychopathology with disturbed autonomy, it is not clear, nor theoretically necessary that they are exclusively *outcomes* of parental control. Evidence has, in fact, been compelling for robust biologic contributions to OCD, whereas parental factors appear to play a larger role in the development of OCP. Indeed, case literatures concerning OCP have more frequently pointed toward controlling, often intrusive parenting (e.g., Millon, Davis, Millon, Escovar, & Meaghan, 2000). Benjamin (2003) suggested that persons with OCP often come from demanding, even coercive parenting environments. Moreover she argued that the controlling emphasis is more often

on punishment for failures than on acknowledgement for success. Yet, here too, systematic investigation of the social context of development of OCP individuals is not extensive. It does appear, however, that the controlling regulatory styles of those with OCP are more likely to be linked with excessive parental controllingness than is the case for OCD.

Introjection and Self-Disparagement: Self-Critical Depression

At the core of a number of pathologies are introjected demands that organize intentional behavior. Although this type of autonomy disturbance involves conflict and tension, individuals with rigid character are often able to behave intentionally and satisfy their introjects. There are other types of introjected demands, however, that individuals cannot easily attain, and these invariably result in experienced failure and self-disparagement. These forms of disturbed autonomy, like those of rigid character, begin with individuals' introjecting the demands of authorities and basing their self-worth on living up to those demands. But here, the pathology and experience are quite different, for the individuals' predominant experience is failure and worthlessness. Thus, not only is their autonomy undermined by introjects and the social conditions that instilled them, but they also feel incompetent and unloved by significant others. Two such pathologies of introjection are self-critical depression and eating disorders. We focus first on self-critical depression.

Research on depression and its etiology has increasingly pointed toward two distinct pathways to the disorder, each of which can build on biological vulnerabilities (Luyten & Blatt, 2013). The first is concerned primarily with internalization of excessive demands for achievement and is characterized by harsh self-criticism and guilt, and the second is concerned more with the loss of relatedness, love, or attachment. Although both of these vulnerabilities involve self-esteem dynamics, and issues of autonomy and relatedness are intertwined in both, the former, self-critical form of depression is particularly relevant to our discussion of autonomy disturbances, internalization, and internally controlling regulation. We thus focus on the self-critical depression syndrome, which has been labeled variously as a disorder of achievement-autonomy (Neitzel & Harris, 1990), introjection (Blatt, 1974), and autonomy (Beck, 1983).

By whatever label, this syndrome involves a type of disturbed autonomy in which people experience dysphoric affect and lethargy resulting from the belief that they are failures. In this disorder there are rigid standards or ideals that have been introjected, along with the belief that failure to attain them means they are unlovable and unworthy. Thus, the punitive introjects, with their ties to contingent self-worth, leave people vulnerable to ongoing self-scrutiny. Because with such introjects there is little individuals can do that is good enough, they will invariably experience a sense of worthlessness. In other words, self-disparagement tends to be ubiquitous. Individuals with such introjects are harsher in their self-judgments than in their judgments of others, and these self-judgments often form the precursors to depressive episodes (Beck, 1983). Their harsh self-judgments may even manifest outwardly, through engagement in nonsuicidal self-injurious behaviors, which reflect the scars of the inner critic (Claes, Soenens, Vansteenkiste, & Vandereycken, 2012). Accordingly it is hardly surprising that adolescent depression and low self-esteem are associated with low autonomy (e.g., Noom, Dekovic, & Meeus, 1999).

The phenomenological functioning underlying *self-critical perfectionism* is particularly relevant to the understanding of depression as involving disturbed autonomy. With respect to significant self-goals people see themselves as responsible yet as incapable. Thus, the absence of felt competence to attain internalized goals results in a sense of amotivation (Deci & Ryan, 1985a). At the same time many of the specific demands on the self to achieve or succeed have the character of *have-to* and *must* revealing their phenomenological character as having an external perceived locus of causality (i.e., as being heteronomous with respect to the self). Consistent with such an analysis, self-critical perfectionism has been found to be associated with more controlled functioning (e.g., Miquelon, Vallerand, Grouzet, & Cardinal, 2005) and increased psychological need frustration over time (Boone, Vansteenkiste, Van der Kaap-Deeder, Soenens, & Verstuyf, 2014).

There has been recent interest in the development of perfectionism, a characteristic related to self-critical depression. In line with our argument that such internally controlling characteristics reflect disturbed autonomy, evidence suggests that individuals with high levels of self-critical perfectionism frequently report having experienced harsh and authoritarian parenting (see, e.g., Flett, Hewitt, & Singer, 1995; Frost, Novara, & Rheaume, 2002; Kawamura, Frost, & Harmatz, 2002). For example, Enns, Cox, and Clara (2002) found support for a model in which harsh parenting led to maladaptive perfectionism, which in turn was associated with an increased proneness to depression.

Such findings suggest perfectionism as a pathway through which controlling environments might increase

depressive symptoms. Consistent with this, investigations of the backgrounds of self-critical depressives point to controlling parental styles. In one illustrative investigation, McCranie and Bass (1984) found that women high in self-critical depression had parents who maintained strict control, demanded high achievement, and were inconsistent and contingent in their conveyance of love. Whiffen and Sassville (1991) reported similar results for both males and females. Research based in SDT concurs, with longitudinal data showing that children growing up in controlling families are more likely to adopt self-critical perfectionistic attitudes, which, in turn, relate to elevated depressive symptoms (Soenens, Luyckx, et al., 2008). Moreover, the adoption of self-critical perfectionistic standards in controlling environments may not elicit depressive symptoms but may also involve self-esteem deficits (e.g., Soenens, Vansteenkiste, Luyten, Duriez, & Goossens, 2005).

Empirical findings are thus consistent with our speculations concerning the nature of the social contexts leading to introjection and internally controlling states. In this frame one can view self-critical depression as a chronic and pervasive state of ego-involvement in which one continuously fails to live up to the demands and is thus punished. Unfortunately, self-critical perfectionism appears to get transmitted from the one generation to the other through controlling parenting (Assor et al., 2004; Soenens, Elliot, et al., 2005). Presumably, the contingent love self-critical perfectionists display vis-à-vis their own functioning gets projected onto their children, such that children experience their parents as displaying conditional regard, which subsequently affects their own functioning.

Indeed, such dynamics between parent and child may be mutually problematic. Using a cross-lagged longitudinal design in two samples of middle and late adolescents, Soenens, Luyckx, Vansteenkiste, Duriez, and Goosens (2008) compared three models concerning the role of parental control in depression. Results generally favored a reciprocal model in which controlling parent behaviors contribute to increases in depression, which in turn enhance parents' tendency to control their teenager. This represents a truly maladaptive cycle between parent and child. This bidirectional relationship suggests that more depressive symptoms in an adolescent can elicit less autonomy support from parents over time, even as autonomy support from parents conduces toward fewer depressive symptoms in their adolescents.

Further, Soenens, Park, Vansteenkiste, and Mouratidis (2012) examined whether this association between parental control and depressive symptoms among adolescents might be culture bound as some cultural relativists have argued, versus whether as SDT suggests, the detrimental effects of psychological control and the loss of autonomy generalize across cultures. Soenens et al. (2012) specifically investigated the relevance of two domain-specific expressions of psychological control (i.e., dependence oriented and achievement oriented) in both Belgian and South Korean adolescents. Analyses showed very similar relations between the psychological control, depressive personality, and depressive symptoms across the two samples, thus supporting the notion that the effects of psychological control generalize across cultures.

In short, there are clear relations between control and these internally controlling forms of depression in which self-critical cognition is salient. Although there is substantial evidence that vulnerability to depression varies, the importance of autonomy thwarting in this expression of psychopathology is becoming ever better documented.

Eating Disorders: Anorexia and Bulimia

In her classic book on the topic, Bruch (1973) described eating disorders as pathologies of autonomy. She argued that these eating pathologies typically involve a struggle for control that takes the form of an obsession with eating and body image, with control over one's body dynamically staving off a pervasive sense of ineffectiveness. Bruch saw people with eating disorders as exemplifying the capacity, as a function of harsh introjects, to manipulate bodily states and desires to preserve the illusion of self-sufficiency and to feel a sense of control with respect to oneself and others. In restrictive anorexia, the introjects around eating and weight are more stable and effective in keeping people's eating behaviors in abeyance, whereas in bulimia there is a more open conflict between rigid controls and akratic eating. Bulimics engage in binge eating—in expressive, uncontrolled consumption—only to be plagued by guilt and self-derogation leading to vomiting or abuse of diuretics.

The psychodynamics highlighted by Bruch were empirically supported in an early study by Strauss and Ryan (1987). They found greater self-oppression and self-rejection in both anorexic and bulimic participants than in a matched control group, suggesting internally controlling dynamics, whereas control-group participants displayed more flexible self-management and self-acceptance. Furthermore, Strauss and Ryan documented a particularly heightened impersonal causality orientation (Deci & Ryan, 1985a) among restrictive anorexics, indicative of an impoverished sense of both autonomy and effectiveness.

Internally controlling forms of regulation are readily apparent in the dynamics of many behaviors entailed in eating disorders, especially those involving restrictive self-control. Whether anorexic or bulimic these patients display inordinate concern with how others view them, and hypertrophied public self-consciousness. As Plant and Ryan (1985) argued, such consciousness potentiates an external perceived locus of causality in which people have to conform to the projected views of others. Although a focus on weight is often paramount, eating disordered patients are typically self-conscious, demanding, and self-critical with regard to many aspects of appearance and behavior. And although restrictive anorexics in particular appear to display a high degree of personal control, the regulatory basis of this control is dictatorial.

Applying SDT to anorexia, Vansteenkiste, Soenens, and Vandereycken (2005) described how a focus on thinness represents an attempt to gain a sense of security and worth, yet the thinness, even when attained, fails to supply the anticipated emotional benefits. The anorexic person is never thin enough, yet they continually work with the belief that positive feelings are a few pounds away. The paradox, however, is that achieving their extrinsic goal is satisfying to some degree, so it further anchors them in this pattern of behavior. These patients often strongly assert that they do feel better when losing weight and believe that the pursuit of weight loss is therefore not the source of distress. Indeed, their own experiences seem to confirm that efforts at weight loss are part of the solution. The transitory nature of relief thus fosters an addictive quality to the internally controlling drive for thinness.

Whereas the restrictive anorexics can display a high degree of control with respect to eating, bulimic patients often find their control overwhelmed by an impulse to binge eat. Binges typically occur at times of high psychological need frustration, thus showing how their introjected regulatory structures are not sufficiently stable to keep the impulse in check. The binge impulse is often a reaction to the experience of need frustration rooted in internal criticism, so it represents an attempt to escape from the painful sense of self that carries the burdensome, introjected standards. For example, Verstuyf, Vansteenkiste, and Soenens (2012) researched the connections between need frustrations and binge eating. In a sample of female adolescents, they showed indeed that an investment in appearance-focused motives led to need frustrations, which in turn predicted increased bulimic symptoms. Along similar lines, a diary study in female adolescents (Verstuyf, Vansteenkiste, Soenens, Boone, & Mouratidis, 2013) indicated that daily variation in psychological need frustration covaried with ups and downs in bulimic symptoms, suggesting that binge eating serves to compensate for painful experience of need frustration.

It is interesting to note that, as the psychic threat gets greater for individuals with the bulimic disorder, the people's regulatory capacities becomes weakened, whereas in people with obsessive-compulsive personality the regulatory capacity often becomes even more rigid and dominant. The lack of stability of the regulatory introjects in the people with bulimia thus allows for the akratic action, but the self-evaluative introjects invariably result in self-disparagement and feelings of depression for having lost control. It is therefore interesting to note in this regard that the long-term course for many restrictive anorexics is a shift to bulimic patterns of coping.

Although again there are multiple contributors to the development of bulimia and related eating disorders, the role of familial factors in setting up the dynamics of introjection and internal control are quite salient. Bruch (1979) has vividly depicted the role of parents in catalyzing anorexia by depriving their daughter of autonomy and the "right to live her own life" (p. 38). Minuchin, Rosman, and Baker (1978) similarly reported high levels of enmeshment and intrusive control in families of patients with eating disorders. Strober and Humphrey (1987) reported that both anorexics and bulimics experience parents as blaming, rejecting, and critical. Strauss and Ryan (1988) found less mutuality of autonomy in the object representations of both bulimic and anorexic participants, and lower expressiveness within their families. Another more recent study found that late-adolescent women hospitalized for eating disorders reported having more psychologically controlling fathers than a matched sample of women without the eating disorders (Soenens et al., 2008).

Summary

Each of the disorders we have considered thus far begins with rigid, demanding, and critical introjects. These rigid structures take varied forms and are more or less stable and effective in controlling the people's actions. In some cases, most notably obsessive-compulsive personality and anorexia nervosa, the disordered individuals can feel a strong sense of personal control and self-efficacy—the OCP can keep personal affairs orderly and anorexics can keep body urges under control. But these, like the other disorders involving salient introjects, constitute disturbed autonomy and can terrorize people with such contingent self-esteem. Findings in this area also highlight

the important difference between personal control and autonomy; being self-controlled can sometimes yield desired outcomes yet not be experienced as autonomous or volitional (Muraven, Gagné, & Rosman, 2008).

We turn now to disorders characterized not by the prevalence of introjects, but rather by the relative absence of internalization. These forms of psychopathology involve the lack of adequate regulatory structures that link one effectively to the socializing context.

Impairments of Internalization in Externalizing Disorders

As we argued earlier, the process of internalization is dependent upon certain affordances in the caregiving environment—namely, autonomy support, structure, and involvement—which together facilitate both attachment to caregivers and a readiness to assimilate the values they model. Some caretaking environments, particularly those characterized by coldness and hostility or by neglect conduce toward poor quality of attachments and lessened internalization of social norms and prosocial behaviors (Johnson, Cohen, Chen, Kasen, & Brook, 2006; Joussemet et al., 2008). In addition, those low in autonomy supports, especially the lack of both perspective taking and attunement to needs and interests, are at risk for both low social skills and negative social interactions (Shields et al., 2001). In fact, parenting environments impact on internalization through their effects on need satisfaction (Ryan et al., 2006). Controlling parents not only restrict opportunities for children to develop self-regulatory capacities, they create emotional and need-related frustrations that can fuel negative behaviors.

In the realm of more severe forms of psychopathology, we are also concerned with more extreme forms of need thwarting, including physical, sexual, and emotional maltreatment, which thwart all three basic needs, with irradiating effects across multiple developmental lines. Such parenting conditions may result either in externalizing problems (Gershoff, 2013) such as conduct disorders, or, especially when the thwarting is extreme, in disorders such as dissociative identity.

Illustrating this with respect to externalizing disorders, Shields, Cicchetti, and Ryan (1994) examined the social-competence self-regulation impairments of children who had been maltreated by caregivers. The setting was a summer camp for both maltreated and economically disadvantaged children, allowing for a comparison of these groups. As predicted, children who had endured maltreatment displayed, as rated by observers, less social competence, more emotional dysregulation, and impaired self-regulation reflected in externalizing disorders as well as internalizing disorders. Indeed, self-regulatory deficits mediated the relations between maltreatment status and social competencies.

In a subsequent study, Shields, Ryan, and Cicchetti (2001) further explored this developmental process by assessing maltreated and nonmaltreated children's representations of the parenting they experienced. They found, as expected, that the representations of parents in maltreated children, as accessed through a projective narrative task, were less coherent and integrated, and more negative and constricted than those of nonmaltreated children. These differences in parental representations were in turn related to the externalizing processes of emotional dysregulation, aggression, and peer rejection in the ecologically valid camp setting.

These issues concerning need thwarting conditions and impairments in self-regulation have particular salience with respect to conduct and oppositional disorders, in both of which internalization, self-regulation, and capacities for relatedness are impaired.

Conduct Disorders and Antisocial Personality

Broadly speaking antisocial personality disorder (APD) applies to persons who lie, steal, manifest an impoverished sense of responsibility, are aggressive and manipulative toward others, and show evidence that these patterns are continuations of behavior disorders earlier in life. Children diagnosed with *conduct disorders*, a frequent antecedent of APD, display control problems, lability, a lack of ability to give and receive affection, and delayed or impaired development of conscience. Further, self-aggrandizement and egocentrism typically characterize their behavior, and their lying about accomplishments highlights their excessive need to be recognized or esteemed by others in an immediate way. Like adults with ADP, these children tend to display a lack of conscience, or deep concern with what is good or right. Related to conduct disorders and APD is the behavioral category of reactive or destructive aggression, the developmental routes of which have been related to those associated with these diagnostic categories.

Etiologic theories of APD have clearly implicated biologic or genetic factors such as poor autonomic reactivity (Burnette & Cicchetti, 2012; Raine, 2013). Yet even with this recognition of potential biologic vulnerabilities, it is clear that such vulnerabilities become amplified in nonnurturing environments. Our contention is that APD is a psychopathology entailing *impaired internalization*, and although internalization may have been compromised by biological factors (Jafee, Moffitt, Caspi, & Taylor, 2003),

we maintain that these impairments can also be linked to, and are interactive with, deficits in the social contextual factors associated with the basic psychological need supports that are essential for internalization to develop.

An internalization perspective looks toward the family environment for the relational resources that support the internalization of values of a prosocial nature, resources that are frequently lacking in the families of persons developing APD. For example, the empirical evidence supports the idea that prosocial values are most likely to be acquired (or expressed) when caregiving is characterized by warmth (Maccoby, 1980), low power assertive discipline (Hoffman, 1960), and autonomy support (Ryan & Hawley, in press).

The experience of growing up in a nurturing, caring, and responsive familial environment undoubtedly facilitates prosocial values both directly and indirectly. A person who grows up in a basic need supportive environment will have been exposed to models of caring and concern about others, from figures to whom they are likely to attach and desire to emulate. Support for this comes from many quarters. For example, Ryan and Connell (1989) reported that children who experience a high quality of relatedness to parents were more autonomously motivated in the prosocial domain. Conversely, a variety of clinical and empirical perspectives have suggested that antisocial personality, oriented towards self-serving, manipulative, and hedonically gratifying acts, has its roots, in part, in cold, inconsistent, and controlling family environments (e.g., Benjamin, 2003). A plethora of studies has also shown that the common backdrop to APD and conduct disorders of childhood includes such factors as maternal depression, loss, high family conflict, other parental pathology, and impoverished conditions of life that fragment the family (Coie & Jacobs, 1993; Loeber & Stouthammer-Loeber, 1986). All of these factors potentiate a situation ultimately lacking in the basic nutrients upon which internalization depends—namely autonomy support, adequate structure and guidance, and concerted, caring involvement.

One limitation of these models of value acquisition, and their implications with regard to the development of externalizing disorders such as APD, is that they do not focus on how or why children might develop nonprosocial or nonmoral attitudes, except as a converse or absence of moral internalization. For example, there are probably very few parents who strive to teach their children to be manipulative, materialistic, or Machiavellian, but there are many children who develop such an orientation. Absence of identification does not explain why one proactively seeks to act in *these* ways. Thus, a fuller model of APD requires both explaining why internalization fails, and why hedonic, aggressive, self-gratifying values predominate instead.

Directly informative in this regard is a six-year longitudinal study by Joussemet et al. (2008), who followed children from six to twelve years of age. Specifically, they identified developmental trajectories in childhood aggression in which they applied SDT. They found that over these years children on average engage in a lower frequency of aggressive acts, presumably reflecting socialization and development. Yet the slope of that trajectory differs among children, such that while for most there are declines in aggressive acts, for others there are maintained or escalating frequencies of aggression. After controlling for a number of other risk factors for being aggressive (e.g., being male, having a reactive temperament, parental divorce/separation), Joussemet et al. found, as they hypothesized based on SDT, that having a mother who was autonomy thwarting rather than autonomy supportive reliably increased the odds of a high aggressive trajectory.

In fact, some research within SDT suggests that controlling parenting can result not only in poor internalization, but also oppositional motivations. In line with this, parental prohibition of moral misdeeds perceived to be controlling was found to predict increased oppositional defiance (Vansteenkiste, Soenens, et al., 2014). Other studies have established the explanatory role of need frustration in the relation between controlling parenting and oppositional defiance and associated problem behaviors (Van Petegem, Beyers, Vansteenkiste, & Soenens, in press). Indeed, it seems that much antisocial behavior can be understood as a reaction to controlling, power-assertive strategies in contexts where attachment security and relatedness is low (Kim, Kochanska, Boldt, & Nordling, 2014).

Ryan, Sheldon, Kasser, and Deci (1996) argued that, to the extent that individuals lack autonomy support and caring involvement (and therefore are deficient in the development of self), they often turn toward extrinsic values to gain and sustain some minimal sense of power, narcissistic importance, and worth, and a corresponding lack of concern with doing for others or contributing to society. In line with this, Kasser et al. (1995), analyzing data from a long term longitudinal sample, found that adolescents who were more materialistic came from homes where both they and their mothers reported that there was less autonomy support, warmth, and security. They also found that more impoverished, high-crime neighborhoods, in confluence with these more controlling and hostile parenting environments, were likely to promote children's placing strong importance on these extrinsic values. Most relevant in this context, Kasser et al.'s analysis of clinical interviews with

participants in an at-risk population (defined in terms of maternal psychopathology and low socioeconomic status) revealed that those with greater centrality of materialistic values were more likely to be clinically diagnosed as conduct disordered. One important aspect of this work was its clarification that environments that fail to support autonomy and relatedness promote a compensatory emphasis on alienated or substitute needs, such as material acquisitions and visible trappings of worth. These, in turn, activate the so-called antisocial personality, albeit in ways often seen as self-centered. The research also highlights the continuity of motivational dynamics between nonclinical and clinical populations, as caregiving factors associated with materialism, in more extreme forms are associated with conduct disorders. This general pattern of findings is also consistent with Coie and Jacob's (1993) analysis of conduct disorders.

From the perspective of SDT, the development of conduct disorders and a self-focused goal orientation both stem directly from inadequate attachment and failed internalization resulting from an externally controlling, inconsistent, and affection-impoverished social context as they interact with vulnerabilities to poor self-regulation. To the extent that social values (and the economic conditions that structure them) disable, distract, or fragment the caretaking environment, then children will be more oriented to narcissistic goals to gain a temporary sense of worth and importance. This model is applicable not only to conduct-disordered children but increasingly also to American culture more generally (Kasser, 2002; Twenge et al., 2010). Put succinctly, the more we create conditions that disrupt the quality and stability of familial relationships, the more narcissistically oriented and antisocial our culture as a whole may become.

In sum, conditions of need thwarting, particularly threats to and deprivations of autonomy and relatedness satisfactions in childhood conduce to lowered attachment and interpersonal connectedness, to poorer development of self-regulation and thus impoverished internalization, and to need substitutes in the form of hedonic and materialistic aims, rather than prosocial ones. While such conditions negatively affect wellness in general (e.g., Kasser et al., 1995) when applied to vulnerable individuals the result is often more entrenched antisocial patterns of behavior.

Severe Need Thwarting in Dissociative Identity and Borderline Personality Disorders

We have argued that essential to the formation of integrated experience and behavioral regulation is support for autonomy, competence, and relatedness. Dissociative identity disorders (DID) and borderline personality disorders (BPD) represent, in most cases, disorders befalling individuals whose life experiences were characterized by not only deprivation, but also active intrusive thwarting, of both autonomy and relatedness needs. In fact, they represent disorders where there has been a failure in the average expectable environments needed for psychological development. For example, Johnson et al. (2006), using a longitudinal sample, reported that low affection (low relatedness) and punitive (low autonomy support) parenting practices were associated with elevated risk for borderline and other personality disorders. Steinberg and Schnall (2001) reported extremely high rates of early physical and sexual abuse among patients with DID. In the histories of people with either disorder, important others who should have been loving and kind to them were instead hurtful and maltreating, violating of the minds and bodies of the people who needed their support. The compensatory results of this are less integrated functioning and diminished sense of autonomy and connection.

Borderline Personality Disorder

Borderline personality disorders have become a predominant concern in clinical settings both because of their increasing incidence and thus the personal resources demanded in their treatment. Studies of BPD highlight many issues in character pathology generally, and they represent a prototypic example of structural damage to the self that has been associated with caregiver failures to support autonomy and relatedness in early development (Ryan, 2005).

The core feature of BPD is the lack of a cohesive and stable sense of self. Associated with this lack of a consistent and organized self are highly labile emotions, interpersonal relations, and self-esteem dynamics. Borderline individuals show the externalizing attributes of impulsivity, along with some of the features of internalizing disorders such as susceptibility to depression, anxiety, and fragmentation in the face of self-esteem related losses. A cardinal feature of BPD is anger, both self- and other-directed, which can result in destructive actions and magnify relationship volatility and internal feelings of being overwhelmed and disintegrated. More generally, patients with BPD have difficulty differentiating internal needs from external reality, and they are tremendously dependent on concrete supports from others to maintain a sense of self. They lack the internal regulatory processes to modulate anxiety, which can escalate to panic proportions, particularly when no one is available to contain and comfort them.

Persons with BPD also often lack a stable identity or sense of purpose (Meissner, 1988). Patients with this disorder may, however, latch on to something or someone in an effort to derive a temporary feeling of cohesion, but these choices are often inappropriate or destructive. Commitments are difficult because borderline individuals lack a stable and cohesive self that can form the basis for sharing in committed relationships or endeavors. Connected with the lack of feeling of identity, patients with BPD may feel empty and isolated (Westen, 1991). Clearly, there is a diminution of their true self whereby they lose connection to their interests and feelings. Patients with this disorder often report feelings of boredom and may engage in impulsive acts, such as substance abuse, careless spending, and binge eating, to counteract such feelings. Closely related to this is borderline individuals' lack of capacities for reflective awareness, especially when inner urges are strong (Bleiberg, 2004; Ryan, 2005).

Phenomenologically, patients with BPD often report being both controlled and helpless with respect to their behavior. They often feel like a victim of circumstances without a sense of responsibility for the direction of their own fate. One late adolescent patient, for example, reported that prior to self-mutilation he entered into a lost state, where the overwhelming impulse to cut came on him (impersonal perceived locus of causality), while at the same time he felt he could obtain relief and release only from dysphoric self-hate by engaging in such acts (suggesting external causality). In no sense did he feel autonomous and volitional in these acts, but rather driven, desperate, and helpless.

Although there is some evidence of genetic contributions to BPD, in that many appear to have exhibited a difficult child temperamental profile, much of the etiology appears to be associated with care-giving environments (e.g., Silk, Lee, & Hill, 1995). As we discussed in attachment research, formation of a stable and cohesive sense of self depends on nurturance in the form of autonomy support and positive involvement. Yet as Sperry (2003) noted, those with BPD have typically experienced severely impoverished care-giving during these early years, with parents' (particularly mothers) having difficulty allowing the children to move toward self-sufficiency and autonomy. Through being both controlling and inconsistent, parents of individuals with BPD fail to bolster the children's integrated self that is essential for the tasks of emotion regulation and identity formation. Having been exposed to caregivers who did not provide autonomy support and were unable to take the children's perspectives, the children failed to develop the sense of reflective awareness that would allow them to be in touch with their own internal states or the internal states of others.

Early caregivers of individuals with borderline disorder have often been described as unavailable, inconsistent, and neglectful, with frequent reports of sexual, emotional, and physical abuse (Sperry, 2003; Zanarini, 1997). In one study, 80% of borderline patients were reported to be physically or sexually abused or to have witnessed serious domestic violence (Herman et al., 1989). Explicating more specifically the affect and behavior regulation difficulties of these patients, Linehan (1993) suggested that patients with borderline disorder often come from families that invalidate the affective experience of their children. There is little tolerance for fears or anxieties in their children, and they do not offer soothing or comfort when needed. Without such care, these children have difficulty internalizing the capacity to self-soothe, resulting in emotion regulation difficulties, especially being unable to tolerate feelings of distress.

According to object relations theorists (Masterson, 1985), the disorder has its roots in the mother–child relationship, particularly during the phase of separation-individuation when the child is striving to experience him or herself as separate from the mother. Part of the phase involves a pushing away from the mother. In families of borderline individuals, the mothers are not able to tolerate movement toward self-sufficiency as it brings up their own fears of abandonment. Consequently, the mothers threaten to withdraw nurturance from the children if they move to act as separate autonomous individuals. Given this level of conflict, there is no "good" object that can support the psychological needs underlying a cohesive sense of self.

The problems of borderline patients thus illustrate how lack of empathy and consistent involvement and autonomy support undermine intrinsic interests and tendencies and self-regulatory functions—all aspects of a healthy autonomous functioning (Ryan, 2005). These developmental thwarts are thus critical to the formation of BPD. Sadly patients with BPD thus have ongoing experiences of lack of need satisfaction, as they have difficulty trusting in relationships and often find themselves acting without autonomy, both contributing to inner distress and continuing cycles of dysregualtion.

Dissociative Identity Disorder

Dissociative disorders are a particularly important issue within SDT given the theory's focus on autonomy as integration and synthesis. That is, SDT defines autonomy as the integrated regulation of behavior, and dissociative disorders, by definition represent the absence of such integrated regulation. There are two broad senses in which

dissociative disorders can represent nonintegration. Some dissociative disorders concern a lack of integration between behavior and experience with a sense of self. This is evident in depersonalization symptoms and when there is amnesic or accessibility barrier for certain event or actions. More extreme is the issue of multiplicity, the idea of more than one center of consciousness, expressing distinct motivations, memories, and attributes.

Dissociative disorders thus fundamentally represent a substantial breakdown in self-integration (Ryan et al., 2006). Related by some to an extreme form of BPD, as well as to severe PTSD, most clinicians and researchers also view most dissociative disorders, and particularly dissociative identity disorder (DID) as frequently a product of severe maltreatment or loss (although see Lynn, Lilienfeld, Merckelbach, Giesbrecht, & van der Kloet, (2012) for a contrary perspective.) Dissociation represents a protective or defensive mechanism, originally arising to protect integrity of a self that is beset with trauma. However, when inability to integrate experiences is chronic or severe, fragmentation of self-functioning and increased tendency to use dissociation to cope with threat occur (Carlson, Yates, & Sroufe, 2011).

DID manifests as compartmentalized functioning, in which traumatic experiences are housed in dissociated, often inharmonious, parts of personality. Yet more distinctly, DID symptoms include alterations between distinct identity states, often accompanied by amnesic barriers between them. Given its traumatic origins, symptoms of DID overlap with those of both BPD post-traumatic stress disorder. Yet DID is also commonly related to depression, associated with both developmental deficits and helplessness within the condition. In part this may be related to a common pattern in which, in an attempt to maintain relatedness to an abusing or harsh caregiver, the child internalizes self-critical and self-blaming views (e.g., Westen, 1991). Low self-esteem and depressive affect thus commonly accompany DID and other dissociative disorders.

DID is an autonomy disturbance insofar as autonomy requires cohesiveness and integration, and the violation of self, especially by caregivers, can shatter integrity. The splitting of personality has the function of defensively shielding the core self from pain or harm (van der Hart, Nijenhuis, & Steele, 2006), but the shifts in identity that characterize DID are typically not experienced as within personal control. Indeed, they have an impersonal perceived locus of causality (Ryan et al., 2006). Depersonalization, another symptom associated with DID, also exemplifies this sense of impersonal causality, as if one's body were acting independently of one's will. As autonomy, or self-rule, depends on a sense of self and an internal perceived locus of causality for behavior, it is clear that autonomy is seriously disturbed when behavior occurs without choice or in a dissociated state.

The perceived lack of personal causation is often part of the despondency and depression these patients report. For example, a patient who came to treatment for depression and for concerns for her memory for events had manifest symptoms of DID. She subsequently revealed repeated sexual abuse by her father that she previously had not reported. Images began to surface, often uncontrollably. As memories unfolded it was also clear that she had been repeatedly threatened if she ever told anyone, a command she had internalized. Various parts of her story, and emotional reactions to it, were represented by different personality systems, each with its own age and makeup, and its own capacity to exert executive control under particular circumstances.

As van der Hart and colleagues (2006) pointed out, whereas healthy personality functioning is characterized by synthetic, coordinated functioning, the defensively precipitated identities in DID are often very simple and focused, and not prone toward integration or harmony with other personality subsystems. Van der Hart et al.'s treatment approach focuses on increasing connectivity between split off parts of personality, using a slow process of exposure and dialog in which compassion and understanding are fostered, along with encouragement to move toward feared or avoided opportunities for self-realization, to further promote such connectivity. Clinicians who have worked with DID know the difficulty of the process approach and the required patience and empathy. Often change requires strong supports and anxiety reduction strategies as well. Adaptation and tools for grounded self-regulation rather than full integration is often an appropriate goal. The goal of course is the recovery of more coherent, integrated functioning, and the resumption of self-development to whatever extent possible.

In sum, a fundamental tenet of developmental psychopathology is that the study of both normal and atypical development should inform each other (Cicchetti, 2006; Cicchettti & Toth, 2009). SDT's research into basic psychological needs richly links psychologies of growth with those of pathology. Evidence shows how the psychological nutrients that facilitate optimal development, when neglected or thwarted can have cascading negative effects across development. Moreover, the kind of need thwarting a child experiences can be related to the kind of pathology he or she manifests. We identified three types of disorders reflecting different types of autonomy disturbance:

internalizing disorders, impairments in internalization, and self-fragmentation. Each reflects a varied way the integrated self-regulation and ownership of actions is disrupted, and each also has autonomy thwarting as a featured symptomatic or etiological factor.

Translational Implications: Autonomy and Autonomy Support in Psychotherapy and Intervention Programs

An interesting question concerns the relations between *etiological factors* such as lack of autonomy, relatedness, and competence supports in development to *ameliorative factors* proposed by various treatment approaches to psychological problems. It seems clear that the absence of need-supportive inputs from caregivers can catalyze an array of negative developmental processes, both behavioral and biological, that eventuate in disordered functioning. Reciprocally, in efforts to treat the immediate harms and cascading negative effects of such social etiological factors, SDT suggests that need supportive treatment and intervention contexts are critical.

Ameliorating or treating existing disorders often entails addressing need deficits both *directly*, by providing a need supportive therapeutic context, and *indirectly*, for example by providing people with skills or opportunities to better satisfy needs in their natural environments. In other words, SDT suggests therapy is, in large part, effective through facilitating the individuals' capacities to act with autonomy, and in acting so, to satisfy all three basic psychological needs (Ryan & Deci, 2008).

Autonomy support is of course important to all schools of psychotherapy, especially because volitional motivation is so necessary to therapeutic engagement and success. For example, Ryan, Lynch, Vansteenkiste, and Deci (2011), reviewing approaches to psychotherapy, and behavior change distinguished between outcome and process focused therapies. Outcome focused approaches (e.g., behavioral and cognitive behavioral treatments) tend to promote autonomy through an emphasis on transparency. Patients are told beforehand what is expected and allowed to select in to treatment, or not. Readiness for treatment can also be facilitated through motivational enhancement therapies, such as brief motivational interviewing (MI; Miller & Rollnick, 2002) as adjuncts to outcome-focused interventions to prepare the individual to volitionally engage the therapy. Ryan et al. pointed out that more *process-oriented* schools of therapy (e.g., psychodynamic and existential approaches) conceive of motivation as a focus of treatment itself rather than something that facilitates pre-treatment readiness. Cultivating autonomous engagement and taking interest in resistances is in fact considered an active and ongoing focus of process-oriented therapies.

Zuroff et al. (2007), drawing on SDT, proposed that autonomy should be considered a common factor in all effective psychological treatments. Supporting their proposal, they reported a study of depressed outpatients who were randomly assigned to receive interpersonal therapy, cognitive-behavioral therapy, or pharmacotherapy with clinical management. Assessments of depression severity were taken before and after treatment, and therapeutic alliance, the clients' autonomous motivation, and therapists' autonomy support were measured at the third session within each group. Zuroff et al. found first, as expected by SDT that therapist autonomy support was associated with greater client autonomous motivation. More strikingly, autonomous motivation was a better predictor of improved outcome than therapeutic alliance across all three treatments. The researchers therefore suggested that the promotion of autonomy is an important factor in treatment across modalities and can be distinguished from therapeutic alliance per se.

Given its focus on facilitating autonomous motivation, not surprisingly SDT has increasingly been used as a guiding framework for clinical interventions and randomized clinical trials in behavior change (Ryan, Patrick, Deci, & Williams, 2008). In fact clinical studies applying SDT attest to the positive consequences of autonomy-supportive treatment climates, including higher treatment attendance, less dropout, less relapse, and enhanced well-being over the course of treatment. Such results have been obtained in various domains such as drug and alcohol dependence (Foote et al., 1999; Ryan, Plant, & O'Malley, 1995; Zeldman, Ryan, & Fiscella, 2004), weight loss (Williams, Grow, Freedman, Ryan, & Deci, 1996), smoking cessation (Williams, McGregor, et al., 2006), general medication adherence (Williams, Rodin, Ryan, Grolnick, & Deci, 1998), HIV-related medication adherence (Kennedy, Gogin, & Nollen, 2004), eating disorders (Van der Kaap-Deeder et al., 2014), and other issues.

Beyond the importance of autonomy for behavior change is its importance in restorative self-development and personality growth. These principles are critical both in psychotherapy and in programs that provide support and training for caregivers. Particularly germane to the issue of motivation in growth-focused settings is a focus on *autonomy support*, which conduces exploration, disclosure, and integration (Weinstein, Przybylski, & Ryan, 2012). Specific behaviors associated with autonomy support include (1) taking the child's or adolescent's internal frame

of reference and welcoming negative feelings and resistance; (2) providing a meaningful rationale for requests and activities; (3) minimizing external controls such as contingent rewards and punishments or conditional regard, instead nurturing inner motivational resources (e.g., challenge, curiosity); (4) providing opportunities for participation, input, and choice; and (5) following the child's rhythm of progress and development (Reeve, 2009; Ryan & Deci, 2008). In autonomy-supportive contexts, pressure to engage in specific behaviors or express specific attitudes is minimized, and individuals are encouraged to base their actions on their own reflective considerations and values. Thus, autonomy for behavior is facilitated insofar as actors are helped to identify and own their reasons for changing or not changing behaviors, and do not feel pressured toward specific outcomes. This in turn leads to greater internalization and maintenance of change.

In addition to experiencing autonomy, internalization also requires that a person experience the confidence and competence to change. In SDT, *competence support* is afforded when practitioners provide the effectance-relevant inputs of structure and feedback (Jang, Reeve, & Deci, 2010; Sierens, Vansteenkiste, Goossens, Soenens, & Dochy, 2009). Clients are afforded the skills and tools for change and are empathically supported when efficacy-related barriers emerge. In the SDT model of change, gaining a sense of competence is facilitated by autonomy, in that, once people are volitionally engaged and have a high degree of willingness to act, they are then most apt to learn and apply new strategies and competencies (Markland et al., 2005). At that point providing additional challenges and structure can be especially helpful.

Finally SDT considers *relatedness support* as critical in both growth and internalization processes. Unconditional positive regard and helpful, but not intrusive, involvement facilitate relatedness experiences, which support interest and curiosity, as well as the trust and connectedness important to internalization. However, unconditional regard is not merely a technique, and it must be perceived as authentic or genuine to have the functional significance of relational support (Ryan, 1995).

These general principles can be actively applied in programs to train teachers (e.g., Aelterman, et al., in press; Cheon, Reeve, & Moon, 2012) or parents to adopt a more need-supportive approach, as to enhance developmental outcomes and lessen psychopathology. For example, Joussemet, Mageau, and Koestner (2013) studied an intervention program called the *How-to Parenting Program,* which included components meant to enhance provision of structure, greater relatedness and more support for autonomy. Results indicated the program succeeded in increasing these parenting attributes relative to baseline, and moreover, the level of children's internalizing and externalizing symptoms were significantly decreased, suggesting program effectiveness.

Future translational efforts must also move beyond tertiary efforts at psychotherapy and behavior change, toward more prevention, and this is a goal for which SDT's body of knowledge is uniquely suited. SDT, as an empirically supported framework, points both to general facilitating factors (e.g., psychological need satisfactions), and criteria for testing any specific strategies for enhancing these factors (i.e., measurements of targeted motivational mediating variables). There is thus great promise ahead for both intervention and process studies of behavior change and for creating conditions in families, schools, and communities that build resilience through fostering capacities for autonomy.

SDT AND THE STUDY OF DEVELOPMENT AND PSYCHOPATHOLOGY: CONCLUSIONS AND IMPLICATIONS

Among the central aims of SDT is the explication of the social-contextual conditions that promote optimal development of self. Thus, we have focused considerable attention on specifying the conditions that both facilitate and undermine optimal development. In this way, basic psychological needs are constructs that link SDT's efforts in understanding growth and positive experience, to the study of ill-being and the development of psychopathology. It is our contention that many of the processes that are integral to nonoptimal functioning in normal populations are also central to various psychopathologies. Indeed, we see continuity in the basic psychological needs for autonomy, competence, and relatedness across people's personality differences, developmental epochs, and cultures, suggesting that thwarting of these basic needs, especially when being severe, is a critical component of psychopathology and more normal or transitory maladjustment.

The developmental antecedents of disturbed autonomy are manifold, with genetic, biological, interpersonal, and sociocultural factors all being relevant (Cicchetti & Dawson, 2002; Ryan & Hawley, in press). Genetic and other biological factors enter transactionally into interpersonal relationships, facilitating or forestalling the quality of these social contextual inputs and thus constituting an obstacle or affordance for experienced need satisfaction, and cultural factors both shape and are emergent from patterns of social and familial functioning. Our focus has

been primarily on the social and familial factors, although our aim was not to provide a complete account of the development of autonomy disturbances. Rather, we have attempted to describe the phenomenological significance of autonomy in normal and pathological development and to show empirically and theoretically how interpersonal factors contribute to the etiology of pathologically disturbed autonomy.

Within this approach, we have viewed the development of autonomy as proceeding most effectively in familial and social contexts that provide autonomy support, optimal structure, and interpersonal involvement. In the absence of these necessary social nutrients—in contexts that thwart satisfaction of the needs for autonomy, competence, and relatedness—disturbed self-development is expected, resulting in the emergence of psychopathology. Psychopathology is thus the result of disorganizing influences, of contexts that thwart or forestall personality integration.

We have reviewed a large number of studies indicating that the development of autonomy—that is, the maintenance of intrinsic motivation, the internalization of values and regulatory processes, the integration of emotions, and the formation of identities—is facilitated by the contextual nutrients of caregiver attention and interest, and of encouragement for exploration and self-initiation. Contexts where interpersonal involvement and autonomy support are absent have been found reliably to diminish autonomous regulation and impair the development of self. The contextual elements that have consistently been found in our studies to impair autonomy and development—namely, controlling or uninvolved parenting—have also been emphasized in the clinical literature on the antecedents of disorders that involve either heteronomous introjects or failures of internalization. Thus, there appear to be clear parallels between the results of the empirical explorations of autonomy dynamics in normative development and the conclusions from clinical studies of psychopathology. Indeed, basic psychological needs represent constructs linking our understanding of both growth and pathology in self-development (Vansteenkiste & Ryan, 2013).

Future Directions

SDT focuses on the role of basic psychological needs in both growth and pathology. What is exciting about the convergence between SDT-grounded research results on both healthy populations and those experiencing psychopathology is that it sets the stage for more integrative empirical investigations in which the concepts of autonomy, competence, and relatedness needs figure heavily. For example, the constructs concerning intrinsic motivation, internalization, and autonomy-supportive relationships all bear on clinical phenomena, and they help us see the continuity of human needs in diverse populations. Whereas theories of internalization have been widely discussed in developmental and clinical literatures by such seminal writers as Freud, Mahler, Erikson, Perry, Piaget, and Kohlberg to name a few, there has been a paucity of empirically based studies of the process, and little specific theory about factors that facilitate versus undermine internalization in clinical settings. SDT's empirical focus provides testable hypotheses, amenable to both experimental and field methods, concerning both how to measure internalization, and its antecedents, consequences, and neuropsychological correlates.

More detailed work on the antecedents of specific autonomy disturbances within the various disorders will also be important. It is clear, for example, that the lack of autonomy support and genuine relatedness by caregivers are antecedent to the development of a wide range of pathologies (Ryan et al., 2006). Yet why an individual develops anorexia nervosa rather than pervasive self-critical depression in familial contexts that are demanding and critical is an example of the kinds of questions that are important to tackle empirically, and in coordination with epigenetic and neuroscience findings. Although failures of autonomy support appear to supply a generalized stressor to integrated development, how this differentially impacts and interacts with varied diatheses and vulnerabilities is an area for further inquiry. We know far too little about the specific pathways to specific disorders, leaving the study of factors that conduce to or elicit particular pathologies an important agenda for continued research.

The theoretical framework of SDT specifically distinguishes autonomy from independence, and this distinction is one we see as particularly critical to the field of developmental psychopathology, as well as to theories of parenting, attachment, and development more generally. Noting that both independence and dependence can be either heteronomous or autonomous can help future researchers disentangle what heretofore has been a very mixed and confused literature on dependence across the life span. In our view, dependence should be viewed not only as a potential problem but also as a positive capacity. The willingness to rely on and receive support from others is a basic human propensity that is fostered when autonomy is supported (Ryan et al., 2005). Future research can thus build on this distinction, examining more clearly the pros and cons of dependencies in different developmental epochs and interpersonal contexts. It can also inform

the growing literatures on psychopathology as it relates to gender and culture, where issues of autonomy and independence have too often been melded and confused (Chirkov, Ryan, & Sheldon, 2011).

More generally, we have pointed to the connections between the development and integration of personality and the phenomenological experience of autonomy in the regulation of behavior. In our view, the issue of autonomy is a critical one for organizational perspectives on developmental psychopathology. The experience of autonomy is a defining feature of organization, whereas disturbances of autonomy that correspond to fragmentation and disorganization in psychological development are also manifest as experiences of control and amotivation. We have further pointed to environmental conditions that either thwart or nurture needs for autonomy, competence, and relatedness as influential factors in development either away from or toward greater organization and integrity, respectively. The differentiated study of how these psychological needs, in interaction with the biological and social conditions of development, result in relative integration and healthy self-regulation supplies a broad and important agenda for future clinical research, and for the development of a knowledge base applicable in clinical interventions.

Because of their fundamental importance in psychological development and their pervasive expression in human behaviors, there is clearly more to know about the nature of basic psychological needs, their evolution, and their mechanistic underpinnings. Moreover, research is only nascent regarding their dynamic interaction with physiological or physical needs (Chen, Van Assche, Vansteenkiste, Beyers, & Soenens, in press). Finally, there is much more research needed about micro- and macrosocial contextual factors impacting their satisfaction or frustration. Progress is rapidly being made on the neuropsychological underpinnings of autonomous and controlled forms of motivation, as we have reviewed. However, much of this knowledge is still at the descriptive level and has yet to be coordinated with genetic studies, and the differential expression of human capacities for growth and defense over development. For example, we know that need thwarting impacts more than immediate experience and behavior and can have cascading effects in multiple areas, from social adjustment to executive functioning (e.g., Bernier, Carlson, & Whipple, 2010; Vansteenkiste & Ryan, 2013). Understanding how need frustrations have these arrayed effects is a matter that will require both more refined empirical and theoretical efforts at every level of analysis.

Developmental psychology, in fact, offers windows into our evolved tendencies and their selective advantages (Ryan & Hawley, in press; Tomasello, 2008). The fact that basic psychological needs universally play a role in healthy psychological development and wellness suggests that further studies, both genetic and comparative, will add to our understanding of their role in adaptation and maladaptation. More generally, SDT has grown very rapidly in recent years, and its body of evidence contains studies of persons in every part of the life span. Yet there needs to be much deeper understanding of both how need satisfactions change over time in character and focus and how they support developmental progressions in multiple developmental lines.

As a universalistic, organismic perspective on development and personality functioning SDT is also inherently a cross-cultural psychology. Cultures, by their behavioral norms, environmental affordances, and familial practices differentially impact need satisfactions and frustrations. Although the cross-cultural research in SDT has largely focused to date on showing commonalities in effects of satisfaction and frustration of basic needs across groups, domains and cultures, as a maturing scientific perspective it is also time for SDT to take closer interest in the differences these contexts set in motion. For instance, there may exist some cross-cultural variability in the way the psychological needs get nurtured and satisfied (Soenens, Vansteenkiste, & Van Petegem, in press). Along with this, greater attention is needed on cultural differences that enhance or undermine the adopted and internalization of need supportive tertiary and preventive interventions.

Beyond culture we also need more empirical research on how conditions of impoverishment and oppression at cultural levels impact developmental outcomes as mediated by need supports and thwarts. Both proximal and global conditions affect human psychological need satisfactions, so just as we need to coordinate the study of psychological needs with their mechanistic underpinnings, understanding the more global social conditions that affect human wellness is critical to research that can inform policies on child welfare, education, and the necessary social supports to facilitate optimal development for children of all social backgrounds. Illustratively, two recent studies demonstrate how differences in socioeconomic status and the presence of income inequalities negatively affect people's physical and mental health (Di Domenico & Fournier, 2014; Gonzales, Swanson, Lynch, & Williams, 2014). Insofar as conditions of poverty and deprivation conduce to psychopathology through the frustration of basic needs, global efforts to address such conditions and to establish greater social justice deserve our attention both as clinical scientists and as world citizens.

REFERENCES

Abuhamdeh, S., & Csikszentmihalyi, M. (2012). The importance of challenge for the enjoyment of intrinsically motivated, goal-directed activities. *Personality and Social Psychology Bulletin, 38,* 317–330. doi: 10.1177/0146167211427147

Aelterman, N., Vansteenkiste, M., Van den Berghe, L., De Meyer, J., & Haerens, L. (in press). Fostering a need-supportive teaching style: Intervention effects on physical education teachers' beliefs and teaching behaviors. *Journal of Sport & Exercise Psychology*.

Ahmad, I., Vansteenkiste, M., & Soenens, B. (2013). The relations of Arab Jordanian adolescents' perceived maternal parenting to teacher-rated adjustment and problems: The intervening role of perceived need satisfaction. *Developmental Psychology, 49*, 177–183.

Ainsworth, M. D. S., Blehar, M. C, Waters, E., & Wall, S. (1978). *Patterns of attachment*. Hillsdale, NJ: Erlbaum.

Amabile, T. M. (1983). *The social psychology of creativity*. New York, NY: Springer- Verlag.

Aunola, K., & Nurmi, J. E. (2005). The role of parenting styles in children's problem behavior. *Child Development*, *76*, 1144–1159.

Appiah, A. K. (2005). *The ethics of identity*. Princeton, NJ: Princeton University Press.

Assor, A. (2012). Allowing choice and nurturing an inner compass: Educational practices supporting students' need for autonomy. In S. L. Christenson, A. L. Reschly & C. Wylie (Eds.), *Handbook of research on student engagement* (pp. 421–439). New York, NY: Springer.

Assor, A., Roth, G., & Deci, E. L. (2004). The emotional costs of parents' conditional regard: A self-determination theory analysis. *Journal of Personality, 72*, 47–88. doi: 10.1111/j.0022-3506.2004.00256.x

Assor, A., Vansteenkiste, M., & Kaplan, A. (2009). Identified versus introjected approach and introjected avoidance motivations in school and in sports: The limited benefits of self-worth strivings. *Journal of Educational Psychology, 101,* 482–497. doi: 10.1037/a0014236

Assor, A., & Tal, K. (2012). When parents' affection depends on child's achievement: Parental conditional positive regard, self-aggrandizement, shame and coping in adolescents. *Journal of Adolescence*, *35*, 249–260. doi: 10.1016/j.adolescence.2011.10.004

Baldwin, A. L (1955). *Behavior and development in childhood*. New York, NY: Dreyden.

Bandura, A. (1989). Human agency in social cognitive theory. *American Psychologist, 44*, 1175–1184. doi: 10.1037/0003-066X.44.9.1175

Barber, B. K. (1996). Parental psychological control: Revisiting a neglected construct. *Child Development, 67*, 3296–3319. doi: 10.1111/j.1467-8624.1996.tb01915.x

Barber, B. K., Stolz, H. E., & Olsen, J. A. (2005). Parental support, psychological control, and behavioral control: Assessing relevance across time, method, and culture. *Monographs of the Society for Research in Child Development, 70*(4, Serial No. 281).

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117,* 497–529. doi: 10.1037/0033-2909.117.3.497

Baumrind, D. (1996). The discipline controversy revisited. *Family Relations, 45,* 405–414. doi: 10.2307/585170

Baumrind, D. (2012). Differentiating between confrontive and coercive kinds of parental power-assertive disciplinary practices. *Human Development, 55,* 35–51. doi: 10.1159/000337962

Bayer, J. K., Sanson, A. V., & Hemphill, S. A. (2006). Parent influences on early childhood internalizing difficulties. *Journal of Applied Developmental Psychology, 27 (6),* 542–559. doi: 10.1016/j.appdev.2006.08.002

Bechara, A., Tranel, D., Damasio, H., & Damasio, A. R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cerebral Cortex, 6*, 215–225 doi: 10.1093/cercor/6.2.215

Beck, A. T. (1983). Cognitive treatment of depression: New perspectives. In P. J. Clayton & J. E. Barrett (Eds.), *Treatment of depression: Old controversies and new approaches* (pp. 265–290). New York, NY: Raven Press.

Belsky, J. (2002). Developmental origins of attachment styles. *Attachment and Human Development, 4 (2),* 166–170.

Benjamin, L. S. (2003). *Interpersonal diagnosis and treatment of personality disorders*. New York, NY: Guilford.

Bernier, A., Carlson, S. M., & Whipple, N. (2010). From external regulation to self-regulation: Early parenting precursors of young children's executive functioning. *Child Development, 81,* 326–339.

Blais, M. R., Sabourin S., Boucher, C, & Vallerand, R. J. (1990). Toward a motivational model of couple happiness. *Journal of Personality and Social Psychology, 59*, 1021–1031. doi: 10.1037/0022-3514.59.5.1021

Blatt, S. J. (1974). Levels of object representation in anaclitic and introjective depression. *Psychoanalytic Study of the Child, 29*, 107–157.

Bleiberg, E. (2004). *Treating personality disorders in children and adolescents: A relational approach*. New York, NY: Guilford.

Blos, P. (1979). *The adolescent passage*. New York, NY: International Universities Press.

Bober, S., & Grolnick, W. S. (1995). Motivational factors related to differences in self-schemas. *Motivation & Emotion,19*, 307–327. doi: 10.1007/BF02856517

Boone, L., Vansteenkiste, M., Soenens, B., Van der kaap-Deeder, J., & Verstuy f, J. (2014). Self-critical perfectionism and binge eating symptoms: A longitudinal test of the intervening role of psychological need frustration. *Journal of Counseling Psychology, 61,* 363–373. doi: 10.1037/a0036418

Bouchard, G., Lee, C. M., Asgary, V., & Pelletier, L. G. (2007). Fathers' motivation for involvement with their children: A self-determination theory perspective. *Fathering*, *5*, 25–41.

Bowlby, J. (1969). *Attachment and loss: Vol. 1: Attachment*. New York, NY: Basic Books.

Bradley, S. J. (2000). *Affect regulation and the development of psychopathology*. New York, NY: Guilford.

Brenning, K., Soenens, B., Braet, C., & Bal, S. (2012). The role of parenting and mother-adolescent attachment in the intergenerational similarity of internalizing symptoms. *Journal of Youth and Adolescence, 41,* 802–816. doi: 10.1007/s10964-011-9740-9

Brenning, K., Soenens, B., Van Petegem, S., & Vansteenkiste, M. (in press). Perceived maternal autonomy support and early adolescent emotion regulation: A longitudinal study. *Social Development*.

Bretherton, I. (1987). New perspectives on attachment relations: Security, communication and internal working models. In J. Osofsky (Ed.), *Handbook of infant development* (pp. 1061–1100). New York, NY: Wiley.

Bruch, H. (1973). *Eating disorders*. New York, NY: Basic Books.

Bruch, H. (1979). *The golden cage: The enigma of anorexia nervosa*. New York, NY: Vintage Books.

Brunstein, P. (1994). Patterns of parent-child interaction in Mexican families: A cross-cultural perspective. *International Journal of Behavioral Development, 17*, 423–446.

Burnette, M. L., & Cicchetti, D. (2012). Multilevel approaches toward understanding anti-social behavior: Current research and future directions. *Development and Psychopathology, 24*, 703–704. doi: 10.1017/S0954579412000314

Cacioppo, J. T., Berntson, G. G., Sheridan, J. F., & McClintock, M. K. (2000). Multilevel integrative analyses of human behavior: Social neuroscience and the complementing nature of social and biological approaches. *Psychological Bulletin, 126*, 829–843. doi: 10.1037//0033-2909.126.6.829

Calkins, S. D., & Johnson, M. C. (1998). Toddler regulation of distress to frustrating events: Temperamental and maternal correlates. *Infant Behavior and Development*, *21*(3), 379–395.

Carlson, E. A., Yates, T. M., & Sroufe, A. L. (2011). Dissociation and the development of self. In P. F. Dell & J. A. O'Neil (Eds.), *Dissociation and the dissociative disorders: DSM V and beyond* (pp. 39–53). New York, NY: Routledge

Cerasoli, C. P., Nicklin, J. M., & Ford, M. T. (2014). Intrinsic motivation and extrinsic incentives jointly predict performance: A 40-year meta-analysis. *Psychological Bulletin, 140*(4), 980–1008. doi: 10.1037/a0035661

Chambers, R. A., Taylor, J. R., & Potenza, M. N. (2003). Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *American Journal of Psychiatry, 160*, 1041–1052. doi: 10.1176/appi.ajp.160.6.1041

Chen, B., Vansteenkiste, M., Beyers, W., Soenens, B., & Van Petegem, S. (2013). Autonomy in family decision making for Chinese adolescents: Disentangling the dual meaning of autonomy. *Journal of Cross-cultural Psychology, 44,* 1184–1209. doi: 10.1177/0022022113480038.

Chen, B., Vansteenkiste, M., Beyers, W., Boone, L., Deci, E. L., Duriez, B. . . . , Verstuyf, J. (in press). Basic psychological need satisfaction, need frustration, and need strength across four cultures. *Motivation and Emotion*.

Chen, B., Van Assche, J., Vansteenkiste, M., Soenens, B., & Beyers, W. (in press). Does psychological need satisfaction matter when environmental or financial safety are at risk? *Journal of Happiness Studies*.

Cheon, S. H., Reeve, J., & Moon, I. S. (2012). Experimentally based, longitudinally designed, teacher-focused intervention to help physical education teachers be more autonomy-supportive toward their students. *Journal of Sport & Exercise Psychology, 34*, 365–396.

Cheung, C. S. S., & Pomerantz, E. M. (2011). Parents' involvement in children's learning in the United States and China: Implications for children's academic and emotional adjustment. *Child Development, 82 (3),* 932–950. doi: 10.1111/j.1467-8624.2011.01582.x

Chirkov, V., & Ryan, R. M. (2001). Parent and teacher autonomy-support in Russian and U.S. adolescents: Common effects on well-being and academic motivation. *Journal of Cross-Cultural Psychology, 32*, 618–635. doi: 10.1177/0022022101032005006

Chirkov, V. I., Ryan, R. M., & Sheldon, K. M. (Eds.) (2010). *Human autonomy in cross-cultural context*. New York, NY: Springer.

Chua, S. N., Wong, N., & Koestner, R. (2014). Autonomy and controlling support are two sides of the same coin. *Personality and Individual Differences, 68*, 48–52. doi: 10.1016/j.paid.2014.04.008

Cicchetti, D. (2006). Development and psychopathology. In D. Cicchetti & D. Cohen (Eds.) *Developmental psychopathology: Theory and methods* (2nd ed., Vol. 1, pp. 1–24). New York, NY: Wiley.

Cicchetti, D., & Dawson, G. (2002). Editorial: Multiple levels of analysis. *Development and Psychopathology*, *14*, 417–420. doi: 10.1017/S0954579402003013

Cicchetti, D., Ganiban, J., & Barnett, D. (1991). Contributions from the study of high-risk populations to understanding the development of emotion regulation. In J. Garber & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp. 15–48). New York, NY: Cambridge University Press.

Cicchetti D., Toth S. L. (2009). The past achievements and future promises of developmental psychopathology: The coming of age of a discipline. *Journal of Child Psychology and Psychiatry*, *50*, 16–25. doi: 10.1111/j.1469-7610.2008.01979.

Cicchetti, D., & Valentino, K. (2006). An ecological transactional perspective on child maltreatment: Failure of the average expectable environment and its influence on child development. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 3. Risk, disorder and adaptation* (2nd ed., pp. 129–201). New York, NY: Wiley.

Claes, L., Soenens, B., Vansteenkiste, M., & Vandereycken, W. (2012). The scars of the inner critic: Perfectionism and non-suicidal self-injury in eating disorders. *European Eating Disorders Review, 20*, 196–202 doi: 10.1002/erv.1158

Coie, J. D., & Jacobs, M. R. (1993). The role of social context in the prevention of conduct disorder. *Development and Psychopathology, 5*, 263–27. doi: 10.1017/S0954579400004387

Curran, T., Hill, A. P., & Niemiec, C. P. (2013). A conditional process model of children's behavioral engagement and behavioral disaffection in sport based on self-determination theory. *Journal of Sport & Exercise Psychology, 35*, 30–43

Danner, E W., & Lonky, E. (1981). A cognitive-developmental approach to the effects of rewards on intrinsic motivation. *Child Development, 52*, 1043–1052. doi: 10.1111/j.1467-8624.1981.tb03147.x

Deci, E. L. (1971). Effects of externally mediated rewards on intrinsic motivation. *Journal of Personality and Social Psychology, 18,* 105–115. doi: 10.1037/h0030644

Deci, E. L. (1972). Intrinsic motivation, extrinsic reinforcement, and inequity. *Journal of Personality and Social Psychology. 22*, 113–120. doi: 10.1037/h0032355

Deci, E. L., & Cascio, W. F. (1972, April). *Changes* in *intrinsic motivation as a function of negative feedback and threats*. Paper presented at the meeting of the Eastern Psychological Association, Boston, MA.

Deci, E. L., Driver, R. E., Hotchkiss, L., Robbins, R. J., & Wilson, I. M. (1993). The relation of mothers' controlling vocalizations to children's intrinsic motivation. *Journal of Experimental Child Psychology, 55*, 151–162. doi: 10.1006/jecp.1993.1008

Deci, E. L., Koestner, R., & Ryan, R. M. (1999). A meta-analytic review of experiments examining the effects of extrinsic rewards on intrinsic motivation. *Psychological Bulletin, 125*, 627–668 doi: 10.1037/0033-2909.125.6.627

Deci, E. L., & Ryan, R. M. (1985a). The general causality orientations scale: Self-determination in personality. *Journal of Research in Personality, 19,* 109–134. doi: 10.1016/0092-6566(85)90023-6

Deci, E. L., & Ryan, R. M. (l985b). Intrinsic motivation and self-determination *in* human behavior. New York, NY: Plenum.

Deci, E. L., & Ryan, R. M. (1995). Human autonomy: The basis for true self-esteem. In M. Kernis (Ed.), *Efficacy, agency, and self-esteem* (pp. 31–49). New York, NY: Plenum.

Deci, E. L., & Ryan, R. M. (2000). The "what" and "why" of goal pursuits: Human needs and the self-determination of behavior. *Psychological Inquiry, 11*, 227–268. doi: 10.1207/S15327965PLI1104_01

Deci, E. L., & Ryan, R. M. (2012). Motivation, personality, and development within embedded social contexts: An overview of self-determination theory. In R. M. Ryan (Ed.), *The Oxford handbook of motivation* (pp. 85–107). New York, NY: Oxford University Press.

Deci, E. L., & Ryan, R. M. (2013). The importance of autonomy for development and well-being. In B. Sokol, F. M. E. Grouzet, & U. Muller (Eds.), *Self-regulation and autonomy: Social, developmental, educational, and neurological dimensions of human contact* (pp. 19–46). New York, NY: Cambridge University Press.

Deci, E. L., Ryan, R. M., Schultz, P. P., & Niemiec, C. P. (2014). Being aware and functioning fully: Mindfulness and interest-taking within

self-determination theory. In K. W. Brown, R. M. Ryan, & J. D. Creswell (Eds.), *Handbook of mindfulness*. New York, NY: Guilford.

Deci, E. L., Schwartz, A. J., Sheinman, L., & Ryan, R. M. (1981). An instrument to assess adults' orientations toward control versus autonomy with children: Reflections on intrinsic motivation and perceived competence. *Journal of Educational Psychology, 73*, 642–650. doi: 10.1037//0022-0663.73.5.642

De Charms, R. (1968). *Personal causation: The internal affective determinants of behavior*. New York, NY: Academic Press.

de Haan, A. D., Soenens, B., Dekovic, M., & Prinzie, P. (2013). Effects of childhood aggression on parenting during adolescence: The role of parental psychological need satisfaction. *Journal of Clinical Child & Adolescent Psychology, 42*, 393–404. doi: 10.1080/15374416.2013.769171

De Naeghel, J., Van Keer, H., Vansteenkiste, M., & Rosseel, Y. (2012). The relation between elementary students' recreational and academic reading motivation, reading frequency, engagement and comprehension: A self-determination theory perspective. *Journal of Educational Psychology, 104,* 1006–1021 doi: 10.1037/a0027800

Depue, R. A., & Lenzenweger, M. F. (2001). A neurological dimensional model. In W. J. Livesley (Ed.), *Handbook of personality disorders: theory, research, and treatment* (pp. 136–176). New York, NY: Guilford.

Di Domenico, S. I., & Fournier, M. A. (2014). Socioeconomic status, income inequality and health complaints: A basic psychological needs perspective. *Social Indicators Research*, DOI 10.1007/s11205-013-0572-8

Di Domenico, S. I., Fournier, M. A., Ayaz, H., & Ruocco, A. C. (2013). In search of integrative processes: Basic psychological need satisfaction predicts medial prefrontal activation during decisional conflict. *Journal of Experimental Psychology: General, 142*, 967–978. doi: 10.1037/a0030257

Dittmar, H. (2011). Material and consumer identities. In S. Schwartz, K. Luyckx, & V. Vignoles (Eds.), *Handbook of identity theories and research* (p. 745). New York, NY: Springer.

Dworkin, G. (1988). *The theory and practice of autonomy*. New York, NY: Cambridge.

Eisenberger, R., & Cameron, J (1996). Detrimental effects of reward: Reality of myth? *American Psychologist, 51*, 1153–1166. doi: 10.1037/0003-066X.51.11.1153

Elkind, D. (1967). Egocentrism in adolescence. *Child Development, 38*. 1025–1034. doi: 10.1111/j.1467-8624.1967.tb04378.x

Enns, M. W., Cox, B. J., & Clara, I. (2002). Adaptive and maladaptive perfectionism: Developmental origins and association with depression proneness. *Personality & Individual Differences, 33*, 921–935. doi: 10.1016/S0191-8869(01)00202-1

Enzle, M. E., & Anderson, S. C. (1993). Surveillant intentions and intrinsic motivation. *Journal of Personality and Social Psychology*, *64*(2), 257–266. doi: 10.1037/0022-3514.64.2.257

Erikson, E. H. (1950). *Childhood and society*. New York, NY: Norton.

Farkas, M. S., & Grolnick, W. S. (2010). Examining the components and concomitants of parental structure in the academic domain. *Motivation and Emotion*, *34*, 266–279. doi: 10.1007/s11031-010-9176-7

Flavell, J. H., Miller, P. H., & Miller, S. A. (2002). *Cognitive development*. Englewood Cliffs, NJ: Prentice Hall.

Flett, G. L., Hewitt, P. L., & Singer, A. (1995). Perfectionism and parental authority styles. Individual Psychology. *Journal of Adlerian Theory, Research & Practice, 51*, 50–60.

Foote, J., DeLuca, A., Magura, S., Warner, A., Grand, A., Rosenblum, A., & Stahl, S. (1999). A group motivational treatment for chemical dependency. *Journal of Substance Abuse Treatment, 17,* 181–192. doi: 10.1016/S0740-5472(99)00003-3

Freud, A. (1966). *The ego and the mechanisms of defense*. New Your, NY: International Universities Press.

Friedman, M. (2003). *Autonomy, gender, politics*. New York, NY: Oxford University Press.

Frodi, A., Bridges, L., & Grolnick, W. S. (1985). Correlates of mastery-related behavior: A short term longitudinal study of infants in their second year. *Child Development, 56*, 1291–1298**.** DOI: 10.2307/1130244

Frost, R. O., Novara, C., & Rheaume, J. (2002). Perfectionism in obsessive compulsive disorder. In R. O. Frost & G. Steketee (Eds.), *Cognitive approaches to obsessions and compulsions: Theory, assessment, and treatment* (pp. 91–105). Amsterdam, The Netherlands: Pergamon Elsevier.

Fu, A. S., & Markus, H. R. (2014). My mother and me: Why tiger mothers motivate Asian American but Not European Americans. *Personality and Social Psychology Bulletin, 40,* 739–749. doi: 10.1177/0146167214524992

Gable, S. L., Reis, H. T., & Elliot, A. J. (2000). Behavioral activation and inhibition in everyday life. *Journal of Personality and Social Psychology, 78,* 1135–1149. doi: 10.1037//0022-3514.78.6.1135

Gershoff, E. T. (2013). Spanking and child development: We know enough now to stop hitting our children. *Child Development Perspectives, 7*, 133–137.

Gonzalez, M. G., Swanson, D. P., Lynch, M., & Williams, G. C. (2014). Testing satisfaction of basic psychological needs as a mediator of the relationship between socioeconomic status and physical and mental health. *Journal of Health Psychology*. doi: 10.1177/1359105314543962

Greenspan, S. I. (1979). *Intelligence and adaptation*. New York, NY: International Universities Press.

Grolnick, W. S. (2002). The psychology of parental control: How well-meant parenting backfires. *Adolescence, 37*, 858–859.

Grolnick, W. S. (2012). The relations among parental power assertion, control, and structure: Commentary on Baumrind. *Human Development, 55,* 57–64. doi: 10.1159/000338533

Grolnick, W. S. (in press). Mothers' motivation for involvement in their children's schooling: Mechanisms and outcomes. *Motivation and Emotion*.

Grolnick, W. S., Bridges, L., & Connell, J. P. (1996). Emotional self-regulation in two-year-olds: Strategies and emotional expression in four contexts. *Child Development, 67*, 928–941. doi: 10.1111/j.1467-8624.1996.tb01774.x

Grolnick, W. S., Bridges, L., & Frodi, A. (1984). Maternal control style and the mastery motivation of one-year-olds. *Infant Mental Health Journal, 5*, 72–82.

Grolnick, W. S., Gurland, S. T., DeCourcey, W., & Jacob, K. (2002). Antecedents and consequences of mothers' autonomy support: An experimental investigation. *Developmental Psychology, 38*, 143–155. doi: 10.1037//0012-1649.38.1.143

Grolnick, W. S., Kurowski, C. O., McMenamy, J. M., Rivkin, I., & Bridges, L. J. (1998). Mothers' strategies for regulating their toddlers' distress. *Infant Behavior and Development, 21,* 437–450. doi: 10.1016/S0163-6383(98)90018-2

Grolnick, W. S., & Pomerantz, E. M. (2009). Issues and challenges in studying parental control: Toward a new conceptualization. *Child Development Perspectives*, *3*, 165–170.

Grolnick, W. S., & Ryan, R. M. (1987). Autonomy in children's learning: An experimental and individual difference investigation. *Journal of Personality and Social Psychology, 52*, 890–898. doi: 10.1037/0022-3514.52.5.890

Grolnick, W. S., & Ryan, R. M. (1989). Parent styles associated with children's self-regulation and competence in school. *Journal of Educational Psychology, 81,* 143–154.

Grolnick, W. S., Ryan, R. M., & Deci, E. L. (1991). The inner resources for school achievement: Motivational mediators of children's perceptions of their parents. *Journal of Educational Psychology, 83*, 508–517. doi: 10.1037/0022-0663.83.4.508

Grolnick, W. S., Weiss, L., McKenzie, L., & Wrightman, J. (1996). Contextual, cognitive, and adolescent factors associated with parenting in adolescence. *Journal of Youth and Adolescence, 25*, 33–54. doi: 10.1007/BF01537379

Grolnick, W. S., Price, C. E., Beiswenger, K. L., & Sauck, C. C. (2007). Evaluative pressure in mothers: Effects of situation, maternal, and child characteristics on autonomy supportive versus controlling behavior. *Developmental Psychology*, *43*, 991–1002. doi: 10.1037/0012-1649.43.4.991

Gross, J. J., & John, O. P. (2002). Wise emotion regulation. In L. F. Barrett & P. Salovey (Eds.), *The wisdom in feeling: Psychological processes in emotional intelligence. Emotions and social behavior*. (pp. 297–319). New York, NY: Guilford Press.

Sokol, B. W., Grouzet, F. M. E., & Muller, U. (Eds.), (2013). *Self-regulation and autonomy: Social, developmental, educational, and neurological dimensions of human contact*. (pp. 191–207). New York, NY: Cambridge University Press.

Grusec, J. E., & Goodnow, J. J. (1994). Impact of parental discipline methods on the child's internalization of values: A reconceptualization of current points of view. *Developmental Psychology, 30*, 4–19. doi: 10.1037/0012-1649.30.1.4

Gurland, S. T., & Grolnick, W. S. (2005). Perceived threat, controlling parenting, and children's achievement orientations. *Motivation and Emotion*, *29*, 103–121. doi: 10.1007/s11031-005-7956-2

Harlow, H. F. (1950). Learning and satiation of response in intrinsically motivated complex puzzle performance by monkeys. *Journal of Comparative and Physiological Psychology, 43,* 289–294. DOI: 10.1037/h0058114

Harter, S. (2012). *The construction of the self*. New York, NY: Guilford.

Hartmann, H. (1964). On rational and irrational action. In *Essays on ego psychology*. New York, NY: International Universities Press. (Original work published 1947)

Herman, J. L., Perry, J. C., van der Kolk, B. A. (1989). Childhood trauma in borderline personality disorder. *American Journal of Psychiatry*, *146*, 490–495.

Hmel, B. A., & Pincus, A. L. (2002). The meaning of autonomy: On and beyond the interpersonal circumplex. *Journal of Personality, 70*, 277–310.

Hodgins, H. S., Koestner, R., & Duncan, N. (1996). On the compatibility of autonomy and relatedness. *Personality and Social Psychology Bulletin*, *22*, 227–237. doi: 10.1177/0146167296223001

Hodgins, H. S., Brown, A. B., & Carver, B. (2007). Autonomy and control motivation and self-esteem. *Self & Identity, 6*, 189–208. doi: 10.1080/15298860601118769

Hodgins, H. S., & Knee, C. R. (2002). The integrating self and conscious experience. In E. L. Deci & R. M. Ryan (Eds.), *Handbook of self-determination research* (pp. 87–100). Rochester, NY: University of Rochester Press.

Hodgins, H., Yacko, H., & Gottlieb, E. (2006). Autonomy and nondefensiveness. *Motivation and Emotion, 30*, 283–293. doi: 10.1007/s11031-006-9036-7

Hoffman, M. L. (1960). Power assertion by the parent and its impact on the child. *Child Development, 31,* 129–143.

Hooyman, A., Wulf, G., & Lewthwaite, R. (2014). Impacts of autonomy-supportive versus controlling instructional language on motor learning. *Human Movement Science, 36,* 190–198. doi: 10.1080/02640414.2014.922693

Iyengar, S. S., & DeVoe, S. E. (2003). Rethinking the value of choice: Considering cultural mediators of intrinsic motivation. In V. Murphy-Berman & J. J. Berman (Eds.), *Nebraska symposium on motivation: Cross-cultural differences in perspectives on self* (Vol. 49, pp. 129–174). Lincoln, NE: University of Nebraska Press.

Jaffee, S. R., Moffitt, T. E., Caspi, A., & Taylor, A. (2003). Life with (or without) father: The benefits of living with two biological parents depend on the father's antisocial behavior. *Child Development, 74 (1),* 109–126. doi: 10.1111/1467-8624.t01-1-00524

Jahoda, M. (1958). *Current concepts of positive mental health*. New York, NY: Basic Books.

Jang, H., Reeve, J., Ryan, R. M., & Kim, A. (2009). Can self-determination theory explain what underlies the productive, satisfying learning experiences of collectivistically oriented Korean students? *Journal of Educational Psychology*, *101*, 644–661. doi: 10.1037/a0014241

Jang, H., Kim, E. J., & Reeve, J. (2012). Longitudinal test of self-determination theory's motivation mediation model in a naturally occurring classroom context. *Journal of Educational Psychology, 104*(4), 1175–1188. doi: 10.1037/a0028089

Jang, H., Reeve, J., & Deci, E. L. (2010). Engaging students in learning activities: It's not autonomy support or structure, but autonomy support and structure. *Journal of Educational Psychology*, *102*, 588–600. doi: 10.1037/a0019682

Jenike, M. A. (1991). Obsessive-compulsive disorder. In B. D. Beitman & G. L. Klerman (Eds.), *Integrating pharmacotherapy and psychotherapy* (pp. 183–210). Washington, DC: American Psychiatric Association.

Johnson, J. G., Cohen, P., Chen, H., Kasen, S., & Brook, J. S. (2006). Parenting behaviors associated with risk for offspring personality disorder during adulthood. *Archives of General Psychiatry, 63*(5), 579–587. doi: 10.1001/archpsyc.63.5.579.

Jordan, J. V. (1991). The relational self: A new perspective for understanding women's development. In J. Strauss & G. R. Goethals (Eds.), *The self: Interdisciplinary approaches* (pp. 136–149).. New York, NY: Springer-Verlag.

Joussemet, M., Mageau, G., & Koestner, R. (2014). Promoting optimal parenting and children's mental health: A preliminary evaluation of the how-to parenting program. *Journal of Child and Family Studies, 23*, 949–964. DOI 10.1007/s10826-013-9751-0

Joussemet, M., Vitaro, F., Barker, E. D., Coté, S., Nagin, D., Zoccolillo, M., & Tremblay, R. E. (2008). Controlling parenting and physical aggression during elementary school. *Child Development*, *79*, 411–425. doi: 10.1111/j.1467-8624.2007.01133.x

Kanat-Maymon, M., & Assor, A. (2010). Perceived maternal control and responsiveness to distress as predictors of young adults' empathic responses. *Personality and Social Psychology Bulletin, 36 (1),* 33–46. doi: 10.1177/0146167209347381

Karavasilis, L., Doyle, A. B., & Markiewicz, D. (2003). Associations between parenting style and attachment to mother in middle childhood and adolescence. *International Journal of Behavioral Development, 27*(2), 153–164. doi: 10.1080/0165025024400015

Kasser, T. (2002). *The high price of materialism*. Cambridge, MA: MIT Press.

Kasser, T., & Ryan, R. M. (1996). Further examining the American dream: Differential correlates of intrinsic and extrinsic goals. *Personality and Social Psychology Bulletin, 22*, 80–87. doi: 10.1177/0146167296223006

Kasser, T., Ryan, R. M., Zax, M., & Sameroff, A. J. (1995). The relations of maternal and social environments to late adolescents' materialistic and prosocial values. *Developmental Psychology, 31*, 907–971. doi: 10.1037/0012-1649.31.6.907

Kasser, V. G., & Ryan, R. M. (1999). The relation of psychological needs for autonomy and relatedness to vitality, well-being, and mortality in a

nursing home. *Journal of Applied Social Psychology, 29*, 935–954. doi: 10.1111/j.1559-1816.1999.tb00133.x

Katz, I., & Assor, A. (2007). When choice motivates and when it does not. *Educational Psychology Review*, *19*, 429–442. doi: 10.1007/s10648-006-9027-y

Kawamura, K. Y., Frost, R. O., & Harmatz, M. G. (2002). The relationship of perceived parenting styles to perfectionism. *Personality & Individual Differences, 32*, 317–327. doi: 10.1016/S0191-8869(01)00026-5

Kennedy, S., Gogin, K., & Nollen, N. (2004). Adherence to HIV medications: Utility of the theory of self-determination. *Cognitive Therapy and Research, 28,* 611–628. doi: 10.1023/B:COTR.0000045568.95219.e2

Kernis, M. H. (2003). Toward a conceptualization of optimal self-esteem. *Psychological Inquiry, 14*, 1–26. doi: 10.1207/S15327965PLI1401_01

Kim, S., Kochanska, G., Boldt, L. B., Nordling, J. K., & O'Bleness, J. J. (2014). Developmental trajectory from early responses to transgressions to future antisocial behavior: Evidence for the role of the parent–child relationship from two longitudinal studies. *Development and Psychopathology, 26*(1), 93–109. doi: 10.1017/S0954579413000850

Knafo, A., & Assor, A. (2007). Motivation for agreement with parental values: Desirable when autonomous, problematic when controlled. *Motivation and Emotion, 31,* 232–245.

Kobak, R. Cassidy, J., & Zir, Y (2004). Attachment-related trauma and post-traumatic stress disorder. In W. S. Rholes & J. A. Simpson (Eds.), *Adult attachment: theory research and clinical implications* (pp. 388–407). New York, NY: Guilford.

Koestner, R., & McClelland, D. C. (1990). Perspectives on competence motivation. In L. Pervin (Ed.), *Handbook of personality: Theory and research* (pp. 527–548). New York, NY: Guilford.

Koestner, R., Bernieri, E., & Zuckerman, M. (1992). Self-regulation and consistency between attitudes, traits, and behaviors. *Personality and Social Psychology Bulletin, 18,* 52–59. doi: 10.1177/0146167292181008

Koestner, R., Ryan, R. M., Bernieri, E., & Holt, K. (1984). Setting limits on children's behavior: The differential effects of controlling versus informational styles on intrinsic motivation and creativity. *Journal of Personality, 52*, 233–248. doi: 10.1111/j.1467-6494.1984.tb00879.x

Krapp, A. (2002). Structural and dynamic aspects of interest development: Theoretical considerations from an ontogenetic perspective. *Learning and Instruction, 12,* 383–409. doi: 10.1016/S0959-4752(01)00011-1

Kroger, J., & Marcia, J. E. (2011). The identity statuses: Origins, meanings and interpretations. In S. J. Schwartz, K. Luyckx, & V. L. Vignoles (Eds.), *Handbook of identity theory and research* (pp. 31–54). New York, NY: Springer.

Kusurkar, R. A., Ten Cate, T. J., Vos, C. M. P., Westers, P., & Croiset, G. (2013). How motivation affects academic performance: A structural equation modelling analysis. *Advances in Health Sciences Education*, *18*, 57–61. doi: 10.1007/s10459-012-9354-3

La Guardia, J. G., Ryan, R. M., Couchman, C. E., & Deci, E. L. (2000). Within-person variation in security of attachment: A self-determination theory perspective on attachment, need fulfillment, and well-being. *Journal of Personality and Social Psychology, 79*, 367–384. doi: 10.1037//0022-3514.79.3.367

Lamb, M. E., & Easterbrooks, M. A. (1981). Individual differences in parental sensitivity: Origins, components, and consequences. In M. E. Lamb & L. R. Sherrod (Eds.), *Infant social cognition: Empirical and theoretical considerations* (pp. 127–154). Hillsdale, NJ: Erlbaum.

Landry, R., Whipple, N., Mageau, G. A., Joussemet, M., Gingras, I., Didio, L., & Koestner, R. (2008). Trust in organismic development, autonomy support, and adaptation among mothers and their children: A self-determination theory approach to parenting. *Motivation & Emotion*, *32*, 173–188. doi: 10.1007/s11031-008-9092-2

Lee, W., & Reeve, J. (2013). Self-determined, but not non-self-determined, motivation predicts activations in the anterior insular cortex: An fMRI study of personal agency. *Social Cognitive Affective Neuroscience, 8,* 538–545. DOI: 10.1093/scan/nss029

Lekes, N., Gingras, I., Philippe, F. L., Koestner, R., & Fang, J. Q. (2010) Parental autonomy-support, intrinsic life goals, and well-being among adolescents in China and north America. *Journal of Youth and Adolescence, 39*, 858–869. doi: 10.1007/s10964-009-9451-7

Lepper, M. R., Greene, D., & Nisbett, R. E. (1973). Undermining children's intrinsic interest with extrinsic rewards: A test of the "overjustification" hypothesis. *Journal of Personality and Social Psychology, 28*, 129–137. doi: 10.1037/h0035519

Levenson, R. W. (1999). The intrapersonal functions of emotion. *Cognition and Emotion*, *13*, 481–504. doi: 10.1080/026999399379159

Linehan, M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY: Guilford.

Loeber, R., & Stouthammer-Loeber, M. (1986). Family factors as correlates and predictors of juvenile conduct disorders. In M. Tonry & N. Morris (Eds.), *Crime and justice* (Vol. 7, pp. 29–149). Chicago, IL: University of Chicago Press.

Loevinger, J. (1976). *Ego development*. San Francisco: Jossey-Bass.

Luyckx, K., Vansteenkiste, M., Goossens, L., & Duriez, B. (2009). Basic need satisfaction and identity formation: Bridging self-determination theory and process-oriented identity research. *Journal of Counseling Psychology, 56,* 276–288. doi: 10.1037/a0015349

Luyckx, K., Soenens, B., Vansteenkiste, M., Goossens, L., & Berzonsky, M. D. (2007). Parental psychological control and dimensions of identity formation in emerging adulthood. *Journal of Family Psychology, 21,* 546–550. doi: 10.1037/0893-3200.21.3.546

Luyten, P., & Blatt, S. J. (2013). Interpersonal relatedness and self-definition in normal and disrupted personality development: Retrospect and prospect. *American Psychologist, 68,* 172–183. doi: 10.1037/a0032243

Lynch, M. F. (2013). Attachment, autonomy, and emotional reliance: A multilevel model. *Journal of Counseling and Development*, *91*, 301–312. doi: 10.1002/j.1556-6676.2013.00098.x

Lynn, S. J, Lillienfeld, S. O., Merckelbach, H., Geisbrecht, T., & van der Kloet, D. (2012). Dissociation and dissociative disorders: Challenging conventional wisdom. *Current Directions in Psychological Science 21*, 48–53.

Maccoby, E. E. (1980). *Social development: Psychological growth and the parent-child relationship*. New York, NY: Harcourt Brace Jovanovich.

Mansour, S., Bruce, K. R., Steiger, H., Zuroff, D. C., Horowitz, S., Anestin, A. S., & Sycz, L. (2012). Autonomous motivation: A predictor of treatment outcome in bulimia-spectrum eating disorders. *European Eating Disorders Review, 20*, 116–122. doi: 10.1002/erv.2154

Markland, D., Ryan, R. M., Tobin, V., & Rollnick, S. (2005). Motivational interviewing and self-determination theory. *Journal of Social and Clinical Psychology, 24,* 811–831. doi: 10.1521/jscp.2005.24.6.811

Markland, D., & Tobin, V. J. (2010). Need support and behavioural regulations for exercise among exercise referral scheme clients: The mediating role of psychological need satisfaction. *Psychology of Sport and Exercise, 11*, 91–99. doi: 10.1016/j.psychsport.2009.07.001

Markus, H. R., Kitayama, S., & Heiman, R. J. (1996). Culture and "basic" psychological principles. In E. T. Higgins & A. W. Kruglanski (Eds.), *Social psychology: Handbook of basic principles* (pp. 857–913). New York, NY: Guilford.

Mask, L., & Blanchard, C. M. (2011). The protective role of general self-determination against the "thin ideal" media exposure on women's body image and eating-related concerns. *Journal of Health Psychology, 16,* 489–499. doi: 10.1177/1359105310385367

Masterson, J. F. (1985). *Treatment of the borderline adolescent: A developmental approach*. New York, NY: Brunner/Mazel.

McCranie, E. W., & Bass, J. D. (1984). Childhood family antecedents of dependency and self-criticism: Implications for depression. *Journal of Abnormal Psychology, 93*, 3–8. doi: 10.1037/0021-843X.93.1.3

McGraw, K. O., & McCullers, J. C. (1979). Evidence of detrimental effect of extrinsic incentives on breaking a mental set. *Journal of Experimental Social Psychology, 15*, 285–294. doi: 10.1016/0022-1031(79)90039-8

Meissner, W. W. (1988). *Treatment of patients* in *the borderline spectrum*. Northvale, NJ: Aronson.

Miklikowska, M., Duriez, B., & Soenens, B. (2011). Family roots of empathy-related characteristics: The role of perceived maternal and paternal need support in adolescence. *Developmental Psychology, 47*(5), 1342–1352. doi: 10.1037/a0024726

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York, NY: Guilford.

Miquelon, P., Vallerand, R. J., Grouzet, F. M. E., & Cardinal, G. (2005). Perfectionism, academic motivation, and psychological adjustment: An integrative model. *Personality and Social Psychology Bulletin, 31,* 913–924. doi: 10.1177/0146167204272298

Miller, W. R., & Rollnick, S. (2002). *Motivational interviewing: Preparing people for change*. New York, NY: Guilford Press.

Millon, T., Davis, R., Millon, C., Escovar, L., & Meagher, S. (2000). *Personality disorders in modern life*. New York, NY: Wiley & Sons, Inc.

Minuchin, S., Rosman, B., & Baker, L. (1978). *Psychosomatic families*. Cambridge, MA: Harvard University Press.

Moller, A. C., Roth, G., Niemiec, C. P., & Deci, E. L. (2014). Perceiving, selecting, and projecting. Manuscript under editorial review.

Mouratidis, A., Michou, A., Vansteenkiste, M., & Lens, W. (2013). Perceived structure and achievement goals as predictors of students' self-regulated learning and affect and the mediating role of competence need satisfaction. *Learning and Individual Differences, 23*, 179–186. doi: 10.1016/j.lindif.2012.09.001

Mouratidis, A., Vansteenkiste, M., Sideridis, G., & Lens, W. (2011). Variation in vitality and interest-enjoyment as a function of class-to-class need-supportive teaching and pupils' autonomous motivation. *Journal of Educational Psychology, 103,* 353–366. doi: 10.1037/a0022773

Muhtae, L., Zhou, Q., Eisenberg, N., & Wang, Y. (2013). Predicting internalizing problems in Chinese children: The unique and interactive effects of parenting and child temperament. *Development and Psychopathology, 25,* 653–667. doi: 10.1017/S0954579413000084

Murayama, K., Matsumoto, M., Izuma, K., & Matsumoto, K. (2010). Neural basis of the undermining effect of extrinsic reward on intrinsic motivation. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 20911–20916. doi: 10.1073/pnas.1013305107

Murayama, K., Matsumoto, M., Izuma, K., Sugiura, A., Ryan, R. M., Deci, E. L., & Matsumoto, K. (2014). How self-determined choice facilitates motivation and performance: A key role of the ventromedial prefrontal cortex. *Cerebral Cortex, 25* (5), 1241–1251. doi: 10.1093/cercor/bht317

Muraven, M., Gagné, M., & Rosman, H. (2008). Helpful self-control: Autonomy support, vitality, and depletion. *Journal of Experimental Social Psychology*, *44*, 573–585.

Neyrinck, B., Vansteenkiste, M., Lens, W., Hutsebaut, D., & Duriez, B. (2006). Cognitive, affective and behavioral correlates of internalization of regulations for religious activities. *Motivation and Emotion, 30,* 321–332. doi: 10.1007/s11031-006-9048-3

Ng, Y., Ntoumanis, N., Thøgersen-Ntoumani, C., Deci, E. L., Ryan, R. M., Duda, J., & Williams, G. C. (2012). Self-determination theory applied to health contexts: A meta-analysis. *Perspectives on Psychological Science, 7,* 325–340. doi: 10.1177/1745691612447309

Nietzel, M. T., & Harris, M. J. (1990). Relationship of dependency and achievement/autonomy to depression. *Clinical Psychology Review*, *10*, 279–297.

Noom, M. J., Dekovic, M., & Meeus, W. H. J. (1999). Autonomy, attachment and psychosocial adjustment during adolescence: A double-edged sword? *Journal of Adolescence, 22*, 771–783. doi: 10.1006/jado.1999.0269

Othmer, E., & Othmer, S. C. (2002). *The clinical interview using DSM-IV-TR: Vols. 1 and 2*. Washington, DC: American Psychiatric Publishing, Inc.

O'Rorke, D. J., Smith, R. E., Smoll, F. L., & Cumming, S. P. (2012). Parent-initiated motivational climate and young athletes' intrinsic-extrinsic motivation: Cross sectional and longitudinal relations. *Journal of Child and Adolescent Behavior, 1*, 109. doi: 10.4172/jcalb.1000109

Parrott, W. G. (1993). Beyond hedonism: Motives for inhibiting good moods and for maintaining bad moods. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control. Century psychology series* (pp. 278–305). Upper Saddle River, NJ: Prentice-Hall, Inc.

Patall, E. A., Cooper, H., & Robinson, C. (2008). The effects of choice on intrinsic motivation and related outcomes: A meta-analysis of research findings. *Psychological Bulletin, 134,* 270–300. doi: 10.1037/0033-2909.134.2.270

Patall, E. A., Dent, A. L., Oyer, M., & Wynn, S. R. (2012). Student autonomy and course value: The unique and cumulative roles of various teacher practices. *Motivation and Emotion, 37 (*1*),* 14–32. DOI: 10.1007/s11031-012-9305-6

Pelletier, L., Dion, S. C., & Levesque, C. (2004). Can self-determination help protect women against socio-cultural influences about body image and reduce their risk of experiencing bulimic symptoms? *Journal of Social and Clinical Psychology, 23,* 61–88. doi: 10.1521/jscp.23.1.61.26990

Pfander, A. (1967). *Phenomenology of willing and motivation* (H. Spiegelberg, Trans.). Evanston, IL: Northwestern University Press. (Original work published 1908)

Piaget, J. (1971). *Biology and knowledge*. Chicago, Ill: University of Chicago Press.

Plant, R., & Ryan, R. M. (1985). Intrinsic motivation and the effects of self-consciousness, self-awareness, and ego-involvement: An investigation of internally controlling styles. *Journal of Personality*. *53*. 435–449. doi: 10.1111/j.1467-6494.1985.tb00375.x

Pomerantz, E. M., & Eaton, M. M. (2001). Maternal intrusive support in the academic context: Transactional socialization processes. *Developmental Psychology, 37*(2), 174–186. doi: 10.1037//0012-1649.37.2.174

Pulfrey, C., Buchs,C., & Butera, F. (2012). Why grades engender performance-avoidance goals: The mediating role of autonomous motivation. *Journal of Educational Psychology, 103,* 683–700. doi: 10.1037/a0023911

Raine, A. (2013). *The anatomy of violence*. New York, NY: Vintage Books

Reeve, J., & Halusic, M. (2009). How K–12 teachers can put self-determination theory principles into practice. *Theory and Research in Education*, *7*, 145–154.

Reeve, J., & Jang, H. (2006). What teachers say and do to support students' autonomy during a learning activity. *Journal of Educational Psychology*, *98*, 209–218. doi: 10.1037/0022-0663.98.1.209

Rogers, C. (1963). The actualizing tendency in relation to "motives" and to consciousness. In M. R. Jones (Ed.), *Nebraska symposium on motivation* (Vol. 11, pp. 1–24). Lincoln, NE: University of Nebraska Press.

Roth, G., & Assor, A. (2010). Parental conditional regard as a predictor of deficiencies in young children's capacities to respond to sad feelings. *Infant and Child Development*, *19*, 465–477. doi: 10.1002/icd.676

Roth, G., & Assor, A. (2012). The costs of parental pressure to express emotions: Conditional regard and autonomy support as predictors of emotion regulation and intimacy. *Journal of Adolescence, 35,* 799–808 doi: 10.1016/j.adolescence.2011.11.005

Roth, G., Benita, M., Amrani, C., Shachar, B. H., Asoulin, H., Moed, A., Bibi, U., & Kanat-Maymon, Y. (in press). Integration of negative emotional experience versus suppression: Addressing the question of adaptive functioning. *Emotion*.

Roth, G., Assor, A., Niemiec, C. P., Ryan, R. M., & Deci, E. L. (2009). The emotional and academic consequences of parental conditional regard: Comparing conditional positive regard, conditional negative regard, and autonomy support as parenting practices. *Developmental Psychology*, *45*, 1119–1142.

Roth, G., Ron, T., & Benita, M. (2009). Mothers' parenting practices and adolescents' learning from their mistakes in class: The mediating role of adolescent's self-disclosure. *Learning and Instruction*, *19*, 506–512 doi: 10.1016/j.learninstruc.2008.10.001

Ryan, R. M. (1982). Control and information in the intrapersonal sphere: An extension of cognitive evaluation theory. *Journal of Personality and Social Psychology, 43*, 450–461. doi: 10.1037//0022-3514.43.3.450

Ryan, R. M. (1993). Agency and organization: Intrinsic motivation, autonomy and the self in psychological development. In J. Jacobs (Ed.), *Nebraska Symposium on Motivation: Vol. 40. Developmental perspectives on motivation* (pp. 1–56). Lincoln, NE: University of Nebraska Press.

Ryan, R. M. (1995). Psychological needs and the facilitation of integrative processes. *Journal of Personality, 63*, 397–427. doi: 10.1111/j.1467-6494.1995.tb00501.x

Ryan, R. M. (2005). The developmental line of autonomy in the etiology, dynamics, and treatment of borderline personality disorders. *Development and Psychopathology*, *17*, 987–1006. doi: 10.1017/S0954579405050467

Ryan, R. M., Brown, K. W., & Creswell, J. D. (2007). How integrative is attachment theory? Unpacking the meaning and significance of felt security. *Psychological Inquiry, 18*, 1–6.

Ryan, R. M., & Connell, J. P. (1989). Perceived locus of causality and internalization: Examining reasons for acting in two domains. *Journal of Personality and Social Psychology, 57*, 749–761. doi: 10.1037/0022-3514.57.5.749

Ryan, R. M., Connell, J. P., & Deci, E. L. (1985). A motivational analysis of self-determination and self-regulation in education. In C. Ames & R. E. Ames (Eds.), *Research on motivation in education: The classroom milieu* (pp. 13–51). New York, NY: Academic Press.

Ryan, R. M., Curren, R. R., & Deci, E. L. (2013). What humans need: Flourishing in Aristotelian philosophy and self-determination theory. In A. S. Waterman (Ed.), *The best within us: Positive psychology perspectives on eudaimonic functioning* (pp. 57–75). Washington, DC: American Psychological Association Books.

Ryan, R. M., & Deci, E. L. (2000a). The darker and brighter sides of human existence: Basic psychological needs as a unifying concept. *Psychological Inquiry, 11*, 319–338. doi: 10.1207/S15327965PLI1104_03

Ryan, R. M., & Deci, E. L. (2000b). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55*, 68–78. doi: 10.1037//0003-066X.55.1.68

Ryan, R. M., & Deci, E. L. (2008). A self-determination approach to psychotherapy: The motivational basis for effective change. *Canadian Psychology, 49,* 186–193. doi: 10.1037/a0012753

Ryan, R. M., & Deci, E. L. (2013). Motivation and development in the context of schools: Research on how classroom environments affect student engagement, well-being, and performance. In B. Sokol, F. M. E. Grouzet, & U. Muller (Eds.), *Self-regulation and autonomy: Social, developmental, educational, and neurological dimensions of human contact* (pp. 191–207). New York, NY: Cambridge University Press.

Ryan, R. M., & Deci, E. L. (2011). Multiple identities within a single self: A self-determination theory perspective on internalization within contexts and cultures. In M. R. Leary & J. P. Tangney (Eds.), *Handbook of self and identity* (pp. 225–246). New York, NY: Guilford.

Ryan, R. M., Deci, E. L., Grolnick, W. S., & La Guardia, J. G. (2006). The significance of autonomy and autonomy support in psychological development and psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Volume 1, theory and methods* (2nd ed., pp. 795–849). New York, NY: John Wiley & Sons.

Ryan, R. M., & Grolnick, W. S. (1986). Origins and pawns in the classroom: Self-report and projective assessments of individual differences in children's perceptions. *Journal of Personality and Social Psychology, 50,* 550–558. doi**:** 10.1037//0022-3514.50.3.550

Ryan, R. M., & Hawley, P. (in press). Naturally good? Basic psychological needs and the proximal and evolutionary bases of human benevolence. In M. Leary & K. W. Brown (Eds.), *The psychology of hypoegoic behavior*. New York, NY: Guilford.

Ryan, R. M., & Kuczkowski, R. (1994). Egocentrism and heteronomy: A study of imaginary audience, self-consciousness, and public individuation in adolescence. *Journal of Personality, 62*, 219–238.

Ryan, R. M., Kuhl, J., & Deci, E. L. (1997). Nature and autonomy: Organizational view of social and neurobiological aspects of self-regulation in behavior and development. *Development and Psychopathology, 9*, 701–728.

Ryan, R. M., La Guardia, J. G., Solky-Butzel, J., Chirkov, V., & Kim. Y. (2005). On the interpersonal regulation of emotions: Emotional reliance across gender, relationships, and cultures. *Personal Relationships, 12,* 145–163. doi: 10.1111/j.1350-4126.2005.00106.x

Ryan, R. M., Legate, N., Niemiec, C. P., & Deci, E. L. (2012). Beyond illusions and defense: Exploring the possibilities and limits of human autonomy and responsibility through self-determination theory. In P. R. Shaver & M. Mikulincer (Eds.), *Meaning, mortality, and choice* (pp. 215–233). Washington, DC: American Psychological Association.

Ryan, R. M., & Lynch, J. H. (1989). Emotional autonomy versus detachment: Revisiting the vicissitudes of adolescence and young adulthood. *Child Development, 60*, 340–356. doi: 10.1111/j.1467-8624.1989.tb02720.x

Ryan, R. M., Lynch, M. L., Vansteenkiste, M., & Deci, E. L. (2011). Motivation and autonomy in counseling, psychotherapy, and behavior change: A look at theory and practice. *Counseling Psychologist, 39*, 193–260. doi: 10.1177/0011000009359313

Ryan, R. M., Mims, V., & Koestner, R. (1983). Relation of reward contingency and interpersonal context to intrinsic motivation: A review and test using cognitive evaluation theory. *Journal of Personality and Social Psychology, 45*, 736–750. doi: 10.1037/0022-3514.45.4.736

Ryan, R. M., Patrick, H., Deci, E. L., & Williams, G. C. (2008). Facilitating health behavior change and its maintenance: Interventions based on self-determination theory. *European Health Psychologist, 10,* 2–5.

Ryan, R. M., Plant, R. W., & O'Malley, S. (1993). Initial motivations for alcohol treatment: Relations with patient characteristics, treatment involvement and dropout. *Addictive Behaviors, 20*, 279–297. doi: 10.1016/0306-4603(94)00072-7

Ryan, R. M., Rigby, S., & King, K. (1993). Two types of religious internalization and their relations to religious orientations and mental

health. *Journal of Personality and Social Psychology, 65*, 586–596. doi: 10.1037/0022-3514.65.3.586

Ryan, R. M., Sheldon, K. M., Kasser, T., & Deci, E. L. (1996). All goals are not created equal: An organismic perspective on the nature of goals and their regulation. In P. M. Gollwitzer & J. A. Bargh (Eds.), *The psychology of action: Linking cognition and motivation to behavior* (pp. 7–26). New York, NY: Guilford.

Ryan, R. M., Stiller, J. D., & Lynch, J. H. (1994). Representations of relationships to teachers, parents, and friends as predictors of academic motivation and self-esteem. *Journal of Early Adolescence, 14*, 226–249.

Savard, A., Joussemet, M., Pelletier, J. E., & Mageau, G. A. (2013). The benefits of autonomy support for adolescents with severe emotional and behavioral problems. *Motivation and Emotion, 37,* 688–700. doi: 10.1007/s11031-013-9351-8

Schaefer, E. S. (1959). A circumplex model for maternal behavior. *Journal of Abnormal and Social Psychology, 59*, 226–235. doi: 10.1037/h0041114

Schafer, R. (1968). *Aspects of internalization*. New York, NY: International Universities Press.

Sears, R. R., Maccoby, E., & Levin, H. (1957). *Patterns of child rearing*. Evanston, IL: Row, Peterson.

Shapiro, D. (1981). *Autonomy and rigid character*. New York, NY: Basic Books.

Shields, A., Cicchetti, D., & Ryan, R. M. (1994). The development of emotional and behavioral self-regulation and social competence among maltreated school-age children. *Development and Psychopathology, 6*, 57–75.

Shields, A., Ryan, R. M., & Cicchetti, D. (2001). Narrative representations of caregivers and emotion dysregulation as predictors of maltreated children's rejection by peers. *Developmental Psychology, 37,* 321–337. doi: 10.1037//0012-1649.37.3.321

Silk, K. R., Lee, S., & Hill, E. M. (1995). Borderline personality disorder symptoms and severity of sexual abuse. *American Journal of Psychiatry, 152,* 1059–1064.

Silk, J. S., Morris, A. S., Kanaya, T., & Steinberg, L. (2003). Psychological control and autonomy granting: Opposite ends of a continuum or distinct constructs? *Journal of Research on Adolescence, 13,* 113–128. doi: 10.1111/1532-7795.1301004

Sierens, E., Vansteenkiste, M., Goossens, L., Soenens, B., & Dochy, F. (2009). The synergistic relationship of perceived autonomy support and structure in the prediction of self-regulated learning. *British Journal of Educational Psychology, 79,* 57–68. doi: 10.1348/000709908X304398

Skinner, B. F. (1953). *Science and human behavior*. New York, NY: Macmillan.

Smetana, J. G., Campione-Barr, N., & Daddis, C. (2004). Longitudinal development of family decision making: Defining healthy behavioral autonomy for middle-class African American adolescents. *Child Development, 75*, 1418–1434. doi: 10.1111/j.1467-8624.2004.00749.x

Sobral, D. T. (2004). What kind of motivation drives medical students' learning quests? *Medical Education, 38*(9), 950–957. doi: 10.1111/j.1365-2929.2004.01913.x

Soenens, B., Berzonsky, M. D., Dunkel, C., Papini, D., & Vansteenkiste, M. (2011). Are all identity commitments created equally? The importance of motives for late adolescents' personal adjustment. *International Journal of Behavioral Development, 35*, 358–369. doi: 10.1177/0165025411405954

Soenens, B., Elliot, A. J., Goossens, L., Vansteenkiste, M., Luyten, P., & Duriez, B. (2005). The intergenerational transmission of perfectionism: Parents' psychological control as intervening variable. *Journal of Family Psychology*, *19*, 358–366. doi: 10.1037/0893-3200.19.3.358

Soenens, B., Luyckx, K., Vansteenkiste, M., Luyten, P., Duriez, B., & Goossens, L. (2008). Maladaptive perfectionism as an intervening variable between psychological control and adolescent depressive symptoms: A three-wave longitudinal study. *Journal of Family Psychology, 22,* 465–474. doi: 10.1037/0893-3200.22.3.465

Soenens, B., Park, S.-Y., Vansteenkiste, M., & Mouratidis, A. (2012). Perceived parental psychological control and adolescent depressive experiences: A cross-cultural study with Belgian and South-Korean adolescents. *Journal of Youth and Adolescence, 35,* 261–272 doi: 10.1016/j.adolescence.2011.05.001

Soenens, B., & Vansteenkiste, M. (2005). Antecedents and outcomes of self-determination in three life domains: The role of parents' and teachers' autonomy support. *Journal of Youth and Adolescence, 34*, 589–604 doi: 10.1007/s10964-005-8948-y

Soenens, B., & Vansteenkiste, M. (2010). A theoretical upgrade of the concept of parental psychological control: Proposing new insights on the basis of self-determination theory. *Developmental Review, 30,* 74–99. doi: 10.1016/j.dr.2009.11.001

Soenens, B., & Vansteenkiste, M. (2011). When is identity congruent with the self? A self-determination perspective. In S. J. Schwartz, K. Luyckx, & V. L. Vignoles (Eds.), *Handbook of identity theory and research* (pp. 381–402). New York, NY: Springer.

Soenens, B., Vansteenkiste, M., Duriez, B., & Goossens, L. (2006). In search of the sources of psychologically controlling parenting: The role of parental separation anxiety and parental maladaptive perfectionism. *Journal of Research on Adolescence, 16,* 539–559. doi: 10.1111/j.1532-7795.2006.00507.x

Soenens, B., Vansteenkiste, M., Duriez, B, Luyten, P., & Goossens, L. (2005). Maladaptive perfectionistic self-representations: The mediational link between psychological control and adjustment. *Personality and Individual Differences, 38,* 487–498. doi: 10.1016/j.paid.2004.05.008

Soenens, B., Vansteenkiste, M., Goossens, L., Duriez, B., & Niemiec, C. (2008). The intervening role of relational aggression between psychological control and friendship quality. *Social Development, 17,* 561–581. doi: 10.1111/j.1467-9507.2007.00454.x

Soenens, B., Vansteenkiste, M., Lens, W., Luyckx, K., Goossens, L., Beyers, W., & Ryan, R. M. (2007). Conceptualizing parental autonomy support: Adolescent perceptions of promotion of independence versus promotion of volitional functioning. *Developmental Psychology, 43,* 633–646. doi: 10.1037/0012-1649.43.3.633

Soenens, B., Vansteenkiste, M., & Sierens, E. (2009). How are parental psychological control and autonomy support related? A cluster-analytic approach. *Journal of Marriage and Family, 71,* 187–202. doi: 10.1111/j.1741-3737.2008.00589.

Soenens, B., Vansteenkiste, M., Vandereycken, W., Luyten, P., Sierens, E., & Goossens, L. (2008). Perceived parental psychological control and eating disordered symptoms: Maladaptive perfectionism as a possible intervening variable. *Journal of Nervous and Mental Disease, 196*, 144–152. doi: 10.1097/NMD.0b013e318162aabf

Soenens, B., Vansteenkiste, M., & Van Petegem, S. (in press). Let's not throw out the baby with the bathwater: Applying the principle of "universalism without uniformity" to autonomy-supportive and controlling parenting. *Child Development Perspectives*.

Sperry, L. (2003). *Handbook of diagnosis and treatment of DSM-IV-TR personality disorders*. New York, NY: Brunner-Routledge

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Science, 9, (2),* 69–74. doi: 10.1016/j.tics.2004.12.005

Steinberg, L., & Silverberg, S. (1986). The vicissitudes of autonomy in adolescence. *Child Development, 57*, 841–851.

Steinberg, M., & Schnall, M. (2001). *The stranger in the mirror*. New York, NY: Cliff Street Books.

Stern, D. N. (1985). *The interpersonal world of the infant*. New York, NY: Basic Books.

Stevenson, M. B., & Lamb, M. E. (1981). The effects of social experience and social style on cognitive competence and performance. In M. E. Lamb & L. R. Sherrod (Eds.), *Infant social cognition* (pp. 375–394). Hillsdale, NJ: Erlbaum.

Strauss, J., & Ryan, R. M. (1987). Autonomy disturbances in subtypes of anorexia nervosa. *Journal of Abnormal Psychology, 96*, 254–258. doi: 10.1037//0021-843X.96.3.254

Stipek, D., & Seal, K. (2001) *Motivated minds: Raising children to love learning*. New York, NY: Henry Holt.

Strober, M., & Humphrey, L. L. (1987). Familial contributions to the etiology and course of anorexia and bulimia. *Journal of Consulting and Clinical Psychology, 55*, 654–659.

Talwar, V., & Lee, K. (2011). A punitive environment fosters children's dishonesty: A natural experiment. *Child Development, 82*, 1751–1758. doi: 10.1111/j.1467-8624.2011.01663.x

Thrash, T. M., & Elliot, A. J. (2002). Implicit and self-attributed achievement motives: Concordance and predictive validity. *Journal of Personality, 70,* 729–756.

Tsai, Y., Kunter, M., Lüdtke, O., Trautwein, U., & Ryan, R. M. (2008). What makes lessons interesting? The role of situational and individual factors in three school subjects. *Journal of Educational Psychology*, *100*, 460–472. doi: 10.1037/0022-0663.100.2.460

Twenge, J., Gentile, B., DeWall, C. N., Ma, D., Lacefield, K., & Schurtz, D. R. (2010). Birth cohort increases in psychopathology among young Americans, 1938–2007: A cross-temporal meta-analysis of the MMPI. *Clinical Psychology Review*, *30*, 145–154. doi: 10.1016/j.cpr.2009.10.005

Vallerand, R. J. (2010). On passion for life activities: The dualistic model of passion. In P. Devine & A. Plant (Eds.) *Advances in Experimental Social Psychology* (Vol. 42, pp. 97–193). London, UK: Elsevier.

Vallerand, R. J., O'Connor, B. P., & Hamel, M. (1995). Motivation in later life: Theory and assessment. *International Journal of Aging and Human Development*, *41*, 221–238. doi: 10.2190/YLFM-DGUE-HRL2-VWLG

Van den Broeck, A., Schreurs, B., De Witte, H., Vansteenkiste, M., Germeys, F., & Schaufeli, W. (2011). Understanding workaholics' motivations: A self-determination perspective. *Applied Psychology: An International Review, 60*(4), 600–621. doi: 10.1111/j.1464-0597.2011.00449.x

Vandereycken, W., & Vansteenkiste, M. (2009). Let eating disorder patients decide: Providing choice might reduce early drop-out from inpatient treatment. *European Eating Disorders Review, 17,* 177–183. doi: 10.1002/erv.917

Van der Hart, O. Nijenhuis, E., & Steele, K. (2006). *The haunted self: Structural dissociation and the treatment of chronic traumatization*. New York, NY: Norton.

Van der Kaap-Deeder, J., Vansteenkiste, M., Soenens, B., Verstuyf, J., Boone, L., & Smets, J. (in press). Fostering self-endorsed motivation to change in patients with an eating disorder: The role of perceived autonomy support and psychological need satisfaction. *International Journal of Eating Disorders*.

Van Petegem, S., Beyers, W., Vansteenkiste, M., & Soenens, B. (2012). On the association between adolescent autonomy and psychosocial functioning: Examining decisional independence from a self-determination theory perspective. *Developmental Psychology, 48*, 76–88. doi: 10.1037/a0025307

Van Petegem, S., Vansteenkiste, M., & Beyers, W. (2013). The jingle-jangle fallacy in adolescent autonomy in the family: In search of an underlying structure. *Journal of Youth and Adolescence, 42,* 994–1014. doi: 10.1007/s10964-012-9847-7

Van Petegem, S., Soenens, B., Vansteenkiste, M., & Beyers, W. (in press). *Rebels with a cause?* Adolescent defiance from the perspective of reactance theory and self-determination theory. Child Development.

Vansteenkiste, M., Niemiec, C., & Soenens, B. (2010).The development of the five mini-theories of self-determination theory: An historical overview, emerging trends, and future directions. In T. Urdan & S. Karabenick (Eds.), *Advances in motivation and achievement, vol. 16: The decade ahead*. Bingley, UK: Emerald Publishing.

Vansteenkiste, M., Sierens, E., Soenens, B., Goossens, L., Dochy, F., Aelterman, N., . . . , Beyers, W. (2012). Identifying configurations of perceived teacher autonomy support and structure: Associations with self-regulated learning, motivation and problem behavior. *Learning and Instruction, 22,* 431–439. doi: 10.1016/j.learninstruc.2012.04.002

Vansteenkiste, M., Simons, J., Lens, W., Soenens, B., & Matos, L. (2005). Examining the motivational impact of intrinsic versus extrinsic goal framing and autonomy-supportive versus internally controlling communication style on early adolescents' academic achievement. *Child Development, 76*, 483–501. doi: 10.1111/j.1467-8624.2005.00858.x

Vansteenkiste, M., Soenens, B., & Vandereycken, W. (2005). Motivation to change in eating disorder patients: A conceptual clarification on the basis of self-determination theory. *International Journal of Eating Disorders, 37,* 207–219. doi: 10.1002/eat.20099

Vansteenkiste, M., Soenens, B., Van Petegem, S., & Duriez, B. (2014). Longitudinal associations between adolescent perceived degree and style of parental prohibition and internalization and defiance. *Developmental Psychology, 50,* 229–236. doi: 10.1037/a0032972

Vansteenkiste, M., & Ryan, R. M. (2013). On psychological growth and vulnerability: Basic psychological need satisfaction and need frustration as a unifying principle. *Journal of Psychotherapy Integration*, *23*, 263–280. doi: 10.1037/a0032359

Verschueren, K., Doumen, S., Vervoort, E., & Vansteenkiste, M. (2013). *How early parent–child interactions are carried forward into elementary school: Relational and motivational pathway to academic success*. Manuscript submitted for publication.

Verstuyf, J., Vansteenkiste, M., & Soenens, B. (2012). Eating regulation and bulimic symptoms: The differential correlates of health-focused and appearance-focused eating regulation. *Body Image: An International Research Journal, 9*, 108–117. doi: 10.1016/j.bodyim.2011.09.003

Verstuyf, J., Vansteenkiste, M., Soenens, B., Boone, L., & Mouratidis, A. (2013). Daily ups-and-downs binge eating symptoms: The role of need satisfaction, self-control and emotional eating style. *Journal of Social and Clinical Psychology, 32,* 335–361.

Vignoles, V. L., Schwartz, J., & Luyckx, K. (2011). Introduction: Towards an integrative view of identity. In S. J. Schwartz, K. Luyckx, & V. L. Vignoles (Eds.), *Handbook of identity theory and research* (pp. 1–30). New York, NY: Springer.

Warneken, F., & Tomassell, M. (2008). Extrinsic rewards undermine altruistic tendencies in 20-month olds. *Developmental Psychology, 44,* 1785–1788 doi: 10.1037/a0013860

Waters, E., Wippman, J., & Sroufe, L. A. (1979). Attachment, positive affect, and competence in the peer group: Two studies in construct validation. *Child Development, 50,* 821–829. doi: 10.1111/j.1467-8624.1979.tb02431.x

Weinstein, N., Deci, E. L., & Ryan, R. M. (2011). Motivational determinants of integrating positive and negative past identities. *Journal of Personality and Social Psychology*, *100*, 527–544. doi: 10.1037/a0022150

Weinstein, N., & Hodgins, H. S. (2009). The moderating role of autonomy and control on the benefits of written emotion expression. *Personality and Social Psychology Bulletin*, *35*, 351–364. doi: 10.1177/0146167208328165

Weinstein, N., Hodgins, H. S., & Ostvik-White, E. (2011). Humor as aggression: Effects of motivation on hostility expressed in humor appreciation. *Journal of Personality and Social Psychology*, *100*, 1043–1055. doi: 10.1037/a0022495

Weinstein, N., Przybylski, A. K., & Ryan, R. M. (2012). The index of autonomous functioning: Development of a scale of human autonomy. *Journal of Research in Personality*, *46*, 397–413. DOI: 10.1016/j.jrp.2012.03.007

Weinstein, N., Przybylski, A. K., & Ryan, R. M. (2013). The integrative process: New research and future directions. *Current Directions in Psychological Science*, *22*, 69–74.

Weinstein, N., & Ryan, R. M. (2010). When helping helps: Autonomous motivation for prosocial behavior and its influence on well-being for the helper and recipient. *Journal of Personality and Social Psychology*, *98*, 222–244. doi: 10.1037/a0016984

Weinstein, N., Ryan, W. S., DeHaan, C. R., Przybylski, A. K., Legate, N., & Ryan, R. M. (2012). Parental autonomy support and discrepancies between implicit and explicit sexual identities: Dynamics of self-acceptance and defense. *Journal of Personality and Social Psychology*, *102*, 815–832. doi: 10.1037/a0026854

Weiss, L. A., & Grolnick, W. S. (1991, April). *The roles of parental involvement and support for autonomy in adolescent symptomatology*. Paper presented at the biennial meeting of the Society for Research in Child Development, Seattle, WA.

Werner, H. (1948). *Comparative psychology of mental development*. New York, NY: International Universities Press.

Westen, D. (1991). Cognitive-behavioral interventions in the psychoanalytic psychotherapy of borderline personality disorders. *Clinical Psychology Review, 11*, 211–230. doi: 10.1016/0272-7358(91)90101-Y

Whiffin, V. E., & Sassville, T. M. (1991). Dependency, self-criticism, and recollections of parenting: Sex differences and the role of depressive affect. *Journal of Social and Clinical Psychology, 10,* 121–133. doi: 10.1521/jscp.1991.10.2.121

Whipple, N., Bernier, A., & Mageau, G. A. (2009). Attending to the exploration side of infant attachment: Contributions from self-determination theory. *Canadian Psychology*, *50*, 219–229 doi: 10.1037/a0016322

Whipple, N., Bernier, A., & Mageau, G. A. (2011). Broadening the study of infant security of attachment: Maternal autonomy-support in the context of infant exploration. *Social Development, 20(*1*),* 17–32. DOI: 10.1111/j.1467-9507.2010.00574.x

Williams, G. C., Grow, V. M., Ryan, R. M., Friedman, Z., & Deci, E. L. (1996). Motivational predictors of weight loss and weight-loss maintenance. *Journal of Personality and Social Psychology, 70*, 115–126. DOI: 10.1037/0022-3514.70.1.115

Williams, G. C., McGregor, H. A., Sharp, D., Levesque, C., Kouides, R. W., Ryan, R. M., & Deci, E. L. (2006). Testing a self-determination theory intervention for motivating tobacco cessation: Supporting autonomy and competence in a clinical trial. *Health Psychology, 25,* 91–101. doi: 10.1037/0278-6133.25.1.91

Williams, G. C., Rodin, G. C., Ryan, R. M., Grolnick, W. S., & Deci, E. L. (1998). Autonomous regulation and long-term medication adherence in adult outpatients. *Health Psychology, 17*, 269–276. doi: 10.1037/0278-6133.17.3.269

Wouters S., Doumen S., Germeijs V., Colpin H., Verschueren K. (2013). Contingencies of self-worth in early adolescence: The antecedent role of perceived parenting. *Social Development*, 242–258. Blackwell Publishers.

Wuyts, D., Vansteenkiste, M., Soenens, B., & Assor, A. (in press). An examination of the dynamics involved in child-invested parental contingent self-esteem. *Parenting: Science and Practice*.

Winnicott, D. W. (1965). *The maturational process and the facilitating environment*. New York, NY: International Universities Press. (Original work published 1960)

Yamauchi, H., & Tanaka, K. (1998). Relations of autonomy, self-referenced beliefs and self-regulated learning among Japanese children. *Psychological Reports, 82*, 803–816. doi: 10.2466/PR0.82.3.803-816

Yarrow, L. J., Rubenstein, J. C., & Pedersen, F. A. (1975). *Infant and environment: Early cognitive and motivational development*. Washington, DC: Wiley.

Zanarini, M. C. (Ed.). (1997). *Role of sexual abuse in the etiology of borderline personality disorder*. Washington, DC: American Psychiatric Association.

Zeldman, A., Ryan, R. M., & Fiscella, K. (2004) Client motivation, autonomy support and entity beliefs: Their role in methadone maintenance treatment. *Journal of Social and Clinical Psychology, 23,* 675–696.

Zuroff, D. C., Koestner, R., Moskowitz, D. S., McBride, C., & Bagby, R. M. (2012). Therapist's autonomy support and patient's self-criticism predict motivation during brief treatments for depression. *Journal of Social and Clinical Psychology*, *31 (*9*)*, 903–932.

Zuroff, D. C., Koestner, R., Moskowitz, D. S., McBride, C., Marshall, M., & Bagby, R. M. (2007). Autonomous motivation for therapy: A new common factor in brief treatments for depression. *Psychotherapy Research, 17*, 137–148. doi: 10.1080/10503300600919380

CHAPTER 10

Roots of Typical Consciousness: Implications for Developmental Psychopathology

PHILIPPE ROCHAT

GENERAL AIM 439
WHAT DOES IT MEAN TO BE CONSCIOUS? 440
Origins of Feeling Experience 440
What Is Representation? 442
EXPERIENTIAL AWARENESS AT BIRTH 443
What Is it Like to Be a Newborn? 444
Experiential Vicariousness at Birth 447
CONSTRAINTS ON EARLY EXPERIENCE 447
Experiential Versus Conceptual Awareness 448
Functional Propensities of Newborns 449
Built-In Motivational and Attention Systems 450
Built-In Sameness Detection System 450
Core Knowledge and Conceptual Primitives 452
Revisiting Infantile Amnesia 453
ROOTS OF INTERSUBJECTIVITY 455
Sociality and Reciprocity in Typical Development 455
Products of Emerging Reciprocation 456
Developing Self- and Social Awareness 457
Social Dependence and Human Symbolic Psychology 459
Unfolding Levels of Sharing and *Conscientiousness* 460
CONCLUSIONS: IMPLICATIONS FOR DEVELOPMENTAL PSYCHOPATHOLOGY 463
Centrality of Mutual Recognition 463
Given Sense of Self-Unity 464
Primordial Possession of the Own Experience 464
REFERENCES 465

Mental illness has to be considered against what supports and might be the roots of typical consciousness: what human consciousness is made of, how it tends to work and develop in the healthy young child. Deviances do exist and are only revealed against what might constitute a typical norm. The etiology, emergence, and evolution of mental ills (main object of developmental psychopathology) do require both a clinical and a normative approach. That is the imperative behind this chapter, attempting to provide some normative look at what appears to constitute typical consciousness in early development.

GENERAL AIM

The tidal wave of infancy data harvested in the past 50 years changed the view of the starting state of typical development, away from strict empiricism (behaviorism) or stage-like, domain general structural constructivism à la Piaget. The discovery of early capacities interpreted as deeply rooted in animal evolution is now part of mainstream theorizing. It is important to remember that such views were demonized for their reductionism only a few decades ago. In recent years, nature did regain some weight in contrast to nurture. The explosion of brain measurement technology contributed to such radical shift in the perennial nature–nurture controversy, a polarized and weight shifting debate. Here, I want to propose a few basic systems and mechanisms accounting for starting state representational development. These systems are supported by a large body of infancy research and tend to be acknowledged by most theorists. General enough, yet falsifiable, these systems and mechanisms cut across and somehow transcend the many theoretical controversies that continue to animate the field of developmental psychology.

First and foremost, I want to describe and discuss a system that allows infants from birth and even in utero, to represent what is of the same, in other words to represent *sameness* of feelings. Sameness detection systems are a necessary pre-condition for the emergence of any forms of consciousness, in other words any forms of awareness of having knowledge about something. Sameness detection is the backbone of the conscious (thinking) mind as William James describes it.

Representations that form the content and format of the conscious mind, is framed here as a dynamic process

of informational transformation that operates at multiple representational levels: analogical sensory representations of what is out there in terms of physical energy (proximal stimulation); more cognitive and conceptual representations capturing what is out there in terms of differentiated objects or entities (distal stimulation); pretend representations of what was there (imagined stimulation); might-be representations of future events (projected stimulation); or could-be representations in terms of conjectures (simulated stimulation).

In the generic sense, representation does capture a transformational process that operates at sensory, perceptual, and conceptual levels. The question is what might be the foundational expression and early development of such process? Ample evidence now suggests that the starting state development of the child is not just sensory and motor, but perceptual and very early on, at least within the first year, also conceptual with a propensity toward essentialism or the default belief that things are made of a set of ontologically deep characteristics (as opposed to surface features). Research shows that from the outset, infants are objective perceivers whose actions are organized around functional goals. They start off from birth being actors and not just complex passive reactors or thermostat-like reflex machines. These action systems have their own motivational components (hunger, comfort, curiosity), tapping into physical and social environmental resources that are indispensible for their survival. This starting state can be viewed as the evolutionary product of prolonged immaturity and hypersocial dependence of human childhood, two uncanny characteristics that are unique to our species.

It is within this general context that we discuss the roots of human consciousness as it typically develops in an attempt to characterize basic prerequisites for its healthy development. The attempt here is to provide some general frame to the perennial reflection in the field of developmental psychopathology regarding what it takes—minimally—to function in an adaptive way and what could go wrong, such as in cases of autism or schizophrenia.

WHAT DOES IT MEAN TO BE CONSCIOUS?

Consciousness comes from the Latin word *conscientia*, meaning "with knowledge" or "knowledge with others" as it was originally meant in ancient Greek philosophy prior to Descartes' ergo sum, his "ego-logy" that announced the modern self-identity of eighteenth-century Enlightenment (Rochat, 2009; Taylor, 1979). In a literal sense, to be without conscious or nonconscious means to exist without knowledge or in the ancient sense, without the potential of sharing knowledge with others (*sine sientia or con-sopio*: literally "stupefied," "benumbed," "put to sleep"). But what does it mean to be minimally conscious and have knowledge, hence not to be benumbed or stupefied?

By definition, consciousness necessarily entails some sensory awareness, some embodied feeling experience corresponding to any affective or emotional experience like pain but also feelings of surprise in the face of the unexpected, comfort in the face of what is expected. In the most generic sense, consciousness would correspond to the process by which sameness among sensations or individuated perceptual events is detected and assembled (synthesized) for further mental processing. Consciousness is the process of meaning creation by linking disparate experiences on the basis of what they share (sameness) and, hence, also necessarily what they don't share (difference). In the most generic sense, consciousness is thus the experience of discrete events (i.e., sensations) that are linked and compared. It is what unifies and gives direction to subjective experience, the primary and necessary root of self-unity and self-agency.

Questions remain as to what are the representations or syntheses that are produced by the typical conscious process? What is the typical content of consciousness early in development? Infancy research has shed new empirical light on the issue and the roots of what might be lacking when psychological development goes astray.

Origins of Feeling Experience

A rich body of neuroanatomical and behavioral evidence points to the fact that implicit feeling awareness—and hence subjective experience—is already present at birth, possibly even in the womb. This is a rather revolutionary idea considering that not so long ago, the idea of feelingless infants (i.e., *infans,* or nonverbal children) was the default assumption.

Until fairly recently, the zeitgeist was to deny infants any form of worthwhile feeling experience (phenomenal awareness). Proof of it is that in the 1940s and 1950s, surgery without anesthesia was routinely performed on infants and young children. Modern surgeons conveniently paralyzed squirming infants injecting Curare or similar paralytic agents. Under such circumstances, adults recalled excruciating pain during surgery; however, patients were not believed, and the practice went on for 20 years. As Dennett (1981) pointed out, "The fact that most of the patients were infants and small children may explain this

credibility gap" (p. 201). Even today, local anesthetics are not routine in painful procedures on newborns such as heel prick and circumcision, even by pediatricians practicing in state-of-the-art maternity hospitals. The zeitgeist continues to be that infants have either no feelings or less feelings or that feeling experience at this early stage might not be as consequential for lack of memory (infantile amnesia; see later section on the topic). Such rationale raises questions when looking at the brain as well as behavior in pre- and postnatal development.

Looking at brain development during the prenatal and postnatal period suggests that the necessary neurological prerequisites for feeling experience might already be in place by *8 weeks* after conception, meaning when the sperm meets the egg! Here is in a nutshell what we know today about the emergence of the brain and neural growth from conception to birth. This emergence is humbling for its pace and complexity, considering that the brain is probably the most complex system in the universe. It reminds us of the great biological forces behind development. It takes only 4 weeks from conception for the neural tube to be formed from layers of cells on the embryonic disc (Hepper, 2002). Only one extra week is needed for the basic five-part structure of the brain (i.e., telecephalon and diencephalon of the forebrain, midbrain, hindbrain, and the spinal cord) to be anatomically differentiated and clearly visible. By 11 weeks of gestation (not 3 months after conception), the medulla, cerebellum, inferior and superior colliculus, and both cerebral hemispheres covering the diencephalon are also clearly visible (Carlson, 1994).

From then on and for a few years, both hemispheres grow in surface areas via folding grooves and convolutions. This growth reflects rapid and exponential connection network among synapses and myelination of axons that provide insulation (fatty white matter) for better transmission of electrochemical nerve impulses. By 2 years of age, the child's brain already weighs 80% of its lifetime maximum (Kretschmann, Kammradt, Krauthausen, Sauer, & Wingert, 1986).

In terms of neural growth, between 10 and 26 weeks gestational age, neurons are produced at a rate of 250,000 a minute, leading to overproduction. Beyond 26 weeks, more than half of the produced nerve cells are selectively pruned and die. The surviving 100 billions will eventually form the adult brain (Oppenheim, 1991). All of this development is indeed humbling. We should always keep it in the back of our mind when thinking about what it means to be present, alive, and having experience in this world.

Regarding connection between cells, an overproduction of synapses also continues beyond birth, with peaking periods that vary across brain regions (Rakic, 1972). Synaptogenesis continuing after birth is not homogeneous and synchronous across brain regions. For example, postmortem data indicate that synaptic density peaks earlier in the auditory cortex (3 months) compared with the middle frontal gyrus (15 months; Huttenlocher & Dabholkar, 1997). This kind of growth asynchrony is reflected, for example, in the sequential development of sense modalities in the womb and beyond (i.e., vision lag).

In short, these facts remind us that brain emergence and the neural (sensory) machinery that necessarily underlies implicit feeling experience are fast, remarkably precise, and predictable in their unfolding. To the naked eye or through the lens of the microscope, it is an explosive and amazingly well-programmed growth. In relation to putative first feeling experience, brain development puts in place within 8 weeks the potential for fetuses to sense the world and hence the necessary prerequisite to eventually feel it: the potential for first minimal, obviously implicit, and very rudimentary feeling experience. Let us not forget, however, that feeling experience rests on the prerequisite of an ability to sense the world, as machine do. In humans, such necessary sensory machinery matures within weeks of conception and long before birth. In its development and within the confine of the maternal womb, fetal research show that it actually recapitulates the emergence order of the main sensory systems in the course of species evolution. The sensory machinery (sense modalities) of fetuses matures in the same sequential order than our ancestral species (Gottlieb, 1971; Lecanuet & Schaal, 1996). Somesthetic sensitivity (skin and body feelings) matures first. This sensitivity corresponds to tactile (skin pressure), vestibular (posture and balance), and pain stimulation (tissue damage). It is followed by the maturation of chemosensory sensitivity that combines olfaction and gustation (i.e., smell and taste), followed by audition (pitch, amplitude, and phrasing of sounds) and finally by vision (light and optic array). From this, we can conclude that the necessary sensory prerequisites of first feeling experience are in place at least by the third semester of gestation. But when can we say that first feeling experiences actually emerge in development? New ways of observing fetuses (three- and four-dimensional sonic echography and image scanning) suggest that already by the last trimester fetuses express emotions such as *happiness* via smiling and *sadness* via frowning of the eye brows and lowering of the corner of the lips in the same way newborns express such mental states at birth and beyond (Hata, Dai, & Marumo, 2010). More intriguing is the fact that nociceptive (pain) receptors appear first in and around the mouth area at

around 7 weeks postconception. They rapidly extend to the palmar surface of the hands by 11 weeks, and the rest of the skin and mucosal surfaces by 20 weeks (Brusseau & Mashour, 2007; Smith, 1996).

In all, this suggests that in trying to find the roots of feeling experience, above and beyond sensing, we should probably look in the womb for its embryonic stage. But what about representation? Does feeling experience entail representational ability? Next, we turn to this question, but first it is necessary to clarify what lies behind the concept of representation and its multiple meanings at various levels of analysis.

What Is Representation?

The term *representation* is widely used across all sciences—from engineering to brain science and cognitive psychology. It is among the most polysemous and mixed-bag concept of all, the meaning of which lies in the eyes and uses of the beholder. Do brain scientists, roboticists, cognitive psychologists, or computer scientists talk about the same thing when using the term representation in their respective fields? It appears that they might refer to one basic process after all—but a process that is happening at incommensurable and irreducible scales, from groups of neurons or electronic chips storing nondescript information to semantic networks forming bodies of knowledge and the creation of meanings about the larger environment. Like philosophers or anthropologists, it is to the latter that developmental psychologists typically refer to when talking about representation.

In the most literal sense, representation means re-presenting or *presenting again*. Etymologically, it thus refers to a transformation from one state of being to another. It is fundamentally a transformative process and its product, for example, the transformation of a physical event (light stimulation in the environment) to a subjective or psychic state that is embodied and stored for further mental processing (sensation of the light hitting the retina).

Representation is thus essentially a process. It is not a thing in itself but a transformation. By analogy, a cloud in the sky is the symptom of an ongoing atmospheric condensation and air mass exchanges, not a thing. Representation (presenting again) captures the transformative process by which information is translated from one system to another, whatever the translation code might be: analog, digital, or symbolic.

Representational process is pervasive in nature, present at all levels of functioning in living organisms: from the transduction of light energy at the cellular level on the retina, giving rise to its sensation; to the mental generation of spatial maps, the emotional expression of mental states; to the macro level of collective habitus or group ways. The process of representation is also present and what characterizes any information processing machines, minus the feeling experience and subjectivity that are exclusive to living and sentient animals like us.

From this perspective, the transformative process of representation exists at all levels of description, from the neurological to the psychological, moral, and societal—each level requiring a different language to account for them. At the low neurological level, the language of mechanical causality applies. The process can be analyzed and accounted for on the basis of highly specific neurochemical factors causing the transduction process underlying any sensory awareness that is the object of psychophysics. Vision neuroscientists can trace precisely the successive representational transformations in the visual cortex leading to higher levels perception and consciousness that is the object of perception theories. From such causal description, neuroscientists capture the necessary transformative, meaning-making process of perception. An example of such representational process at a cellular level is captured by pioneer neuroscientist Donald O. Hebb in his seminal 1949 book on the *Organization of Behavior*: "...A repeated stimulation of specific receptors will lead slowly to the formation of an 'assembly' of association-area cells which can act briefly as a closed system after stimulation has ceased; this prolongs the time during which the structural changes of learning can occur and constitutes the simplest instance of a representative process (image or idea)" (p. 64).

At the psychological, sociocognitive, and interpersonal level, any causal account of representation is much more elusive as they depend on an intricacy of complex interacting factors such as age, individual, and group (cultural) differences. As social scientists, the best we can do is to capture representational patterns. These patterns are documented in the same way natural scientists document varieties of species, eventually coming up with some accounts as to what might be some of the underlying causes of the observed diversity.

At the macro level, the developmental approach has the distinct advantage of allowing for the description of representation not only for what they are at various ages but also for how they systematically unfold in ontogeny. The credo behind the developmental approach is that the constitutive elements of human representational systems are best revealed in their change and as new forms come about in ontogeny. However, for representational systems

to become organized around concepts that unify and give direction to subjective experience, some prerequisites are necessary. It includes—as we will consider in subsequent sections—built-in action attractors and sameness detection systems supporting the basic ability to pay attention and register what remains invariant in the midst of what otherwise would be mental chaos and confusion, meaningless, and maladaptive experience.

Three General Levels of Mental Synthesis

Philosopher Immanuel Kant's (1724–1804) views on the mind continue to be valid and helpful today when dealing with the issue of trying to distinguish levels of representation as a transformative cognitive process. Following Kant (see Brook, 1994), sensory inputs from the world need to be unified to become conscious experiences about something. In other words, for the sense datum to become knowledge about the world, it needs to be synthesized within a temporal and spatial structure at three levels: (1) the transformation of sensory apprehension into intuitions or percepts; (2) the coordination of intuitions or percepts in reproductive imagination (what would correspond to mental simulation in today's neuroscientific jargon); and (3) the recognition of concepts in coordinated intuitions (conceptualization of a priori categories).

Kant proposes that the unity of our conscious experience rests on these three kinds of synthesis, an idea that still prevails in current cognitive sciences. Within the Kantian framework, one can argue that newborns do engage in the synthesizing of sense data, certainly at the first level proposed by Kant and probably also at the second level. The third level seems to be evident only in a few months down the road, possibly before the first birthday as suggested by Jean Mandler (1988, 1992). Mandler and now many other researchers provide numerous empirical evidence that at least by 9–12 months infants manifest object categorization that is based on ontological concepts such as containment, agency, continuity, or nonobvious inferred property like weight, animacy as opposed to inanimacy, self-propulsion as opposed to driven by passive movement, even intentional as opposed to accidental or random actions (see also Wellman & Gelman, 1992, for further detailed discussion). Core knowledge and essentialist interpretations of infant cognition are continuously challenged by researchers within a more empiricist (Humean) tradition that emphasizes perceptual learning over ad hoc representational capacities in the young child (see, e.g., Rakison & Poulin-Dubois, 2001).

However, there is unquestionably a trend toward an ever growing body of research suggesting that in the first year, infants are already engaging in a perceptual analysis expressing the three kinds of syntheses proposed by Kant, presumably the foundation of unity in consciousness. But what about newborns, and what about infants at birth? Do they show signs of unity in their experience? The most probable answer is yes.

EXPERIENTIAL AWARENESS AT BIRTH

Based on empirical facts accumulated over the past 50 years in the booming field of infancy research, we now have more empirical ammunition to speculate about what it might be like to encounter the world by seeing, feeling, or hearing at the origins of life. We better understand not only how newborns experience the world from the perspective of psychophysics (what range of stimulus thresholds infants detect across the various sense modalities) but also what attracts them in their exploration of objects in the environment and what information they appear to pick up, store, and eventually retrieve as newborns and even as fetuses during the last two trimesters of gestation. We know that newborns see colors and use dynamic information, preferring moving rather than static things (Rochat, 2001). We also know that infants are innately attracted to particular configurations like moving faces, regions of high contrast on a visual display, high-pitch sounds, and human voices with particular contours (i.e., motherese). We know why they crave sugar, why they appear to innately savor sweet tastes, and why they show repulsion and strong rejection of bitter tastes.

Topping all of these well-established empirical facts, there is now a vast amount of habituation and other operant conditioning studies with newborns showing that infants from birth are fully attuned to novel as opposed to familiar experiences. Probably one of the greatest and most striking discoveries in developmental psychology over the past few decades is that most of what is demonstrated in newborns is also shown in healthy fetuses during the last trimester of gestation: they habituate, learn, store experiential information, and demonstrate comparable threshold across sensory modalities. Furthermore, what they learn in the womb is readily transferable ex utero. Facts show that few-hours-old newborns prefer to suck in certain ways on a pacifier and to hear the voice of their mother over the voice of a female stranger (De Casper & Fifer, 1980). They orient significantly more, showing preference in smelling a gauze impregnated with the mother's amniotic fluid over the fluid of a stranger that just gave birth (Marlier, Schaal, & Soussignan, 1998a, 1998b).

What can be safely inferred from these now well recognized and numerous facts (see Rochat, 2001) is that not only are newborns sophisticated learners, perceivers and even knowers, but more importantly that they have from the start a very rich affective and emotional life. We are not born affectively "neutral" but on the contrary alive with forces pushing and pulling us around: born with strong desires and untamed affective needs as Freud speculated in a highly controversial book over a century ago (Freud, 1905). This rich and untamed affective life cannot, however, be reduced to an early sensory experience that would be undifferentiated and disorganized. What newborns experience of the world is not a blob of chaotic multimodal sensations befalling onto the senses as "a blooming, buzzing, confusion," confusion taken to mean "chaos" and not the literal sense of con-fusion or *fusion with* that suggests some harmony of experience.

We now know that newborn experience is anything but chaos. Healthy neonates are quick to learn, they attend and respond to specific physical events felt in and out of the body, manifest emotions (in the literal sense of moving out feelings via scream and other clearly readable basic facial expressions that are innate). In all, this experience is not an affectively neutral experience that would be mainly attached to the functioning of automatic reflexes. This is not the case since learning is involved from the start, infants acting rather than responding in a world that has values: comfort, pain, relief, and an intense orientation toward particular affective states linked to satiety, specific odors and tastes, dim lights, contrasted visual contours or high-pitch speech sounds. This experience rests on an embodied semantics of approach and avoidance, dangers and strong attractors or pulls.

Because of learning, newborns' experience is not repetitive but rather cumulative: a present experience is influenced by what happened prior. It can be therefore assumed that if successive experiences might be homologous, they are never absolutely the same. This inference simply means that newborn experience does not rest on the feelings that might accompany automatic reflexes as for example the feeling of something hitting my knee and the proprioceptive sensation of my knee automatically jerking forward. Such machine-like experience does exist in newborns, in the same way it persists in adults with the feeling experience that accompanies nonvolitional responses like a knee jerk or the obligatory chill one feels running down the spine while stepping on what was thought to be a snake but in fact is a piece of dead wood laying around.

However, what it is like to be a neonate cannot be reduced to the sensations that arise from such responses. Infants at birth are more than reflex machines: from the outset they desire. They are fast to learn, showing unmistakable orientation toward specific and meaningful experiences: food, comfort, and an optimum level of stimulation (e.g., not too loud, not too bright, but with a lot of contrast and movement).

What Is it Like to Be a Newborn?

Daniel Stern (1991), in an unusual book titled *Diary of a Baby: What Your Child Sees, Feels, and Experiences*, took on the task to imagine what it is like to be a young child from 6 weeks to 4 years. Stern writes from the perspective of the child and reconstructs what it must feel like to be hungry, to wake up, or to stare at a moving spot of light. Stern infers the world of sensations of a fictive newborn (named Joey), based on what we now know about perception, learning, and affect regulation in infancy. The world of Joey at 6 weeks is for Stern a world of sensations. Although this world is nameless and nonobjectified, not yet explicit or conceptualized, it is a world rich of motion and moving impressions, where pieces of space are contrasted, detached, move, meet, overlap, vanish. It is a world made of rhythms and changes of pace in which the infant detects forces expressed in movements: acceleration, deceleration, shrinking, and looming of forms and shapes that are more or less capturing the attention of the child-like magnets of various forces that wax and wane. The world that Stern describes has phenomenal *qualities*. It is experienced from a particular embodied point of view, the first-person perspective that Stern tries to capture. It is a world of sensations, but a world with a point of experiential origin that is the body, the referential point in space where sensations arise while the infant experiences being awake and alive in the world via multiple sensory channels that are *all open at the same time*, either actively when the infant is on his own or more passively while cared and manipulated by others. This in-unison working of the senses does not however entail chaos but rather a pull toward a primitive harmonious order (of phenomenal awareness) that will become eventually objectified, brought to explicit cognitive coherence and scrutiny (consciousness proper), a phenomenal consciousness that has, in addition, cognitive accessibility. (See the discussion and distinction proposed by philosopher Ned Block [2007]).

Stern's assumption, in his speculation, is that the world of sensations of the newborn is not just undifferentiated or diffuse. It is a world of experiences with distinct qualities and values that are anchored in the body, lived from within and therefore carrying with them a subjective perspective.

Such speculation regarding what it must be like to be a newborn finds support in numerous research studies pointing to a minimal *self-awareness* at birth, therefore evidence that even neonates have subjectivity and the propensity to experience the world from their own embodied perspective.

Among all the existing speculations on what might animate the psychic life of newborns, assuming that there is one prior to language, which was not easily accepted until fairly recently, ideas emerging from Freud's psychoanalytical approach have been the most prolific as well as the most controversial. Freud (1905) was the first to place desire in the instinctive behaviors expressed by newborns, and the first to identify early embodied sensory experience as being the cradle of the person, with its often, debilitating characteristics emerging in development. To introduce the notion of infantile sexuality was a revolutionary act of courage at the time and still is to a large extent.

Freud's pulsion theory of psychosexual development outlined in *Three Essays on the Theory of Sexuality* (Freud, 1905/2000) was a paradigm shift. With it he took on the taboo idea that infants from birth might be driven by erotic desires that quickly extend and transcend survival instincts evolved by the species. In the history of ideas, de facto, Freud offered with his pulsion theory the most comprehensive account of what might drive behavior at birth, aside from conditioning.

Obviously, Freud's theory was not the first published account on the questions of early experience and what it might be like to be an infant, but Freud's account was the most thorough and inquisitive to be proposed on what might constitute the psychoaffective forces driving behavior at birth. This account opened a whole new vista on what might be the constitutive elements of subjective life.

Although Freud's pulsion theory and the theoretical concepts inferred continue to be criticized, it was the first to ground subjective life, from the outset, in the experience of the body as we perceive and act in the world, an embodiment of psychic life, its somatic grounding that is now routinely vindicated by current research in the cognitive and affective neurosciences (see Barsalou, 2008; Damasio, 1995; Gallese, 2007; Gazzaniga, Ivry, et al., 1998, for a review).

The important contribution of Freud's pulsion theory is that it grounds psychic life in the feeling of the body, particularly certain bodily regions (oral, anal, genital) invested successively by the young child in his or her development. At the core of this theory, there is the pleasurable quest for bodily feelings (excitability) and its control (search and suppression): a drive-reduction account that remains a powerful causal account of what might motivate psychic life at the outset. There is also the general, rightful intuition that at birth, the body is the primordial locus of exploration and meaning making.

However, the concepts of *autoeroticism* and primary narcissism expressed by the infant, both at the core of Freud's (1905) pulsion theory, need serious revision in light of recent progress in infancy research. The Freudian idea of a first drive toward autoeroticism and the view that early psychic life is primarily revolving around a basic form of narcissism are now problematic. Autoeroticism as a primary drive reduces early experience of being in the world to some sort of blind, circular, nonobjectified, and autistic quest toward bodily excitation and suppression. We now know that there is much more than blind autoeroticism in the life of newborns.

Starting State Synesthesia

Throughout our lives we try to establish what can be counted on and relied upon to survive and make sense of being alive in this world. This quest is already embodied in the neonate, and that is the built-in focus on what can be expected and trusted in a world that is by definition constantly changing, associated to a subjective experience that is fundamentally dynamic, the stream of consciousness William James writes about.

The question is what mechanisms might jumpstart the sense of sameness expressed by infants at birth? What might drive newborns to focus their attention and learning on what remains the same in the midst of constant changes? Based on recent discoveries in behavioral neurosciences, some basic mechanisms might jumpstart the innate sense of sameness expressed by newborns: in particular what would amount to a starting experiential state of acute *synesthesia*.

Synesthesia corresponds to the spontaneous, implicit metaphorical experience of a sensation or percept in one modality that is simultaneously experienced in another. For example, one might experience the particular timber or pitch of a sound with the vivid experience of a specific color, the experience of time duration corresponding to the obligatory experience of a particular spatial layout or form (Simner et al., 2006). Neuroscientists have now established the embodied (neurobiological) reality of such synesthesic experiences that, according to existing surveys, are part of the life of approximately 5% of all adults (Hubbard, Arman, Ramachandran, & Boynton, 2005; Spector & Maurer, 2009).

What is of interest to us here is the idea proposed and tentatively documented with infancy research by Spector and Maurer (2009)—that adult cases of synesthesia

might in fact be remnant and magnifying cases of intersensory connections that are present at birth, pruned, and somehow inhibited in the course of typical perceptual development. But these connections are expressed in muted forms in *all adults,* as Spector and Maurer put it. Accordingly, synesthesia could be the natural starting state of our subjective sensory experience. We would indeed start off with a conflation of all sensory modalities as suggested by William James (1890) in his statement about blooming, buzzing, confusion. Here is what James had to say:

> The physiological condition of (the) first sensible experience is probably nerve-currents coming in from many peripheral organs at once. .... In a new-born brain, this gives rise to an absolutely pure sensation. But the experience leaves its "unimaginable touch" on the matter of the convolutions, and the next impression which a sense organ transmits, produces a cerebral reaction in which the awakened vestige of the last impression plays its part. Another sort of feeling and a higher grade of cognition are the consequences; and the complication goes on increasing till the end of life, no two successive impressions falling on an identical brain, and no two successive thoughts being exactly the same. (pp. 7–8)

This experiential conflation or pure sensory experience elegantly described by James is possibly the symptom of a major competence and not an incompetence as it has been taken by most infancy researchers over the past 50 years.

Infants are born with the ready-made opportunity to link experiences from the various sense modalities, experiences that co-occur and tend to be qualitatively linked, corresponding to particular feeling tones and profiles.

From the start, intermodal systems might exist that allow these sensory experiences to coalesce into the affective core of subjective experience that ultimately gives it *values*: values in rudimentary polarized terms such as pleasure or displeasure, stress or calm, soothing or enhancing, attunement or disharmony, bonding or estrangement. All these represent affective meanings (good or bad feelings) that are at the core of subjective experience, particularly in early development.

This affective core cannot be simply dissociated from subjective experience, as abstract and rational such experience might be later in development: as for example in the epistemic pleasures and satisfactions in discovering a theorem, in the building of a coherent argument, or in the reaching of an agreement with others.

But what kind of empirical evidence is there that supports the assertion of a rich primitive sensory conflation, a conflation that would harmonize rather than confuse early experience?

In relation to *synesthesia*, there is an abundance of empirical evidence showing that infants from birth are readily able to process information across sensory modalities. One-month-old infants are reported to discriminate an object they see projected on a screen based on the previous experience of an analogous object explored with their mouth only (i.e., a smooth spherical pacifier or a bumpy spherical pacifier with a knobee texture; Meltzoff & Moore, 1979). In another series of highly controlled, careful psychophysical studies on newborns in the early 1980s, Lewkowiz and Turkewitz (1980) demonstrated that neonates transfer learning from the auditory to the visual modality. Following visual habituation to either a bright or a dimmed light, they responded differently to corresponding soft or intense sounds in the auditory domain.

In support of such unitary or common functioning of the senses at the outset, an even older neurobehavioral study by Wolff and collaborators (Wolff, Matsumiya, Abrohms, van Velzer, & Lombroso, 1974) shows that if the tactile stimulation of the newborn's wrist evokes activation of the somatosensory cortex, this activity is significantly enhanced when the infant hears also a white noise. Such auditory-tactile interaction is not found in adults, a phenomenon that appears to be specific to the perceptual experience of newborns.

As additional developmental evidence on an early unitary functioning of the senses, let me mention the work of Neville and collaborators showing that if infants respond to spoken language with, as expected, enhanced activity in the auditory cortex, unlike adults and children, they also respond with enhanced activity in the visual cortex (Neville, 1995).

Finally, in support of the natural primacy of synesthetic experience, Mondloch and Maurer (2004) show in a series of studies that 2–3-year-old children tend to be naturally inclined to perceive the same pitch–lightness, color–letters, or sound–shape correspondences typically expressed by synesthetic adults (but also to some extent by nonsynesthetic adults). Young toddlers tend, for example, to systematically perceive that a higher pitch sound goes with a brighter color; a nonsense word made of rounded vowels goes with a jagged shape (e.g., te-ta-ke goes with a sharp-edged form), or that the letter A goes with the color red).

In all, these few empirical examples taken from the developmental literature on synesthesia, and there are many other, support the idea of a highly organized intermodal and resonating experience at birth. Early perceptual experience is made of rich sensory correspondences and implicit "a-modal" representations that can be said to be

metaphorical because they transcend the particularities of the sense modalities as singular perceptual systems. It is, and this is important, an experience that carries rich conflation and correspondences, not the cognitive confusion that has been assumed by many infancy researchers, including myself, since James's misconstrued blooming, buzzing confusion.

Experiential Vicariousness at Birth

In addition to starting state synesthesia, there is also abundant empirical evidence and even precise animal models substantiating the possibility of an innate capacity for vicariously experiencing self-produced and other-produced action: the experiential (phenomenological) equivalence between the observation and the execution of the same action. As a quick reminder, in the original experiments, Rizzolatti and his team from Parma found that the responses of single nerve cells recorded in area F5 of the premotor frontal cortex of the macaque monkey discharge equally when the monkey itself performs a specific action (e.g., reaching for a peanut) or when the same monkey observes another monkey (or another person) perform the same action (Rizzolati, Fadiga, Gallese, & Fogassi, 1996). These cells are thus multimodal by nature and are activated when a particular action is performed by the individual or seen performed by another.

This discovery has had much resonance as it might provide some biological validation to the idea that there might be a deeply rooted system matching self and others' representations, a mirroring system that could be the constitutive element of higher order phenomena like empathy, language learning, and basic embodied intersubjectivity. Note, however, that these ideas remain highly controversial (see Hickok, 2014). Plausible yet indirect behavioral evidence of mirror systems functional at birth is provided by the numerous research on facial imitation in neonates, the matching reproduction of facial expression, tongue gestures or emotional displays, actions that are seen repetitively being performed by an adult model at close visible range and that are systematically reproduced by the infant (e.g., Meltzoff & Moore, 1977, 1997). Such imitative responses in neonates suggest that typical infants are born with the necessary mechanism that would allow for the experience of an equivalence between the perception and the execution of actions (Lepage & Théoret, 2007). In James's terminology, infants would be born with the opportunity to experience the *sameness* of what is done by the self or what is seen done by somebody else or vice versa. Rather than in a state of cognitive confusion, infants would therefore be able from the start to experience and exploit in future learning, the *analogical link* between the products of two different agents: something self-generated and the same thing generated by others, in the same way that they would be able to experience the analogical link between the varieties of sensory experiences in their incipient *synesthesia*.

It is worth noting that the importance placed here on an innate sense of sameness expressed by neonates is also at the root of analogical reasoning and processing, the mechanism by which novel situations are understood in reference to what is familiar (the same) and that developmental psychologists view as a core mechanism of cognitive development (see Gentner, Holyoak, & Kokinov, 2001). The analog format of early representations that would allow for sameness detection across domains is demonstrated in multiple infancy works documenting the existence of a general system supporting the comparison and determination of relative magnitude in terms of number but also spatial extent, frequency, and duration (see Lourenco & Longo, 2011, for a thorough review of exiting infancy research and lively debate around the issue). Such general magnitude system, presumed to be innate, would cut across numerical, spatial, and temporal domains, therefore entailing some starting state analogical and synesthetic abstraction. As suggested by Walsh (2003, p. 486):

> Given the utility of relational inferences on quantities and the power of a statistical picture of the physical world that would extract co-variance of time, space and quantity, it would perhaps be as maladaptive for the infant brain not to use a common metric as it is difficult for an older child to unbind these three elements when learning mathematics.

CONSTRAINTS ON EARLY EXPERIENCE

In the short history of modern developmental psychology, theoretical controversies have revolved primarily around the relative role of innate biology (i.e., nature expressed in maturational and evolutionary factors) and the relative role of experience and learning factors (i.e., nurture). Progress in infancy research during the last 40 years strongly supports a marked theoretical shift from either strict empiricism (behaviorism) or constructivism à la Piaget (1967/1971) and toward a third solution to the nature–nurture controversy, not unlike the *diathesis–stress* model found in recent theories of developmental psychopathology (see Cummings et al., 2000).

Truthfully, 50 years of infancy research certainly debunk the myth of the tabula rasa and reinforce the major role played by evolved propensities to experience and construe

the world in specific ways, what philosopher Jerry Fodor coined *modularity of mind* in a highly influential book of the early 1980s (Fodor, 1983). However, these propensities change and are transformed in the course of the first weeks, when infants swiftly manifest new fluencies in innate propensities and also develop new capacities that are qualitatively different and more powerful to resolve problems, adapt to new situations, construe what is going to happen next. They also rapidly become better at picking up invariants over change, parsing such invariants to be memorized for further inferences.

Admittedly, when the tabula rasa was debunked (e.g., Pinker, 2002), it created blind spots on the actual process of learning and the mechanisms underlying developmental changes. The particular focus of dissident infancy researchers then was inspired by dynamic system theories and the self-organizing emergences of new forms in physics (Thelen & Smith, 1996) or epigenetic phenomena in the realm of biology by which experience crucially determines the phenotypic expression of genes (Gottlieb, 1971; Oyama, 2000).

In spite of such neglect, the majority of infancy researchers have focused on accounting for innate or precocious competencies in acting, perceiving, and representing objects, people, and the self.

Experiential Versus Conceptual Awareness

Newborns' experience of the world is rich from the start—within the polarity of pleasure and pain, restfulness and agitation, approach and avoidance. Newborns cry and fuss when hungry or tired. They show irrepressible smiles with eyes rolling to the back of their head after a good feed. They feel something, expressing unmistakable pleasure and pains. These expressions have adaptive functions, forming crucial signals for caregivers on whom newborns rely to survive. But how much unity and embodied self-awareness can be ascribed to such emotional, obviously not yet objectified experience of being alive in the world?

To address this question, it is necessary to distinguish two basic forms of being in the world: the *experiential* and the *conceptual*. This is not a new approach, supported and proposed by recent research and theories in cognitive neuroscience, in the footsteps of James proposing a distinction between the I or experiential self and the Me or conceptual (objectified) self. Damasio (1999) emphasized the fundamental difference between core consciousness and extended consciousness about the self and events that are construed over time and emerging with language. Similarly, Edelman and Tononi (2000) called for a distinction between primary- and symbolic- (language- and narrative-) driven consciousness. A large body of research in neuroscience supports the experiential diversity of being aware in the world, including blind-sight, hypnotic dissociation of pain, and other highly relativist (as opposed to real or core) perceptual phenomena (see Gazzaniga, Ivry, & Mangun, 1998). There are different kinds of awareness, not all necessarily requiring re-cognition, language, or the capacity to represent representations as in meta-cognition. It is justified to talk about infra- or prelinguistic awareness. There are indeed markedly different ways of being aware and conscious, as opposed to nonconscious or unconscious (Rochat, 2009).

Newborns are not yet conceptually aware of being themselves alive in the world, obviously. However, they are *experientially* aware. Newborns, when not sleeping, are not merely in a wakeful state of confusion between what they feel and what causes them to feel. If they see a face or are struck by an object, they do not become this face or this object. Although not yet conceptualizing them as objects of reflection, they do not confound them with their own subjective feeling or sensory experience. This can be assumed to the extent that newborns feelings and behaviors cannot be simply reduced to automatic reflex responses, like the mechanical adjustments of a thermostat or any kind of automata.

Infants are born predisposed to act with purpose, oriented toward indispensable resources in the environment, be it food, comfort, or protection. These innate functional action systems are what unify the experiential awareness of newborns. It is also what justifies the ascription of selfhood from the outset of development.

Although babies are born with poor contrast sensitivity and grating acuity (Banks & Shannon, 1993), infancy researchers investigating newborn vision demonstrate that despite the obvious developmental lag of the modality, active perceptual processing does take place at birth. For example—and relevant to our discussion—using habituation and novelty preference paradigms researchers have established that newborn infants, only a few hours old, when awake and alert, perceive the real (distal) size of objects, not the varying (proximal) sizes projected onto the retina. Newborns perceive size constancy of objects (Granrud, 1987), most likely via visuo-proprioceptive convergence cues from both eyes as they line their gazes and focus onto the distal object (Kellman & Aterberry, 2006). In all, this kind of empirical evidence suggests that newborn infants have feeling experience and are not just limited to sensing what is recorded at the proximal level of the receptors (i.e., the retina). Early perceptual competency

of perceiving a world that is distal and objectified in relation to the self forms the necessary core for the perception and control of self-produced action in relation to nonself things in the world.

Functional Propensities of Newborns

Newborns are born actors and not just passive "reactors" to stimulations (any stimulations) as pioneer child psychologists tended to describe the starting state of development: reflex montages or systems following Piaget, pure autoeroticism according to Freud, or random reactions as suggested by James. In addition to automatic reflex responses, that are clearly part of the child's survival kit and innate endowment, newborns also act in certain organized and oriented ways that are not merely automatic in nature. Thumbs are brought to the mouth for sucking, eyes are moved to track objects selectively, and leg movements are organized in coordinated stepping or kicking patterns. Even in the womb, fetuses manifest actions with some invariant patterns and a compression of degrees of freedom that is oriented outward, toward the environment, making movements of the infant and related perceptions more than merely random or disorganized.

In recent years, research based on the ultrasonic observations of fetuses demonstrates the remarkable continuity between prenatal and postnatal behaviors (Prechtl, 1984). Fetuses and newborns show the same rich repertoire of organized patterns of movements that are not simply random, but rather preadapted to tap into vital environmental resources such as food or comfort. These movements are not of a solipsistic and endogenous nature like tics or twitches. Rather, they are outward oriented, coming to closure with particular external consequences (e.g., food or thumb in mouth; skin or eye contact with particular objects in the environment). Such movements are actions, not mere reflexes.

Reflexes and action systems correspond to two radically different control systems and hence to two radically different psychologies. The beating of the heart, the movements of the lungs in breathing, the shaking of the whole body under a cold spell, the knee-jerk response, or the blinking of eyelids in response to an air puff all belong to the first kind of bodily movements. They are autonomic and reflex responses of the organism. The control of such movements is endogenous and self-contained. These movements consist in highly predictable stimulus–response loops. They are in essence automatic, *triggered* by particular stimulations.

Following physiologist Sherrington's (1906) first account of this kind of bodily movements, the control is encapsulated and rigidly prescribed within the organism as reflex arcs. It clearly involves subcortical neural networks as surgically decorticated animals continue to express such movements. The control of such movements can be described as closed feedback systems like thermostats controlling for constant temperature inside a house. Closed control systems are simple when considered in isolation, but they are complex when considered in interaction with each other. Each is calibrated to respond to particular ranges of stimulation from the environment that are internal and external to the body. Each system controlling for a particular autonomic/reflex response is also adapted to interact with myriad other similar systems that, in concert, maintain the integrity of the organism as a whole living and adapting system.

In brief, autonomic and reflex movements are controlled by closed-loop feedback systems that ensure basic physiological functioning. They keep the individual organism alive, but such movements do not involve any perception or any particular higher order treatment of basic physiological signals or sensations. In a way, this kind of bodily movement is sensitive but psychologically blind to its responding environment. The movements are triggered by nonspecified circumstances.

The second kind of innate bodily movement is *action systems*. They are more than autonomic or reflex responses, also expressed from birth and prominent during the first six weeks of life. Such movements are distinct from the first kind on two basic grounds. First, they are movement systems consisting of actions that are oriented toward particular functional goals. These systems are by definition adapted to tap into available resources that exist outside the individual organism, in the surrounding environment: food, surfaces, objects, or people. Second, these movements are organized into systems that are flexible, capable of changing based on previous experiences, and adjusting to novel circumstances. They allow room for learning, controlled by open-loop feedback systems. Although still unintentional, this second kind of movement entails perception and learning, some psychology, and presumably higher order cortical involvement.

Infants at birth show more than autonomic/reflex arcs (Rochat, 2001 for a general review). Sucking, grasping, stepping, rooting, or head turning are too often construed as reflexes or automatic responses triggered by non-specific stimulations. Multiple studies show in fact that such movements need to be construed as actions rather than reflexes, actions that are already oriented toward particular features and resources in the environment: for example, faces or objects with a certain shape, texture, consistency, or smell.

Above and beyond the search for optimum visual stimulation and in particular toward maximum light contrast, immediately after birth newborns track with their eyes objects that move close by in their field of view. More impressive is the fact that they do so preferentially when the objects consist of face-like displays. Research shows that they tend to track more canonical face-like displays (two adjacent dots for eyes above vertically aligned two dots for nose and mouth) compared with noncanonical face-like displays with the same but scrambled features (Morton & Johnson, 1991). Similarly, newborns tend to suck differentially on pacifiers that are more or less mimicking the biological nipple of the mother. They suck less and increase oral exploration as a function of the eccentricity of a pacifier compared with the biological nipple in terms of texture and consistency (Rochat, 1983; Rochat & Senders, 1991). We found the same kind of results when recording newborns' grasping of objects varying in texture and consistency that are placed in one of their palms (Rochat, 1987). Researchers have even established that newborn infants are significantly more inclined to orient their face toward gauze impregnated with their own mother's amniotic fluid or breast milk compared with gauze impregnated with the amniotic fluid or the breast milk of another woman who just gave birth (Marlier et al., 1998a, 1998b).

If newborns orient and root to smells or face-like displays, if they suck and grasp at objects introduced in their mouth or in their hands, they do so with discrimination and preference. This kind of movement is not made of autonomic, reflex responses triggered by nonspecific stimulation. It is under the control of previous experiences (learning) and intrinsically oriented toward particular environmental resources. It calls for some psychology engaging more than subcortical structures. However, such control is not yet intentional proper. It arises in the context of adaptive actions generated by the single individual in relation to physical objects and the physical aspects of people. It does not depend on reciprocal communication and shared experience with others or intersubjectivity.

Built-In Motivational and Attention Systems

Born in a world of values, from the start infants experience the basic affective polarity of approach and avoidance, events that are either qualitatively attractive or repulsive. It is well documented, for example, that from birth infants are attracted to sweet tastes and avoid bitter or sour tastes (Steiner, 1979). This built-in motivational system is linked to automatic positively rewarding or negatively rewarding mechanisms. Reward mechanisms via endogenously triggered opioid (endorphin) pathways are now well supported by numerous animal models (see, e.g., Barr, Paredes, Erikson, & Zukin, 1986; Blass, Fitzgerald, & Kehoe, 1987). In other words, infants are born prepared to be reinforced in certain ways in their learning and channeled in their experience and to be drawn to a particular carving of attractive objects in the environment, including their mother's smell and the taste of maternal amniotic fluid or the colostrum laced with sweetness, a reward needed for newborns to survive (see Marlier et al., 1998a, 1998b).

At a more epistemic level, infants are also born with typical ways of attending the visual world. Visual activity of the newborn is endogenously organized and not simply reflex-like, not just triggered by light stimulations.

In a seminal book, Marshall Haith (1980) systematically documented via an eye tracker the visual behavior of neonates in the dark, looking at a homogeneous ambient light environment, and when presented with various contrasted contours (e.g., solid horizontal, vertical or oblique black and white bars). Haith reveals the "systematic rules that babies look by"—the title of his book. His research shows that infants are oriented toward maximum information in the environment guided or rewarded toward maximum rate of cortical firing, which corresponds to the zeroing the fovea and scanning where there is maximum contrast in the environment. Newborns eyes are attracted by the defining edges of a contour. Once located, these edges are systematically scanned back and forth to generate maximum cortical firing rate. From his series of studies, Haith concludes that newborn function visually according to precise rules driven by specific reward contingencies (maximum cortical firing rate, or high firing rate principle according to Haith). These innate rules constrain infants from the start to carve meaningful perceptual information, hence specific representations about their environment, an interesting alternative to either radical tabula rasa (nurture) or nativist (nature) perspective. As Haith (1980) pointed out:

> Perceptual theorists agree that homogeneous surfaces are uninformative, it is the edges and boundaries of stimuli that carry the important information. One could imagine a sequence in which babies first scan areas of high contour density for their firing potential. Such activity would facilitate neural growth and, as associative areas and memory capabilities develop, these same stimulus regions would provide information for synthesizing objects and placing them in a spatial-temporal perspective. (p. 124)

Built-In Sameness Detection System

If we have learned one thing in recent years from studying babies—and there has been a huge wave of interest in

studying infants in the past 40 years—it is that from birth, and even prior to that based on the numerous evidence of fetal learning (Lecanuet & Schaal, 1996), infants are active in processing invariant information over changes. In their inclination to scrutinize novelty hides a deep look for sameness. They avidly look for regularities in the environment, and this is the name of the game from the outset: we are born and built in a way that what we are primarily preoccupied with is the detection of what remains the same in the midst of constant changes.

Throughout our lives we try to establish what can be counted on and relied upon, building trust and coherence. This quest is already embodied in the neonate, and that is the built-in focus on what can be expected and trusted in a world that is by definition constantly changing, associated to a subjective experience that is fundamentally dynamic.

But prior to developing these ideas, it is important to insist that the focus on *sameness* in the environment, which seems to be the core aspect of infant behavior and development, remains a core aspect of the human mind all through the life span. It might even be thought to be at the root of morality and the conception of justice and equity across human cultures. As James wrote over a century ago: "The mind can always intend, and know when it intends, to think of the Same . . . . This sense of sameness is the very keel and backbone of our thinking" (James, 1890, p. 459).

The past 40 years of booming infancy research did certainly debunk a great deal of strong-held common assumptions: that babies were born cognitively helpless and passive, their behavior disorganized. Prior to this research, it was not uncommon to construe infants as born blind and oblivious of the world surrounding them, a blank slate in need of fundamental growth and learning, often thought to be born in a vegetative state that kept them alive and tentatively able to receive indispensable care and protection from others. These views have certainly changed, but the fact that human children are pretty helpless at birth should not be overlooked, particularly when comparing them to the infants of other species. These ancient views were not that counterintuitive after all.

Compared with other species, humans are indeed born too soon, greatly immature and markedly dependent on others to survive. This is due to the combination of the proportionally larger brains we evolved as a species, together with the narrowing of the female's birth canal associated with bipedal locomotion, a posture uniquely evolved by our species and linked to protracted external gestation, namely, that we are born much sooner compared with other primate species (Montague, 1961; Trevarthan,1987). We start standing and roaming the world on our own by 12 months, and it takes many, many long childhood years to separate from our own original niche and to become autonomous to reproduce this cycle of development with new progenies.

The premature human birth leads to a state of protracted dependence during approximately one-fifth of our life. This remarkable dependence shapes our psychology from the outset. It is a simple, straightforward fact yet is probably the major determinant of what makes us psychologically unique in the animal kingdom.

The new wave of infancy research shows not that infants are born much more mature than previously thought but that infants are born much better equipped to tap into and exploit the prolonged state of dependence in which they are born. As Bruner (1972) wrote years ago, there are "uses of immaturity" (p. 1) by the young child. Numerous research tapping into preferential looking, and sucking, visual familiarization; violation of expectations; and other clever habituation and dishabituation paradigms show that infants from birth and even prior in the confine of the womb are remarkably quick to learn (see Rochat, 2001, for a review).

The most solid and reliable finding is indeed that healthy young infants get easily bored and are particularly inclined to seek novel information. From birth on, infants expect particular outcomes to occur based on past experiences and show a natural inclination to build up on new expectations.

Two-month-olds are attuned to complex probabilistic algorithms or conditional probability that one particular event will be followed by another, for example, in their ability to discriminate among strings of speech sounds they hear successively or the frequency of lights flashing at different locations in the environment (Haith et al., 1997; Saffran et al., 1996).

Infants show all this remarkable learning ability while not having to worry about being fed, getting enough cuddling, or living in wet diapers. Their protests are typically heard, and they are able to explore and encounter the world around them in playful ways. Childhood is, for the most part, a prolonged immaturity that translates into a prolonged, socially secured, and assisted opportunity for a free license to learn and to explore, to fantasize, and to realize these fantasies in the unbridled works of children's imagination. But children's free license to explore and to play is not just free and self-organized; it is also highly constrained by early core representations of objects, self, and others and by innate propensities to imitate and mirror the mental states of those interacting with the self.

Between birth and 2 months, noticeable changes occur, particularly in the social domain. By 6 weeks, infants universally begin to respond to faces with smiles that are not just automatic or linked to feeding or satiety but that are socially elicited, taking place in face-to-face

exchanges and active emotional coregulation. This is what is generally recognized as unmistakable demonstration of primary intersubjectivity or first sign of an infant's active sense of shared experience with others. These face-to-face exchanges are, in the broad sense, aimed at coregulating feelings and at creating mutual affective attunement, a *sameness of feelings* with others in a mutually affective proto-dialog and emotional entrainment that has been extensively documented in the past 30 years.

This mutual affective entrainment is typically geared toward the maintenance and coregulation of a shared happy, often exuberant, state first initiated by the adult but also increasingly initiated by the infant, particularly from approximately 7 months (Rochat, 2001; Striano & Rochat, 1999). Note, however, that such coregulation around a shared state does not require the context of face-to-face exchanges particularly nurtured in Western industrial cultures. It also occurs via different sensory channels when babies are tied to the back of an adult, days in and days out, or being carried on the hips of older siblings.

In the realm of social exchanges and intersubjectivity with a focus on shared feelings, the name of the social game is again about sameness detection. It is about the mutual monitoring of *sameness* in reciprocal affects and emotional expression, including the timing of such expression that specifies mutuality: whether, for example, the mother is more or less responsive to changes in the emotional expression of the infant and vice versa or whether the infant is more or less responsive to the mother (Bigelow & Rochat, 2006).

We now know that by 2 months infants become very much attuned to this relative mutuality of emotional responses, showing reliable negative responses when their expectation is violated, as in the case of the famous still-face situation (Tronick, Als, Adamson, Wise, & Brazelton, 1978). From at least 2 months, infants detect and overtly react to the violation of mutuality expectation; in other words, they react to the fact that others are not responding with *equivalent* emotional responses. What developmental research shows is that possibly from 2 months, and certainly by 7 months, infants create and promote similarities, hence sameness in feelings. They express the active propensity to create equivalent experiences with others.

What is intriguing is that this propensity becomes what is often identified as the central piece of cognitive and symbolic development, in particular language development. This central piece is the emergence by 9 months of *secondary intersubjectivity* with the active sharing of attention in reference to objects in the environment (i.e., joint attention; see Tomasello, 1995). Once again, this important development rests on the new active sense and monitoring by the child of equivalence (thus sameness) in the focus of visual attention between self and others. The name of the game remains essentially the same.

The sense of sameness is the broad concept used here to capture the natural inclination already expressed at birth to bridge experiences and draw analogies between things that are intrinsically distinct, between physical objects, but also between self and others. The sense of sameness not only pertains to linking physical objects, self, and people because they phenomenally look alike or share the same qualities but also to spatial-temporal relations among things and probabilistic co-occurrences of events: that something entering one end of a tunnel typically tends to reappear some time later from the other end; or that if I smile and coo toward someone I expect this person to somehow respond in comparable ways.

As mentioned previously, research demonstrates that these latter aspects (contingency and conditional probability detection) are expressed very early on. They are other expressions of infants' propensity to sense a link between things as belonging to a same set, a same chunk, or category of experiences.

It appears to be all part of the embodied propensity to sense sameness or equivalence (i.e., same value, same meaning). It is part of the analogous sense that is at the origins of concepts, symbols, and other representational signs that *stand* for something that exists in the world as separate entities: my own reflection in the mirror that I recognize and identify as the same as my embodied self. What is peculiar in human development, however, is that from around 2 years of age typical children start explicitly to recognize and identify themselves not just in mirrors but also through the evaluative eyes of others. They become *self-conscious*, what can be viewed as one of the major trademarks of our species around which developmental psychopathologies could revolve and be triggered (Rochat, 2009, 2014). But what might be the experiential (phenomenal) and representational (cognitive) origins of such development? As a preliminary discussion, we want to posit what is meant by representation, which we framed earlier as content and format of consciousness. It is a concept that can have multiple meanings across domains, including physics, engineering, neurobiology to psychology, and the cognitive sciences.

Core Knowledge and Conceptual Primitives

If from birth infants are motivated objective perceivers and actors, questions remain as to what they might construe

and represent about the world at a more conceptual level. Beyond mere sensory awareness, we now know that neonates are perceptually engaged as they feed, orient, control their posture, reach toward things, and explore objects and events, including their own body and how it affects the world. But what might they abstract and eventually memorize of what they sense and perceive of the world?

By 6 months, an abundant literature demonstrate that the typically developing infant has highly specific expectations about the world, in particular what pertains to the self, physical objects, and people (see Rochat, 2001). If those expectations are learned from experience, they are learned very fast, so fast that such learning has to be channeled by what could amount to core knowledge or some of conceptual primitives that are the evolutionary product of natural selection. Such cognitive primitives would strongly scaffold early experience, and there is now an abundance of empirical evidence pointing to such innate conceptual constraints, although the nature of such constraints remains highly controversial and source of various theories that continue to animate the field of infancy research (Carey & Gelman, 1991; Haith, 1998; Wellman & Gelman, 1992).

In relation to physical objects, numerous studies demonstrate that from at least 5 months of age, and sometime even earlier, infants construe objects as persisting in their existence when disappearing from sight (Clifton, Rochat, Litovsky, & Perris, 1991), expecting midsize objects to be substantial (i.e., to occupy space, to be spatially continuous, subject to gravity, and not existing at two different places at the same time— Baillargeon, 2004; Kellman, Spelke & Short, 1986; Spelke, Breinlinger, Macomber, & Jacobson, 1992). Infants detect who is agent and patient of an event action, inferring causality above and beyond mere associative learning (Leslie, 1982), even when the protagonists act at a distance, as in the case of chase scene between predator and prey (Rochat et al., 2004). In the first year, infants are already construing the goals and intentions of others who are acting on objects (Woodward, 2009), and by the middle of the second year they appear to anticipate the actions of others holding false beliefs about where things are in the environment (Onishi & Baillargeon, 2005). False belief understanding, hence theories of mind, typically reported as emerging across cultures between the age of 3 and 5 years (Callaghan et al., 2005), would actually project much deeper roots at an implicit level and in the context of nonverbal tasks.

The new wave of cognitive science in infancy has now established that within a few months and certainly prior to the emergence of syntactic, symbolic-based language, infants infer and hence represent a great deal of nonobvious aspects of the world. The products of such inference are what infancy researchers present and discuss as core knowledge (Kinzler & Spelke, 2007) or conceptual primitives (Carey, 2010; Mandler, 2004), infants inclined from the outset to make sense of reality with the default assumption that the world is made of entities that have hidden essence corresponding to defining (essential) ontological or constitutive characters (Gelman, 2003; Gelman & Bloom, 2000). Theoretically, what comes out of this new cognitive science of the budding child is that we might have evolved, as a species, the unique propensity to see and construe of the world in essentialist terms, constrained and guided from at least 2 months of age by what would amount to innate core knowledge or representations in the physical as well as the social domains, including the self. Traces of essentialism and core representation are now documented in infants as young as 5–6 months of age who appear to discriminate and prefer animated puppets or abstract protagonists wearing googly eyes that behave prosocially (e.g., help) rather than antisocially (e.g., hinder) (Hamlin et al., 2007). Six-month-old infants show surprise when an animated large cube backs out and shows deference to a smaller triangle, both wearing animal-like googly eyes (Thomson et al., 2011). They appear to infer social hierarchy and norms in terms of relative physical power. Same-age infants are even shown to recognize essential characteristics of social group formation by inferring that in-group members are supposed to act in similar ways (Powell & Spelke, 2013).

In relation to affectivity, recent findings uphold Bowlby's original idea that by the end of the first year and with the emergence of various forms of attachment to the primary caretaker, infants construct a representational, hence implicitly conceptual working model of their social and affective environment. One-year-olds who are either securely or insecurely attached to their mother based on the Ainsworth Stranger Situation manifest opposite expectations when viewing an animation movie with a large oval figure representing the mother was either helping or ignoring a smaller oval figure representing a baby. Securely attached infants looked longer and hence presumably are more surprised when the mother ignored the baby as insecurely attached infants showed the reverse: more surprise when the mother helped and nurture the baby (Johnson, Dweck, & Chen, 2007).

Revisiting Infantile Amnesia

First coined by Freud, the phenomenon of infantile amnesia invites to speculate that there might be a radically different mental organization at birth, or even *none*

whatsoever, newborns simply driven by unrepressed and chaotic pleasure orientation. It does indeed leave open the possibility of a different and incomparable experience to what older children and we as adults experience and represent of the world.

The memory black hole of the first months outside of the womb and in the world has naturally enticed philosophers to think of a primary representational incompetence, the incompetence of infants to create memories, even memories stored for later retrieval. This absence of conscious recollection from our life prior to the third birthday is universal. It is pervasive despite the claims of highly speculative therapies and other rather unscrupulous psychoanalysts reconstructing from patient hearsay what young infants might feel and what might be meaningful events for them.

If we consider infantile amnesia as the symptom of an original incompetence, their inability to store and represent sensory information, it is also presumably the symptom of an original incapacity to synthesize sensory impressions into the concepts that give the mind its conscious unity. Children, prior to 2–3 years of age, would be incapable of giving sensory experience its unified mindfulness. Translated in Kantian terms, infantile amnesia would be symptomatic of mental blindness. Babies' intuitions of the world and of their own body arising from sensory experience would be blind, not yet transcended into concepts and representations, not yet synthesized into bodies of knowledge that can be consciously retrieved. Historically, this is also what the founders of modern psychology assumed. Wilhelm Wundt, who established the first experimental psychology laboratory in Leipzig in the late nineteenth century, considered that infants could not help in the scientific understanding and conceptualizing of the adult mind. He writes in his *Outline of Psychology* (1897): "The results of experiments which have been tried on very young children must be regarded as purely chance results, wholly untrustworthy on account of the great number of sources of error. For this reason, it is an error to hold, as is sometimes held, that the mental life of adults can never be fully understood except through the analysis of the child's mind" (translated into English in 1907; cited in Kessen, 1965).

Progress in neuroscience might also have reinforced this intuition as we now have ample evidence that the brain of the young child continues to develop in marked ways during the first two to three years of life, particularly in the prefrontal regions of the neocortex, which are involved in the higher order synthesis of neural information as in advanced executive function, inhibition in problem solving, and intentional actions (e.g., Zelazo, 2004). Furthermore—and this is what delimits infancy from childhood—by the second year children become symbolic, increasingly proficient with language, and beginning to manifest an unambiguous conceptual sense of who they are (Bates, 1990). Their vocabulary becomes full of personal pronouns and adjectives like *I, me,* and *mine*. All these mental changes occur by the second to third year of life and correlate also to what is typically reported as our earliest, reliable memories.

From this point on, the veil of amnesia appears to be lifted. Memories are stored to become potentially retrievable and communicable in narrative forms (Dennett, 1992; Nelson & Fivush, 2004). From then on only, it would therefore be legitimate to postulate that the child possesses a mind that is explicitly conceptual, showing unity in the Kantian sense. This unity also implies a conceptual sense of who the child is as an entity among other entities, a person among other persons in the world. From the time they speak, children identify (recognize) themselves in mirrors and show embarrassment. They start to show off, begin to lie if necessary, and to engage in pretense. Arguably, the child's experience rises to mindfulness proper. It is unified over time and space. Representations of representations are synthesized and organized into abstract concepts that can be mentally manipulated at will to generate new truths and true previsions about future states of the world.

In short, historically, there has been a natural inclination, albeit with good reason, for many thinkers of the mind to believe that there might be a lack of representational unity at birth. The lack of unity would persist until children develop the ability to synthesize representations of the world that are memorable and organized along the continuum of time and space. If a concept of self is an a priori condition of unity in consciousness—as was suggested by Kant—it would be erroneous to speak of any notion of self prior to language, prior to the explicit ability to remember, conceptualize, and recognize the world symbolically, in particular within symbolic conventions. This, of course, would extend to any other non-symbolic animals, that do not possess language, namely creatures that are not capable of representing representations, not capable of organizing thoughts around a priori truths and within a continuous timeline that gives hindsight to the direct sensory experience of the world.

There are marked qualitative shifts in how and what the mind processes between birth and the onset of language, particularly when the child starts to remember an increasing number of past events in the explicit narrative formats of autobiographical memories (Nelson & Fivush, 2004). However, much research shows now that the phenomenon

of infantile amnesia is not due to a lack of unity or sense of self, as alluded to by the founders of modern psychology. In fact, infantile amnesia is becoming increasingly a misnomer given the flow of empirical evidence that demonstrates long-term procedural memory in infants of only a few months, infants who presumably should be deep into our memory black hole period (e.g., Bauer, 1996; Meltzoff, 1995; Rovee-Collier & Hayne, 2000). In addition, numerous studies show that the timing of first explicit memories (typically between 2 and 4 years) can vary greatly among individuals depending on memory content, gender, family structure, and culture (Nelson & Fivush, 2004).

It thus appears that children develop autobiographical memory progressively, incrementally, and parallel with language development. It does not emerge abruptly as if children were overcoming the obstacle of a generalized amnesia, hence a disorganized mind incapable of having organized representations of representation, not functioning rationally on the basis of a priori concepts, only finding unity and selfhood by their third birthday.

ROOTS OF INTERSUBJECTIVITY

By the second month, there is a behavioral revolution. Typical infants open up to the world in unmistakable ways. When not sleeping or crying, they spend markedly more time in a wakeful state, actively and spontaneously tracking and exploring objects, particularly faces in the environment (Wolff, 1987). When attending to faces, they begin to spend significantly more time exploring internal features, namely, eyes and mouth, compared with outside features such as forehead contours and hairline, which are preferentially attended to by infants less than 1 month of age (Haith, Bergman, & Moore, 1977). More significant is the fact that by 6 weeks of age, in a face-to-face situation infants begin to manifest smiling. Such smiling expression is elicited by the social engagement of others as they typically try to create a shared positive emotional experience with the infant.

From this point on, infants enter the give and take of interpersonal conversation—a privileged context in which they can differentiate their first-person perspective from the third-person perspective of the social partner with whom they converse. They develop a sense of shared experience or primary intersubjectivity, which in face-to-face exchanges is a first form of triadic exchanges that is turned toward the self of the infant, the infant being the prime topic of the communicative exchanges with the adult. From 2 months on and until approximately 9 months, the main topic of communication is the infant himself and not yet the objects that surround the infant and the adult (secondary intersubjectivity; see Tomasello, 1995; Trevarthen, 1979).

Sociality and Reciprocity in Typical Development

The sense of reciprocity is expressed very early in the life of the healthy child. By two months, infants start to engage in face-to-face proto-conversations, first manifesting signs of socially elicited smiles toward others (Rochat, 2001; Sroufe, 1996; Wolff, 1987). Such emotional coregulation and affective attunement are more than the mirroring process underlying neonatal imitation and emotional contagion evident immediately after birth (Meltzoff & Moore, 1977; Sagi & Hoffman, 1976; Simner, 1971). From this point on, infants express a new sense of shared experience with others in the context of interactive, typically face-to-face plays, what Trevarthen (1979) first labeled as *primary intersubjectivity*.

When infants start to engage in proto-conversation, they are quick to pick up cues regarding what to be expected next from the social partner. In general they are quick to expect that following an emotional bid on their part, be it via a smile, a gaze, or a frown, the other will respond in return. Interestingly, adult caretakers in their response are typically inclined to reproduce, even exaggerate the bid of the child. If the child smiles or frowns, we are inclined to smile or frown back at her with amplification and additional sound effects. There is some kind of irrepressible affective mirroring on the part of the adult (Gergely & Watson, 1999).

The complex mirror game underlying social cognition does manifest itself from approximately 2 months of age and from then on, infants develop expectations and representations as to what should happen next in this context. The still-face experimental paradigm that has been extensively used by infancy researchers for over 30 years provides good support for this assertion (see the original study by Tronick et al., 1978). Infants are disturbed when the interactive partner suddenly freezes while staring at them (Rochat & Striano, 1999). They manifest unmistakable negative affects, frowning, suppressing bouts of smiling, looking away, and sometimes even starting to cry. In general, they become avoidant of the other person, presumably expecting them to behave in a different, more attuned way toward them.

This reliable phenomenon is not just due to the sudden stillness of the adult, as the infant's degree of negative responses varies depending on the kind of facial expression (i.e., happy, neutral, or fearful) adopted by the adult while suddenly still (Rochat, Striano, & Blatt, 2002). Also, it appears that beyond 7 months old, infants become

increasingly active rather than avoidant and unhappy, showing initiative in trying to reengage the still-face adult. Typically, they touch her, tap her, or clap hands to bring the still-face adult back into the play, with an intense gaze toward her (Striano & Rochat, 1999).

Numerous studies based on this still-face paradigm and studies using the double video paradigm, in which the infants interact with their mother, whom they see on a TV, either live or in replay (Murray & Trevarthen, 1985; Nadel, Carchon, Kervella, Marcelli, & Réserbat-Plantey, 1999; Rochat, Neisser, & Marian, 1998), all show that early on infants develop social expectations as to what should happen next or what should happen while interacting with others. The difficult question is what do these expectations actually mean psychologically for the child? What does it mean for 2-month-olds to understand that if they smile toward an individual this individual should "normally" smile back at them? What does it mean that they pick up the fact that amplified and synchronized mirroring from the adult is an invitation for more bouts of interaction?

One could interpret these expectations as basic, possibly subpersonal, and automatic. Accordingly, face-to-face interactions are information-rich events for which infants are innately wired to pick up information, attuned and prepared from birth to attend to and eventually recognize familiar voices and faces (e.g., De Casper & Fifer, 1980; Morton & Johnson, 1991). From birth, infants would be attuned to perceptual regularities and perceptual consequences of their own actions, wired to prefer faces, human voices, and contingent events as opposed to any other objects, any other noises, or any other random events. Accordingly, this would be enough for young infants to build social expectations and manifest apparent eagerness to be socially connected as shown by studies using the still-face experimental paradigm or the double video system. But there is more than what meets the eyes of an engineering look at the phenomenon (Rochat, 2009). It is more than just mechanical and requires another, richer look to capture its full psychological meaning.

This proposal is based on evidence of major developmental changes in the ways that children appear to connect with others and reciprocate. Infants rapidly go beyond mirroring and imitation to reciprocate with others in increasingly complex ways, adding the explicit social negotiation of *values* to the process. This development corresponds to the unfolding of primary and secondary (i.e., triadic exchanges of the infant with people in reference to objects in the environment by 7–9 months), and also a *tertiary* level of intersubjectivity from at least 3 years of age. Table 10.1 summarizes the road map of various levels of intersubjectivity unfolding in typical development between birth and approximately 4 years of age.

Each transition represents a major extension. The extension from primary to secondary intersubjectivity is well accounted for in the literature (Bruner, 1983; Trevarthen, 1979; Trevarthen & Hubley, 1978; Tomasello, 1995; see Table 10.1). At the tertiary level of intersubjectivity, objects and situations in the environment are not just jointly attended to (secondary intersubjectivity) but also become *jointly evaluated* via negotiation, until eventually some kind of a mutual agreement is reached, also a crucial progress in the development of sociality and morality as we will see next.

Products of Emerging Reciprocation

When infants begin to open up to their social environment by reciprocating via smiling and cooing toward others interacting with them, and because of the obligatory propensity of others to mimic or *reproduce* with marked exaggeration what infants express (i.e., affective mirroring; Gergely & Watson, 1999), infants have the

TABLE 10.1 Levels of Intersubjectivity Unfolding in Typical Development

	Type	Context	Behavioral index	Process	Age
I	Mirroring	Face-to-face engagement	Imitation	Automatic simulation	Birth
II	Primary intersubjectivity	Reciprocal dyadic exchanges	Proto-conversation, social expectations	Emotional coregulation	2 months
III	Secondary intersubjectivity	Triadic exchanges about things	Joint attention; social referencing	Intentional communication and intentional coexperience	9 months
IV	Tertiary intersubjectivity	Triadic exchanges about the value of things	Self-recognition and embarrassment, use of possessives, claim of ownership, prosocial behaviors	Projection and identification of self onto others	20 months
V	Ethical stance	Decision regarding the value of things, what is right vs. wrong	Claim of ownership, sharing, distributive justice, theories of mind	Value negotiation with others, narration, meta-representation of reputation	From 4 years

Source: This table is based on the table included in Rochat & Passos-Ferreira, 2008.

unique opportunity to *objectify* themselves in relation to others. Face-to-face exchanges, turn taking, and the proto-conversations dominated by affective mirroring allow for self-objectification and the objectification of first vs. third person perspective. All this forms a privileged context in which infants can learn to distinguish their own and others' perspectives on the self, the basic prerequisite of intentional and referential communication. This context is the template for the intentionality expressed in relation to physical objects, intentionality that starts to be manifested by infants by the time they begin to smile in the context of reciprocal social exchanges (Rochat & Striano, 1999).

Infants might learn also to objectify themselves in the exploration of their own dynamic traces in objects they acted upon: a mobile they kicked or a ball they pushed (Piaget, 1952; Rochat, 1995, 2002; Watson, 1995). The effect of self-generated actions on the object does indeed reflect the dynamic and the amount of energy produced by the infant who can pause and contemplate traces of himself in such effects. However, in this context, the differentiation between the first- and third-person (object) perspective is possible only on a trial-and-error basis. Interactions with physical objects do not carry the bidirectionality of attention, the mutuality and tutoring guidance that a reciprocating adult typically offers to the child. Reciprocal exchanges are intrinsically referential in relation to each of the protagonists (infant and adult). They specify on-line, in a unique way, the alternating perspectives of each protagonist because of the give and take, reciprocal, and coconstructed format of conversational exchanges.

The learning of a differentiation between the first- and third-person perspective is facilitated by reciprocal exchanges. Infants can eventually generalize what they learn in interaction with others to their interaction with physical objects, rather than the reverse. It is reasonable to postulate the precedence of one format of exchange (social interaction) over the other (action on physical objects) to explain the emergence of intentionality in development, assuming of course that we do not postulate innate module for such stance.

Social partners (e.g., caretakers) work hard from the outset to reveal themselves intentional in communication and are quickly perceived as such by the infants. Children will eventually also perceive others as intentional *outside* of face-to-face communicative contexts, when observing them interacting and acting on physical objects. The generalization of an understanding of others as intentional with both objects and other people aside from the self opens up new, crucial opportunities for observational and imitative learning. These are often identified as basic mechanisms of cultural transmission that are considered, by some, to be unique to our species (Tomasello, 1999; Tomasello, Kruger, & Ratner, 1993).

Developing Self- and Social Awareness

For decades now, the mirror mark test has been used as an acid test of conceptualized self-awareness from both a developmental and comparative perspective (Amsterdam, 1968, 1972; Gallup, 1970). Self-directed behaviors toward a mark surreptitiously put on the face and discovered in the mirror would attest of self-concept, in other words an objectified sense of the self (but see also Mitchell, 1993; Rochat & Zahavi, 2011 for alternative views on the mirror mark test). What the individual sees in the mirror is Me, not another person, a feat that is not unique to humans since chimpanzees, orangutans, dolphins (Parker, Mitchell, & Boccia, 1995), and now magpies as well as elephants are also reported to pass the test (Plotnick & DeWaal, 2006; Prior, Schwarz, & Güntürkün, 2008).

The majority of children pass the mirror mark test by 21 months (Bertenthal & Fisher, 1978), although it depends on culture (Broesch, Callaghan, Henrich, & Rochat, 2011). But beyond the mirror mark test and what its passing might actually mean in terms of emerging self-concept, there is an early and universal reaction to mirrors that, in my view, is most revealing of human psychology. This reaction is the typical expression of an apparent uneasiness and social discomfort associated with mirror self-experience. The same is true for seeing photographs of one's self, or hearing the recording of one's own voice. Across cultures, mirror self-experience is *uncanny*, an expression of deep puzzlement. This is evident even by adults growing up with no mirrors and who manifest terror when confronted for the first time with their own specular image (see Carpenter, 1976). Looking at the self in a mirror puts people, young and old, in some sort of arrested attention and puzzlement. Mirror self-experience is indeed an uncanny experience (Rochat & Zahavi, 2011).

In general, aside from the landmark passing by a majority of children of the mirror mark test from around the second birthday, mirror self-experience develops to become incrementally troubling and unsettling for the healthy child. Such development is not observed by young autistic children, impaired in their reading of others' mind (Baron-Cohen, 1995), but passing the mirror mark test (Neuman & Hill, 1978). They will remove the mark from their faces when they perceive it but do not show the signs of coyness and embarrassment so typical of nonautistic children (Hobson 2002, p. 89). It appears that for autistic

children there is a different meaning attached to the mark they discover on their faces that they eventually touch and remove. This meaning would not entail the same kind of self-evaluation or self-critical stance in reference to the evaluative gaze of others expressed in typical children via self-conscious emotions. Autistic children's passing of the mirror test is not self-conscious proper and does not appear to entail any sense of reputation as defined earlier.

In her pioneer research on children's reactions to mirror and establishing (in parallel with Gallup, 1970) the mirror mark test, Amsterdam (1968, 1972) describes four main developmental periods unfolding between 3 and 24 months. In the *first period* of mainly sociable behaviors toward the specular image, infants between 3 and 12 months tend to treat their own image as a playmate. A *second period* is accounted for by the end of the first year, in which infants appear to show enhanced curiosity regarding the nature of the specular image, touching the mirror or looking behind it. By 13 months a *third period* begins, during which infants show marked increase in *withdrawal behaviors* by crying, hiding from, or avoiding looking at the mirror. Finally, Amsterdam accounts for a *fourth period* starting at around 14 months but peaking by 20 months when the majority of tested children demonstrate embarrassment and coy glances toward the specular image as well as clowning. These changes index the self-reflective and ultimately the unique self-conscious psychology unfolding in human ontogeny. Such psychology is the product of a complex interplay of cognitive and affective progress that take place during this early period of child development (Amsterdam & Lewitt, 1980), something that Charles Darwin inferred observing his own child long before the recent wave of experimental works around the mirror mark test.

In his book on the expression of the emotions in man and animals, Darwin (1872/1965) was struck by the unique and selective human crimsoning of the face, a region of the body that is most conspicuous to others: "Blushing is the most peculiar and the most human of all expressions" (p. 309).

Observing blushing in his son from approximately 3 years of age and not prior, Darwin highlighted the mental states that seem to induce human blushing:

> It is not the simple act of reflecting on our own appearance, but the thinking what others think of us, which excites a blush. In absolute solitude the most sensitive person would be quite indifferent about his appearance. We feel blame or disapprobation more acutely than approbation; and consequently depreciatory remarks or ridicule, whether of our appearance or conduct, causes us to blush much more readily than does praise. (p. 325)

These observations capture something fundamental and distinctive about humans, a unique motivation behind their social cognition: the exacerbated quest for approbation and affiliation with others, the unmatched fear of being rejected by others (see Rochat, 2009).

The expression of embarrassment in front of mirrors by 2 to 3 years is associated with the child's growing metacognitive abilities, in particular the child's growing ability to hold multiple representations and perspectives on the same thing, including the self. The recognition of self in the mirror is also for the child the recognition of how the self is publicly perceived.

From the point of view of neurophysiology, there is an apparent link between the emergence of metacognitive abilities around 2–3 years and the documented orderly maturation of the rostrolateral region of the prefrontal cortex. The growth of this prefrontal cortical region would correlate with the development of new levels of consciousness, in particular the transition from minimal to metacognitive levels of self-consciousness (Bunge & Zelazo, 2006; Zelazo, Gao, & Todd, 2007).

Elsewhere (Rochat, 2009), I interpreted the negative affective connotation of mirror self-experience (e.g., embarrassment and self-conscious emotions as opposed to positive jubilation) as expression of a universal tendency to hold an overestimated representation about the self that is at odds with what is actually seen by others, the latter truly revealed in the mirror. First-person (private) perspective on the self is generally overestimated compared with third-person (public) perspective. This interpretation is supported by the well-documented illusory superiority phenomenon found in adults (Ames & Kammrath, 2004; Beer & Hughes, 2010; Hoorens, 1993).

Arguably, such overestimation would bring about the experience of a generalized gap between private (first-person) and public (third-person) self-representations, a gap that is the source of basic psychic tension and anxiety, the expression of a generalized social phobia, and universal syndrome expressed from 2–3 years of age (Rochat, 2009).

An alternative interpretation would be that young children shy away from their reflection in the mirror, not because they are self-conscious but rather because they wrongly construe the presence of another child staring at them with some kind of a persistent still face, hence to be avoided. But this is doubtful considering, as we have seen, that very early on infants discriminate between seeing themselves or seeing someone else in a video (Bahrick, Moss, & Fadil, 1996; Rochat & Striano, 2002).

By showing embarrassment and other so-called secondary emotions (Lewis, 1992), young children demonstrate

a propensity toward an evaluation of the self in relation to the social world (the looking-glass self first proposed by Cooley, 1902). They begin to have others in mind, existing through in addition to with others.

Children begin to express secondary emotions such as shame or pride in parallel by 2 to 3 years of age and these are probably linked to the emergence of symbolic and pretend play. Such play entails, if not at the beginning but at least by 3 to 4 years of age, some ability to simulate events and roles, to take and elaborate on the perspective of others (Harris, 1991; Striano, Tomasello, & Rochat, 2001; Tomasello, 1999; Tomasello, Striano, & Rochat, 1999).

The process of imagining what others might perceive or judge about the self, whether this imagination is implicitly or explicitly expressed, is linked to the cognitive ability of running a simulation of others' minds as they encounter the self. There are fantasies and phantasms involved, the stuff that feeds the self-conscious mind and characterizes a metacognitive level of self-awareness (i.e., the construal and projection of what others might see and evaluate of us).

Social Dependence and Human Symbolic Psychology

For centuries the question of the origins and development of consciousness intrigued philosophers, moralists, educators, biologists, and more recently psychologists and neuroscientists: from Augustine, Rabelais, Rousseau, and Darwin to Freud and Piaget. All speculated how the experience of being in the world comes into place and is shaped in the early days of psychic life, from the moment infants come out from the obligatory, in appearance lethargic and silent 9 months gestation in the womb. Over 16 centuries ago, for example, in what is often considered the first self-narrative in the history of Western thought, Augustine in his *Confessions* expressed the idea that the origins of what one knows about the self is primarily social. Self-knowledge would be learned from others, particularly women because of the primal maternal bond:

> I give thanks to you, lord of heaven and earth .... For you have granted to man that he should come to self-knowledge through the knowledge of others, and that he should believe many things about himself on the authority of the womenfolk. Now, clearly, I had life and being; and, as my infancy closed, I was already learning signs by which my feelings could be communicated to others. (Augustine, 398 AD/2007. Confessions, 1.6.10.).

Augustine's intuition that we first learn about ourselves through contact with *the womenfolk* reflects a basic fact to which any speculation on early subjective experience should indeed refer to: that infants rely on others to live and survive. Necessary submission to the cares of others, the mother in particular, is the point of psychic origin. If neonates do their share to survive—breathing, eating, as well as orienting, approaching and avoiding—they are nonetheless born is a profound state of helplessness and dependence. This is the bottom line, particularly pronounced in the human infant who is born too soon after nine months of slow gestation (Rochat, 2001).

As already mentioned, the human birth creates a unique ecology of behavioral growth compared with other animal species, an ecology of protracted *exterogestation*, hence a particularly long and marked dependence due to immaturity (Bruner, 1972; Montagu, 1961). It is an ecology that has been constrained, among other contingent and cascading factors, by the evolution of bipedal locomotion. After nine months in the womb, the head circumference of the normally developing fetus becomes dangerously large for the mother and for itself.

Disproportionately big compared with other primate species, it also reaches a growth limit in relation to the birth canal of humans that evolved to be narrower with changes in pelvic bone configuration, reflecting itself the bipedal locomotion evolved by the *Homo* genus (Gould, 1977; Montagu, 1961; Trevarthan, 1987).

Human protracted exterogestation creates a unique case of altriciality in the animal kingdom, an unusual need for care from others and an exacerbated dependence on others to survive beyond the third year (when most children of all cultures today expand the circle of close caretakers to socialize on their own with peers in the more formal circle of school).

This state of protracted dependence is the specific context in which human consciousness and the human psyche take roots in their development. That is the original bath that we need to consider if we want to figure what it is like to be a newborn: it is first and foremost the experience of relying on generous caregivers, themselves conscious and reflective, willing to give freely and abundantly attention and care. It defines human experience at the outset. Attachment and dependence are evident in other animals. However, what is unique compared with any other mammalian species, is that human dependence is (1) protracted and (2) engages explicitly conscious (as opposed to just minimally conscious) others that have beliefs and communicate symbolically with one another.

In this primary context, how might it feel to be a newborn? What kind of sensuous experience might arise is such a great state of immaturity and dependence?

In *The Family Idiot*, Sartre's (1971/1981) biography of nineteenth-century writer Gustave Flaubert, he ponders this question at length and in remarkable depth. In the first part of this long essay ("The Constitution"), Sartre describes in detail the initial experiential state of total abandon to the cares of others. Sartre speculates that it is in this early submission to specific maternal gestures, attitudes and attention that one can find the roots of Flaubert's apparent passivity as a child and subjective disconnection as an adult, possibly also the source of his genius as inventor of the modern novel: "Gustave as a child, is not *made* to act; what he feels is dizzying submission to this constitutive nature experienced within him as the product of *others*" (p. 48; author's translation from French). He goes on a few pages later:

> The newborn, molded everyday by dispensed cares, internalizes the maternal activity of his own passive "being there", in other words the infant internalizes the maternal care activity as the passivity that conditions all the pulsions, and all the internal rhythms of desires, speeds, accumulated storms, schemas that reveal at the same time organic constancies and unspeakable wishes – in brief that his own mother, buried deep into that body, becomes the *pathetic* structure of affectivity. (p. 58)

Sartre (1971/1981) insists on the inescapable determinant of the mother as a whole person that determines the affective core experience of cared newborns. Maternal constitution, relative sensitivity, and life story is inescapably reflected in the kind of care, attention, and gestures mothers are able to dispense to their infant. Indeed, in the experiential life of newborns that revolves primarily in the passive reception of cares molded by others, there is more than the serving of basic survival needs such as food, warmth, and hygiene. There is also, as Sartre points out, the transmission of unique trans-generational idiosyncrasies or family truth:

> ... When the mother breastfeeds or clean her infant, she expresses herself, like anybody else, in her personal truth, which, naturally, sums up in it all her life, from her own birth; in the meantime, she realizes a rapport that changes depending on circumstances and people – for which she is the *subject* and that we can call maternal love. .... But, at the same time, by this love and through it, by that person, skillful or unskillful, rough or tender, the way her history made her, the child is manifested to himself. (p. 57)

In summary, Sartre (1971/1981) proposes astutely that experiential awareness of what it might be like to be a newborn is highly dependent on the subjective experience of others. It would be, in large part, socially determined and intersubjective to start with, in the context of dispensed cares. In other words, it would depend primarily on the interaction of a mother–infant experiential point of view, from which each partner would extract their own mutually defining meanings, not unlike the dialectic of master and slave: the newborn would extract a sense of imposed passivity and submission, the mother would extract a sense of duty and fulfillment in the instinctive call for maternal love and protection: a constitutive rapport of force.

Unfolding Levels of Sharing and *Conscientiousness*

With the intentional communication about objects that emerges by 9 months of age via social initiatives and explicit bouts of joint attention (secondary intersubjectivity), infants break away from the primary context of face-to-face exchanges. They become referential beyond the dyadic exchanges to include objects that surround the relationship. Social exchanges also include conversations about things outside of the relationship, becoming triadic in addition to being dyadic. Exchanges become object oriented or objectified, in addition to being the expression of a process of emotional coregulation. Infants now willfully try to capture and control the attention of others in relation to themselves via objects in the environment. At this point, however, the name of the game is limited to the sharing of attention just for the sake of it. Children measure the extent to which others are paying attention to them and what they are doing. They begin to check back and forth between the person and the object they are playing with (Tomasello, 1995), or they begin to bring an event to the attention of others by pointing or calling for attention to share the experience with them. However, such initiative ends there, and is typically not followed through in further conversation or coregulation. For infants, secondary intersubjectivity in triadic exchanges is a new means to control their social environment, in particular the proximity of others as they gain new degrees of freedom in roaming about the environment (Rochat, 2001). By becoming referential, infants also open the gate of symbolic development. They develop a capacity for dual representation whereby communicative gestures stand for and become the sign of something else (e.g., a pointing gesture as standing for a thing out there to be shared with others). Communication becomes intentional, transcending the process of emotional coregulation and affective attunement that characterizes early face-to-face, proto-conversational exchanges (i.e. primary inter-subjectivity). Yet it remains restricted to the monitoring of whether others are, or are not, co-experiencing with the child.

Nevertheless, with the emergence of intentional communication and the drive to co-experience events and things in the environment, infants learn and begin to develop shared meanings about things. To some extent, they also begin to develop shared values about what they experience of the world, but this development remains limited. For example, when facing dangers or encountering new situations in the environment, they are now inclined to refer to the facial expressions of others that are paying attention to the same events (Campos & Sternberg, 1981; Striano & Rochat, 2000). The meaning of a perceived event (e.g., whether something is dangerous or threatening) is now referred to others' emotional responses, to some extent evaluated in relation to others, but it ends there. The process does not yet entail any kind of negotiation regarding the value of what is experienced. The world is essentially divided into either good (approach) or bad (avoidance) things and events. Such basic social referencing emerges at around 9 months, in parallel to the propensity of infants to share attention with others and to communicate with them intentionally (Rochat & Striano, 1999; Tomasello, 1999).

By the middle of the second year of life, triadic exchanges develop beyond basic social referencing and the sense of co-experience with others that is the trademark of secondary intersubjectivity. The child now begins to engage in active negotiation regarding the values of things co-experienced with others. They manifest *tertiary intersubjectivity*, a sense of shared experience that rests on complex ongoing exchanges unfolding over time: things that happened in the past, are manifest in the present and are projected by the child into the future. The prototypical expression of this new level of inter-subjectivity is the expression of secondary emotions such as embarrassment or guilt.

In relation to the self, by 20 months, children begin to represent what others perceive of themselves and gauge this representation in relation to values that are negotiated. If they see themselves in a mirror and notice a mark surreptitiously put on their face, they will be quick to remove it and often display coy behaviors or acting out (Amsterdam, 1972; Rochat, 2003). They begin to pretend and mask their emotions (Lewis, 1992). In general, they become self-conscious, negotiating and actively manipulating what others might perceive and evaluate of themselves (Lewis, 1992; Rochat, 2009). From this point on (18–20 months of age), children project and manipulate a public self-image, the image they now identify and recognize in the mirror. It is an image that is objectified and shared with others, a represented public self-image that from now on will be constantly updated and negotiated in relation to others. Interestingly, by 20 months, children's linguistic expressions begin also to include the systematic use of possessives, children starting to claim ownership over things with imperative expressions such as "Mine!" (Bates, 1990; Tomasello, 1998). Such expressions demarcate the value of things that are jointly attended in terms of what belongs to the self and what belongs to others. This value begins to be negotiated in the context of potential exchanges, bartering, or donations. With the explicit claim and demarcation of property, the child develops a new sense of reciprocity in the context of negotiated exchanges of property, whether objects, feelings, or ideas. At around the same age, children also begin to demonstrate prosocial behaviors, engaging in acts of giving and apparent benevolence by providing help or spontaneously consoling distressed others (Zahn-Waxler, Radke-Yarrow, Wagner, & Chapman, 1992). Self-concept, ownership claim, and a new concern for others bring the child to the threshold of moral development and the progressive construction of an explicit sense of justice (Damon, 1994). What follows in development is a new level of social reciprocity that is increasingly organized around an ethical stance taken by the child. But this ethical level of reciprocity develops between 3 and 5 years of age and beyond as shown by our recent investigation of young children's sense of fairness in sharing across cultures.

There is a developmental trend from a reluctance to share to subtle, more reciprocal exchanges. For example, children between 3 and 5 years of age become significantly more flexible and systematic in adjusting their successive bids while engaged in bartering exchanges of stickers or toys. They are increasingly inclined to up their bids until an agreement is reached. This developmental trend is also associated with an increased understanding by the child of others' mental states, a trend that appears to cut across cultures (Callaghan et al., 2005).

As children start to claim explicit ownership and invest affects into objects of devotion, they do so by first manifesting unmistakable exclusivity in their possession, a blunt reluctance to exchange. They show overwhelming egocentrism. When the child begins to say "Mine!" she not only implies that "It is not yours," but also is explicitly stating defensive exclusivity, a reluctance to even contemplate sharing and an unmistakable claim that she wants to keep it for herself.

In cross-cultural observations (Rochat et al., 2009), we confirmed that this egocentric trend is a universal trend. We found it in 3-year-olds and to a lesser extent in 5-year-olds from all over the world, who are growing up in highly contrasted physical, social, economical, and

cultural environments. It happens in children living in rich or poor neighborhoods and in cultures fostering radically different values regarding private property. We observed this trend in children in small, highly collectivist villages of rural Peru or in small isolated fishing communities in Fiji. This same trend occurs in children growing up in violent and lawless as well as affluent neighborhoods of Rio de Janeiro; unschooled kids begging and living on the streets of Recife in Brazil; young children attending a Communist Party–controlled preschool in Shanghai, China; and in middle-class North America (Atlanta, Georgia).

In general, we found that across cultures, between 3 and 5 years, there is a robust developmental trend toward more equity in sharing. In conditions where the child was one of the two recipients, 3-year-olds tended to distribute overwhelmingly more candies to themselves, whether equity was possible or not. By 5 years, however, this trend was still evident but significantly tamed. Children continued to favor themselves and are selfish but markedly less. Interestingly this trend was the same in children from all cultures but was reduced in children growing up in small rural and collective communities (i.e., Peru and Fiji in our sample). In development, there is thus a universal drift in active sharing from massive to reduced selfishness between 3 and 5 years of age, a trend moderated by the cultural environment of the child. Despite the significance of cultural factors, the trend toward increased altruism/prosociality in sharing is remarkably robust from the time children begin to be explicit in claiming ownership.

Culture appears to play a role in the developmental pace at which the child becomes inclined to share with greater equity, but the general trend is there regardless of marked variations. In China, children were tested in a preschool that emphasized primarily group activities and sharing. Children always play, sing, and learn as members of a group, rarely as individuals isolated from the group. Such attempts are much less frequent in middle-class North American preschools, such as those of the children we tested in Atlanta. In Fiji or in Peru, the tested children lived in small, close-knit communities where public and shared properties dominate over ostentatious private ownership. When they exist, preschools in these regions are known to emphasize synchronized group activities in children.

The stability of this developmental trend is particularly striking when considering the three groups of Brazilian children. Each group grows in highly contrasted economical and social circumstances within the same national and cultural borders. A group of children lived in the poor and insecure environment of a favela in Rio de Janeiro, an environment dominated by young drug lords that terrorize and dictate law and order. Another group was composed of privileged children, of the same age, from an affluent private preschool situated just a few miles away from the favela. The third group of Brazilian children was composed of 3- and 5-year-old unschooled street kids from the city of Recife, a few hundred miles northeast of Rio. These children spent their days unsupervised by adults, begging on the street, collecting refuse, and typically spending the night with an extended family living in precarious, unsanitary slums close to public dumps.

One could easily presume that the drive to own, and not to share, in the young children of the favela, and particularly the street kids of Recife, might be different compared with the privileged children of Rio. Our research shows that it is not the case. All of these children demonstrate the same developmental trend toward a significant decrease in selfishness and increase in more equitable sharing between 3 and 5 years.

In typical development, young children become more equitable in their sharing, regardless of their economical and cultural circumstances because they enter the culture of their species (*Homo Negotiatus*), a culture that is fundamentally based on reciprocal exchanges. Hoarding and coercion are antithetical to this culture. If it exists, it is an anomaly, due to particularly stressful circumstances (war, disaster, rebellion, madness). It is not cardinal to the culture of *Homo Negotiatus*, unlike any other animal species that are not designed to have others in mind in their social exchanges and their sharing of resources.

We construct equity as well as agree on values by an active process of approximation and mutual monitoring. This process takes form within reciprocal exchanges. We do so by negotiation and ultimately by caring about reputation, namely our relative proximity with others. What happens between 3 and 5 years, is a marked progress in this process that channels children away from greed and immediate gratification. The product of this development is the emergence of a moral space in which children begin to care about reputation.

Children between 3 and 5 develop an understanding that they are potentially liable and that they build a history of transactions with others. Needless to say, parents and educators foster this development in all cultures, but this fostering is essentially the enforcement of the basic rules of reciprocity, the constitutive elements of human exchanges. Children are channeled to adapt to these rules they depend on to maintain proximity with others. From this, they begin to build a moral space in relation to others, a moral space that is essentially based on the basic rules of reciprocity. It is a moral space that is constantly in the

making, constantly revised, and in which equity is endlessly approximated by way of negotiation. Philosopher Charles Taylor (1989) notes in his book on the *Sources of the Self*: "What we are constantly losing from sight is that being a self is inseparable from existing in a space of moral issues, to do with identity and how one ought to be. It is being able to find one's standpoint in this space, being able to occupy a perspective in it" (p. 112). Consciousness and conscientiousness are indeed inseparable in typical development.

CONCLUSIONS: IMPLICATIONS FOR DEVELOPMENTAL PSYCHOPATHOLOGY

In this chapter and based on current progress in infancy research, we considered what seems to constitute the roots of typical consciousness. A large body of recent works indicate that healthy infants are born equipped to represent categorical features of the environment that are relevant for their survival such as faces (i.e., face-like displays); food (i.e., associated with particular taste or smell); but also less pragmatically, things that are of the same (i.e., selective process of sameness detection). Infants are born in a world of values. These values are represented in terms of specific attractor forces. There is a predetermined approach–avoidance polarity that channels the infant's attention and give direction to perception and action from the outset. As we have seen, newborns do not just sense the world but perceive it in terms of what it affords in for action: food, contact, novel vs. familiar experience, what should be avoided or approached. More than reflex machines limited in experiencing automatically proximal stimulations, research shows that they are perceivers of distal objects. As objective (distal) perceivers of the world, they are born implicitly aware of themselves as differentiated entities among other entities. Contrary to what was assumed by pioneer developmental psychologists like Piaget or Freud, newborns are now shown to have minimal self-awareness, not born is an initial state of fusion with the environment. In fact, one could argue that much of developmental psychopathologies and their deleterious effects affect what appears to be a given starting state of human development. Some aspects of this starting state, topic of this chapter, can have particularly deleterious consequences with the onset of neurodevelopmental disorders like schizophrenia or autism. Such instances can be viewed as the *undoing* of the typical propensity displayed very early on to engage in mutual recognition, experience self-unity as well to have a deep sense of possessing (i.e., controlling) one's own experience.

As illustration, and to conclude, we review next some of these deleterious undoing of typical consciousness. The intent is to emphasize aspects that are naturally a given from a very young age and too often taken for granted by the healthy individual. The importance of such aspects is clearly revealed in instances autism and schizophrenia where foundations of typical consciousness are undermined.

Centrality of Mutual Recognition

Kanner (1943), in his description of infantile autism, noted that these children appear to have "an innate inability to form the usual biologically provided affective contact with people, just as other children come into the world with innate physical and intellectual handicaps" (p. 23). Kanner went on to insist on what he sees as the extreme autistic aloneness of these children, their social isolation. Interestingly, for novice yet well-intended healthy adults who might try to engage a child diagnosed with autism, there is always a great deal of discomfort, frustration, and sense of being thwarted, of becoming unsettled and unsure of themselves (Greenspan & Wieder, 2006; Sigman & Capps, 1997). These children are difficult to figure out, removed, unpredictable, unreachable. Looking through or beside you, they behave as if you were transparent, *invisible*, nonexistent, *nonconsequential,* an experience that is a typical source of great discomfort for the well-intended parent or caretaker, and presumably a permanent discomfort for the autistic child withdrawn into his world.

The symptomatic trademark of autistic children is the depleted sociality experience by anybody trying to engage them and connect with them. The social current and cocreation of meanings that normally arise among communicating individuals are either hindered or plainly absent. It takes a great deal of expertise and exercises from parents, educators and therapists to contact these children, a difficult and courageous enterprise that requires sometimes infinite patience (e.g., Greenspan & Wieder, 2006).

What makes the raising of an autistic child so much more difficult and exhausting compared with raising a healthy, even hyperactive child is the fact that there is no room for mutual recognition, no room for reciprocal acknowledgment. The love parents of autistic children might express, often inexhaustibly, remains unmatched in its return. In this context, parents have difficulties recognizing themselves in the impact they have on their child. Inversely, the child is impaired in recognizing himself in what he does to his parents. Autism causes *mutual* blind mindedness, mutual invisibility, and it is a source of great

discomfort, obviously for the trying parents, but also for the disconnected child.

Given Sense of Self-Unity

At some basic level, feeling experience carries with it a fundamental sense of unity or *self-evidence*. It is that my feelings experiences of the world are mine, can only be mine, and nobody else. This is true no matter what, and to the extent that one doesn't take drugs or shows symptoms of schizophrenic autism or any other kinds of psychosis.

As continental phenomenological philosophers in the tradition of Husserl, Merleau-Ponty, Sartre, and many others but more recently Gallagher and Zahavi (2009) would claim, this self-evidence comes for free with any kinds of feeling experience. It is an implicit given. In the same way, James Gibson (1979) in his ecological approach to perception proposes that perceiving the world always necessarily entails *coperceiving* oneself as perceiver and actor in the world. One implies the other, no matter what, like the two sides of the same coin. This is a simple yet powerful intuition that is theoretically helpful, accounts for much of what we see in the development of children and in devastating instances of psychopathology like schizophrenia. For these empirical reasons, there are good reasons to endorse such implicit self-evidence that feeling would necessarily entail. However, one should acknowledge other philosophical and psychological perspectives adopting radically different epistemological starting points. These views are that the mine-ness of experience is more likely an illusion and a cognitive reconstruction rather than a necessary given. Accordingly, self-perception would be more the product of an inference and a mental representation rather than an implicit direct counterpart of any acts or direct perception, as Gibson suggested. Cognitive reconstruction views, however, appear untenable when considering very young infants as well as adults suffering mental and other self-disorders found particularly in schizophrenia but also other psychotic symptoms.

Research show that we are born with the basic intuition of what belongs to our own feeling experience and what is not. Few hour-olds infants do show unambiguous discrimination between self- and non–self-touch. For example, newborns root significantly more in the direction of another person's finger that touches one of their cheeks compared to when one of their own finger of hand comes spontaneously in contact to the same cheek. They discriminate between self-produced double touch of simultaneous cheek and finger touching and the single touch of a non–self-finger (Rochat & Hespos, 1997). This simple observation tells us, among other empirical evidence found in neonates that we are born with the potential for the implicit sense of exclusive mine-ness of experience, something that is probably the necessary phenomenal prerequisite of psychological development, its necessary subjective seed. That my feelings are mine is self-evident and does not have to be an indirect representational reconstruction. If that were the case, it is doubtful that neonates would show discrimination between double and single touch experience. Such empirical evidence would imply that there is from birth the potential self-evidence property of feeling experience.

Primordial Possession of the Own Experience

A look at the anomalous experience of psychotic adults who suffer from great cognitive disorganization and uncontrollable hallucinations within the schizophrenia spectrum, demonstrates how fundamental and actually basic self-evident mine-ness experience is, the sine qua non condition of our sanity. It shows clearly that self-evident *mine-ness* of feeling experience is the cornerstone of the ways we create meanings and bring coherence to our experience of being alive in this world: something that is ineffable (hard to put words on) and pre-reflective (implicit) by nature.

Loss of self-evident mine-ness of experience can be pathological and long lasting, as it can be temporary and provoked by drugs and other trance inducing rituals that are so pervasive across religions and cultures. Speaking tongues and other possessed states are symptomatic of self-evident feeling experience transmuted (mentally delegated) to a fictive entity that intrudes individual consciousness, be it the devil or a dead ancestor. Trance states can transform the implicit self-evidence typically accompanying feeling experience into the embodied *allo*-evidence of another. Possessed individuals are literally invaded and under the spell of another. There is experiential loss of self-control, until the individual snaps out of their trance state. Aside from religious rituals, hypnosis is a good, more secular example of trance-like state. It continues to be successfully exploited in psychotherapy and psychoanalysis, in the footsteps of Charcot (1825–1893) in his pioneer research on hysteria and other neurotic symptoms shown to be of psychological origin, as well as Freud in his early practice of psychoanalysis (Breuer & Freud, 1895/2000). Such induced temporary disturbance and loss of subjectivity is similar in some fundamental ways to chronic psychosis like schizophrenia.

Refined examination of the subjective, first person perspective accounts of hundreds of schizophrenic individuals reveals that first and foremost, schizophrenia and allied

symptoms are disorders of minimal self-experience including at its core, self-evident *mine-ness experience (*Parnas et al., 2005). It is typical for individuals suffering the atrocious ills of schizophrenia to report that although they rationally, thus explicitly know that their thoughts originate from within them and are not hallucinations proper, these thoughts are nonetheless *not* felt as generated by them. There is no implicit sense of self-agency in thinking or generating thoughts, they are thus somehow possessed rather than in possession as discussed above. It is a loss of thought *ipseity* (from the Latin word *ipse*, self), in other words the loss in the mine-ness of thinking one's own thoughts. Losing this basic and implicit sense of self is devastating, of almost unimaginable horror magnitude as probably best conveyed by Elyn Saks's (2007) vivid account of her own, very personal and detailed journey into schizophrenia:

> This experience is much harder, and weirder, to describe than extreme fear or terror. Most people know what it is like to be seriously afraid. If they haven't felt it themselves, they've at least seen a movie, or read a book, or talked to a frightened friend—they can at least imagine it. But explaining what I've come to call "disorganization" is a different challenge altogether. Consciousness gradually loses its coherence. One's center gives way. The center cannot hold. The "me" becomes a haze, and the solid center from which one experiences reality breaks up like a bad radio signal. There is no longer a sturdy vantage point from which to look out, take things in, assess what's happening. No core holds things together, providing the lens through which to see the world, to make judgments and comprehend risk. Random moments of time follow on another. Sights, sounds, thoughts, and feelings don't go together. No organizing principle takes successive moments in time and puts them together in a coherent way from which sense can be made. And it's all taking place in slow motion (p. 13)

Delusions and hallucinations are an intrinsic part of the diagnosis of schizophrenia and the self-disorganization, including voices as running commentaries on the person's behavior or thoughts, some sort of inescapable hyper-reflectivity but from a third-party perspective that invades the individual who lost basic self-evidence, the loss of an implicit trust that subjective experience *belongs* to oneself, a feeling experience that is not self-specifying anymore but rather diffused and confused with a world that is chaotic: the forceful, uncontrollable voices and intrusive thoughts that have no overarching narrative structure, no meaningful organization.

Such psychopathologies point to the fact that to function and experience the world normally, self-evidence and the basic feeling experience that one's thoughts and perceptions belong to oneself are a necessity—these are indispensible, absolute prerequisites to avoid mental invasion and the loss of minimal self-reference. How easy it is for us to take for granted the fact that these gestures, this voice, such vision *belong* to no one else and no other source than *me*. Without this basic self-evident feeling experience, we are at a loss. Losing it is indeed devastating: it is the primordial prerequisite of a healthy psychological development that starts in the womb.

REFERENCES

Ames, D. R., & Kammrath, L. K. (2004). Mind-reading and metacognition: Narcissism, not actual competence, predicts self-estimated ability. *Journal of Nonverbal Behavior*, *28*(3), 187–209.

Amsterdam, B. (1972). Mirror self-image reactions before age two. *Developmental Psychobiology*, *5*, 297–305.

Amsterdam, B. K. (1968). *Mirror behavior in children under two years of age*. Doctoral dissertation, University of Michigan. Order No. 6901569; University Microfilms, Ann Arbor, MI 48106.

Amsterdam, B. K., & Levitt, M. (1980). Consciousness of self and painful self-consciousness. *Psychoanalytic Study of the Child*, *35*, 67–83.

Anand, K. J. S., & Hickey, P. R. *(*1987). Pain and its effects in the human neonate and fetus. *New England Journal of Medicine*, *317*, 1321–1329.

Augustine. (2007). *Confessions, 1.6.10*. New York, NY: Barnes and Noble Classics. (Original work published 398 AD.)

Bahrick, L., Moss, L., & Fadil, C. (1996). Development of visual self-recognition in infancy. *Ecological Psychology*, *8*(3), 189–208.

Baillargeon, R. (2004). Infants' physical world. *Current Directions in Psychological Science*, *13*, 89–94.

Banks, M. S., & Shannon, E. S. (1993). Spatial and chromatic visual efficiency in human neonates. In C. E. Granrud (Ed.), *Carnegie-Mellon Symposium on Cognitive Psychology* (pp. 1–46). Hillsdale, NJ: Erlbaum.

Baron-Cohen, S. (1995). *Mindblindness: an essay on autism and theory of mind*. Boston, MA: MIT Press/Bradford Books.

Barr, G., Paredes, W., Erikson, K. L., & Zukin, S. R. (1986). Opioid receptor-mediated analgesia in the developing rat. *Developmental Brain Research*, *29*, 145–152.

Barsalou, L. W. (2008). Grounded cognition. *Annual Review of Psychology*, *59*, 617–645.

Bates, E. (1990). Language about me and you: Pronominal reference and the emerging concept of self. In D. Cicchetti & M. Beeghly (Eds.). *The self in transition: Infancy to childhood* (pp. 165–182). Chicago, IL: University of Chicago Press.

Bauer, P. J. (1996). Recalling past events: From infancy to early childhood. *Annals of Child Development*, *11*, 25–71.

Bauer, P. (2006). *Remembering times of our lives: Memory in infancy and beyond*. Mahwah, NJ: Lawrence Erlbaum Publishers.

Beer, J. S., & Hughes, B. L. (2010). Neural systems of social comparison and the "above average" effect. *Neuroimage*, *49*, 2671–2679.

Bertenthal, B., & Fisher, K. (1978). Development of self-recognition in the infant. *Developmental Psychology*, *14*, 44–50.

Bigelow, A. E., & Rochat, P. (2006). Two-month-old infants' sensitivity to social contingency in mother–infant and stranger–infant interaction. *Infancy*, *9*(3), 313–325.

Blass, E., Fitzgerald, E., & Kehoe, P. (1987). Interaction between sucrose, pain and isolation distress. *Pharmacology, Biochemistry and Behavior*, *26*, 483–489.

Block, N. (2007). Consciousness, accessibility, and the mesh between psychology and neuroscience. *Brain and Behavioral Sciences*, *30*, 481–548.

Breuer, J., & Freud, S. (2000). *Studies on hysteria*. New York: Basic Books. (Original publication in German 1895.)

Broesch, T., Callaghan, T., Henrich, J., & Rochat, P. (2011). Cultural variations in children's mirror self-recognition. *Journal of Cross-Cultural Psychology*, *42*(6) 1018–1029.

Brook, A. (1994). *Kant and the mind*. Cambridge, UK: Cambridge University Press.

Bruner, J. (1972). Nature and uses of immaturity. *American Psychologist*, *27*(8), 687–708.

Bruner, J. S. (1983). *Child's talk*. New York, NY: Norton.

Brusseau, R. R., & Mashour, G. A. (2007). Subcortical consciousness: Implications for fetal anesthesia and analgesia. *Behavioral and Brain Sciences*, *30*(1), 86–87.

Bunge, S. A., & Zelazo, P. D. (2006). A brain-based account of the development of rule use in childhood. *Current Directions in Psychological Science*, *15*, 118–121.

Callaghan, T., Rochat, P., Lillard, A., Claux, M. L., Odden, H., Itakura, S., Tapanya, S., & Singh, S. (2005). Synchrony in the onset of mental-state reasoning: Evidence from five cultures. *Psychological Science*, *16*(5), 378–384.

Campos, J., & Sternberg, C. (1981). Perception, appraisal, and emotions: The onset of social referencing. In M. Lamb & L. Shjerrod (Eds.), Infant social cognition: Empirical and theoretical considerations (pp. 273–314). Hillsdale, NJ: Lawrence Erlbaum Publishers.

Carey, S. (2010). *The origin of concepts*. New York, NY: Oxford University Press.

Carey, S., & Gelman, R. (Eds.). (1991). *The epigenesis of mind: Essays on biology and cognition*. Hillsdale, NJ: Erlbaum Associates.

Carlson, B. M. (1994). *Human embryology and developmental biology*. St. Louis, MO: Mosby.

Carpenter, E. (1976). The tribal terror of self-awareness. In P. Hockings (Ed.), *Principles of visual anthropology*. Berlin, Germany: Walter de Gruyter GmbH & Co.

Clifton, R. K., Rochat, P., Litovsky, R., & Perris, E. E. (1991). Representation guides infant reaching in the dark. *Journal of Experimental Psychology: Human Perception and Performance*, *17*(2), 323–329.

Cooley, C. H. (1902). *Human nature and the social order*. New York, NY: Charles Scribner's Sons.

Cummings, M. E., Davies, P. T., & Campbell, S. B. (2000). *Developmental psychopathology and family process: Theory, research, and clinical implications*. New York, NY: Guilford Press.

Damassio, A. (1999). *The feeling of what happens: Body and emotion in the making of consciousness*. New York, NY: Harcourt Brace & Company.

Damassio, A. R. (1995). *Descartes' error: Emotion, reason and the human brain*. New York, NY: Avon Publishers.

Damon, W. (1994). Fair distribution and sharing: The development of positive justice.n B. Puka (Ed.), *Fundamental research in moral development* (pp. 189–254). New York, NY: Garland Publishing.

Darwin, C. (1965). *The expression of the emotions in man and animals*. Chicago, IL: Chicago University Press. (Original publication 1872.)

DeCasper, A. J., & Fifer, W. P. (1980). Of human bonding: Newborns prefer their mother's voices. *Science*, *208*, 1174–1176.

Dennett, D. C. (1981). *Brainstorms: Philosophical essays on mind and psychology*. Cambridge, MA: MIT Press Bradford Books Series.

Dennett, D. C. (1987). *The intentional stance*. Cambridge, MA: MIT Press.

Dennett, D. C. (1992). The self as the center of narrative gravity. In F. S. Kessel, P. M. Cole, & D. L. Johnson (Eds.), *Self and consciousness: Multiple perspectives* (pp. 103–115). Mahwah, NJ: Lawrence Erlbaum Publishers.

Edelman, G. M., & Tononi, G. (2000). *A universe of consciousness: How matter becomes imagination*. New York: Basic Books.

Fodor, J. A. (1983). *Modularity of mind: An essay on faculty psychology*. Cambridge, MA: MIT Press.

Freud, S. (2000). *Three essays on the theory of sexuality*. New York, NY: Basic Books Classics series. (Original publication 1905.)

Gallagher, S., & Zahavi, D. (2008). *The phenomenological mind: An introduction to philosophy of mind and cognitive science*. New York, NY: Routledge.

Gallese, V. (2007). Before and below "theory of mind": Embodied simulation and the neural correlates of social cognition. *Philosophical Transactions of the Royal Society, Biological Sciences*, *362*(1480), 659–669.

Gallup, G. (1970). Chimpanzees: Self recognition. *Science*, *167*(3914), 86–87.

Gazzaniga, M., Mangun, G., & Ivry, R. (1998). *Cognitive neuroscience: The biology of the mind*. New York, NY: Norton.

Gelman, S. A. (2003). *The essential child: Origins of essentialism in everyday thought*. New York, NY: Oxford University Press.

Gelman, S. A., & Bloom, P. (2000). Young children are sensitive to how an object was created when deciding what to name it. *Cognition*, *76*, 91–103.

Greenspan, S., & Wieder, S. (2006). *Engaging autism*. Cambridge, Philadelphia, PA: Da Capo Press.

Gentner, D., Holyoak, K. J., & Kokinov, B. (Eds.). (2001). *The analogical mind: Perspectives from cognitive science*. Cambridge, MA: MIT Press.

Gergely. G.., & Watson. J. S. (1999). Early socio-emotional development; Contingency perception and the social-biofeedback model. In P. Rochat (Ed.), *Early social cognition: Understanding others in the first months of life* (pp. 101–136). Mahwah, NJ: Lawrence Erlbaum Associates. Inc.

Gibson, J. J. (1979). *Ecological approach to visual perception*. New York, NY: Houghton-Mifflin.

Gottlieb, G. (1971). Ontogenesis of sensory functions in birds and mammals. In A. Tobach, L. R. Aronson, & E. Shaw (Eds.), *The biopsychology of development* (pp. 67–128). New York, NY: Academic Press.

Gould, S. J. (1977). *Ontogeny and phylogeny*. Cambridge, MA: Harvard University Press.

Granrud, C. E. (1987). Size constancy in newborn human infants. *Investigative Ophtamology and Visual Science*, *28*(Supplement), 5.

Hamlin, J. K., Wynn, K., & Bloom, P. (2007). Social evaluation by preverbal infants. *Nature*, *450*, 557–559.

Haith, M. (1980). *The rules that babies look by*. Mahwah, NJ: Lawrence Erlbaum Publisher.

Haith, M. M. (1998).Who put the cog in infant cognition: Is rich interpretation too costly? *Infant Behavior and Development*, *21*, 167–179.

Haith, M. M., Bergman, T., & Moore, M. J. (1977). Eye contact and face scanning in early infancy. *Science*, *198*, 853–855.

Haith, M. M., Wass, T. S., & Adler, S. A. (1997). Infant visual expectations: Advances and issues. *Monographs of the Society for Research in Child Development*, *62*(2), 150–160.

Harris, P. (1991). The work of the imagination. In A. Whiten (Ed.), *Natural theories of mind* (pp. 283–304). Oxford, UK: Blackwell.

Hata, T., Dai, S. Y., & Marumo, G. (2010). Ultrasound for evaluation of fetal neurobehavioural development: From 2-D to 4-D ultrasound. *Infant and Child 529 Development*, *19*(1), 99–118.

Hebb, D. O. (1949). *Organization of behavior*. New York, NY: John Wiley & Sons.

Hepper, P. G. (2002). Prenatal development. In A. Slater & M. Lewis, *Introduction to infant development*. 42–56. New York, NY: Oxford University Press.

Hickok (2014). *The myth of mirror neurons*.

Hobson, R. P. (2002). *The cradle of thought*. New York, NY: Macmillan.

Hoorens, V. (1993). Self-enhancement and superiority biases in social comparison. *European Review of Social Psychology*, *4*(1), 113–139.

Hubbard, E. M., Arman, A. C., Ramachandran, V. S., & Boynton, G. M. (2005). Individual differences among grapheme–color synesthetes: Brain–behavior correlations. *Neuron*, *45*, 975–985.

Huttenlocher, P. R., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *Journal of Comparative Neurology*, *387*(2), 167–178.

James, W. (1950). *The principles of psychology*. New York, NY: Dover Publishers. (Original publication 1890.)

Johnson, S. C., Dweck, C., & Chen, F. S. (2007). Evidence for infants' internal working model of attachment. *Psychological Science*, *18*(6), 501–502.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, *2*, 217–250.

Kellman, P. J., & Arterberry, M. E. (2006). Infant visual perception. In D. Kuhn, R. S. Siegler, W. Damon, & R. M. Lerner (Eds.), *Handbook of child psychology: Vol 2, Cognition, perception, and language* (6th ed., pp. 109–160). Hoboken, NJ: Wiley.

Kellman, P. J., Spelke, E. S., & Short, K. R. (1986). Infant perception of object unity from translatory motion in depth and vertical translation. *Child Development*, *57*(1), 72–86.

Kessen, W. (1965). *The child*. New York, NY: Wiley.

Kinzler, K. D., & Spelke, E. S. (2007). Core systems in human cognition. In C. von Hofsten & K. Rosander (Eds.), *Progress in brain research* (Vol. 164, Chapter 14). North Holland: Elsevier B.V. pp. 257–264.

Kretschmann, H. J., Kammradt, G., Krauthausen, I., Sauer, B., & Wingert, F. (1986). Brain growth in man. Bibliotheca anatomica, *28*, 1–26.

Lecanuet, J. P., & Schaal, B. (1996). Fetal sensory competencies. *European Journal of Obstetrics & Gynecology and Reproductive Biology*, *68*, 1–23.

Lepage, J. P., & Théoret, H. (2007). The mirror neuron system: Grasping others' actions from birth? *Developmental Science*, *10*(5), 513–529.

Leslie A. M. (1982). The perception of causality in infants. *Perception*, *11*(2), 173–186.

Lewis, M. (1992). *Shame: the exposed self* . New York, NY: Free Press.

Lewkowicz, D. J., & Turkewitz, G. (1980). Cross-modal equivalence in early infancy: Auditory–visual intensity matching. *Developmental Psychology*, *16*, 597–607.

Lourenco, S. F., & Longo, M. R. (2011). Origins and the development of generalized magnitude representation. In S. Dehaene and E. Brannon (Eds.), *Space, Time, and Number in the Brain: Searching for the Foundations of Mathematical Thought* (pp. 225–244).

Mandler, J. M. (1988). How to build a baby: On the development of an accessible representational system. *Cognitive Development*, *3*, 113–136.

Mandler, J. M. (1992). How to build a baby II: Conceptual primitives. *Psychological Review*, *99*, 587–604.

Mandler, J. M. (1994). From perception to conception. In P. van Geert, L. P. Mos, & W. J. Baker (Eds.), *Annals of theoretical psychology* (Vol. 10, pp. 43–57). New York, NY: Plenum Press.

Mandler, J. M. (2004). *The foundations of mind: The origins of conceptual thought*. New York, NY: Oxford University Press.

Marlier, L., Schaal, B., & Soussignan, R. (1998a). Neonatal responsiveness to the odor of amniotic and lacteal fluids: A test of perinatal chemosensory continuity. *Child Development*, *69*(3), 611–623.

Marlier, L., Schaal, B., & Soussignan, R. (1998b). Bottle-fed neonates prefer an odor experienced in utero to an odor experienced postnatally in the feeding context. *Developmental Psychobiology*, *33*, 133–145.

Meltzoff, A. N. (1995). Understanding the intentions of others: Re-enactment of intended acts by eighteen-month-old children. *Developmental Psychology*, *31*(5), 838–850.

Meltzoff, A. N., & Borton, R. W. (1979). Intermodal matching by human neonates. *Nature*, *282*, 403–404.

Meltzoff, A. N., & Moore, M. K. (1977). Imitation of facial and manual gestures. *Science*, *198*(4312), 75–78.

Meltzoff, A. N., & Moore, M. K. (1997). Explaining facial imitation: A theoretical model. *Early Development and Parenting*, *6*, 179–192.

Merskey, H., & Bogduk, N. (Eds.). (1994). *Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms*, 2nd Edition. New York, NY: IASP Press.

Mitchell, R. W. (1993). Mental models of mirror-self-recognition: Two theories. *New Ideas in Psychology*, *11*(3), 295–325.

Mondloch, C., & Maurer, D. (2004). Do small white balls squeak? Pitch–object correspondences in young children. *Cognitive, Affective, and Behavioral Neuroscience*, *4*, 133–136.

Montagu, A. (1961). Neonatal and infant immaturity in man. *Journal of the American Medical Association*, *178*(23), 56–57.

Morton, J., & Johnson, M. H. (1991). CONSPEC and CONLERN: A two-process theory of infant face recognition. *Psychological Review*, *98*(2), 164–181.

Murray, L., & Trevarthen, C. (1985). Emotional regulation of interactions between two-months-old and their mothers. In T. M. Field & N. A. Fox (Eds.), *Social perception in infants* (pp. 177–197). Norwood, NJ: Ablex.

Nadel, J., Carchon, I., Kervella, C., Marcelli, D., & Réserbat-Plantey, D. (1999). Expectancies for social contingency in 2-month-olds. *Developmental Science*, *2*(2), 164–173.

Nelson, K., & Fivush, R. (2004). The emergence of autobiographical memory: A social cultural developmental theory. *Psychological Review*, *111*(2), 486–511.

Neuman, C. J., & Hill, S. D. (1978). Self-recognition and stimulus preference in autistic children. *Developmental Psychobiology*, *11*, 571–578.

Neville, H. (1995). Developmental specificity in neurocognitive development in humans. In M. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 219–231). Cambridge, MA: Bradford.

Onishi, K. H., & Baillargeon, R. (2005). Do 15-month-old infants understand false beliefs? *Science*, *8*, 255–258.

Oppenheim, R. W. (1991). Cell death during development of the nervous system. *Annual Review of Neuroscience*, *14*, 453–501.

Oyama, S. (2000). *The ontogeny of information: Developmental systems and evolution* (2nd ed.). Durham, NC: Duke University Press.

Parker, S. T., Mitchell, R. W., & Boccia, M. L. (1994). *Self-awareness in animals and humans*. Cambridge, UK: Cambridge University Press.

Parnas, J.,Moller, P., Kircher, T. Thalbitzer, J. Jansson, L., Handset, P. & Zahavi, D., … et al. (2005) EASE: Examination of Anomalous Self-Experience. *Psychopathology*, *38*, 236–258.

Piaget, J. (1952). *The origins of intelligence in children*. New York, NY: International Universities Press. (Original publication 1936.)

Piaget, J. (1971). *Biology and knowledge*. Chicago, IL: University of Chicago Press. (Original publication 1967.)

Pinker, S. (2002). *The blank slate: The modern denial of human nature*. New York, NY: Penguin Books.

Plotnik, J., & de Waal, F. B. M. (2006). Self-recognition in an Asian elephant. *Proceedings of the National Academy of Sciences USA*, *103*(45), 17053–17057.

Powell, L. J., & Spelke, E. S. (2013). Preverbal infants expect members of social groups to act alike. *Proceedings of the National Academy of Sciences*, *110*, E3965–E3972.

Prechtl, H. F. R. (Ed.). (1984). *Continuity of neural functions: From prenatal to postnatal life*. Oxford, UK: Blackwell Scientific Publications.

Prior, H., Schwarz, A., & Güntürkün, O. (2008). Mirror-induced behavior in the magpie (Pica pica): Evidence of self-recognition. *PLoS Biol*, *6*(8): e202. doi: 10.1371/journal.pbio.0060202.

Rakic, P. (1972). Model of cell migration to the superficial layers of the fetal monkey neocortex. *Journal of Comparative Neurology*, *145*, 61–84.

Rakison, D. H., & Poulin-Dubois, D. (2001). The developmental origin of the animate-inanimate distinction. *Psychological Bulletin*, *2*, 209–228.

Rizzolatti, G., Fadiga, L., Gallese, V., & Fogassi, L. (1996). Premotor cortex and the recognition of motor actions. *Brain Research, Cognitive Brain Research*, *3*(2), 131–141.

Rochat, P. (1983). Oral touch in young infants: Response to variations of nipple characteristics in the first months of life. *International Journal of Behavioral Development*, *6*, 123–133.

Rochat, P. (1987). Mouthing and grasping in neonates: Evidence for the early detection of what hard or soft substance afford for action. *Infant Behavior and Development*, *10*, 435–449.

Rochat, P. (2001). *The infant's world*. Cambridge, MA: Harvard University Press.

Rochat, P. (2002). Origins of self concept. In G. Bremner & A. Fogel (Eds.), *Blackwell handbook of infant development* (pp. 191–212). Malden, MA: Blackwell Publishers.

Rochat, P. (2009). *Others in mind—Social origins of self-consciousness*. New York, NY: Cambridge University Press.

Rochat, P. (2014). *Origins of possession: Owning and sharing in development*. Cambridge, UK: Cambridge University Press.

Rochat, P. (Ed.). (1995). *The self in infancy: Theory and research advances in psychology* (Series No. 112). Amsterdam, The Netherlands: North Holland, Elsevier Science Publishers.

Rochat, P., Dias, M. D. G., Guo, L., Broesch, T., Passos-Ferreira, C., & Winning, A. (2009). Fairness in distributive justice by 3- and 5-year-olds across seven cultures. *Journal of Cross-Cultural Psychology*, *40*, 327–348.

Rochat, P., & Hespos, S. J. (1997). Differential rooting response by neonates: Evidence of an early sense of self. *Early Development and Parenting*, *6*(3–4), 105–112.

Rochat, P., Neisser, U., & Marian, V. (1998). Are young infants sensitive to interpersonal contingency? *Infant Behavior & Development*, *21*(2), 355–366.

Rochat, P., & Passos-Ferreira, C. (2008). *Homo negotiatus*: Ontogeny of the unique ways humans own and share. In S. Itakura & I. Fujita (Eds.), *Origins of the social mind: Evolutionary and developmental views* (pp. 141–156). New York, NY; Berlin: Springer Publishers.

Rochat, P., & Senders, S. J. (1991). Active touch in infancy: Action systems in development. In M. J. Weiss & P. R. Zelazo (Eds.), *Infant attention: Biological constraints and the influence of experience* (pp. 412–442). Mahaw, NJ: Ablex Publishers.

Rochat, P., & Striano, T. (1999). Social cognitive development in the first year. In P. Rochat (Ed.) *Early Social Cognition*, Lawrence Erlbaum Associates, Mahaw, NJ: pp. 3–34.

Rochat, P. & Striano, T. (2002). Who is in the mirror: Self-other discrimination in specular images by 4- and 9-month-old infants. *Child Development*, 73, 35–46.

Rochat, P., Striano, T., & Blatt, L. (2002). Differential effects of happy, neutral, and sad still faces on 2-, 4-, and 6-month-old infants. *Infant and Child Development*, *11*(4), 289–303.

Rochat, P., Striano, T., & Morgan, R. (2004). Who is doing what to whom? Young infants' developing sense of social causality in animated displays. *Perception*, *33*(3), 355–369.

Rochat, P., & Zahavi, D. (2011). The uncanny mirror: A re-framing of mirror self-experience. *Cognition and Consciousness*, *20*, 204–213.

Rovee-Collier, C., & Hayne, H. (2000). Memory in infancy and early childhood. In E. Tulving & F. I. M. Craik (Eds.), *The Oxford handbook of memory* (pp. 267–282). New York, NY: Oxford University Press.

Saffran, J., Aslin, R., & Newport, E. (1996). Statistical learning by 8-month-old infants. *Science*, *274*(5294), 1926–1928.

Sagi, A., & Hoffman, M. L. (1976). Empathic distress in newborns. *Developmental Psychology*, *12*, 175–176.

Saks, E. (2007). *The center cannot hold: My journey through madness*. New York, NY: Hyperion.

Sartre, J.-P. (1971/1981). *The family idiot: Gustave Flaubert, 1821–1857* (Vol. *1*). (Trans. Carol Cosman). Chicago, IL: University of Chicago Press.

Sherrington C. S. (1906). *The integrative action of the nervous system*. New Haven, CT: Yale University Press.

Sigman, M., & Capps, L. (1997). Children with autism—A developmental perspective. Cambridge. MA: Harvard University Press.

Simner, M. L. (1971). Newborn's response to the cry of another infant. *Developmental Psychology*, *5*, 136–150.

Simner, J., Sagiv, N., Mulvenna, C., Tsakanikos, E., Witherby, S., Fraser, C., Scott, K. & Ward, J. (2006). Synesthesia: The prevalence of atypical cross-modal experiences. *Perception*, *35*, 1024–1033.

Smith, S. (1996). Commission of inquiry into fetal sentience. *CARE*.

Spector F., & Maurer D. (2009). Synesthesia: a new approach to understanding the development of perception. *Developmental Psychology*, *45*(1), 175–189.

Spelke, E. S., Breinlinger, K., Macomber, J., & Jacobson, K. (1992). Origins of knowledge. *Psychological Review*, *99*, 605–632.

Sroufe, L. A. (1996). *Emotional development: The organization of emotional life in the early years*. New York, NY: Cambridge University Press.

Steiner, J. E. (1979). Human facial expressions in response to taste and smell stimulation. *Advances in Child Development and Behavior*, *13*, 257–296.

Stern, D. (1991). *The diary of a baby: what your child sees, feels, and experiences*. London, UK: Fontana Press.

Striano, T., & Rochat, P. (1999). Developmental link between dyadic and triadic social competence in infancy. *British Journal of Developmental Psychology*, *17*(4), 551–562.

Striano, T., & Rochat, P. (2000). Emergence of selective social referencing. *Infancy*, *1*(2), 253–264.

Striano, T., Tomasello, M., & Rochat, P. (2001) Social and object support for early symbolic play. *Developmental Science*, *4*(4), 442–455.

Taylor, C. (1989). *Sources of the Self: the making of modern identity*. Cambridge, MA: Harvard University Press.

Thelen, E., & Smith, L. (1996). *A dynamic systems approach to the development of cognition and action*. Cambridge, MA: Bradford/M.IT Press.

Thomsen, L., Frankenhuis, W. E., Ingold-Smith, M., & Carey. S. (2011). Big & mighty: Preverbal infants mentally represent social dominance. *Science*, *331*, 477–480.

Tomasello, M. (1995). Joint attention as social cognition. In C. J. Moore & P. Dunham (Eds.), *Joint attention: Its origins and role in development* (pp. 103–130). Hillsdale, NJ: Lawrence Erlbaum Publishers.

Tomasello, M. (1998). One child early talk about possession. In J. Newman (Ed.), *The linguistic of giving*. 87–109. Amsterdam, The Netherlands: John Benjamins.

Tomasello, M. (1999). *Cultural origins of human cognition*. Cambridge, MA: Harvard University Press.

Tomasello, M., Kruger, A. C., & Ratner, H. H. (1993). Cultural learning. *Brain and Behavioral Sciences*, *16*, 495–552.

Tomasello, M., Striano, T., & Rochat, P. (1999). Do young children use objects as symbols? *British Journal of Developmental Psychology*, *17*(4), 563–584.

Trevarthen, C. (1979). Communication and cooperation in early infancy: A description of primary intersubjectivity. In M. M. Bullowa (Ed.), *Before speech: The beginning of interpersonal communication* (pp. 321–347). New York, NY: Cambridge University Press.

Trevarthen, C. (1980). The foundations of intersubjectivity: Developments of interpersonal and cooperative understanding in infants. In D. R. Olson (Ed.) *The Social foundations of language and thought: Essays in honor of Jerome S. Bruner*. 134–146. New York, NY: Norton.

Trevarthan, W. R. (1987). *Human birth: An evolutionary perspective*. Hawthorne, NY: Aldine de Gruyter.

Trevarthen, C., & Hubley, P. (1978). Secondary intersubjectivity: confidence, confiding and acts of meaning in the first year. In A. Lock (Ed.), *Action, gesture and symbol: The emergence of language* (pp. 183–229). London, UK: Academic Press.

Tronick, E., Als, H., Adamson, L. B., Wise, S., & Brazelton, T. B. (1978). The infant's response to entrapment between contradictory messages in face to face interaction. *Journal of the American Academy of Child Psychiatry*, *17*, 1–13.

Walsh, V. (2003). A theory of magnitude: Common cortical metrics of time, space and quantity. *Trends in Cognitive Sciences*, *7*, 483–488.

Watson, J. S. (1995). Self-orientation in early infancy: The general role of contingency and the specific case of reaching to the mouth. In P. Rochat (Ed.), *The self in infancy: Theory and research* (Advances in Psychology, Vol. *112*, pp. 375–394). Amsterdam, The Netherlands: North-Holland/Elsevier Science Publishers.

Wellman, H. M., & Gelman, S. A. (1992) Cognitive development: Foundational theories of core domains. *Annual Review of Psychology*, *43*, 337–375.

Wolff, P. H. (1987). *The development of behavioral states and the expression of emotions in early infancy*. Chicago, IL: University of Chicago Press.

Wolff, P., Matsumiya, Y., Abrohms, I. F., van Velzer, C., & Lombroso, C. T. (1974). The effect of white noise on the somatosensory evoked responses in sleeping newborn infants. *Electroencephalography and Clinical Neurophysiology*, *37*, 269–274.

Woodward, A. L. (2009). Infants' grasp of others' intentions. *Current Directions in Psychological Science*, *18*, 53–57.

Zahn-Waxler, C., Radke-Yarrow, M., Wagner, E., & Chapman, M. (1992). Development of concern for others. *Developmental Psychology*, *28*, 126–136.

Zelazo, P. D. (2004). The development of conscious control in childhood. *Trends in Cognitive Sciences*, *8*, 12–17.

Zelazo, P. D., Gao, H. H., & Todd, R. (2007). The development of consciousness. In P. Zelazo, M. Moscovitch, & E. Thomson (Eds.) *Cambridge handbook of consciousness*. New York, NY: Cambridge University Press.

CHAPTER 11

I-Self and Me-Self Processes Affecting Developmental Psychopathology and Mental Health

SUSAN HARTER

INTRODUCTION 471
Summary 473
How the I-Self Can Contribute to Mental Health and Well-Being 473
HOW THE I-SELF CONTRIBUTES POSITIVELY TO THE DEVELOPMENTAL CONSTRUCTION OF THE ME-SELF, INCLUDING JAMESIAN I-SELF FUNCTIONS 474
James's I-Self Functions: Self-Awareness, Self-Agency, Self-Continuity, and Self-Coherence 474
Very Early Childhood (Ages 2–4) 475
Early to Middle Childhood (Ages 5–7) 475
Middle-to-Late Childhood (Ages 8–10) 477
Interlude: Limitations of Piaget's Theory of Adolescent Cognitive Development, Applied to the Self 478
Early Adolescence (Ages 11–13) 478
Middle Adolescence (Ages 14–16) 479
Late Adolescence (Ages 17–19) 480
SELF-ENHANCEMENT AND SELF-SERVING BIASES: THE I-SELF/ME-SELF CONSPIRACY 481
Self-Enhancement Strategies During Very Early Childhood 481
Self-Enhancement During Early to Middle Childhood (Ages 5–7) 482
Self-Enhancement During Middle to Late Childhood (Ages 8–10) 483
Self-Enhancement During Early Adolescence (Ages 11–13) 484
Self-Enhancement During Middle Adolescence (Ages 14–16) 485
Self-Enhancement During Late Adolescence (Ages 17–19) 486
SOCIALIZATION PRACTICES THAT CAN COMPROMISE JAMESIAN I-SELF FUNCTIONS 488
CROSS-CULTURAL DIFFERENCES IN SELF-ENHANCEMENT 488
SELF-EVALUATIONS IN GLOBAL SELF-ESTEEM AND SELF-CONCEPT: I-SELF AND ME-SELF FUNCTIONS 489
WHY CAN'T THOSE WITH LOW SELF-ESTEEM ALTER THE PERCEPTIONS OF THEIR OVERALL LACK OF WORTH? 491
CROSS-CULTURAL DIFFERENCES IN SELF-ESTEEM 493
DEPRESSION AND SUICIDAL BEHAVIORS 494
Different Pathways to Depression 495
Depression as a Combination of Sadness and Anger 496
The Perceived Directionality of Low Self-Esteem and Depressed Affect 496
EATING-DISORDERED BEHAVIOR 496
THE ROLE OF SELF PROCESSES, HUMILIATION, AND VIOLENT REVENGE 498
The Role of Humiliation 499
Cyberbullying 502
SUMMARY, THUS FAR 503
The I-Self's Role in Promoting Mental Health Among our Children and Adolescents 503
Awareness 504
Sense of Agency 504
Self-Efficacy as an I-Self Process 504
Mastery Motivation 504
Intrinsic Versus Extrinsic Motivation 505
The Concept of Flow 506
Self-Continuity 506
The Impoverished Self 507
The I-Self Function of Self-Coherence 507
CROSS-CULTURAL DIFFERENCES IN SELF-COHERENCE 510
MINDFULNESS AS AN I-SELF PROCESS: A BUDDHIST PERSPECTIVE 511
FURTHER IMPLICATIONS FOR MENTAL HEALTH: THE ROLE OF THE POSITIVE PSYCHOLOGY MOVEMENT 512
The Role of Positive Emotions 512
Positive Affectivity 513
Emotional Intelligence 513

Emotional Competence 513
Emotional Creativity 514
POSITIVE PSYCHOLOGY'S EMPHASIS ON ISSUES RELATED TO PERCEPTIONS OF CONTROL: COGNITIVE PLUS EMOTIONAL COMPONENTS 516
Sense of Control 516
Self-Determination 516
Optimism 516
Hope 516
Curiosity 516
CONCLUSIONS 517
Coda: Putting the I-Self in Perspective 518
REFERENCES 518

INTRODUCTION

I was incredulous when a bright 2-year-old with whom I was once conversing looked at me quizzically and asked, with tremendous earnestness: "Am I me?" Was this budding young philosopher wrestling with a distinction that William James (1892) introduced over 120 years ago, namely, the I-self versus the me-self? I was tempted to think so. Of course at his age, he was more likely struggling to sort out the linguistic conventions governing the use of the pronouns I and me. Nevertheless, I found his query quite profound. It set the stage for examining how I-self and me-self processes change with age, from a developmental perspective.

James (1890, 1892) himself wrestled with the distinction between the I-self and the me-self in adults and never arrived at an explanation that was, to him, intellectually satisfying. He concluded that the I-self was an incorrigible construct, an elusive concept that defied clear explication. Many scholars to follow retained this distinction in their own theorizing, where the I-self has been afforded such characteristics as a sense of *agency*, as the *observer,* one's intellectual pulse and window on the world and as the cognitive engine that drives the creation of the me-self (see reviews in Harter, 1999, 2012). However, too many theorists have paid only lip service to the role of the I-self, failing to translate this concept into researchable or applied formulations that document its specific functions.

Moreover, the I-self has been overshadowed by the me-self, which took center stage in the self literature (see Harter, 2012; Wylie, 1979, 1989), particularly with regard to measurement issues. The *me-self* reflects one's image in the psychological mirror, the self as *observed* and personally described, namely, one's *concept* of self at both domain-specific levels (e.g., perceptions of one's competence, physical appearance, social acceptability) as well as more global self-esteem. Unfortunately, the me-self in the mirror became an entity over which the individual myopically preened, a self that demanded enhancement, promotion, and protection, often reflected in self-serving biases and self-deception (see Harter, 2012; Leary, 2004). As the self literature evolved, the I-self was increasingly given short shrift, particularly from a developmental perspective.

It is the intent of this chapter contribution to resurrect the I-self, to restore the I-self to its rightful place as a critical cognitive-developmental process, with implications for both normative and pathological development. Critical to this endeavor is the premise that we must adopt a neo-Piagetian perspective (see Fischer, 1980; Fischer & Bidell, 2006; Harter, 1999, 2012), charting how changing cognitive-developmental processes define the I-self. These processes, in turn, dictate how the content and structure of me-self evaluations will correspondingly change with age. However, it will be argued that we need to broaden and revitalize the I-self's job description, beyond that of the mere architect of the me-self, beyond efforts to package, protect, enhance, and distort the me-self. This is the dark side of the I-self's preoccupation that leads to pathological conditions. This job definition will be reviewed. However, momentarily fast-forwarding, I will also address how there are better ways for the I-self to spend its precious time that can contribute to mental health.

As a backdrop, I first review how developing I-self processes contribute to the construction of the me-self. I will address these processes across six levels of development, very early childhood, early to middle childhood, and late childhood as well as early adolescence, middle adolescence, and late adolescence. Those positive Jamesian (1892) I-self functions, *self-awareness*, a sense of *agency*, as well as the *continuity* and *coherence* of the self will be included in this developmental analysis. In first describing self-development across these six substages, the focus will be on (1) normative development as well as what will be labeled (2) normative-developmental *liabilities,* psychological speed bumps along the road that are typical for many children and adolescents but do *not* represent minefields that are signals of psychopathology. Typically, with appropriate support and good-enough child rearing, children and adolescents will healthily proceed, lurching toward normative development as emerging adults and beyond. However, not all are so blessed.

Research supported by grants to the author by NICHD and the W.T. Grant Foundation.

What might go awry? I will conceptualize psychopathology as various *deficiencies in me-self* development where anomalies and clinical symptoms are primarily manifest. Yet the I-self is a coconspirator, as we explore the dark side of the contribution of the I-self. One can ask whether this less than admirable preoccupation has been forced upon the I-self by societal and cultural demands as well as certain socialization practices, for example, less than optimal parental child-rearing practices, including various forms of abuse in the extreme.

Thus, next, the dark side of the I-self's job description will be discussed, namely, how from a Western perspective the I-self should be gainfully employed in protecting and enhancing the me-self, deceptively packaging it as a valued commodity in the psychological market place. For example, conditional support from parents—behave according to my agenda for you if you want my love and support—can lead to false self manifestations that compromise the more positive I-self functions (Harter, Marold, Whitesell, & Cobbs, 1996). Moreover, severe and chronic abuse can seriously interfere with other I-self roles such as self-awareness, a sense of agency, as well as the continuity and coherence of the I-self (see Harter, 1999).

I will then review how the dark side of self-development can also include how the I-self conspires to keep the me-self at the heart of those developmental psychopathologies that intimately involve self-evaluations. Five such pathological conditions that involve these self processes will be examined: (1) self-aggrandizement, self-serving biases, self-deception, and narcissism; (2) negative self-evaluations in the form of low self-esteem and poor self-concept; (3) depressive reactions, syndromes, and suicidal behaviors; (4) eating-disordered behaviors; and (5) the role of negative self-processes that can lead to violent revenge in the face of bullying.

1. Self-Aggrandizement, Self-Serving Biases, Self-Deception, and Narcissism

There is a contemporary belief that the self is cause for concern in our culture, among certain self-theorists (see Harter, 2012; Leary, 2004). Many adults in our society seem transfixed by the need for self-enhancement, self-serving biases, falsely inflating their self-esteem for personal gain. These manifestations on the psychological landscape shade into increasing *narcissism,* a psychological condition that takes on different manifestations at different developmental levels, as will be traced in this chapter. Similarly, the cognitive *skills* to engage in self-aggrandizement and the *motives* for so doing will differ across developmental levels, an issue that will be tracked across the six stages.

2. Negative Self-Evaluations in the Form of Low Self-Esteem and Poor Self-Concept

Normatively, these evolve in a predictable developmental sequence, but at each stage there is the potential for derailment. Thus, we will evaluate what factors can interfere with healthy or optimal self-esteem, leading to precariously low self-esteem. In addition, very negative self-evaluations are revealed in particular domain-specific self-concepts. We will deal with those socialization practices that interfere with optimal self-esteem development, including such parental practices as *conditional support*, which involves behavioral demands of the child if he/she wishes to insure parental love. However, children do not perceive conditional parental behavior to be genuine love. In addition, abuse by caretakers can interfere with both I-self and me-self processes, provoking negative self-evaluations (Harter et al., 1996).

3. Depressive Reactions, Syndromes, and Suicidal Behaviors

There has been considerable research (see reviews in Harter, 1999, 2012) revealing that depressive reactions are intimately related to negative self-concept and low self-esteem. In the most severe cases, these can lead to suicidal gestures, which for some, meet with tragic outcomes that result in the termination of one's life. Taking its toll is abuse by caregivers, insensitive responses by peers, and most recently bullying, including cyberabuse, which has become the new look in cruelty, destroying children's and adolescent's sense of worth. In extreme cases, victims are driven to engage in previously unthinkable acts of self-destruction. We will bring this literature up to date.

4. Eating-Disordered Behaviors

Although there are many different theories and approaches to this serious problem among our youth, much of the research reveals that self-processes are highly implicated. Negative perceptions of one's body image, relative to the punishing and impossible cultural standards that are touted and blasted on TV, movies, and social media, contribute. These seriously erode the bodily self-images of our children and adolescents, and not just for females. Males are now held to higher standard of physical attractiveness. Our own research, as well as that of others, reveals that these negative self-perceptions of appearance lead directly to low self-esteem or self-worth, which in turn result in eating-disordered behavior (see Harter, 2012). These reactions, in turn, can lead to active suicidal attempts or more passive behavior such as lack of intake in food that can stunt biological growth processes and in some cases result in death.

5. The Role of Negative Self-Processes Leading to Violent Revenge in the Face of Bullying

The previous decade has seen a flurry of activity and interest in bullying and prevention programs within the schools that are certainly welcome, to stem the tide of this type of abuse. Our own approach has been to view these phenomena as intimately related to the self and our own model of the causes and consequences of self-esteem identifies several various self and social processes (Harter, 1999, 2012). Clearly, the victims of abuse and harassment have displayed low self-esteem and related attributes that we have identified in our model (for example, inadequacy in domains valued by peers) that make them targets of harassment. But why, one might ask, has it been the *victims* that have emerged, in the 12 high-profile cases of school shootings, who are the perpetrators of such violence? Why did they, in particular, seek revenge? Our own scrutiny of the most widely publicized cases has revealed that in every case these White, middle-class, suburban, or rural boys had not only been harassed and physically abused but had been *publicly humiliated*. At a broader level, we have argued that there is not a simple, direct link between being bullied and revenge. Rather, it has to be mediated by an *emotional reaction*, which we have identified as *humiliation,* with correlates of anger, frustration, shame, and depression (Harter, 2012). However, humiliation seems to be the primary trigger that eventually evokes the rage of revenge. These, and other models that implicate the self, will be explored.

Summary

There are many contributions to psychopathological behaviors in children and adolescents, as this entire handbook will illuminate. This particular chapter will focus on those factors that involve the *self*, more specifically the interface of the I-self and the me-self, an approach that may be complementary to other perspectives. The field needs those that are adept at integrating such approaches, which hopefully this volume will do.

As a developmentalist, I will approach the topic by first introducing a framework in which I distinguish between Jamesian I-self processes and me-self processes. I argue that the self has not received its rightful due as an active construct or that too much of the self-literature has focused on the me-self, providing an unbalanced view of self processes. Thus, next I identify six stages of developmental, three within childhood and three within adolescence, since there are rapid changes within childhood and adolescence in both I-self and me-self processes; we can no longer treat childhood and adolescence as two broad, general stages. Both normative self-representations and potential normative liabilities will be highlighted. Finally, I will argue that certain forms of developmental psychopathology are largely manifest in me-self aberrations but that the I-self plays a role, dictated by societal and cultural values including conventions and expectations.

Thus, I first briefly trace how the I-self contributes to me-self development, normatively, including some predictable *liabilities* that are *not* pathological and usably abate under normal, child-rearing conditions. Unlike certain purely descriptive treatments of mere me-self changes, I indicate how changes in I-self development contribute to the manifestations of the me-self at different stages. Furthermore, unlike other treatments, I honor the role of the Jamesian specific I-self processes that he so brilliantly identified.

The chapter then turns to the dark side of the I-self's job description in having to package the me-self in a manner that would be acceptable to societal dictates about what the me-self should look like, behave like, how it should display itself, sometimes falsely, against the wishes of one's authentic self. This is most evident in the mandate to package the self, to falsify the self, to engage in self-aggrandizement that can lead to self-serving behaviors that border on and include narcissism.

Against this background, we visit the six stages of childhood and adolescence, applying this developmental thinking to five different areas of developmental psychopathology, which are manifest differently at each of the six stages. To review, these forms of developmental psychopathology that intimately involve the *self,* are listed as follows:

1. Self-aggrandizement, self-serving biases, and self-deception, that can evolve into narcissism
2. Negative self-evaluations in the form of low self-esteem and poor self-concepts
3. Depressive reactions, syndromes, and suicidal behaviors
4. Eating-disordered behaviors
5. The role of negative self-processes, including humiliation, which can lead to violent revenge in the face of bullying

How the I-Self Can Contribute to Mental Health and Well-Being

The chapter will end with an emphasis on how I-self processes can spearhead more mentally healthy preoccupations that temper some of the negative pathological effects of the me-self and can supplant such processes

in order to allow the I-self its rightful due as a positive force in development. We will enlarge and broaden upon this vision, casting the I-self in a more positive light, as a key player in promoting psychological health. Links to contemporary theorizing that provides an antidote to some of these disruptions in adaptive I-self functioning will be discussed. This new and more optimistic focus on human behavior suggests that we devote more energy to appreciating and fostering a number of such processes that can be cast in I-self/me-self parlance. These include self-awareness, self-efficacy, sense of agency, mastery motivation, the concept of flow, self-continuity and self-coherence, mindfulness, from a Buddhist perspective, which includes a focus on egolessness, the quiet ego, humility, and compassion. From a Buddhist perspective, far more fringe benefits will accrue if the I-self averts its myopic gaze away from protecting the me-self through a distorted lens of self-aggrandizement. Consistent with the premises of the field of positive psychology (see Lopez & Snyder, 2009; Snyder & Lopez, 2002) more beneficial outcomes will ensue from an exploration of the I-self's role in promoting *authenticity*, which, in today's Western world, unfortunately seems to be in short supply.

From this relatively new field of positive psychology (see Lopez & Snyder, 2009), other constructs will be selected that can be interpreted within an I-self/me-self perspective, highlighting the role of the I-self in promoting mental health and well-being. These include the role of self-acceptance, the role of positive emotions, positive affectivity, emotional intelligence, emotional competence, emotional creativity, cognitive creativity, sense of control, self-determination, optimism, hope, and curiosity.

HOW THE I-SELF CONTRIBUTES POSITIVELY TO THE DEVELOPMENTAL CONSTRUCTION OF THE ME-SELF, INCLUDING JAMESIAN I-SELF FUNCTIONS

Developmental approaches to the self have primarily focused on age-related changes in the *me-self*, although they have not been theoretically driven. For example, Montemayor and Eisen (1977) provided compelling examples of the changes in self-descriptions across three broad developmental periods: early childhood, later childhood, and adolescence. Young children's descriptions were very concrete, focusing on observable behaviors (e.g., I can run fast, I live in a big house); those in later childhood were more likely to include socially relevant traits or attributes (e.g., I am popular, I am nice), whereas adolescents were more likely to refer to more general, abstract personality characteristics (e.g., I am intelligent, I am moody) (see Damon & Hart, 1988; Harter, 1999, 2012; Rosenberg, 1979). Age-related differences in the me-self were primarily described as developmental changes in the *content* of self-representations.

The cognitive revolution within the field of psychology in the 1980s and 1990s brought with it the premise that the self was primarily a cognitive construction, namely, a *theory* about the self (see Epstein, 1991; Markus, 1980; Sarbin, 1980). These scholars addressed this contention for adults, forcefully arguing that our species has been designed to cognitively create *theories* about our world, including the construction of a *theory of self*, which makes meaning of our personal experiences.

With the advent of neo-Piagetian formulations of cognitive development (Case, 1991, 1992; Fischer, 1980; Fischer & Bidell, 2006), attention turned to how changing cognitive structures determined the very nature of the self theory that children and adolescents construct at different periods of development (see Harter, 1999). As cognitive processes undergo normative-developmental change, so will the *content* and very *structure* or organization of self undergo alterations. That is, the particular cognitive limitations and advances at each developmental period will dictate the emerging features of the self-portrait. I first suggested (Harter, 1999) a new conceptual framework, arguing that the influential cognitive-developmental processes be thought of as *I-self* processes. These, in turn, are central to corresponding changes in the content and structure of the *me-self*, namely, the resulting self-descriptions as the product, the image in the mirror, as it were. I have subsequently (Harter, 2012) expanded upon this conceptualization and will share some of that thinking here. I have distinguished between three levels of childhood (early, middle, and late) as well as three levels of adolescence (early, middle, and late). The reader interested in illustrative cameos of representative self-description at each of these six stages is referred to Harter (2012).

James's I-Self Functions: Self-Awareness, Self-Agency, Self-Continuity, and Self-Coherence

Although James (1892) struggled with the concept of the I-self, he nevertheless afforded it certain functional properties, certain positive characteristics. However, he did not speak to how these functions might be defined differently, or change, as a result of development. Thus, these will also briefly be addressed over the six periods of development that have been our focus. For the most part, this will cast the I-self in a more favorable light, with a job description more promising than the negative portrayal of the dark side

of the I-self's more odious duties. A developmental analysis of these positive functions, as well as how the I-self's role contributes to both normative descriptive characteristics of the me-self, including transient liabilities, will now be addressed.

Very Early Childhood (Ages 2–4)

During very early childhood, the I-self can craft only very concrete cognitive representations of the me-self, observable features of the self (e.g., I am a boy, I have a TV in my room, I have a kitty that is orange). Damon and Hart (1988) refer to these as categorical identifications, reflecting the fact that the young child understands the self only as separate, taxonomic attributes that may be *physical* (e.g., I have blue eyes), *active* (e.g., I can run real fast), *social* (e.g., My mommy and daddy love me), and *psychological* (I am happy). As Rosenberg (1979) cogently observed, the young child acts as a demographer or radical behaviorist in that his or her self-descriptions are limited to characteristics that are observable.

From a cognitive-developmental perspective, the me-self representations of this period are highly differentiated or isolated from one another; that is, the I-self of the young child is incapable of integrating these compartmentalized representations of self and thus self-descriptive accounts appear quite disjointed. This lack of coherence is a general cognitive-characteristic that pervades the young child's thinking across a variety of domains. As Piaget (1960) observed, young's children thinking is transductive, in that they reason from particular to particular, in no logical order. From the perspective of self-development, the I-self cognitive processes are unable to coordinate the attributes of the me-self.

Neo-Piagetians have elaborated on these processes. For example, Case (1992) refers to this level as *interrelational*, in that young children can forge rudimentary links in the form of discrete event-sequence structures that are defined in terms of physical dimensions, behavioral events, or habitual activities. However, they cannot integrate two such structures, in part because of working memory constraints that prevent young children from holding several features of the self in mind simultaneously (see also Griffin, 1992). Fischer's (1980) formulation is very similar. He labels these initial structures *single representations*. Such structures are highly differentiated from one another because the child is incapable of integrating single representations into a coherent self-portrait. Thus, the I-self processes of this period allow for little coherence in the self-descriptive narrative, which constitutes piecemeal and seemingly random, unrelated features of the me-self.

However, those characteristics that might be considered normative limitations are not really liabilities in any serious sense of the term. In fact, as has been argued elsewhere (Harter, 1999, 2012) they may serve motivational functions that propel young children to develop new skills in the absence of subjective cognitive scrutiny.

Jamesian I-Self Functions

Very young children (ages 2–4) have emerged from an earlier stage in which they mastered bodily self-awareness, they know what they look like as the *self-recognition* mirror paradigm has documented (see Berthenthal & Fischer, 1978; Lewis & Brooks-Gunn, 1979; Rochat, 2003). Subsequently, during very early childhood, self-awareness takes on more psychological manifestations, namely, the verbalization of self-referential attributes and behaviors. That is, linguistically the young child can now *describe* the self.

One feature of self-awareness at this substage of early childhood is the labeling of *internal states*, primarily basic emotions. By age 3, most children are able to describe their experiences of being happy, mad, sad, and scared (Bretherton & Beeghley, 1982; Harter & Whitesell, 1989; Kring, 2008).

A second manifestation of the I-self is a sense of *agency*, the conviction that one has control over one's actions and thoughts (see Lewis, 2008; Thomson, 2006). As a causal agent, one's actions have a predictable impact on others or the environment. Thus, a prototypical young child might describe how he can kick a ball really far and can count up to 100 if there is an audience to listen.

A third rudimentary I-self function at this age level is the sense of *continuity* or physical *permanence* over time (Nelson, 2003; Rochat, 2003). There is the realization that the self is invariant over time, even given changes in outward appearance (e.g., wearing different clothes, changing their hair). There is considerable agreement (Fivush & Haden, 2003) that *autobiographical memory* is critical to a sense of continuity. Creating self-*narratives* represents a primary mechanism by which a sense of continuity is established. Finally, the I-self must construct a sense of *self-coherence*, meaning that the different parts of the self are unified. This I-self function is less at issue at this developmental level because the self is highly differentiated, if not fragmented. Self-coherence will, however, because an important issue in mid- to late adolescence, as our own work has documented (Harter, 2012).

Early to Middle Childhood (Ages 5–7)

The hallmarks of me-self descriptions at this age level include competencies (e.g., I am good at my schoolwork),

physical skills (I can climb high on the jungle gym), and social skills (I have a lot of friends). However, the self-portrait is one of total virtuosity (e.g., I can do lots of stuff, real good! If you are good at things you can't be bad at things. Maybe *other* kids can't do certain things, but not me!). Thus, all-or-nothing thinking is still pervasive.

With regard to the cognitive-developmental advances of this period, what I am labeling as *I-self processes*, children at this age begin to display a rudimentary ability to inter-coordinate concepts that were previously compartmentalized (see Case, 1985; Fischer, 1980). For example, they can form a category or representational set that combines a number of their competencies (for example, good at running, jumping, climbing and throwing, or knowing their numbers, letters, and words). However, all-or-none thinking persists. In Case's (1985) model and its application to the self (Griffin, 1992), this stage is labeled *unidimensional* thinking. At this age, such black and white thinking is supported by another new cognitive process that emerges. The novel acquisition is the child's ability to link or relate representational sets to one another, to map representations onto one another, to use Fischer's terminology. However, of particular interest, with implications for self-development, is one type of representational mapping that is extremely common in the thinking of young children, namely, a link in the form of *opposites*. For example, in the domain of physical concepts, young children can oppose up versus down, tall versus short, thin versus fat or wide, although they cannot yet meaningfully coordinate these representations.

The ability to construct opposites, an I-self process, can also be observed within the descriptions of self and others, where the child's ability to oppose *good* and *bad* is especially salient. The child develops a rudimentary concept of the self as good at many skills. Because good is defined as the opposite of bad, this cognitive construction precludes the young child from being bad. Thus, an oppositional mapping (Fischer's term) takes the necessary form of "I'm good and therefore I can't be bad." However, others can be bad. Thus, this I-self structure leads the child to over-differentiate favorable and unfavorable attributes. These limitations extend to the inability to integrate such attributes as *nice* and *mean* (Fischer & Biddell, 2006), *smart* and *dumb* (Harter, 1986), or emotions such as *happy* and *sad* (Harter & Buddin, 1987). This mapping structure leads to the persistence of me-self descriptions laden with virtuosity.

Thus, the inability to acknowledge that one can possess both positive and negative attributes or emotions represents a normative cognitive liability that is a hallmark of this period of development. Moreover, due to greater linguistic abilities, the child is now able to verbally express his or her staunch conviction that one cannot possess both positive and negative characteristics, simultaneously. As one 5-year-old interviewee vehemently asserted: "Nope, there's no way you could be both smart and dumb, you only have one mind!" An interesting 5-year-old's insight, from the perspective of how the me-self is dependent upon I-self processes!

What might be considered to be the limitations of this period, for example, all-or-none thinking, the inability to coordinate positive and negative attributes (e.g., being both nice and mean) do *not* appear to be major normative liabilities in terms of the functioning of young children. Parents and teachers, who know children well, who have seen them pass through these phases normatively are not troubled by these manifestations. Toward the end of this period, parents or teachers may engage in dialogue with children to begin the exploration into how one can be both nice and mean, happy and sad, providing age-appropriate relevant examples that may facilitate the child's development to the next normative stage. As always, cognitive development can be enhanced with appropriate social interventions.

Jamesian I-Self Functions During Early to Middle Childhood

Self-awareness, self-agency, and self-continuity I-self processes undergo several developmental advances at this next level, ages 5 to 7, that Rochat (2003) described as one of *metacognitive self-awareness*. Children become more cognizant of how they are viewed in the public eye, attempting to simulate how the minds of others are constructing an image of them. Improved, but still limited, perspective-taking skills allow children to observe others evaluating the self (Selman, 1980, 2003).

This awareness is consistent with Nelson's (2003) concept of a *cultural self* that emerges during this period. The child's autobiographical self-history, codified in narratives, comes to be crafted in accordance with a cultural framework that dictates roles, institutions, and values. For example, these may include occupational choices that are likely to reflect gender stereotypes such as fireman, doctor, and professional athlete (for boys) and teacher, nurse, and mother (for girls). Behavioral norms are also salient (e.g., I'm learning why it is important for girls and boys to act differently). Bem (1985) observed that gender schemas not only dictate the *content* of stereotypes but also highlight that it is initially important to adhere to these cultural directives.

In the construction of *narratives*, children take an increasingly active role in telling their autobiographical

story, displaying a greater sense of *self-agency*. They incorporate their own personal experiences and mastery efforts and their successes. In addition, children, as architects of their narratives, are more likely to underscore *intentions* and future plans. These promote a greater sense of *self-continuity*, as children project their narratives into the future.

Middle-to-Late Childhood (Ages 8–10)

In contrast to the more concrete behavioral representations of younger children, older children are much more likely to describe the me-self in terms of generalizations such as *popular, helpful, nice*, *smart*, *mean*, and *dumb*. Children at this age level can now acknowledge that they can possess both positive and negative attributes. In addition to making reference to cognitive competencies *and* limitations, self-attributes become increasingly *interpersonal*, as relations with others become more salient dimensions of the self (see Damon & Hart, 1988; Harter, 2006).

From the standpoint of emerging cognitive-developmental processes that define the I-self, atttributes represent traits in the form of generalizations, based upon the integration of more specific behavioral features of the self (see Fischer, 1980; Siegler, 1991). For example, a generalization that one is smart, can be based upon the integration of scholastic success in both language arts and social studies. One may also feel dumb based on a generalization combining poor performance in both math and science. Popular may also combine several behaviors such as being nice, helpful, and good at keeping secrets.

A second I-self cognitive advance is the realization that one's self-attributes can be both positive and negative, in contrast to the all-or-none thinking that dominated the two earlier periods of childhood. As one 9-year-old thoughtfully observed, "I now realize that I can be both smart and dumb, you aren't just one or the other." Thus, what were formerly contradictory opposing attributes that could not co-exist in one's self-portrait can now be acknowledged by the I-self as realistic me-self descriptions that can simultaneously be integrated. These processes also apply to *emotion* concepts in that children can now also appreciate the fact that they can experience both a positive emotion (e.g., happy) and a negative emotion (e.g., sad) simultaneously (Harter & Buddin, 1987).

Children at this age can, for the first time, construct a higher order concept of their global self-worth or self-esteem. This construction involves, as James (1892) contended for adults, an evaluation of their competence or adequacy across those domains of their life in which success is *important*. Our own research has provided considerable documentation that this theory of the causes of self-esteem is alive and well, beginning at about age 8 (see Harter, 1999).

Also central to the construction of a concept of one's global self-worth is the contribution of Charles Horton Cooley (2002), who argued that a different process guided the formation of such a global concept. In his formulation of the looking-glass self, he argued that significant others function as psychological mirrors into which we gaze, to attempt to determine what *others* think of the self. These perceptions are then integrated into one's personal construction of their perceived worth as a person. Our own findings document Cooley's contention, at this period of development, not as an alternative theory competing with James' formulation, but one that complements it (Harter, 1999). Thus, advances in the I-self's cognitive ability to integrate diverse information about the me-self's competence as well as the importance of success in different domains (bolstering James' formulation) as well to take the perspective of another's evaluation of one's self and integrate it in one's global sense of worth of esteem (Cooley's contention) each emerge at this period of development.

Liabilities at this stage may ensue, if certain perceptions are less than accurate. For example, a child may overestimate or underestimate the level of one's competence in a given domain, given lack of experience in coordinating objective evidence of success (in the classroom, on the playing field, in social situations) into realistic subjective self-perceptions. Similarly, a child may not yet be sufficiently skilled at reading the perspectives of others who may be judging the self. Perspective-taking undergoes developmental change, as Selman's work (1980, 2003) has clearly demonstrated. Thus, there are subsequent skills to master in order to arrive at a realistic evaluation of the self, particularly one's authentic self, a topic to which we shall return, as we move into the stages of adolescence. However, there is definite improvement in perspective taking during later childhood, as we next address, in identifying James's I-self functions.

Jamesian I-Self Functions During Middle to Late Childhood

Perspective-taking skills improve dramatically between the ages of 8 and 10, facilitating increasing *self-awareness* (see Selman, 1980, 2003) and decreasing egocentrism. The standards and opinions of others become more internalized because the child can more clearly determine whether external expectations are met.

Narratives reflect a more mature sense of *agency* and *self-continuity*. Autobiographical memory is primarily dictated by personal experiences as the child becomes the major author of his or her life story, not merely the

narrator, previously incorporating input from parents. Agency is also observed in perceptions of both academic and social *self-efficacy*, expectations about what one can achieve in challenging circumstances (Bandura, 1977, 2006; Maddox & Gosselin, 2003). For example, a child may know how she can do well in certain school subjects and understands what it takes to be liked. The personal ownership of narratives, in consort with greater memory capacities, increased linguistic abilities, and a sense of self-efficacy all provide an increasing sense of *self-continuity*. Moreover, the child's *cultural self* (Nelson, 2003) expands during these years, as the child further adopts the standards and values of the larger society.

Interlude: Limitations of Piaget's Theory of Adolescent Cognitive Development, Applied to the Self

Adolescence represents a dramatic developmental transition, with pubertal and related physical changes as well as cognitive-developmental advances. With these new I-self cognitive acquisitions, adolescent development introduces the ability to think abstractly (Case, 1985; Fischer, 1980; Fischer & Bidell, 2006; Flavell, 1985; Harter, 1999, 2012; Higgins, 1991; Piaget, 1960). From a Piagetian perspective, the capacity to form abstractions emerges with the stage of Formal Operations in early adolescence. These newfound acquisitions, according to Piaget, should equip the adolescent with the hypothetico-deductive skills to create a formal theory. This observation is critical to the topic of self-development, given the claims of many earlier scholars (e.g., Epstein, 1973, 1981; Greenwald, 1980; Kelly, 1955; Markus, 1980; Sarbin, 1962) that the self is a personal epistemology, a cognitive construction, a theory that should possess the characteristics of any formal theory. Therefore, a self-theory should meet those criteria by which any good theory is evaluated. These criteria include the degree to which a theory is parsimonious, empirically valid, internally consistent, coherently organized, testable, and useful. Given Piaget's formulation, entry into the period of formal operations should make the construction of such a theory possible, be it a theory about entities in the world or a theory about the self.

As will become apparent, however, the self-representations constructed by the I-self during early and middle adolescence fall far short of these criteria. Self-representations are not coherently organized, nor are the postulates of the self-portrait internally consistent. Moreover, many self-attributes fail to be subjected to tests of empirical validity; as a result, they can be extremely unrealistic. Nor are self-representations particularly parsimonious, quite the contrary. There is little self-coherence. Thus, the Piagetian framework fails to provide an adequate explanation for the dramatic developmental changes in the me-self that can be observed across the substages of adolescence. Nor does it account for why the self does not meet the requisite criteria to qualify as a formal theory. A *neo*-Piagetian approach is needed to understand how changes in cognitive-developmental I-self processes result in both the organization and content of a very different me-self, at three levels: early adolescence, middle adolescence, and late adolescence. Each of these periods is next addressed, beginning with early adolescence.

Early Adolescence (Ages 11–13)

Many of the me-self descriptions to emerge in early adolescence represent *abstractions* about the self, based on newfound I-self cognitive abilities that integrate trait labels into higher order self-concepts (see Case, 1985; Fischer, 1980; Fischer & Bidell, 2006: Flavell, 1985; Harter, 1999; Higgins, 1991). For example, the young adolescent can construct an abstraction of the self as intelligent, by combining such traits as smart, curious, and creative. Alternatively, one may create an abstraction that the self is an airhead, given situations in which one feels dumb, scholastically inadequate, or just downright stupid. Similarly, an adolescent could construct an abstraction that he or she is an extrovert (integrating the traits of talkative, cheerful, and funny in social situations) as well as that he or she is also an introvert in certain interpersonal situations (where one is shy, uncomfortable, and nervous). Thus abstractions represent advances in the form of more cognitively complex representations now constructed by the I-self, whereby trait labels can now be appropriately integrated into even higher order generalizations that apply to the me-self.

Although the ability of the I-self to construct such abstractions represents a cognitive advance, these representations are highly compartmentalized; that is, they are quite distinct from one another (Case, 1985; Fischer, 1980; Fischer & Bidell, 2006; Harter, 1999; Higgins, 1991). For Fischer, these single abstractions are therefore over-differentiated, and therefore the young adolescent can only think about each abstraction as an isolated self-attribute. According to Fischer, structures that were initially observed in childhood reappear, but at a new abstract level. Thus, just as single representations were compartmentalized during early childhood, Fischer argues that when the adolescent first moves to the level of abstract thought, the I-self lacks the ability to integrate the many single abstractions that are constructed to define the me-self in different relational contexts. As a result, young adolescents will engage in all-or-none thinking at an

abstract level. For Fischer, movement to a qualitatively new level of thought brings with it lack of cognitive control on the part of the I-self. Therefore, adolescents at the level of single abstractions can only think about compartmentalized self-attributes, one at a time, but not simultaneously. Thus, contrary to earlier models of mind (Piaget, 1960), in which formal operations supposedly usher in newfound cognitive-developmental abilities that should allow one to create an integrated theory of self, fragmentation of self-representations during early adolescence is more the rule than the exception (Fischer & Ayoub, 1994; Harter, 1999, 2012; Harter & Monsour, 1992).

Compartmentalization of these abstract attributes can also be observed in the tendency for the young adolescent to be *unconcerned* about how certain postulates appear inconsistent across different roles and relationships. Thus, the fact that the self-portrait lacks consistency or coherence at this stage of early adolescence is not distressing. However, the inability to integrate seemingly contradictory characteristics of the self (e.g., intelligent vs. airhead, extrovert vs. introvert, cheerful vs. depressed) has the psychological advantage of sparing the young adolescent potential *conflict* over opposing attributes in his or her self theory (Harter, 1999; Harter & Monsour, 1992).

Moreover, as Higgins (1991) observes, the increased differentiation functions as a cognitive buffer, reducing the possibility that negative attributes in one sphere may spread or generalize to other spheres (see also Linville, 1987; Simmons & Blyth, 1987). Thus, although the construction of multiple selves across roles and relationships sets the stage for attributes to be contradictory, most young adolescents do not identify potential inconsistencies or experience conflict within their self-portrait, given the compartmentalized structure of their self-representations. However, the inability to integrate one's various attributes at this stage has liabilities for important Jamesian I-self functions to next be discussed.

Jamesian I-Self Functions During Early Adolescence (Ages 11–13)

Early adolescence marks the beginning of intense self-consciouness, a heightened concern with the reflected appraisals of others (Harter, 1999, 2012; Lerner, 2002; Rosenberg, 1986). S*ocial awareness* increases dramatically, leading to a greater self-awareness of how one's attributes are viewed by others, particularly because different significant others hold varying opinions of the young adolescent. This preoccupation and uncertainty contributes to the beginning of introspection, self-reflection, and rumination (see Selman, 1980, 2003), particularly about one's negative characteristics. Thus, self-awareness is a mixed blessing.

Moreover, the compartmentalization of role-related attributes, as multiple selves abound, also represents a double-edged sword. It spares young adolescents the psychic pain of acknowledging contradictory attributes in their self-portrait. However, it interferes with the I-self functions of agency, and self-continuity, particularly its corollary of self-coherence. There is no integrated sense of self with which one can imbue intention and future directions.

Middle Adolescence (Ages 14–16)

New cognitive I-self processes emerge during middle adolescence, giving the self-portrait a very new look (Fischer & Bidell, 2006; Harter, 1999, 2012). Whereas in the previous stage single abstractions were psychologically isolated from each other, during middle adolescence the I-self acquires the ability to make *comparisons* between single abstractions. Fischer labels these new structures abstract mappings; the adolescent can now map constructs about the self onto one another, that is, directly compare them. Such mappings force the individual to compare and contrast different attributes as they become increasingly salient. It should be noted that abstract mappings have features in common with the representational mappings of children, in that the cognitive links that are initially forged take the form of *opposites*. During adolescence, these opposites reflect contradictory *abstractions* about the self (e.g., extrovert vs. introvert, tolerant vs. intolerant, responsible vs. irresponsible, good-looking vs. unattractive).

Noteworthy is the fact that the abstract mapping structure that the I-self imposes on the me-self has serious limitations as a means of relating two concepts of self to one another. The ability to merely compare attributes simultaneously forces the individual to recognize troublesome inconsistencies within the self. However, the individual cannot yet truly integrate such self-representations in a manner that would resolve apparent contradictions. As a result, the awareness of these opposites causes considerable intra-psychic conflict, confusion, and distress (Harter, 1999; Harter & Monsour, 1992) given the inability to coordinate these seemingly contradictor self-attributes. For example, one adolescent interviewee agonized over whether she is an extrovert or an introvert (Am I just acting like an extrovert, am I just trying to impress them, when really I am an introvert?). Another adolescent expressed consternation over a different contradiction (So which am I, responsible or irresponsible, how can the same person be both?). Thus, the cognitive-developmental limitations of the I-self during middle adolescence contribute to the emergence of what James (1892) identified as the conflict

of the different me's, leading to major normative liabilities, to be overcome at the subsequent level.

Jamesian I-Self Functions During Middle Adolescence (Ages 14–16)

Development is not necessarily a linear progression toward some teleological end point. There is clearly a blip on the psychological radar screen at midadolescence. This point is often missed in classic treatments of development. *Self-awareness*, a Jamesian I-self function, previously manifest as the unreflective self-acceptance of one's attributes during earlier periods of development vanishes. What were previously unexamined self-truths, the basis for a durable self-image, now become troublesome self-hypotheses. The tortuous search for the self is made more difficult given the painful proliferation of multiple me-selves that now crowd the self-landscape (Harter, 1999). Self-awareness becomes quite intense, yet the images are not stable or enduring. It is much like a lighthouse beacon that flits from buoy to buoy, providing only fleeting glimpses of potentially troubled waters. By analogy, awareness quickly shifts from role to role where one's image of self is defined quite differently. Thus, a kaleidoscopic self emerges (Harter, 2012).

Another related I-self function, a sense of *self-coherence*, is compromised given the contradictory multiple selves that cannot be integrated. The self as a *causal agent* can also be problematic, given that the construction of multiple selves that do not speak with one voice. Thus, there is no efficacious self at the helm, precluding the perception that one is a causal agent in control. As such, it is difficult for adolescents in this period to project a clear sense of themselves into the future, and as a result, the search for *self-continuity*, links between one's past, present, and future selves, must be put on hold. For many adolescents, our own daughter included, one manifestation is the ever changing swirl of potential occupational choices, many of which seem contradictory. While it is healthy to consider options, it is difficult for those in midadolescence to logically think through how to make meaningful decisions. Nonjudgmental listening on the part of parents and other significant adults is critical, scaffolding a consideration of the pro's and con's supporting their own thinking, without heavy-handed suggestions or adult decisions.

Late Adolescence (Ages 17–19)

In contrast to middle adolescence, in late adolescence the many potentially contradictory attributes are no longer described as characteristics in opposition to one another. Thus, one can be both an extrovert as well as an introvert, responsible as well as irresponsible, both depressed and cheerful as well as conscientious as a student but also lackadaisical. With regard to the last of these attribute pairs, one of our interviewees explained: "That's normal, I mean you can't just be a total 'grind,' you'd be pretty boring if you were. You have to be *flexible*." He further explicated why introversion doesn't conflict with extroverted behaviors: "You have to be *different* around other people. It would be weird to be the same kind of person on a date or with my friends at a football game or with my mother!"

Some important I-self cognitive acquisitions allow the older adolescent to overcome some of the liabilities of the previous period, where opposing attributes were viewed as contradictory and a cause of internal conflict. The general I-self cognitive advances during late adolescence involve the construction of *higher order-abstractions* that allow for the meaningful intercoordination of single abstractions (see Case, 1985; Fischer, 1980; Fischer & Bidell, 2006; Harter, 1999, 2012). For example, the fact that one is both introverted and extroverted can be integrated through the construction of a higher order abstraction in which the self is adaptive. The observation that one is both depressed and cheerful can be integrated under the personal rubric of moody. Similarly, flexible can allow one to coordinate conscientiousness with the tendency to be lackadaisical. Thus, what were formerly troublesome inconsistencies in the me-self can now comfortably coexist.

In the English language we actually have particular linguistic terms that codify the integration of concepts that might appear to be contradictory. One of our older teenage interviewees utilized two such words in his description of his feelings about going off to college. "I want to be independent of my family but still want to remain connected to my parents, so I guess you could say that I am *ambivalent*. I am excited about going to college but sad over leaving my parents; it's rather *bittersweet*." Such higher order abstractions provide self-labels that bring meaning and therefore legitimacy to what formerly appeared as troublesome contradictions within the self. They allow the older adolescent to *normalize* potentially inconsistent attributes within their personality. Thus, we have observed how at every developmental level, the I-self is at the helm, in creating an age-appropriate portrait of the me-self.

Jamesian I-Self Functions During Late Adolescence (Ages 17–19)

Limitations of the preceding period are overcome as a result of cognitive-developmental acquisitions during late adolescence (Case, 1985; Fischer, 1980). One critical cognitive advance is the ability to construct higher order

abstractions that provide a meaningful integration of single abstractions that previously represented contradictions (e.g., depressed and cheerful no longer conflict because they are combined into the higher order abstraction of moody). The older adolescent can also resolve potentially contradictory attributes (e.g., studious versus lackadaisical) by asserting that one is flexible or adaptive, thereby subsuming apparent inconsistencies under more generalized abstractions about oneself. Each of these new acquisitions sharpens the accuracy of the I-self's *self-awareness,* a Jamesian I-self function. Through these mechanisms, conflict over previous contradictions in one's self-portrait abates (Harter & Monsour, 1992). Thus, self-awareness is also reflected in the older adolescent's recognition of these cognitive strategies and their effectiveness.

Characteristics reflecting personal beliefs, values, and standards become even more internalized. The focus on future or possible selves (see Markus & Nurious, 1986) also gives the older adolescent some sense of direction, albeit often idealistic. Thus, compared to midadolescence, the teenager at this stage demonstrates a greater sense of *agency* as well as self-continuity. In addition, the ability to construct higher order abstractions that resolve the many contradictions of the previous period allows for a much more integrated and *coherent* self-portrait. In addition, older adolescents can now construct a self with a greater sense of *self-continuity* that can aid in plans and projections for the future. Theoretically, this would be the ideal; however, there are impediments given cultural dictates and demands that can lead to pathologies, to which we next turn.

SELF-ENHANCEMENT AND SELF-SERVING BIASES: THE I-SELF/ME-SELF CONSPIRACY

We have observed how the I-self, defined as age-appropriate cognitive processes, constructs a changing me-self at different stages during childhood and adolescence, a worthy goal. However, there is a downside to this endeavor. The I-self is also required to devote time and energy in efforts to enhance and package the me-self in a way that meets with societal expectations. However, this protective preoccupation with the self is a contemporary cause for concern.

There is not unequivocal support for such a position. Some (see Alicke & Sedikes, 2009) make a case for how self-enhancement is *normative*, in describing optimistic cognitive illusions, viewing the self as having more personal control than might be warranted, and better life circumstances, than others would describe about them. In so doing, they emphasize the many positive, self-protective strategies. Those not engaging in such biases are more likely to experience depressive reactions.

However, we concur with Leary (2004) that there are costs of self-enhancing strategies, self-serving biases, unrealistically inflated self-perceptions, self-aggrandizement, narcissistic fantasies of omnipotence, and other illusions about the self that have been amply documented among adults, leading Leary (2004) to refer to the *curse of the self*. Adults engage in downward social comparisons to make themselves look better, they claim to be better than average, they take more responsibility for their successes than failures (blaming external factors for one's personal misfortunes), and they believe that they are special or have unique talents (the false uniqueness effect). Finally, they are unaware of these biases, acknowledging them in others but not in themselves. These self-serving behaviors threaten relationships with others and have personal costs, one of which is they jeopardize *authenticity*. The true self can become seriously compromised for adults by narcissistic tendencies to inflate, becloud, and distort the real inner self, in desperately seeking social approval, admiration and associated gain (see Harter, 2012). In sum, the I-self is at the helm, actively packaging the me-self, in the service of meeting these potentially destructive goals.

However, just what are the *developmental prerequisites* necessary to engage in these processes? At each of the same six developmental levels, three in childhood and three in adolescence, we can ask three questions:

1. What determines the need or *motive* to pursue various self-enhancement goals at a given substage? Why might one be driven to enhance and protect the self?
2. At each developmental level, what cognitive *skills* or *mechanisms* are available to the I-self to engage in such self-serving strategies? We cannot assume that the mature I-self processes accessible to adults are operative during the developmental stages of childhood and adolescence. At what point, therefore, do self-serving biases merge into narcissism and become more pathological?
3. What are the *consequences* of engaging in developmentally determined self-enhancement strategies or self-serving biases, and when do they become truly compromising?

Self-Enhancement Strategies During Very Early Childhood

Critical to this assessment is the observation that very young children's self-reports are overly positive and inaccurate, for several reasons that involve the developmental limitations of the I-self. First, young children lack the

cognitive ability to engage in *social comparison for the purpose of self-evaluation*. From a cognitive-developmental perspective (Piaget, 1960), social comparison requires that one be able to hold two dimensions in mind simultaneously, actively comparing an evaluation of one's own characteristics to another's. This exceeds the cognitive capacity of the very young child's I-self.

Second, one can observe the rudimentary use of *temporal comparison*, an awareness that one's current skills greatly exceed those of the not-so-distant past (see Harter, 2012), leading to an overestimation of young children's abilities, by social-comparative standards. For example, young children may boast that they can run faster than when they were younger, which is true. Temporal comparisons are particularly salient and gratifying given that skill levels change rapidly during this age period and thus improvement is quite noticeable. An appreciation for these advancements contributes to the overestimation of one's competencies.

A third limitation of the I-self processes in young children is their inability to distinguish between their *actual* self-attributes and their *ideal* self-attributes. As a result, self-evaluations are unrealistically positive since the fusion or confusion of the two judgments favors idealization. These overly positive evaluations reflect the cognitive limitations of the I-self rather than purposeful deceit (Harter, 2012).

Fourth, very young children lack the *perspective-taking* ability to understand and therefore incorporate the perceived opinions held by significant others (Harter, 2006; Selman, 1980, 2003; Thompson, 2006). Thus, the inability to fully comprehend that adults may be critical of them leads very young children to persist in overly positive self-evaluations. The I-self's cognitive prerequisites for Cooley's (1902) looking-glass self formulation, in which the opinions of others are recognized and incorporated into the me-self, are lacking in very young children.

Fifth, cognitive limitations also lead to young children's inability to acknowledge that they can possess both positive and negative self-attributes (Case, 1985; Fischer, 1980; Fischer & Bidell, 2006; Harter, 1999, 2012). That is, the all-or-none thinking characteristic of this age level extends to children's evaluations of themselves. They must be unilaterally positive in most young children.

For all of these reasons, very young children's self reports will naturally be overly positive and inaccurate. However, do they constitute the conscious self-enhancement efforts that have been described for adults? While some (see Trzesniewski, Kinal, & Donnellan, 2010) view these normative self-biases as similar to the types of self-enhancing strategies observed in adults, my own developmental perspective suggests a different interpretation. First, young children do not need to tactically enhance perceptions of competence because their self-evaluations are already normatively inflated! Thus, they do not possess the *motives* that propel adults' tendency to enhance themselves, namely, to protect or conceal fragile or negative self-images. Second, the *mechanisms* employed by adults are not yet in place (e.g., downward social comparisons, false uniqueness processes, attempts to reduce ideal-real self-image disparities, social perspective taking). Third, the consequences are quite different. The normative exhibitionistic displays of young children are typically endearing and meet with social adult approval. In contrast, the self-centered grandiosity among adults, evoked to protect a damaged self, blinds narcissists to their shortcomings and does not elicit support from others; rather it provokes rebuffs or rebukes.

The very young child's desire to be the center of attention, *normative narcissism*, is age appropriate compared with pathological forms of narcissism where these concerns are excessive (see Kernberg, 1975, who has articulated the distinction between normative and pathological narcissism). The exhibitionism among healthy children is typically warm and engaging, whereas in pathological narcissism, the demands for constant admiration are defensive and generally not socially welcome.

One can also observe the seeds of the construction of a false me-self, by the I-self, given processes that build upon unrealistic self-perceptions, compromising one's sense of authenticity. The emergence of *language*, another I-self function, represents a double-edged psychological sword in this regard. Language clearly promotes heightened levels of relatedness and allows for the creation of a personal narrative. Stern (1985), however, also alerted us to the liabilities of language, which can drive a wedge between two simultaneous forms of interpersonal experience, as it is lived and as it is verbally represented. The young child can distort his or her immediate experience and create a fantasized or falsified construction of the self. Such distortions may contribute to the rudimentary formation of an inauthentic self, if the child accepts the falsified version of experience.

Self-Enhancement During Early to Middle Childhood (Ages 5–7)

Because the I-self of young children is not yet cognitively equipped to develop a general concept of one's worth as a person (see Harter, 2012), there is no global

self-esteem to enhance! The normative overestimation of competencies described for the previous period continues, for the most part, to persist, including: (1) the inability of the I-self to engage in *social comparison* for the purpose of self-evaluation; (2) the use of *temporal comparison* highlighting greater skill levels compared with previous competencies; (3) the inability to differentiate one's *actual* self attributes from one's *ideal* attributes; (4) the *lack of perspective-taking,* which precludes an appreciation for the evaluations of significant others; and (5) the inability to acknowledge that the me-self is composed of both positive and negative characteristics. As such, self-perceptions continue to be inaccurate. These I-self limitations obviate the need to protect the me-self; there is no obvious *motive*. These unrealistically positive perceptions of competence are reinforced by all-or-none thinking. However, basically, young children do not possess the skills to engage in defensive or protective strategies. They may lie or blame a sibling in an attempt to conceal a transgression (Wilson, Smith, & Ross, 2003). However, their efforts are usually quite transparent and thus not that effective as self-protective strategies.

I-self processes identified in very early childhood will continue to set the stage for the development of false self-behavior. As noted for the previous stage, language provides the linguistic vehicle through which the child can falsify his or her experiences. As language becomes more proficient, and as the child plays an increasingly active role in constructing his or her autobiographical narrative, the development of a falsified self becomes increasingly possible. As one of our young interviewees revealed in describing his life story, "Some parts are make-believe but mostly they're true!" Thus, the child has a basic notion that the content can be distorted. Wolf (1990) has described several mechanisms such as deceit and fantasy, whereby the child at this age, as author of his or her self narrative, can select, edit, or change the facts in the service of personal goals, hopes, or wishes (see also Dunn, 1988). This linguistic-cognitive backdrop of I-self processes can pave the way for a lack of authenticity.

Narcissism continues to be *normative* at this age level. This includes fantasies of success, wealth, power, and fame that are probably less than realistic. These include positive autobiographical stories, peppered with fanciful projections into the future, grandiose aspirations and wishes about who or what one will become when one grows up. The normative narcissism at this age level is associated with more conscious exhibitionism; there is more awareness of the impact on one's audience, as the child shows some budding showmanship. Nevertheless, these behaviors are still within the normative bounds of age-appropriate narcissism.

Self-Enhancement During Middle to Late Childhood (Ages 8–10)

Advances heralded as hallmarks of development usher in the potential for various self-protective strategies to emerge. First and foremost, the newfound I-self capacity to forge a concept of one's global self-esteem, unavailable to younger children (see Harter, 2012) raises the psychological specter that feelings of worth may need to be protected or defended. Greater perspective-taking skills allow the older child to realize that there is a wider, observing audience whose members are also evaluating the self. This, in turn, can provoke self-consciousness and the need to develop strategies to ensure the I-self's positive self-evaluations of the me-self. The increased ability to engage in social comparison also makes one's self-appraisals more vulnerable. Thus, advances during this period paradoxically may produce new *needs,* demanding that new I-self skills be devoted to the protection and enhancement of the me-self. What new skills might promote such strategies?

First, sensitivity to social comparison allows one to capitalize on this awareness and to submit others to *downward social comparison*. Thus, the I-self can implicitly or explicitly compare oneself with peers considered inferior, thereby enhancing the me-self. Older children can now adopt the *better-than-average* strategy as well as make attributions that one is more successful than others in areas of greatest social importance, thereby elevating their self-esteem.

Thus, increased perspective-taking skills emerge, but they are not necessarily utilized toward the greater good of peers. They might also be employed in the service of *impression management*, playing to the prevailing peer standards of what is cool or awesome. Newfound perspective-taking skills can also be used to one's advantage, by manipulating peers to meet one's own needs and desires (Selman, 1980, 2003). The older child can engage in psychological attempts at persuasion, convincing others to view things from his or her own perspective. One is reminded of Tom Sawyer, who conned his peers into believing that his job of painting the fence was a desirable enterprise.

Older children can also engage in a practice first identified by James (1892), who viewed self-esteem as a balance between perceptions of domain-specific competencies and the *importance* of success attached to these domains. The I-self can *discount* the importance of domains in which one is not meeting one's expectations, thereby preserving and protecting the me-self.

Therefore, the period of middle to late childhood ushers in new critical acquisitions that forecast the need to engage in self-enhancement strategies. First, there is the emergence of the concept of one's global self-esteem and second, the acknowledgement of negative self-appraisals, present new *motives* for self-protection and enhancement. Third, new cognitive-developmental skills (e.g., social comparison abilities, enhanced perspective-taking skills and discounting) equip the older child with the *capacity* to engage in self-protection, self-enhancement, and self-presentation.

Another potential negative is that true-self behavior may become eroded, as the older child can better evaluate the self unfavorably, with the realization that significant others, particularly peers, may also have unfavorable views of one's me-self. This vulnerability may alter the self that is presented to the social world, leading to frantic efforts to regain favor in the eyes of others. Thus, false-self behavior may become more prevalent, given increasing I-self management skills designed to protect a potentially fragile me-self.

Earlier *normative* narcissistic features begin to fade in middle to late childhood, as children become better able to accept their flaws, adopting a more balanced perspective on their strengths and weaknesses (Bardenstein, 2009). However, new *pathological* manifestations of narcissism emerge (Bleiberg, 1994). The older narcissistic child lacks empathy for others' feelings and intentions. Grandiosity, an inflated sense of self coupled with impulsivity, precludes a tolerance for outcomes that are not immediately successful. Others are blamed for one's personal deficits, compromising relationships (see Bardenstein, 2009; Kernberg, 1998). The narcissist's sense of superiority and entitlement leads to the exploitation and manipulation of peers. Preoccupied with protecting a fragile sense of self, narcissistic children devalue others and if criticized, display rage. Narcissists may self-select relationships with weaker peers who will reinforce their grandiose self views (Carlson & Gjerde, 2009). They seek to dominate social interactions, to impress others and to gain admiration rather than to establish genuine friendships or close relationships (Thomaes, Bushman, Stegge, Olthof, & Denissen (2008).

Self-Enhancement During Early Adolescence (Ages 11–13)

Normative strategies on the part of the I-self to protect and enhance the me-self are evident in early adolescence. The heightened preoccupation with others' perceptions of oneself, coupled with a focus on the discrepancy between the real and ideal self, conspire to produce an escalation in the *need* to protect and enhance the self. As one young adolescent told us, "It's important to act as if you really like yourself." Thus, there is an appreciation of the powerful motives for self-protection and self-enhancement. Moreover, young adolescents have certain requisite skills.

The increasing emphasis on *social comparison* provides a psychological stimulus for the strategy of *downward* social comparison. Although overt comparisons are frowned upon, successful students have the information to conclude that they are more scholastically competent than others, thereby protecting or enhancing the self. The criteria for social acceptance are quite clearly communicated by peers, leading the more popular young teenagers to conclude, through downward social comparison, that they are higher on the totem pole of popularity. Since only a few can rise to the top of the scholastic and social ladders, most young adolescents are vulnerable, fueling their motivation to enhance, or at least protect, their self-images.

Cognitive I-self skills to engage in certain adult attributional biases become available to the young adolescent. For example, successes can be attributed to internal, stable characteristics such as intelligence whereas failures are due to external factors such as an excessively difficult test. As a young adolescent interviewee recounted, "If I do well on tests, I tell myself it's because I'm smart. If I don't do well, it's because the test was too hard or the teacher didn't teach us what we needed to know." Another attributional bias for lack of success is the claim that one simply didn't study hard enough, implying that one could have done better, with increased personal effort.

Young adolescents also make use of Greenwald's (1980) concept of beneffectance, a self-protective strategy in which one's central or important attributes are touted as positive, whereas negative characteristics are relegated to the periphery of one's concept of self. However, these tactics can coexist with an I-self who may become preoccupied with protecting a me-self that is becoming increasingly false or phony.

Early adolescence brings with it (1) a greater appreciation of the meaning of false self behavior, (2) its greater salience in one's vocabulary, and (3) a preoccupation with its detection. Not until about age of 12 or 13 can young adolescents clearly define false self-behavior (Harter, 2012). Definitions focus on concealing what you really think or feel and saying things that you don't really believe. One adolescent participant struggled consciously with this issue: "I try to tell myself that what *I* think is the most important, that I should just be my true self. I shouldn't be phony and act like I'm somebody else. Except I *do*!" Young adolescents are sleuths and appear driven to detect hypocrisy. While they acknowledge some false

self behavior in themselves, they are far more likely to observe it in others (e.g., parents and peers). Anthony (1982) has described a new vocabulary that emerges at this stage. Young adolescents subject others' beliefs to scrutiny and criticism, attempting to expose the contradictions in parental arguments, values, and behavior. Thus, parents, in particular, are accused of hypocrisy, deflecting blame from the adolescent, which, in turn, should supposedly protect the me-self.

However, the young adolescent's self-perceptions are not necessarily *accurate*.

During the preceding period of late childhood, a number of cognitive-developmental advances promoted more realistic self-evaluations. However, the progression toward more accurate judgments is not necessarily linear. As noted earlier in the chapter, there are *liabilities* of advances to new levels of cognitive development, in this case, early adolescence (Harter, 2012). The young adolescent can now begin to construct abstractions about the self, for example, extravert and introvert. However, the more abstract the representation, the farther removed it is from concrete behavioral evidence. As a result, self-evaluations can lack convincing documentation, thereby leading to inaccurate self-evaluations.

Moreover, narcissism becomes a potential danger to the self at this age. The features of narcissistic personality disorders begin to emerge and can crystallize during early adolescence (Bleiberg, 1994) and become more stable (Bukowski, Schwartzman, Santo, Bagwell, & Adams, 2009). These include an omnipotent sense of self, refusal to acknowledge one's shortcomings, and unrealistic demands for public affirmation. Narcissistic disturbances invariably stem from concerns that one is ignored or not given sufficient attention, often coupled by doubts about one's ability to evoke positive responses from others.

Another source of narcissistic vulnerability is the realization that one's actual self does not conform to one's lofty aspirations (Joffe & Sander, 1967). Joffe and Sander observe that narcissistic vulnerability evokes *shame* given the deflation that accompanies the inability to measure up to one's ideal. Those with pathological forms of narcissism defensively deny any signs that they are failing to achieve self ideals. Their self-narrative contains illusions of power, omnipotence, control, perfection, and invulnerability resulting in a highly distorted sense of self. They cannot see past their own reflection. These self-aggrandizing fantasies conceal very fragile and unstable self-esteem, requiring high-maintenance personal efforts to obtain compensatory attention and admiration (Rhodewalt, 2006), efforts that may be doomed to failure.

Self-Enhancement During Middle Adolescence (Ages 14–16)

Our developmental analysis has focused on several themes, including the needs or motives fueling attempts to protect and enhance the self and, second, the skills to enact the various strategies that have been identified in adulthood. Although certain skills are in place (e.g., the ability to engage in downward social comparisons) contradictions between multiple selves may preclude a thoughtful consideration of just which selves should be protected or enhanced. The adolescent may be overwhelmed with the enormity of the challenge. Given that the adolescent at this stage is ill equipped to resolve the various contradictions in his or her self-portrait, he or she will be very ineffective in employing self-protective or self-enhancing strategies. One adolescent expressed this distress as follows. When she puts on her extraverted face, she asks, "Am I just trying to impress the other kids, when I am really an introvert?" Thus, there would appear to be a motive to engage in self-enhancement, although efforts are not particularly successful. The desire to enhance the self is powerful, although the skills may not be accessible.

Moreover, what may pass as normative narcissistic attempt to protect the self must be distinguished from more pathological forms of narcissism (Bleiberg, 1994; Lapsley & Stey, 2012). These theorists discuss this distinction within the context of a major developmental challenge during midadolescence, namely, the need to begin the process of separating from parents, to individuate, while at the same time remaining appropriately connected. In healthy adolescent development, teenagers maintain basically good relationships with parents, despite the squabbles and normative conflicts (Bleiberg, 1994). They build, and strive toward, an ideal self as they explore their expanding world. They struggle with the construction of realistic self-images in their pursuit of future goals, and in the process create their own personal fable. At this age, their narratives contain moderate feelings of *omnipotence*, a perception of *uniqueness*, and a sense of *invulnerability*, features of *adolescent egocentrism*, as initially described by Elkind (1967), further examined by Harter (2012). How, then, do these presumed normative characteristics differ from pathological manifestations of narcissism?

Lapsley and Stey (2012) offered several contrasts. Normative narcissism reflects (1) grandiosity without the exploitation of others, (2) illusions without a sense of entitlement, (3) expressions of invulnerability without shame, (4) a sense of omnipotence that doesn't risk the isolation from others, (5) the desire for realistic admiration versus the unrealistic demands for attention and preening

self-preoccupation, and (6) positive affect and warm relationships with others versus derision, lack of empathy, and envy. Thus, these distinctions mark the boundaries between normal and dysfunctional narcissism. Dysfunctional narcissism represents failed attempts at self-enhancement. Extreme grandiosity provokes desperate maneuvers on the part of the I-self to protect the me-self's very precarious self-esteem and illusory control. They display relentless addiction-like strivings to continually assert their superiority (Morf, Horvath, & Torchetti, 2001). However, such efforts are eventually unsuccessful, leading to consequences such as peer rejection because such an inflated self-portrait reflects unrealistic bravado. This, in turn, provokes negative social feedback. In addition, dysfunctional narcissism also leads to the alienation from one's authentic self (see Given-Wilson, McIlwain, & Warvurton, 2010; Nicholls & Stukas, 2012), particularly if they broadcast supposed evidence for their self-glorification (Wallace, 2011).

These processes also interface with the potential for the construction, by the I-self, of a false, me-self, which can become more pathological. The proliferation of multiple selves at this developmental juncture, coupled with the ability to detect potential contradictions between self-attributes displayed in different roles, naturally ushers in concern over which attributes define the true self (Harter, Bresnick, et al., 1997). Many of our participants have spontaneously agonized over which was their true self, the real me. An illustration of this preoccupation can be seen in the confessions of one 15-year-old girl. When she switches from being cheerful with friends to being nasty with parents, she asks, "Which one is the real me?" In obsessing over being both an extrovert and an introvert, she laments, "I don't know who I really am!" In admitting that she is both irresponsible with friends but responsible on the job, she queries: "So which am I, responsible or irresponsible, how can the same person be both?" She ends her self-reflection disclosing that she thinks a lot about "who is the real me, and I try to figure it out when I write in my diary, but I can't resolve it." Thus, from a normative perspective, the adolescent at this age level is ill equipped to solve these dilemmas. If the adolescent *ruminates* excessively about these contradictions, then more pathological thinking ensues. That is, the frantic I-self seems unable to protect the kaleidoscopic me-self (Harter, 2012).

Self-Enhancement During Late Adolescence (Ages 17–19)

Clearly, in late adolescence, cognitive advances in the form of higher order abstractions and efforts to *normalize* potential contradictions serve to reduce the distressing conflict and negative self-appraisals. In addition, efforts to discount lack of success in domains that are deemed unimportant are increasingly utilized as strategic attempts to maintain high self-esteem. As one older adolescent put it to us: "Let's face it, I'm not that popular, you have to be a jock in our high school to be really popular, but I have never been into sports, they just aren't important to me, that isn't where I put my energies. I have my own circle of friends, so being popular doesn't really appeal to me." Discounting the importance of athletic competence and popularity, domains in which one is not that successful is but one of many self-protective strategies.

However, other strategies, identified in the adult literature, become even more available to older adolescents, as their repertoire expands, allowing them to engage in self-enhancement, self-serving biases, and narcissistic tendencies. For example, the *better-than-average effect* can be observed in another adolescent's declaration that "I am more moral than the average person my age," a stance that also requires downward social comparison. Thus, most of the self-protective and self-enhancing strategies exhibited by adults are available to older adolescents. In addition, older adolescents, no longer as overwhelmed with the contradictions and confusion experienced in midadolescence, may be in a better position to facilely or skillfully execute and combine strategies, as the I-self attempts to better protect the me-self.

The issue of authenticity remains on the personal radar screen of older adolescents, although they are less likely to agonize about whether they, or their parents, are phony or hypocritical. That said, it remains a cultural preoccupation that one cannot avoid. The English language is rife with terms that speak to our societal concern with false self-behavior. As Lerner (1993) pointed out in her intriguing book *The Dance of Deception,* we have a vast vocabulary that signifies deceit. Verb forms refer to *fabricating, withholding, concealing, distorting, falsifying, pulling the wool over someone's eyes, posturing, charading, faking*, and *hiding behind a façade*. Adjectives include *evasive, elusive, wily, phony, fake, artificial, two-faced, hypocritical, manipulative, calculating, pretentious, crafty, conniving, duplicitous, deceitful*, and *dishonest*. Noun forms include *charlatan, chameleon, impostor, hypocrite, a fake*, and a *fraud*.

Given the cognitive and linguistic advances of older adolescents, their own vocabulary is likely to expand, as they become increasingly aware of how society chooses to verbally frame such deceitful activities and personal characteristics. Many manifestations primarily refer to verbal expression in which one's true belief and attitudes are suppressed and in their place, false opinions are endorsed.

However, as a strategy to gain or maintain approval, from both peers and parents, opting into this dance of deception may ultimately backfire. Our own findings reveal that those who display higher levels of false-self behavior within a given relational context (suppressing their voice, supplanting it with false assertions) report less peer approval and lower self-esteem (Harter, Waters, & Whitesell, 1997). Greater true self behavior is associated with higher self-esteem. Thus, those admitting to false self behavior devalue their worth as a person, raising a causal question for future research: Namely, which comes first? Does true versus false self behavior *precede* the corresponding high versus low self-esteem? Alternatively, does one's evaluation of his or her overall worth as a person dictate the perceived authenticity of the self?

Concerns about the prevalence of false-self behavior shade into links to *narcissistic* tendencies during the period of late adolescence. Characteristics of both normative and pathological narcissism emerged as salient during the previous period of midadolescence (Bleiberg, 1994; Lapsley & Stey, 2012) when a major developmental challenge is to begin, in earnest, namely, to separate from the family, to individuate. This theme can be activated or exacerbated for those in *late* adolescence when the end of high school looms large and separation issues come to the forefront if one is college-bound or moving out of the home due to job or new age-appropriate relationship demands. Leaving home can trigger excessive separation anxiety that, for those whose upbringing has put them on a path to maladaptive narcissism, can provoke rage at parents who are perceived as the perpetrators.

These reactions can be exacerbated in future adulthood when narcissistic individuals present themselves as grandiose and arrogant, creating an inflated impression of their self-worth; the motive is to avoid internalized feelings of inadequacy (LeCours, Briand-Malenfant, & Torchetti, 2011; Ruggiero, Veronese, Castiglioni, & Sassaroli, 2011). The causal role of pathological narcissism also has been found to result in aggressive behavior (Bushman & Thomaes, 2001; Thomaes, Bushman, Segge, Olthof, & Denissen, 2008). As Bushman and Thomas (2001) observe, when the narcissistic ego deflates, narcissistic aggression inflates. Others focus on the self-conscious emotions, namely, hubris (excessive pride), which becomes deflated and can be replaced by shame (Tracy, Cheng, Martense, & Robins, 2012). As Campbell and Buffard (2008) point out, while there are immediate benefits to narcissistic behaviors (feel-good temporary affective experiences), these backfire in that they are not well received by others, in most social circles.

Those who fit what Bleiberg (1994) has described as *histrionic exhibitionism* may become especially troubled over separation issues provoked by impending adulthood. Their excessive dependency upon parents, and their inordinate need for admiration that may not be forthcoming in their new environment, will not bode well at this developmental juncture. A healthier reaction to these new demands (see Bleiberg, 1994; Lapsley & Stey, 2012) can be observed among our own study participants in normative samples. For example, one older adolescent admitted to being ambivalent about going off to college and that leaving home will be bittersweet. Although he was looking forward to leaving home and going to college, he acknowledged, "It's a little nervous," and, "I'll probably always be somewhat dependent on my parents." He tries to reassure himself that "my parents will always be there for me."

Healthy reactions notwithstanding, Twenge, Konrath, Foster, Campbell, and Bushman (2008) reviewed convincing evidence that narcissism has clearly been on the rise over the past few decades in the United States. Other findings reveal that contemporary youth in this country display narcissistic over-confidence. Campbell, Goodie, and Foster (2004) reported findings indicating that today's students have markedly higher and more unrealistic educational expectations and aspirations for success. By way of illustration, slightly more than half of recent high school students predicted that they would earn graduate or professional degrees, even though only 9% of 25- to 34-year-old high school graduates actually obtain such credentials.

Thus, narcissism has become a societal cause for concern. Cultural pundits observe that a sense of *entitlement* among our youth is on the rise. Furthermore, it would seem that increasingly greater numbers of parents and educators are co-conspirators in bolstering unrealistically positive perceptions of the competencies and the overall self-worth of their children and adolescents. For example, the national frenzy to enhance the self-esteem of our youth is apparent within schools systems as well as around the family dinner table. (See Harter, 2012, for an extensive treatment and critique of the *self-esteem movement*.)

Recently (Harter, 2012), I shared my concern over the costs of the contemporary preoccupation with enhancing if not aggrandizing the self among adults in our culture (see Leary, 2004), which is descending the developmental ladder. Self-enhancement strategies, self-serving biases, the inflation of self-esteem, unrealistic self-appraisals, and narcissistic illusions of omnipotence are all pernicious processes that compromise personal authenticity and jeopardize interpersonal relationships. In the present chapter,

I have addressed three developmental questions for each stage. First, what *needs* or motives, if any, are there to engage in self-protective and self-enhancing strategies? Second, what *skills* exist that allow one to execute such biases? Third, what are the consequences? Several needs and skills emerge in late childhood and proliferate during adolescence. Thus, there is cause for concern, not only for adults but also for older children and teenagers who may be driven to engage in self-serving strategies that compromise personal authenticity and relationships with others. Unfortunately, this only taxes the already harried I-self to keep pace in its efforts to falsely package the me-self so that it will be socially acceptable and presentable to the world at large.

SOCIALIZATION PRACTICES THAT CAN COMPROMISE JAMESIAN I-SELF FUNCTIONS

I-self cognitive processes do not develop in a vacuum. Benevolent parents, positive childrearing practices can certainly facilitate the developmental trajectory that has been identified. However, negative socialization practices, such as conditional support, where children are instructed in the psychological hoops they must jump through to win parental approval and love, backfire. Instead, they instill false self behavior in children who try to please parents and meet *their* agenda. Moreover, severe neglect and abuse, can compromise the emergence of these positive I-self functions at each developmental level.

Chronic and severe abuse typically provokes a variety of *dissociative* symptoms in which there is a massive attempt to protect the psyche by efforts to eliminate the abusive events from consciousness. These psychological efforts serve to numb the pain and anxiety leading to psychogenic *amnesia* that interferes with the victim's lack of *awareness* of self (Brier, 1992; Cicchetti & Toth, 2006). The defensive structures driven by abuse will also compromise the victim's sense of *agency* (Putnam, 1993; Westen, 1993). The dissociative symptomatology that is defensively mobilized by the abuse victim seriously interferes with a *sense of coherence*, the consolidation of features of oneself that would be experienced as integrated (Briere, 1992; Fischer & Ayoub, 1994; Putnam, 1993; Westen, 1993). Thus, all of the Jamesian I-self functions are compromised by negative socialization practices, the most severe of which is chronic abuse. A comprehensive treatment of this topic, including an extensive review of the extant literature on the effects of abuse, with illustrative examples, can be found in Harter (1999, 2012).

CROSS-CULTURAL DIFFERENCES IN SELF-ENHANCEMENT

The self-enhancement strategies and self-serving biases, while not unique to Americans have been found to be more prevalent among North Americans, compared to those in Eastern, Asian cultures. Americans are notorious for not only viewing themselves positively but also making *unrealistic* evaluations of their positive attributes (Cross & Gore, 2003; Heine, 2007; Heine, Lehman, Markus, & Kitayama, 1999). This self-enhancing style takes other forms, as described earlier. For example, Americans take credit for their successes but attribute failures to external or situational factors. Another tactic is to make downward social comparisons, highlighting the less adequate performance of others, thereby looking more competent. A meta-analysis by Mezulis, Abramson, Hyde, and Hankin (2004) reveals strong tendencies for Americans to demonstrate a self-serving, attributional bias, namely, making more internal, stable, and global attributions for positive, compared with negative, personal outcomes. Asian samples, in contrast, did not show such biases. The authors concluded that there is no *universal* attribution positivity bias.

These self-enhancement strategies more common in Western cultures can be contrasted with the *self-effacing* tendencies among Eastern cultures that involve *modesty* and *self-criticism* toward the goal of *self-improvement*. Heine (2003) defined self-enhancement as the tendency to dwell on, elaborate, and exaggerate positive aspects of the self, relative to personal weaknesses. In contrast, self-effacing tendencies and self-improvement involve the tendency to dwell on, elaborate, and exaggerate *negative* aspects of the self, relative to personal strengths, in an effort to correct perceived shortcomings (see also Kanagawa, Cross, & Markus, 2001).

In another meta-analysis of published cross-cultural studies on self-enhancement (Heine & Hamamura, 2007), pervasive and pronounced differences between East Asians and Westerners were revealed. Westerners demonstrated a clear self-serving bias whereas East Asians did not (with Asian Americans falling in between). This evidence converges with other findings indicating East Asians typically do not self-enhance.

The vast majority of cross-cultural analyses have focused on the Japanese culture. However, others have addressed these issues in South Korea and China. Kim, Kim, Kam, and Shin (2003) examined self-enhancing versus self-effacing tendencies among Koreans. College students were read two scenarios, one in which the protagonist

was bragging about positive accomplishments and a second in which the main character emphasized negative self-perceptions. The results revealed that the negative self-presentation was judged more favorably than the positive self-presentation, suggesting the role of self-effacement.

Wang and Ollendick (2001) have reviewed literature revealing self-effacing tendencies observed in Chinese culture. Their findings highlight the value that Chinese society places on modesty and harmonious social relationships. They argue that self-effacing behavior will be more likely to obtain favorable social responses from others that, in turn, will serve to maintain harmony. In contrast, the self-enhancing tendencies observed among European Americans function to support an elevated sense of self-esteem.

Wirtz and Chiu (2008) offered an interesting perspective on the type of *ego* that differentiates those in Western and Eastern cultures. They describe the self-enhancing strategies and self-serving biases among Westerners in terms of Greenwald's (1980) concept of the *totalitarian ego*. Greenwald has likened these self-protective and self-promotional tendencies to a controlling political state. Just as a totalitarian state exercises control of the media, relies on propaganda, and regulates political life, the self devotes effort toward information control and self-serving personal propaganda through a variety of self-enhancing cognitive biases. According to Greenwald, egocentrism, illusions of control, self-serving biases, and the manipulation of personal memory all operate to glorify the self. They review literature suggesting that the self in Eastern cultures is more restrained or subdued. These suggest that the Eastern self, in contrast to the totalitarian ego, constitutes a more reserved representation that has been labeled the *quiet ego*. This theme is revisited in a concluding section of this chapter.

Cross-cultural differences in *autobiographical memory* parallel the distinctions raised above. For Americans, the content of memory focuses on distinction and personal accomplishment, whereas autobiographical memory in Eastern cultures emphasizes connectedness and interpersonal relationships. Leichtman, Wang, and Pillemer (2003) summarized findings that adults who have grown up in individualistic, Western cultures recount earlier, lengthier, and more detailed childhood memories than do adults who have grown up in more collectivist cultures. American parents support their children's autobiographical narrative accounts that underscore personal preferences, needs, judgments, opinions, successes, and personal control compared to those of children from Korea and China (see also Wang & Leichtman, 2000). These researchers further documented, in the stories of Chinese children, the greater orientation toward social engagement, concern with moral correctness, respect for authority, and themes of responsibility. Thus, there is considerable evidence for cross cultural differences in what can broadly be characterized as self-enhancing tendencies displayed by Westerners in contrast to the self-effacing strategies manifest in Easterners leading to different behavioral stances. Westerners, on average, emphasize the individualistic goals of personal success whereas Easterners, on average, adopt a stance toward harmonious social relationships.

Interestingly, despite these cross-cultural differences, a recent study reveals that narcissism is on the rise among Chinese youth (Cai, Kwan, & Sedikides, 2012), just as in the United States, though it is still higher in this country. More specifically, those from higher socioeconomic classes, those from one-child-only families (given the political edict, a generation ago), and those reporting individualistic values scored higher on measures of narcissism. Social-cultural changes (e.g., the influence of capitalistic economic practices, the one child per family legislation) have contributed to the rise of narcissism among certain Chinese, according to the authors. Other observers have pointed to how four doting Chinese grandparents vie for the attention of a single grandchild, which may lead to children's greater sense of exaggerated self-esteem and entitlement.

SELF-EVALUATIONS IN GLOBAL SELF-ESTEEM AND SELF-CONCEPT: I-SELF AND ME-SELF FUNCTIONS

The I-self is also responsible for another me-self construction in the form of an overall evaluation of one's worth as a person, typically referred to as global self-esteem or self-worth. Unfortunately, many children and adolescents report distressingly low levels of self-esteem. However, from a cognitive-developmental perspective, such a representation is not within the child's repertoire until approximately age 8. That is, it cannot be verbalized in young children.

Young children do exude self-esteem in their behavior, what we have labeled *behaviorally presented self-esteem* (see Haltiwanger, 1989; Harter, 1999). Teachers report that preschoolers who have high self-esteem display confidence, curiosity, initiative, and independence. In addition, they react adaptively to stress, adjusting to changes and transitions, they tolerate frustration, they persevere, and they are able to handle criticism and teasing. Those children who manifest low self-esteem in their behavior rarely display these positive features and have difficulty in reacting to

change, stress, and frustration. An interesting question is whether young children with low behaviorally presented self-esteem also report low self-esteem when they are older and can verbalize their worth as a person. If the negative parenting practices leading to low self-esteem *persist*, for example, lack of affection, conditional support, neglect, excessively punitive discipline, and abuse (see Harter, 1999), then one would expect continuity over time. That is, an individual who displayed low self-esteem in his or her behavior as a young child would also, as an older child report negative feelings of worth on self-esteem questionnaires.

This ability to form a linguistic concept of global self-esteem does not emerge until about the age of 8 (see Harter, 1999). Two historical scholars originally addressed the potential causes of verbalized self-esteem in adults. James's (1892) formulation focused on the cognitive prerequisites in that perceived success in domains deemed important is weighed against perceptions of actual accomplishments. If perceived successes are congruent with one's pretensions, then high self-esteem will ensue. In contrast, if perceptions of success fall short of one's aspirations, then low self-esteem will be experienced. For Cooley (2002), who proposed a looking-glass self model of self-esteem, the internalization of the opinions of significant others, as social mirrors, dictated the level of global self-esteem. However, these processes do not emerge until middle childhood.

Our own research (see Harter, 1999, 2012) finds strong support for the formulations of both James (1892) and Cooley (2002). We first identified age-appropriate domains, namely, scholastic competence, athletic competence, social competence, physical appearance, and behavioral conduct, assessing older children perceptions of competence or adequacy in each domain. To examine James's theory, we also asked older children to report on the *importance of success* in each domain. In this manner, we could determine if there was a discrepancy between perceived competence or adequacy and the importance of success in each domain. The high self-esteem child is not necessarily a super-star in all domains. Rather, the I-self is able, as James hypothesized, to *discount* the importance of success for domains in which one does not feel competent or adequate. In contrast, the low self-esteem child is unable to discount the importance of success for domains in which he or she feels inadequate. We will return to why the I-self is unable to engage in this discounting process, leading to very negative and debilitating self-evaluations for some individuals.

We have also documented the fact that Cooley's (1902) looking-glass self model of self-esteem is alive and well, in the lives of older children and adolescents. Self-reports of approval from parents, as well as classmates, are highly predictive of global self-esteem (Harter, 1999). Older children with high self-esteem provide favorable accounts of the positive support and approval they receive. Of more importance to this chapter are the self-reports of low self-esteem children who experience lack of social support from parents and peers, reporting low approval as well as overt rejection. Moreover, those who report *conditional* support from parents and peers also suffer in terms of low self-esteem. Thus, if parents make their love contingent upon unrealistic demands on their children, this practice will erode the child's self-esteem, as our findings have documented (Harter, 1999).

Self-esteem becomes more nuanced in early adolescence, with implications for those who evaluate their sense of worth unfavorably. The term *self-esteem* becomes salient in the young adolescent's vocabulary. As one adolescent participant concluded, "It's a big deal to show that you have high self-esteem." Moreover, young teenagers begin to form meta-theories about the causal nature of constructs that involve themselves and others. For example, they form meta-theories about the directionality of their perceived attractiveness and self-esteem and about the causal links between peer approval and self-esteem.

From a Jamesian (1892) perspective, perceived adequacy in domains of importance strongly predicts self-esteem, as young adolescents come to appreciate (Harter, 1999). A preoccupation with the congruence or discrepancy between ideal and real self-representations intensifies during early adolescence. Moreover, perceptions of physical appearance are particularly salient in predicting overall worth (see Harter, 2012). The majority of young adolescents admit that they are basing their self-esteem on their perceived appearance. Those whose self-esteem is dependent on meeting the punishing societal standards report lower perceptions of their appearance, lower global self-esteem, and greater feelings of depression (see Harter, 1999). As one young adolescent admitted, "How much I like myself as a person depends on how I look. I'd like to be good looking, like the models and the TV and movie stars, but that's impossible. I'm too short and I weigh too much."

Although perceived approval is an important determinant of one's level of self-esteem, young adolescents who are *preoccupied* with the importance of the opinions of peers are also at risk (Harter, Stocker, & Robinson, 1996). They admit to greater *fluctuations* in both approval and self-esteem, lower *levels* of approval and self-esteem, and are more distracted in the classroom. As one adolescent described it, "How much I like myself really depends on

what other kids think about me, I have to admit it. One day, like if I get invited to a party, I'll feel great about myself. But then the next day, if somebody I know ignores me in the hall or doesn't say something nice, then I feel terrible about myself. Sometimes I think too much about this during my classes and I don't concentrate on what the teacher is saying." These adolescents, drawn like magnets to the social mirror, are also more likely to report that their lack of support leads to their being depressed, a topic to which we shall return. Fast-forwarding to adulthood, Cambron, Acitelie, and Petit (2008) reported that women who base their self-esteem on interpersonal relationships (friendships, romantic involvement) are more at risk for depression.

Those in middle-to-late adolescence face different potential assaults to their self-esteem that become more differentiated in terms of the various social sources of approval that impact one's sense of personal worth. Contradictory messages from different significant others can lead to self-esteem volatility, what Rosenberg (1979) called the *barometric self*. Differential approval across roles will also lead to different levels of role-specific self-esteem in each context, what we termed *relational self-esteem* (Harter, Waters, & Whitesell, 1998). As one adolescent interviewee explained, "How much I like myself as a person goes back and forth, my self-esteem is higher with kids who know me than with my parents," a pattern that our findings have documented. Rosenberg's concept of the barometric self refers to volatility in the overall *level* of *global* self-esteem. Because adolescents report that their self-attributes, as well as their self-esteem, vary across relationships, we have coined the term the *kaleidoscopic self* to capture this greater complexity.

Relational self-esteem with *classmates* is the most predictive of global self-esteem (Harter, Waters, & Whitesell, 1998). As one adolescent observed, "What all of the other kids think of you is so important to how much you like yourself as a person." As reported elsewhere (Harter, 1999), the approval of close friends is not as highly related to self-esteem where there is less variability (scores are uniformly high given that, by definition, a close friend likes you). The perceived approval from classmates presumably reflects a more objective or credible index of validation from those whom Mead (1934) referred to as *the generalized other*. Those who cannot garner the support of this general body of peers will suffer greatly, in terms of low relational self-esteem with peers as well as global self-esteem.

Normatively, global self-esteem declines between early to midadolescence (see Harter, 1999). The increased penchant for *introspection*, an I-self function, can contribute to lowered self-esteem by facilitating the contrast between one's *real* and *ideal* self-concepts. This produces a heightened awareness of the *discrepancy* between the real self (e.g., I can get pretty obnoxious and intolerant) and one's ideal (e.g., I'd like to be friendly and tolerant all of the time, that's the kind of person I *want* to be, and I'm disappointed in myself when I'm not). Thus, consistent with James's (1892) formulation, the more one's aspirations or ideals outstrip perceptions of one's actual capabilities across different domains, the lower one's self-esteem.

The realm of physical appearance is particularly critical (see Harter, 1999, 2012). Perceived attractiveness looms large in importance, yet most cannot meet the punishing societal standards so salient in our culture. The I-self, as cognizer, also looks outward and is well aware of the cultural preoccupation with attractiveness, as well as its criteria. The demand that females be excessively thin, tall, long-legged, with ample breasts and a pretty face, framed in flattering hair, is an ideal that 95% of females in this culture cannot attain (Kilbourne, 1999). As a result, aspirations far outweigh perceived appearance, contributing to lower self-esteem (see Harter, 2012). One female adolescent participant admitted that she wants to look like Beyonce but knows she doesn't and never could, and this sets up a painful discrepancy. In reality, she falls far short of the cultural standards for beauty. Similar dynamics are emerging for males who attempt to emulate the cultural criteria of male attractiveness that now include height, muscularity, rippled abs, and great hair (see Harter, 2012; Kiang & Harter, 2006). These issues continue into late adolescence and beyond, given our societal cultural obsession with attractiveness and the impossible criteria to which both females and males are subjected. We will return to the implications of this cultural preoccupation, in the subsequent section on eating disorders.

WHY CAN'T THOSE WITH LOW SELF-ESTEEM ALTER THE PERCEPTIONS OF THEIR OVERALL LACK OF WORTH?

Our model of the determinants of global self-esteem documents the fact that both the Jamesian (1892) formulation, emphasizing the discrepancy or congruence between perceptions of success in domains deemed important, and Cooley's (1902) looking-glass self theory of the role of approval from significant others, both contribute to self-esteem. Thus, there seem to be two clear pathways to increasing the self-esteem of those who suffer from

negative self-evaluations of their worth. First, one needs to reduce pernicious discrepancies. Second, one needs to find niches in which to garner support. These strategies have broader implications for those engaged in intervention efforts to enhance self-esteem, and I have suggested some possible pathways toward such a goal (Harter, 1999). Yet there are reasons to realistically question even my own former optimism.

James (1892), a brilliant theorist, was quite sanguine about how one might increase one's self-esteem. He postulated two potential routes to self-esteem enhancement. One could lower one's aspirations by discounting the importance of success in areas where one was deficient. Alternatively, one could raise one's level or actual competence or personal adequacy (e.g., through skills training or other efforts to overcome one's limitations). Both of these strategies will serve to reduce the discrepancy between perceived competency or adequacy and the importance of success; decreasing this discrepancy should, in turn, increase one's level of self-esteem.

Although such interventions are theoretically appealing, they may not be that plausible given the realities of the lives of children and adolescents in our contemporary culture. There are two psychological roadblocks to discounting the importance of the specific domains we have selected, in the face of perceived deficiencies. First and foremost, these domains were initially chosen because most children and adolescents, in interviews, identified these arenas as extremely important. That is, we are living in a society where scholastic competence, athletic competence, social competence, physical attractiveness, and appropriate behavioral conduct are highly valued and are sought after by the majority of youth in the cultural mainstream. Thus, personal aspirations and standards in these domains are typically quite high, making it difficult for those feeling inadequate to discount their importance. The domain of physical appearance is particularly hard to discount.

Second, these domains are highly valued by significant others, notably parents and peers. Our own findings reveal that children and adolescents report that scholastic competence and behavioral conduct are extremely important to parents. In contrast, social competence, physical appearance, and athletic competence are judged to be particularly important to peers. Moreover, importance to others is highly correlated with importance to the self; competence/adequacy in domains important to others is just as highly correlated with self-worth as is competence in domains important to the self. Thus, it would appear to be extremely difficult for children and adolescents to discount the importance that represents standards set by significant others whom they wish to please (see also Baumeister, 1993). The inability to discount these aspirations is particularly debilitating for those who acknowledge deficiencies in these areas. Moreover, limitations in these domains will also make it difficult to garner the support of parents and peers, also so critical to maintaining positive self-esteem.

Increasing one's competence and corresponding self-evaluations as an alternative strategy for decreasing the discrepancy between competence/adequacy and the importance of success would also appear to be problematic for many youth. First, there are undoubtedly natural limits to the extent to which children and adolescents can increase their actual competence or adequacy. Although a given individual may be highly motivated to improve his or her scholastic performance, athletic ability, or physical appearance, there may be little that one can realistically do, given certain ceilings on intellectual and physical potential.

Moreover, there may also be limitations placed on perceptions of competence/adequacy given the standards imposed by social comparison, an I-self process by which the me-self is compared with others. The process of comparing the self to others begins in middle childhood and increases with development, as most children adopt the cultural preoccupation with how individuals differ from one another, with competition, with who is the best, and who ascends to the top of ladder. Thus, how one measures up to peers and societal standards becomes the filter through which judgments about the self pass. Thus, even if motivated, most individuals will fall short, relative to the punishing peer standards that provide the metric for self-evaluation. As a result, it is difficult for many to reduce the discrepancy between perceived competence/adequacy and the importance of success, hampering the desire to enhance one's self-esteem (see Harter, 1993). Thus, debilitating low self-esteem persists.

Baumeister (1993), in his book *Self-Esteem: The Puzzle of Low Regard*, presents other explanations for why low self-esteem is so resistant to change. Moreover, Swann's (1996) *Self-Verification Theory* provides an alternative interpretation for why negative self-evaluations are so intractable. People, Swann argues, are powerfully motivated to retain their self-perceptions, even if negative, because they provide a road map for what to expect from others and how to behave. Feedback to the contrary, even if positive, is psychologically distressing and disruptive because it derails people from their life's agenda. Thus, low self-esteem is likely to persist because the I-self has come to consistently define the me-self. This discussion has focused on the determinants or antecedents of self-esteem, addressing the causes of low self-esteem. We next turn to

cross-cultural differences in self-esteem. Following this topic, we address correlates and consequences of low self-esteem, including depression, suicidal behaviors, eating disordered behaviors and humiliation, in the face of severe peer harassment, leading to violent revenge.

CROSS-CULTURAL DIFFERENCES IN SELF-ESTEEM

There are clear differences between Western and Eastern conceptions of self-esteem that are critical to recognize, before Westerners encourage Eastern cultures to adopt their frameworks, paradigms, and instruments. Clearly, self-esteem is a value touted by Americans, as witnessed by the many attempts to protect and enhance it, as has been demonstrated. However, high self-esteem, based on individual accomplishments, is not revered as a psychological commodity in most Eastern cultures. Rather, harmonious relationships represent the primary sources of self-respect (Diener & Diener, 2009; Kang, Shaver, Sue, Min, & Zing, 2003). Kang et al. further reported that high self-esteem predicted *life satisfaction* for European Americans but not for Koreans and the Chinese. Rather, *relationship quality* was found to be the path to life satisfaction for these Asian groups (but was not highly predictive for Americans).

These findings relate to an interesting linguistic cultural observation by Wang and Ollendick (2001), who studied self processes in China. In the Chinese language, there is no equivalent term for self-esteem! The same is true for Japanese. This would appear to reflect the point above, namely, that self-esteem is not a core value and therefore is not represented or codified in their respective languages.

Yet this has not deterred American psychologists from subjecting Asian children, adolescents, and adults to instruments designed for US culture, assuming that such measures are useful and can be meaningfully interpreted. We cannot make such assumptions (Harter, 2012). Wang and Ollendick (2001) concurred, arguing that this approach is problematic because investigators assume that the construct of self-esteem has the same meaning in China, compared with the United States, leading to findings that are potentially misleading and inaccurate.

This conclusion was borne out by a colleague, Professor Lee C. Lee (personal communication, April 15, 1987) who employed our global self-esteem subscale with first-generation Chinese immigrant children who came to New York. Lee indicated to me that these items lacked meaning for her sample, concluding that the concept of global self-esteem, as defined in American culture, was not appropriate to include on an instrument administered to Chinese children.

Others have found the psychometric properties of our Self-Perception measures to be inadequate. Meredith, Abbott, and Zheng (1991) reported that only 20 of the 36 items (almost half) on our Self-Perception Profile for Children (Harter, 1985) factored appropriately in a Chinese sample. Similarly, in both this and another Chinese sample employed by Stigler, Smith, and Mao (1985), the reliabilities were unacceptably low. Moreover, in both studies, the Global Self-Esteem was particularly problematic. Culturally, inappropriate *content* on a given instrument will invariably contribute to an inadequate scale *structure* and to unacceptable psychometric properties.

A further concern inheres in the fact that for the Chinese (Meredith et al., 1991; Stigler et al., 1985) and for Japanese (Maeda, 1997; Sakura, 1983) and for Koreans (Rhee, 1993), the mean domain-specific self-concept scores and the Global Self-Esteem ratings were considerably lower than scores in the United States, Canada, Australia, and Western Europe samples. There are several interpretations for this pattern. First, the Eastern Asian penchant for modesty and self-criticism, part of their self-effacing orientation, would predictably lead to lower scores. The contrasting American style of self-enhancement with its positive bias would ensure relatively high scores.

Another explanation for the more negative self-evaluations of Asian samples can be traced to our structured-alternative question format (Harter, 1985). This particular question format (see Harter, 2012) invites if not requires some degree of social comparison with one's peers. Stigler et al. (1985) indicate that overt social comparison is frowned upon in Chinese culture, where individual differences in competence or ability are downplayed, in the service of social harmony. Thus, Chinese children's unwillingness to report that they may be superior to others should lead to a pattern of scores that may not reveal their private perceptions of personal competence.

Thus, the inappropriate content of a Western instrument, the lack of meaningful items, a problematic question format (with our own measures that pull for social comparison), self-effacing or self-critical response tendencies, and inadequate psychometric properties all conspire to make these measures methodologically inappropriate and culturally insensitive. Other thoughtful investigators (e.g., McCrae, 2001; Tsui, Nifadkar, & Ou, 2009) have pointed to problems in translation, including the typical back-translation procedure. See Harter (2012), where these different issues are reviewed in more detail and where it is urged that those in different cultures take stock of these

problems and perhaps initially design interview questions to address their specific research questions and hypotheses. Responses may then evolve into a culturally sensitive measure that would directly bear on their an appreciation of their own cultural values and yield more meaningful results. Using cultural standards, establishing cutoff scores for what constitutes pathology, would be preferable for normative and applied research.

A final criticism of much cross-cultural research is that too many studies are conducted with samples of convenience, namely, university students. Such students, albeit in their culture of origin, are not representative community samples. As McCrae (2001) thoughtfully pointed out, undergraduates may not be accurate proxies for more nationally appropriate samples that are representative of age, socioeconomic status (SES), education, and experience in a given culture. Watkins, Mortazavi, and Trofimova (2000) echoed this concern. The dangers of generalizing from a single group such as college students became evident in their own research. Moreover, they make another important point: University students around the globe have been westernized in many respects, they may be wrestling with or rejecting the cultural values of their family of origin and are a poor choice for cross-cultural research, because they are not representative of their culture at large.

Thus, there are clear challenges in the assessment of self-constructs across cultures. The field must attend to these critiques and seek solutions to the problems plaguing assessment procedures as well as the selection of representative cultural participants. Solving these dilemmas is critical to provide a window through which we can observe cultural differences that are both accurate and meaningful. However, more important to this chapter is a focus on what self-esteem processes might be pathological, leading to low self-esteem, and related symptoms of depression and eating disorders, if not other manifestations. We, they, need to employ culturally sensitive definitions for both normative, including transient liabilities, and pathological conditions that may involve the self, culturally defined.

DEPRESSION AND SUICIDAL BEHAVIORS

In the previous section, we reviewed the causes or antecedents of global self-esteem, which focused on perceptions of competence/adequacy in domains deemed important, as well as approval from significant others. However, if the I-self's evaluations of the me-self do not directly impact an individual's day-to-day functioning, then an understanding of such representations will contribute little to our comprehension of the behavior of children and adolescents. Why should we care about self-esteem unless we can demonstrate that it plays a vital role in individuals' lives? The efforts of self theorists and researchers may be misguided if self-research reduces to a cognitive epiphenomenon that has little impact on everyday functioning.

Considerable research now demonstrates the powerful link between self-esteem and depression (Harter, 1999). There is clear historical precedent for including negative self-evaluations as one of a constellation of symptoms experienced in depression, beginning with Freud's (1952) observations of the low self-esteem displayed by adults suffering from depressive disorders. Others within the psychoanalytic tradition have continued to afford low self-esteem a central role in depression (Bibring, 1953; Blatt, 1974).

Numerous theorists and researchers to follow have addressed the manifestations of depression in children, adolescents, and adults, focusing heavily on cognitive components involving the self (see Abramson, Metaksky, & Alloy, 1989; Baumeister, 1990; Beck, 1975; Hammen & Goodman-Brown, 1990; Higgins, 1989; Kovacs & Beck, 1986; Nolen-Hoeksema, Girus, & Seligman, 1986; Pyszczynski & Greenberg, 1987; Seligman & Peterson, 1986). In our own studies (see Harter, 1999) we have consistently found that among older children and adolescents, self-esteem is highly related to affect along a continuum of cheerful to depressed (with correlations ranging from .72 to .80). These findings are consistent with those of other investigators (Battle, 1987; Beck, 1975; Kaslow, Rehm, & Siegel, 1984).

More recent findings confirm the strong relationship between negative self-evaluations, low self-esteem, and depression (Berg & Klinger, 2009: Lee-Flynn, Pomaki, DeLongis, Biesanz, & Piterman, 2011: Robles-Pina, DeFrance, & Cox, 2008; Montague, Enders, Dietz, & Canendish, 2008: Sadhwani, 2012; Schwartz, Klimstra, Luyckx, Hale, & Meeus, 2012; Sturman, Flett, Hewitt, & Rudolph, 2009). Among clinic samples of inpatient adolescents with psychiatric diagnoses of depression, we have also documented a powerful link between self-esteem and self-reported depressed affect (Harter, Marold, & Whitesell, 1992). Moreover, these effects are exacerbated in those who have been the victims of sexual abuse (see Turner, Finkelhor, & Omrad, 2010).

Our own findings, consistent with those of others, led to the development of a model of the causes (competence/adequacy in domains of importance in addition to peer and parental support), correlates, and consequences

of self-esteem (Harter, 1999; Harter, Marold, & Whitesell, 1992). Initially we postulated that self-esteem served as a mediator between the causes of global feelings of worth and depression. However, it became apparent that the exceedingly high correlation between self-esteem and depressed affect did not permit a clear interpretation of the directionality of these two constructs. Moreover, when we added *hopelessness* to our battery of measures, following the lead of Kovacs & Beck (1986), we found it to be equally correlated, at high levels, to both depression and self-esteem. Thus, for the purpose of statistical modeling, we combined self-esteem, depression, and hopelessness into what we labeled the Depression/Adjustment Composite. This composite, in turn, was predictive of *suicidal ideation,* as an outcome. Baumeister (1990) has postulated that many suffering from depression and its correlates are driven to consider suicide as a solution, as a form of escape from the painful cognitions and affects about the self and the disapproving reactions and rejection by significant others.

More recent studies have also documented the strong link between depression and suicidal behaviors (see Au, Lau, & Lee, 2012; Santos, Saraiva, & DeSousa, 2009). Others have examined the specific components or symptoms of depression and their relationship to suicidal behaviors. For example, core mood disturbance symptoms (i.e., depressed affect) and self-punitive thinking have been found to be the major predictors of suicidal ideation (Keilp et al., 2012).

The incidence of suicide among adolescents has tripled in recent decades, leading to efforts to identify the determinants of this major mental health threat to our youth (see Noam & Borst, 1994; Pfeffer, 1988). Several broad classes of risk factors implicated in depression and suicidal behavior have captured the attention of researchers, including biological precursors, epidemiological correlates, and social/psychological stressors. Our own model has focused on an examination of this third class of risk factors, consistent with the emphasis and urging of others (see Spirato, Brown, Overholser, & Fritz, 1989).

The following poignant self-disclosure by one of the adolescents in our studies provides a personal cameo of the model that has evolved in our research:

> I look in the mirror and most days I don't like what I see, I don't like how I look. I don't like myself as a person. So I get depressed, bummed out. Plus, my family has rejected me and that makes me feel pretty lousy about myself. My mother is really on my case because I'm not living up to what she wants me to be. If I get A's in school, she's nice and is proud of me but if I don't she doesn't approve of me. You could say that how she treats me is conditional on how I do. Mostly she tells me I am a failure and I am beginning to believe it. Doing well in school has always been important to me, but now I feel like I'll never amount to anything. There's no way I am ever going to be able to please her, it's pretty hopeless. I don't get much support from other kids, either. I probably never will because I'm an introvert. I don't even try to make friends. So a lot of the time I get depressed. When I am depressed I feel very sad because other people have hurt me, but I'm also angry at them, for not caring and for rejecting me. I feel so depressed that I often think about just killing myself. Life is worthless, but so is death. So what's the use?

Different Pathways to Depression

Although most of the components of our model appear in this adolescent's personal vignette, not all depressed adolescents report all of the precursors of depression we have identified. Inspection of hundreds of profiles has revealed six different routes to the depression composite (see Harter, 1999; Harter & Whitesell, 1996). A central tenet for those studying psychopathology is that there are multiple pathways to a given disorder (Cicchetti & Rogosh, 1996; DeKlyen & Greenberg, 2008). We first identified six subgroups, all of whom reported low self-esteem, depressed affect, and hopelessness. One subgroup does report all of the predictors in the model, reporting incompetence/inadequacy in domains that are viewed as important to peers (physical appearance, social competence, and athletic competence) and important to adults (scholastic competence and behavioral conduct). They also reveal corresponding lack of approval from both peers and parents.

A second subgroup reported inadequacy in the domains judged important to both peers and parents, as well as lack of parent support. Children from this group that we interviewed seemed to be in denial about the lack of acceptance from peers, for a variety of reasons (see Harter, 1999). A third group acknowledged lack of competence/inadequacy in the domains deemed important to peers and parents, as well as low peer support. However, they did *not* report lack of parent support, presumably, interview data revealed, because they had benevolent parents who provided unconditional love, accepting their child's deficiencies; in some cases, they were congenitally caused.

A fourth pathway to the depression composite (low self-esteem, depressed affect, and hopelessness) was characterized by negative perceptions in domains important to peers only, with accompanying lack of peer support. These individuals reported positive self-evaluations in the domains of scholastic competence and behavioral conduct and appeared to garner the approval of parents.

However, interviews revealed the greater importance of the peer culture and the need to meet with the approval of classmates, which they were not garnering because of their perceived inadequacies, and this overshadowed success in the domains that were rewarded by parents.

A fifth pattern was manifest by those who did report success in the peer-important domains, accompanied by peer support, but who were nevertheless more focused on their lack of adequacy in the domains important to parents, scholastic competence and behavioral conduct, leading to depression. Interviews indicated that for some, placement in special education classes because of either scholastic or behavioral difficulties, accompanied by lack of approval from parents, were largely responsible. The sixth and last pattern was initially puzzling. These depressed youth reported low levels of success in both the domains important to peers and parents, yet surprisingly they felt the approval of both peers and parents. Interviews revealed that these individuals were extremely self-critical, with very high and unrealistic standards for their own behavior, and did not feel that the approval of others was credible (see Blatt's, 1995, discussion of self-critical depression). The identification of these six patterns has implications for those involved in intervention efforts to ameliorate low-self esteem, depression, and hopelessness. Given that there is not one pathway to these manifestations, it behooves us to identify individual differences in the specific causes leading to depressive behaviors.

Depression as a Combination of Sadness and Anger

Another line of our research has examined the phenomenological experience of depression as a combination of sadness and anger, consistent with the clinical literature, which points to the *comorbity* of internalizing and externalizing symptoms (e.g., Rutter, 1989). Moreover, it is also congruent with our own research on mixed or multiple emotions (reviewed in Harter, 1999). Our studies reveal that the vast majority of adolescents (80%) report that depression involves the co-occurrence of sadness and anger. However, a key question became anger at whom? The classic Freudian interpretation is that depression involves anger turned *inward* against the self (Freud, 1968). However, our own research has revealed that most of the causes that adolescents cite for being depressed involve actions of the others against the self. Correspondingly, they report that they are angry at *others* when they feel depressed. We have speculated that adolescents may not yet have developed the inhibitions or defenses that would prevent them from expressing their feelings toward the significant others whom they perceive to be the cause of their depression-related anger.

The Perceived Directionality of Low Self-Esteem and Depressed Affect

As described earlier, statistical modeling could not determine the directionality of self-esteem and affect, given that they were so highly correlated. This issue is reminiscent of the debate, in the 1980's, about the primacy of cognitions versus emotions (Izard, 1984; Lazarus, 1984; Zajonc, 1984). Our own solution was to go to the font of wisdom itself, the adolescent who experiences both low self-esteem and depression. We simply asked them: Which comes first? Do you first feel depressed and then don't like yourself as a person, or is it that you don't like yourself as a person and this makes you feel depressed? Our findings revealed two groups, one of which endorsed the first directionality orientation and another that endorsed the second orientation (see Harter, 1999).

Those for whom depression preceded low self-esteem described actions of *others* against self, revealing three categories of examples: (1) perceived rejection by others; (2) conflict with others; and (3) the experience of the *loss* of another (through their death or the termination of a relationship). Those for whom low self-esteem preceded feelings of depression cited dissatisfaction with particular *self-attributes* for three primary reasons: (1) negative evaluations of their appearance; (2) lack of competence (scholastic or athletic); and (3) dissatisfaction with their behavior or conduct in social interactions. In Harter (1999) we give many examples of the explanations of adolescents, for each of these orientations. For those working directly with depressed adolescents, we suggest, based on these findings, that it would be important to assess their perceptions of the directionality between self-esteem and depressed affect, since this will have implications for intervention. In conclusion, it is clear that there is a powerful, but complex link, between self-esteem and depression, indicating how the me-self is involved in this form of psychopathology.

EATING-DISORDERED BEHAVIOR

Our own research has identified one-self-concept domain that robustly affects global self-esteem across the life span, namely, perceived physical appearance. The I-self harshly judges the appearance of the me-self, which, in turn, negatively affects the evaluation of one's overall sense of esteem.

This link is profoundly impacted by cultural standards of appearance for each gender, given that the culture clearly touts physical attractiveness as the measure of one's worth as a person (see Harter, 1999, 2012). The empirical findings have revealed that the correlations between perceived attractiveness and global self-esteem range from the .60s to .80s, in our own studies. Moreover, investigators have demonstrated that these relationships are not merely statistical but are very much embedded in the consciousness of individuals well aware of this link.

In our own research (Kiang & Harter, 2006), we have found strong support for a model in which contemporary cultural values are highly endorsed. These values include the perceptions that being attractive will lead to higher self-esteem, that meeting societal standards of appearance will make people more popular, and that people who are overweight are discriminated against. However, there is enough variability in these scores to determine that such awareness predicts perceptions of one's own appearance, which in turn predicts level of self-esteem, depression, and eating-disordered behavior. Specifically, those endorsing these cultural values reported more negative views of their appearance, lower self-esteem, more psychological correlates of eating disorders, and more eating-disordered behaviors (see also Smith, 2010). Other studies with undergraduate females have indicated that when self-worth is based upon perceptions of physical appearance, particularly among those displaying vulnerable narcissism, such individuals are at risk for eating disorders (Gordon & Domback, 2010; Vartanian, 2009; Welch & Ghaderi, 2013).

Our own study (Kiang & Harter, 2006) was conducted with college students. However, the seeds of such a model are sown in childhood and adolescence. Children and teenagers of both genders are well aware of the prevailing norms for desirable appearance. For adult females in the 2000s, one must be tall, very thin, weigh very little (ideally around 110–115 pounds), and have ample breasts and of course a pretty face and stunning hair, an unattainable combination for more than 90% of the female population. The average American woman is 5 feet, 4 inches, tall and weighs 140 pounds. Standards have been extremely punishing for females across the decades.

Kim and Sundar (2012) concurred, observing that the discrepancy between one's bodily ideal and perceptions of one's actual bodily self is associated with both depression and eating-disordered behavior. They attribute the cause to intensified exposure to unrealistic images of others, for example, celebrities and media models (see also Bergstrom, Neighbors, & Malheim, 2009, on the media's impact on body image disturbance). Important to these dynamics is whether females *internalize* the thin ideal now so prominent in contemporary culture (Dittmar, Halliwell, & Stirling, 2009). Processes involving a *feared* body image, based on ideal-real discrepancies, also contribute (Woodman & Hemings, 2008).

Other recent research cites the role of *self-criticism* in eating disordered behaviors. In a study of adolescent females in Israel, self-derogation emerged as a strong predictor of eating-disordered symptoms (Fenning, Hadas, Itzhaky, Roe, Apter, & Shahar, 2008). In another investigation, bulimic symptoms in females were associated with a history of emotional and sexual abuse, leading to depressive symptoms and self-criticism, in the form of expressed bodily dissatisfaction (Dunkley, Masheb, & Grilo, 2010).

These standards are not lost on our children and adolescents. Children succumb to the same discouragement of not being able to emulate the looks of models, singers, and movie stars in the limelight. Meeting these exacting standards becomes particularly critical during early adolescence as teenagers face inevitable pubertal changes that signal their impending adulthood. Thus, they adopt adult standards as the physical markers for what defines attractiveness, appeal, and social acceptability, all of which determine one's self-esteem.

What is new, within the last two decades, is the fact that the bar has been raised for males in our society. No longer is appeal to be judged primarily by status, wealth, position, and power but by physical standards of attractiveness, as well. Muscular build, abs, biceps, physique, and hair on the head as well as face have all come to define the new ideals for men (see Harter, 2012; McFarland & Kaminski, 2009).

The genetic throw of the dice leads some young adolescent males and females to fare better than others on the competitive battleground, a minefield on the road to winning the attractiveness war. For example, early-maturing girls are at a distinct disadvantage given the current emphasis on thinness and height because on average they are typically heavier and shorter compared with later-maturing girls, who tend to be thinner and taller. The pattern is just the opposite for adolescent males. Early-maturing boys tend to be taller and more muscular, which gives them a physical edge. Thus, beginning in earnest at this age, evaluations of one's appearance take on critical implications for one's global self-esteem. The I-self is attentive to the cultural standards of attractiveness comparing the me-self to the ideal, which in turn determines one's level of self-esteem. Thus, those not meeting the gold standard are at serious risk for pathological forms of low self-esteem, depression, and possible suicide, as well

as for eating disorders that can be life threatening. While this preoccupation initially becomes quite salient in early adolescence, it continues throughout the life span.

Although the etiology of various eating disorders is quite complex and beyond the scope of this chapter, it is clear that appearance-related issues, body dissatisfaction, low self-esteem, and depression are implicated in eating disordered behaviors (see also Wertheim, Paxton, & Blaney, 2009). Moreover, others continue to emphasize the negative impact of cultural messages about appearance and body ideals (Smith, 2012). Woodruff-Burton (2012) provided a particularly interesting perspective on the cultural ideology of the consumer's image of the ideal or more desirable body. *Postmoderism* has supposedly offered the consumer freedom to choose from a wide range of cultural identities in self-expression. However, particularly for women, paradoxically, there is *little* flexibility or latitude in the contemporary ideals for feminine beauty. Malson (2009) expressed a similar concern, suggesting that the world would be a better place without mass-media saturation of ideals that dictate the pernicious effects of such images. She cites the hyperconformity to a culturally dominant, media-promulgated idealization of female slenderness and the culture's culpability in the production of eating disorders.

Our understanding of these processes is informed by the cognitive-behavioral therapy (CBT) model as it applies to eating disorders (Pike, Devlin, & Loeb, 2004). According to one CBT principle, the self-esteem of an individual with an eating disorder is overly determined by perceptions of body weight, shape, and appearance. In fact, one of the psychiatric diagnostic criteria for anorexia and bulimia is a disturbance in body image. Individuals with eating disorders imbue body weight, shape, and appearance with intense meaning and extreme importance and are acutely sensitive to minute changes in these dimensions, according to Pike et al., in their review of the literature. Weight fluctuations imperceptible to others can dominate the experience of those with eating disorders, having a significant effect on self-esteem and depressed mood.

In their studies of those with extreme eating disorders, Levine and Harrison (2004) found that the importance of weight and shape is significantly correlated with eating-disordered symptomatology. They, and others, argue that this pattern reflects the internalization of society values concerning appearance and the beauty ideal of thinness (Geller, Srikameswaran, Cockell, & Zaitsoff, 2000). Moreover, the overemphasis on weight and shape is motivated by the attempt to compensate for feelings of low self-esteem. Thus, for the individual with an eating disorder, the pursuit of thinness is the only pathway to enhancing self-esteem (see also Showers & Larson, 1999).

As Pike et al. (2004) observed, the societal values that make thinness a holy virtue, and beauty a ticket to happiness, are fundamental to the maintenance of an eating disorder. Achieving the cultural standards of attractiveness is thought to remediate the detrimental experience of low self-esteem. Thus, the fragile victims of eating disorders cling with tenacity to the hope that attainment of the ideal bodily self will enhance their flagging self-esteem. Anderson-Fye and Becker (2004) further observed a link between (desired) upward mobility and eating disorders, given the belief that social status will be enhanced by adhering to societal standards of beauty.

Concerns over weight and body shape continue to dominate more recent studies of eating disorders. For example, Serpell, Neiderman, Roberts, and Lask (2007) documented the fact that adolescent girls with eating disorders reported more negative evaluations of their weight and shape than those without such disorders. Geller et al. (2002) broadened their approach. In addition to shape and weight, they found that those adolescent females who based their self-esteem on intimate relationships displayed more eating-disordered symptoms and reported greater body dissatisfaction and more negative self-esteem. In a related study by Sanchez and Kwang (2007), females who derived their self-esteem from romantic relationships acknowledged their greater body shame that, in turn, predicted bulimic symptoms. In summary, there is clear evidence that appearance-related dissatisfactions and associated low self-esteem contribute heavily to the dynamics of eating-disordered behaviors in normative as well as clinical populations.

THE ROLE OF SELF PROCESSES, HUMILIATION, AND VIOLENT REVENGE

On a gloomy day, April 20, 1999, I turned on the afternoon news and was appalled to learn that 15 minutes from my Colorado mountain home, at Columbine High School, terrified students were being rushed out of the building, as 11 students lay dead, one teacher was dying, and many others teenagers were wounded. This was one of 12 high-profile school shootings between 1996 and 2005. The immediacy, the proximity, of this event spurred my own interest, as a psychologist, in the motives for such mass killings and caused me to study the histories of these several all-male, all-White, suburban or rural older children and teens; what would lead them to such atrocities? These were not

the acts of inner-city gang members from big cities or of identified conduct-disordered youth. Just what might be the dynamics of their behavior, many of us asked? Puzzling was the fact that the shootings seem to be indiscriminantly aimed at random targets, rather than specified individuals. Once the horror of it passed, we began to piece together a picture of what might be the psychological precursors of such tragic and desperate actions.

An examination of the media accounts of the school shooters made it obvious that many of the determinants of our model of self-esteem, and its consequences, could be found in the lives of these adolescents (see Harter, Low, et al., 2003). These youth had been the victims of peer rejection, harassment, taunting, and physical abuse. They did not display the attributes that would garner peer acceptance or parental support. Many reported rejection by parents, as well by peers. They seemed to suffer from low self-esteem, by the accounts of those adults who knew them, as well as depression. And they certainly did not appear to be hopeful about the future. Some were suicidal, three actually suicided, and a few others were prevented from suicidal attempts by police on the scene. It thus seemed to us that many of the dynamics in our model of the causes and consequences of self-esteem could be identified in the lives of these youth (see Harter, 2012). Others have documented that victims of bullying present more negative self-concepts than a control group (Houbre,Targuinio, & Lanfranchi, 2010). These authors also observed that victims experience low self-esteem, depression, and difficulty making friends.

Although our model addressed only *lack* of peer support, it became apparent that it was more serious—these young males actively experienced severe peer rejection. Other investigators (see Bellmore & Gillessen, 2006) also reported that adolescents perceived as victims of bullying and acute harassment came to view themselves as disliked by peers, perceived themselves as socially inadequate, and reported negative social self-concepts and low self-esteem (see also Ferrell-Smith, 2012; Jenkins & Denaray, 2012). Others (see McGee & DeBernardo, 1999; Leary, Kowalski, Smith, & Phillips, 2003) also described these same features; boys who were regarded as unattractive, socially inadequate, were loners, were cruelly bullied, teased, rejected, and ostracized, as a result.

Moreover, it has been documented that *ostracism* not only leads to depression but also causes individuals to lose their willingness to control their impulses (Crescioni & Baumeister, 2009; DeWall, Gilman, Sharif, & Rice, 2012). Other research (Denson, Dewall, & Finkel, 2012; Denson, Pederson, Friese, Hahn, & Roberts, 2011) also documented a link between lack of self-control and aggression. Similarly, Winstok (2009) reported that reactive violence among adolescent males is linked to low self-control capabilities. Recent findings also reveal that when faced with acute peer rejection, victims often aggress against the very peers from who they desire acceptance, leading to further rejection (Rejinjes et al., 2011).

Thus, as a first attempt at unraveling these dynamics, we expanded our model to include peer rejection, angry feelings of aggression, as well as violent ideation, in addition to suicidal ideation (Harter, Low, et al., 2003). Consistent with the clinical literature on the comorbidity of internalizing and externalizing symptoms, we found a correlation of $r = .55$, between suicidal and violent ideation toward others. Thus, the determinants and correlates in our model of self-esteem put adolescents at risk for endangering their own, as well as others' lives. The I-self, attentive to the social reactions of others to the me-self, incorporates these reactions and shapes the me-self accordingly. Thus, our most recent model of the causes and consequences of self-esteem and its correlates now includes peer rejection (not just lack of approval), anger (not just depression), and violent ideation (not just suicidal ideation). The model is displayed in Figure 11.1.

The Role of Humiliation

However, the story of the revenge that those who experience such victimization at the hands of peers who reject them does not stop there. The picture of the role of self processes also includes self-conscious *emotions*. One, in particular, was clear in the media accounts of the school shooters: as victims of bullying and harassment, they were all *humiliated*. Humiliation is an emotion that involves the debasement of the self by others, as a cause, and if the attacks against the self become chronic, the victim reacts with angry revenge (Elison & Harter, 2007). In the extreme, as has been revealed in our own society where access to guns is rampant, chronically bullied male youth, whose self-esteem has been compromised and who have, in response, felt humiliated, ultimately responded angrily and desperately, killing others. Our own approach to the issue of the effects of bullying, on the victims, differs somewhat from other conceptualizations in postulating that the emotion of humiliation is a key *mediator* of the violent revenge that the victims ultimately act upon. Chronic bullying leads to low self-esteem and related humiliation, which, in turn, fuels attempts at retribution, and sadly, acts of attempted mass killing. Emotions are powerfully implicated in human behavior, as many thoughtful emotion

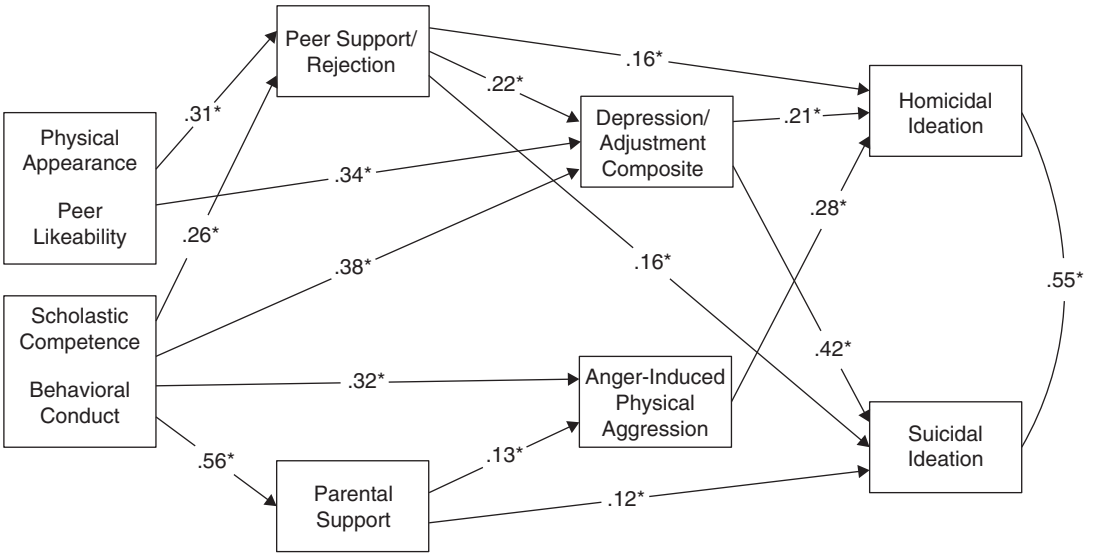


Figure 11.1 Path-Analytical Model for Predictors of Homicidal Ideation and Suicidal Ideation

theorists and researchers have documented (e.g., Fridja, 1986; Lewis & Haviland-Jones, & Barrett, 2008; Saarni, Campos, Camras, & Witherton, 2006), and we are simply echoing a similar framework.

Interestingly, humiliation, as one of a family of *self-conscious* emotions (see Tracy, Robins, & Tangney, 2007) that includes shame, guilt, and embarrassment, has been given short shrift. This, despite the fact that many children and adolescents are humiliated, in schools, playgrounds, and neighborhoods, every day of their lives. Yet there has been surprisingly little empirical research on this critical emotion (Harter, 2012). This is true not only in the research literature but also in the clinical literature. Negrao, Bonanno, Noll, Putnam, and Trickett (2005) noted that attention to humiliation has also be conspicuously absent in the childhood sexual abuse literature, where shame and anger have been the primary focus.

Thus, we have embarked on a program of research over the past decade to document the many critical features of humiliation. For example, we have found that it is critical that there is an *audience* that witnesses acts to demean the victim, psychologically, threatens their sense of self-esteem, that engages in hostile laughter, mocking the victims. In addressing humiliation, it is critical to distinguish it from other self-conscious emotions, shame, guilt, and embarrassment. There are similarities but also differences (see Elison & Harter, 1906; Harter, 2012; Tracy & Robins, 2004). We will summarize these, with a focus on humiliation, addressing the *causes,* implications for the *self,* role of the *other*, *emotional* correlates, and *behavioral consequences* (see also Torres & Bergner, 2012).

The Dimensions of Humiliation

One dimension addresses the *causes* of humiliation. Clearly, bullying, taunting, harassment, mocking, and torture, in the extreme, at the hands of *others* have been identified as powerful, prototypical antecedents (see Elison & Harter, 2007; Gilbert, 1997; Harter, 2012). Ostracism has also been implicated (Hartling, 2007). These causes differ from the antecedents of *shame*, which typically involves a *moral transgression* where, in the parlance of this chapter, the I-self is ashamed of the me-self, for the violation of cultural and personal standards of behavior (Lewis, 1987). Several theorists have argued that humiliation is more *other-focused,* whereas shame is more *self-focused* (Gilbert, 1997; Klein, 1991; Miller, 1988; Sarpharie, 1993). Humiliation can also be distinguished from embarrassment in that it involves the *hostile intent* of others who deliberately engage in overt acts designed to denigrate the victim, to demean them in a manner that lowers their social status (Lacey, 2011). Moreover, the derogation of the victim is judged to be *unfair* or undeserved (Combs, Campbell, Jackson, & Smith, 2010; Gilbert, 1997; Hartling & Luchetta, 1999; Klein, 1991; Statman, 2000; Torres & Bergner, 2010).

Humiliation has definite implications for the self. If the self is seriously devalued in the eyes of others, one loses one's dignity and experiences a major loss of self-esteem, leading to humiliation as a very intense, painful emotional reaction. Klein (1991) described the experience as loss of face, an invasion of the self, at the hands of others. This experience is exacerbated if the personal attack is directed

at one's perceived weaknesses. For example, if the self is viewed as a klutz, then spilling food in a restaurant, and then tripping in an attempt to recover the food on the floor, will reinforce one's trait attributions of clumsiness, intensifying the experience of humiliation. Moreover, in a dissertation by McCarley (2009), we found evidence that the lower the global *self-esteem* that one brings to a potentially humiliating experience, what Tangney and Dearing (2002) referred to as *trait* self-esteem, the more intense the emotional reaction. However, Tangney and Dearing also pointed out that a drop in more transitory or *state* will also be experienced as a *result* of being humiliated.

The specific role of the *other* is also critical in that an *audience* is a prerequisite for the experience of humiliation (Harter, Low, et al., 2003; Harter, Kiang, et al., 2003). Our findings have revealed that humiliation is intensified if the observing others laugh or join in the mockery by the perpetrators. In addition, we have documented the fact that the larger the audience, the more intense are the feelings of humiliation. This feature differentiates humiliation from shame or guilt in that the latter emotions can be personally experienced in the absence of an audience.

There are also clear *emotional correlates* of humiliation. It is likely that embarrassment will be experienced. However, as Elison and Harter (2007) pointed out, the humiliated person will probably first be embarrassed; yet if one merely feels embarrassed by an event of their own doing, for example, walking out of a restroom with toilet paper on his or her shoe, one may feel embarrassed, but not necessarily humiliated. People would only feel humiliated if there was a witnessing audience who began to laugh at them and mock them cruely. However, the emotional correlates that accompany humiliation extend beyond embarrassment. The victim also feels anger toward the perpetrator, if not rage (see Leidner, Sheikh, & Ginges, 2012). The entire constellation of associated emotions also includes sadness and depression, as well as anxiety and fear over future humiliation and attacks against the self.

Intense emotional correlates provide causal links to the *behaviors* that can be provoked. Humiliation and the associated emotion of anger are highly likely to fuel acts of revenge or retaliation, as in the case of the school shooters who were driven to violence given a chronic history of victimization (Combs et al., 2010; Elison & Harter, 2007; Harter, Low, et al., 2003; Hartling, 2007; Jackson, 2000); Leary, Kowalski, Smith, & Phillips, 2003; Smith, 2008; Trumbull, 2008; Walker & Knauer, 2011). Scheff (2011) went so far as to claim that the humiliation–revenge cycle is the most dangerous element in human history. Smith, Webster, Parrott, and Eyre (2002) reported that humiliation, more so than shame, was strongly correlated with hostility (see also Garbarino, 1999). In contrast, shame is more likely to cause the individual to want to escape, hide, or disappear (Harter, Kiang, et al., 2003).

Humiliation and Violence Within the School System

In our own research (Harter, Low, et al., 2003), we have explored young adolescents' reactions to hypothetical events that simulated the types of harassment experienced by the school shooters, albeit it somewhat milder forms, through the creation of vignettes. Two features of these vignettes were noteworthy. First, there is a peer audience that laughs at the victim in a mocking fashion. Second, each vignette ends with a statement to the effect that this was not the first time something like this had happened, implying a history of insult and ridicule, given that being chronically harassed was a feature of the experience of the actual school shooters. We first determined the mean level of rated humiliation, which was 3.4 on a 4-point scale (1 to 4), clearly indicating that the vignettes we created did, as intended, evoke feelings of humiliation. Young adolescents in this normative sample were asked to indicate how likely they would be to react violently, in retaliation or revenge and to rate each of the variables in our revised model. We first divided the sample into violent and nonviolent ideators and then examined their ratings on each of the variables in the model.

Violent ideators, compared with the nonviolent ideators, reported greater anger and blame toward the perpetrators and, interestingly, greater difficulty in getting a humiliating event out of their mind, suggesting the role of rumination. Violent ideators also acknowledged more depressed affect, lower self-esteem, and greater hopelessness. In examining the antecedents of self-esteem and its correlates, violent ideators reported significantly lower feelings of peer likability and physical attractiveness, along with lower feelings of peer approval and higher peer rejection. Parent approval also differentiated the two groups. Violent ideators also report a greater preoccupation with violent media (e.g., movies, TV, video games) as well as more interest in, and access to, weapons and bombs.

Finally, in this same study, we sought to determine whether teachers could identify those students at risk for violence. Thus, teachers were asked to independently rate violent tendencies in their own students. A comparison of teacher ratings with students' own ratings of violent ideation revealed that teachers were largely unaware that a significant number of adolescents in their own classes would harbor thoughts of revenge or retaliation, should they be humiliated. This is not to indict teachers, given that

their job description does not include such detection. The point is that there may be self-report strategies that can help to identify such individuals who are at risk, not only for harmful acts toward others but also toward themselves.

Within the last decade, others have turned their attention to the role of humiliation and violence within the school setting. Phillips (2007) described the dynamics of bullying and verbally and physically attacking victims. The behaviors on the part of bullies are purposeful strategies to affirm masculine norms of toughness, strength, dominance, and control (also see Pollack's, 1998, description of the boy code). Olweus (2008) described bullying as one of the biggest social problems affecting children in our schools today (see also Jenkins & Demaray, 2012; Tenenbaum, Varjas, Meyers, & Parris, 2011). Mattaini (2008) also addressed this concern, observing that rates of harassment, bullying, threats, coercions, and humiliation, and the intentional and hostile exclusion in the peer environment are much higher that even most adults realize (see also Williams & Nida, 2009).

Employing a daily report methodology, Nishina (2012) found that humiliation increased on school days on which adolescent students reported public peer victimization. Meltzer, Vostonis, Bebbington, & Dennis (2011) studied retrospective reports of being bullied in schools during childhood, in a psychiatric sample of adults. Being the victim of bullying led to the experience of suffering defeat and humiliation, which in turn provoked feelings of entrapment, hopelessness, depression, and suicidal ideation or attempts in adulthood. Miller (2012) also pointed to self-destructive behavior, including suicide.

These dynamics extend to our college campus environments, where bullying, in the form of hazing, has become institutionalized (Denmark, Klara, & Baron, 2008). Moreover, it is not merely peers who engage in humiliation tactics; adults in positions of authority (e.g., sports coaches) also use practices such as belittling, rejecting, and emotional abuse to command respect and demand higher levels of performance.

Of course, school officials and parents have become painfully aware of bullying and humiliation within the schools, as it has risen in the consciousness of today's culture, in part due to much greater media coverage. Post-Columbine, there have been several copycat school rampage shootings, which rightfully should be distinguished from other forms of violence (e.g., inner-city gang members targeting single victims in drive-by shootings), as I have also argued (Harter, 2012). Larsin (2009) made a related point, namely, that rampage shootings are to be distinguished from other forms of school violence, for example, a targeted assault on a single individual and gang-related vendettas.

Sadly, awareness of retaliatory violence in the form of mass public shootings has been enhanced by the subsequent murderous actions of a previous university student at Georgia Tech.

Most recently (in 2012), society has witnessed the attempted mass killing of innocent shoppers at a mall in Oregon, the murderous intent of the shooter to kill as many as possible in a movie theater in Aurora, Colorado, and the senseless shooting of 20 young children and six adults in an elementary school in Newtown, Connecticut. We know less, at this point, about the psychological makeup of these shooters, particularly since two of the three killed themselves, attesting to the link between violent acts toward others as well as toward the self. Whether low self-esteem or a history of humiliation was part of their own personal story has yet to be determined; we can only speculate.

These events have provoked an intense new political agenda on the part of the Obama administration, toward enacting tougher gun control legislation, at odds with the powerful National Rifle Association. Others argue for more mental health services to identify potentially deranged individuals as well as preventative measures in terms of support systems and interventions to support people who may well be at risk. Toward this goal, our own studies, as well as the efforts of others, have identified a number of psychological factors within the self-system that contribute to violent and suicidal ideation among our youth.

Cyberbullying

In recent years, a new trend has emerged, both in this country and abroad, namely, *cyberbullying*. This type of harassment is defined as sending demeaning messages on the internet (e.g., Facebook, twitter), email, or texting that involve public or private derogatory comments directed at negative characteristics about the self that are intended to ruin one's reputation (Menesini, Calussi, & Nocentinie, 2012). Pyzalski (2012) operationalized it as a form of bullying understood as peer aggression whereby a bully or perpetrator attacks someone perceived to have lower status, where there is an imbalance of power between the victim and the perpetrators. Whereas children and adolescents, at the end of the school day, can escape the direct form of bullying, be it physical or verbal, they cannot avoid cyber bullying if they are connected to social media, emails, or texting. Thus, it becomes a particularly pernicious form of persecution, all the more so because

it can be done anonymously and oppressively, with little space for escape. One's own room is no longer safe if one is connected to the Internet and these other forms of social media communication.

There is a small, but growing, literature on the topic, although most authors acknowledge that there has been little solid empirical research on the topic. Admittedly, this is a difficult topic to research. That said, there are a few articles that focus on the characteristics of the *bullies*: that they lack the moral values that would prevent such acts by either traditional or cyberbullies (see Menesini, Nocentinin, & Camodeca, 2013), researchers from Italy. Perren and Gutzwiller-Helfenfiner (2012), from Switzerland, also report that a lack of moral emotions and moral values predicted cyberbullying. Another study from Portugal (Almeida, Correia, Marinho, & Garcaia, 2012) documented the role of moral disengagement and lack of empathy. Some investigators cite the need for *self-enhancement* among cyberbullies (Menesini et al., 2012).

Other studies focus on the characteristics of the *victims* of bullying through cyberspace tactics. Some have pointed to the victim's *low self-esteem* (Brighi et al., 2012; Holfeld & Grabe, 2012), *social anxiety* (Holffeld & Grabe, 2012), and *loneliness* (Brighi et al., 2012). While there are similarities between the traditional bullying victim dynamics and cyberbullying, there has not yet been a focus on the humiliation experiences by victims. One report from Luxenberg investigators (Konig, Gollwitzer, & Steffgen, 2010) suggests that some forms of cyberbullying are conducted by victims of bullying who are seeking vengefulness and justice. However, there has yet to be empirical research on the importance of *humiliation* as a mediator between the experience on the part of cyberbullied victims and their acts of revenge and just through what media or behaviors it might be enacted. Thus, much more attention needs to be directed toward these issues, both in this country and abroad.

SUMMARY, THUS FAR

This contemporary excursion into James's (1892) insightful recognition of the I-self, for too long put on the intellectual shelf, has been something of a roller-coaster ride. It began with a developmental perspective, describing how the I-self, defined as changing cognitive abilities, shapes the me-self across three stages of childhood and three stages of adolescence. This discussion also addressed those positive I-self functions identified by James, which included self-awareness, a sense of agency, the continuity of the self and its companion, self-coherence. Although James intended these terms to reflect the adult self, their origins were traced across the six developmental levels.

Next ensued a discussion of the dark side of the demands put upon the I-self in contemporary culture, tracing at each of these same developmental levels, whether and how the I-self had the motives and skills to engage in the self-enhancement tactics and self-serving biases that are observed in adults. Next, we examined how the conspiracy between the I-self and the me-self contributed to five different, though related, forms of developmental psychopathology in which the self was intimately related: (1) narcissism; (2) low and debilitating global self-esteem; (3) depression reactions including suicidal ideation; (4) eating-disordered behaviors; and (5) violence in the form of victims' actions of revenge in the face of harassment and bullying.

However, the point was made that I-self and me-self processes do not develop in a vacuum, the child's socialization experiences impact how these processes unfold. Many benevolent parents support the development of adaptive healthy I-self functioning. In contrast, conditional parent support, where the parental agenda is intrusively foist upon the child, can suppress the child's true self, leading to false self-manifestations. The severe and chronic abuse of children at the hands of caretakers can derail healthy development, compromising such I-self functions as self-awareness, a sense of agency, of self-continuity and of self-coherence. While we have seated certain forms of developmental psychopathology at the interface of what has been labeled a co-conspiracy between the I-self and the me-self, the I-self also has the potential to provoke positive, mental *health*. I would be remiss to not flip the coin given that, as students of developmental psychopathology, our ultimate goal is to understand and ensure healthy functioning. Thus, we turn next to this topic, affording the I-self its rightful position in promoting mental health.

The I-Self's Role in Promoting Mental Health Among our Children and Adolescents

As noted, James (1892) identified several I-self functions, self-awareness, sense of agency, sense of self-continuity, and self-coherence. However, he did not map out how these might be manifest at different developmental levels, nor did he thoughtfully explore the implications for either psychopathology or mental health, to which I now turn. That has been the goal of this chapter.

Awareness

Awareness involves not only self-awareness but also an appreciation for the outer world. Admittedly, one I-self job definition is to scrutinize the inner world of the me-self. But the I-self also directs its awareness, its gaze, to the outer world of reality. It is literally the eye that perceives the world beyond the self with the potential to make many interesting observations, such as sunsets and rainbows and the beauty of nature. It becomes aware of others and the gratification of meaningful relationships. It looks for opportunities to improve one's capabilities, it looks for challenge at an appropriate level. This may occur in one's work situation leading to greater job satisfaction. It may involve leisure activities, reading, writing, gardening, sports, travel, new educational ventures, wonderment, and falling in love. It involves getting a life. Whatever one's choice, such activities can only be pursued if the I-self is unencumbered by its stultifying demands to defensively package and protect a potentially fragile Me-Self.

Sense of Agency

The positive and natural desire to be a causal agent can be captured in many manifestations, including (1) self-efficacy, (2) mastery attempts, (3) intrinsic motivation, and (4) the concept of flow. We will address each of these and then turn to the issues of self-consistency and self-coherence.

Self-Efficacy as an I-Self Process

James's (1892) identification of a sense of agency as a critical I-self function has much in common with a more recent construct in the field, that of *self-efficacy* (Bandura, 1977, 2006). Self-efficacy is actually a belief on the part of the I-self that one can successfully make things happen, it is an I-self observation about one's sense of agency, projected into the future, both near and far. I repeatedly find that self-efficacy is often confused with *self-concept*. (I encounter this confusion when people interested in our own instruments indicate that they are looking for a measure of self-efficacy, whereas our self-perception profiles tap present domain-specific self-concepts and self-esteem, *not* self-efficacy.) So what is the difference? Perceptions of competence or adequacy are *me-self* constructions, namely, the *product* of the self-evaluation process, the *observed* self in the psychological mirror. Self-efficacy is an I-self cognitive belief about one's ability to be successful in the future.

Self-efficacy, since Bandura (1977) first introduced the concept, has permeated the field of psychology as well as many related disciplines. It obviously spoke volumes to many who welcomed the introduction of a construct that captured this dynamic force in human nature. Static self-perceptions did not reflect this vibrant process, the belief that one held the personal power to be efficacious. What are the implications of conceptualizing self-efficacy as an I-self process? How might it be relevant to the goals of this chapter, namely, to identify how I-self and me-self processes contribute, not only to developmental psychopathology but to conceptions of mental health?

Bandura (2006) recently gave us some clues. He embedded this construct within the general debate about psychological *traits* that transcend context versus attributes that are situation-specific, that are very much bound by context. His conclusion is that self-efficacy is best conceptualized as context-specific. One may feel efficacious in one domain but not necessarily in others or all domains. To take a contemporary example, a bright computer nerd may feel that he can contribute brilliantly to the latest social media frenzy but feels far less efficacious in the realms of romantic pursuits or reality TV. Whether one conceives of self-efficacy as trait-like or more situation-specific, however, is not really germane to the issue of pathology versus mental health. The point is that self-efficacy is really a characteristic of the I-self, an active force, be it cognitive or physical activity in which one acts on the world, with the belief that one can achieve an effect in the immediate or distant future. It is one embodiment of James's I-self function of *agency*. It is the I-can force that makes us move confidently forward.

Mastery Motivation

The concept of mastery motivation appears in the work of many scholars and is most frequently invoked in dealing with manifestations in children and adolescents. Originally, it appeared in the writings of Robert White (1959), who described the concept of effectance motivation, a driving force beginning in infancy that propelled the child toward curiosity, exploration, and mastery attempts that were designed to have an effect on one's world. Whether this is kicking one's foot to make the mobile above one's head, attached to a string, jiggle or move, or attempt to master linguistic utterances in order to have an effect on one's caregivers, this is the I-self in action. Among young children, it might amount to kicking a soccer ball to score a goal or using language to impact the significant others in one's world, through communication, where there is

definitely a goal in mind. Thus, mastery attempts became intimately related to *motivational goals* that propel healthy development, very definitely an I-self function.

We see the concept of mastery in Deci and Ryan's self-determination theory (Deci & Ryan, 2000; Ryan & Deci, 2000, 2009), who documented children's strivings for both competence and autonomy. These natural strivings are contrasted to a preoccupation with meeting the external goals established by significant others, captured in contingent reward systems and conditional approval (Harter, 1999), which can compromise these normative and necessary I-self functions. Socializing agents and caregivers, are, therefore, quite relevant.

In a recent creative integration of attachment theory and goal orientation theory, Rusk and Rothman (2010) pursued these themes. From each of their respective perspectives, they argued that caretakers can endanger the pursuit of self-validation goals in contrast to facilitating the I-self functions of cognitive openness, curiosity, mastery, exploration, learning, and effective problem solving for its own sake. They drew upon the attachment theorizing of Mikulincer (1997, 1998), describing how certain caregivers' attachment styles can interfere with these functions. From Mikulincer's perspective, those who are compromised by negative attachment styles (e.g, anxious-ambivalent or -avoidant) that stem from lack of parental responsiveness or attunement will turn to an underlying concern with approval to falsely enhance self-worth rather than a focus on mastery and learning goals. For goal-oriented theories, a major distinction is drawn between *self-validation* and *learning or mastery goals* (see Dweck, 1999; Dweck & Master, 2009; Kaplan & Maehr, 2007; Mikulincer & Shaver, 2007).

Dweck's (1999) work can readily be brought to bear upon this distinction and can be cast into I-self versus me-self terms. Dweck has identified those children who adopt an *entity* theory of characteristics (such as intelligence, social skills) who view such attributes as fixed and immutable. They cannot be altered. She contrasts these individuals to those who adopt who she labels as *incremental* theorists, who see the promise of improvement and change, due to their own personal efforts. The relevance to the me-self versus I-self processes is that those who adopt an entity perspective have much in common with self-validation tendencies; these are individuals who are desperately trying to protect and falsely enhance the me-self. Those who adopt an *incremental* perspective are much more aligned with I-self mastery goals. Thus, the message is that the incremental perspective serves an important I-self function in the service of mental health.

Intrinsic Versus Extrinsic Motivation

Intrinsic motivation, defined by such dimensions as curiosity, exploration, preference for challenge, and the joy of learning for its own sake (Harter, 1981; White, 1959) can be intimately linked to the two aforementioned themes, self-efficacy and mastery motivation. As such, we view them as I-self processes where the energy is directed outward toward interesting pursuits that are rewarding for their own sakes, versus a preoccupation with the fragile me-self, preening nervously in the mirror. Over the years, my own research group has directed a great deal of attention to the distinction between intrinsic versus extrinsic motivation, trying to unravel the correlates and consequences of both orientations. In this chapter, I have revisited this distinction linking intrinsic motivation, as an I-self function to mental health, whereas extrinsic motivation seems more aligned with an orientation toward seeking extrinsic rewards from significant others that focus on protecting the self. This latter perspective, in turn, interferes with true mastery goals and the potential intrinsic joy of competence. But do the consequences of these two orientations really matter?

Our own findings reveal a very high, positive correlation between intrinsic motivation and perceived competence scholastically, including greater pleasure in such accomplishments. These seem more consistent with the I-self functions of mastery and intrinsic motivation. Those who report an extrinsic orientation, more akin to Dweck's students who report an entity interpretation, seem more intent on self-validation goals, a more me-self focus with its compromising features.

However, our findings (Harter, 1981, 1999) reveal, sadly, that intrinsic motivation systematically declines as children move through the school system. We and Eccles (see Eccles & Roeser, 2009) have given trenchant analyses of how the decline in intrinsic motivation is a function of certain educational practices within school systems (see review in Harter, 2012). The purpose of this chapter is not to review these debates. However, there is a subplot here. As a review of the self-esteem movement (Harter, 2012) indicates, the emphasis on falsely enhancing students' views of their skills, competence, and value, places the focus on the me-self and its negative consequences, compromising the I-self functions that can lead to mental health.

The Concept of Flow

Closely aligned to concepts of intrinsic motivation is the construct of flow, introduced initially by Csikszentmialyi (1997). Flow is characterized by a complete absorption in what one is doing, a pleasurable, optimal state of being that seems akin to a Jamesian definition of the I-self. There are links to intrinsic motivation in that one is engaged in rewarding activities, in and of their own sakes, without consideration of extrinsic rewards. Whether these are leisure pursuits or related to aspects of one's profession, typically the dynamics involve perceived challenges that stretch existing skills beyond, but not exceeding, one's capabilities. There are clear proximal goals that allow one to make this determination. Moreover, the process is very pleasurable.

Particularly critical to this chapter is that the flow experience is relatively free of self-consciousness; there is the loss of awareness that one is a social actor. Rather, there is the perception that the activity is intrinsically rewarding, there is no extrinsic goal in sight. Intense concentration is one of the defining features of flow, it is clearly an in the moment experience. There is a loss of a definition of self, of the self-consciousness that so defines the me-self. Flow marks the fading of the me from self-awareness as attention is taken up entirely by the challenges in which one is engaged (Nakamura & Csikszentihalyi, 2002). The ultimate goal is the growth of the individual, as the optimal level of challenge stretches one's existing skills and who one is as a person. The flow experience is thus a force of expansion, consistent with James' intended goal of the I-self.

Self-Continuity

Self-continuity, for James (1892), referred to an I-self function in which there was a sense of continuity or the permanence of the self over time. For young children, this involves the realization that the self is invariant over time, even given changes in outward appearance, for example, wearing different clothes, changing one's hair style, growing taller (Nelson, 2003; Rochat, 2003). Very young children have not yet acquired such a concept. As Piaget (1960) documented, when questioned about whether their gender could change, young boys will acknowledge that they could become a girl, and could grow up to be a mommy, and young girls similarly believe that becoming a boy is a possibility, and they could grow up to be a daddy. Thus, very young children do not display self-continuity; they lack a sense of even physical *self-conservation* (Harter, 1983).

However, by age 5 or 6, children do develop a sense of the permanence of their physical self. There is considerable agreement that *autobiographical memory* is critical to a sense of continuity (Fivush & Haden, 2003). Creating *self-narratives* represents a primary mechanism through which a sense of the continuity of the self is established. Initially, narratives are highly scaffolded by parents, who dictate and reinforce aspects of the child's experience that they feel are important to codify and remember (Fivush & Hudson, 1990; Haden, 2003: Nelson, 2003; Nelson & Fivush, 2004). Increasingly, these autobiographical accounts are *coconstructed* by parent and child, in that the child takes on a greater role in their construction. By mid to late childhood, the child takes the major responsibility for the content and structure of his or her narrative, one's budding life story. Interestingly, there is no demand for total accuracy or completeness. Certain experiences can be omitted, others embellished, at the whim of the narrator. However, when the narrator takes charge of the construction of his or her autobiographical story, then a sense of continuity permeates the continuing narrative.

The I-self function of *agency* contributes to a sense of self-continuity. The perception that the self is continuous provides the individual with a sense of purpose and the direction that one's life will take. It allows one to appreciate who I am and will continue to be. There is a core and enduring self at the helm, in the face of more superficial change. As such, it contributes to a mentally healthy and adaptive approach to the future.

Clinicians have become aware of the importance of a sense of self-continuity when dealing with adult clients who have been severely and chronically abused as children or adolescents. Such individuals typically display forms of pathology where there is the loss of significant chunks of their autobiographical memory and some may never have had the opportunity to create such narratives (see Putnam, 1993). These amnesic gaps in memories, the loss of significant phases of their lives can be extremely derailing, when there is no sense of self-continuity to provide direction in the form of who one is as a person.

For some, this takes the form of low self-esteem, depression, and in some cases suicidal ideation. For others, a different condition may arise, what we have labeled an impoverished self (Harter and Marold, reported in Harter, 1999, 2012). I first encountered such a phenomenon when, in a seminar on the self for experienced clinicians, one astute woman asked me, "But what do you do with a child who *has no self*?" This struck me as a very profound question, for which I did not have a good answer, but it provoked me, in collaboration with clinician Donna Marold, to flesh out what such a developmental history might look like.

The Impoverished Self

The history of what we labeled as an *impoverished self* has it roots in the early socialization practices of caregivers who fail to assist the child in the co-construction of a positive, personally rich, and coherent self-narrative. This typically happens given parents, who because of their own dysfunction (e.g., depression, borderline pathology) and other inadequacies do little in the way of fostering the child's self-narrative (see discussion in Harter, 2012). Marold, in her clinical practice, observes that children with an impoverished self come to therapy with the lack of a descriptive, evaluative vocabulary to define the self and there is little in the way of autobiographical memory or a personally meaningful narrative. The liability is that they have few personal referents or self-concepts around which to organize present experiences. As a result, the behavior of such children will often appear to be disorganized, without purpose. Moreover, to the extent that a richly defined self promotes motivational functions in terms of guides to regulate behavior and to set future goals, such children may appear aimless, with no clear pursuits.

Marold has astutely observed that these children do not have dreams for the future, whereas most children do have future aspirations, as naïve as they may appear to be. They want to be a hero on a professional sports team, or a firefighter, an astronaut, a doctor, or a teacher, a nurse, a veterinarian. However, the parents of children with an impoverished self do not foster goals, nor do they engage in such simple acts as posting the child's artwork or school papers on the refrigerator door. They do not have family rituals such as cooking the child's favorite food or reading (and rereading!) cherished bedtime stories. Marold (see Harter, 2012) has suggested several interventions for working with such children to basically recreate a sense of self-continuity that has been neglected or lost, or create one that has never been established. By observing the consequences of those socialization factors that can interfere with the development of the I-self's sense of continuity over time, we can appreciate its contribution to the healthy adaptation of individuals.

Chandler et al. (2003) addressed the role of a lack of self-continuity among adolescents. They provide findings that those adolescents who fail to develop a strategic justification for the continuity of the self are more prone to suicidal behavior. They lack earlier developmental solutions and cannot create a new workable rationale, which brings with it a lack of personal connectedness to one's past, current, or future self, leading to a sense of futility. Thus, those who "lose the thread of their own personal persistence are at special risk for suicide" (p. 116).

The value of establishing a sense of self-continuity, through the continued construction of autobiographical narratives, carries over into adulthood (McAdams, 1997). In developing and embellishing a self-narrative, the individual creates a sense of continuity over time as well as coherent connections among self-relevant life events. In constructing such a life story, the I-self is assigned an important agentic role as author, temporally sequencing the Me-selves into a coherent self-narrative that provides personal meaning and a sense of future direction. Moreover, as these psychologists argue, narrative construction is a continuous process, as we not only craft but also revise the story or our lives, creating new blueprints that facilitate further architectural development of the self. As such, this cognitive reframing can serve to promote healthier self-development.

The I-Self Function of Self-Coherence

Self-coherence, for James (1892), referred to how, at a given point in time, the various facets of one's personality form a unified, coherent whole. Personal characteristics are consistent with one another and make for a sense of the integrity of one's self-portrait. However, in contrast, in other discussions of the self, James (1890) pointed to the different Me-selves that the I-self constructs. He observed: "Many a youth who is demure enough before his parents and teachers, swears and swaggers like a pirate among his tough young friends" (p. 169). Often, James conceded, these different selves could be discordant, if not contradictory. He labeled this experience *the conflict of the different me's* and described how the incompatibility of potential roles continues into adulthood. As he observed, "A man has as many social selves as there are individuals who recognize him and carry an image of him in their mind" (p. 190).

So how can this conceptual conundrum be resolved, how can there be a *coherent* self in the face of the conflict of the multiple me's? Surprisingly, for this intellectual giant, James had no ready answer. He seemed to emphasize the troublesome contradictions between disparate selves rather than the I-self function of insuring compatibility or coherence. In fact, he accused the I-self of being an elusive if not incorrigible construct, and eventually abandoned his interest in the I-self, never really wrestling with this dilemma. The subsequent discussion will attempt a contemporary explanation that might resolve this apparent contradiction.

It should first be acknowledged that theorists in the first half of the twentieth century did not embrace

James's (1890) pluralistic contentions. There was historical resistance to the notion of discordant selves. Rather, most endorsed a self-consistency ethic. Scholars seemed more enamored with the view that the self was integrated, unified, and rational (Allport, 1961; Horney, 1950; Jung, 1928; Lecky, 1945; Maslow, 1954; Rogers, 1951). For example, Allport emphasized the importance of a sense of *inward unity*. Lecky highlighted the theme of self-consistency and the need to maintain the integrity of the self. Later in the century, Epstein (1973; 1981) argued that one's self-theory must meet the criterion of *internal consistency*, which he formalized as the unity principle, arguing that there is a basic need to ensure the unity and coherence of the conceptual system that defines the self.

However, these formulations, emphasizing the supposed coherence of the self then met with new resistance toward the end of the twentieth century that continued into the twenty-first century, on two fronts: one was empirical, the other sociocultural. The empirical challenge came in the form of findings such as our own, described earlier, revealing a normative liability of adolescent development, more consistent with James's view of the *conflict of the multiple me's* (see Harter, Bouchey, et al., 1997). Our findings demonstrated that beginning in middle adolescence, the different me-selves that the I-self constructs in various roles (with parents, teachers, classmates, close friends, romantic partners, on a part-time job) can be quite inconsistent, if not totally contradictory, causing intra-psychic conflict and emotional distress.

The second challenge came in the philosophical shift from an era viewed as *modernism*, where the emphasis was on the consistency, coherence, and the rationality of human behavior to the current period of *postmodernism* (see Gergen, 1991; Lifton, 1993). Postmodernism initially not only stressed the reality of the multiple selves that contemporary society thrusts upon the self, but focused on the perils of such multiplicity, describing the landscape as cluttered with potentially treacherous minefields. Individuals were described as psychologically compromised by their inability to navigate the contradictory psychological pathway that represented a segmented self.

However, on both fronts, the empirical and the sociocultural, the pendulum has once again shifted. Our own findings (Harter, Bouchey, et al., 1997) have revealed, as described earlier, that the normative *liability* of perceived contradictions between one's multiple selves during midadolescence undergoes a transformation in later adolescence and beyond. Two processes have been documented (Harter, 2012). First, cognitive-developmental advances in the form of higher-order abstractions (Fischer, 1980) can be constructed by the I-self, allowing the older adolescent or adult to integrate what were previously viewed as inconsistencies. For example, the earlier perceptions that one was depressed with parents but cheerful with peers can be integrated into the conclusion that one is *moody* or flexible, thereby precluding the perception of conflict.

The second process involves the *normalization* of previous conflicts within the self. Thus, the older adolescent comes to the conclusion that it is normal and socially acceptable to be different in different roles or social contexts, that it would be weird, in the words of one young man, to be the same self (see Harter, 2012). Moreover, normalization will be more likely if the older adolescent receives the reassuring support of significant adults (see Fischer and Bidell, 2006, on the importance of social support at higher developmental levels during adolescence). As observed earlier in this chapter, most *normative liabilities* are transient as cognitive-developmental and socialization factors come to the rescue. From the perspective of transformational interventions, parents and other significant adults (e.g., counselors or therapists) can provide support for an understanding that it is normal to maintain different selves, providing examples from the teenager's own life situations.

The post-modern era also witnessed a compatible shift in thinking about the multiplicity of the self. The construction of multiple selves was now viewed as a realistic and adaptive stance to the demands of modern life. Thus, it became appropriate to display different selves in different life contexts, shifting one's psychological stances accordingly. Paulhus and Marin (1988) are among those who have endorsed the *functional flexibility* of multiple selves that can be called upon, confidently and adaptively, across different relational contexts.

How do these considerations bear upon the issue of the *coherence* of the self and the I-self's contribution to the mental health of the individual? Our own research and thinking suggests that this requires a redefinition of coherence. No longer do perceived *unity*, consistency, and total integration seem to be the appropriate consideration. That is, the I-self is no longer required to integrate one's diverse role-related attributes into one big happy sense of unity. However, the I-self is not off the hook by this declaration. It must come to the rescue by providing some solution as to how the different facets of the self can comfortably co-exist, and in the process achieve a new sense of coherence.

Our own research also provides some clues, consistent with the observations of Paulus and Marin (1988), in examining certain features of *acculturation* and *bicultural*

adaptation. Tara Mehta, who came to our graduate program from India, was interested in the adaptation of youth from India and other Eastern Asian countries, to the United States, to Chicago, specifically. We became interested in the claims of Berry (2005), a much respected researcher who described the phenomena of acculturation and bicultural adaptation.

Berry (2005) challenged the prevailing uni-dimensional models in which individuals were supposedly required to either retain and vigorously defend the values of their culture of origin, or reject those values and totally embrace American values and traditions. He argued that there were different *combinations* of the endorsement of these two sets of values, for example, (1) rejecting the values of one's culture of origin and embracing American values (which he labeled rejection), (2) retaining the values of one's own culture and rejecting American values (labeled assimilation,) or (3) both retaining one's native cultural values while also adopting American values and traditions (which he labeled integration).

Although we respect his reformulation, Mehta and I challenged his assumption that those endorsing both sets of values truly felt integrated. Such individuals are clearly dealing with the issue of different selves from a more complex multicultural perspective. Our framework was more consistent with the position of Hong, Morris, Chiu, and Benet-Martinez (2000) who observed that two cultural forms of self-appraisal may alternatively be triggered in everyday life, leading to what they labeled cultural frame switching. Thus, such individuals would appear to reflect a bicultural form of adaptation rather than feeling that their different self-appraisals are experienced as a unified, coherent, whole. However, we felt that research was necessary.

To examine this hypothesis empirically, Mehta and I (see Mehta, 2005, described in Harter, 2012) provided young adult respondents (most originally from India) four written options, each of which was accompanied by a puzzle-like drawing that visually depicted each choice (Figure 11.2). We sought to tap four different orientations toward those who fell in the bicultural quadrant, displaying the values of both their culture of origin and American values: (1) true *integration* (first drawing at the top); (2) *unhealthy fragmentation* (second drawing); (3) *healthy differentiation* (third drawing); and (4) *no interest/reflection* on these issues.

Mehta (2005) and I predicted that the majority of these East Asian Indian participants would select the healthy differentiation option and that relatively few would endorse the integrated choice. These predictions were supported in that 70% did select the healthy differentiation option,

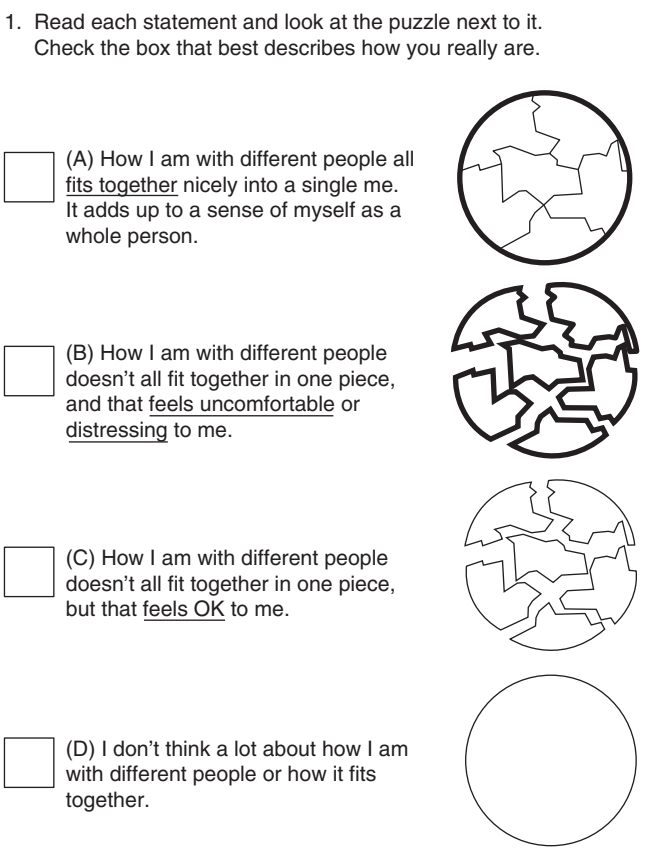


Figure 11.2 Measure to Assess Integration or Differentiation of Multiple Selves

whereas only 13% opted for the integrated choice. Another 13% chose the unhealthy fragmentation description, with only 4% selecting the no interest/reflection choice.

Based on this pattern of findings, we suggested that those who endorse both sets of values should not be conceptualized as displaying the integration of their self attributes, given that this is not their subjective experience, based on our data. Rather, bicultural appears to be the more appropriate designation. Our conclusion is consistent with the perspective of Friedman and Liu (2009), who define biculturalism as the ability to *comfortably understand and act in accordance* with the norms, ways of thinking, and attitudes of two cultural systems. The bicultural individual also has the ability to *shift* between these two systems appropriately, as the situation demands, as Hong et al. (2000) argued.

The findings from this study bear upon the definition of self-coherence as we complete our discussion of the I-self's role in promoting psychological health, beginning with James' contentions about unity of the self. At the same time, he insightfully described the conflict of the different me's, a contradiction that he never really confronted or

resolved. Our own solution is to redefine *self-coherence*, in that different self attributes can *cohere*, stick together (which is the dictionary definition), but not meld. This is the essence of our own conceptualization, operationally defined as healthy differentiation. This is the I-self's task, which will contribute to psychological adaptation for all who realistically experience different self attributes in different social contexts.

For those counseling individuals who are struggling with issues about how to reconcile values of a non-American culture of origin with the values and expectations of American society, the promotion of *healthy differentiation* should be the goal. In terms of transformational interventions, it may also be helpful to employ our visual depiction of the orientations we have studied empirical. That is, research and clinical goals can be integrated. By visually presenting and explaining the different options, one could dissuade the adolescent from thinking that their various selves should feel totally unified. They should be encouraged *not* to feel guilty, ashamed, or conflicted if they honor the values of their country of origin, with family or peers of their original ethnicity but also display American values with their American peers, be they students or coworkers. These roles can comfortably coexist. Counselors or therapists who have done so in their own life would be particularly valuable.

Next, there will be a discussion of cross-cultural differences in how self-coherence is conceptualized and experienced. As the countries of the world become less isolated, and as mobility increases, as in the world of business, there are more demands to think biculturally. However, we must all respect long-standing basic cross-cultural differences in views about self-consistency and self-coherence and how such differences can affect interpersonal relationships, across cultures. Then the stage shifts to broader challenges for the I-self, in the service of mental health.

CROSS-CULTURAL DIFFERENCES IN SELF-COHERENCE

American scholars over the decades have wrestled with the issue of whether the self was experienced as a unified construct versus differentiated, leading to further controversy about whether disparate selves were adaptive versus problematic if not pathological. However, eastern Asian views have remained relatively constant on this issue. Eastern perspectives have espoused a different conceptualization, in that they are more tolerant of inconsistency, ambiguity, and contradiction (Nisbett, Peng, Choi, & Norenzayan, 2001), what these authors refer to as *naïve dialecticism*. When confronted with a potential inconsistency, Chinese participants will attempt to resolve the dilemma through compromise, reasoning that most positions have merit. This type of thinking can be traced to Eastern philosophical notions of Yin and Yan, which are opposing but also integrated forces in the world.

There has been considerable documentation of this cultural distinction within the past decade. Spencer-Rodgers, Peng, Wang, and Hou (2004) examined cultural differences in reasoning about psychological contradictions and the effects of naïve dialecticism on self-evaluations. Their research reveals differences between residents of mainland China and Westerners in their spontaneous self-descriptions. Chinese participants reported more evaluative contradictions. Spencer-Rodgers and colleagues more recently replicated their findings with the Chinese and extended them to the Japanese who also display naïve dialecticism, demonstrating the contradictory, changeable nature of the East Asian concept of self (Spencer-Rogers, Boucher, Mori, Wang, & Peng, 2009). Others (see Hamamura, Heine, & Paulus, 2008) also documented the East Asian tendency toward dialectical thinking, manifest in a tolerance for contradictory beliefs.

Other treatments also focus on the situational specificity that is valued in Eastern cultures, where multiple selves are the norm. Suh (2002) makes explicit reference to such multiplicity that East Asian cultures endorse, which are experienced as comfortably coexisting realities. Suh's research questions the classic thesis in Western psychology that identifies *consistency* as a prerequisite for psychological well-being. Koreans, in his study, viewed themselves as more flexible across interpersonal contexts, which predicted well-being and positive social evaluations, whereas perceptions of identity consistency was a better predictor for North Americans. Spencer-Rodgers et al. (2009) further researched the naïve dialecticism that defines East Asian's tolerance for contradiction and the expectation of situational change in the self.

A similar theme was addressed by Nezlek, Sorrentino, Yasunaga, Otsubo, Allen, and Kouhara (2008), who demonstrated that the self-esteem of Japanese participants changed more in reaction to daily social events (be they positive or negative) than it did for North Americans. Their findings highlight the greater sensitivity of the Japanese to social context in contrast to North Americans. A related study (Tafarodi, Lo, Yamaguchi, Lees, & Katsura, 2004) revealed that East Asian cultures place more emphasis on the contextual adjustment of a person's behavior. Such an adjustment translates into greater variation in the outwardly presented self across contexts. These differences were manifest in comparisons of Canadian adults with Chinese and Japanese participants.

In summary, self-coherence takes on a somewhat different look in East Asian cultures than our revised definition for Americans, where we referred to *healthy differentiation* of one's attributes (Harter, 2012). A commonality involves the notion that the self is differentiated along various culturally appropriate roles, that there is situation specificity in the display of one's attributes. However, East Asians have more tolerance for inconsistency, ambiguity, and contradiction. In contrast, Western thought is fashioned after models of Aristotelian logic that are linear and analytic. Clarity is emphasized with little tolerance for ambiguity. Western formal reasoning requires that contradictions be resolved, only one argument can be logically correct (Nisbett et al., 2001). Although in American culture, adolescents struggle with inconsistency and contradictions in their self-portraits, the ultimate developmental goal is for these inconsistencies and contradictions to be resolved through the logical normalization of such inconsistencies, aided by more mature cognitive processes in the form of higher order abstractions.

MINDFULNESS AS AN I-SELF PROCESS: A BUDDHIST PERSPECTIVE

A central thesis of Buddhism is the cultivation of *egolessness*, which involves the denial of those ego-enhancing strategies that dominate the Western preoccupation with the self. We shall see how mindfulness comes to be a handmaiden for how this can be accomplished. According to one Buddhist scholar, Trungpa Rinpoche (1976), "We build up an idea, a preconception, that self and other are solid and continuous, and once we have this idea, we manipulate our thoughts to confirm it and are afraid of any contrary evidence" (p. 13). According to Rinpoche, we create an intense *watcher,* which is actually a very complicated bureaucracy that we construct to hopefully protect and enhance the self. In I-self, me-self terminology, the watcher represents the rather frenetic, encumbered, and at times, desperate I-self, preoccupied with strategies for managing the impression that the me-self is making on the world.

However, from a Buddhist perspective, one must go beyond this form of self-observation, one must dismantle the complicated bureaucracy that has been created to preserve the falsified illusion of the me-self. The I-self should not be burdened with the impossible task of packaging the me-self so as to be an acceptable psychological commodity in the social marketplace. Rather, the I-self should be directing its energies *outward*, exercising its capacity to enjoy and share life experiences, as opposed to turning inward in its preoccupation with the fabrication of a me-self fitfully designed and distorted to be acceptable to society at large.

How, from a Buddhist perspective, should the I-self achieve these lofty goals? A major answer lies in the cultivation of *mindfulness* that requires a thoughtful awareness of who and where one is in the moment. Here, it is important to contrast the goal of living *in* the moment from living *for* the moment. Living *in* the moment may not always be idyllic, it might call up an awareness that one has to wash the dishes or pay the bills. However, it can be contrasted with living *for* the moment (e.g., let's just get drunk tonight and blow off what we need to prepare for tomorrow). Contemporary psychologists, bridging Eastern and Western perspectives, concur. Langer (2009) describes mindfulness as a flexible state of mind that sensitizes us to our context in the present and opens us up to novel ways of thinking. It is to be aware and attentive in the here and now; this is the essence of being mindful.

Buddhism also encourages us to drop the elaborate fabrication that represents an embellished storyline of our life. Too often we create a Cecile B. DeMille production in which we are simultaneously the director, the producer, and the heroic protagonist: This is my life, the second greatest story ever told! Interestingly, our self dramas typically focus on the past and the future, but not the present. We rarely stop to ask the more critical question: What is going on *right now*? This question is at the core of mindfulness. The goal of mindfulness is the awareness of one's present surroundings, including an accurate perception of oneself.

How, from a Buddhist perspective, can one achieve such mindfulness? In the Buddhist tradition, meditation becomes the pathway to such realizations. A discussion of the essence of meditation practice is beyond the scope of this chapter. However, it begins with the very meaning of the name Buddha, which derives from the word meaning to be awake. A major goal of meditation is to promote mindfulness by reducing our constant mental chatter. We typically engage in considerable cluttered thinking that is reflected in our self-absorbed internal monologues as well as dialogues between our multiple selves. Such thinking diverts our attention from a more mindful focus on the present, which both Eastern and certain Western scholars agree is more adaptive. There have been thoughtful treatments of the meditation process by American psychologists (e.g., Benson, 1975, 1985; Leary, 2004; Langer, 2009; Shapiro, 2009), exploring its benefits, many of which correspond to the Buddhist perspective.

The goals of meditation and the concept of mindfulness, designed to remedy the excesses of the Western self, have much in common with a relatively new emphasis on *quieting the ego* (Wayment & Bauer, 2008). As observed,

in reviewing the many self-enhancement techniques, the Western ego is quite noisy, excessively so in the case of narcissism. Thus, the quiet ego has been contrasted to the noisy, self-aggrandizing ego that requires a self-serving megaphone aimed at audiences whom one hopes will be attentive. However, often the self-promoting proclamations eventually fall on deaf ears.

One feature of the quiet ego is particularly relevant to I-self processes, namely, *detached awareness* (Wayment & Bauer, 2008). Detached awareness refers to the person's awareness of self and situation that is nondefensively detached from egoistic appraisals. In I-self, me-self parlance, the I-self is relieved of its duties to protect and enhance the me-self. Wayment and Bauer also emphasize the role of *humility*, which they view as self-awareness tempered by self-compassion (see also Neff, 2008). In the same vein, Tangney (2009) offered a thoughtful treatment of humility, highlighting one feature that is directly related to the quiet ego. She describes humility as a forgetting of the self, which is *not* a denial of the self. Rather, it requires the recognition that one is but part of the larger universe, putting the self in perspective as a small cog in a much larger world wheel. That said, Tangney is quick to assert that humility is not self-deprecation. It involves the acceptance of one's limitations, as well as strengths, humility respects that while one has a certain degree of personal power, one is not omnipotent. One becomes unselved, in Tangney's view, analogous to the quiet ego.

FURTHER IMPLICATIONS FOR MENTAL HEALTH: THE ROLE OF THE POSITIVE PSYCHOLOGY MOVEMENT

A major goal of this chapter has been to cast developmental psychopathology into a framework of the Jamesian I-self and me-self, arguing that certain I-self/me-self interactions can result in psychopathology. However, it behooves us to translate this framework into our ultimate goals, which are mental health and well-being. Toward this objective, many positive I-self functions have been illuminated. Similar processes have been highlighted in the recent positive psychology movement (see Lopez & Snyder, 2009; Seligman & Csikszentmihalyi, 2000; Snyder & Lopez, 2002). These will briefly be reviewed in keeping with the goals of this chapter.

Keyes (2009), in outlining the criteria for mental health, identified a number of features, including the role of self-acceptance. In the terminology of this chapter, the I-self accepts the me-self, exuding confidence and appreciation for the me-self, without shading into narcissism. The I-self is happy with the me-self, proud of the me-self, although conversely, angry at, or disappointed with, the me-self. In the extreme, such negativity can lead to several forms of psychopathology that have been identified.

Historically, the field of psychopathology has been devoted to a consideration of dysfunctional, negative emotions (see Freud, 1905; Saarni et al., 2006). Emotions such as depression, anxiety, guilt, and shame have been viewed as debilitating. Thus, efforts, initially through psychotherapy but more recently through various prescription drugs, have been designed to reduce negative emotions, as the pathway to mental health. There are two limitations to this approach. From a medical perspective, often pills are not the answer. We do need to respect the fact that negative emotions do have an evolutionary function. Fear alerts us to danger, anger, at an appropriate level, can mobilize us to productive action, and yes, depression, in some circumstances such as grief, can help us to cope with losses.

I am reminded of a compelling popular magazine story (not a research study but a worthy topic.) A mother had lost her 10-year-old son in an unfortunate traffic accident. She went to see a psychiatrist for help in dealing with the tragedy. He proscribed Prozac (the popular drug at the time), and she indeed did feel less depression. Ultimately she resented the medication because it robbed her of the opportunity to appropriately grieve the loss of her son. So we should not be so eager to blot out the negative emotions as the focus of a translational intervention or treatment.

More recently, there has been a shift toward the functional role of *positive* emotions, long ignored as frivolous or irrelevant reactions. Scholars have taken seriously their function. Solid research is proliferating. This is not, in the words of a song some years ago: Don't worry, be happy. Nor is it a girlfriend's advice to just go shopping, buy new shoes, and you'll feel better. I next turn to this new literature, which thoughtfully addresses and researches the functional role of positive emotions.

The Role of Positive Emotions

Frederickson and colleagues, in identifying key components of mental health, from the perspective of self psychology, focus on the role of positive emotions (see Cohn & Frederickson, 2009; Frederickson, 1998). They put forth what is labeled a Broaden-and-Build Theory of positive emotions.

The *broadening* function, an I-self process, refers to the claim that positive feelings serve to widen the array

of thoughts and actions available to the individual, they produce more flexible response tendencies and solutions to life's challenges. As a result of a broadened mind set, one has access to more personal coping resources, which, in turn, enhance the likelihood of success in the future. This broadening function makes the individual more receptive or open to new information and promotes enhanced creativity (see also Isen's, 2000, discussion of the role of positive affect).

According to the *build* function of this theory, once positive emotions broaden the range of potential solutions then the individual's I-self can build upon these options in order to construct enduring personal resources. This building process provides a source of agency for achieving important life goals. Findings from Cohn and Fredericksson (2009) reveal that positive emotions are associated with the ability to develop a more long-range perspective that facilitates the purposeful development of plans and aspirations for the future. Thus, positive emotions can assist the individual in facing and coping with life stressors and thus in promoting mental health.

Positive Affectivity

In a similar vein, Watson and Naragon (2009) invoked the concept of *positive affectivity*, a relatively stable trait that also facilitates well-being. These theorists relate positive affectivity to what has been labeled the big five factor model of personality (see Watson & Clark, 1992). Of particular relevance to a treatment of positive I-self functions is the dimension of *openness to experience*. That is, the I-self not only directs attention to the me-self but to the outer world. It observes a beautiful sunset, it appreciates the beauty to be found in other people. Those high on positive affectivity are more likely to be open to such experiences, furthering a sense of subjective well-being.

Emotional Intelligence

The role of affect in promoting mental health can also be observed in the construct of emotional intelligence (Salovey, Mayer, Caruso, & Yoo, 2009), another theme within the positive self psychology movement. Four processes are key to this formulation that are consistent with additional functions of the I-self:

1. *Perceiving emotions* (e.g., the ability to accurately identify emotions in the self and others)
2. *Understanding emotions* (e.g., the ability to identify the causes and consequences of emotions)
3. *Employing emotions to facilitate problem solving* (e.g., applying creative solutions)
4. *Managing emotions* (e.g., the ability to be open to feelings, both pleasant and unpleasant and utilize them productively)

Emotional Competence

Emotional intelligence bears a close resemblance to the construct of *emotional competence* (Saarni, 1999). The difference is that emotional competencies are graded along a developmental continuum from the most basic to the more mature and complex abilities. Eight levels are identified. At *level 1*, the capacities involve an awareness of one's emotional states, including the possibility that one may experience multiple emotions (see also Harter, 2012). At *level 2,* the individual must develop skills to discern others' emotions based upon situational and expressive clues upon situational and expressive clues that require some degree of cultural consensus as to their emotional meaning. At *level 3,* an important component to emotional competence is the mastery of a culturally relevant vocabulary of emotion labels, namely, verbal facility with commonly employed emotion terminology. At *level 4*, emotional competence also makes reference to the capacity for empathic and sympathetic involvement in another's emotional experiences. At *level 5,* one must further develop skills in understanding that one's inner emotional state need not correspond to its outer expression, both in oneself and others. At *level 6,* emotional competence also refers to the development of skills to cope adaptively with aversive emotions and distressing circumstances by using self-regulatory strategies that weaken the intensity, frequency, or temporal duration of negative emotional states that are destructive. At *level 7*, individuals must come to realize that the nature of a relationship is largely defined by how emotions are communicated within that relationship, where ideally one displays genuineness and authenticity in the expressive display of emotions. At *level 8*, the highest level of emotional competence, *emotional efficacy* is the designated goal. The individual at this level of maturity is very much in touch with his or her emotions and can understand, accept, and respect his or her emotional experiences. Moreover, a person at this level develops a moral belief system about what constitutes the desirable emotional balance in a relationship and strives to meet that goal.

All of these skills, which I would argue demand the attention of the I-self, evolve over time, just as our initial cognitive-developmental analysis described how I-self

functions develop with age as cognitive-developmental skills build upon one another over the six stages that were identified in the overall sequential framework. The developmental acquisition of these emotional competencies, like the mastery of the skills identified under *emotional intelligence*, are viewed as essential components that support mental health and subjective well-being.

These levels have profound implications for translational or clinical interventions. They provide a blueprint for a sequence of states through which one must be guided, somewhat independent of age. One must first assess where an individual is on this progression of stages. Here, it will be important to determine, empirically, how just to assess one's position on this hierarchy, a challenging research task. Is it a developmental hierarchy, first, and how does one investigate this claim? This is the first challenge. It is an appealing notion, particularly because of its translational or clinical implications. But how can such a sequence be empirically validated, across developmental levels? A talented psychologist or psychiatrist might be able, in a clinical interview, to estimate at what level an individual may be, in terms of their emotional skill level. However, in order for this framework to be more useful to many, there must be a thoughtful research program to justify these interesting claims that truly broaden our developmental thinking.

Another new direction might be whether an individual's level along this hierarchy is associated with particular forms of developmental psychopathology. This would represent an invaluable connection between developmental thinking and individual differences that are captured in our categorization of psychopathological disorders. For example, are sociopathic individuals arrested at a particular developmental level? Are borderline pathologies associated with a given level of emotional understanding? This type of union would be welcome in integrating developmental approaches with the developmental psychopathological identification of specific syndromes. Treatment implications might follow.

Emotional Creativity

A related construct put forth by Averill (2009), in support of the tenets of positive psychology, is emotional creativity. There are features in common with emotional intelligence and emotional competence, although there are some nuances that include a focus on creativity as applied to emotions. Two of the components share features in common with these other perspectives. For example, one dimension is labeled Preparation where sample items on an assessment instrument include: When I have strong emotional reactions, I search for the reasons for my feelings; I pay attention to other people's emotions so that I can better understand my own. A second dimension is labeled Effectiveness/Authenticity, where sample items include: My emotions help me achieve my goals in life; The way I experience and express my emotions helps me in my relationships with others; My outward emotional reactions accurately reflect my inner feelings. The third dimension is labeled Novelty and seems to distinguish it from the other frameworks in bringing in the issue of *creativity*. Sample items here include: My emotional reactions are different and unique; I have felt combinations of emotions that other people probably have never experienced: I sometimes experience feelings and emotions that cannot be easily described in ordinary language; I like to imagine situations that call for unusual, uncommon, or unconventional emotional reactions.

The application of this formulation, particularly the dimension involving creativity and novelty sets this framework apart from the previous conceptualizations that related emotional skills and competencies to greater mental health. Averill (2009) discussed these issues in the context of some pathological conditions where the components have been analyzed, and delved into the links between dimensions of spirituality and mysticism, a fascinating direction. The pattern of findings is mixed, although those identifying themselves as mystics score higher on the novelty dimension of emotional experience. The picture is less clear for the one neurotic complex condition selected. However, this work vaults us into the next topic on the issue of whether creativity, outside of the emotional domain, contributes to mental health or its opposite. These arguments will next be reviewed, including how some have seemed to resolve the controversies in a meaningful manner.

Creativity

The more general issue of creativity is relevant to the field of positive psychology as well as to the framework of this chapter, in terms of I-self and me-self functions. It would seem that the I-self is the creative engine, looking inward, looking to the outer world, being open to experience, and trying to balance these observational perspectives. However, as Simonton (2009) cogently argued, there are some controversies in the domain of creativity that must be resolved, where I think the ensuing discussion is reasonably successful.

From a historical perspective, Guilford (1967) was one of thoughtful intellects who made the distinction between

divergent thinking and *convergent* thinking. Divergent thinking was the ability to generate many alternative ides of considerable variety and originality. These were cognitive spewed out, put on the intellectual table, as it were, to later be sifted through for merit and logic and selecting accordingly, the latter convergent process. However, divergent thinking was equated with creativity. (This was the expansive first draft phase as it were, of ideas, drawings, musical notes, and the list goes on, though it depended upon the quality of what was generated! And who was doing the judging, the self or others, and just which others?)

Simonton (2009) first came up with a general observation that creative people tend to have a wide range of interests, greater openness to new experiences, seek more such opportunities, and display more cognitive flexibility and boldness. However, here comes the controversy. Does creativity facilitate mental health? Or, alternatively does it represent that fine line between genius and insanity that puts many so-called creative geniuses over the edge into the land of pathology?

Simonton (2009) attempted to solve the dilemma by postulating two forms of creativity. There is what is termed a *small-c* version of creativity in which those of us have creative flashes that facilitate our problem-solving activities at home, in school, in our work, and are sources of pleasure and pride. (I wasn't thrilled with the terminology because for those of us who can generate such creative flashes in our own home or school or work, it can feel like a big "ahha" experience that is very gratifying. It is no small feat. But I understand the point.) And these experiences may well contribute to subjective well-being and mental health.

The contrast is Big-C creativity, which is demonstrated by those few who make grand creative contributions in their given field (art, literature, music, intellectual endeavors, inventions), contributions to culture, to history, to their own posterity. We will know their names (they have not known ours!). Yet so often, these contributors, the Mozart, Bethoven, many artists, DaVinci, Van Gogh, intellectual giants, Copercnious, Einstein, Darwin, etc. there looms this fine-line argument, were they geniuses, yes, were they also mentally unstable, perhaps. Did the genius contribute to pathology, or did the pathology contribute to the genius? The age-old problem of directionality in psychology where it still difficult to tease about just which caused which?

But this distinction between small-c and Big-C creativity partially separates those for whom creativity can be in the service of mental health (small-c processes) from those in the Big-C category striving to take their art to the big worldwide stage where they want to make a major mark in terms of cultural and historical contributions. And mentally suffer the consequences. If I could be so bold as to make a suggestion, within the humble framework I have tried to develop, I would conjecture that those wanting to be on the big stage of life and culture and the world, making an impact, have had an I-self that eventually becomes overwhelmed, first trying to open the artist to experiences that facilitate his or her craft but then must be totally devoted to packaging the me-self in a manner that will be received favorably by larger and larger audiences on the world's stage. And that contributes to their undoing, ultimately. Obviously, there are trait-like temperamental variables that they bring to their enterprises, as well as socialization experiences, child-rearing, but ultimately when the me-self looks so large that the I-self can no longer protect it, then narcissism and other pathologies creep in.

As an example, I draw upon a marvelous opportunity we in Denver had at the Denver Art Museum this fall and winter. They brought in a most exquisite display of van Gogh's works, dozens of original works brought in from all over the world, filling several rooms, with an excellent audio description of the exhibit where the works were arranged chronologically. Kudos to Christoph Heinrich, the director of the Denver Art Museum, Kira van Lil, an art history professor, and Timothy Standring, the show's curator, and their staff, for a superb and stunning exhibit.

Although the text of the various paintings did not attempt to intentionally pathologize van Gogh, I attended the event with a colleague who is an astute clinician, Shauna Ries, and brought my own psychological lens—it is hard not to in our professions. Van Gogh has certainly been the object of speculation as to his mental health for various reasons, the very nature of his paintings, his bouts of anxiety, his cutting off an ear, his very ungratifying social and intimate life, and his early, untimely, death. My colleague, by the end of the exhibit, studying the various paintings with this background knowledge, came to a possible speculation that he suffered from a pretty severe case of obsessive-compulsive disorder that might have contributed to his reported distress; she felt it in his paintings. We also found it interesting that his many self-portraits, which occurred during the three years preceding his death, might have portended his own prediction of his death, by a self-inflicted gun shot to the chest that was either a suicidal gesture or an accident, as some biographers claim. We speculated that he wanted to leave or preserve a visual memory of his visage for centuries to come, to personalize his legacy, in perpetuity. Biographers say he painted them looking into the mirror, the ultimate me-self obsession.

The point of this excursion into van Gogh was just to bring to life the issue of various forms of creativity some of which might support mental health or alternatively might be more associated with forms of psychopathology that, combined with genius, led to distress for these individuals. Yet we reap the benefits of many of their artistic and other works. The bottom line is that we must value the creativity in our own lives, using it to our greatest advantage to solve intellectual, work-related, and relational challenges toward the goal of subjective well-being and mental health. This will not preclude our fascination with those whose creativity took them over the top, ultimately, for our own cultural, historical, and artistic enjoyment, but at what price to these creators? It would seem that we need both forms of creativity for cultures to successfully survive. And emotional constructs must head the list in terms of these considerations.

POSITIVE PSYCHOLOGY'S EMPHASIS ON ISSUES RELATED TO PERCEPTIONS OF CONTROL: COGNITIVE PLUS EMOTIONAL COMPONENTS

Several themes in the most recent treatments of positive psychology (see Lopez & Snyder, 2009) would appear to echo earlier treatments in this chapter about bringing the I-self's role into mental health considerations. Thus, just the overlap will be briefly presented.

Sense of Control

Thompson (2009) has written about the concept of personal control, which is closely related to self-efficacy (Bandura, 2006). The definition of a sense of control is the judgment that one has the means to obtain desired outcomes and to avoid undesirable outcomes. Thompson wrote that the beneficial features of a sense of control have evolved through evolution, linking it to positive emotions. People who experience positive emotions and a sense of well-being when they had control were more likely to manipulate the environment in ways that increased the chances that their offspring would survive. Those with a drive for control were morel likely to survive and pass on their genes, resulting in an innate desire for control. As such, with the I-self at the helm, control can be a positive force for mental health outcomes and should be fostered through formal and informal interventions, in the therapy room and the classroom.

Self-Determination

Another related construct is self-determination, which is rooted in concepts about self-agency, a Jamesian I-self function (see chapter on self-determination by Wehmeyer, Little, & Sergent, 2009). Little, Hawley, Henrich, and Marsland (2002) defined human agency as "... the sense of personal empowerment which involves both knowing and having with it takes to achieve one's goal" (p. 390). These concepts merge with Deci and Ryan's (2002) focus on the human needs for competence, autonomy, and relatedness. For Deci and Ryan, the socializing environment (e.g., child rearing, schooling) can thwart or contribute to displays of self-determination. People who are causal agents, exhibiting I-self functions, can direct their energies toward achieving or maintaining desired outcomes. As such, the construct of self-determination has much in common with the next topics to be discussed, hope and optimism, more characterized as cognition-emotion hybrids that will contribute to positive mental health.

Optimism

Carver, Scheier, Miller, and Fulford (2009) put a potentially more emotional face on issues of efficacy and control. Still, there is a cognitive focus on expectations for the future, reflecting the pursuit of goals, including desired states and actions. There is an associated expectancy or confidence that the goal can be obtained. Realistic optimism is associated with subjective well-being and enhanced coping skills, both predictive of positive mental health. Pollyanna visions of success, and excessive spin, as we see from politicians, will, in the long run, undermine adaptive outcomes.

Hope

Hope (Rand & Cheavens, 2009) is another construct that is closely aligned with optimism and a sense of agency (Snyder, 1994). An important principle of Snyder's thinking requires the perceived ability to first generate routes to a goal and then execute the determination to successfully bring about that outcome. To the extent that there is a hopeful outcome that is achieved, it will contribute to feelings of well-being. Hope theory shares in common many of the features expressed in the treatment of positive emotions, and thus I will not attempt to be redundant.)

Curiosity

Curiosity qualifies as another I-self function that can impact well-being, and is cited in treatments of the positive

psychology movement. The curious person is open to new experiences and seeks novelty and challenge. As such, theorists have likened it to dimensions of flow, as earlier discussed, and viewed it as another dimension of intrinsic motivation (see Harter, 1981, 2012; Kashdan & Silva, 2009). Curiosity evokes interests and behaviors that are pleasurable, in and of themselves, often stretching the person to new heights. Upon seeking and investing efforts in novel and challenging activities, people with greater curiosity expand their knowledge, skills, goal-directed efforts, and self-awareness (Ainley, Hidi, & Berndorff, 2002). As such, it contributes to well-being.

A complete review of all of the processes that define the emerging emphasis on positive psychology is beyond the scope of this chapter. However, the interested reader is directed toward Lopez and Snyder (2009), where other topics such as forgiveness, gratitude, resilience, and love are also explored.

CONCLUSIONS

This contemporary excursion into James's (1892) insightful recognition of the I-self, for too long put on the intellectual shelf, has been something of a roller-coaster ride. It began with a developmental perspective, describing how the I-self, defined as changing cognitive abilities, shapes the me-self across three stages of childhood and three stages of adolescence. More positive thinking then addressed those positive I-self functions identified by James, which included self-awareness, a sense of agency, the continuity of the self, and its companion self-coherence. Although James intended these terms to reflect the adult self, their origins in this chapter are traced across developmental level. However, the point was made that I-self processes do not develop in a vacuum; the child's socialization experiences impact how these processes unfold. Because the I-self has been neglected, particularly from a developmental perspective, considerably more research needs to be conducted to unravel just how the I-self is constructed. Moreover, its various functions require further investigation because it is the cognitive engine that drives the construction of the me-self (see Harter, 2012).

Next, ensued a discussion of the dark side of the demands put upon the I-self in contemporary culture, tracing at each of these same developmental level whether and how the I self had the motives and skills to engage in the self-enhancement tactics and self-serving biases that are observed to exist in adults. Beginning in midadolescence, teenagers do begin to develop the adult-like capabilities, often exaggerated, which can shade into narcissism, including other pathological forms. This provided a segue into an application of self-processes to severe forms of developmental psychopathology, invoking I-self/me-self functions. Five different applications were presented, taking a developmental approach across three stages of childhood and three stages of adolescence: (1) self-aggrandizement, self-serving biases, self-deception, and narcissism; (2) negative self-evaluations in the form of low self-esteem and poor self-concept; (3) depressive reactions, syndromes, and suicidal behaviors; (4) eating-disordered behaviors; and (5) the role of negative self-processes that can lead to violent revenge in the face of bullying. However, these will require further empirical work, moving beyond mere speculation.

Yet a resurrection of the I-self can end on a note of hope, as I have demonstrated. For this reason, discussion turned to many positive functions that the I-self can perform in the service of mental health and well-being: self-awareness, self-efficacy, sense of agency, mastery motivation, the concept of flow, self-continuity and self-coherence, and mindfulness, from a Buddhist perspective, which includes a focus on egolessness, the quiet ego, humility, and compassion. From a Buddhist perspective, far more fringe benefits will accrue if the I-self averts its myopic gaze away from protecting the me-self through a distorted lens of self-aggrandizement. Consistent with the premises of the field of positive psychology (see Lopez & Snyder, 2009; Snyder & Lopez, 2002) more beneficial outcomes will ensue from an exploration of the I-self's role in promoting *authenticity*, which, in today's Western world, unfortunately seems to be in short supply. These topics, as well, require empirical verification rather than philosophical speculation.

From this relatively new field of positive psychology (see Lopez & Snyder, 2009), other constructs were selected that can be interpreted within an I-self/me-self perspective, highlighting the role of the I-self in promoting mental health and well-being: self-acceptance, the role of positive emotions, positive affectivity, emotional intelligence, emotional competence, emotional creativity, cognitive creativity, sense of control, self-determination, optimism, hope, and curiosity.

Obviously, a comprehensive review of the many features that define the positive psychology literature is beyond the scope of this chapter, as this comes to a close. However, it is noteworthy that these contemporary antidotes to the negative characterization of the self-serving ego represent a

breath of fresh air in terms of resurrecting the I-self. They provide a needed sense of balance, including the search for authenticity, themes that should guide further inquiry and soften our indictment of the me-self, which has become victimized and held hostage by contemporary society, casting the I-self as a potential villain. However, further research is needed before the I-self can be vindicated.

Coda: Putting the I-Self in Perspective

I have asked myself, in crafting this chapter, whether I am reinventing an historical wheel, namely, the age-old homunculous that those in my generation were warned against in Psychology 101. Originally invoked in the practice of Alchemy, scholars conjured up the concept of a little internal man who was infused with the power of directing our thoughts, perceptions, feelings, and behaviors. This notion engendered some appeal in early 1800 theories of psychology, prior to more sophisticated theories of mind. Serious debate ensued in the literature and the notion was eventually defeated, the arguments were deemed fallacious and circular, by the intellectuals who later confronted these issues more empirically (see Ryle, 1949).

I bring this up because in resurrecting the I-self, I do not believe that I am regressing to such earlier thoughts. However, I do tend to personalize the I-self in my writing, because I believe that it is a phenomenological experience in people's lives. As such, it tells a compelling story. Too many psychologists have paid lip service to James's (1892) dichotomy, as some sort of academic reverence, but finding little usefulness in their own work. Forgive me for trying to make it relevant, but it is very useful distinction from a developmental perspective. It has clinical implications, for example, asking clients to reflect on why the I-self is constructing negative me-self self-evaluations. There is also much research needed in terms of further investigating the processes underlying the construction of the me-self, if we truly give the I-self its due.

Moreover, I think we can relate to this distinction given our own personal experiences. I would ask the reader to think about the following: Have you ever said to yourself: I'm so mad at myself, I am so disappointed in myself, I am really proud of myself, I am so happy with myself for what I just accomplished. Or, on a really pleasant social or intimate occasion, we fleetingly observe, I really felt like myself. So let us ask ourselves, Just who is the I doing the talking here? And just who is the object of this reflection? I rest my case! I hope James is smiling in his grave. Or perhaps he and his thinking, in the eyes of contemporary scholars, have been resurrected.

REFERENCES

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness and depression: A theory-based subtype of depression. *Psychological Review*, *96*, 358–372. doi: 10.1037/0033-295X.96.2.358

Ainley, M., Hidi, S., & Berndorff, D. (2002). Interest, learning, and the psychological processes that mediate their relationship. *Journal of Educational Psychology*, *94*, 545–561. doi: 10.1037/0022-0663.94.3.545

Alicke, M., & C. Sedikides (Eds.). (2010). *Handbook of self-enhancement and self-protection*. New York, NY: Guilford Press.

Allport, G. W. (1961). *Pattern and growth in personality*. New York, NY: Holt, Rinehart, & Winston.

Almeida, A., Correia, I., Marino, S., & D'Jamila, G. (2012). Virtual but not less real: A study of cyberbullying and its relations to moral disengagement and empathy. In L. Quin, D. Cross, & P. K. Smith (Eds.), *Cyberbullying in the global playground: Research from international perspectives* (pp. 223–244). New York, NY: Wiley-Blackwell.

Anderson-Fye, E. P., & Becker, A. E. (2004). Sociocultural aspects of eating disorders. In J. K. Thompson (Ed.), *Handbook of eating disorders and obesity* (pp. 565–589). New York, NY: Wiley.

Anthony, E. J. (2002). Normal adolescent development from a cognitive viewpoint. *Journal of American Academy of Child Psychiatry*, *21*, 318–327.

Au, A., Lau, S., & Less, M. T. Y. (2012). Suicide ideation and depression: The moderation effects of family cohesion and social self-concept. *Adolescence*, *44*, 851–868.

Averill, J. R. (2009). Emotional creativity: Toward "spiritualizing the passions." In C. R. Snyder & S. J. Lopez (Eds.), *Oxford handbook of positive psychology* (pp. 249–257). Oxford University Press.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change *Psychological Review*, *84*, 191–215. doi: 10.1037/0033-295X.84.2.191

Bandura, A. (2006). Toward a psychology of human agency. *Perspectives on Psychological Science*, *1*, 164–180. doi: 10.1111/j.1745-6916.2006.00011.x

Bardenstein, K. K. (2009). The cracked mirror: Features of narcissistic personality disorder in children. *Psychiatric Annals*, *39*, 147–155.

Battle, J. (1987). Relationship between self-esteem and depression among children. *Psychological Reports*, *60*, 1187–1190.

Bauer, J. J. (2008). How the ego quiets as it grows: Ego development, growth stories, and eudaimonic personality development. In H. A. Wayment & J. J. Bauer (Eds.), *Transcending self-interest: Psychological explorations of the quiet ego* (pp. 199–210). Washington, DC: American Psychological Association.

Bauer, J. J., and Wayment, H. A. (2008). The psychology of the quiet ego. In H. A. Wayment & J. J. Bauer (Eds.), *Transcending self-interest: Psychological explorations of the quiet ego* (pp. 7–20). Washington, DC: American Psychological Association.

Baumeister, R. F. (1990). Suicide as escape from self. *Psychological Review*, *97*, 90–113.

Baumeister, R. F. (Ed.). (1993). *Self-esteem: The puzzle of low self-regard*. New York, NY: L. Plenum Press.

Beck, A. T. (1975). *Depression: Causes and treatments*. Philadelphia, PA: University of Pennsylvania Press.

Bellmore, A. D., & Cillessen, A. H. (2006). Reciprocal influences of victimization, perceived social preference, and self-concept in adolescence. *Self and Identity*, *3*, 209–229.

Bem, S. (1985). Androgeny and gender schema theory. In T. B. Sonderegger (Ed.), *Nebraska symposium on motivation* (Vol. 32, pp. 180–226). Lincoln, NE: University of Nebraska Press.

Benson, H. (1975). *The relaxation response*. New York, NY: William Morrow.

Benson, H.(1985). *Beyond the relaxation response*. New York, NY: Times Books.

Berg, D., & Klinger, D. A. (2009). Gender differences in the relationship between academic self-concept and self-reported depressed mood in school children. *Sex Roles*, *61*, 501–509. doi: 10.1007/s11199-009-9652-2

Bergstrom, R. L., Neighbors, C., & Malheim, J. E. (2009). Media comparisons and threats to body image: Seeking evidence of self-affirmation. *Journal of Social and Clinical Psychology*, *28*, 264–280. doi: 10.1521/jscp.2009.28.2.264

Berry, J. W. (2005). Acculturation. In W. Friedlmeier, P. Chakkarath, & B. Schwartz (Eds.), *Culture and human development*. New York, NY: Psychology Press.

Bertenthal, B. L., & Fischer, K. W. (1978). Development of self-recognition in the infant. *Developmental Psychology*, *14*, 44–50.

Bibring, E. (1953). The mechanism of depression. In P. Greenacre (Ed.), *Affective disorders: Psychoanalytic contribution to their study* (pp. 13–48). New York, NY: International Universities Press.

Blatt, S. J. (1974). Levels of object representation in anaclitic and introjective depression. *Psychoanalytic Study of the Child*, *29*, 107–157.

Bleiberg, E. (1984). Narcissistic disorders in children. *Bulletin of the Menninger Clinic*, *48*, 501–517.

Bleiberg, E. (1994). Normal and pathological narcissism in adolescence. *American Journal of Psychotherapy*, *48*, 29–51.

Bretherton, I., & Beeghly, M. (1982). Talking about internal states: The acquisition of an explicit theory of mind. *Developmental Psychology*, *18*, 529–548.

Briere, J. (1992). *Child abuse trauma: Theory and treatment of the lasting effects*. Newbury Park, CA: Sage.

Brighi, A., Melotti, G., Guarini, A., Genta, M. L., Ortega, R., Mora-Merchan, J.A., Smith, P. K., & Thompson, F. (2012). Self-esteem and loneliness in relation to cyberbullying in three European countries. In Q. Li, D. Cross, & P. K. Smith (Eds.), *Cyberbullying in the global playground: Research from international perspectives*. Malden, MA: Blackwell.

Bukowski, W., M., Schwartzman, A., Santo, J., Bagwell, C., & Ryan, A. (2009). Reactivity and distortions in the self: Narcissism, types of aggression, and the functioning of the hypothalamic-pituitary-adrenal axis during early adolescence. *Development and Psychopathology*, *21*, 1249–1262. doi: 110.1017/S0954579409990149

Bushman, B. J., & Thomaes, S. (2001). When the narcissistic ego deflates, narcissistic aggression inflates. In W. K. Campbell & J. D. Miller (Eds.). *The handbook of narcissism and narcissistic personality disorder: Theoretical approaches, empirical findings, and treatments* (pp. 319–329). Hoboken, NJ: Wiley.

Cai, J., Kwan, V., & Sedikides, C. (2012). A sociocultural approach to narcissism: The case of modern China. *European Journal of Personality*, *26*, 529–535. doi: 10.1002/per.852

Campbell, W. K., & Buffardi, L. E. (2008). The lure of the noisy ego: Narcissism as a social trap. In H. A. Wayment & J. J. Bauer (Eds.), *Transcending self-interest*. Washington, DC: American Psychological Association.

Campbell, W. K., Goodie, A. S., & Foster, J. D. (2004). Narcissism, confidence, and risk attitude. *Journal of Behavioral Decision Making*, *17*, 297–311. doi: 10.1002/bdm.475

Cambron, M. J., Acitelli, L. K., & Pettit, J. W. (2008). When women's self-esteem hinges on relationships: A contingent self-esteem model of gender differences in depression. In P. R. Bancroft (Ed.), *Major depression in women* (pp. 123–136). Hauppauge, NY: Nova Biomedical Books.

Carlson, K. S., Gjerde, P. F. (2009). Preschool Personality Antecedents of Narcissism in Adolescence and Emergent Adulthood: A 20-Year Longitudinal Study. *Journal of Research in Personality*, *43*(4): 570–578.

Carver, C. S., Scheier, M. F., Miller, C. J., & Fulford, D. (2009). Optimism. In C. R. Snyder & S. J. Lopez (Eds.), *Oxford handbook of positive psychology* (pp. 303–312). New York, NY: Oxford University Press.

Case, R. (1985). *Intellectual development: Birth to adulthood*. New York, NY: Academic Press.

Case, R. (1991). Stages in the development of the young child's first sense of self. *Developmental Review*, *11*, 210–230. doi: 10.1016/0273-2297(91)90010-L

Case, R. (1992). *The mind's staircase*. Hillsdale, NJ: Erlbaum.

Chao, R. K. (1996). Chinese and European American mothers' views about the role of parenting in children's school success. *Journal of Cross-Cultural Psychology*, *27*, 403–423. doi: 10.1177/0022022196274002

Chandler, M. J., Lalonde, C. E., Sokol, B. W., & Hallett, D. (2003). Personal persistence, identity development, and suicide: A study of native and non-native North American adolescents. *Monographs of the Society for Research in Child Development*, *68*(2), Series No. 273, 1–130.

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597–600. doi: 10.1017/S0954579400007318

Cicchetti, D., & Toth, S. (2006). Developmental psychopathology and preventive intervention. In K. A. Renninger & I. E. Sigel (Volume Eds.) and W. Damon & R. M. Lerner (Editors-in-Chief), *Handbook of child psychology* (6th ed., Vol. 4, Child Psychology in Practice, pp. 497–547). New York, NY: Wiley.

Cohn. M. A., & Frederickson, B. L. (2009). Positive emotions. In S. J. Lopez & C. R. Snyder (Eds.), *Handbook of positive psychology* (pp. 13–24). Oxford, UK: Oxford University Press.

Combs, D. J. Y., Campbell, G., Jackson, M., & Smith, R. H. (2010). Exploring the consequences of humiliating a moral transgressor. *Basic and Applied Social Psychology*, *32*, 128–143. doi: 10.1080/01973531003738379

Cooley, C. H. (1902). *Human nature and the social order*. New York, NY: Charles Scribner's Sons.

Cooper, C., R., Grotevant, H. D., & Condon, S. M. (1983). Individuality and connectedness both foster adolescent identity formation and role taking skills. In H. D. Grotevant & C. R. Cooper (Eds.), *Adolescent development in the family: New directions for child development* (pp. 43–59). San Francisco, CA: Jossey-Bass.

Crescioni, A. W., & Baumeister, R. F. (2009). Alone and aggressive: Social exclusion impairs self-control and empathy and increased hostile cognition and aggression. In M. J. Monica (Ed.), *Bullying, rejection, and peer victimization: A social cognitive neuroscience perspective* (pp. 251–277). New York, NY: Springer Publishing Co.

Cross, S. E., & Gore, J. S. (2003). Cultural models of the self. In M. R. Leary & J. P. Tangney (Eds.), *Handbook of self and identity* (pp. 536–566). New York, NY: Guilford Press.

Csikszentmialyi, M. (1997). *Finding flow*. New York, NY: Basic Books.

Damon, W., & Hart, D. (1988). *Self-understanding in childhood and adolescence*. New York, NY: Cambridge University Press.

Deci, E. L., & Ryan, R. M. (2000). The "what" and the "why" of goal pursuits: Human needs and the self-determination of behavior. *Psychological Inquiry*, *11*, 227–268. doi: 10.1207/S15327965PLI1104_0

DeKlyen, M., & Greenberg, M. T. (2008). Attachment and psychopathology in childhood. In J. Cassidy & P. R. Shaver (Eds.), *Handbook*

of attachment: Theory, Research, and clinical applications (2nd ed., pp.1637–1665). New York, NY: Guilford.

Denmark, F., Klara, M., & Baron, E. (2008). Bullying and hazing: A form of campus harassment. In M. A. Paludi (Ed.), *Understanding and preventing campus violence* (pp. 27–40). Westport, CT: Praeger Publishers/Greenwood Publishing Group.

Dennison, S., & Stewart, A. (2006). Facing rejection: New relationships, broken relationships, shame, and stalking. *International Journal of Offender Therapy and Comparative Criminology*, *50*, 324–337. doi: 10.1177/0306624X05278077

Denson, T., F., DeWall, C. N., & Finkel. (2012). Self-control and aggression. *Current Directions in Psychological Science*, *21*, 20–25. doi: 10.1177/0963721411429451

Denson, T. F., Pederson, W. C., Friese, M., Hahm, A., & Roberts, L. (2011). Understanding impulsive aggression: Angry rumination and reduced self-control are mechanisms underlying the provocation-aggression relationship. *Personality and Social Psychology Bulletin*, *37*, 850–862. doi: 10.1177/0146167211401420

Dewall, C. N., Finke, E. J., & Denson, T. F. (2011). Self-control inhibits aggression. *Social and Personality Psychology Compass*, *5*, 458–472. doi: 10.1111/j.1751-9004.2011.00363.x

DeWall, C. N., Gilman, R., Sharif, V., Carboni, I., & Rice. (2012). Left out, sluggardly, and blue: Low self-control mediates the relationship between ostracisms and depression. *Personality and Individual Differences*, *53*, 832–837. doi: 10.1016/j.paid.2012.05.025

Diener, E., & Diener, M. (2009). Cross-cultural correlates of life satisfaction and self-esteem. In E. Diener (Ed.), *The collective works of Ed Diener* (pp. 71–91). New York, NY: Springer Science and Business Media.

Dittmar, H., Halliwell, E., & Stirling, E., (2009). Understanding the impact of thin medial models on women's body-focused affect. *The roles of thin-ideal internalization and weight related self-discrepancy activation in experimental exposure effects. Journal of Social and Clinical Psychology*, *28*, 43–72. doi: 10.1521/jscp.2009.28.1.43

Dunkley, D. M., Masheb, R. M., & Grilo, C. M. (2010). Childhood maltreatment, depressive symptoms, and body dissatisfaction in patients with binge eating disorder: The mediating role of self-criticism. *International Journal of Eating Disorders*, *43*, 274–281. doi: 10.1002/eat.20796

Dunn, J. (1988). *The beginnings of social understanding*. Cambridge, MA: Harvard University Press.

Dweck, C. S. (1999). *Self-theories: Their role in motivation, personality, and development*. Philadelphia, PA: Psychology Press.

Dweck, C. S., & Master, A. (2009). Self-theories and motivation: Students' beliefs about intelligence. In K. R. Wentzel & A. Wigfield (Eds.), *Handbook of motivation at school* (pp. 123–140). New York, NY: Routledge/Taylor & Francis Group.

Eccles, J. S., & Roeser, R. W. (2009). Schools, academic motivation, and stage-environment fit. In R. M. Lerner & L. Steinberg (Eds.), *The handbook of adolescent psychology* (3rd ed., Vol. 1: Individual Bases of Adolescent Development). New York, NY: Wiley.

Elison, J., & Harter, S. (2007). Humiliation: Causes, correlates, and consequences. In J. L. Tracy, R. W. Robins, & J. P. Tangney, *The self-conscious emotions: Theory and research* (pp. 310–329). New York, NY: Guilford Press.

Elkind, D. (1967). Egocentrism in adolescence. *Child Development*, *38*, 1025–1034.

Epstein, S. (1973). The self-concept revisited or a theory of a theory. *American Psychologist*, *28*, 405–416.

Epstein, S. (1981). The unity principle versus the reality and pleasure principles, or the tale of the scorpion and the frog. In M. D. Lynch, A. A. Norem-Hebeisen, & K. Gergen (Eds.), *Self-concept: Advances in theory and research* (pp. 82–110). Cambridge, MA: Ballinger.

Epstein, S. (1991). Cognitive-experiential self theory: Implications for developmental psychology. In M. R. Gunnar & L. A. Sroufe (Eds.), *Self processes and development: The Minnesota symposium on child development* (Vol. 23, pp. 111–137). Hillsdale, NJ: Erlbaum.

Exline, J. J. (2008). Taming the wild ego: The challenge of humility. In H. A. Wayment & J. J. Bauer (Eds.), *Transcending self-interest: Psychological explorations of the quiet ego* (pp. 53–62). Washington, DC: American Psychological Association.

Fennig, S., Hadas, A., Itzhaky , Roe, D., Apter, A., & Shahar, G. (2008). Self-criticism is a key predictor of eating disorder dimensions among inpatient adolescent females. *International Journal of Eating Disorders*, *41*, 762–765. doi: 10.1002/eat.20573

Ferrell-Smith, F. (2012). Bullying puts teens at risk. In C. Watkins (Ed.), *Teens at risk* (p. 51). Farmington Hills, MI: Greenhaven Press.

Fischer, K. F. (1980). A theory of cognitive development: The control and construction of hierarchies of skills. *Psychological Review*, *87*, 477–531. doi: 10.1037/0033-295X.87.6.477

Fischer, K. F., & Ayoub, C. (1994). Affective splitting and dissociation in normal and maltreated children: Developmental pathways for self in relationships. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium on developmental psychopathology: Disorders and dysfunctions of the self* (Vol. 5, pp. 149–222). Rochester, NY: University of Rochester Press.

Fischer, K. W., & Bidell, T. R. (2006). Dynamic development of action and thought. In N. Eisenberg (Volume Ed.), W. Damon, & R. Lerner (Editors-in-Chief), *Handbook of child psychology* (6th ed., Vol. 1, Theoretical models of human development, pp. 313–399). New York, NY: Wiley.

Fivush, R., & Haden, C. A. (Eds.), (2003). *Autobiographical memory and the construction of a narrative self*. Mahwah, NJ: Lawrence Erlbaum.

Fivush, R., & Hudson, J. A. (Eds.), (1990). *Knowing and remembering in young children*. New York, NY: Cambridge University Press.

Flavell, J. H. (1985). *Cognitive development* (2nd ed.). Englewood Cliffs, NJ: Prentice-Hall. *Personality, 37*, 469–486.

Frederickson, B. L. (1998). What good are positive emotions? *Review of General Psychology*, *2*, 300–319.

Freud, S. (1952). *A general introduction to psychoanalysis*. New York, NY: Washington Square Press.

Freud, S. (1968). Mourning and melancholia. In J. Strachey (Ed. and Trans.), *The standard edition of the complete works of Sigmund Freud* (Vol. 14, 237–260). London, UK: Hogarth Press. (Original work published 1917.)

Friedman, R. & Liu, W. (2009). Biculturalism in Management: Leveraging the Benefits of Intrapersonal Diversity, Understanding Culture: theory, Research and Applications. pp. 343–360.

Frijda, N. H. (1986). *The emotions*. Cambridge, UK: Cambridge Universities Press.

Garbarino, J. (1999). Lost Boys: Why Our Sons Turn to Violence and How We Can Save Them. *Reaching Today's Youth: The Community Circle of Caring Journal*, *3*(4), 7–10.

Geller, J., Srikameswaran, S., Cookell, S. J., & Zaitsoff, S. L. (2000). The assessment of shape and weight-based self-esteem in adolescents. *International Journal of Eating Disorders*, *28*, 339–345.

Gergen, K. (1991). *The saturated self: Dilemmas of identity in contemporary life*. Basic books.

Gilbert, P. (1997). The evolution of social attractiveness and its role in shame, humiliation, guilt and therapy. *British Journal of Medical Psychology*, *70*, 113–147. doi: 10.1111/j.2044-8341.1997.tb01893.x

Given-Wilson, Z., McIlwain, D., & Warburton, W. (2011). Meta-cognitive and interpersonal difficulties in overt and covert narcissism. *Personality and Individual Differences*, *50*, 1000–l005.

Gordon, K. H., & Dombeck, J. J. (2010). The associations between two facets of narcissism and eating disorder symptoms. *Eating Behaviors*, *11*, 288–292. doi: 10.1016/j.eatbeh.2010.08.004

Greenwald, A. G. (1980). The totalitarian ego: Fabrication and revision of personal history. *American Psychologist*, *7*, 603–618. doi: 10.1037/0003-066X.35.7.603

Griffin, S. (1992). Structural analysis of the development of their inner world: A neo-structural analysis of the development of intrapersonal intelligence. In R. Case (Ed.), *The mind's staircase* (pp. 189–206). Hillsdale, NJ: Erlbaum.

Guilford, J. P. (1967). *The nature of human intelligence*. New York, NY: McGraw-Hill.

Haden, C. A. (2003). Joint encoding and joint reminiscing: Implications for young children's understanding and remembering of personal experiences. In R. Fivush & C. A. Haden (Eds.), *Autobiographical memory and the construction of a narrative self* (pp. 49–70). Mahwah, NJ: Erlbaum.

Haltiwanger, J. (1989, April). *Behavioral referents of presented self-esteem in young children*. Paper presented at the meeting of the Society for Research in Child Development, Kansas City, MO.

Hamamura, T., Heine, S., & Paulhus, D. (2008). Cultural differences in response styles: The role of dialectical thinking. *Personality and Individual Differences*, *44*, 932–942. doi: 10.1016/j.paid.2007.10.034

Hammen, C., & Goodman-Brown, T. (1990). Self-schemas and vulnerability to specific life stress in children at risk for depression. *Cognitive Therapy and Research*, *14*, 215–227.

Harter, S. (1981). A new self-report scale of intrinsic versus extrinsic orientation in the classroom: Motivational and information components. *Developmental Psychology*, *17*, 300–312. doi: 10.1037/0012-1649.17.3.300

Harter, S. (1983). Developmental perspectives on the self-system. In P. Mussen & E. M. Hetherington (Eds.), *Handbook of child psychology: Vol. 4, Socialization personality, and social development* (4th ed., pp. 275–385). New York, NY: Wiley.

Harter, S. (1985). *The Self-Perception Profile for Children*. Unpublished manual, University of Denver, Denver, CO.

Harter, S. (1986). Processes underlying the construction, maintenance, and enhancement of the self-concept in children. In J. Suls & A. G. Greenwald (Eds.), *Psychological perspectives on the self* (Vol. 3). Hillsdale, NJ: Lawrence Erlbaum.

Harter, S. (1993). Causes and consequences of low self-esteem in children and adolescence. In R. F. Baumeister (Ed.), *Self-esteem: The puzzle of low self-regard* (pp. 87–116). New York, NY: Plenum.

Harter, S. (1998). The effects of child abuse on the self-system. In B. B. Rossman & M. S. Rosenberg (Eds.), *Multiple victimization of children: Conceptual, developmental, research, and treatment issues*. New York, NY: Haworth Press.

Harter, S. (1999). *The construction of the self*. New York, NY: Guilford Press.

Harter, S. (2006). Self-processes and developmental psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Handbook of developmental psychopathology* (2nd ed.). New York, NY: Wiley.

Harter, S. (2012). *The construction of the self: Developmental and sociocultural foundations* (2nd ed.). Guilford Press: New York.

Harter, S., Bresnick, S., Bouchey, H. A., & Whitesell, N. R. (1997). The development of multiple role-related selves during adolescence. *Development and Psychopathology*, *9*, 835–854.

Harter, S., & Buddin, B. (1987). Children's understanding of the simultaneity of two emotions: A five-stage developmental acquisition sequence. *Developmental Psychology*, *23*, 388–399. doi: 10.1037/0012-1649.23.3.388

Harter, S., Kiang, L., Whitesell, N. R., & Anderson, A. V. (2003, April). *A prototype approac to the emotion of humiliation in college students*. Poster session presented at the biannual meeting of the Society of Research in Child Development, Tampa, FL.

Harter, S., Low, S., & Whitesell, N. R. (2003). What have we learned from Columbine: The impact of the self-system on suicidal and violent ideation among adolescents. *Journal of Youth Violence*, *2*, 3–26. doi: 10.1300/J202v02n03_02

Harter, S., Marold, D. B., & Whitesell, N. R. (1992). A model of psychosocial risk factors leading to suicidal ideation in young adolescents. *Development and Psychopathology*, *4*, 167–188. doi: 110.1017/S0954579400005629

Harter, S., Marold, D. B., Whitesell, N. R., & Cobbs, G. (1996). A model of the effects of parent and peer support on adolescent false self behavior. *Child Development*, *67*, 360–374. doi: 10.1111/j.1467-8624.1996.tb01738.x

Harter, S., & Monsour, A. (1992). Developmental analysis of conflict caused by opposing attributes in the adolescent self-portrait. *Developmental Psychology*, *28*, 251–260.

Harter, S., Stocker, C., & Robinson, N. S. (1996). The perceived directionality of the link between approval and self-worth: The liabilities of a looking glass self orientation among young adolescents. *Journal of Research on Adolescence*, *6*, 285–308.

Harter, S., Waters, P., & Whitesell, N. R. (1997). False self behavior and lack of voice among adolescent males and females. *Educational Psychologist*, *32*, 153–173.

Harter, S., Waters, P., & Whitesell, N. R. (1998). Relational self-worth: Differences in perceived worth as a personal across interpersonal contexts. *Child Development*, *69*, 756–766. doi: 10.1111/j.1467-8624.1998.tb06241.x

Harter, S., & Whitesell, N. R. (1989). Developmental changes in children's understanding of single, multiple and blended emotion concepts. In C. Saarni & P. L. Harris (Eds.), *Children's understanding of emotion* (pp. 835–854). Cambridge, UK: Cambridge University Press.

Harter, S., & Whitesell, N. R. (1996). Multiple pathways to self-reported depression and adjustment among adolescents. *Development and Psychopathology*, *9*, 835–854.

Hartling, L. (2007). Humiliation: Real pain, a pathway to violence. *RBSE, Revista Brasileira de Sociologia da emoção 6*, 276–290.

Hartling, L. M., & Luchetta, T. (1999). Humiliation: Assess the impact of derision, degradation, and debasement. *Journal of Primary Prevention*, *19*, 259–278. doi: 10.1023/A:1022622422521

Heine, S. J. (2003). An exploration of cultural variation in self-enhancing and self-improving motivations. In R. A. Diensbier (Series Ed.), V. Murphy-Berman, & J. J. Berman (Volume Eds.), *Cross-cultural differences in perspectives on the self* (Vol. 49, pp. 101–128). Lincoln, NE: University of Nebraska Press.

Heine, S. J. (2007). Culture and motivation: What motivates people to act in the ways that they do? In S. Kitayama & D. Cohen, *Handbook of cultural psychology* (pp. 714–733). New York, NY: Guilford Press.

Heine, S. J., & Hamamura, T. (2007). In search of East Asian self-enhancement. *Personality and Social Psychology Review*, *11*, 1–24.

Heine, S. J., Lehman, D. R., Markus, H. R., & Kitayama, S. (1999). Is there a universal need for positive self-regard? *Psychological Review*, *106*, 766–794.

Higgins, E. T. (1989). Self-discrepancy theory: Why patterns of self-belief cause people to suffer? In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 22, pp. 22–63). New York, NY: Academic Press.

Higgins, E. T. (1991). Development of self-regulatory and self-evaluative processes: Costs, benefits, and tradeoffs. In M. R. Gunnar & L. A. Sroufe (Eds.), *Self-processes and development: The Minnesota symposium on child development* (Vol. 23, pp. 125–166). Hillsdale, NJ: Erlbaum.

Holfeld, B., & Grabe, M. (2002). An examination of the history, prevalence, characteristics, and reporting of cyberbullying in the United States. In L. Qing, D. Cross, & P. Smith (Eds.), *Cyberbulling in the global playground: Research from international perspectives* (pp. 117–142). New York, NY: Wiley-Blackwell.

Hong, Y., Morris, M. W., Chiu, C., & Benet-Martinez, V. (2000). Multicultural minds: A dynamic constructivist approach to culture and cognition. *American Psychologist*, *55*, 709–720.

Horney, K. (1950). *Neurosis and human growth*. New York, NY: Norton.

Houbre, B., Tarquinio, C., & Lanfranchi, J. (2010). Expression of self-concept and adjustment against repeated aggressions: The case of a longitudinal study on school bullying. *European Journal of Psychology of Education*, *25*, 105–123. doi: 10.1007/s10212-009-0005-x

Isen, A. M. (2000). Positive affect and decisions making. In M. Lewis & J. M. Haviland-Jones (Eds.). *Handbook of emotion* (2nd ed., pp. 417–435). New York, NY: Guilford Press.

Izard, C. E. Emotion-cognition relationships and human development. In C. E. Izard, J., Kagan, & R. B. Zajonc (Eds.), *Emotions, cognition, and behavior* (pp. 389–413). Cambridge, UK: Cambridge University Press.

Jackson, M. A. (2000). Distinguishing shame and humiliation. *Dissertation Abstracts International*, *61*(4), 2272. (UMI No. 9968089)

James, W. (1890). *Principles of psychology*. Chicago, IL: Encyclopedia Britannica.

James, W. (1892). *Psychology: The briefer course*. New York, NY: Henry Holt.

Jenkins, L. N., & Demaray. M. K. (2012). Social support and self-concept in relation to peer victimization and peer aggression. *Journal of School Violence*, *11*, 56–74. doi: 10.1080/15388220.2011.630958

Joffe, N. G., & Sandler, J. (1967). Some conceptual problems involved in the consideration of disorders of narcissism. *Journal of Child Psychotherapy*, *2*, 56–66.

Jung, C. G. (1928). *Two essays on analytical psychology*. New York, NY: Dodd, Mead.

Kanagawa, C., Cross, S. E., & Markus, H. R. (2001). "Who am I?" The cultural psychology of the conceptual self. *Personality and Social Psychology Bulletin*, *27*, 90–103. doi: 10.1177/0146167201271008

Kang, S., Shaver, P., Sue, S., Min, K., & Jing, H. (2003). Culture-specific patterns in the prediction of life satisfaction: Roles of emotion, relationship quality, and self-esteem. *Personality and Social Psychology Bulletin*, *29*, 1596–1608. doi: 10.1177/0146167203255986

Kaplan, A., & Maehr, M. L. (2007). The contributions and prospects of goal orientation theory. *Educational Psychology Review*, *19*, 91–110.

Kashdan, T. B., & Silvia, P. J. (2009). Curiosity and interest: The benefits of thriving on novelty and challenge. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 367–374). Oxford, UK: Oxford University Press.

Kaslow, N. J., Rehm, L. P., & Siegel, A. W. (1984). Social-cognitive and cognitive correlates of depression in children. *Journal of abnormal child psychology*, *12*(4), 605–620.

Keilp, J. G., Grunebaum, Gorlyn, M., LeBlanc, S., Burke, A. K., Galalvy, H., Oquendo, M., & Mann, J. J. (2012). Suicidal ideation and the subjective aspects of depression. *Journal of Affective Disorders*, *140*, 75–81. doi: 10.1016/j.jad.2012.01.045

Kelly, G. A. (1955). *The psychology of personal constructs*. New York, NY: Norton.

Kernberg, O. F. (1975). *Borderline conditions and pathological narcissism*. New York, NY: Jason Aronson.

Kernberg, O. F. (1986). Further contributions to the treatment of narcissistic personalities. In A. Morrison (Ed.), *Essential papers on narcissism* (pp. 244–292). New York, NY: New York University Press.

Kernberg, P. E. (1998). Developmental aspects of normal and pathological narcissism. In E. F. Ronningstam (Ed.), *Disorders of narcissism: Diagnostic, clinical, and empirical implications* (pp. 103–120). Washington, DC: American Psychiatric Press.

Keyes, C. (2009). Toward a science of mental health. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 89–96). Oxford, UK: Oxford University Press.

Kiang, L., & Harter, S. (2006). Sociocultural values of appearance and attachment processes: An integrated model of eating disorder symptomatology. *Eating Behaviors*, *7*, 134–151. doi: 10.1016/j.eatbeh.2005.08.001

Kilbourne, J. (1999). *Deadly persuasion: Why women must fight the addictive power of advertising*. New York, NY: Free Press.

Kim, J., Kim, M., Kam, K., & Shin, H. (2003). Influence of self-construals on the perception of different self-perception styles in Korea. *Asian Journal of Social Psychology*, *6*, 89–101. doi: 10.1111/1467-839X.t01-1-00013

Kim, Y., & Sundar, S. S. (2012). Visualizing ideal self vs. actual self through avatars: Impact on preventive health outcomes. *Computers in Human Behavior*, *28*, 1356–1364. doi: 10.1016/j.chb.2012.02.021

Klein, D. C. (1991). The humiliation dynamic: An overview. *Journal of Primary Prevention*, *12*, 93–121. doi: 10.1007/BF02015214

Konig, A., Gollwitzer, M., & Steffgen, G. (2010). Cyberbullying as an act of revenge? *Australian Journal of Guidance and Counseling*, *20*, 210–224. doi: 10.1375/ajgc.20.2.210

Kovacs, M., & Beck, A. T. (1977). An empirical-clinical approach toward a definition of childhood depression. In J. G. Schulterbrandt & A. Raskin (Eds.), *Depression in childhood: Diagnosis, treatment, and conceptual models* (pp. 1–25). New York, NY: Raven Press.

Kovacs, M., & Beck, A. T. (1986). Maladaptive cognitive structures in depression. In J. C. Coyne (Ed.), *Essential papers on depression* (pp. 212–239). New York, NY: New York Universities Press.

Kring, A. M. (2008). Emotion disturbances as transdiagnostic processes in psychopathology. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., pp. 691–708). New York, NY: Guilford.

Lacey, D. (2011). The role of humiliation in the Palestinian/Israeli conflict in Gaza. *Psychology of Society*, *4*, 76–92.

Langer, E. (1989). *Mindfulness*. New York, NY: Da Capo Press/Perseus Books Group.

Langer, E. (2009). Mindfulness versus positive evaluation. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 279–294). New York, NY: Oxford University Press.

Lapsley, D. K., & Stey, P. C. (2012). Adolescent narcissism. In R. Levesque (Ed.), *Encyclopedia of adolescence*. New York, NY: Springer.

Larkin, R. W. (2009). The Columbine legacy: Rampage shootings as political acts. *American Behavioral Scientist*, *52*, 1309–1326. doi: 10.1177/0002764209332548

Leary, M. R. (2004). *The curse of the self: Self-awareness, egotism, and the quality of human life*. Oxford, UK: Oxford University Press.

Leary, M. R., Kowalski, R. M., Smith, L., & Phillips, S. (2003). Teasing, rejection, and violence: Case studies of the school shootings. *Aggressive Behavior*, *29*, 202–214. doi: 10.1002/ab.10061

Lecky, P. (1945). *Self-consistency: A theory of personality*. New York, NY: Island Press.

Lecours, S., Briand-Malenfant, R., & Descheneaux, E. (2013). Affect regulation and mentalization in narcissistic personality disorder. In J. S. Ogrodniczuk (Ed.), *Understanding and treating pathological narcissism* (pp. 129–145). Washington, DC: American Psychological Association.

Lee-Flynn, S., Pomaki, G., DeLongis, A., Biesanz, J. C., & Puterman, E. (2011). Daily cognitive appraisals, daily affect, and long-term depressive symptoms: The role of self-esteem and self –concept clarity in the stress process. *Personality and Social Psychology Bulletin*, *37*, 255–268. doi: 10.1177/0146167210394204

Leichtman, M. D., Wang, Q., & Pillemer, D. B. (2003). Cultural variations in interdependence and autobiographical memory: Lessons from Korea, China, India, and the United States. In R. Fivush & C. A. Haden (Eds.), *Autobiographical memory and the construction of a narrative self: Developmental and cultural perspectives* (pp. 73–98). Mahwah, NJ: Lawrence Erlbaum.

Leidner, B., Sheikli, H., & Ginges, J. (2012). Affective dimensions of intergroup humiliation. *Plus ONE*, *7*, 2012, 463–475.

Lerner, H. G. (1993). *The dance of deception*. New York, NY: Harper Collins.

Levine, M. P., & Harrison, K. (2004). In J. K. Thompson (Ed.), *Handbook of eating disorders and obesity. Media's role in the perpetuation and prevention of negative body image and disordered eating*, 695–717. New York, NY: Wiley.

Lewis, H. B. (1987). *The role of shame in symptom formation*. Hillsdale, NJ: Erlbaum.

Lewis, M. (2008). Self-conscious emotions: Embarrassment, pride, shame, and guilt. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., 742–756). New York, NY: Guilford.

Lewis, M., & Brooks-Gunn, J. (1979). *Social cognition and the acquisition of the self* . New York, NY: Plenum.

Lewis, M., & Haviland-Jones, J. M. (Eds.). (2000). *Handbook of emotions* (2nd ed.). New York, NY: Guilford.

Lewis, M., Haviland-Jones, J. M., & Barrett, L. F. (Eds.). (2008). *Handbook of emotions* (3rd ed.). New York, NY: Guilford.

Linville, P. W. (1987). Self-complexity as a cognitive buffer against stress-related illness and depression. *Journal of Personality and Social Psychology*, *52*, 663–676.

Little, T. D., Hawley, P. H., Henrich, C. C., & Marsland, K. Three views of the agentic self: A developmental synthesis. In E. L. Deci & R. M. Ryan (Eds.), *Handbook of self-determination research* (pp. 389–404). Rochester, NY: University of Rochester Press.

Lopez, S. J., & C. R. Snyder (Eds.). *Oxford handbook of positive psychology*. New York, NY: Oxford University Press.

Maddux, J. E., & Gosselin, J. T. (2003). Self-efficacy. In M. R. Leary & J. P. Tangney (Eds.), *Handbook of self and identity* (pp. 218–238). New York, NY: Guilford Press.

Maeda, K. (1997). *The Self-Perception Profile for Children administered to a Japanese sample*. Unpublished data, Ibaaraki Prefectural University of Health Sciences, Ibarki, Japan.

Malson, H. (2009). Appearing to disappear: Postmodern femininities and self-starved subjectivities. In H. Malson & M. Burns (Eds.), *Critical feminist approaches to eating disorders* (pp. 135–145). New York, NY: Routledge/Taylor & Francis Group.

Markus, H. R. (1980). The self in thought and memory. In D. M. Wegner & R. R. Vallacher (Eds.), *The self in social psychology* (pp. 42–69). New York, NY: Oxford University Press.

Markus, H. R., & Nurious, P. (1986). Possible selves. *American Psychologist*, *41*, 954–969.

Maslow, A. (1954). *Motivation and personality*. New York, NY: Harper & Row.

Mattaini, M. (2008). Creating a violence-free school climate/culture. In C. Franklin, M. B. Harris, & P. Allen-Meares (Eds.), *The school practitioner's concise companion to preventing violence and conflict* (pp. 27–39). New York, NY: Oxford University Press.

McCarley, K. (2009). *The humiliation experience: Causes, emotional correlates, and behavioral consequences*. Unpublished doctoral dissertation, University of Denver, Denver, CO.

McCrae, R. R. (2001). Trait psychology and culture: Exploring intercultural comparisons. *Journal of Personality*, *69*, 819–846.

McFarland, M. B., & Kaminski, P. L. (2009). Men, muscles, and mood: The relationship between self-concept, dysphoria, and body image disturbances. *Eating Behaviors*, *10*, 68–70. doi: 10.1016/j.eatbeh.2008.10.007

Mead, G. H. (1934). *Mind, self, and society from the standpoint of a social behaviorist*. Chicago, IL: University of Chicago Press.

Mehta, T. (2005). *Multiple selves in South Asian adolescents*. Unpublished doctoral dissertation. University of Denver, Denver, CO.

Meltzer, H., Vostanis, P., Ford, T., Bebbington, P., & Dennis, M. S. (2011). Victims of bullying in childhood and suicide attempts in adulthood. *European Psychiatry*, *26*, 498–503. doi: 10.1016/j.eurpsy.2010.11.006

Menesini, E., Calussi, P., & Nocentini, A. (2012). Cyberbullying and traditional bullying: Unique, additive, and synergistic effects on psychological health symptoms. In L. Qing, D. Cross, & P. K. Smith (Eds.), *Cyberbullying in the global playground: Research from international perspectives* (pp. 245–262). New York, NY: Wiley-Backwell.

Menesini, E., Nocentini, A., & Camodeca, M. (2013). Morality, values, traditional bullying, and cyberbullying in adolescence. *British Journal of Developmental Psychology*, *31*, 1–14. doi: 10.1111/j.2044-835X.2011.02066.x

Meredith, W. H., Abbott, D.A., & Zheng, F. M. (1991). Self-concept and sociometric outcomes: A comparison of only children and sibling children from urban and rural areas in the Peoples' Republic of China. *Journal of Psychology*, *126*, 411–421.

Mezulis, A., Abramson, L., Hyde, J., & Hankin, B. (2004). Is there a universal positivity bias in attributions? A meta-analytic review of individual, developmental, and cultural differences in the self-serving attributional bias. *Psychological Bulletin*, *130*, 711–747. doi: 10.1037/0033-2909.130.5.711

Mikulincer, M. (1997). Adult attachment style and information processing: Individual differences in curiosity and cognitive closure. *Journal of Personality and Social Psychology*, *72*, 1217–1230. doi: 10.1037/0022-3514.72.5.1217

Mikulincer, M. (1998). Adult attachment style and affect regulation: Strategic variations in self-appraisals. *Journal of Personality and Social Psychology*, *75*, 420–435. doi: 10.1037/0022-3514.75.2.420

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York, NY: Guilford Press.

Miller, D. N. (2012). Youth suicidal behavior in the context of school violence. In S. R. Jimmerson, A. B. Nickerson, M. Mayer, & M. J. Furlong (Eds.), *Handbook of school violence and school safety: International research and practice* (2nd ed.), pp. 203–214. New York, NY: Routledge.

Miller, S. (1988). Humiliation and shame: Comparing two affect states as indicators of narcissistic stress. *Bulletin of the Menninger Clinic*, *52*, 40–51.

Montague, M., Enders, C., Dietz, S., Dixon, J., & Cavendish, W. M. (2008). A longitudinal study of depressive symptomatology and self-concept in adolescents. *Journal of Special Education*, *42*, 67–78. doi: 10.1177/0022466907310544

Montemayor, R., & Eisen, M. (1977). The development of self-conceptions from childhood to adolescence. *Developmental Psychology*, *Vol. 13*(4), 314–319.

Morf, C. C., Horvath, S., & Torchetti, L. (2010). Narcissistic self-enhancement: Tale or (successful?) self-portrayal. In M. D. Alicke & C. Constantine (Eds.), *Handbook of self-enhancement and protection* (pp. 399–424). New York, NY: Guilford.

Nakamura. J., & Csikszemtmihali M. (2002). The concept of flow. In C. R. Snyder & S. Lopez (Eds.), *Handbook of positive psychology* (pp. 89–106). New York, NY: Oxford University Press.

Neff, K. D. (2008). Self-compassion: Moving beyond the pitfalls of a separate self-concept. In H. A. Wayment & J. J. Bauer (Eds.), *Transcending self-interest: Psychological explorations of the quiet ego* (pp. 95–106). Washington, DC: American Psychological Association.

Negrao, C., Bonanno, G., Noll, J., Putnam, F., & Trickett, P. (2005). Shame, humiliation, and childhood sexual abuse: Distinct contributions and emotional coherence. *Child Maltreatment*, *10*, 350–363. doi: 10.1177/1077559505279366

Nelson, K. (2003). Narrative and self, myth and memory: Emergence of the cultural self. In R. Fivush & C. A. Haden (Eds.), *Autobiographical memory and the construction of a narrative self* (pp. 3–28). Mahwah, NJ: Erlbaum.

Nelson, K., & Fivush, R. (2004). The emergence of autobiographical memory: A socio-cultural developmental theory. *Psychological Review*, *111*, 486–511. doi: 10.1037/0033-295X.111.2.486

Nelson, L. J. (2003). Rites of passage in emerging adulthood: Perspectives of young Mormons. *New Directions in Child and Adolescent Development*, *100*, 33–49.

Nezlek, J., Sorrentino, R., Yasunaga, S., Otsubo, Y., Allen, M., & Kouhara, S. (2008). Cross-cultural differences in reactions to daily events as indicators of cross-cultural differences in self-construction and affect. *Journal of Cross-Cultural Psychology*, *39*, 685–702. doi: 10.1177/0022022108323785

Nicholls, E., & Stukas, A. A. (2011). Narcissism and the self-evaluation maintenance model: Effects of social comparison threats on relationship closeness. *Journal of Social Psychology*, *15*, 201–212. doi: 10.1080/00224540903510852

Nisbett, R. E., Peng, K., Choi, I., & Norenzayan, A. (2001). Culture and systems of thought: Holistic versus analytic cognition. *Psychological Review*, *108*, 291–310. doi: 10.1037/0033-295X.108.2.291

Nishina, A. (2012). Microcontextual characteristics of peer victimization experiences and adolescents' daily well-being. *Journal of Youth and Adolescence*, *41*, 191–201. doi: 10.1007/s10964-011-9669-z

Noam, G. G., & Borst, S. (Eds.). (1994). *Children, youth and suicide: Developmental perspectives*. San Francisco, CA: Jossey-Bass.

Nolen-Hoeksema, S., Girgus, J. S., & Seligman, M. E. P. (1986). Learned helplessness in children: A longitudinal study of depression, achievement, and explanatory style. *Journal of Personality and Social Psychology*, *51*, 435–442.

Olweus, D. (2008). Social problems in school. In A. Slater & G. Bremner (Eds.), *An introduction to developmental psychology* (pp. 437–454). Malden, MA: Blackwell Publishing.

Paulhus, D. L., & Martin, C. L. (1988). Functional flexibility: A new conceptualization of interpersonal flexibility. *Journal of Personality and Social Psychology*, *55*, 88–101.

Perren, S., & Gutzwiller-Helfenfinger, E. (2012). Cyberbullying and traditional bullying in adolescence: Differential roles of moral disengagement, moral emotions, and moral values. *European Journal of Developmental Psychology*, *9*, 195–209. doi: 10.1080/17405629.2011.643168

Pfeffer, C. R. (1988). Risk factors associated with youth suicide: A clinical perspective. *Psychiatric Annals*, *18*, 652–656.

Phillips, D. (2007). Punking and bullying: Strategies in middle school, high school, and beyond. *Journal of Interpersonal Violence*, *22*, 158–178. doi: 10.1177/0886260506295341

Piaget, J. (1960). *The psychology of intelligence*. Patterson, NJ: Littlefield-Adams.

Pike, K. M., Devlin, M. J., & Loeb, K. (2004). Cognitive-behavioral therapy in the treatment of anorexia, bulimia nervosa, and binge-eating disorder. In J. K. Thompson (Ed.), *Handbook of eating disorders and obesity* (pp. 130–162). New York, NY: Wiley.

Pollack, W. (1998). *Real boys*. New York, NY: Random House.

Putman, F. W. (1993). Dissociation and disturbances of the self. In D. Cicchetti & S. Toth (Eds.), *Rochester Symposium Developmental Psychopathology: Disorders and dysfunctions of the self* (Vol. 5, pp. 251–266). Rochester, NY: University of Rochester Press.

Pyszcznski, T., & Greenberg, J. (1987). Self-regulatory perseveration and the depressive self-focusing style: A self-awareness theory of reactive depression. *Psychological Bulletin*, *102*, 1–17.

Pyzalski, J. (2012). From cyberbullying to electronic aggression: Typology of the phenomenon. *Emotional and Behavioral Difficulties*, *17*, 305–317. doi: 10.1080/13632752.2012.704319

Rand, K. L., & Cheavens, J. S. (2009). Hope theory. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 323–334). New York, NY: Oxford University Press.

Reijntjes, A., Thomaes, S., Kamphuis, J. H., Bushman, B. J., de Castro, B. O., & Telch, J. J. (2011). Explaining the paradoxical rejection-aggression link: The mediating effects of hostile intent attributions, anger, and decreases in state self-esteem on peer rejection-induced aggression in youth. *Personality and Social Psychology Bulletin*, *37*, 955–963. doi: 10.1177/0146167211410247

Rhee, U. (1993). Self-perceptions of competence and social support in Korean children. *Early Child Development and Care*, *85*, 56–66. doi: 10.1080/0300443930850107

Rhodewalt, F. (2006). Possessing and striving for high self-esteem. In M. H. Kernis (Ed.), *Self-esteem: Issues and answers* (pp. 281–287). New York, NY: Psychology.

Rinpoche, T. (1976). *The myth of freedom*. Berkeley, CA: Shambhalla Books.

Rochat, P. (2003). Five levels of self-awareness as they unfold early in life. *Consciousness and Cognition: An International Journal*, *12*, 717–731.

Robles-Pina, R., Defrance, E., & Cox, D. I. (2008). Self-concept, early childhood depression and school retention as predictors of adolescent depression in urban Hispanic adolescents. *School Psychology International*, *29*, 426–441.

Rogers, C. R. (1951). *Client-centered therapy*. Boston, MA: Houghton Mifflin.

Rosenberg, M. (1979). *Conceiving the self* . New York, NY: Basic Books.

Rosenberg, M. (1986). Self-concept from middle childhood through adolescence. In J. Suls & A. G. Greenwald (Eds.), *Psychological perspective on the self* (Vol. 3, pp. 107–135). Hillsdale, NJ: Erlbaum.

Ruggiero, G., Veronese, G., Castiglioni, M., & Sasssaroli. (2011). Grandiose fantasies and low self-esteem: An experimental study. *Psychological Studies*, *56*, 368–372. doi: 10.1007/s12646-011-0095-9

Rusk, N., & Rothbaum, F. (2010). From stress to learning: Attachment theory meets goal orientation theory. *Review of General Psychology*, *14*, 31–43. doi: 10.1037/a0018123

Rutter, M. (1989). Isle of Wight revisited: Twenty-five years of child psychiatric epidemiology. *Journal of the American Academy of Child Adolescent Psychiatry*, *28*, 633–653. doi: 10.1097/00004583-198909000-00001

Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development and well-being. *American Psychologist*, *55*, 68–78. doi: 10.1037/0003-066X.55.1.68

Ryan, R. M., & Deci, E. L. (2009). Promoting self-determined self-engagement. In K. R. Wentzel & A. Wigfield (Eds.), *Handbook of motivation at school* (pp. 171–196). New York, NY: Routledge.

Ryle, G. (1949). The Concept of Mind.

Saarni, C. (1999). *Developing emotional competence*. New York, NY: Guilford Press.

Saarni, C., Campos, J. J., Camras, L. A., & Witherington, D. (2006). Emotional development: Action, communication, and understanding.

In N. Eisenberg (Volume Ed.), W. Damon, & R. Lerner (Editors-in-Chief), *Handbook of child psychology* (6th ed., Vol. 3, *Social, emotional, and personality development,* pp. 226–229). New York, NY: Wiley.

Sadhwani, I. (2012). Effect of self-concept on adolescent depression. *Journal of Psychosocial Research*, *7*, 147–152.

Salovey, P., Mayer, J. D., Caruso, D., & Yoo, S. H. (2009). The positive psychology of emotional intelligence. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (pp. 237–248). New York, NY: Oxford University Press.

Sakura, S. (1983). Development of the Japanese version of Harter's Perceived Competence Scale for Children. *Japanese Journal of Educational Psychology*, *31*, 245–249.

Sanchez, D., & Kwang, T. (2007). When the relationship becomes her: Revisiting women's body concerns from a relationship contingency perspective. *Psychology of Women Quarterly*, *31*, 401–414. doi: 10.1111/j.1471-6402.2007.00389.x

Santos, J. C., Saraiva, C., & De Sousa, L. (2009). The role of expressed emotion, self-concept, coping, and depression in parasuicidal behavior: A follow-up study. *Archives of Suicide Research*, *13*, 358–367. doi: 10.1080/13811110903266590

Sarbin, T. R. (1962). A preface to a psychological analysis of the self. *Psychological Review*, *59*, 11–22.

Schwartz, S. J., Klimstra, T. A., Luyckx, K., Hale, W., & Meeus, W. H. J. (2012). Characterizing the self-system over time in adolescence: Internal structure and associations with internalizing symptoms. *Journal of Youth and Adolescence*, *41*, 1208–1225. doi: 10.1007/s10964-012-9751-1

Scheff, T. J. (2011). Social-emotional origins of violence: A theory of multiple killing. *Aggression and Violent Behavior*, *16*, 453–460.

Seligman, M. E. P., & Peterson, C. (1986). A learned helplessness perspective on childhood depression: Theory and research. In M. Rutter, C. E. Izard, & P. B. Read (Eds.), *Depression and clinical perspectives* (pp. 223–249). New York, NY: Guilford.

Seligman, M., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist*, *55*, 5–14. doi: 10.1037/0003-066X.55.1.5

Serpell, L., Neiderman, M., Roberts, V., & Lask, B. (2007). The Shape- and Weight-Based Self-Esteem Inventory in adolescent girls with eating disorders and adolescent controls. *Psychotherapy Research*, *17*, 321–327.

Selman, R. L. (1980). *The growth of interpersonal understanding*. New York, NY: Academic Press.

Selman, R. L. (2003). *The promotion of social awareness*. New York, NY: Russell Sage Foundation.

Shapiro, S. L. (2009). Meditation and positive psychology. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 601–610). New York, NY:

Showers, C., & Larson, B. (1999). Looking at body image: The organization of self-knowledge about physical appearance and its relation to disordered eating. *Journal of Personality*, *67*, 659–700. doi: 10.1111/1467-6494.00069

Siegler, R. S. (1991). *Children's thinking* (2nd ed.). Englewood Cliffs, NJ: Prentice-Hall.

Simmons, R. G., & Blyth, D. A. (1987). *Moving into adolescence: The impact of pubertalchange and school context*. New York, NY: Aldine de Gruyter.

Simonton, D. K. (2009). Creativity. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 261–269). New York, NY: Oxford University Press.

Smith, D. (2008). Globalization, degradation, and the dynamics of humiliation. *Current Sociology*, *56*, 371–379. doi: 10.1177/0011392107088230

Smith, A. J. (2010). Body image, eating disorders, and self-esteem problems during adolescence. In M. H. Guindon (Ed.), *Self-esteem across the lifespan: Issues and interventions* (pp. 125–141). New York, NY: Routledge/Taylor & Francis Group.

Smith, R. H., Webster, J. M., Parrott, W. G., & Eyre, H. L. (2002). The role of public exposure in moral and nonmoral shame and guilt. *Journal of Personality and Social Psychology*, *83*, 13–18.

Snyder, C. R. (1994). *The psychology of hope: You can get there from here*. New York, NY: Free Press.

Snyder, C. R., & Lopez, S. J. (Eds.). (2002). *Handbook of positive psychology*. New York, NY: Oxford University Press.

Spencer-Rodgers, J., Peng, K., Wang, L., & Hou, Y. (2004). Dialetical self-esteem and east–west differences in psychological well-being. *Personality and Social Psychology Bulletin*, *30*, 1416–1432. doi: 10.1177/0146167204264243

Spencer-Rodgers, J., Boucher, H., Mori, S., Wang, L., & Peng, K. (2009). The dialectical self-concept: Contradiction, change, and holism in East Asian cultures. *Personality and Social Psychology Bulletin*, *35*, 29–44. doi: 10.1177/0146167208325772

Spirato, A., Brown, L., Overholder, J., & Fritz, G. (1989). Attempted suicide in adolescence: A review and critique of the literature. *Clinical Psychology Review*, *9*, 335–363.

Statman, D. (2000). Humiliation, dignity and self-respect. *Philosophical Psychology*, *13*, 523–540. doi: 10.1080/09515080020007643

Stern, D. (1985). *The interpersonal world of the infant*. New York, NY: Basic Books.

Stigler, J. W., Smith, S., & Mao, L. (1985). The self-perception of competence by Chinese Children. *Child Development*, *56*, 1259–1270.

Sturman, E. D., Flett, G. L., Hewitt, P. L., & Rudolph, S. G. (2009). Dimensions of perfectionism and self-worth contingencies in depression. *Journal of Rational-Emotive and Cognitive-Behavior Therapy*, *27*, 213–231.

Suh, E. (2002). Culture, identity consistency, and subjective well-being. *Journal of Personality and Social Psychology*, *83*, 1378–1391.

Swann, Jr.,, W. B. (1996). *Self-Traps: The elusive question for high self-esteem*. New York, NY: W.H. Freeman.

Tafarodi, R., Lo, C., Yamaguchi, S., Less, W., & Katsura, H. (2004). The inner self in three countries. *Journal of Cross-Cultural Psychology*, *35*, 97–117. doi: 10.1177/0022022103260462

Tangney, J. P. (2009). Humility. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 483–490). New York, NY: Oxford University Press.

Tenenbaum, L, Varjas, K., Myers, J., & Parris, L. (2011). Coping strategies and perceived effectiveness in fourth through eighth grade victims of bullying. *School Psychology International*, *32*, 263–287. doi: 10.1177/0143034311402309

Thomaes, S., Bushman, B. J., Stegge, H., & Olthof, T., & Denissen, H. (2008). Trumping shame by blasts of noise: Narcissism, self-esteem, shame, and aggression in young adolescents. *Child Development*, *79*, 1792–1801. doi: 10.1111/j.1467-8624.2008.01226.x

Thompson, R. A. (2006). The development of the person: Social understanding, relationships, conscience, self. In N. Eisenberg (Volume Ed.), W. Damon, & R. Lerner (Editors-in-Chief), Handbook of child psychology (6th ed., Vol. 3, Social, emotional, and personality development, pp. 24–98). New York, NY: Wiley.

Thompson, S. (2009). The role of personal control in adaptive functioning. In S. J. Lopez & C. R. Snyder (Eds.), *The Oxford handbook of positive psychology* (2nd ed., pp. 279–294). New York, NY: Oxford University Press.

Torres, W. J., & Bergner, R. M. (2010). Humiliation: Its nature and consequences. *Journal of American Academy of Psychiatry and the Law*, *38*, 195–201.

Tracy, J. L., Cheng, J. T., Martens, J. P., & Robins, R. W. (2012). The affective core of narcissism: Inflated by pride, deflated by shame. In W. K. Campbell, & J. Miller (Eds.), *Handbook of narcissism and narcissistic personality disorder* (pp. 330–343). New York: NY, Wiley.

Tracy, J. L., & Robins, R. W. (2004). Putting the self into self-conscious emotions: A theoretical model. *Psychological Inquiry*, *15*, 103–125. doi: 10.1207/s15327965pli1502_01

Tracy, J. L., Robins, R. W., & Tangney, J. P. (Eds.). (2007). *The self-conscious emotions: Theory and research*. New York, NY: Guilford.

Trent, L. M., Russell, G., & Cooney, G. (1994). Assessment of self-concept in early adolescence. *Australian Journal of Psychology*, *46*, 21–28.

Trumbull, D. (2002). Humiliation: The trauma of disrespect. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry*, *36*, 643–660.

Trzesniewski, K. H., Kinal, M. P., & Donnellan, M. B. (2010). Self-enhancement and self-protection in a developmental context. Self-enhancement and self-protection in a developmental context. In M. D. Alicke & C. Sedikides (Eds.), *Handbook of self-enhancement and self-protection* (pp. 341–357). New York, NY: Guilford Press.

Tsui, A., S., Nifadkar, S. S., & Ou, A. Y. (2009). Nagging problems and modest solutions in cross-cultural research. In R. S. Wyer, C. Chiu, & Y. Hong (Eds.), *Understanding culture: Theory, research, and application*. New York, NY: Psychology Press.

Turner, H. A., Finkelhor, D., & Ormrod, R. (2011). The effects of adolescent victimization on self-concept and depressive symptoms. *Child Maltreatment*, *15*, 76–90.

Twenge, J. M., Konrath, S., Foster, J. D., Campbell, W. K., & Bushman, B. J. (2008). Egos inflating over time: A cross-temporal meta-analysis of the narcissistic personality inventory. *Journal of Personality*, *76*, 875–901. doi: 10.1111/j.1467-6494.2008.00507.x

Vartanian, L. (2009). When the body defines the self: Self-concept clarity, internalization, and body image. *Journal of Social and Clinical Psychology*, *28*, 94–126. doi: 10.1521/jscp.2009.28.1.94

Wallace, H. M. (2011). Narcissistic self-enhancement. In W. R. Campbell & J. D. Miller (Eds.), *The handbook of narcissism and narcissistic personality disorder: Theoretical approaches, empirical findings, and treatments* (pp. 309–318). Hoboken, NJ: Wiley.

Wallace, M., Barry, F., Zeigler-Hill, D., & Green, B. (2012). Locus of control as a contributing factor in the relation between self-perceptions and adolescent aggression. *Aggressive Behavior*, *38*, 213–221. doi: 10.1002/ab.21419

Walker, J., & Knauer, V. (2001). Humiliation, self-esteem, and violence. *Journal of Forensic Psychiatry and Psychology*, *22*, 724–741. doi: 10.1080/14789949.2011.617542

Wang, Q., & Leichtman, M. D. (2000). Same beginnings, different stories: A comparison of American and Chinese children's narratives. *Child Development*, *71*, 1329–1346. doi: 10.1111/1467-8624.00231

Wang, Y., & Ollendick, T. (2001). A cross-cultural and developmental analysis of self-esteem in Chinese and Western children. *Clinical Child and Family Psychology Review*, *4*, 253–271. doi: 10.1023/A:1017551215413

Watkins, D., Mortazavi, S., & Trofimova, I. (2000). Independent and interdependent conceptions of self: An investigation of age, gender, and culture differences in importance and satisfaction ratings. *Cross-Cultural Research: Journal of Comparative Social Science*, *34*, 113–134. doi: 10.1177/106939710003400202

Watson, D., & Clark, L. A. (1992). On traits and temperament: General and specific factors of emotional experience and their relation to the five-factor model. *Journal of Personality*, *60*, 441–476. doi: 10.1111/j.1467-6494.1992.tb00980.x

Watson, D., & Naragon, K. (2009). Positive affectivity: The disposition to experience positive emotional states. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 207–215). New York, NY: Oxford University Press.

Wayment, H. A., & Bauer, J. J. (2008). *Transcending self-interest: Psychological explorations of the quiet ego*. Washington, DC: American Psychological Association.

Wehmeyer, M. L., Little, T. D., & Sergeant, J. (2009). Self-determination. In S. J. Lopez & C. R. Snyder (Eds.), *Oxford handbook of positive psychology* (2nd ed., pp. 357–366). New York, NY: University of Oxford Press.

Welch, E., & Ghaderi, A. (2013). Eating disorders and self-esteem. In A. Andershed (Ed.), *Girls at risk: Swedish longitudinal research on adjustment* (pp. 35–56). New York, NY: Springer Science and Business Media.

Wertheim, E. H., Paxton, S. J., & Blaney, S. (2004). Risk factors for the development of body image disturbances. In J. K. Thompson (Ed.), *Handbook of eating disorders and obesity* (pp. 463–494). New York, NY: Wiley.

Westen, D. (1993). The impact of sexual abuse on self structure. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium developmental psychopathology: Disorders and dysfunctions of the self* (Vol. 5, pp. 223–250). Rochester, NY: University of Rochester Press.

Wilson, A., Smith, M., & Ross, H. (2003). The nature and effects of young children's lies. *Social Development*, *12*, 21–45. doi: 10.1111/1467-9507.00220

Williams, K., & Nida, S. (2009). Is ostracism worse than bully? In M. J. Harris (Ed.), *Bullying, rejection, and peer victimization: A social cognitive neuroscience perspective* (pp. 279–296). New York, NY: Springer Publishing Company.

Winstok, Z. (2009). From self-control capabilities and the need to control others to proactive and reactive aggression among adolescents. *Journal of Adolescence*, *32*, 455–466. doi: 10.1016/j.adolescence.2008.08.006

Wirtz, D., & Chiu, C. (2008). Perspectives on the self in the east and the west: Searching for the quiet ego. In H. A. Wayment & J. J. Bauer (Eds.), *Transcending self-interest: Psychological explorations of the quiet ego* (pp. 149–158). Washington, DC: American Psychological Association.

Wolf, D. P. (1990). Being of several minds: Voices and versions of the self in early childhood. In D. Cicchetti & M. Beegley (Eds.), *The self in transition: Infancy to childhood* (pp. 183–212). Chicago, IL: University of Chicago Press.

Woodruff-Burton, H. (2012). Lived consumer bodies: Narcissism, bodily discourse, and women's pursuit of the body beautiful. In C. Otnes, & L. T. Zayer (Eds.), *G*ender, culture, and consumer behavior (pp. 195–221). New York, NY: Routledge/Taylor & Francis Group.

Woodman, T., & Hemmings, S. (2008). Body image self-discrepancies and affect: Exploring the feared body self. *Self and Identity*, *7*, pp. 413–429. doi: 10.1080/15298860701800225

Wylie, R. C. (1979). *The self-concept: Theory and research on selected topics* (Vol. 2). Lincoln, NE: University of Nebraska Press.

Wylie, R. C. (1989). *Measures of self-concept*. Lincoln, NE: University of Nebraska Press.

Yang, K. (2003). Beyond Maslow's culture-bound linear theory: A preliminary statement of the double-Y model of basic human needs. In R. A. Diestbier (Series Ed.), V. Murphy-Berman, & J. J. Berman (Eds.), *Cross-cultural differences in perspectives on the self, Nebraska symposium* (Vol. 49, pp. 175–256). Lincoln NE: University of Nebraska Press.

Zajonc, R. B. (1984). On the primacy of affect. *American Psychologist*, *39*, 117–123. doi: 10.1037/0003-066X.39.2.117

Zeigler-Hill, V., & Jordon, C. H. (2011). Behind the mask: Narcissism and implicit self-esteem. In W. K. Campbell & J. D. Miller (Eds.), *The handbook of narcissism and narcissistic personality disorder: Theoretical approaches, empirical findings, and treatments* (pp. 101–115). Hoboken, NJ: John Wiley & Sons Inc.

CHAPTER 12

Peer Relations and Developmental Psychopathology

MITCHELL J. PRINSTEIN and MATTEO GILETTA

INTRODUCTION 527
A Developmental Psychopathology Framework 528
Organization of This Chapter 530
INFANCY AND EARLY CHILDHOOD 530
Social Withdrawal 530
SCHOOL-AGE CHILDREN 533
Peer Status 533
Peer Victimization 542
Friendship 551
ADOLESCENCE 556
Peer Influence 557
CONCLUSION AND FUTURE DIRECTIONS 566
REFERENCES 568

INTRODUCTION

Few youth likely would report that their experiences with peers are irrelevant to their emotional and behavioral development. A majority of children, in fact, spend considerable time with peers; many infants and young children spend more time engaged in peer interactions than with adults, including their parents (Ellis, Rogoff, & Cromer, 1981). Many school-age children indicate that their interactions with peers are a significant source of distress or enjoyment. Children readily can identify specific peers with whom they have friendships, and without whom they feel particularly lonely. By adolescence, youth are more likely to engage in emotional disclosure with peers than with adults, and most utilize feedback from peers as a major determinant for their own developing values, behaviors, and sense of self (Furman & Buhrmester, 1992). Theorists too have highlighted the unique role of *horizontal relationships* with peers in the formation of basic developmental competencies (e.g., Piaget, 1932; Vygotsky, 1981), and diagnosticians have noted difficulties in sustaining adaptive interactions with peers as criteria for major psychiatric disorder (e.g., American Psychological Association [APA], 2013). There is little doubt that peer experiences represent highly salient and developmentally meaningful constructs in understanding youths' maladaptive emotional and behavioral trajectories.

However, peer experiences have been a focus of behavioral scientists' empirical work for a relatively brief period of time. Following decades of a psychoanalytically oriented conceptualization of psychopathology that strongly emphasized early parent–child relations and unresolved libidinal impulses as well as a mid-twentieth-century postwar focus on psychological symptoms among adults (particularly veterans), the study of peer experiences was fairly dormant. Moreover, an understanding of psychopathology more broadly among youth remained elusive.

Studies of young adults experiencing a range of maladaptive outcomes (e.g., significant distress, psychopathology, criminal behavior) brought renewed attention to the study of peers. Roff (1961) revealed that dishonorably discharged military servicemen were more likely than others to have early persistent and severe peer difficulties noted within their early childhood school records. Cowen, Pederson, Babigian, Isso, & Trost (1973) similarly noted that adults receiving psychiatric treatment were more likely than others to have had negative reputations among their peers over a decade earlier; in fact, peer status and reputations discriminated treatment-seeking adults from others more effectively than did a variety of other known predictors of psychopathology (e.g., intellectual ability, physical health, self-esteem, and teacher-reported psychological adjustment). Results such as these unwittingly set an agenda for peer research for decades to come. For the remainder of the twentieth century, dozens of empirical studies were conducted to examine the putative effects of early peer status and reputations on later psychological adjustment. Studies used a host of measures to operationalize peer status and reputations, and examined a wide range of outcomes representing broad and narrow indices of psychopathology. Like some of these early studies, the field also became skewed toward the study of males. In

1987, Parker and Asher published a seminal qualitative review indicating that children who possessed negative status and reputations among their peers were at greatest risk for school dropout, criminal behavior, and other broad indices of psychopathology. This review, and the dozens of studies that were used to support its conclusions, instantiated the peer context as a critical domain for examination among developmental psychopathologists. Few empiricists would deny that peer experiences significantly contribute to youths' development, and may have an etiological role in the development of psychopathology.

It has been almost 30 years since the publication of Parker and Asher's (1987) review. Research on peer experiences currently is abundant and has yielded at least two critical contributions. First, based on a large body of descriptive work, a wide range of peer experiences now has been articulated and operationalized. Although initial work on peer experiences focused almost exclusively on peer status and reputations, and on the construct of peer acceptance–rejection in particular, contemporary work now recognizes that youths' peer relationships, social behaviors, as well as youths' preferences and reputations within the peer milieu each represent related, but distinct peer constructs, varying in form or function across development. Importantly, there remains much work to do in this regard. Although many discrete constructs have been identified, it is clear that youth have developed sophisticated ways of interacting with one another, and regrettably, even more peer behaviors that can cause significant distress among youth. With the advent of more sophisticated technologically mediated forms of peer interaction and a growing awareness of multicultural differences in behaviors among peers, researchers remain challenged to keep abreast of the rapidly changing peer experiences that are so salient and meaningful to youth themselves.

A second substantial contribution offered by recent peer relations research has been the elucidation of cross-sectional and longitudinal correlations among each of many peer relations constructs and indices of maladjustment or psychopathology. Consistent with the tradition set by the groundbreaking work briefly reviewed already, most of this work has focused on peer predictors of psychopathology, rather than vice versa. Most work has focused on externalizing symptom outcomes and health risk behaviors; relatively little research has examined associations among peer relations constructs and internalizing psychopathology. Research has focused on the psychological risks of normative youth who have experienced peer difficulties more than on the peer experiences of clinically referred youth who already have been diagnosed with disorders. Extant data also have offered surprisingly little predictive specificity, and the field of peer relations research often has been critiqued for lacking theoretical frameworks that may organize and guide studies on the putative role of peer experiences on youths' maladjustment. Nevertheless, prior work has offered an excellent foundation for understanding whether peer experiences may be relevant predictors of psychopathology. Much of this work has used robust, follow-forward (sometimes long-term) longitudinal designs to demonstrate the distal effects of peer dysfunction on later maladjustment, often above and beyond competing predictors.

A Developmental Psychopathology Framework

Although the study of peer relations constructs, and associations between peer relations and psychopathology have been remarkably active areas of inquiry, much work is needed to help understand the putative role of peers in youths' development. It is clear that peers provide a context in which development occurs. However, it may be less clear whether peer experiences merely reflect existing developmental incompetence, or whether these experiences may contribute to new deficits. Alternatively, peer experiences may compound existing vulnerabilities, conjointly altering developmental trajectories. Much of this dialectic regarding risk factor models has been discussed for years. In earlier editions of this volume, for instance, researchers articulated several possibilities (Parker, Rubin, Erath, Wojslawowicz, & Buskirk, 2006). Suggesting that difficulties among peers may be a manifestation of areas of underlying developmental incompetence, or symptoms of psychopathology, an *incidental model* was proposed to explain apparent but not etiological links between peer difficulties and psychopathology. Incidental models are based an assumption of developmental continuities in psychological difficulties. For instance, aggressive behavior in early childhood manifests as disruptive behavior disorders symptoms later in development. Associations between early aggressive behavior and rejection by peers may reveal the social consequences of childhood aggression, but this model suggests no incremental contribution of peer rejection on the prediction of later disruptive behavior symptoms after considering heterotypic continuity in externalizing symptom presentations. In contrast, a "causal model" suggests an association between peer experiences and later psychological symptoms, even after accounting for prior levels of maladjustment. The causal model suggests a direct and independent association between peer difficulties and later psychopathology.

Developmental psychopathology theories and recent research increasingly suggest more sophisticated and necessarily complex models to understand associations between environmental experiences, including peer experiences, and individual development. Developmental psychopathology approaches emphasize multiple levels in which developmental occurs, and the inextricable co-evolution of developmental processes at biological, psychological, social, and cultural levels (Cicchetti & Lynch, 1993). Two tenets of developmental psychopathology theory are especially relevant.

First, transactional models of development suggest that individuals' transactions within the environment are mutually reciprocal and evolve over time (Sameroff, 2000). Linear risk factor models have been most amenable to empirical examination but do not reflect the process by which theorists believe psychopathology develops. Rare is the instance in which a single environmental experience acts in isolation as a predictor of an individual's maladaptive behavior. Rather, an ongoing exchange between an individual youth's behavior, the environmental response to this behavior, and the youth's subsequent reaction to this response (and so on) is most likely to promote the development of psychopathology through modification of developmental competence trajectories.

Theories of contextualism stress the reciprocal influences between an individual's continual development and the evolving social context in which an individual is embedded (Cicchetti & Aber, 1998). Individuals' own characteristics and vulnerabilities affect the social environment just as social experiences can alter the development of individuals' characteristics (Sroufe, 1997). Such transactions are not limited to psychological factors; social experiences can exert effects on myriad biological processes, even including genetic expression (Slavich & Cole, 2013).

Time is an especially important factor within transactional models but further can confound efforts at empirical measurement and testing. Cyclical iterations (i.e., person–environment transactions) may occur within microseconds or over the course of years. Because it is unknown when the observable effects of a specific stimulus may become evident, and the duration of this lag in itself likely is due to factors that vary between people, linear risk factor models are especially unlikely to fit many youths' experience. Rather, transactional models suggest that the product of these cyclical iterations is a change in youths' developmental trajectory, or a new array of potential outcomes and experiences. Altered developmental trajectories bring forth new potential behaviors, and different environmental responses, thus further eliciting complexities in the association between environmental stimuli and individuals' responses. Aggressive behavior may lead to negative reputations among peers, and the resulting ostracism or exclusion may deprive youth of normative opportunities to develop prosocial play behavior, for example. Children with deficits in prosocial behavior may be especially likely to form deviant peer groups that promote the development of antisocial behavior. Externalizing psychopathology is caused neither by peer rejection nor by prior aggressive behavior but rather the altered developmental trajectory in social behavioral development produced by conjoint effects between aggression and peer rejection.

Moreover, transactional models assume developmental variation over time. Not only do youths' repertoire of behaviors become increasingly differentiated and nuanced over time, but also changes in the social environment are predictable. Adult supervision decreases as youth mature. Behavioral expectations also are modified over the life span, as are concomitant norms for (in)appropriate social behavior and the form of social consequences. Each of these factors further is influenced by youths' history and their prior behavior.

Second, a dynamic systems perspective also is necessary for understanding the role of peer relations on the development of psychopathology (e.g., Magnusson & Stattin, 2006). Systems of development include various developmental competencies (e.g., emotional, cognitive, social) and contexts (e.g., biological, peer, family, school, neighborhood)—literally including contexts as innate as one's cellular development and as broad as community or cultural influence. Developmental systems are mutually informative and influential as youth mature. Experiences with peers are embedded within broader systems of development and have a potential impact on numerous competencies outside the peer or even social domain more broadly. Increasingly, research has embraced multilevel investigations conceptualizing social environmental experiences as relevant for understanding cognitive and biological developmental processes that promote psychopathology. For instance, recent work suggests that chronic social rejection and concomitant negative cognitive appraisals may have long-lasting effects on the central and peripheral nervous systems. These effects likely are the consequence of epigenetic modifications at the molecular level that alter gene expression and subsequently may compromise immune functioning, alter brain activity, and perhaps promote psychopathology symptoms (Slavich & Cole, 2013). A related line of research suggests that negative social experiences may trigger neural responses similar to the effects of physically injurious experiences

on the affective experience of pain. In other words, social pain triggers brain regions in a manner similar to physical pain and may have implications for affective regulation and the development of psychopathological symptoms (Eisenberger, 2012; MacDonald, & Leary, 2005).

The study of peer processes as relevant contributors to other developmental systems, and to multilevel processes has gained attention only recently. Yet this is an especially important direction for ongoing study in the relevance of peer experiences in the development of psychopathology.

Organization of This Chapter

This chapter was written to accomplish several objectives. First, the chapter offers an overview of the constructs frequently examined within peer relations research. This overview is not exhaustive, as it would be impossible to provide sufficient coverage of the many topics that peer relations researchers have examined. Key constructs that have received the greatest attention within the study of psychopathology have been highlighted. For each, operationalization of theoretical constructs is discussed. A second goal of this chapter is to review patterns of consistent findings linking peer relations constructs with indices of psychopathology. Third, theories of psychopathology with implications for understanding the role of peers are noted and used to interpret results within the peer relations field. Due to the unfortunate segregation of research into increasingly artificial subdisciplines of psychology (e.g., developmental, clinical, cognitive, neuroscience), prior research in peer relations rarely has integrated conceptual frameworks from the broader discipline. This chapter attempts to offer some directions for further collaboration and cross-pollination within social science fields. Last, a main emphasis of this chapter is to note efforts to understand peer processes from a developmental psychopathology perspective, incorporating transactional and dynamic systems frameworks and emerging research examining broader multilevel models.

The chapter is organized developmentally, beginning with a review of research examining infancy and early childhood, followed by work on school age-children and adolescence. Naturally, the organization of this chapter into half-decade segments is an artificial categorization of both the nature of developmental processes and the approach used within some peer relations research. Nevertheless, this offers a useful framework for organizing a large body of research. The depth of coverage of work across developmental periods is broadly commensurate with the focus on each phase of youth in the peer relations literature.

INFANCY AND EARLY CHILDHOOD

Infants and youth in early childhood have meaningful peer interactions. Newborns attend not only to their parents but also to peers through mutual eye contact, touching, and vocalizing (Vandell, Wilson, & Buchanan, 1980). Over the first two years of life, toddlers begin to engage in some play behaviors with peers, developing rudimentary reciprocal turn-taking skills, sharing, and directed vocalizations. By 3 years of age, most children can express empathy and engage in some conflict regulation skills (Mueller & Brenner, 1977). Toddlers exhibit surprisingly advanced social knowledge; they can identify a peer's distress and provide comfort through emotional support (Caplan & Hay, 1989). Within the third year, children also can express fairly stable preferences for one another, reporting which children they like the most or least (Denham, McKinley, Couchoud, & Holt, 1990). Ages 3 to 4 years are accompanied by increased sophistication in social behaviors. Play interactions include increasingly complex scenarios and games, which include an increase in the communication of rules, negotiation of leadership skills, and peers' mutual use of social consequences (Fein, 1981). Although children express preferences for one another earlier in life, reciprocated friendships become apparent by the age of 3 to 4 years. These friendships involve a clear preference for mutual companionship and increased levels of positive behavior. Children express different types of affect among friends compared with nonfriend peers (Gershman & Hayes, 1983).

Social Withdrawal

Not all children adapt to the early peer environment in similar ways, however. Although most children actively seek interactions with other peers, some children do not. Social withdrawal is defined as a consistent display of solitary behavior when encountering unfamiliar or familiar peers (Rubin, Bowker, & Kennedy, 2009). Social withdrawal is differentiated from *active isolation* (i.e., rejection and exclusion by others), instead referring to children's own choice to remain alone. Building on approach and avoidance theories, at least three subgroups of internally motivated social withdrawal have been articulated (Coplan & Armer, 2007). First, anxious-withdrawn children include those who wish to interact with other peers (i.e., high social-approach motivation) but withdraw to fearfulness and anxiety that accompany these social situations (i.e., high social-avoidance motivation). A second subgroup includes unsociable (or socially disinterested) children who

lack a strong desire to interact with other peers (i.e., low social-approach motivation) but on the contrary do not experience emotional dysregulation (e.g., anxiety) in social situations (i.e., low social-avoidance motivation). Finally, socially avoidant children show a low social approach and a high social avoidance motivation. Although at least from a theoretical point of view avoidant children can be identified, this subgroup of children has received remarkably little empirical attention, likely due to the challenges of operationalizing this construct.

Despite the theoretical relevance of differentiating between social withdrawal subtypes and the growing body of research focusing on unsociable children, to date most extant work either has operationalized social withdrawal as a unidimensional construct (without distinguishing internal motivations of social isolation) or exclusively has examined anxious and fearful withdrawn children. Consequently, comparisons within the literature among studies of withdrawn children can be difficult. Moreover, related research on behavioral inhibition, shyness, and anxious solitude also overlap with studies of social withdrawal (see Rubin & Coplan, 2010, for a review). Next, social withdrawal is discussed broadly, and particular attention is dedicated toward research that has differentiated internal motivations underlying social isolation.

Research has suggested that social withdrawal may have its origin in behavioral inhibition, a temperamental trait that can be identified already within the first years of life (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001). Young toddlers who are behaviorally inhibited display negative emotional and motor reactivity in front of unfamiliar social and nonsocial stimuli, including people and objects (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984). Behavioral inhibition has been proposed to have a biological basis and is associated with a number of unique physiological responses (see Fox, Henderson, Marshall, Nichols, & Ghera, 2005). When confronted with novel stimuli (e.g., visual images), inhibited children demonstrate right frontal electroencephalogram (EEG) asymmetry, increased heart rate variability, and heightened cortisol release compared with their uninhibited peers. Such findings suggest that behavioral inhibition is characterized by amygdala hyperactivity—a hypothesis that has received further direct support in recent functional magnetic resonance imaging (fMRI) studies (e.g., Schwartz et al., 2012).

Studies examining the homotypic and heterotypic continuity of behavioral inhibition throughout childhood (as well as adolescence) have revealed only moderate continuity. In selected samples of inhibited toddlers, approximately 70% show signs of behavioral inhibition later in childhood; within unselected samples only half of toddlers show behavioral continuity in inhibited behavior (for a review, see Degnan & Fox, 2007). Thus, although behavioral inhibition may be an antecedent to social withdrawal, not all inhibited toddlers will display withdrawal behaviors later in childhood when in the presence of other peers, and, accordingly, not all socially withdrawn children show behavioral inhibition earlier in life. In this regard, ample work has been dedicated to identify potential (intrinsic and extrinsic) moderating factors that alter the development of behavioral inhibition and influence the pathway from behavioral inhibition to social withdrawal and, eventually, to psychopathology (Degnan & Fox, 2007). For instance, Rubin, Burgess, and Hastings (2002) demonstrated that inhibited 2-year-old toddlers who had psychologically overcontrolling mothers (i.e., intrusive and derisive mothers) were more likely to exhibit social withdrawal (e.g., reticent behavior) in the presence of unfamiliar peers by age 4 years compared with inhibited toddlers with nonovercontrolling mothers. Notably, some researchers have emphasized that children who display withdrawal behaviors in the presence of unfamiliar (or familiar) peers may be specifically at risk for developing psychopathology compared with children who are inhibited exclusively among nonsocial situations or among unfamiliar adults. Thus, social withdrawal is a distinct and perhaps more risky phenotype compared with behavioral inhibition. Compared with behaviorally inhibited youth (generally), socially withdrawn children in particular may have altered peer experiences and ultimately greater risk for developing psychological disorders (Rubin, Bowker, & Kennedy, 2009).

To date, there is ample evidence that socially withdrawn children have unique peer experiences that likely deprive youth of opportunities to develop typical social competencies (Rubin, Coplan, & Bowker, 2009). For instance, in dyadic free play interactions, socially withdrawn children rarely initiate interactions with other peers and more frequently engage in solitary behaviors, such as reticent behavior (i.e., prolonged observation of other children without accompanying play and being unoccupied; Coplan, Rubin, Fox, Calkins, & Stewart, 1994), and solitary-passive play (i.e., exploration of objects or constructive activity while playing alone). Compared with nonwithdrawn children, socially withdrawn children also are less socially assertive and have limited social problem-solving strategies (Stewart & Rubin, 1995). Thus, not only do withdrawn children interact less frequently with their peers, but also when they do they appear to be less socially skilled. Perhaps not surprisingly, therefore, social withdrawal is

associated with short- and long-term difficulties in peer relationships (Rubin, Bowker, & Gazelle, 2010). Young children engaging in reticent behavior receive fewer interactional initiations from peers (Chen, DeSouza, Chen, & Wang, 2006) and are more likely than others to be excluded, rejected, and victimized by other children, both concurrently and later in development (Gazelle & Ladd, 2003).

Socially withdrawn children are equally as likely as nonwithdrawn children to have at least one mutual stable friend (e.g., Rubin, Wojslawowicz, Rose-Krasnor, Booth-LaForce, & Burgess, 2006); however, their friendships are likely to be less adaptive in at least two ways. First, withdrawn children tend to have friends who are similarly as withdrawn as they are (Haselager, Hartup, van Lieshout, & Riksen-Walraven, 1998). Second, the friendships of withdrawn children are characterized by limited positive interactions and lower relationship quality (Rubin et al., 2006).

Notably, differences in play behavior and social skill are evident across subtypes of withdrawn children. Compared with anxious-solitary children, unsociable youth can demonstrate competent social behaviors when in a social interaction (Harrist, Zaia, Bates, Dodge, & Pettit, 1997). Anxious-solitary children demonstrate consistent social deficits, however. Both unsociable and anxious-solitary children are at risk for being rejected by peers, at least in early childhood (Coplan & Weeks, 2010). Later in development, unsociable children do not appear to be at as great a risk for peer difficulties as are anxious-solitary youth (Coplan et al., 2012). The few studies examining socially avoidant youth suggest that, similarly to anxious-solitary youth, they are likely to experience difficulties with other peers (e.g., Bowker & Raja, 2011).

Social Withdrawal and Internalizing Distress

In light of the temperamental characteristics of withdrawn children and the interpersonal difficulties to which they likely are exposed, the link between social withdrawal and symptoms of psychopathology is not surprising. Social withdrawal is a concurrent predictor of social anxiety, depressive symptoms, and loneliness (e.g., Rubin, Chen, & Hymel, 1993; Strauss, Forehand, Smith, & Frame, 1986; Vanhalst, Luyckx, & Goossens, 2014). Gazelle, Workman, and Allan (2010) revealed that compared with their nonanxious-solitary agemates, anxious-solitary children are more likely to be diagnosed with anxiety or mood disorders.

Social withdrawal during early childhood also has long-term effects on internalizing symptoms. Early social withdrawal prospectively predicts depressive symptoms and loneliness in middle childhood (Hymel, Rubin, Rowden, & LeMare, 1990). Similarly, shy-inhibited temperament in childhood prospectively predicts anxiety in adolescence (Prior, Smart, Sanson, & Oberklaid, 2000). It has been suggested that the effects of social withdrawal on internalizing problems may increase with age (Rubin et al., 2009); that is, early social withdrawal may become a stronger risk factor for internalizing symptoms during middle and late childhood than in early childhood. Social withdrawal also has been associated with negative self-perceptions and depressogenic attributional styles (Burgess & Younger, 2006). When subtypes of social withdrawal have been examined, results suggest that anxious-solitary (or shy-withdrawn) and avoidant children are both at risk for internalizing outcomes, while unsociable children may not be at risk for developing psychopathology (Coplan et al., 2012).

Thus, substantial evidence suggests that socially withdrawn children, and anxious-solitary youth in particular, are at increased risk for internalizing psychopathology. Of course, a parsimonious interpretation of research findings might therefore suggest continuity in anxious/inhibited behavior across development, in which temperamental traits are expressed as social difficulties and ultimately internalizing disorder later in development. However, social withdrawal appears to affect the continuity of anxious behavior in meaningful ways.

Recent research has examined hypotheses utilizing such a developmental psychopathology perspective. First, studies suggest that the effect of social withdrawal on later symptoms of psychopathology occur via peer relationships (e.g., Boivin, Hymel, & Bukowski, 1995; Katz, Conway, Hammen, Brennan, & Najman 2011). For instance, Katz and colleagues (2011) revealed that indicators of social withdrawal measured at age 5 years did not have a direct effect on depression diagnosis at age 20; instead, an indirect pathway was observed, in which social withdrawal at age 5 years predicted social impairment at age 15, which in turn increased the probability of a major depressive disorder diagnosis at age 20.

Second, empirical evidence indicate that prospective bidirectional effects may occur between social withdrawal and peer relationships throughout childhood and adolescence. Although most studies have demonstrated that social withdrawal may increase children's risk for experiencing peer difficulties, other work also has documented that in childhood and adolescence peer relationships prospectively may affect social withdrawal (e.g., Booth-LaForce et al., 2012; Gazelle & Ladd, 2003; Oh et al., 2008). For example, peer factors in middle childhood distinguish among children with different developmental trajectories of social withdrawal from middle childhood (Grade 5) to

early adolescence (Grade 8) (Oh et al., 2008). Interestingly, higher peer exclusion in middle childhood enhanced children's probability of membership in an increasing rather than low stable trajectory of social withdrawal. Moreover, among children who followed an increasing trajectory, those who did not have a mutual best friend, and a stable friendship at baseline, revealed steeper increases in social withdrawal, compared with children with a best friend and a stable friendship. These findings indicate that peer relationships, both at the group and dyadic levels, may alter the development of children social withdrawal.

Finally, a number of studies have demonstrated that withdrawn children who also experience peer difficulties (e.g., peer rejection) are particularly at risk for internalizing problems. Bukowski, Laursen, and Hoza (2010) examined a specific type of cascade model (i.e., snowballing cascade) to see the combined effect of withdrawal and peer relationships (i.e., peer exclusion and friendship) on trajectories of preadolescents' depressive symptoms over a three-year period. Results indicated that friendship moderated the conjoint effect of social withdrawal and peer exclusion on the development of depressive symptoms. Rejected-withdrawn children who also did not have a mutual best friend had steeper increasing trajectories of depressive symptoms; however, rejected-withdrawn children who were involved in a mutual best friendship had declining trajectories of depression.

In sum, evidence is accumulating to support a developmental cascade multilevel model involving mutually influential transactions between young children's temperamental traits and evoked environmental experiences that conjointly influence internalizing symptom trajectories. This model suggests that behavioral inhibition in the first years of life among children may evoke overprotective caregiver responses that in turn may further exacerbate children's inhibition and withdrawal behaviors during interactions with peers (Rubin, Coplan, & Bowker, 2009). Socially withdrawn behaviors, and resulting peer difficulties, may conjointly influence the development of later internalizing symptoms.

SCHOOL-AGE CHILDREN

By the time children reach 5 to 6 years of age and enter formal schooling in most communities, most have developed *peer competence*, including the capacity to engage in mutual, cooperative peer interactions that are characterized by positive affect. Yet considerable variability in children's peer competence is evident. Difficulties emerge in children's ability to develop reciprocated interest among peers, both within the overall peer group and within dyadic interactions. For instance, some children develop negative reputations within the peer group, leading to rejection, unpopularity, or victimization from peers. Some children also experience significant difficulties forming stable friendships or relationships characterized by positive friendship qualities. Each of these specific peer experiences (i.e., peer status, peer victimization, and friendships) has been examined as distinct constructs within the peer relations literature.

Peer Status

One of the most frequently used indices to capture children's overall success within the peer group is their level of acceptance or rejection among peers. The use of peer nominations to capture children's peer acceptance or rejection (i.e., peer likeability) has been common for many years, and this construct probably has been examined more frequently than almost any other aspect of children's peer relations. Moreover, although research certainly has examined peer acceptance/rejection prior to the school-age period, and well into adolescence, most prior work has measured this construct among youth during the school-age years. Recent developments in the conceptualization of peer status, particularly its developmental heterogeneity, have led to semantic obfuscation, however. In prior literature, this construct has been referred to as peer status, sociometric status, social preference, likeability, or even popularity. We elect to refer to this construct as peer acceptance/rejection to remain most consistent with the manner in which this construct is operationalized.

Due to known biases in self-, parent-, and teacher-reported indices of youths' overall peer experiences and the high ecological validity associated with the use of an aggregated peer report measure to understand children's experiences among peers, the use of peer ratings or nominations is considered a gold standard for assessing peer acceptance/rejection. Quite simply, peer nomination measures customarily involve research participants' unlimited selection of peers from a roster of classmates/grademates that they like the most and like the least, yielding two orthogonal scores. A standardized difference between each child's standardized tally of positive and negative nominations, respectively, is computed as a continuous measure of peer acceptance/rejection (referred to as social preference), while a standardized sum between these standardized tallies can be used as an index of visibility within the peer network (i.e., social impact). A commonly used algorithm can be employed to categorize scores of peer acceptance, rejection, social preference, and social impact into five

sociometric status groups: popular, rejected, neglected, controversial, and average (see Coie & Dodge, 1983). Notably, the classification of some youth as popular based on this assessment of peer acceptance/rejection may be a misnomer, as subsequent research recently has identified a unique construct that more closely resembles what most youth consider to represent popularity.

Children's peer acceptance/rejection appears to be quite stable across time and contexts. Longitudinal work suggests that children's peer status categories remain consistent over multiple developmental transitions, with moderate stability over several consecutive assessments (see Cillessen, Bukowski, & Haselager, 2000). Innovative experimental work also demonstrates that children's level of peer acceptance/rejection among familiar peers is very quickly replicated within groups of unfamiliar peers (Coie & Kupersmidt, 1983; Dodge, 1983). Similarly, a meta-analysis including more than 18,000 participants has shown high stability for the continuous measures of peer acceptance, rejection, and social preference across childhood as well as adolescence (Jiang & Cillessen, 2005). Such intriguing results suggest that peer status may serve as a marker for other child traits or difficulties that persist across lengthy developmental periods or across multiple contexts.

Indeed, many determinants of peer status have been identified in studies of children's behavioral and physical characteristics, cognitive and emotional competencies, and family factors. Some of these predictors are relevant to consider as possible precursors of psychopathology in themselves. Perhaps not surprisingly, the most robust predictors of children's peer acceptance/rejection appear to be youths' own social behaviors. Children who are aggressive are highly likely to be rejected by their peers. Rejected children also exhibit greater internalizing difficulties than others (e.g., anxiety, depression, withdrawal) and demonstrate poorer social skills (Newcomb, Bukowski, & Pattee, 1993). In contrast, popular youth often are rated as social leaders with high ratings of prosocial skill (e.g., helping others, kind) and athleticism. Neglected youth exhibit few differences from their peers with average sociometric status other than less frequent social interaction and somewhat elevated levels of social withdrawal. Interestingly, neglected peer status is the least stable of the nonaverage status groups over time (Cillessen et al., 2000). Controversial youth possess a blend of traits that are common among both rejected youth (i.e., aggressive behavior, breaking rules) and popular youth (i.e., positive social interactions, extroversion, and good humor). At younger ages, higher levels of intelligence are associated with higher levels of peer acceptance; by preadolescence, higher levels of intelligence, particularly when combined with children's effortful presentation of academically oriented goals, are associated with lower levels of peer acceptance (Juvonen & Murdock, 1995). Correlates of high intelligence, including high problem-solving skills, high academic achievement, and teacher preferences, also are associated with popular peer status among young school-age children. Numerous studies have demonstrated that markers of facial and body attractiveness also predict high levels of peer acceptance, while child obesity has been associated with peer rejection (Bell & Morgan, 2000; Dodge, 1983).

In addition to children's physical and behavioral characteristics, family determinants of children's peer status also have been identified. Some of these family determinants are markers for the social environment in which children are raised (Parke & Ladd, 1992). For example, maternal psychopathology, particularly depressive symptoms, and mothers' own social skill deficits are associated with their children's peer rejection. Parents' engagement in authoritarian or lax parenting, low emotional expressiveness, and parent–child play behavior characterized by power assertion all are associated with children's peer rejection. Children who demonstrate insecure attachment relationships also are more likely than others to experience rejection by peers (Parke & Ladd, 1992). Parents' management of their children's social experiences also is associated longitudinally with their child's peer acceptance/rejection; however, associations are dependent upon the developmental appropriateness of these parenting behaviors. Parents who actively initiate social interaction for their young children, and closely monitor their children's peer interactions have children more likely than others to experience peer acceptance. However, these same parenting behaviors are associated with peer rejection when measured among older children. Like other aspects of development, results suggest that parents' scaffolding of social interaction experiences appear to produce maximally adaptive outcomes (Parke & Ladd, 1992).

Peer Acceptance/Rejection and Externalizing Symptoms

It should be noted that many of the child and family determinants of school-age children's peer acceptance/rejection also are known risk factors for psychological symptoms among youth. For instance, children's aggression with peers and poor problem-solving skills are known risk factors for later externalizing symptoms. Maternal depression, insecure attachment relationships, and maladaptive parenting also have been associated longitudinally with children's own internalizing and externalizing symptoms.

Thus, a critical question among researchers examining peer acceptance/rejection as a putative risk factor for psychopathology has been whether peer experiences offer incremental predictive value in the prediction of later psychopathology. Studies using prospective longitudinal designs, predicting psychological symptoms while controlling for initial levels of psychopathology, largely have supported this hypothesis.

Over 30 prospective longitudinal studies have demonstrated that peer rejection is associated with later aggressive, delinquent, oppositional, and illegal behavior (e.g., Coie, Terry, Lenox, Lochman, & Hyman, 1995). Findings suggest long-term effects (i.e., peer rejection predicts outcomes up to 10 years later; Laird, Pettit, Dodge, & Bates, 2005) and robust effects across multiple informants of peer rejection (i.e., peer and teacher reported; Coie et al., 1995). Moreover, results indicate similar effects at different developmental stages. Peer rejection predicts externalizing symptoms in early childhood (e.g., kindergarten and first grade; Dodge et al., 2003), middle childhood (e.g., Coie et al., 1995), and adolescence (e.g., eighth to tenth grade; Prinstein & Cillessen, 2003). Results are consistent among studies predicting elevations on symptom checklists (Kupersmidt & Patterson, 1991), the presence of externalizing diagnoses (Miller-Johnson et al., 2002), and criminal offenses according to public records (Nelson & Dishion, 2004). It appears that these findings are similar for predominantly Caucasian samples (Lewin, Davis, & Hops, 1999), exclusively African American samples (Rabiner, Coie, Miller-Johnson, Boykin, & Lochman, 2005), and ethnically heterogeneous samples (Prinstein & La Greca, 2004).

Yet, despite the many studies examining a main effect of peer rejection as a risk factor for psychopathology, little theory initially had been offered to suggest why peer rejection may lead to externalizing symptoms. Indeed, children's peer acceptance/rejection likely does not act in isolation as a risk factor, but it may be one part of broader person–environment transactions that promote maladaptation. Recent theory and research suggests bidirectional effects between peer rejection and psychopathology. Moreover, there is some evidence that children's experiences with peers act in concert with perturbations in other developmental systems, offering a more thorough understanding of how peer rejection may contribute to trajectories of psychopathology.

Aggressive behavior and peer rejection are mutually reinforcing; results reveal bidirectional effects between peer rejection and externalizing problems beginning in early childhood (Sturaro, van Lier, Cuijpers, & Koot, 2011). For instance, youths' aggressive behavior, particularly reactive aggression, may be a response to exclusion, victimization, or other manifestations of rejection by peers; simultaneously, in most contexts, aggressive behavior elicits aversive responses from others (Dodge et al., 2003). Interestingly, the stability of peer rejection is greater among aggressive youth than among nonaggressive youth (Cillessen, van IJzendoorn, Van Lieshout, & Hartup, 1992). The combination of peer rejection and aggression delays developmental competencies more pervasively than either of these characteristics alone (Cillessen et al., 1992) and predicts later externalizing symptoms especially potently (Bierman & Wargo, 1995). For example, Bierman and Wargo (1995) revealed that compared with rejected/nonaggressive or nonrejected/aggressive youth, rejected/aggressive boys exhibited the highest risks for later externalizing symptoms. Coie and colleagues (1995) demonstrated that compared with others rejected/aggressive boys had the steepest increasing trajectories of self-reported externalizing problems beginning in the third grade and consistent levels of elevated parent-reported behavior problems beginning in the sixth grade for over four years. Additionally, Lochman and Wayland (1994) revealed that peer rejection moderated the association between aggressive behavior and externalizing symptoms four years later.

Some of the especially pernicious effects of peer rejection and aggression on later externalizing symptoms may be due to interplay between youths' interpersonal functioning and their social-cognitive development. In particular, much past work has focused on the manner in which children's peer experiences may be associated with specific social information processing behaviors and how these behaviors may mediate or moderate longitudinal associations with later psychological symptoms.

Crick and Dodge's (1994) social information processing model most often is cited in the study of peer relations and social information processing. The model indicates that individuals' responses within interpersonal interactions (i.e., to a specific social stimulus) are mediated by several specific cognitive steps; intra- and interindividual variability at each of these steps is associated with interpersonal schema, scripts, and data from past experiences. The presence of cognitive biases at any of these steps alters the trajectory of subsequent processes, thus leading to aberrant or maladaptive behavior. Specifically, this model suggests that individuals first encode social stimuli, generate an interpretation (i.e., causal and intent attributions) of encoded information, and form social goals. Behavioral strategies are generated, selected, and enacted that follow from these social goals.

Rejected, aggressive, and rejected/aggressive children are at greater risk than others to develop a unique cognitive bias suggesting a tendency to interpret ambiguous or benign social stimuli as due to hostile intent (i.e., a hostile attribution bias) (e.g., Crick & Dodge, 1996). This hostile attribution bias is associated significantly with a tendency to generate aggressive solutions to hypothetical provocation dilemmas and to behave aggressively toward peers (Dodge, Bates, & Pettit, 1990). Moreover, a hostile attribution bias is associated with a greater risk for continued and persistent aggressive behavior across development (Dodge, Pettit, Bates, & Valente, 1995). Research suggests that individuals with a hostile attribution bias may be especially likely to generate, select, and enact aggressive behaviors (Orobio de Castro, Veerman, Koops, Bosch, & Monshouwer, 2002). Effects between hostile attribution biases and aggression are significantly stronger among peer-rejected children (Orobio de Castro et al., 2002).

Recent work offers compelling evidence for transactions among peer rejection, aggression, and a hostile attribution bias in a manner that is consistent with dynamic systems theory. Examining a developmental cascade model, Lansford, Malone, Dodge, Pettit, and Bates (2010) demonstrated mutually reinforcing, and gradually escalating longitudinal associations between these three constructs over time within a sample of young children over four years (i.e., kindergarten to grade three). Moreover, results suggested direct and indirect effects between these constructs across 12 time points (Lansford et al., 2010). Dodge, Greenberg, Malone, (2008) further demonstrated that a hostile attribution bias and peer rejection are included within a sophisticated developmental cascade model linking adverse social contexts and harsh parenting with later externalizing behaviors, deviant peer group affiliation, and serious violence in adolescence.

Research on children's peer relations most often examines community-based samples, examining risks for elevated, if not clinically significant, levels of psychopathology. However, studies of clinically referred youth similarly support a link between peer rejection, social-emotional-cognitive deficits, and externalizing symptoms. For instance, studies of maltreated youth consistently offer evidence for poor emotion regulation strategies, greater physiological reactivity, and concomitant rejection by peers (e.g., Rogosch, Cicchetti, & Aber, 1995). Peer rejection also may partially mediate the continuity of externalizing symptoms among maltreated youth (Kim & Cicchetti, 2010).

The peer relations of children with attention-deficit/hyperactivity disorder (ADHD) have been examined somewhat frequently; results suggest that these children are particularly likely to be rejected by peers (see Landau & Moore, 1991, for a review). Given that many of the behavioral symptoms most associated with ADHD (e.g., talking and fidgeting excessively, impulse control problems, inattention) are socially aversive and that children with ADHD tend to be aggressive, links with peer rejection are not entirely surprising. Erhardt and Hinshaw (1994) found that after only three days of interacting with previously unfamiliar peers at summer camp, children with ADHD earned lower liking ratings and were overwhelmingly more rejected than campers without ADHD. Aggression and disruptiveness, two major behavioral features of ADHD, were the strongest predictors of negative peer status across both groups. In another study utilizing peer sociometric methods, Hoza and colleagues (2005) found that a disproportionate number of boys and girls with ADHD were rejected by age- and sex-matched peers. Notably, peer rejection among children with ADHD has been linked to an increased risk for externalizing problems. Specifically, high levels of peer rejection assessed approximately 20 months postclinical treatment for ADHD prospectively predicts externalizing problems over a subsequent four-year period (Mrug et al., 2012).

Children with ADHD also demonstrate certain social cognitive deficits, including both cue encoding and cue interpretation biases (see literature on positive illusory biases; Hoza, Murray-Close, Arnold, Hinshaw, & Hechtman, 2010). Indeed, despite their low sociometric status and probable experience of peer rejection, children with ADHD tend to perceive their own social acceptance as no worse than controls (Hoza, Pelham, Milich, Pillow, & McBridge, 1993) or even see their social performance and behavior as more favorable than their peers' (Hoza, Waschbusch, Pelham, Molina, & Milich, 2000). Recent work indicates that among children with ADHD, these social-cognitive deficits are associated longitudinally with diminished social skills, peer rejection, and increases in aggressive behavior. Moreover, peer rejection reciprocally predicts exacerbations in aggressive behavior, positive illusory biases, and social skill deficits, supporting a dynamic systems theory (Murray-Close et al., 2010).

It should be noted that much of the research examining links between peer acceptance/rejection and externalizing symptoms has focused on boys. Given substantial gender differences in the prevalence of many externalizing symptoms, particularly during the years in which much of this research was conducted, a focus on boys' externalizing

symptoms offered an important opportunity to examine predictors of behaviors that are of high public health priority. However, recent data regarding sharply increasing levels of externalizing behaviors among young females have prompted greater attention to gender diversity.

As among boys, results indicate that peer rejection, particularly in combination with aggressive behavior, is an especially potent predictor of later externalizing behavior among girls (Prinstein & La Greca, 2004). However, in the past 20 years, research has identified a set of aggressive behaviors that may be more common among girls than the types of overt and physically violent behaviors assessed in prior work on boys (Crick & Grotpeter, 1995; Galen & Underwood, 1997; Lagerspetz, Bjorkqvist, & Peltonen, 1988). Studies of peer reputations and later externalizing behaviors sometimes have focused specifically on the prediction of social aggression or relational aggression. Unlike physical forms of aggression, social and relational forms of aggression utilize one's social reputation or an interpersonal relationship as a target for harm (e.g., through gossip, social exclusion, withdrawal of friendship support). As with the prediction of overt/physical externalizing symptoms among boys, peer rejection is a significant predictor of social and relational aggression. Werner and Crick (2004) revealed that peer rejection among children was associated with higher levels of girls' relational aggression one year later. Comparatively little work has examined whether links between peer acceptance/rejection and social/relational forms of aggression also may be best understood through social-cognitive deficits, however, extant research suggests that processes may be similar to the results for overt aggression (Mathieson et al., 2011).

Peer Acceptance/Rejection and Internalizing Symptoms

Compared with work examining externalizing outcomes, studies examining links between peer acceptance/rejection and internalizing symptoms are remarkably rare. This may be due to mixed results in extant research. When examining internalizing symptoms as a broad domain of psychopathology (e.g., from an overall symptom checklist) peer rejection is a significant statistical predictor (e.g., Coie, Lochman, Terry, & Hyman, 1992). Some evidence suggests that peer rejection may act in combination with other behavioral traits, such as aggression (Coie et al., 1992) or withdrawal (Bell-Dolan, Foster, & Christopher, 1995) to predict internalizing symptoms longitudinally. Peer rejection also may be an especially potent predictor of internalizing difficulties when rejection is chronic over multiple years (Burks, Dodge, & Price, 1995). Theoretical accounts for why peer acceptance/rejection may lead to internalizing symptoms are perhaps best conceptualized to the study of specific symptom domains.

For example, a rather straightforward hypothesis suggests that peer rejection may be associated with (1) social isolation, (2) failures to establish close, supportive friendships; or (3) increased risk for aversive peer experiences that reinforce a sense of lack of belongingness; each of these experiences may contribute to loneliness. Although loneliness is not specifically an internalizing disorder in itself, it is a symptom of mood disorders, and an especially important predictor of suicidality. Indeed, research examining specific internalizing outcomes that follow peer rejection has revealed especially potent longitudinal effects for loneliness, including work across the school-age years (Ladd & Burgess, 1999) and at the adolescent transition (Boivin et al., 1995). Some evidence for a link between peer rejection, isolation, and loneliness also is available. Specifically, Pedersen and colleagues (2007) suggested that peer rejection is prospectively associated with loneliness over time, both directly and partially mediated by fewer mutual friends (Pedersen, Vitaro, Barker, & Borge, 2007).

Longitudinal associations between peer rejection and depressive symptoms have been more elusive, however. Some research has revealed links between peer rejection and later depressive symptoms (Kiesner, 2002; Panak & Garber, 1992). For instance, depressed youth are more likely to receive negative sociometric nominations than are nondepressed children (Kennedy, Spence, & Hensley, 1989). However, other research has failed to replicate these effects (e.g., Woodward & Fergusson, 1999). Recent work alternatively has begun to conceptualize peer rejection as a potential stressor within contemporary cognitive theories of depression. Cognitive models generally suggest that stressful experiences can activate dysfunctional cognitive schemas (Beck, 1976), affecting social-cognitive processing of subsequent social stimuli. Stressful experiences may influence one's perception of stimuli as aversive (i.e., threatening or affirming low self-worth), or affect one's interpretation of stimuli through the use of maladaptive explanatory styles (e.g., a depressogenic attributional style; Abramson, Metalsky, & Alloy, 1989). Research supports transactional, reciprocal associations between depressive symptoms and the development of negative cognitions (Garber, Keiley, & Martin, 2002).

Cognitions have been cited as important mediators or moderators of the links between peer rejection and depressive symptoms in prior work. Some of these cognitions pertain to youths' perceptions of their own peer status. For example, youths' perceptions of rejection by peers are more

proximal longitudinal predictors of depression (e.g., Kistner, Balthazor, Risi, & Burton, 1999) and may mediate the association between actual peer rejection and later depressive symptoms (Panak & Garber, 1992). Youths' underestimations of their levels of peer acceptance are predicted by their depressive symptoms (De Los Reyes & Prinstein, 2004; Rudolph & Clark, 2001), and these underestimations reciprocally are associated with later increases in depression (Cillessen & Bellmore, 1999).

Youth with specific cognitive dispositions also appear to be at heightened risk for the deleterious effects of peer rejection on depressive symptoms. For example, consistent with cognitive vulnerability models of depression, youth with a depressogenic attributional style (i.e., a tendency to interpret negative life events as due to internal, global, and stable causes, and positive events to external, unstable, and specific causes) appear to be at greater risk for increases in depressive symptoms if also rated by peers as rejected, compared with nonrejected peers (Panak & Garber, 1992). Consistent with self-discrepancy theory (Higgins, 1987) youth who place high value on their level of peer status, yet are rejected by peers (i.e., a condition that would produce cognitive dissonance) appear to be especially at risk for increases in depressive symptoms over time (Prinstein & Aikins, 2004). In sum, research has indicated that compared with externalizing symptoms, internalizing outcomes are less consistently associated with early peer rejection. However, research increasingly suggests that peer rejection may be a relevant stressor interacting with prior vulnerabilities for internalizing symptoms, consistent with a transactional hypothesis. For instance, a recent study demonstrated that the role of peer rejection on the development of youths' internalizing problems could be better conceptualized within a cascade model, in which externalizing problems contribute to the development of internalizing psychopathology via negative peer relationships. Specifically, high levels of externalizing problems in kindergarten were found to predict negative peer relationships, including peer rejection and peer victimization, in first grade. In turn, transactional effects were revealed between peer rejection and peer victimization from first to third grade; ultimately, these effects resulted in peer rejection in third grade to predict internalizing (as well as externalizing) problems in fourth grade (van Lier & Koot, 2010). Conversely, among maltreated youth, evidence suggests that adaptive levels of emotional regulation are associated with greater peer acceptance, ultimately lowering the longitudinal risk for internalizing symptoms (Kim & Cicchetti, 2010).

Peer Rejection and Immune System Activity: A Biological Pathway to Internalizing Symptoms

There are many theoretical reasons to suspect that peer rejection is especially relevant to the development of internalizing symptoms, particularly depression. However, research in the peer relations literature has not yet fully taken advantage of opportunities to integrate with related theories or disciplines. For example, research focusing on social genomics and psychophysiological processes related to interpersonal stress events suggests that the innate immune system may play an important role in understanding the long-term effects of stressful interpersonal experiences on the development of psychopathology. In short, social adversity is related to immune system responses that in turn may affect the synthesis of monoamines implicated in depressive symptoms. A brief review is offered below.

One of the key functions of the innate immune system is to eliminate pathogens (e.g., bacteria) and to enhance wound healing by activating inflammatory response in white blood cells (i.e., leukocytes). The activity of the innate immune system is genetically regulated and begins when, in response to infections and injuries extracellular signals activate the release of intracellular proteins, specifically the transcription factor nuclear factor kappa-B (NF-kB), from its inhibitory protein. The activated NF-kB moves from the cytoplasm of the cell to the nucleus where it activates genes that encode pro-inflammatory cytokines (i.e., cell-signaling proteins). Pro-inflammatory cytokines (e.g., interleukin-1; tumor necrosis factor-α (TNF-α)) are protein molecules that drive the immune system response by attracting leukocytes (e.g., macrophages) toward the injured or infected body areas in order to expel the pathogens or heal the wound.

Researchers recently have acknowledged that the social environment also may regulate the activity of the genes involved in the innate immune response (see Irwin & Cole, 2011; Slavich & Cole, 2013). Specifically, it has been suggested that adverse social experiences (i.e., interpersonal stress) may influence the expression of inflammatory genes. The effect of interpersonal stress on inflammatory activity may, at least partially, occur via the endocrine and sympathetic nervous system (SNS). This process starts when social stressors activate a series of adaptive physiological responses within the central nervous system involving both the hypothalamic-pituitary-adrenal (HPA) axis and the SNS. The HPA axis and the SNS stimulation can subsequently influence the activity of pro-inflammatory genes in the leukocytes. For instance, glucocorticoids released by

the HPA axis inhibit pro-inflammatory activity. Specifically, glucocorticoids (e.g., cortisol) bind to glucocorticoid receptors (i.e., intracellular proteins) in the cytoplasm of leukocytes and move in the nucleus where they reduce the activity of one of the major pro-inflammatory transcription factor, NF-kB. Conversely, catecholamines (e.g., norepinephrines) released by the SNS in several body tissues to stimulate a fight-or-flight reaction activates the transcription factor NF-kB, thus promoting pro-inflammatory activity.

Notably, all these biological responses are expected to be regulated by the central nervous system, meaning that only if the individual perceives the external social stimuli as threatening these processes occur (Slavich & Cole, 2013). In other words, the way individuals perceive external social stimuli, rather than the stimuli themselves, give rise to these biological responses.

An increasing number of observational and experimental studies have provided empirical support for the influence of social stress on the immune system activity. First, it has been demonstrated that chronic interpersonal stress may lead to greater pro-inflammatory reactions in response to other psychological stressors (e.g., recent episodic stress) or pathogenic agents (e.g., bacteria). For example, chronic interpersonal stress was found to lead to higher production of interleukin-6 (IL-6), a pro-inflammatory cytokine, in response to bacterial stimuli (Miller & Chen 2010). Second, high levels of biomarkers indicating (anti- and) pro-inflammatory activity have been found in individuals who experienced (chronic) interpersonal stress, even in the absence of external agents (e.g., pathogens). Specifically, these studies revealed that greater stress was associated with a) increasing levels of messenger RNA (mRNA) for the transcription factor NF-kB (e.g., Murphy, Slavich, Rohleder, & Miller, 2013), and b) increasing levels of systemic pro-inflammatory activity in the bloodstream, as reflected in high levels of circulating pro-inflammatory markers, including IL-6, or C-reactive protein (CRP) (Fuligni et al., 2009). Third, evidence supporting the hypothesis that chronic social stress may lead to sustained inflammation comes from genome-wide studies comparing transcriptional activity of individuals with and without histories of chronic stress. Among individuals with chronic stress, genes bearing pro-inflammatory activity are up-regulated whereas genes bearing anti-inflammatory activity are down-regulated (e.g., Cole et al., 2007).

Two important aspects from this work are herein potentially noteworthy for studies of peer relationships. First, research suggests that early-life stress, early interpersonal stressors, and chronic interpersonal stress experienced later in life may be related to pro-inflammatory activity. That is, acute forms of stress during developmentally sensitive periods (e.g., early childhood) as well as chronic forms of stress occurring at different developmental periods may both result in enhanced inflammation (Miller, Chen, & Parker, 2011). For instance, children raised in a stressful family context, characterized by maltreatment and harsh parenting, tend to show higher inflammatory activity later in life (for a review, see Miller et al., 2011). Accordingly, during adolescence and adulthood, recent experiences with chronic interpersonal stress are associated with higher inflammatory activity (e.g., Miller, Rohleder, & Cole, 2009). Second, among the interpersonal stressors that may influence the immune system, social stressors that directly threaten individual social connection, including social rejection, may be especially powerful. For example, Murphy and colleagues (2013) examined adolescent females' inflammatory activity during six-month intervals over two years. During some of these six-month intervals, adolescents experienced social rejection experiences, but during other time intervals they did not. Social rejection experiences were associated with adolescent females' higher inflammatory activity, as indicated by greater levels of mRNA for the transcription factor NF-kB and the inhibitory factor I-kB, as well as a lower ratio of NF-kB/I-kB. Recent findings from a large population-based study (Copeland et al., 2014) indicate that being bullied during childhood and adolescence prospectively predicted higher levels of inflammatory markers (CRP) several years later during early adulthood, even after accounting for prior levels of inflammation and a number of relevant physical and mental health covariates (e.g., body mass index, early life-adversity). Interestingly, contrary to bully victims, children who were identified as bully perpetrators showed lower levels of inflammatory markers during early adulthood than children who were not involved in bullying. These social rejection experiences may be particularly harmful as they may directly threaten the innate human need to belong (Slavich, O'Donovan, Epel, & Kemeny, 2010). In sum, it seems that throughout childhood and adolescence, interpersonal stress events, including social rejection, may regulate the expression of genes involved in the immune system and may therefore lead to chronic mild inflammation.

The effect of stress on inflammatory activity may derive from innate adaptive biological responses to prepare the body to deal with potential injuries and infections arising from adverse physical threats (see Slavich & Cole, 2013). However, under conditions of chronic stress or during

developmentally sensitive periods (e.g., early childhood), these adaptive biological reactions may lead to stable physiological changes (i.e., allostatic load), including mild levels of inflammatory activity, which, in turn, may have long-term consequences on individuals' psychological well-being and similarly increase risk for psychopathology.

To date, the specific molecular pathways through which stress gets under the skin and leads to sustained levels of inflammation have not yet been fully elucidated. One of the most empirically supported hypotheses suggests that the glucocorticoid receptor (GR), a ligand-activated transcription factor, may play a crucial role in linking social stress to stable pro-inflammatory activity (see Irwin & Cole, 2011). The GR is an intracellular protein that regulates gene expression by binding to glucocorticoids. The GR has a major role in inhibiting both HPA axis (i.e., glucocorticoids release) and pro-inflammatory activity (acting on NF-kB; see Meaney, 2010). Two main lines of research have provided evidence supporting the importance of GR to explain the link between stress and chronic inflammation. First, studies have shown that GR-mediated gene transcription was reduced in individuals with a history of chronic stress, a phenomenon referred to as GR resistance (or GR desensitization; see Miller et al., 2011). Specifically, findings from this work suggest that exposure to chronic stress may reduce immune cells sensitivity to the anti-inflammatory effect of glucocorticoids (Cohen et al., 2012). Notably, this phenomenon seems to depend not on the unavailability of GR, as indicated by similar levels of mRNA for GR in individuals with and without chronic stress (Miller, Chen et al., 2009). Instead, it may be the case that posttranslational modifications are responsible for GR resistance, explaining why high levels of glucocorticoids and pro-inflammatory cytokines may co-exist even with available quantities of GRs (see Miller et al., 2011).

Second, evidence from animal studies have demonstrated that exposure to early-life stress may result in epigenetic modifications (e.g., DNA methylation) in the glucocorticoid receptor gene in the hippocampus. Specifically, these studies revealed that in rats, high maternal care (e.g., high licking and grooming) in the first week of life was associated with higher expression of the GR gene in the hippocampus (Weaver et al., 2004). Notably, because GR has a negative-feedback loop on glucocorticoids release, higher GR expression led to lower HPA axis reactivity to stress. A similar epigenetic variation in response to chronic stress (or early life stress) also may occur in immune cells, which may contribute to maintain chronic mild levels of inflammation. Although evidence for DNA methylation of the GR gene in immune cells has not been provided yet, this hypothesis would converge with recent work suggesting that epigenetic modifications are system-wide (that is, they occur across different types of cells; Szyf & Bick, 2013). These molecular modifications may explain how immediate physiological responses resulting from stress exposure may yield long-term physiological changes, including inflammation.

Of particular relevance, sustained levels of inflammation resulting from exposure to chronic interpersonal stress may be linked to a higher risk of depressive symptoms (see Slavich & Irwin, 2014, for a review). The key role that inflammation may exert on the development of psychopathology is supported by the known effects of pro-inflammatory cytokines on central nervous system activity (Miller, Maletic, & Raison, 2009). Throughout different molecular pathways, inflammatory activity in the periphery of the body may reach the brain and activate pro-inflammatory responses by microglial cells (i.e., brain immune system cells) in the central nervous system (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008). In the brain, pro-inflammatory cytokines (e.g., TNF-α) elicit sickness behaviors which imply emotional, motor and cognitive alterations (Irwin & Cole, 2011). Notably, these changes strongly correspond to the behavioral symptoms which characterize depression, including social withdrawal, fatigue, and physical inactivity. This has led researchers to hypothesize that pro-inflammatory cytokines in the brain may induce depression. Specifically, pro-inflammatory cytokines have been suggested to influence the release of monoamines (e.g., serotonin and dopamine) in the brain, which may have direct effects on the development of psychopathology in general, and depression in particular (Miller, Maletic, et al., 2009). Prior work has provided support for this hypothesis by showing that high levels of inflammation are associated with increased risk for depression (e.g., Miller, Maletic, et al., 2009). For example, high levels of circulating CRP and IL-6 in the bloodstream were found to predict depressive symptoms over a 12-year period (Gimeno et al., 2009). More importantly in this context, other recent work has specifically provided some empirical support for the hypothesis that inflammation may give rise to one of the biological pathways linking social stress to depression. In an observational study (Miller & Cole, 2012), pro-inflammatory activity and depression were found to cluster together among female adolescents who experienced high levels of adversity during their childhood. Of specific interest, high levels of IL-6 led to increased risk for depression six months later only among adolescents who reported childhood adversity; however, this association was not found in female

adolescents who did not experience childhood adversity. In another study (Eisenberger, Inagaki, Rameson, Mashal, & Irwin, 2009), the effect of pro-inflammatory cytokines on depressive symptoms was examined in an experimental design. Specifically, participants' pro-inflammatory activity was experimentally manipulated by administrating either endotoxin (i.e., microbial stimuli) or placebo. Subsequently, participants in both conditions were exposed to a social rejection task while assessing neural activity using fMRI. As expected, administration of endotoxin increased both participants' pro-inflammatory activity, measured as levels of circulating IL-6, and depressive symptoms. Moreover, although no differences in neural responses to social exclusion were found between participants in the endotoxin and placebo condition, circulating levels of IL-6 in response to endotoxin emerged to be positively associated with neural activity in the anterior and posterior insula of the brain. Notably, neural activity in response to the social stress task mediated the association between inflammation and depressive symptoms (among females only).

In sum, research suggests that chronic social rejection is associated with unique immune system, epigenetic, and neuropsychological responses that have clear implications for the study of peer relationships, and potentially peer rejection in particular. Unfortunately, few studies have operationalized youths' chronic social rejection in a manner that is consistent with developmental theory or findings within the peer relations literature. This remains an important direction for future work.

Peer Acceptance/Rejection and Health Risk Behaviors

Health risk behaviors have been examined relatively infrequently within the literature on peer status compared with research that has focused on traditional diagnostic symptoms. However, some work has examined the association between peer acceptance/rejection and substance use, largely supporting the hypothesis that peer rejection in childhood is associated with long-term substance use (i.e., nicotine, alcohol, and marijuana) outcomes, especially among boys (Dishion, Capaldi, Spracklen, & Li, 1995; but also see Allen, Porter, McFarland, Marsh, & McElhaney, 2005). One study suggests that children rejected at ages 10–13 years are eight times more likely than sociometrically average children to abuse alcohol as young adults up to 10 years later (Zettergren, Bergman, & Wångby, 2006).

Similar to findings on externalizing outcomes, results also indicate that a combination of peer rejection and aggression is an especially potent predictor of substance use outcomes. Rejected/antisocial eighth-grade students are more likely to use tobacco and alcohol two years later compared with rejected, nonantisocial or peer accepted students (French, Conrad, & Turner, 1995). Among girls in fourth through sixth grade, peer rejection moderates the long-term association between peer aggression and adolescent substance use. Among girls with low social preference scores (i.e., peer rejection), peer aggression was significantly associated with heavy episodic drinking, marijuana, and hard drug use six years later; however, among girls with high social preference scores, peer aggression was not a significant predictor of later substance use (Prinstein & La Greca, 2004).

Although studied rarely, peer acceptance/rejection may be associated longitudinally with several additional health risk behaviors. However, findings have yielded sometimes conflicting patterns of results. For example, Miller-Johnson and colleagues (1999) revealed that childhood aggression, but not peer rejection, significantly predicted teenage motherhood (see also Feldman, Rosenthal, Brown, & Canning, 1995). Underwood, Kupersmidt, and Coie (1996) revealed that girls with childhood controversial sociometric status (i.e., both liked and disliked by peers) and aggressive girls were more likely to give birth in adolescence, had experienced more births than other adolescent mothers, and had given birth to their first child at earlier ages compared with other adolescent mothers. Brendgen, Wanner, and Vitaro (2007) revealed indirect associations between peer rejection and early sexual initiation for girls via lower levels of self-esteem.

With regard to weight-related risk behaviors, Lieberman, Gauvin, Bukowski, and White (2001) found that higher levels of girls' friendships nominations from peers (i.e., a possible proxy for peer acceptance) were associated with lower levels of body self-esteem and higher levels of dieting and bulimic behavior (Lieberman et al., 2001). Yet Graham, Eich, Kephart, and Peterson (2000) examined associations between peer ratings of likeability among peers, revealing that peer acceptance was associated with lower levels of body dissatisfaction (see also Rancourt & Prinstein, 2010). Research examining links between peer factors and self-injury are rare, but at least some short-term longitudinal evidence suggests that peer rejection is associated with later increases in suicidal ideation (Heilbron & Prinstein, 2010).

Social information processing models may be relevant for understanding links between childhood peer rejection and adolescent health risk behaviors, perhaps due to the mediating effects of social problem-solving skills deficits or broader emotion dysregulation that influences both cue interpretations and later behavioral enactment (Lemerise & Arsenio, 2000). Consistent with a broader dynamic

systems theory, it also has been suggested that childhood peer rejection inhibits adaptive social skill development, consequently narrowing later friendship choices to other similarly skilled peers. Together, these friendships between rejected and maladjusted (e.g., aggressive) youth form deviant peer groups, creating a powerful context for the socialization of a large array of risk behaviors.

Data offer support for the critical role of peer rejection and low social skills in the development of deviant peer group affiliation, particularly when peer rejection is combined with school failure and poor parental management (e.g., Dishion, Patterson, & Griesler, 1994). Patterson, Dishion, and Yoerger (2000) further suggest that early involvement with deviant peers at age 10 is associated with increases in new forms of risk behavior by age 14. Accordingly, in a recent study testing a developmental cascade model of adolescent substance use, peer rejection assessed in early childhood was associated with an increased probability in substance use engagement (i.e., cigarette and marijuana use) throughout high school, via affiliation with deviant peers in seventh grade (Lynne-Landsman, Bradshaw, & Ialongo, 2010). An indirect pathway also was found between affiliation with deviant peers in seventh grade and substance use trajectory membership during high school via eighth-grade conduct problems. These findings clearly emphasize how peer rejection may have long-term effects on health behaviors by affecting other peer domains such as friend affiliation.

Peer-Perceived Popularity

Peer status is a construct that has important developmental heterogeneity in its operationalization. Among preschool-age and young school-age children, youths' peer status can be understood as an index of how much children are well liked. However, by the middle of the school-age period, a separate construct emerges that reflects the extent to which youth have reputations of popularity. Here, *popularity* refers to an individual's levels of status, dominance, access to resources, and prestige (Vaughn & Waters, 1981), which are not isomorphic constructs to individual's likeability among peers. This distinct construct has been examined for decades by sociologists, human ethologists, and within ethnographical research (Cillessen, Schwartz, & Mayeux, 2011). However, it was more formally introduced to the study of peer relationships in the developmental psychopathology literature in the late 1990s (Parkhurst & Hopmeyer, 1998), and subsequent work revealed that it becomes further differentiated from peer acceptance/rejection as children grow older (Cillessen & Mayeux, 2004). As with peer acceptance/rejection, popularity traditionally is measured using peer nominations. Youth select peers who are most and least popular, and a standardized difference score between standardized tallies of nominations on each item is computed to provide a continuous measure of popularity, or social reputation. Popularity is only moderately correlated with peer acceptance/rejection by early adolescence (Parkhurst & Hopmeyer, 1998), and a substantial proportion of youth high in popularity meet criteria for the controversial sociometric status group.

Research examining popularity reveals somewhat surprising associations with measures of adjustment. Substantial research indicates that peer rejection is both a determinant and consequence of aggressive behavior. In contrast, reciprocal associations between high levels of popularity and aggressive behavior have been revealed (Cillessen & Rose, 2005). High levels of popularity also are associated with several health risk behaviors, including alcohol use, sexual risk behavior, and engagement in drug use (Prinstein, Choukas-Bradley, Helms, Brechwald, & Rancourt, 2011). Given the relatively new distinction between peer acceptance/rejection and peer popularity, it is unknown whether popularity may be relevant to the development of psychopathology. As discussed in turn, research suggests that popularity may be highly relevant for identifying the most influential peers within a social context, however. Peer influence proves to be a powerful process related to psychopathology risk, especially in adolescence.

In sum, compared with research conducted only 15–20 years ago, research on peer status has become fairly dormant. Rarely do researchers examine peer sociometric status categories. Moreover, studies of peer acceptance/rejection have been conducted infrequently, in part due to greater restrictions among human subjects committees and disproven fears among school administrators regarding the potential harm that peer nominations may cause other children (see Bell-Dolan, Foster, & Sikora, 1989). This may be quite unfortunate, as peer acceptance/rejection has proven to be a powerful variable that can be measured quite well and is remarkably stable over time, suggesting an important source of chronic interpersonal rejection and stress.

Peer Victimization

Although somewhat neglected for several decades within the peer relations research literature, the study of peer victimization recently has become quite popular, particularly since the onslaught of school shootings and youth suicides that have been attributed to peer victimization experiences.

Peer victimization certainly is far more common among youth rejected by peers; however, the two constructs are not isomorphic. For example, compared with peer rejection, peer victimization is correlated differently (and sometimes in different directions) with aggression. As discussed below, this difference is due, in part, to different aggressive functions and to only recently articulated differences between aggression (broadly defined) and bullying behavior (Salmivalli & Peets, 2009).

Peer victimization refers to experiences in which one has been the target of intentional, repeated, aggression from peers. A subtle but important distinction has been offered between being the recipient of peer victimization (i.e., a more inclusive term representing broadly defined peer aggressive acts) and bullying behavior, which is focused specifically on situations in which the target is less powerful than the perpetrator of peer aggression (Salmivalli & Peets, 2009).

Moreover, there is substantial heterogeneity in aggressive behavior, which has been a focus of recent research. Aggressive behavior varies not only in function, but also in form. Semantic issues are problematic in the literature, and there is a need for uniformity to help create a more consistent dialogue in this rapidly growing area of research. A brief review of forms and functions of aggression is offered.

Aggression and Bullying

Compared with the study of peer victimization, studies of aggressive behavior are as old as psychological research itself. Almost a century ago, Dollard, Doob, Miller, Mowrer, and Sears (1939) posited that aggression served a function of behaviorally expressing internal feelings of frustration (i.e., the frustration-aggression hypothesis). *Frustration* is defined as an affective response to an inability to achieve one's goal. Thus, this aggressive function is referred to as reactive aggression and it is considered to be hot-blooded and reflective of impulsivity and poor emotional regulation skills. Frustration can be a reaction to interpersonal goal-blocking (e.g., a peer taking away a toy), or it can be internally derived (e.g., failure to accomplish a solitary task). For interpersonal sources of frustration, reactive aggression is associated with short-term behavioral reinforcement, but long-term consequences. For example, aggression directed toward a peer who has caused frustration can produce immediate results (e.g., return of the stolen toy). However, in most contexts aggression is regarded as an inappropriate behavior, and thus is associated with long-term decreases in peer status and sanctions from adults. High levels of reactive aggression among youth are associated with higher levels of prior maltreatment experiences, and lower social preference (Dodge, Lochman, Harnish, Bates, & Pettit, 1997). Higher levels of youth's reactive aggression also are associated with unique physiological responses to stress. Hubbard and colleagues revealed that compared with predominantly proactively aggressive youth, children with high levels teacher-reported reactive aggression evidenced greater parasympathetic reactivity in response to an experimentally induced stressor (Hubbard et al., 2002).

Aggressive behavior is not always a behavioral response to frustration. Proactive aggression is conceptualized as cold-blooded, planful, manipulative, and strategic (Dodge & Coie, 1987). Proactive aggression involves the use of aggression to obtain a specific reward. Rewards may include instrumental goals (i.e., access to resources) or person-oriented goals, typically to assert dominance. Successful uses of proactive aggression (i.e., acts that produce the desired reward) are behaviorally reinforced, as articulated by social learning theories (Bandura, 1973).

Proactive and reactive functions of aggression can be identified in a variety of species and are considered to be inherent characteristics of social groups. Ethologists contend that aggression contributes to social organization by enforcing a social hierarchy, group structure, and transparent, if not equitable allocation of resources among group members. Characteristics of aggressive behavior observed among nonhuman species have been directly adapted to the study of youth (Vaughn & Waters, 1981).

Bullying has been defined as a specific example of proactive aggression (Hartup, 1974). Bullying is directed toward members of one's social group who occupy lower levels on the status hierarchy. Interestingly, ethological research suggests that the highest members of a status hierarchy typically are unchallenged and do not need to use aggression to maintain their social ranking. Aggression is more common among members of social hierarchy at near-adjacent positions, who realistically may vie for a reorganization within their group. An explicit goal of bullying is to elevate the perception of the perpetrator's dominance by reducing the power of the victim. Note that proactive and reactive uses of aggression have different social-psychological correlates. Proactive uses of aggression are associated longitudinally with increases in (reputationally based) popularity; reactive aggression is associated with increases in peer rejection (Cillessen & Rose, 2005; Prinstein & Cillessen, 2003). Proactive aggression is associated with later delinquency, while reactive aggression is associated with greater risk for internalizing symptoms, aggression within relationship contexts (e.g., dating violence), and social isolation (Brendgen, Vitaro,

Tremblay, & Lavoie, 2001; Vitaro, Brendgen, & Tremblay, 2000; Vitaro, Gendreau, Tremblay, & Oligny, 1998).

Unfortunately, peer bullying and victimization have not been conceptualized in a manner consistent with these theories within the peer relations literature. Peer aggression rarely is examined with regard to the functions of aggressive behavior, and assessments of aggression often conflate reactive and proactive functions of aggression. Consequently, assessments of peer victimization most often capture the victims of peers' aggressive behavior, but only recently has effort been made to specifically identify peers who have been victimized in manner that matches theoretical accounts of bullying.

Forms of Aggression and Victimization

In addition to variation in the functions of aggression, several different forms of aggressive behavior have been identified. Decades ago, research in this area was dominated by studies of mostly physically (i.e., hitting, kicking, pushing, etc.) and sometimes verbally (i.e., name calling, threatening) aggressive acts. Perhaps not surprisingly, these forms of aggression (alternately referred to as overt, direct, or physical aggression) largely were perpetrated by boys, and victims also were more likely to be male. Gender socialization likely contributes to the perception that exerting dominance among males involves acts that threaten boys' physical strength and feelings of power.

Compared with boys, girls are socialized to place greater value in their social abilities as a component of self-worth (Bem, 1981). Assertions of dominance among girls involve acts that render victims socially weak (e.g., friendless, alienated, or impaired by damaged social reputations). Aggressive acts targeting social standing have been referred to as relational, social, and indirect aggression (Crick & Grotpeter, 1995; Galen & Underwood, 1997; Lagerspetz et al., 1988). Examples include acts meant to damage social relationships, such as excluding others, silent treatments, threatening friendship withdrawal, and acts that are meant to damage social reputations, such as telling rumors. Relational forms of aggression may become more prevalent than overt aggression as age increases, reflecting both the greater sanctions that accompany overt aggression among older youth and older children's increasing cognitive abilities that is required to successfully execute some relationally aggressive acts. Active debate on gender differences in the perpetrators of these different forms of aggression remains; however, recent meta-analytic evidence suggests that gender differences may be minimal, and both forms of aggression (i.e., physical/overt and social/relational) are highly correlated (Card, Stucky, Sawalani, & Little, 2008). The victims of overt or relational aggression are equally distributed across gender.

The assessment of peer aggression, bullying, and victimization raises complex validity issues. As with many peer constructs, overt and relational aggression often is assessed using sociometric procedures. This approach builds on a long-standing traditional assumption within the peer relations literature regarding the conceptualization of peer aggression as group-level phenomena, observable by a broad sample of peers. Peer-nominations yield measures of one's reputations or preferences within an entire peer group (i.e., within a class or grade). Consensus has not been obtained regarding the conceptualization of relational aggression as a group- or dyadic-level peer experience, however. Some relationally aggressive acts (e.g., spreading rumors) involve the larger peer group more than others (e.g., threatening friendship termination). In fact, some forms of relational aggression may be most successfully executed when the victim is unaware of who the perpetrator is. Peer reports of relationally aggressive behavior therefore may oversample estimates of perpetrators who engage in relationally aggressive acts quite visibly and conspicuously.

The assessment of peer victimization also can utilize peer-reported approaches, raising similar questions regarding the ability to adequately capture experiences that are known only among members of a dyad or small friendship group. Alternatively, a large proportion of studies have been conducted utilizing self-reported measures of peer victimization. Compelling arguments for self-reported assessments of peer bullying and victimization are presented by Solberg and Olweus (2003). Yet, while this approach allows for a more thorough assessment of aggressive acts from the perspective of the person who has been targeted by them, data ultimately yield a measure of *perceptions* of peer victimization. Perceptions of peer victimization are relevant for understanding youths' own experience, and likely are relevant for understanding links with maladjustment. However, substantial affective biases can influence the retrospective recall and self-report of peer victimization (Des Los Reyes & Prinstein, 2004).

Last, in addition to the different functions of aggression and forms of aggression/victimization, recent technological developments have stimulated an interest in the medium in which aggression and victimization take place. Presumably, most prior research on peer aggression and victimization focused on the study of social interactions that appeared in person (i.e., face to face) or perhaps using the telephone (i.e., voice to voice). Today, an increasing number of peer victimization experiences occur using online media, such as

Facebook and YouTube. Cyber victimization is especially pernicious and may have unique consequences for maladjustment. Unlike other contexts in which victimization may occur, cyber victimization utilizes a much larger audience to torment youth; indeed, aggression literally can occur on an international stage. Moreover, using the Internet, it is especially easy for aggressors to act with complete anonymity, and perhaps engage in especially brutal attacks consequently. Research suggests that cyber victimization occurs with remarkable frequency, and certainly with particularly dire consequences in some cases (Smith et al., 2008). Yet, research still is needed to determine whether cyber victimization is associated with unique adjustment consequences compared with the effects of victimization occurring in traditional contexts. Studies that investigate both online and offline victimization have documented a strong overlap between these two phenomena. For instance a recent study examining joint developmental trajectories of online and offline victimization from age 12 to age 19.5 years old documented that all adolescents who followed a low trajectory of offline victimization also followed a low trajectory of online victimization. Accordingly, all adolescents who belonged to a moderate online victimization trajectory belonged either to a moderate or to a high offline victimization trajectory, indicating that online victimization always occurred in the presence of offline victimization (Sumter, Baumgartner, Valkenburg, & Peter, 2012).

Peer Victimization as a Predictor of Psychological Symptoms

One might assume that peer victimization is associated with psychological sequelae and dysfunctional behavior. The media has publicized several severe acts of violence (i.e., homicide, suicide) attributed to peer victimization experiences. The implementation of antibullying policies within many states and within several countries outside of North America also suggests that peer victimization is widely regarded as an important risk factor for severe psychological distress and symptoms of psychopathology.

Interestingly, however, empirical data have not suggested that peer victimization has deleterious effects of the magnitude that has been suggested by these well-publicized incidents and policy decisions. Certainly, there is strong evidence that peer victimization is associated with later adjustment difficulties (e.g., Hanish & Guerra, 2002; Schwartz, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1998). Meta-analyses also have indicated that peer victimization indeed is associated concurrently with a wide range of adjustment indices, including depression, loneliness, self-esteem, and anxiety (Hawker & Boulton, 2000). Recent meta-analyses also summarized prospective associations, revealing significant bi-directional associations between peer victimization and internalizing symptoms (Reijntjes, Kamphuis, Prinzie, & Telch, 2010), and peer victimization and externalizing symptoms (Reijntjes et al., 2011). Most surprising, however, is that effect sizes are relatively weak for the putative effects of victimization on symptoms of psychopathology (Cohen, 1988; Reijntjes et al., 2010), particularly when examining these associations in longitudinal designs that use different informants to measure each construct (i.e., reducing method variance issues). It is estimated that the vast majority of youth are victimized by their peers, at least at some point. Consequently, research on peer victimization has begun to consider potential alternate hypotheses to help conceptualize associations between peer victimization and psychopathological symptoms within more complex and dynamic theoretical models. At least five alternate hypotheses have received recent attention. First, peer victimization consequences should be considered in the context of the determinants of peer victimization. It may be that victimization is most relevant as a predictor of continuities or discontinuities in prior maladjustment. Second, peer victimization may predict maladjustment only for a subset of victims, such as those with other vulnerability factors, or those who may have unique cognitive or behavioral responses to being victimized. The study of moderators of the association between peer victimization and adjustment has been especially active and fruitful. Third, recent work has differentiated youth who are chronic versus sporadic victims of peer aggression. Chronic peer victimization may be a unique and far more rare experience with severe psychological consequences. Fourth, peer victimization may not be directly associated with psychological symptoms but may exert important effects within more complex transactional or dynamic models of psychopathology. Fifth, and relatedly, peer victimization may be conceptualized as a stressor with consequences for the development of psychophysiological stress-response systems. Recent work suggests unique biological responses among victims of peer aggression that may be associated with a variety of behavioral and physical health outcomes. Each of these hypotheses will be discussed in more detail.

In addition to these five recent emphases in current research on peer victimization, research in affective neuroscience has suggested that at least one form of peer victimization (i.e., social exclusion) may be associated with pain responses akin to the pain experienced from physical

stimuli. This exciting direction for future research will also be briefly reviewed.

Determinants and Consequences of Peer Victimization

Peer victimization often is depicted as a stressful experience that may promote maladaptation among otherwise well-adjusted youth. To be clear, there is no doubt that many victims of severe peer aggression or bullying indeed are random targets who have evidenced no prior vulnerabilities to psychopathology. However, data suggest that some youth may be more vulnerable than others to peer victimization. Two subtypes of victims have been identified. It should be noted that most of this research has focused on the determinants of overt victimization; research examining characteristics of relational forms of victimization is relatively rare. One group of youth especially prone toward aggression from peers is characterized by indices of psychological and physical passivity. Passive victims have greater levels of internalizing distress (higher levels of depressive and anxiety symptoms, lower self-esteem) and may be physically smaller and weaker than their bullies (Perry, Hodges, & Egan, 2001). Consistent with theories regarding proactive functions of aggression, children who exhibit characteristics that suggest they may be easily dominated by others may be easy targets for peers who wish to demonstrate their own power and potentially affirm or increase their own positions within the social hierarchy. A second group vulnerable to peer victimization has been referred to as aggressive victims. Aggressive-victims are characterized by poor emotional regulation skills, hostile attribution biases, and unlike nonvictimized bullies, they tend to be less strategic, even arbitrary, in their selection of aggression targets (Schwartz, 2000). The use of aggression among aggressive victims appears to most closely resemble the description of reactive aggression. Aggressive victims are especially likely to exhibit symptoms of psychopathology (Schwartz, 2000). Nevertheless, aggression is a strong predictor of peer victimization (Brendgen, Boivin, Vitaro, Girard, et al., 2008), and evidence even suggests that the association between aggression and peer victimization is partially explained by genetic factors (Boivin et al., 2013).

Given that at least some victims have been selected due to their prior levels of psychological symptoms or distress (i.e., internalizing symptoms among passive victims, and externalizing symptoms among aggressive victims), it perhaps is not surprising that peer victimization is not prospectively associated with the *onset* of psychopathology. Indeed, recent work that examines reciprocal associations between psychopathology symptoms and peer victimization do not consistently reveal deleterious effects of victimization on symptoms after controlling for the effects of symptoms on victimization (cf., Kochel, Ladd, & Rudolph, 2012; Leadbeater & Hoglund, 2009; McLaughlin, Hatzenbuehler, & Hilt, 2009; Snyder et al., 2003). Rather, research suggests that both internalizing and externalizing symptoms likely precede peer victimization experiences in childhood and early adolescence (Rudolph et al., 2014; Vaillancourt, Brittain, McDougall, & Duku, 2013).

However, victimization experiences may predict continuities and discontinuities in the experience of symptoms, or links between different types of symptoms. For instance, van Lier and colleagues (2012) demonstrated that peer victimization contributes to the link between youths' externalizing behaviors and their subsequent internalizing symptoms. Children externalizing problems assessed at six years of age predicted internalizing behaviors at 8 years of age by enhancing children's (perceived) peer victimization at 7 years (van Lier et al., 2012).

Moderators of Peer Victimization Effects

Estimates of the proportion of children who are targets of peer aggression are strikingly high (e.g., 75%; Hoover, Oliver, & Hazler, 1992), and victimization episodes are extremely frequent within a school setting (Craig & Pepler, 1997). Of course, it is not expected that significant psychological symptoms may be observed in 75% of youth, however. Rather, it is likely that peer victimization is most strongly associated with psychological symptoms in the context of other risk factors, or of unique peer victimization responses. The study of peer victimization moderators has yielded several interesting effects supporting the notion that only a subset of peer victims are at risk.

Victimization may be most strongly associated with maladjustment in the context of cognitive biases or maladaptive coping. For instance, among children with attributional styles characterized by characterological self-blame, or a tendency to interpret ambiguous experiences as due to self-critical weaknesses, peer victimization is associated with later internalizing symptoms (Graham & Juvonen, 1998; Perren, Ettekal, & Ladd, 2013; Prinstein, Cheah, & Guyer, 2005). Among children with adaptive attributional styles and unbiased social information processing, no significant associations between peer victimization and later adjustment are evident. Youths' coping responses to peer victimization also relates to clinical outcomes. Emotion-focused coping in the context of peer victimization may be especially associated with lower

risk for internalizing symptoms (e.g., Kochenderfer-Ladd, 2004). Evidence indicates that the deleterious effects of peer victimization also are most pronounced among youth with genetic vulnerabilities compared with victimized youth without genetic markers for psychopathology (Benjet, Thompson, & Gotlib, 2010; Brendgen, Boivin, Vitaro, Girard, et al., 2008).

Chronic Versus Sporadic Peer Victims

Unfortunately, the vast majority of longitudinal studies examining peer victimization as a putative risk factor for psychopathology have employed a two-time-point design. These studies often examine peer victimization at an initial time point, and indices of maladjustment at both time points to understand peer victimization as a prospective risk factor for increases in symptoms over time. Thus, a single snapshot of peer victimization is captured, yielding many false positives. For many youth, peer victimization is a transient experience and in fact may be a rite of passage for the majority of children at some point in their development. Data regarding the test–retest reliability of peer victimization assessments yield only moderate consistency over annual time periods indicating that a substantial proportion of children identified as peer victims at one time point may no longer be experiencing significant victimization at all just a few months later (Zwierzynska, Wolke, & Lereya, 2013). For instance, Snyder and colleagues' (2003) playground observations revealed that over 90% of youth were victims of peer aggression at least once during the kindergarten and first-grade years. Overidentifying peer victims (i.e., including many false positives) from a single-time-point snapshot would attenuate apparent associations between peer victimization and later psychopathology.

Moreover, note that recent work on physiological concomitants of interpersonal stress has emphasized the recurrence of interpersonal stressors, rather than single stressful experiences, as relevant for stable physiological changes (e.g., in the stress response systems). Thus, the operationalization of peer victimization as a chronic stressor may provide more overlap with research in related fields.

However, relatively few studies have differentiated chronic versus sporadic victims of peer aggression. Extant data suggest that a small proportion of youth are chronic victims, with high levels of victimization reported over multiple time points (e.g., Hawker & Boulton, 2000). In a recent epidemiological study of young twins (Bowes et al., 2013), approximately 13% of children were identified as being chronic victims of frequent bullying during the transition from primary to secondary school. Notably, children who were chronic victims were found to be at higher risk for poorer mental health and academic outcomes compared with their peers who were never bullied or who were bullied only in primary (age 7–10) or secondary school (age 12). Research utilizing a person-centered approach to examine the developmental course of victimization across childhood and adolescence has been especially useful in this area. For instance, Barker and colleagues (2008) identified three distinct developmental trajectories of peer victimization during preschool years. Between the ages of 3 and 6 years, approximately 4% of children experienced high chronic levels of victimization, 25% were classified as belonging to a moderate/increasing victimization trajectory and 71% to a low/increasing trajectory (Barker et al., 2008). Compared with more sporadic victims, chronic victims are at greater risk for psychopathology, including depressive symptoms and broader internalizing symptoms (Biggs et al., 2010; Nylund, Bellmore, Nishina, & Graham, 2007; Zwierzynska et al., 2013).

Peer Victimization Within a Dynamic Systems Framework

Recent research has examined peer victimization as a factor that contributes to more complex processes. For instance, some research suggests that peer victimization can interact with other intra- and interpersonal domains to predict psychopathology. For example, Schwartz, Lansford, Dodge, Pettit, and Bates (2013) revealed that the association between harsh parental discipline in kindergarten and trajectories of academic achievement over six consecutive years was exacerbated under conditions of peer victimization. Another recent study demonstrated that the interplay between peer victimization and genetic makeup may increase risk for depressive symptoms among children with a history of maltreatment (Banny, Cicchetti, Rogosch, Oshri, & Crick, 2013). Specifically, child maltreatment was positively associated with depressive symptoms via peer victimization (both overt and relational) and these indirect effects were moderated by the children's genotype, with children who carried the long/long variant of the serotonin transporter gene (5-HTTLPR) being at greater risk for depressive symptoms, than children with a short allele.

Peer victimization may be especially relevant to the development of cognitions about the self or others; these cognitions may then be relevant predictors of symptoms. For instance, Troop-Gordon and Ladd (2005) and Ladd and Troop-Gordon (2003) provided some evidence supporting the notion that the effect of peer victimization on children's development of internalizing problems may be mediated by third factors, including self and peer beliefs. Similarly, the prospective effects of peer victimization on

externalizing symptoms likely occur at least in part via third factors, such as hostile attributions (Perren et al., 2013). Other work has suggested that peer victimization may exacerbate certain specific symptom dimensions. For instance, studies of antisocial behavior in youth have become especially focused on an understanding of callous/unemotional traits that appear to be relevant for prolonged, chronic antisocial behavior and psychopathy in adulthood. Callous/unemotional traits can be due to a heritable predisposition, or acquired following negative environmental experiences. A recent study using the Avon Longitudinal Study of Parents and Children dataset demonstrated that peer victimization at the age of 8 years prospectively predicted both internalizing symptoms and callous/unemotional traits at age 13 years via youths' irritability at age 10 years (Barker & Salenkin, 2012). In sum, there is very little research that has examined peer victimization in the broader context of other developmental risks or systems; however, extant work suggests that this may be an important direction for the future.

Psychophysiological Correlates of Peer Victimization

A growing area of research suggests that peer victimization may be best conceptualized as a stressor and consequently may be associated with changes in physiological stress responses. Most studies in this area have examined associations between peer victimization and the HPA axis, whereas relatively little attention has been devoted to the other main stress response system, the sympathetic-adreno medullary (SAM) system. The HPA axis involves central and peripheral system responses to stress. Specifically, the perception of threat or stress leads neurons in the medial parvocellular region of the paraventricular nucleus of hypothalamus to release corticotrophin-releasing hormone (CRH) and arginine vasopressin (AVP), which signals the pituitary gland to release adrenocorticotropic hormone (ACTH) within the body. ACTH in turn crosses the blood–brain barrier and triggers the adrenal gland to release glucocorticoids, which in humans include cortisol.

As noted already, glucocorticoids have a crucial function in regulation gene expression across different cells within the brain as well as throughout the body. Specifically, glucocorticoids can bind to glucocorticoid receptors (GRs; ligand-activated transcription factors), which are located in the cytoplasm of the cells. When glucocorticoids bind to GRs they translocate into the nucleus of the cell, where they activate gene transcription. Moreover, in a negative feedback loop, glucocorticoids inhibit the activity of the anterior pituitary, hypothalamus, and hippocampus, which ultimately down-regulate glucocorticoid release. The effects of glucocorticoids on gene transcription result in a series of physiological outcomes within the body, including down-regulation of immune responses (i.e., pro-inflammatory activity) and the sympathetic nervous system.

In the context of environmental challenges these physiological changes have important adaptive functions and prepare the organism to deal with stressful and potentially threatening stimuli. In other words, HPA axis responses, similar to other physiological responses to stress, represent accommodations to the environmental requests to maintain a homeostatic equilibrium (i.e., allostatic accommodation; Ganzel, Morris, & Wethington, 2010). Yet extreme and sustained stress may lead to *allostatic load*, or the cumulative physiological wear-and-tear that result from the organism's effort to adapt to adverse environmental conditions. Under conditions of high allostatic load, the organism loses its flexibility to respond to the environment, resulting in *fixed automatisms* that can have long-term negative outcomes (see Ganzel et al., 2010). For instance, high chronic levels of cortisol in the body can damage the hippocampus; similarly, dysregulated HPA axis may alter the immune system activity, which ultimately may increase risk for psychopathology.

Research investigating the effects of stressors on HPA axis activity has examined both diurnal cortisol levels and reactive cortisol. Cortisol production typically follows a circadian rhythm over the course of the day. High cortisol levels are evident 30 minutes postawakening and tend to decrease throughout the day reaching the lowest levels about 30 minutes after sleep onset in the evening. Because the HPA axis is a self-regulating system, atypical patterns of cortisol release, resulting from chronic adversity, can lead to longer-term changes in diurnal cortisol production.

Ample research has suggested that both chronically enhanced or blunted diurnal cortisol levels are associated with psychopathology, although this has been examined among adults substantially more than among youth. Relatively little work has examined the link between peer victimization experiences and diurnal cortisol. Extant research consistently indicates that high levels of peer victimization are associated with lower levels of diurnal cortisol (Murray-Close, 2013). For instance, compared with nonvictimized adolescents, victimized adolescents showed a blunted cortisol diurnal pattern that, in turn, was found to associate with higher health problems (e.g., fatigue, stomachache). Thus, the link between peer victimization and health problems was partially mediated by cortisol levels (Knack, Jensen-Campbell, & Baum, 2011).

Reactive cortisol refers to cortisol release in response to acute stressors, and is commonly measured in a laboratory setting after exposing participants to experimentally induced acute stressor (e.g., giving a speech in front of an audience). Given that HPA axis reactions peak approximately 20 minutes after exposure to stress, typically a pretask assessment occurs just before the stress task; subsequently, cortisol is collected one or more times poststressor. Adaptive HPA axis responses typically are observed when participants show moderate cortisol increases in response to a stressor and then return to baseline poststressor, indicating adaptive changes of the body in response to the stress stimuli, and thus the physiological capacity to summon the necessary resources for acute stressors that arise. Conversely, hyper- (exaggerated reactivity) and hypocortisolism (underreactivity to stress) represent dysregulated response patterns that are associated with psychopathology. Most studies examining the effect of peer victimization on HPA axis reactivity to acute stress have demonstrated that higher levels of peer victimization are associated with dysregulated HPA axis reactivity, even after controlling for other known predictors of reactive cortisol response (Calhoun et al., 2014). Ouellet-Morin and colleagues (2011) investigated whether chronic experiences of peer victimization affected cortisol responses to a stress task in a sample of discordant monozygotic twins. They found that twins who experienced chronic victimization showed a blunted cortisol reaction compared with their nonvictimized twins. By using a twin design, this study controlled for genetic and familiar environmental effects, thus providing strong evidence that peer victimization may alter physiological responses to stress. Studies using unselected samples also suggest that repeated peer victimization is associated with allostatic load characterized by hyporeactivity rather than hyperreactivity.

Thus, peer victimization may confer risks for changes in youths' biological stress-response system, perhaps leading to physical or mental health consequences. Recent research also suggests a biological sensitivity model in which the longitudinal association between peer victimization and youths' depressive symptoms is exacerbated among youth with high levels of anticipatory cortisol (i.e., cortisol levels measured before the stress task; Rudolph, Troop-Gordon, & Granger, 2011).

Peer Victimization and Social Pain

Recent work has suggested that social stressors such as exclusion, rejection, or perhaps victimization may lead to physically painful responses. Theory and research suggest that this may be an adaptive response meant to alter subsequent social behavior. This relatively new area of research has important implications for peer victimization research that have not yet been fully realized.

Social scientists have long recognized that human beings have a fundamental motivation to connect to other people, to form and maintain social ties (Baumeister & Leary, 1995). This basic need to belong may have an evolutionary basis. For example, living in groups allowed our ancestors to share tasks (e.g., gathering food), and remain protected from external threats (e.g., predators). Thus, from an evolutionary perspective, experiences of social disconnection, such as social exclusion and rejection, may directly threaten individuals' safety and survival. The negative emotional states arising from social disconnection, also referred to as social pain, may therefore have strong adaptive functions, similar to the ones of physical pain (MacDonald & Leary, 2005). Recent work has revealed an overlap between social and physical pain and has indicated that social pain may share the same physiological mechanisms of physical pain. Two distinct components of physical pain have been identified, including a sensory and an affective component. Whereas the sensory component provides information on the intensity, location, and quality of the pain, the affective component specifically encodes the unpleasant feelings associated with the pain. As a consequence of these negative feelings, the affective component, rather than the sensory one, elicits behaviors aimed to terminate, reduce, or escape the pain. Social pain relies on the same physiological processes of affective pain (MacDonald & Leary, 2005). This would explain why even in the absence of injuries, social experiences could still be perceived as painful. In sum, experiencing pain in response to social disconnection is evolutionary adaptive because it reduces individuals' likelihood to approach social contexts that may potentially elicit social disconnection and because it prepares individuals to properly react when episodes of social disconnections occur.

Extant work has empirically supported the notion that physical and social pain share common biological pathways. Over the last decade, a particularly promising area of research has focused on examining the neural substrates associated with social pain. Specifically, a growing number of studies have employed fMRI to investigate neural activity of individuals exposed to social rejection using a number of different experimental paradigms (for a review, see Eisenberger, 2012). For instance, in the most commonly used paradigm, participants are instructed to take part to a virtual ball-tossing game (i.e., Cyberball). Participants are told to play via the Internet with other two

individuals, which in reality are two computer-simulated players. Participants are exposed to both an inclusion and exclusion condition. In the inclusion condition, they receive the ball from the other virtual players during the entire game session. Conversely, in the exclusion condition, after initially receiving the ball participants are subsequently excluded from the rest of the game session, during which the two virtual players keep on throwing the ball to each other. By comparing neural responses across the two conditions, this work has documented the neural underpinnings of social pain and supported the notion that social pain may be processed by the same neural circuitry of affective physical pain. Specifically, compared with the inclusion condition, social exclusion is associated with higher activation of the dorsal anterior cingulate cortex (dACC) and anterior insula (AI), two brain regions that are involved in processing affective physical pain (e.g., Eisenberger, Lieberman, & Williams, 2003). Moreover, in multiple studies the activation of these regions was found to positively correlate with participants' report of social distress (see Eisenberger, 2012).

Notably, studies revealing the neural substrates of social pain provide crucial information to understand how interpersonal experiences that are perceived as potential threats for social connection may impact psychological health. The brain regions involved in processing social pain, in particular the dACC and the AI, are part of a broader neural network (including also the amygdala and the periaqueductal gray), which is implicated in responding to threating stimuli (Eisenberger & Cole, 2012). That is, the activation of these brain regions gives rise to a number of physiological processes that, evolutionary, prepared individuals to deal with potential threatening external stimuli. Specifically, both the dACC and the AI can elicit the endocrine and autonomic nervous systems. For instance, greater dACC activity has been linked to greater HPA axis and sympathetic nervous system responses (see Eisenberger & Cole, 2012). In turn, in a type of biological cascade, the HPA axis and SNS can regulate immune system activity. Accordingly, a recent study has provided compelling evidence for the association between activity in these regions and the immune system (Slavich, Way, Eisenberger, & Taylor, 2010). Higher dACC and AI responses to an experimental rejection task were found to associate with higher levels of immune system activity after exposure to a different social stress task (as indicated by higher levels of TNF-α, a pro-inflammatory cytokine).

Again, from an evolutionary perspective, the activation of the immune system is adaptive as it prepares the human body to respond to infections and bacteria arising from injuries that potentially are more likely to occur when in social isolation or when facing a threatening situation. However, experiencing chronic episodes that undermine social connection may lead to stable high levels of cortisol and pro-inflammatory cytokines (as a result of immune system activity) in the body, which may have a strong long-term impact on individual physical and mental health. As such, these neural responses may represent the first step of a number of biological processes that potentially may connect the interpersonal context to individual psychopathology.

Clearly, this area of research has direct applications for the study of peer relationships, including peer victimization, and youth development. However, despite promising evidence indicating the neural correlates of social rejection, to date little work specifically has been conducted among youth. Extant studies focusing on social pain inconsistently have replicated adult findings in youth samples. For example, experimentally induced social rejection elicited increased activity of the AI in youth (e.g., Moor et al., 2012). Yet evidence supporting the role of the dACC is somewhat less consistent (e.g., Masten et al., 2009). In addition, the subgenual anterior cingulate (subACC), a brain region that did not emerge to be salient among adults, is implicated in a number of youth studies (e.g., Masten et al., 2009; see also Eisenberger, 2012). The subACC is not directly involved in the affective circuitry of physical pain, yet it is implicated in processing affective states. Interestingly, enhanced activity in the subACC has been associated with higher levels of depressive symptoms in clinical samples. Moreover, higher subACC activation in response to a social rejection task was prospectively associated with youth depressive symptoms over a one-year period (Masten et al., 2011). Future research examining neural correlates of social pain among youth is strongly needed to corroborate existing findings, underscore potential developmental differences and better elucidate how brain neurocognitive processes linked to negative peer experiences may contribute to the development of psychological symptoms.

In sum, research on peer victimization has been prolific in recent years, and there are numerous societal forces (e.g., news media, parent advocacy groups, school district and legal systems) promoting anti-bullying efforts. Research has lagged behind a bit, however. Associations between peer victimization and psychopathology are less straightforward than previously thought. Peer victimization clearly is a relevant construct within complex, dynamic systems models of development. However, main effects of peer victimization on adjustment are somewhat less obvious within

the research literature. Greater attention to chronic peer victimization experiences, and the role of peer victimization within the development of youths' psychophysiological stress responses are needed in future work.

Friendship

As children reach the school-age years, many have achieved the capacity to form stable, true friendships with peers. Within the psychological literature (but notably differently from within sociological or anthropological studies), friendships often are described as relationships that are voluntary, mutual, and horizontal/bilateral (i.e., among peers of equal power; Bagwell & Schmdit, 2011). Compared with interactions with other (i.e., nonfriend) peers, interactions with friends are more frequent, are characterized by greater positive affect, and involve more reciprocal play (Newcomb & Bagwell, 1995). Some of these qualities are evident even within toddler-age youth; however, it is unclear whether young children are simply responding favorably to familiar peers or whether true friendships have emerged (Furman, 1982). Friendships among toddler and preschool years also are more strongly dictated by parental choices than are friendships developing during the school-age years. By school age, children are able to engage in reciprocal communication patterns (i.e., sharing and clarifying feelings, sharing goals for play, conflict resolution) that are thought to be a cornerstone of friendship formation and maintenance (Gottman, 1983). As children mature through the school-age years, friendships involve increasing levels of loyalty, affection, and companionship. In preadolescence, friendships evolve further to include emotionally intimate exchanges, self-disclosure, mutual support, and a general ability to understand and meet each other's psychological needs (Laursen & Hartup, 2002).

Importantly, children's friendship experiences appear to be fairly distinct from their overall experiences within the larger peer group. Although peer rejected children typically have fewer friends than nonrejected youth, and are more likely than others to befriend other rejected youth, they are equally likely to report at least one very best friendship that includes an array of positive and negative friendship qualities at levels sometimes comparable to other children's friendships (e.g., Parker & Asher, 1993). Thus, within the peer relations literature, great efforts have been made to differentiate children's group level peer experiences (e.g., peer status and reputations, peer victimization), from their dyadic peer experiences (e.g., friendships), as distinct aspects of peer functioning with unique correlates.

It is surprisingly difficult to measure the number of children's friends, perhaps because no formal definition exists for distinguishing friendships of different levels (i.e., best friends, close friends, or friends) or to identify friends from other amicable relationships (acquaintances or peers that children hang out with). Most commonly, children are asked to identify an unlimited number of their friends or their closest friends from a roster of peers. Demand characteristics are high for such a task, and youth are highly reluctant to list fewer friends than they believe is considered appropriate by the experimenters. An unlimited nomination procedure yields a very large range of the number of friends children will report, further obfuscating the definition of friendship. The common use of the Internet, and social network sites such as Facebook in particular, have only further obscured the meaning of the word *friend* within our society. Contemporary research combines several approaches by asking youth to nominate an unlimited number of their closest friends and also to identify their top three friends from this larger list. This approach allows for at least an ordinal clarification between youth who are identified as best friends versus other close friends. However, the use of school- or grade-wide rosters for this task often presents limitations in identifying friends from across multiple contexts.

In many studies, investigators determine whether children's friendship nominations have been reciprocated in an effort to identify true friendships that meet most investigators' a priori definitions of close friendship. Reciprocity typically is only possible to determine if children's selected friend also is a consented participant and has the opportunity to complete a similar nomination procedure. Reciprocal nominations may reveal that a target child's very best friend agrees that the target is a very best friend (i.e., ranked as their #1 friend), a close friend (i.e., ranked within their top three friends), a friend (selected as a friend, but not among their top three closest friends), or not a friend at all. Thus, some advocate a continuous rather than a dichotomous approach for studying friendships (see Berndt & McCandless, 2009). Data indicate that approximately 50% of children's very best friend nominations are reciprocated as very best friends (Parker & Asher, 1993). Approximately 75–80% of youth have at least one reciprocated friendship (Parker & Asher, 1993). Reciprocated friendships indeed may be characterized by higher levels of friendship quality and are more likely to be stable than other friendships (but see Poulin & Chan, 2010). Children who do not have a reciprocated best friend are more likely to be rejected by peers and are more likely to be victims of peer aggression (Hodges, Boivin, Vitaro, & Bukowski, 1999). Friendless children also have higher levels of depression than children with friends (Pedersen et al., 2007). Compared with

children with nondepressed friends, friendless children show increasing trajectories of depression over time (Brendgen, Lamarche, Wanner, & Vitaro, 2010).

Some children are less likely to have stable friendships, suggesting possible reciprocal associations between psychopathology, the peer environment, and subsequent psychological reactions to changes in this environment. In the context of psychological symptoms, the termination of a friendship may deprive youth of needed social support or a context in which more adaptive skill development may occur.

Yet relatively little empirical attention has been dedicated to examining which factors may contribute to friendship (in)stability over time (Poulin & Chan, 2010). Three consistent predictors have been revealed in extant research. First, aggressive and antisocial youth tend to have difficulties in maintaining their friendship over time. For instance, early adolescents who engage in high levels of overt aggression are less likely than their nonaggressive peers to have long-lasting friendships (Ellis & Zarbatany, 2007a). Second, youth with internalizing symptoms and socially withdrawn youth tend to have shorter friendships. Interpersonal models of depression (Coyne, 1976a, 1976b; Joiner & Katz, 1999), suggest that depressed individuals engage in a variety of socially alienating behaviors that increase their experience of interpersonal stress and also contribute to truncated friendships (e.g., Prinstein, Boreli, Cheah, Simon, & Aikins, 2005). There also is evidence that compared with other youth, anxious-solitary preadolescents are less likely to remain in a stable best friendship through an entire academic year (Ladd, Kochenderfer-Ladd, Eggum, Kochel, & McConnell, 2011).

Last, data indicate that similarity between youth and their friends may contribute to friendship stability. In other words, friendships between similar youth (i.e., similar levels of self-esteem, aggression, substance use) tend to last longer (Hafen, Laursen, Burk, Kerr, & Stattin, 2011). Using stochastic actor-based modeling (Snijders, van de Bunt, & Steglich, 2010) recent work has suggested that similar levels of depressive symptoms, happiness, and substance use also predict friendship maintenance over time, at least at the level of a unilateral friendship (DeLay, Laursen, Kiuru, Nurmi, & Salmela-Aro, 2013; van Workum, Scholte, Cillessen, Lodder, & Giletta, 2013; Van Zalk, Kerr, Branje, Stattin, & Meeus, 2010).

Initial research on friendships focused on the psychological correlates of friendship versus friendlessness and on factors that may help to explain why some children are able to maintain friendships. More research attention has concerned the study of friendship qualities and to some extent examinations of the characteristics of children's friends (Hartup, 1996).

Friendship Quality

Children or their friends easily can report their relationship quality with specific friends using a variety of available checklists measuring friendship quality (e.g., Furman, 1998). Children can report their own friendship quality, data from children's friends can be used to jointly determine friendship quality of a dyad, or, rarely, friendship quality has been examined using observational methods. Interestingly, rejected children report higher levels of positive friendship qualities than their average-status friends, highlighting a possible social-cognitive bias that may compromise the validity of self-reported friendship quality among some youth (Brendgen, Little, & Krappmann, 2000).

Most research has identified a group of positive friendship qualities (e.g., companionship, emotional disclosure, affection, reliable alliance, intimacy, support) and negative friendship qualities (i.e., conflict, dominance, power assertion, criticism). Positive and negative friendship qualities are orthogonal constructs, rather than two ends of a single relationship quality continuum. Research consistently has documented gender differences in some positive friendship qualities (e.g., emotional disclosure, intimacy) but not all (e.g., companionship). Moreover, there is some evidence that specific behaviors (e.g., shared social interactions) foster intimacy among males more than among females, while emotional disclosure may promote intimacy among females more than males (Hussong, 2000). Gender differences in negative friendship qualities are not reported consistently.

Interestingly, recent work using quantitative twin-study genetic designs indicates that genetic factors may explain a substantial proportion of variability in positive friendship quality. In other words, there is greater similarity among monozygotic twins than among dizygotic twins in the levels of positive friendship quality with best friends (Brendgen, 2012; Pike & Atzaba-Poria, 2003). Genetically determined factors, such as temperament may partially explain how genetic factors influence friendship quality (Pike & Atzaba-Poria, 2003). Results suggest an interesting example of combined rGE and GxE effects related to the role of friendships in children's adjustment. Consistent with a transactional framework, biological (i.e., genetic factors) covary with, influence the selection of, or evoke particular social environments (i.e., friends and friendship quality). These friendship experiences within the social environment then affect an individual's biological systems, altering trajectories toward psychopathology. Specifically, correlated genetic environmental factors (rGE effects)

may influence the ability to make friends (e.g., phenotypes related to appearance, behavioral inhibition, or social skill may predict the ability to make friends) and the level of friendship quality within these friendships. In the context of adverse events, these relationships may buffer maladaptive biological (e.g., autonomic nervous system) stress responses that promote maladaptive behavioral responses, or mitigate the risks associated with other genetic risks for psychopathology.

Harry Stack Sullivan (1953) famously posited that friendships provide a context that facilitates several critical development tasks at various different phases of life. For instance, at entry to the formal schooling years, friendships offer a context for social skill (e.g., cooperation, conflict resolution) development; beginning in preadolescence, friendships offer opportunities to share emotional intimacy, to give and receive social support, and to validate an emerging sense of identity (Sullivan, 1953). Friendships offer opportunities to learn and practice perspective-taking skills, empathy, and altruism (Buhrmester & Furman, 1986). Piaget (1932) and Vygotsky (1981) emphasized the unique context afforded by peer–peer interactions compared with adult–child interactions for promoting cognitive development. In short, many theories have suggested that friendships are highly relevant for understanding adolescent development, and by extension, may be a relevant factor in understanding adjustment difficulties.

Yet at least within the developmental psychology literature, little empirical evidence supports the claim that friendship difficulties confer risk for psychopathology. Most consistent has been support for links between friendship and loneliness. Specifically, research suggests that the absence of a close friend, fewer numbers of close friends, and lower levels of positive friendship quality all are associated with higher reports of loneliness (Bukowski, Hoza, & Boivin, 1993). Some evidence similarly suggests that low levels of positive friendship qualities also are associated at least concurrently with lower self-concept and higher levels of anxiety or depression (Bagwell & Schmidt, 2011). High levels of negative friendship qualities similarly are associated with anxiety symptoms (La Greca & Harrison, 2005). Longitudinal main effects between friendship or friendship quality and later symptoms of psychopathology have been inconsistent, however (Bagwell & Schmidt, 2011). On notable exception has been work examining the role of friendship experiences on school adjustment. For instance, Ladd and colleagues report that higher numbers of close friends and an absence of conflictual friendships are associated longitudinally with better school adjustment (i.e., more positive attitudes to kindergarten transition) among young children (Ladd, Kochenderfer, & Coleman, 1996).

Far more consistent are findings that indicate that both the presence of close friends and of positive friendship qualities serve as moderators in the association between known adverse risk factors and later adjustment difficulties. This is consistent with several theories, and even results from recent neuroscience studies. A large body of research has supported the buffering effects of social support on psychoneuroimmunological functioning (Cohen & Wills, 1985; for a review, see Hostinar, Sullivan, & Gunnar, 2014). Neuroscience research similarly suggests unique benefits of close relationships. For instance, recent research examined fMRI scans of three groups of adult women experiencing anticipatory anxiety (i.e., receiving random electrical shocks; Coan, Schaefer, & Davidson, 2006). In one group, participants held the hand of their romantic partner, one group held the hand of a stranger, and a third group did not hold anyone's hand during the experiment. Compared with the other two groups, women holding their partner's hand had significantly lower levels of brain activation in areas implicated in threat–response (e.g., ventral anterior cingulate cortex), emotional regulation (i.e., right dorsolateral prefrontal cortex, caudate), and emotion-related homeostatic functions (i.e., superior colliculus; Coan et al., 2006). Moreover, among participants with higher levels of partner relationship quality, neural responses were even more dramatic (Coan et al., 2006).

Friendships similarly may provide buffering effects in the context of stress. Friends and positive friendship qualities may provide an outlet for emotional disclosure and comfort that promotes adaptive coping with negative life events. Alternatively, friendships may provide a corrective context to redirect developmental trajectories that had been distorted due to stressors in other life domains (Bagwell & Schmidt, 2011). For instance, research suggests that the presence of a close friend, and high levels of positive friendship quality each appear to buffer the deleterious effects of negative parenting and family dysfunction on both internalizing and externalizing outcomes (Bukowski, Motzoi, & Meyer, 2009). Close support within friendship also appears to moderate the association between peer victimization and emotional maladjustment (Hodges et al., 1999). Friendships also buffer the negative effects of natural disasters, chronic illness, and peer rejection; maladaptation following each type of stressor is lower among youth with close, supportive friendships compared with youth without high quality friendships (e.g., Laursen, Bukowski, Aunola, & Nurmi, 2007; Wasserstein & La Greca, 1996).

Recent evidence suggests that friendship also may moderate biological risks and physiological stress reactions. For instance, Masten, Telzer, Fuligni, Lieberman, and Eisenberger (2012) indicate that time spent with friends during adolescence predicts lower brain activity in the dACC and AI in response to an experimentally induced stress task two years later. Notably, these brain regions are associated with processing of negative affect and pain, suggesting that friendship participation may have longitudinal effects on the ability to cope with negative affect and on reduced experience of emotional distress (Masten et al., 2012). Brendgen and colleagues' (2013) examination of a twin-sample indicates that participation in a single reciprocated close friendship mitigates the genetic risk for depressive symptoms among girls (Brendgen et al., 2013). Regarding physiological stress responses, research has examined friendship qualities as moderators of children's HPA axis response to naturalistic or in vivo lab-based stressors. For instance, Adams, Santo, and Bukowski (2011) indicated that the presence of a close friend during daily stressors was associated with lower levels of cortisol secretion. Similarly, in another study peer exclusion was associated with HPA axis dysregulation, as indicated by flatter diurnal cortisol, especially among children with fewer friends and lower quality friendships (Peters, Riksen-Walraven, Cillessen, & de Weerth, 2011). Calhoun and colleagues (2014) demonstrated that higher levels of positive interactions with a best friend following an experimentally induced social stressor were associated significantly with more dramatic HPA axis recovery.

In sum, while some children are friendless and at greater risk for internalizing distress, a majority of children successfully form and maintain reciprocal and supportive friendships. The ubiquity of this experience may explain relatively weak main effects between friendship quality and indices of psychopathology. However, friendships provide a context for social support or re-routed developmental competence trajectories that appear consistently to offer stress-buffering effects. Thus, friendships are promoted as adaptive and even necessary experiences among youth. Interventions aimed at increasing children's ability to form and maintain stable, close friendships have been established, and professionals advocate that youth be given opportunities to develop voluntary relationships with their peers.

Characteristics of Friends

Peer interactions establish a context in which development occurs. The characteristics of one's friends potentially have a substantial effect on development and on youths' (mal)adaptive trajectories, therefore. Consequently, researchers have been especially interested in the factors that predict which peers a child may befriend. Results suggest that children's risks for psychopathology actively contribute to their selection into social contexts that may reinforce their own psychopathology. Thus, friendship can serve as maintenance or exacerbating factor for youth at risk.

Research findings frequently have supported a proximity–similarity model of friendship formation (Blass & Schwarcz, 1982). Not surprisingly, spatial proximity predicts friendship formation. Although findings may change as Internet-based friendships continue to expand, extant data currently indicate that children living closely together within neighborhoods or sharing the same context (i.e., classroom, school) have an increased probability of becoming friends compared with children at greater physical distance from one another (Preciado, Snijders, Burk, Stattin, & Kerr, 2012). Thus, factors that covary with neighborhood or school selection (e.g., SES) may be likely to be experienced at similar levels among friends.

Many children also are remarkably likely to select friends who are similar to themselves. Younger youth are most likely to choose friends who have similar observable characteristics to themselves (e.g., gender, attractiveness), while older youth select friends with similar attitudes, values, popularity, and behavioral preferences (i.e., homophilic selection; Kandel, 1978). Most relevant is a tendency for individuals with psychopathology to select friends with similar types of psychopathology. For instance, adolescents who engage in delinquent behaviors are more likely to select as friends peers who similarly engage in delinquent behaviors, and youth with depressive symptoms are more likely to select other depressed youth as friends (Burk, Steglich, & Snijders, 2007; Van Zalk et al., 2010).

Research examining homophilic selection largely has built on the idea that adolescents are more likely to befriend similar peers because interpersonal attraction is higher among individuals with similar characteristics. For example, the similarity–attraction hypothesis (Byrne & Griffith, 1973) posits that individuals may evaluate positive interactions with similar others as more favorable because they represent rewarding contexts by validating their own behaviors. Yet more recently it has been argued that selection based on similarity may not always be a matter of choice. Indeed, youth experiencing psychological maladjustment may wish to befriend well-adjusted peers, but because they may experience more difficulties in forming healthy friendships, they may become friends with other isolated, troubled youth. This selection process, referred to as *default selection*, has been demonstrated in the study of

deviant peer affiliation. Importantly, youth with a variety of social deficits may become friends by default, exposing each to a range of psychopathology among their friends that could prove deleterious to their own adjustment.

Parents also play a role in the types of friends that their children select. Among young children, parents explicitly select their children's playmates and provide monitoring and supervision of their children's interactions with friends (Parke & Ladd, 1992). Not surprisingly, mothers' own level of social skill and the size of mothers' social networks are associated significantly with the number of their children's friends (Homel, Burns, & Goodnow, 1987). Later in development, parenting behavior is a relevant predictor of the characteristics of their children's friends. Most frequently examined have been the predictors of youths' entry into deviant peer groups. Findings indicate that parental communication, monitoring, and prohibition predict youths' affiliation with deviant peers. However, effects are somewhat inconsistent. Some research suggests that high levels of parental monitoring are associated with lower levels of children's deviant peer affiliation (e.g., Dishion, Poulin, & Skaggs, 2000). Yet other research suggests that prohibitive parenting may enhance children's affiliation with deviant peers (e.g., Keijsers et al., 2012). Parents' sensitivity to children's desire for autonomy is likely an important consideration. Parenting practices that support youth autonomy (i.e., autonomy-supportive styles) may have the desired effects in reducing negative peer affiliations, yet parental practices that tend to deny youth autonomy (and are accompanied by a controlling style) may increase the probability of deviant peer affiliation (Soenens, Vansteenkiste, & Niemiec, 2009). An interesting recent study employing social network analyses offers supporting evidence for this hypothesis (Tilton-Weaver, Burk, Kerr, & Stattin, 2013). Using network data, including friends' own reports of their deviant behavior, results indicated that high parental rules reduced the probability that children would select deviant peers as friends only when accompanied by low (perceived) parental overcontrol. Conversely, high parental rules had the opposite effects among adolescents who felt overcontrolled.

Research examining gene–environment interplay (i.e., passive, selective, and evocative rGE; Rutter, Moffitt, & Caspi, 2006) suggests that genetic factors also may play a significant role by influencing the social environment that children select (Brendgen, 2012). In other words, some genetic factors may help determine who youth select as friends. Data mostly support selective rGE effects in relation to deviant friendship affiliation. Quantitative genetic studies using twin-designs indicate that 20–42% of variance in deviant friendship selection (i.e., deviance reported by adolescents or parents, rather than by friends themselves) may be explained by genetic factors (Brendgen, 2012). Using Add Health data, allowing for the measurement of friendship reciprocity and friend-reported risk behavior, genetic factors accounted for over 60% of variance in youths' tendencies to select similarly substance-using friends (Harden, Hill, Turkheimer, & Emery, 2008). The Add Health data set has revealed similar genetic effects on children's selection of friends with similar levels of intelligence, depression, aggression, and academic achievement (Brendgen, 2012; Guo, 2006). However, these selective rGE effects appear to be greatest among adolescents, and less so among younger children, perhaps reflecting the increasing autonomy youth have in selecting their friends as they mature (Brendgen, 2012).

Molecular genetic studies also offer some evidence for genetic effects influencing friendship selection. Results from the Add Health study suggested an association between the number of risk alleles (i.e., 10-repeat alleles) of the dopamine transporter (DAT1) gene (i.e., associated with less inhibitory control) and the number of substance using friends among male youth who also came from high-conflict family backgrounds (Beaver, Wright, & Delisi, 2008). Social network studies (e.g., Fowler, Settle, & Christakis, 2011) have further suggested that individuals may select friends who are genetically similar (the frequency of the A1 allele of DRD2, associated with alienation and antisocial behavior) or who possess complementary traits; however, evidence has been inconsistent (Brendgen, 2012).

The Dark Side of Friendships

Ironically, it appears that some friendships or behaviors exhibited within the context of a friendship actually may do more harm than good. In addition to the many adaptive opportunities afforded by this context, friendships also can be a crucible for the development of harsh, maladaptive, and even dangerous behavior. Much of this dark side of friendship pertains to the characteristics of the friends that are selected by youth and the potential for peer socialization among these friends; this will be discussed in more detail later. In addition, recent work has identified numerous interpersonal processes that are unique to the friendship context and appear to be important risk factors for psychopathology. Some of these ideas have been articulated within theories of adult psychopathology, but rarely have been applied to youth.

Interpersonal models of depressive symptoms, for example, posit that individuals with high levels of depressive symptoms are especially likely to experience interpersonal stressors. Moreover, depressed individuals

are prone toward dependent interpersonal stressors, in which their own behavior may partially contribute to the stressful event (Rudolph et al., 2000). Indeed, depressed individuals appear more likely than nondepressed people to evoke more sad affect from conversation partners, and to engage in a variety of interpersonal behaviors that contribute to interpersonal rejection and exacerbations of depression (i.e., frequently referred to as a downward spiral; Coyne, 1976a, 1976b).

Similar phenomena have been observed among youth and help to elucidate friendship behaviors that are associated with deleterious outcomes. For example, data indicate that youth with sad affect and histories of peer rejection are more likely than others to engage in excessive reassurance-seeking behavior (Prinstein, Borelli et al., 2005). Reassurance seeking typically involves requests from close others (e.g., friends) to reinforce feelings of positive self-worth or of relationship stability. However, friends' compliance with these requests often is not sufficient; a reassurance seeker may continue to make repeated requests, followed by repeated disbelief in the reassurance received (Coyne, 1976a, 1976b). Eventually, and ironically, close friends from whom excessive reassurance is sought experience declines in their perceptions of positive friendship quality, threatening friendship stability (Prinstein, Boreli, et al., 2005). Interpersonal rejection from friends is perceived as a reification of the reassurance seeker's initial doubts, contributing to increased depressive symptoms.

A second interpersonal phenomenon relevant to depressive symptoms similarly highlights specific friendship behaviors that ironically lead to increases, rather than decreases in maladjustment. Rose (2002) initially noted that close friendship discussions involving excessive self-disclosure and problem discussion with a focus on excessive speculation and sharing of negative feelings, but without a problem-solving orientation (i.e., co-rumination) appeared to simultaneously predict both positive and negative outcomes among youth. Due to emphasis on self-disclosure and emotional validation during co-rumination, this interaction style is associated with high levels of friendship intimacy. It also appears to be more common among females than males. However, co-rumination also is associated with higher levels of depression and anxiety symptoms, particularly among girls (Rose, Carlson, & Waller, 2007). Additional studies of co-rumination have demonstrated that co-rumination varies among youth with different depression histories (Stone, Uhrlass & Gibb, 2010) and also is associated longitudinally with later major depressive episodes as well as the length of severity of these episodes (Stone, Hankin, Gibb, & Abela, 2011). Hankin, Stone, and Wright (2010) further revealed cumulative and cascading effects among co-rumination, interpersonal stressors, and trajectories of internalizing symptoms, offering compelling support for a cycle of mutually reinforcing transactions between children's psychological states and their dyadic experiences.

In sum, research suggests that the vast majority of youth have close friends characterized by positive relationship qualities. Children tend to befriend youth who are similar to themselves in behavioral characteristics and even psychological symptoms. Friends provide a context for support and assistance in times of distress. Consistent buffering effects of friendship have been observed, mitigating the psychological and physiological effects of adverse events. Friendships may even serve protective functions against the development of psychopathology among those at heightened genetic risk. However, friendships also provide a context for rehearsal and reinforcement of maladaptive interpersonal behaviors that increase the risk for psychopathology.

ADOLESCENCE

The transition to adolescence is a critical developmental vulnerability period for a range of psychological symptoms, including conduct disorder, depression, eating disorders, health risk behaviors to name only a few. Adolescence also is a developmental period characterized by marked changes in peer relationships. Adolescents spend considerably more time with their peers in the absence of adult supervision, and these peer interactions are characterized by much greater sophistication, intensity, and self-scrutiny.

Many of the constructs discussed earlier in this chapter remain relevant in adolescence. As in earlier years, adolescents vary in their overall peer reputations and levels of likeability. As noted already, peer popularity becomes especially salient as a discrete construct at the adolescent transition, however, with broad implications for adolescents' social goals and opportunities for social interactions. Although empirical data are not yet available, some evidence suggests that peer status may become somewhat less salient by late adolescence, particularly as youth reach the completion of their secondary education. Peer victimization most certainly continues during adolescence, however, social/relational forms become increasingly common. Compared with the relatively benign consequences to social/relational aggressors, the legal sanctions associated with physical forms of bullying become especially threatening, likely contributing to its decrease in frequency. Friendship behaviors become remarkably sophisticated

during adolescence; relationships are increasingly defined by high levels of emotional disclosure, loyalty, and social boundaries. Cross-gender friendship emerges for many at the adolescent transition, allowing for the development of heterosexual romantic interactions for many youth. Adolescence also is characterized by several informal peer groupings that define the peer milieu at this developmental stage. Interconnected networks of friends form peer cliques (i.e., interaction-based groups) with unique identities, rules, and expectations. Reputation-based peer crowds also form (e.g., jocks, brains, burnouts), particularly in North America, highlighting differences in adolescents' adoption of peer- or adult-prescribed values as they progress through identity development (Brown, 1990). Notably, these peer crowds vary considerably in their members' engagement in a variety of significant health risk behaviors (La Greca, Prinstein, & Fetter, 2001).

It frequently has been suggested that adolescence is a period marked by heightened susceptibility to peer influence; however, compared with the emphasis on the previously discussed peer relations constructs, less attention has been dedicated toward the study of peer influence. Peer influence effects are relevant to a wide range of psychological domains, including many different psychological symptoms. A brief review of this research is now offered.

Peer Influence

It is surprising that peer influence has been a somewhat understudied phenomenon among peer relations researchers. The apparent strong associations between adolescents' attitudes/behaviors and the attitudes/behaviors of their peers are among the most consistent and potent effects in all of developmental psychopathology research. Peer influence effects also have broad implications for many sectors of society. Among psychologists, there has been interest in peer influence as a normative phenomenon (among developmental psychologists), to illustrate persuasion and influence theories (among social psychologists), and to understand risk for psychopathology (among clinical psychologists). In fact, recent peer influence findings have indicated that the aggregation of exclusively deviant peers within treatment or recreational settings sometimes can produce iatrogenic treatment outcomes (see Dishion & Andrews, 1995; McCord, 1992). Sociologists and public health investigators have a strong investment in understanding peer influence dynamics to construct models of communication and contagion. Economists examine adolescent peer influence as a multiplier of policy interventions designed to reduce health risk behavior. Criminologists have noted that adolescents are more likely to commit crimes in the presence of their peers, while adults are more likely to commit the same crimes while alone (Gardner & Steinberg, 2005). Research also has documented peer contagion of criminal activities among prison inmates (Bayer, Hjalmarsson, & Pozen, 2009). Educators and education policymakers have begun to reflect on peer influence theories to determine students' placements into academically tracked classrooms or detention centers that exclusively include other deviant youth (Vigdor, 2008). Political scientists understand changes in public opinion using peer influence dynamics, just as advertising executives' marketing strategies are based quite explicitly on many of the basic principles that underlie peer influence effects. In short, peer influence effects are relevant for many, but remarkably little empirical attention has been dedicated to some basic, fundamental questions regarding these strikingly prevalent phenomena.

Adolescence likely is a period of heightened vulnerability to peer influence effects. This assertion is supported by developmental theory and findings from social neuroscience (Gardner & Steinberg, 2005). As noted earlier, data indicate that, compared with childhood, adolescence is accompanied by substantial increases in the frequency of adolescents' interactions with peers and concomitant decreases in parental monitoring. Changes in adolescents' social context yield increased opportunities to interact with peers autonomously, develop more sophisticated interpersonal behaviors, and experiment with new social roles. For instance, adolescents' peer relationships are characterized by higher levels of intimacy and emotional disclosure than in childhood, and begin to invest in peers as primary sources of social and emotional support (Harter, Stocker, & Robinson, 1996). In addition, adolescents develop relationships with broader social networks than in childhood and become increasingly cognizant of their status within the overall peer group hierarchy.

Developmental changes in youths' ability to form a stable sense of self-concept, contemplate the future, and engage in more sophisticated perspective-taking skills facilitate a unique period of identity development in adolescence. Identity development occurs in the context of adolescents' new social experiences. Through reflected appraisal and social comparison processes, peers offer an especially salient source of feedback and an opportunity for self-other contrast as adolescents experiment with their own values, preferences, and developing self-concept. Data suggest that, compared with younger children, adolescents become especially attuned to the peer context when making behavioral decisions. By early adolescence, youths' motivation to engage in behaviors that may earn

favorable status among peers usurps their desire to engage in behaviors primarily rewarded by adults (Juvonen & Murdock, 1995).

Adolescence also is a period of substantial changes in brain development, including rapid neuronal myelination and neural pruning, allowing for more rapid processing between brain regions (Giedd, et al., 1999). However, areas of the brain develop at different rates and maturation periods (Dahl, 2004). Research suggests that limbic and paralimbic regions of the brain, responsible for affectively motivated and reward-oriented behaviors, develop more quickly than the prefrontal cortex, responsible for rational decision making, contemplation, and inhibition (i.e., a dual systems model; e.g., Dahl, 2004). Indeed, studies using fMRI indicate that the nucleus accumbens (within the limbic system, responsible for processing rewards) is more likely to be activated during a monetary reward task among adolescents compared with younger children or adults (Galvan et al., 2006). Adolescence also involves increased circulation of sex hormones that bind to receptors in the limbic system, also contributing to heightened sensation-seeking. Decreases in dopamine production in adolescence lead to lower sensations of pleasure, and possibly adolescents' engagement in more extreme behaviors to reach similar levels of reward.

While changes in the limbic system occur early in adolescence, the development of the prefrontal cortex lags behind, with ongoing myelination, synaptic pruning, and greater integration with other brain regions continuing into early adulthood (Dahl, 2004). Consequently, adolescents' motivations toward reward-oriented behavior (i.e., risk behaviors) easily overpower logical reasons that may inhibit them to behave riskily. Data indicate that adolescents are less likely than adults to show activated neural responses in the medial prefrontal cortex when processing the loss of a reward, suggesting that adults are more likely to avoid harm or punishment (Ernst et al., 2005). Note that research clearly indicates that adolescents and adults are equally capable of recognizing the risks involved with specific behaviors, but the conscious awareness of these risks influences behavior less than the perception of immediate rewards. Thus, adolescents' decisions to engage in risk behaviors are likely influenced by social and affective factors and not by cognitive limitations in understanding risk (Cauffman et al., 2010).

Adolescence is associated with greater attention to social cues, rewards, and sanctions across many species—not only in humans. Gonadal hormone production during puberty is associated with increased oxytocin receptors in the amygdala and striatum, promoting increased attention to social stimuli (Albert, Chein, & Steinberg, 2013). Adolescents are especially attuned to social cues regarding inclusion/exclusion and social evaluation and are more invested in mentalizing, or thinking, about their peers' perspectives on their own behavior (Somerville, 2013). Clearly, these psychological and biological changes provide a perfect storm to increase susceptibility to peer influences in adolescence.

Indeed, recent data indicate that the mere presence of peers strongly influences adolescents' (but not adults') decisions to engage in risk behaviors and their biological responses to these behaviors. Gardner and Steinberg (2005) demonstrated that adolescents, but not children or adults were more likely to engage in risky behavior on a computer analog task when peers were present, compared with when alone. O'Brien, Albert, Chein, and Steinberg (2011) reported that late adolescents were more likely to pursue immediate rewards (as opposed to delayed rewards) when with peers, than when alone. Notably, these peer effects are significant even when adolescents believed they were being observed by an unknown peer (Weigard, Chein, Albert, Smith, & Steinberg, 2014), suggesting that peer influences on reward sensitivity may not necessarily be attributable to the familiarity of observing peers. Chein, Albert, O'Brien, Uckert, and Steinberg (2011) revealed that during a computerized risk-taking task the presence of peers led to increased activation in the limbic reward-processing systems among adolescents, but not adults. Steinberg and colleagues (Logue, Chein, Gould, Holliday, & Steinberg, 2014) revealed that even adolescent mice, but not adults, are more likely to consume alcohol in the presence of peers than when alone.

However, it should be acknowledged that recent evidence contradicting the dual-systems models also has been reported (see Crone & Dahl, 2012; Pfeifer & Allen, 2012). For example, studies have shown that tasks that require cognitive control are associated with both age-related increases and decreases in the activation of the prefrontal cortex. Moreover, other work has documented that activation of the limbic regions (e.g., ventral striatum) are associated with positive outcomes in adolescence (e.g., higher resistance to peer influence) rather than maladjustment. Drawing on these findings, researchers have begun to question the validity of the dual-systems models, suggesting that more complex developmental processes may characterize brain development throughout adolescence. For instance, researchers have suggested that brain regions involved in processing social motivation as well developmental and physiological changes associated with

puberty should be taken into account to better understand the behaviors associated with brain development during adolescence (Crone & Dahl, 2012; Pfeifer & Allen, 2012).

Although the specific mechanisms responsible for potentially higher peer influence susceptibility remain a topic of debate, most agree that adolescence is a period in which youth are especially likely to engage in behaviors at levels similar to that of their peers. Associations between adolescents' and their peers' behavior are due to two tenets of *homophily* (Kandel, 1978). *Selection* effects refer to the tendency for individuals to befriend individuals who possess similar attitudes and behaviors. *Socialization* effects suggest that individuals' behavior is influenced by the behavior of others. There is strong support for both selection and socialization effects among adolescents. Moreover, there is growing support to suggest that homophily effects are relevant to a broad array of adjustment indices. Perhaps most frequently studied, data indicate that adolescents befriend others who engage in levels of aggressive and substance use behavior similar to their own. Moreover, substantial data suggest that adolescents' perceptions of their friends' engagement in aggressive and substance use behavior is among the strongest longitudinal predictors of adolescents' own engagement in similar behavior (Prinstein & Dodge, 2008). Peer socialization effects have been revealed for adolescents' aggressive and delinquent/illegal behavior, alcohol, cigarette, marijuana use, and engagement in related health risk behaviors (e.g., sexual risk behavior; Brechwald & Prinstein, 2011). More recently, research has suggested that peer socialization effects also may be relevant for understanding adolescents' depressive symptoms (Giletta et al., 2011) weight-related behavior and body dissatisfaction (Rancourt et al., 2013), engagement in nonsuicidal self-injury (Prinstein et al., 2010), and happiness (Fowler & Christakis, 2008; van Workum et al., 2013). Research examining the presence of selection or socialization effects consistently suggests that peer experiences are among the most reliable predictors of adolescents' own mal/adaptation.

Peer Influence Contexts

Despite the seeming ubiquity of peer influence effects, remarkably little research has been conducted on several fundamental issues that are necessary for better understanding, and ultimately preventing deleterious peer influence. For instance, at present little is known regarding variability in peer influence across different types of peer relationships. A predominant approach for measuring peer influence involves the assessment of adolescents' engagement in a clinically relevant behavior as well as adolescents' perceptions of their friends' engagement in the same behavior. Unidirectional longitudinal associations between adolescents' perceptions of their friends' behavior and their own behavior are examined to reveal potential peer socialization effects. However, recent research has suggested that this approach may impede accurate estimation of peer influence phenomena for at least three reasons. First, studies have varied considerably in the operationalization of a friend who may serve as a source of peer influence. Current developmental psychopathology methods advocate the use of peer nomination procedures to assist adolescents in the identification of specific individuals who are their closest friends (and a single very best friend); in many cases participants' responses can be used to determine whether friendship nominations are reciprocated. Current evidence is mixed regarding the differing potential for influence within reciprocated or unreciprocated friendships. Supporting the hypothesis that close, mutual relationships may provide more opportunity for information exchange and social reinforcement, some research has suggested that stronger socialization effects are observed in reciprocated, compared with unreciprocated friendships (Stevens & Prinstein, 2005). However, other research suggests that socialization effects may be as strong, or stronger within unreciprocated friendships, suggesting that emulating another adolescents' behavior may be a strategy adolescents use to develop a closer relationship with an admired peer (Bot, Engels, Knibbe, & Meeus, 2005).

Investigators' methods for assessing friendships as a potential context for peer influence rarely account for the transient nature of many adolescents' relationships. As noted earlier, many adolescent friendships are short-lived; still other friendships may vary in closeness, such that a very best friend at the time of an initial assessment may become merely a best friend, a friend, or an acquaintance before the follow-up assessment has occurred in a traditional longitudinal design. Currently it is unknown whether there is a minimal dose of exposure to a friend's attitudes or behaviors that is necessary to produce a socialization effect. Therefore, it is unknown whether stable friendships may exert greater influence than unstable friendships; however, recent evidence suggests that stable friendships may be more potent context for peer influence (Laursen, Hafen, Kerr, & Stattin, 2012).

Second, studies that have examined adolescents' reports of their best friends' behavior as a predictor in peer influence research more precisely are measuring the socialization effects of adolescents' *perceptions* of their friends'

behavior. This may be a highly relevant construct to consider; studies suggest that adolescents' perceptions of their friends' behavior may be more proximal predictors of their own behavior and may even mediate the association between adolescents' friends' behavior and adolescents' own behavior (Fromme & Ruela, 1994). However, it should be noted that estimates of peer influence effects relying on adolescents' perceptions of their peers' behavior likely overestimate the magnitude of these effects due to shared method variance. Moreover, substantial theory and research indicate that individuals are notoriously poor at accurately estimating the attitudes and behavior of others. False consensus effects demonstrate that individuals overestimate the similarities between their own and others' behaviors (Ross, Greene, & House, 1977). The uniqueness bias indicates that people underestimate how many of their peers may engage in desirable behaviors that they themselves engage in (Goethals, Messick, & Allison, 1991). Pluralistic ignorance theories suggest that even when engaging in behaviors as frequently as others, individuals may erroneously estimate the underlying attitudes of others as different from their own. For example, a reluctant drinker may estimate higher levels of pro-alcohol attitudes among others who are drinking at levels identical to their own. Empirical work has demonstrated that adolescents' engagement in health risk behaviors is associated with their overestimations of others' engagement in similar behaviors (Prentice, 2008). Research examining the effects of peer socialization relying on an external informant (e.g., friends' own report) of peers' behavior has supported peer socialization effects; however, effect sizes may be somewhat attenuated.

Third, accumulating evidence suggests that adolescents may be influenced within many types of peer relationships. A substantial focus of prior work has been the study of peer influence within adolescents' very best friendships. Emerging evidence suggests that socialization also may occur within adolescents' romantic relationships (Simon, Aikins, & Prinstein, 2008). Data also indicate that adolescents are highly influenced by the behavior of high status peers, with whom they may not have a friendship. For example, experimental research indicates that when presented with identical injunctive social norms, ostensibly communicated either by high-status (i.e., popular and liked) or low-status (i.e., unpopular and rejected) peers (i.e., electronic confederates), adolescents' publically and privately demonstrated greater conformity to high status peers (Cohen & Prinstein, 2006). Adolescents are more likely to behave aggressively when they observe high-status peers doing so compared with when they observe low-status peers engage in identical behavior (Cohen & Prinstein, 2006). Research on media influences has frequently proposed that adolescents' behavior can be socialized by peers whom they have not even met. Empirical data sorely are needed regarding the relative power of different influential agents, as are data on the manner in which adolescents reconcile conflicting messages from different socializing forces in their peer network. Recent data suggest that the most potent peer contexts for socialization may vary by the behavior that is being influenced. For example, recent work has suggested that socialization of depressive symptoms may be most likely to occur within best friendship dyads, while socialization of alcohol use occur within larger social networks (Giletta et al., 2012). This may reflect differences in the mechanisms underlying peer influence effects for different domains of adolescent adjustment and the contexts in which these mechanisms are most likely to occur.

Although most research examining peer influence effects focuses on socialization processes within a dyadic relationship (i.e., very best friendships), researchers agree that very best friendships most often occur within the context of broader friendship groups. Only recently has research begun to understand socialization processes within this broader context. For instance, an especially popular trend has been the use of statistical software that can estimate simultaneously the magnitude of selection and socialization effects among all of an individual's multiple dyadic friendships.

An especially popular recent approach has involved stochastic actor-based modeling (Snijders et al., 2010). This approach is grounded on the notion that individual behaviors and social networks are dynamic and interrelated entities. That is, behavioral changes of individuals within a social network (i.e., actors) are strongly related to the characteristics of the social network and vice versa. Hence, stochastic actor-based models provide a methodological tool to investigate the co-evolution of social networks and individual behaviors over time. These models address several limitations of earlier peer influence analytic approaches. Of most relevance, many observational studies (i.e., nonexperimental studies) do not disentangle selection and socialization processes because they do not take into account that unobserved changes may occur between two discrete observation moments (for a more detailed description, see Steglich, Snijders, & Pearson, 2010). In contrast, stochastic actor-based models assume that unobserved changes occur between two discrete time points. These continuous changes are assumed to occur one at a time via so-called micro steps, which are directly controlled by

actors. That is, actors can change (i.e., increase or decrease) either their behavior or a network tie (i.e., they can form or dissolve a friendship). Actors' decisions to make and select a particular micro step depend exclusively on their current behavior and on the current state of the network, regardless of the previous state (i.e., Markov process). Using a continuous-time Markov chain Monte Carlo (MCMC) approach (Snijders, Steglich, & Schweinberger, 2007), overall changes in actor's behaviors and friendship ties between two discrete time points are estimated through an iterative simulation procedure. This procedure generates unstandardized parameters and their standard errors, which describe changes in actors' behaviors and friendship ties. Notably, when estimating socialization effects, these models control not only for selection effects, but also other confounding effects related to the structure of the social network (i.e., endogenous effects), such as the tendency to reciprocate friendship ties or to form cohesive groups (e.g., triadic relationships). In stochastic actor-based models, peer influence effects typically are operationalized as the tendency of adolescents to become more similar to the average level of behavior among the peers whom they nominated as friends.

Since the first study demonstrating how stochastic actor-based modeling could be applied to estimate socialization effects within adolescent friendship networks (Burk et al., 2007), an increasing number of studies have employed this approach to examine peer influence effects. Overall this growing area of research has provided compelling evidence indicating that peer influence effects contribute to behavioral similarity between adolescents and their friends, even after controlling for selection and network structural effects. Socialization effects in adolescent friendship networks have been documented in relation to a variety of behaviors, including health risk behaviors (e.g., substance use, eating behaviors) and externalizing behaviors (e.g., aggression, delinquency; for a review, see Veenstra, Dijkstra, Steglich, & Van Zalk, 2013). For instance, a number of studies have demonstrated that although adolescent similarity in aggression and substance use is partially due to the tendency of adolescents to select as friends peers with similar levels of aggression and substance use respectively, adolescents also become more similar to their friends' aggression and substance use, as result of peer influence network effects (Veenstra et al., 2013). A few studies also have provided evidence of socialization effects within social networks in relation to internalizing behaviors, including depressive symptoms, loneliness, and social anxiety (Mercer & DeRosier, 2010). Notably, peer influence effects have been demonstrated across different types of friendship networks, including class and grade networks as well as school and outside-school networks. Moreover, these peer influence effects have been reported across different developmental periods, from middle childhood throughout adolescence.

It should be noted that although research using stochastic actor-based modeling has offered a notable contribution to studies of peer influence, this approach typically measures peer socialization effects as the extent to which individuals and their friends' behaviors become more similar to one another over time. It is unclear whether the peer influence effects estimated have direct relevance to the development of psychopathology, therefore. Increasing similarity between adolescents' and their friends' behaviors, as estimated in stochastic actor-based modeling, may reflect adolescents' increases or decreases in the frequency of maladaptive behavior. For instance, network influence effects measured in stochastic actor-based modeling related to aggressive behaviors may indicate that adolescents become more similar to their friends by increasing their levels of aggressive behaviors (conforming to the behavior of their more aggressive friends) or by decreasing their levels of aggressive behaviors (converging to the behavior of their less aggressive friends). Importantly, distinguishing the direction of peer influence effects is a methodological challenge that pertains to many analytic approaches. Most peer influence research tends to neglect the hypothesis that changes in adolescents' behaviors could also reflect positive friends' influence (Berndt & Murphy, 2002). Recent advances in stochastic actor-based modeling (i.e., creation and endowment effects) may allow disentangling deleterious from beneficial effects of peer socialization.

In addition, note also that stochastic actor-based modeling relies on a conceptualization of friendship networks as a summary of (i.e., an average across) all of an individual's friendship dyads. Differences in the strength of the relationships among some of these friends typically are not accounted for. Peer influence effects consequently may be mitigated by averaging socialization among best, close, and casual friends. In addition, social networks should not be confused as similar to peer cliques. Stochastic actor-based modeling does not measure peer influence within cliques.

In fact, extant data suggest that peer influence also occurs within cliques, offering an additional interesting contribution to understanding peer socialization. Developmental theory suggests that peer cliques represent organized groups of peers defined by interconnected friendships, with discrete boundaries, hierarchies, and social norms. Cliques represent more than an amalgam of

multiple friendship dyads. Cliques include a core nucleus of individuals as well as more peripheral ties (Lansford et al., 2009). Cliques have distinct rules and sanctions that differ from how these parameters are expressed within the constituent friendship dyads. Clear hierarchies within peer cliques are associated with different levels of power, different frequencies of contact, and potentially varying levels of peer influence.

Unfortunately, peer cliques are remarkably difficult to operationalize and examine. The notorious difficulties associated with accurately categorizing youths' best friends, friends, and acquaintances (concepts that youth themselves have difficulty articulating with adequate reliability) are compounded when attempting to understanding the breadth of adolescent peer cliques.

Nevertheless, recent work indicates that peer socialization occurs in friendship cliques, and adolescents' position in their clique may significantly alter their susceptibility to clique norms. For instance, Rancourt, Conway, Burk, and Prinstein (2013) revealed that within all-male peer cliques, the average level of dieting behavior of adolescents' cliquemates was associated longitudinally with adolescents' own levels of dieting behavior. These same researchers demonstrated peer clique-level socialization of depressive symptoms, with adolescents' gender and position in their peer clique acting as significant moderators of peer socialization effects. Specifically, compared with other adolescents, the longitudinal association between adolescents' cliquemates' depressive symptoms and adolescents' own depressive symptoms (i.e., both self- and peer-reported) was significantly stronger for female adolescents and adolescents at the periphery of their peer cliques (Conway, Rancourt, Adelman, Burk, & Prinstein, 2011).

Peer Influence Mechanisms

There is little doubt that peer influence processes affect adolescents' development and can promote adolescents' maladaptation. However, it is difficult to reduce peer influence effects. Indeed, efforts to dissuade adolescents from befriending risky peers largely are unsuccessful. Thus, it is critical to identify malleable processes that underlie adolescents' tendencies toward peer conformity.

Recall that developmental theory suggests that peer influence occurs most dramatically during a period also associated with adolescents' heightened sensitivity to peer feedback and acceptance; this developmental period also is associated with rapid identity development. Two theories thus are especially relevant for understanding possible peer influence mechanisms. First, social learning theories contend that individuals adopt behaviors to gain rewards or avoid punishment. Specifically, behaviors are learned through modeling, direct operant reinforcement in the form of social reward or sanctions, and vicarious reinforcement through observational learning (Bandura, 1973). Second, identity-based theories suggest that emulation of idealized others' behavior and adherence to perceived social norms within a valued reference group help confer a favorable sense of self; the adoption of a favorable sense of self is intrinsically rewarding (Festinger, 1954). Accordingly, research on peer influence mechanisms has focused on social reinforcements and identify-enhancing reinforcements that may underlie peer influence effects.

There is substantial evidence that adolescents are likely to engage in behaviors that they believe will earn them social reinforcement. For many adolescents, high peer status is socially reinforcing. This is particularly evident when examining reputation-based forms of peer status, or popularity. Indeed, many of the behaviors especially relevant to peer influence (i.e., aggressive and health-risk behaviors) are associated with high popularity among peers, and research therefore has focused on understanding the social norms of popular peers in particular (Mayeux, Sandstrom, & Cillessen, 2008; Rancourt & Prinstein, 2010). Juvonen and Ho (2008) revealed that middle school students who associate peer-directed aggressive behavior with high popularity (coolness) in the first semester of middle school demonstrated increased antisocial behavior in the second year at that school. Adolescents are more likely to engage in these behaviors and endorse deviant-related attitudes if they believe that doing so has been endorsed by high-status peers (Cohen & Prinstein, 2006). If adolescents are led to believe that these same deviant-related attitudes are endorsed by low-status peers, adolescents demonstrate anticonformity, adopting opposite-valenced attitudes (Cohen & Prinstein, 2006). Related research suggests that failure to adhere to dominant or valued social norms may be met with social punishment in the form of exclusion and rejection by peers (Juvonen & Galvan, 2008).

However, not all adolescents are motivated toward popularity. Some adolescents may be especially influenced toward behaviors that are associated with social norms of peer subgroups that occupy a less central position within the larger peer network. For instance, work has suggested that rejected/aggressive youth may be especially likely to affiliate with deviant peer groups, who actively reject the behavioral norms of the overall peer context and instead adopt a local set of social norms that may be more salient to these adolescents' identity development (Dishion et al., 1995).

Social reinforcements occur not only within the broader peer context but also within dyadic peer relationships; this is an especially important area of inquiry given the large focus of prior work on dyadic friendships as a primary context for peer socialization. In a series of groundbreaking studies, Dishion and colleagues revealed that adolescent dyads engage in subtle but important differences in nonverbal reinforcements during discussions of deviant and nondeviant topics. Within friendship dyads of adolescent males without histories of deviant behavior, utterances regarding deviant activities are followed by neutral affect, whereas discussion of prosocial activities often are followed with nonverbal reinforcements, such as smiling, nodding, and laughing. However, the opposite pattern is evident within friendship dyads of deviant adolescents; deviant talk is followed by positively reinforcing utterances and gestures. This interaction pattern (i.e., referred to as deviancy training) elucidates a process by which peer socialization may occur (Dishion, Spracklen, Andrews, & Patterson 1996). It has been posited that deviancy training represents an attractor state for deviant adolescents (Granic & Dishion, 2003). In other words, deviancy training may be an interaction that deviant adolescents favor; thus, they are especially likely to get stuck in this maladaptive interaction pattern, subsequently predicting ongoing deviant behavior (Granic & Dishion, 2003). Adolescents' exposure to deviancy training is associated longitudinally with increases in problem behavior in middle adolescence (e.g., Dishion, Eddy, Haas, Li, & Spracklen, 1997), and young adulthood (Dishion, Nelson, & Bullock, 2004). In one study, deviancy training observed in a 10-minute dyadic interaction sample predicted criminal arrest rates at least two years later (Dishion et al., 1996), and deviant talk within group therapy contexts has been proposed as a possible explanation for iatrogenic effects of this therapeutic approach among deviant teens with conduct disorder. Interestingly, the effects of deviancy training have been observed among females as well as males (Piehler & Dishion, 2007) and even among kindergarten-age children (Snyder, Schrepferman, Bullard, McEachern, & Patterson, 2012).

In addition to social reinforcement theories, studies of peer influence mechanisms indicate that peer conformity may serve identity-enhancement functions. In other words, peer conformity may be motivated by internal needs and ultimately self-evaluation. Identity-based theories have been discussed at length within the social psychology literature. Generally, these theories suggest that individuals are likely to engage in behaviors that help them establish or maintain a positive sense of self-concept (e.g., Markus & Wurf, 1987). Peers' behaviors offer an opportunity for social comparison. Thus, adolescents may evaluate themselves in reference to their perceptions of others' behavior (i.e., social norms) and modify their behavior in a manner that will confirm a favorable sense of self. Although several derivations of this theory exist, relatively few have been applied to the study of actual risk behaviors among adolescents in a naturalistic context.

For instance, the prototype/willingness model proposes that peer conformity may be the result of two distinct processes (Gibbons, Gerrard, Reimer, & Pomery, 2006). First, individuals evaluate the behavior of those that match their own idealized selves (i.e., prototypes). Behaviors that are perceived to be associated with a desired self-image increase one's favorable attitudes toward the behavior (Ajzen & Fishbein, 1975). Second, the perception that a behavior is similar to the behavior of a desired self or prototype leads to a perception of potential social rewards (i.e., engagement in this behavior will lead to a more favorable self-evaluation). This perception of potential social rewards increases an individual's willingness to engage in a behavior spontaneously should an opportunity become available. In combination, an individual's perception of social norms that align a behavior with their idealized self and an increasing willingness to engage in the behavior conjointly predict behavior engagement (Gibbons et al., 2006).

Blanton and Christie's (2003) deviance regulation theory also emphasizes an underlying motivation to establish a positive self-concept as a basis for peer conformity. While the prototype/willingness model emphasizes a desire to enhance self-concept by achieving similarity with favorable prototypes, deviance regulation theory suggests that individuals are most motivated to achieve a balance between similarity and differentiation from their peers. Such a balance is achieved by consistently aligning one's behaviors with members of a salient and desired reference group through conformity, while still maintaining a sense of uniqueness that engenders feelings of autonomy and distinctiveness from others. Both intrinsic (e.g., sense of self-worth) and extrinsic (e.g., social approval) reinforcements are thought to provide feedback to adolescents, thus potentially serving to maintain the behavior (Blanton & Christie, 2003).

Similarly, Berger's (2008) marketing model of social memes (i.e., fads, trends) posits that social comparison processes and alignment with salient, desirable groups (or nonconformity to undesirable groups) may explain peer socialization. Berger contends that individuals initially engage in a behavior that they feel helps differentiate them

from others, particularly through identification with a special subgroup that matches their desired identity. For example, if alcohol use initially is associated with a typical high status schoolmate, then high status adolescents may be especially likely to adopt this behavior to signal their membership in this in-group. However, when alcohol use begins to become adopted among other, lower status adolescents (i.e., among out-groups), it loses its value as a marker for in-group status and will quickly fade as a preferred behavior among the high status adolescents themselves (see Berger, 2008).

Understanding the mechanisms of peer influence effects that promote youths' maladaptive behavior will require greater empirical attention. At least two neglected putative mechanisms warrant further explanation. First, deviance regulation theory and Berger's model suggest that differentiation from others may be a powerful motivation for peer conformity (or anticonformity). This elucidates an especially pernicious peer influence mechanism that has not been studied extensively, namely, anticonformity effects. By deliberately engaging in behaviors that violate adaptive social norms, adolescents may have the impression that they are not susceptible to the values of their peers, and thus not susceptible to peer socialization. Yet anticonformity is indeed a form of peer socialization, albeit one in which adolescents do not necessarily realize that they are being influenced by others. Second, surprisingly few studies have examined indirect models of peer influence, in which adolescents' behaviors (i.e., X) may be influenced by different, but perhaps related attitudes or behaviors (i.e., Y) of their peers. To date, peer influence research has almost exclusively examined a direct modeling hypothesis, suggesting that adolescents' peers' engagement in a specific behavior (X) will be associated with adolescents' engagement in this identical behavior (X). Several other possibilities deserve consideration. For instance, indirect peer influence may occur through socialization of predisposing, underlying characteristics, which are differentially expressed among members of a friendship dyad. Socialization of impulsive behavior, for instance, may be associated with either drug use or nonsuicidal self-injury, perhaps based on adolescents' own prior experiences (see Giletta, Burk, Scholte, Engels, & Prinstein, 2013). Indirect peer influence also may occur by promoting similarly themed behaviors. A longtime marijuana using friend's initial experimentation with hard drugs may be influential in another adolescent's decision to experiment with marijuana for the first time. Importantly, it is likely that the mechanism of peer influence varies based on the behavior that is subject to socialization. This issue requires greater attention.

Peer Influence Moderators

Some adolescents may be more susceptible to peer influence than others. However, remarkably few research studies have examined the construct of peer influence susceptibility. Theory also is lacking. It is unknown whether peer influence susceptibility is a stable trait or a characteristic that varies substantially within an individual across time, development, or contexts. It also is not known whether adolescents can reflect upon their own level of peer influence susceptibility or whether like many instances of peer socialization itself, this is a process that occurs outside of an individuals' awareness. Susceptibility has been measured using self-report checklists, responses to hypothetical vignettes, and performance-based measures that utilize deceptive elements (Allen, Porter, & McFarland, 2006; Prinstein, Brechwald, & Cohen, 2011; Steinberg & Monahan, 2007). The concurrent and predictive validity of such instruments has been a main focus of research to date.

Although little is known regarding the construct of peer influence susceptibility itself, research increasingly has examined correlates of susceptibility, often referred to as moderators of peer influence. Studies of peer influence moderators typically identify constructs that appear to moderate the association between adolescents' and their peers' behavior. At least four levels of moderators have been identified; notably, moderators likely interact across each of these levels. First, and most frequently examined, are characteristics of the influenced adolescent (i.e., the target adolescent) that may moderate the potency of peer influence effects. Data regarding the moderating effects of gender have yielded inconsistent findings. However, evidence is accumulating to suggest that age may be relevant, with older adolescents and young adults perhaps less susceptible to peer influence, at least for deviant acts, than are younger adolescents (Monahan, Steinberg, & Cauffman, 2009). Research suggests these age effects may be due to higher levels of self-regulation and inhibitory control (Gardner, Dishion, & Connell, 2008). Data also indicate that measures of internalized distress, such as low self-concept, or high social anxiety, may increase the strength of associations between adolescents' and their peers' behavior (Cohen & Prinstein, 2006); however, these effects are not consistently observed (Larsen, Overbeek, Vermulst, Granic, & Engles, 2010). Additionally, poor family functioning appears to augment susceptibility to peer influence (Vitaro, Brendgen, & Tremblay, 2000).

More recently, research has examined possible genetic factors that function as peer influence moderators. For

example, Harden and colleagues (2008) revealed that the impact of best friends' substance use on target adolescents' use was most pronounced for adolescents with the highest genetic vulnerability for using tobacco and alcohol. Similarly, in two samples of young twins, youth were most likely to be physically aggressive if they were both genetically at risk for this behavior and were exposed to highly aggressive peers (Brendgen, Boivin, Vitaro, Bukowski, et al., 2008). Friends' alcohol use was significantly associated with adolescents' own alcohol use more strongly among adolescents with a genetic predisposition for alcohol-based reward responses (i.e., DRD4 VNTR polymorphism; Larsen, van der Zwaluw, et al., 2010).

Second, research has examined characteristics of the influencer that may augment peer socialization effects. There is some evidence to suggest that higher status peers exert greater influence over lower status peers (Cohen & Prinstein, 2006; Laursen et al., 2012; Prinstein, 2007). Similarly, more central members of an adolescents' peer clique appear to exert greater influence over more peripheral members rather than vice versa (Conway et al., 2011). Some data suggest that certain groups (i.e., those with greater centrality in the overall peer context) may exert greater influence over its members than less central groups (Ellis & Zarbatany, 2007b).

A third group of moderators includes relationship attributes between the target and the influencing peer that may alter peer susceptibility. Recent research examining relationship qualities through observational coding has suggested that the effects of deviancy training appear to be strongest within dyads low in entropy (i.e., highly organized and predictable) and high in dyadic mutuality (i.e., reciprocal, shared understanding). Questionnaire-based studies suggest that high levels of positive friendship quality also magnify peer socialization effects for depression symptoms (Prinstein, 2007), substance use (Urberg, Luo, Pilgrim, & Degirmencioglu, 2003), and prosocial behavior (Barry & Wentzel, 2006). As noted already, studies examining friendship reciprocity as a moderator of peer influence effects have wrestled with competing hypotheses. Reciprocated friendships may provide a context for high levels of communication, emotional disclosure, and shared understanding that promotes socialization. On the other hand, an unreciprocated friendship suggests that one member of the dyad desires to develop a closer relationship with the other and may therefore be especially susceptible to socialization. Extant research findings support both hypotheses (cf. Bot et al., 2005; Hall & Valente, 2007; Mercken, Candel, Willems, & de Vries, 2007; Stevens & Prinstein, 2005).

Last, peer susceptibility may vary based on contextual factors, including the context in which socialization processes occur (i.e., in school/home, presence/absence of adults), the number of peers involved, including potential allies for adolescents (Asch, 1952), or even the specific behavior that is at hand. Berndt (1979) initially suggested that susceptibility to peer pressure depended on the social and legal value associated with the target behavior. Alternatively, adolescents' level of prior engagement in specific behaviors (i.e., abstention versus prior experimentation) may affect peer susceptibility. For instance, adolescents who have already initiated alcohol use may be more prone to peer influence toward risky alcohol use than someone who has never consumed alcohol before (Maxwell, 2002). Within studies of social network influence effects, stochastic actor-based modeling offers a unique opportunity for examining contextual factors that could alter socialization effects. For example, a recent study examining socialization of risk attitudes (e.g., substance use and deviant behaviors) revealed stronger influence effects in class networks in which risk attitudes were more strongly associated with peer status than in class networks in which risk attitudes were not or less associated to status (Rambaran, Dijkstra, & Stark, 2013).

Peer Influence Effects Within Multilevel Investigations

Studies of peer influence that adopt a dynamic systems perspective are rare. However, recent work in neuroscience has offered some evidence to suggest more complex models of peer influence effect. For instance, G×E studies indicate that peer influence likely occurs in a manner consistent with a genetic diathesis-stress hypothesis. Results from quantitative twin-design genetic studies consistently indicate that friendships with deviant or substance using peers significantly increases delinquency, alcohol use, and substance use among adolescents with genetic risks (Guo, Elder, Cai, & Hamilton, 2009; Harden et al., 2008; van Lier et al., 2007). Boardman, Saint Onge, Haberstick, Timberlake, and Hewitt (2008) revealed that youth with genetic risks for nicotine use were more likely to smoke when in a school setting with a context that appeared to offer social rewards for smoking (i.e., in schools where smoking was associated with peer popularity).

Evidence from molecular genetic studies similarly supports the genetic diathesis stress effects of deviant peer influence. Lee (2011) revealed a significant interaction between the high-risk MAOA genotype (i.e., dopamine synthesis) and deviant peer affiliation in the prediction of adolescent criminal behavior and Latendresse and colleagues (2011) revealed a similar moderating effect of

deviant peer affiliation in the association between CHRM2 gene (i.e., sensation seeking, disinhibition) and adolescents' externalizing behavior (Brendgen, 2012). Larson and colleagues (2010) revealed that adolescents with the long allele of the DRD4 gene (i.e., alcohol craving/use) demonstrated more alcohol use conformity in a heavy-drinking peer group than adolescents without genetic risk.

In sum, substantial research has suggested that peer influence is a real, powerful, and consistent phenomenon with implications for the development of externalizing, internalizing, and health risk behaviors. The empirical base offers substantial evidence to suggest that adolescents' best friendships serve as a context that can exacerbate risks for psychopathology. Both main effects and interactive effects have been revealed, indicating that the best friendship context may exert direct socialization effects on the development of symptoms, or may interact with preexisting risk factors that are more likely to be expressed in the context of deleterious peer influence. Research increasingly suggests that other peer relationships (e.g., romantic relationships, cliques, networks) and other sources of peer influence (e.g., popular peers) also may be relevant for understanding peer influence effects. Ongoing work also has helped to elucidate the specific mechanisms that explain why adolescents conform to their peers, as well as moderators that help to identify specific youth who may be especially susceptible or resilient to peer influence. Research that integrates peer influence effects into broader dynamic systems models is needed to help understand more complex socialization processes that are relevant to development.

CONCLUSION AND FUTURE DIRECTIONS

For many decades, peer relations researchers have provided empirical data demonstrating that peers contribute meaningfully to youths' development, and to the development of psychopathology. This has been a fruitful and successful endeavor. Few would argue that peers present an important context in which development occurs and that negative peer experiences confer unique risks for maladjustment. Indeed, peers matter for development, and peer experiences can provide risk or protection for psychopathology.

In fact, in many related literatures, including research in associated social and biological science disciplines, there has been substantial appreciation for the importance of social experiences within children's and adolescents' lives. Research indicates that adverse interpersonal experiences, including social rejection among peers, may produce physical pain, affect short- and long-term physiological stress-response systems, compromise the human immune system, activate previously dormant genes, and alter neurochemistry in a manner that augments risk for psychopathology. Conversely, supportive peer relationships can mitigate genetic risks for psychopathology and are associated with activation of brain regions that promote adaptive emotional processing.

Findings from related disciplines also have indicated that peers exert a unique role in the development of youths' attitudes and behaviors. In adolescence, the peer context is a crucible for developing identities; peer interactions are remarkably important for understanding the development of values, consumer choices, the effectiveness of educational policies and clinical interventions, and have clear effects on the development of maladaptive cognitions and behaviors. In sum, the need to substantiate the importance of peer relations has been met.

Perhaps most important given the current state of the literature is the integration of theories and findings from the peer relations literature with the ongoing advances in these related disciplines. For many decades, peer relations researchers have articulated a variety of discrete constructs that help to explain the complex social worlds of children and adolescents. Research has revealed a clear picture of normative developmental peer competencies as well many constructs that characterize atypical development. This chapter offered a brief review of several peer experiences that have been examined most thoroughly as concurrent and longitudinal correlates of psychopathology (e.g., social withdrawal, peer status, peer victimization, friendlessness and friendship quality, and peer influence). Importantly, the distinctions between these constructs, and the methodological advances that have been offered to examine each of these discrete peer experiences, have not been reflected within ongoing work in related disciplines. There is substantial opportunity for peer relations research to integrate with this related work to develop more holistic models for understanding the associations among peer experiences, development, and psychopathology. The field of psychological science has begun to embrace transdisciplinary approaches to inquiry, and team science collaborations; peer relations researchers should be at the forefront of this movement, as their expertise is sorely needed.

In addition to the need for better integration with related scientific disciplines, future research on child and adolescent peer relationships also would benefit by attending to several conceptual issues. First, as is evident in this review, studies in this area only recently have begun to consider the role of peer relationships within the broader context of multiple developmental systems. Reciprocal, transactional

associations between peer experiences, and other developing competences or influences also have been considered in earnest only recently. As has hopefully been demonstrated in this chapter, peer experiences are relevant not only as direct predictors of psychopathology, but also as relevant factors in the development of skills, cognitions, and biopsychosocial processes that are antecedents (or products) of psychopathology. The peer literature would benefit from a consideration of complex developmental models.

Second, greater attention to multiculturalism is needed, including a more thorough understanding of diversity (e.g., gender, ethnicity, sexuality) within a single culture and of cross-cultural differences that may be relevant for understanding associations between peer relations and psychopathology. With regard to diversity, most prior research has examined the peer group as a homogenous context, with measurement of peer status or peer influence, for example, in reference to the entire group at-large. This is not consistent with many youths' peer experiences, however. Subgroups reflecting diversity within the overall peer milieu (i.e., same-ethnic peers, same-gender peers) may be highly relevant for some youth, particularly within large school or community settings, and perhaps especially with youths' increasing participation in peer groups online. In many cases, youths' experiences with these subgroups may deviate considerably from their experiences in the overall peer group, suggesting different pathways between peer experiences and psychopathology. For instance, recent work has revealed that while popularity is a strong longitudinal predictor of adolescents' alcohol use trajectories among White/Caucasian youth, it is not a relevant predictor among African American adolescents at all (Choukas-Bradley, Giletta, Neblett, & Prinstein, 2015). Research capturing these diverse experiences is needed.

In recent years, more work has been conducted with samples of youth from regions outside of North America, offering an opportunity to replicate and extend findings from studies predominantly conducted in the United States and Canada. Developmental psychopathology models emphasize transactions between developmental systems residing not only within individuals, but also multiple environmental systems including broader community and cultural influences (e.g., the exosystem). Research is needed to examine how these cultural norms may influence the meanings and functions of peer relationships. Transactions between peer experiences and the exosystem would help not only to interpret possible cross-cultural differences in peer studies but also to inform theories on how peers affect development universally.

Last, it will be important for research examining links between peer relationships and psychopathology to be informed by theories within the clinical psychology literature. A broad approach for examining adjustment outcomes, including a wide range of possible symptoms and markers of dysfunction has been used too often in prior research. Such an approach lacks predictive specificity and precludes the articulation of specific theories to help understand results. Such a broad, even exploratory approach likely originates from the desire to demonstrate that peers are relevant to adjustment. A focus on multifinality models assumes a focus on predictors of interest rather than a concentrated effort to understand a particular disorder or outcome. Although research must continue to examine the broad implications of negative peer experiences, such efforts should be balanced by studies examining specific clinical outcomes, incorporating extant theories of psychopathology, and understanding how peer experiences are relevant within those existing models.

Related, peer relations science could be especially helpful to inform clinical assessment, prevention and intervention. This chapter highlights myriad distinct constructs that each deserve discrete attention in the assessment of youth social functioning but rarely are included in traditional clinical assessments. It is not sufficient to assess children's peer relationships by asking whether a child gets along well with peers; rather, as suggested by Hartup (1996), an assessment should minimally inquire whether a child is liked by peers, whether a child has a friend, and what that child's friend is like. A more thorough assessment would involve an understanding of youths' desire to interact with peers, their provision or receipt of bullying behaviors, youths' participation within multiple friendships and mutual antipathies (Hartup, 2003), the emergence of cross-gender and perhaps romantic attractions among peers, as well as the behaviors that youth engage in when with, and perhaps influenced by peers. The development of clinical assessment instruments that more comprehensively evaluate youths' peer competence would be of value not only to understand psychopathology risk factors but also to help adequately assess levels of social impairment that are part of the criteria for almost all *DSM* diagnoses.

Peer relations research also has substantial translational implications for prevention and intervention efforts. One notable current issue pertains to bullying prevention programs. Within the United States, there has been a substantial call for antibullying legislation and peer victimization prevention programs. Unfortunately, these policy efforts have been advanced without adequate attention to

scientific progress and findings. As reviewed already, peer aggression and victimization must be understood in the context of aggressive functions (i.e., proactive, reactive); it may be unrealistic to expect that all forms of peer aggression will be reduced using similar intervention methods. Scholars developing preventive intervention approaches that specifically target bullying behavior as a subtype of proactive peer aggression have been more successful, however (Kärnä, Voeten, Little, Poskiparta, Alanen, et al., 2011; Kärnä, Voeten, Little, Poskiparta, Kaljonen, et al., 2011). It will be critical to ensure that antibullying policies and efforts reflect current scientific findings to have maximal impact.

Research regarding friendship and peer influence similarly has implications for practice. Note that predominant models of psychotherapy typically involve amelioration of youth mental health symptoms through contact with children and adolescents themselves and with youths' parents and perhaps teachers. Adolescents' peers rarely are included in the treatment process, yet findings suggest that many maladaptive cognitions and behaviors are developed and reinforced within close friendship interactions. Interestingly, clinicians rarely consider whether their therapeutic progress may be undone by peer socialization processes. Conversely, there is underutilized potential to use these same peer processes to promote adaptive development. Just as co-rumination may foster the development of maladaptive cognitive processing, for instance, therapist-guided friendship interactions may assist adolescents with adaptive coping and problem solving. The peer context is increasingly important to youth as they mature; thus, it makes sense that this context should be considered and perhaps even recruited as particularly potent sources of support and behavior modification. In sum, the integration of basic developmental psychopathology research on youths' peer relations with more applied work on treatment development, efficacy, or mechanisms of treatment effectiveness is a relatively untapped domain with great promise for meaningful contributions.

For many children and adolescents, peers offer a context for enjoyment, exploration, autonomy, and maturation. For many, the peer context provides opportunities for learning and rehearsing new skills, ideas, and behaviors. Peers foster growth and allow youth to achieve developmental competencies needed for successful adaptation. However, some youth experience the peer context quite differently. Whether experienced as a feared setting to avoid, an environment characterized by rejection, torment, and loneliness, or an atmosphere that fosters experimentation with risky and dangerous behavior, the peer context can be destructive. Research understanding how youth relate to their peers always will be critical for understanding adaptive and maladaptive development.

REFERENCES

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review*, *96*(2), 358–372. doi: 10.1037/0033-295X.96.2.358

Adams, R. E., Santo, J., & Bukowski, W. M. (2011). The presence of a best friend buffers the effects of negative experiences. *Developmental Psychology*, *47*(6), 1786–1791. doi: 10.1037/a0025401

Ajzen, I., & Fishbein, M. (1975). A Bayesian analysis of attribution processes. *Psychological Bulletin*, *82*(2), 261–277. doi: 10.1037/h0076477

Albert, D., Chein, J., & Steinberg, L. (2013). The teenage brain: Peer influences on adolescent decision making. *Current Directions in Psychological Science*, *22(2)*, 114–120. doi: 10.1177/0963721412471347

Allen, J. P., Porter, M. R., & McFarland, F. C. (2006). Leaders and followers in adolescent close friendships: Susceptibility to peer influence as a predictor of risky behavior, friendship instability, and depression. *Development and Psychopathology*, *18*, 155– 172. doi: 10.1017/S0954579406060093

Allen, J. P., Porter, M. R., McFarland, F., Marsh, P., & McElhaney, K. (2005). The two faces of adolescents' success with peers: Adolescent popularity, social adaptation, and deviant behavior. *Child Development*, *76*(3), 747–760. doi: 10.1111/j.1467-8624.2005.00875.x

American Psychiatric Association (2013). Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5).

Asch, S. E. (1952). Group forces in the modification and distortion of judgments. In *Social psychology* (pp. 450–501). Englewood Cliffs, NJ: Prentice-Hall, Inc. doi: 10.1037/10025-016

Bagwell, C. L., & Schmidt, M. E. (2011). The friendship quality of overtly and relationally victimized children. *Merrill-Palmer Quarterly*, *57*(2), 158–185. doi: 10.1353/mpq.2011.0009

Bandura, A. (1973). *Aggression: A social learning analysis*. Englewood Cliffs, NJ: Prentice-Hall.

Banny, A. M., Cicchetti, D., Rogosch, F. A., Oshri, A., & Crick, N. R. (2013). Vulnerability to depression: A moderated mediation model of the roles of child maltreatment, peer victimization, and serotonin transporter linked polymorphic region genetic variation among children from low socioeconomic status backgrounds. *Development & Psychopathology*, *25*(3), 599–614.

Barker, E. D., Boivin, M., Brendgen, M., Fontaine, N., Arseneault, L., Vitaro, F. … Tremblay, R. E. (2008). Predictive validity and early predictors of peer-victimization trajectories in preschool. *Archives of General Psychiatry*, *65*(10), 1185–1192. doi: 10.1001/archpsyc.65.10.1185

Barker, E. D., & Salekin, R. T. (2012). Irritable oppositional defiance and callous unemotional traits: is the association partially explained by peer victimization? *Journal of Child Psychology and Psychiatry*, *53*(11), 1167–1175.

Barry, C., & Wentzel, K. R. (2006). Friend influence on prosocial behavior: The role of motivational factors and friendship characteristics. *Developmental Psychology*, *42*(1), 153–163. doi: 10.1037/0012-1649.42.1.153

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, *117*(3), 497–529. doi: 10.1037/0033-2909.117.3.497

Bayer, P., Hjalmarsson, R., & Pozen, D. (2009). Building criminal capital behind bars: Peer effects in juvenile corrections. *Quarterly Journal of Economics*, *124*(1), 105–147.

Beaver, K. M., Wright, J., & DeLisi, M. (2008). Delinquent peer group formation: Evidence of a gene × environment correlation. *Journal of Genetic Psychology: Research and Theory on Human Development*, *169*(3), 227–244. doi: 10.3200/GNTP.169.3.227-244

Beck, A. T. (1976). *Cognitive therapies and emotional disorders*. New York, NY: New American Library.

Bell, S. K., & Morgan, S. B. (2000). Children's attitudes and behavioral intentions toward a peer presented as obese: Does a medical explanation for the obesity make a difference? *Journal of Pediatric Psychology*, *25*(3), 137–145. doi: 10.1093/jpepsy/25.3.137

Bell-Dolan, D. J., Foster, S. L., & Christopher, J. (1995). Girls' peer relations and internalizing problems: Are socially neglected, rejected, and withdrawn girls at risk? *Journal of Clinical Child Psychology*, *24*(4), 463–473. doi: 10.1207/s15374424jccp2404_10

Bell-Dolan, D. J., Foster, S. L., & Sikora, D. M. (1989). Effects of sociometric testing on children's behavior and loneliness in school. *Developmental Psychology*, *25*(2), 306–311. doi: 10.1037/0012-1649.25.2.306

Bem, S. L. (1981). Gender schema theory: A cognitive account of sex typing. *Psychological Review*, *88*(4), 354–364.

Benjet, C., Thompson, R. J., & Gotlib, I. H. (2010). 5-HTTLPR moderates the effect of relational peer victimization on depressive symptoms in adolescent girls. *Journal of Child Psychology and Psychiatry*, *51*(2), 173–179.

Berger, J. (2008). Identity signaling, social influence, and social contagion. In M. J. Prinstein & K. A. Dodge (Eds.), *Understanding peer influence in children and adolescents* (pp. 181–199). New York, NY: Guilford Press.

Berndt, T. J. (1979). Developmental changes in conformity to peers and parents. *Developmental Psychology*, *15*(6), 608.

Berndt, T. J., & McCandless, M. A. (2009). Methods for investigating children's relationships with friends. In K. H. Rubin, W. M. Bukowski, & B. Laursen (Eds.), *Handbook of peer interactions, relationships, and groups* (pp. 63–81). New York, NY: Guilford Press.

Berndt, T. J., & Murphy, L. M. (2002). Influences of friends and friendships: Myths, truths, and research recommendations. In R. V. Kail (Ed.), *Advances in child development and behavior*, *Vol. 30* (pp. 275-310). San Diego, CA US: Academic Press.

Bierman, K., & Wargo, J. B. (1995). Predicting the longitudinal course associated with aggressive-rejected, aggressive (nonrejected), and rejected (nonaggressive) status. *Development and Psychopathology*, *7*(4), 669–682. doi: 10.1017/S0954579400006775

Biggs, B. K., Vernberg, E., Little, T. D., Dill, E. J., Fonagy, P., & Twemlow, S. W. (2010). Peer victimization trajectories and their association with children's affect in late elementary school. *International Journal of Behavioral Development*, *34*(2), 136–146. doi: 10.1177/0165025409348560

Blanton, H., & Christie, C. (2003). Deviance regulation: A theory of action and identity. *Review of General Psychology*, *7*(2), 115–149. doi: 10.1037/1089-2680.7.2.115

Blass, T., & Schwarcz, P. (1982). The relative importance of four determinants of attraction. *Journal of Social Psychology*, *117*(1), 145–146. doi: 10.1080/00224545.1982.9713417

Boardman, J. D., Saint Onge, J. M., Haberstick, B. C., Timberlake, D. S., & Hewitt, J. K. (2008). Do schools moderate the genetic determinants of smoking? *Behavior Genetics*, *38*(3), 234–246. doi: 10.1007/s10519-008-9197-0

Boivin, M., Brendgen, M., Vitaro, F., Forget-Dubois, N., Feng, B., Tremblay, R. E., & Dionne, G., (2013). Evidence of gene-environment correlation for peer difficulties: Disruptive behaviors predict early peer relation difficulties in school through genetic effects. *Development and psychopathology*, *25*(1), 79–92.

Boivin, M., Hymel, S., & Burkowski, W. M. (1995). The roles of social withdrawal, peer rejection, and victimization by peers in predicting loneliness and depressed mood in childhood. *Development and Psychopathology*, *7*(4), 765–785. doi: 10.1017/S0954579400006830

Booth-LaForce, C., Oh, W., Kennedy, A. E., Rubin, K. H., Rose-Krasnor, L., & Laursen, B. (2012). Parent and peer links to trajectories of anxious withdrawal from Grades 5 to 8. *Journal of Clinical Child & Adolescent Psychology*, *41*(2), 138–149.

Bot, S. M., Engels, R. E., Knibbe, R. A., & Meeus, W. J. (2005). Friend's drinking behaviour and adolescent alcohol consumption: The moderating role of friendship characteristics. *Addictive Behaviors*, *30*(5), 929–947. doi: 10.1016/j.addbeh.2004.09.012

Bowker, J. C., & Raja, R. (2011). Social withdrawal subtypes during early adolescence in India. *Journal of Abnormal Child Psychology*, *39*(2), 201–212. doi: 10.1007/s10802-010-9461-7

Bowes, L., Maughan, B., Ball, H., Shakoor, S., Ouellet-Morin, I., Caspi, A. … Arseneault, L. (2013). Chronic bullying victimization across school transitions: The role of genetic and environmental influences. *Development & Psychopathology*, *25*(2), 333–346.

Brechwald, W. A., & Prinstein, M. J. (2011). Beyond homophily: A decade of advances in understanding peer influence processes. *Journal of Research on Adolescence*, *21*(1), 166–179.

Brendgen, M. (2012). Genetics and peer relations: A review. *Journal of Research on Adolescence*, *22*(3), 419–437. doi: 10.1111/j.1532-7795.2012.00798.x

Brendgen, M., Boivin, M., Vitaro, F., Bukowski, W. M., Dionne, G., Tremblay, R. E., & Pérusse, D. (2008). Linkages between children's and their friends' social and physical aggression: Evidence for a gene–environment interaction? *Child Development*, *79*(1), 13–29.

Brendgen, M., Boivin, M., Vitaro, F., Girard, A., Dionne, G., & Pérusse, D. (2008). Gene–environment interaction between peer victimization and child aggression. *Development and Psychopathology*, *20*(2), 455–471.

Brendgen, M., Lamarche, V., Wanner, B., & Vitaro, F. (2010). Links between friendship relations and early adolescents' trajectories of depressed mood. *Developmental Psychology*, *46*(2), 491–501. doi: 10.1037/a0017413

Brendgen, M., Little, T. D., & Krappmann, L. (2000). Rejected children and their friends: A shared evaluation of friendship quality? *Merrill-Palmer Quarterly*, *46*(1), 45–70.

Brendgen, M., Vitaro, F., Bukowski, W. M., Dionne, G., Tremblay, R. E., & Boivin, M. (2013). Can friends protect genetically vulnerable children from depression? *Development and Psychopathology*, *25*(2), 277–289. doi: 10.1017/S0954579412001058

Brendgen, M., Vitaro, F., Tremblay, R. E., & Lavoie, F., (2001). Reactive and proactive aggression: Predictions to physical violence in different contexts and moderating effects of parental monitoring and caregiving behavior. *Journal of abnormal child psychology*, *29*(4), 293–304.

Brendgen, M., Wanner, B., & Vitaro, F. (2007). Peer and teacher effects on the early onset of sexual intercourse. *American Journal of Public Health*, *97*(11), 2070–2075. doi: 10.2105/AJPH.2006.101287

Brown, B. B. (1990). Peer groups and peer cultures. In S. S. Feldman & G. R. Elliott (Eds.), *At the threshold: The developing adolescent* (pp. 171–196). Cambridge, MA: Harvard University Press.

Buhrmester, D., & Furman, W. D. (1986). The changing functions of friends in childhood: A neo-Sullivanian perspective. In *Friendship and social interaction* (pp. 41–62). New York, NY: Springer.

Bukowski, W. M., Hoza, B., & Boivin, M. (1993). Popularity, friendship, and emotional adjustment during early adolescence. In B. Laursen (Ed.), *Close friendships in adolescence* (pp. 23–37). San Francisco, CA: Jossey-Bass.

Bukowski, W. M., Laursen, B., & Hoza, B. (2010). The snowball effect: Friendship moderates escalations in depressed affect among avoidant and excluded children. *Development and Psychopathology*, *22*(4), 749–757. doi: 10.1017/S095457941000043X

Bukowski, W. M., Motzoi, C., & Meyer, F. (2009). Friendship as process, function, and outcome. In K. H. Rubin, W. M. Bukowski, & B. Laursen (Eds.), *Handbook of peer interactions, relationships, and groups* (pp. 217–231). New York, NY: Guilford Press.

Burgess, K. B., & Younger, A. J. (2006). Self-schemas, anxiety, somatic and depressive symptoms in socially withdrawn children and adolescents. *Journal of Research in Childhood Education*, *20*(3), 175–187.

Burk, W. J., Steglich, C. G., & Snijders, T. B. (2007). Beyond dyadic interdependence: Actor-oriented models for co-evolving social networks and individual behaviors. *International Journal of Behavioral Development*, *31*(4), 397–404. doi: 10.1177/0165025407077762

Burks, V., Dodge, K. A., & Price, J. M. (1995). Models of internalizing outcomes of early rejection. *Development and Psychopathology*, *7*(4), 683–695. doi: 10.1017/S0954579400006787

Byrne, D. G., & Griffith, W. (1973). Interpersonal attraction. *Annual Review of Psychology*, *24*, 317–336. doi: 10.1146/annurev.ps.24.020173.001533

Calhoun, C. D., Helms, S. W., Heilbron, N., Rudolph, K. D., Hastings, P. D., & Prinstein, M. J. (2014). Relational victimization, friendship, and adolescents' hypothalamic-pituitary-adrenal axis responses to an in vivo stressor. *Development and Psychopathology 26*(03), 605–618.

Caplan, M. Z., & Hay, D. F. (1989). Preschoolers' responses to peers' distress and beliefs about bystander intervention. *Journal of Child Psychology and Psychiatry*, *30*(2), 231–242. doi: 10.1111/j.1469-7610.1989.tb00237.x

Card, N. A., Stucky, B. D., Sawalani, G. M., & Little, T. D. (2008). Direct and indirect aggression during childhood and adolescence: A meta-analytic review of gender differences, intercorrelations, and relations to maladjustment. *Child Development*, *79*(5), 1185–1229. doi: 10.1111/j.1467-8624.2008.01184.x

Cauffman, E., Shulman, E. P., Steinberg, L., Claus, E., Banich, M. T., Graham, S., & Woolard, J. (2010). Age differences in affective decision making as indexed by performance on the Iowa Gambling Task. *Developmental Psychology*, *46*(1), 193–207. doi: 10.1037/a0016128

Chein, J., Albert, D., O'Brien, L., Uckert, K., & Steinberg, L. (2011). Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Developmental Science*, *14*(2), F1–F10.

Chen, X., DeSouza, A. T., Chen, H., & Wang, L. (2006). Reticent behavior and experiences in peer interactions in Chinese and Canadian children. *Developmental Psychology*, *42*(4), 656–665. doi: 10.1037/0012-1649.42.4.656

Choukas-Bradley, S., Giletta, M., Neblett, E. W., & Prinstein, M. J. (2015) Ethnic Differences in Associations Among Popularity, Likability, and Trajectories of Adolescents' Alcohol Use and Frequency. *Child Development*, *86*(2), 519–535.

Cicchetti, D., & Aber, J. L. (1998). Contextualism and developmental psychopathology. *Development and Psychopathology*, *10*(2), 137–141.

Cicchetti, D., & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. *Psychiatry: Interpersonal and Biological Processes*, *56*(1), 96–118.

Cillessen, A. N., & Bellmore, A. D. (1999). Accuracy of social self-perceptions and peer competence in middle childhood. *Merrill-Palmer Quarterly*, *45*(4), 650–676.

Cillessen, A. N., & Mayeux, L. (2004). From censure to reinforcement: Developmental changes in the association between aggression and social status. *Child Development*, *75*(1), 147–163. doi: 10.1111/j.1467-8624.2004.00660.x

Cillessen, A. N., & Rose, A. J. (2005). Understanding popularity in the peer system. *Current Directions in Psychological Science*, *14*(2), 102–105. doi: 10.1111/j.0963-7214.2005.00343.x

Cillessen, A. N., Bukowski, W. M., & Haselager, G. T. (2000). Stability of sociometric categories. In A. N. Cillessen & W. M. Bukowski (Eds.), *Recent advances in the measurement of acceptance and rejection in the peer system* (pp. 75–93). San Francisco, CA: Jossey-Bass.

Cillessen, A. N., Schwartz, D., & Mayeux, L. (2011). *Popularity in the peer system*. New York, NY: Guilford Press.

Cillessen, A. H., van IJzendoorn, H. W., Van Lieshout, C. F., & Hartup, W. W. (1992). Heterogeneity among peer-rejected boys: Subtypes and stabilities. *Child Development*, *63*(4), 893–905. doi: 10.2307/1131241

Coan, J. A., Schaefer, H. S., & Davidson, R. J. (2006). Lending a hand: Social regulation of the neural response to threat. *Psychological Science*, *17*(12), 1032–1039. doi: 10.1111/j.1467-9280.2006.01832.x

Cohen, G. L., & Prinstein, M. J. (2006). Peer contagion of aggression and health risk behavior among adolescent males: An experimental investigation of effects on public conduct and private attitudes. *Child Development*, *77*(4), 967–983. doi: 10.1111/j.1467-8624.2006.00913.x

Cohen, J. (1988). *Statistical power analysis for the social sciences*. Hillsdale, NJ: Lawrence Erlbaum.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, *98*(2), 310–357. doi: 10.1037/0033-2909.98.2.310

Cohen, S., Janicki-Deverts, D., Doyle, W. J., Miller, G. E., Frank, E., Rabin, B. S., & Turner, R. B. (2012). Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proceedings of the National Academy of Sciences*, *109*(16), 5995–5999.

Coie, J. D., & Dodge, K. A. (1983). Continuities and changes in children's social status: A five-year longitudinal study. *Merrill-Palmer Quarterly*, *29*(3), 261–282.

Coie, J. D., & Kupersmidt, J. B. (1983). A behavioral analysis of emerging social status in boys' groups. *Child Development*, *54*(6), 1400–1416. doi: 10.2307/1129803

Coie, J. D., Lochman, J. E., Terry, R., & Hyman, C. (1992). Predicting early adolescent disorder from childhood aggression and peer rejection. *Journal of Consulting and Clinical Psychology*, *60*, 783–783.

Coie, J., Terry, R., Lenox, K., Lochman, J., & Hyman, C. (1995). Childhood peer rejection and aggression as predictors of stable patterns of adolescent disorder. *Development and Psychopathology*, *7*(4), 697–713. doi: 10.1017/S0954579400006799

Conway, C. C., Rancourt, D., Adelman, C. B., Burk, W. J., & Prinstein, M. J. (2011). Depression socialization within friendship groups at the transition to adolescence: The roles of gender and group centrality as moderators of peer influence. *Journal of Abnormal Psychology*, *120*(4), 857–867. doi: 10.1037/a0024779

Coplan, R. J., & Armer, M. (2007). A "multitude" of solitude: A closer look at social withdrawal and nonsocial play in early childhood. *Child Development Perspectives*, *1*(1), 26–32.

Coplan, R. J., & Weeks, M. (2010). Unsociability in middle childhood: Conceptualization, assessment, and associations with socioemotional functioning. *Merrill-Palmer Quarterly*, *56*(2), 105–130.

Coplan, R. J., Rose-Krasnor, L., Weeks, M., Kingsbury, A., Kingsbury, M., & Bullock, A. (2012). Alone is a crowd: Social motivations, social withdrawal, and socioemotional functioning in later childhood. *Developmental Psychology*, *49*(5), 861. doi: 10.1037/a0028861

Coplan, R. J., Rubin, K. H., Fox, N. A., Calkins, S. D., & Stewart, S. L. (1994). Being alone, playing alone, and acting alone: Distinguishing among reticence and passive and active solitude in young children. *Child Development*, *65*(1), 129–137. doi: 10.2307/1131370

Copeland, W. E., Wolke, D., Lereya, S. T., Shanahan, L., Worthman, C., & Costello, E. J. (2014). Childhood bullying involvement predicts low-grade systemic inflammation into adulthood. *Proceedings of the National Academy of Sciences*, *111*(21), 7570–7575.

Cowen, E. L., Pederson, A., Babigian, H., Isso, L. D., & Trost, M. (1973). Long-term follow-up of early detected vulnerable children. *Journal of Consulting and Clinical Psychology*, *41*(3), 438–446. doi: 10.1037/h0035373

Coyne, J. C. (1976a). Depression and the response of others. *Journal of Abnormal Psychology*, *85*(2), 186–193. doi: 10.1037/0021-843X.85.2.186

Coyne, J. C. (1976b). Toward an interactional description of depression. *Psychiatry: Journal for the Study of Interpersonal Processes*, *39*(1), 28–40.

Craig, W. M., & Pepler, D. J. (1997). Observations of bullying and victimization in the school yard. *Canadian Journal of School Psychology*, *13*(2), 41–59. doi: 10.1177/082957359801300205

Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin*, *115*(1), 74–101. doi: 10.1037/0033-2909.115.1.74

Crick, N. R., & Dodge, K. A. (1996). Social information-processing mechanisms on reactive and proactive aggression. *Child Development*, *67*(3), 993–1002. doi: 10.2307/1131875

Crick, N. R., & Grotpeter, J. K. (1995). Relational aggression, gender, and social-psychological adjustment. *Child Development*, *66*(3), 710–722. doi: 10.2307/1131945

Crone, E. A., & Dahl, R. E. (2012). Understanding adolescence as a period of social–affective engagement and goal flexibility. *Nature Reviews Neuroscience*, *13*(9), 636–650. doi: 10.1038/nrn3313

Dahl, R. E. (2004). Adolescent brain development: a period of vulnerabilities and opportunities. Keynote address. *Annals of the New York Academy of Sciences*, *1021*(1), 1–22.

Dantzer, R., O'Connor, J. C., Freund, G. C., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience*, *9*(1), 46–57. doi: 10.1038/nrn2297

De Los Reyes, A., & Prinstein, M. J. (2004). Applying depression-distortion hypotheses to the assessment of peer victimization in adolescents. *Journal of Clinical Child and Adolescent Psychology*, *33*(2), 325–335. doi: 10.1207/s15374424jccp3302_14

Degnan, K., & Fox, N. A. (2007). Behavioral inhibition and anxiety disorders: Multiple levels of a resilience process. *Development and Psychopathology*, *19*(3), 729–746. doi: 10.1017/S0954579407000363

DeLay, D., Laursen, B., Kiuru, N., Salmela-Aro, K., & Nurmi, J. (2013). Selecting and retaining friends on the basis of cigarette smoking similarity. *Journal of Research on Adolescence*, *23*(3), 464–473. doi: 10.1111/jora.12017

Denham, S. A., McKinley, M., Couchoud, E. A., & Holt, R. (1990). Emotional and behavioral predictors of preschool peer ratings. *Child Development*, *61*(4), 1145–1152. doi: 10.2307/1130882

Dishion, T. J., & Andrews, D. W. (1995). Preventing escalation in problem behaviors with high-risk young adolescents: Immediate and 1-year outcomes. *Journal of Consulting and Clinical Psychology*, *63*(4), 538–548. doi: 10.1037/0022-006X.63.4.538

Dishion, T. J., Capaldi, D. M., Spracklen, K. M., & Li, F. (1995). Peer ecology of male adolescent drug use. *Development and Psychopathology*, *7*(4), 803–824. doi: 10.1017/S0954579400006854

Dishion, T. J., Eddy, J. M., Haas, E., Li, F., & Spracklen, K. (1997). Friendships and violent behavior during adolescence. *Social Development*, *6*(2), 207–223.

Dishion, T. J., Nelson, S. E., & Bullock, B. M. (2004). Premature adolescent autonomy: Parent disengagement and deviant peer process in the amplification of problem behaviour. *Journal of adolescence*, *27*(5), 515–530.

Dishion, T. J., Patterson, G. R., & Griesler, P. C. (1994). Peer adaptations in the development of antisocial behavior: A confluence model. In L. Huesmann (Ed.), *Aggressive behavior: Current perspectives* (pp. 61–95). New York, NY: Plenum Press.

Dishion, T. J., Poulin, F., & Skaggs, N. (2000). The ecology of premature autonomy in adolescence: Biological and social influences. In K. A. Kerns, J. M. Contreras, & A. M. Neal-Barnett (Eds.), *Family and peers: Linking two social worlds* (pp. 27–45). Westport, CT: Praeger Publishers/Greenwood Publishing Group.

Dishion, T. J., Spracklen, K. M., Andrews, D. W., & Patterson, G. R. (1996). Deviancy training in male adolescents friendships. *Behavior Therapy*, *27*(3), 373–390. doi: 10.1016/S0005-7894(96)80023-2

Dodge, K. A. (1983). Behavioral antecedents of peer social status. *Child Development*, *54*(6), 1386–1399. doi: 10.2307/1129802

Dodge, K. A., Bates, J. E., & Pettit, G. S. (1990). Mechanisms in the cycle of violence. *Science*, *250*(4988), 1678–1683. doi: 10.1126/science.2270481

Dodge, K. A., & Coie, J. D. (1987). Social-information-processing factors in reactive and proactive aggression in children's peer groups. *Journal of Personality and Social Psychology*, *53*(6), 1146–1158. doi: 10.1037/0022-3514.53.6.1146

Dodge, K. A., Greenberg, M. T., & Malone, P. S. (2008). Testing an idealized dynamic cascade model of the development of serious violence in adolescence. *Child Development*, *79*(6), 1907–1927. doi: 10.1111/j.1467-8624.2008.01233.x

Dodge, K. A., Lochman, J. E., Harnish, J. D., Bates, J. E., & Pettit, G. S. (1997). Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. *Journal of Abnormal Psychology*, *106*(1), 37–51. doi: 10.1037/0021-843X.106.1.37

Dodge, K. A., Pettit, G. S., Bates, J. E., & Valente, E. (1995). Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. *Journal of Abnormal Psychology*, *104*(4), 632–643. doi: 10.1037/0021-843X.104.4.632

Dollard, J., Miller, N. E., Doob, L. W., Mowrer, O. H., & Sears, R. R. (1939). *Frustration and aggression*. Oxford, UK: Yale University Press.

Eisenberger, N. I. (2012). The pain of social disconnection: examining the shared neural underpinnings of physical and social pain. *Nature Reviews Neuroscience*, *13*, 421–434.

Eisenberger, N. I., & Cole, S. W. (2012). Social neuroscience and health: Neurophysiological mechanisms linking social ties with physical health. *Nature Neuroscience*, *15*(5), 669–674. doi: 10.1038/nn.3086

Eisenberger, N. I., Inagaki, T. K., Rameson, L. T., Mashal, N. M., & Irwin, M. R. (2009). An fMRI study of cytokine-induced depressed mood and social pain: the role of sex differences. *Neuroimage*, *47*(3), 881–890.

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science*, *302*(5643), 290–292. doi: 10.1126/science.1089134

Ellis, S., Rogoff, B., & Cromer, C. C. (1981). Age segregation in children's social interactions. *Developmental Psychology*, *17*(4), 399.

Ellis, W. E., & Zarbatany, L. (2007a). Explaining friendship formation and friendship stability. The role of children's and friends' aggression and victimization. *Merrill-Palmer Quarterly*, *53*(1), 79–104. doi: 10.1353/mpq.2007.0001

Ellis, W. E., & Zarbatany, L. (2007b). Peer group status as a moderator of group influence on children's deviant, aggressive, and prosocial behavior. *Child Development*, *78*(4), 1240–1254. doi: 10.1111/j.1467-8624.2007.01063.x

Erhardt, D., & Hinshaw, S. P. (1994). Initial sociometric impressions of attention-deficit hyperactivity disorder and comparison boys: Predictions from social behaviors and from nonbehavioral variables. *Journal of Consulting and Clinical Psychology*, *62*(4), 833–842. doi: 10.1037/0022-006X.62.4.833

Ernst, M., Nelson, E. E., Jazbec, S., McClure, E. B., Monk, C. S., Leibenluft, E, Blair, J., & Pine, D. S. (2005). Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. *Neuroimage*, *25*(4), 1279–1291.

Fein, G. G. (1981). Pretend play in childhood: An integrative review. *Child Development*, *52*(4), 1095–1118. doi: 10.2307/1129497

Feldman, S. S., Rosenthal, D. R., Brown, N. L., & Canning, R. D. (1995). Predicting sexual experience in adolescent boys from peer rejection and acceptance during childhood. *Journal of Research on Adolescence*, *5*, 387–411.

Festinger, L. (1954). A theory of social comparison processes. *Human Relations*, 7117–7140. doi: 10.1177/001872675400700202

Fowler, J. H., & Christakis, N. A. (2009). Dynamic spread of happiness in a large social network: Longitudinal analysis over 20 years in the Framingham Heart Study. *British Medical Journal*, *338*(7685), 1–13.

Fowler, J. H., Settle, J. E., & Christakis, N. A. (2011). Correlated genotypes in friendship networks. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, *108*(5), 1993–1997. doi: 10.1073/pnas.1011687108

Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., & Ghera, M. M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. *Annual Review of Psychology*, *56*, 235–262. doi: 10.1146/annurev.psych.55.090902.141532

Fox, N. A., Henderson, H. A., Rubin, K. H., Calkins, S. D., & Schmidt, L. A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development*, *72*(1), 1–21. doi: 10.1111/1467-8624.00262

French, D. C., Conrad, J., & Turner, T. M. (1995). Adjustment of antisocial and nonantisocial rejected adolescents. *Development and Psychopathology*, *7*(04), 857–874.

Fromme, K., & Ruela, A. (1994). Mediators and moderators of young adults' drinking. *Addiction*, *89*, 63–71.

Fuligni, A. J., Telzer, E. H., Bower, J., Cole, S. W., Kiang, L., & Irwin, M. R. (2009). A preliminary study of daily interpersonal stress and C-reactive protein levels among adolescents from Latin America and European backgrounds. *Psychosomatic Medicine*, *71*(3), 329–333. doi: 10.1097/PSY.0b013e3181921b1f

Furman, W. (1982). Children's friendships: Integrative efforts in a new area. *Psyccritiques*, *27*(5), 349–350. doi: 10.1037/021133

Furman, W. (1998). The measurement of friendship perceptions: Conceptual and methodological issues. In W. M. Bukowski, A. F. Newcomb, & W. W. Hartup (Eds.), *The company they keep: Friendship in childhood and adolescence* (pp. 41–65). New York, NY: Cambridge University Press.

Furman, W., & Buhrmester, D. (1992). Age and sex differences in perceptions of networks of personal relationships. *Child Development*, *63*(1), 103–115. doi: 10.2307/1130905

Galen, B., & Underwood, M. K. (1997). A developmental investigation of social aggression among children. *Developmental Psychology*, *33*(4), 589–600. doi: 10.1037/0012-1649.33.4.589

Galvan, A., Hare, T. A., Parra, C. E., Penn, J., Voss, K., Glover, G., & Casey, B. J. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience*, *26*(25), 6885–6892. doi: 10.1523/JNEUROSCI.1062-06.2006

Ganzel, B. L., Morris, P. A., & Wethington, E. (2010). Allostasis and the human brain: Integrating models of stress from the social and life sciences. *Psychological Review*, *117*(1), 134–174. doi: 10.1037/a0017773

Garber, J., Keiley, M. K., & Martin, N. C. (2002). Developmental trajectories of adolescents' depressive symptoms: Predictors of change. *Journal of Consulting and Clinical Psychology*, *70*(1), 79–95. doi: 10.1037/0022-006X.70.1.79

Gardner, M., & Steinberg, L. (2005). Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Developmental Psychology*, *41*(4), 625–635. doi: 10.1037/0012-1649.41.4.625

Gardner, T. W., Dishion, T. J., & Connell, A. M. (2008). Adolescent self-regulation as resilience: Resistance to antisocial behavior within the deviant peer context. *Journal of Abnormal Child Psychology*, *36*(2), 273–284. doi: 10.1007/s10802-007-9176-6

Gazelle, H., & Ladd, G. W. (2003). Anxious solitude and peer exclusion: A diathesis-stress model of internalizing trajectories in childhood. *Child Development*, *74*(1), 257–278. doi: 10.1111/1467-8624.00534

Gazelle, H., Workman, J., & Allan, W. (2010). Anxious solitude and clinical disorder in middle childhood: Bridging developmental and clinical approaches to childhood social anxiety. *Journal of Abnormal Child Psychology*, *38*(1), 1–17. doi: 10.1007/s10802-009-9343-z

Gershman, E. S., & Hayes, D. S. (1983). Differential stability of reciprocal friendships and unilateral relationships among preschool children. *Merrill-Palmer Quarterly*, *29*(2), 169–177.

Gibbons, F. X., Gerrard, M., Reimer, R. A., & Pomery, E. A. (2006). Unintentional behavior: A subrational approach to health risk. In D. de Ridder & J. de Wit (Eds.), *Self-regulation in health behavior* (pp. 45–70). West Sussex, UK: John Wiley & Sons, Ltd.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A. … Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, *2*(10), 861–863. doi: 10.1038/13158

Giletta, M., Burk, W. J., Scholte, R. J., Engels, R. E., & Prinstein, M. J. (2013). Direct and indirect peer socialization of adolescent nonsuicidal self-injury. *Journal of Research on Adolescence*, *23*(3), 450–463. doi: 10.1111/jora.12036

Giletta, M., Scholte, R. H., Prinstein, M. J., Engels, R. C., Rabaglietti, E., & Burk, W. J. (2012). Friendship context matters: Examining the domain specificity of alcohol and depression socialization among adolescents. *Journal of Abnormal Child Psychology*, *40*(7), 1027–1043.

Giletta, M., Scholte, R. J., Burk, W. J., Engels, R. E., Larsen, J. K., Prinstein, M. J., & Ciairano, S. (2011). Similarity in depressive symptoms in adolescents' friendship dyads: Selection or socialization? *Developmental Psychology*, *47*(6), 1804–1814. doi: 10.1037/a0023872

Gimeno, D. D., Kivimäki, M. M., Brunner, E. J., Elovainio, M. M., De Vogli, R. R., Steptoe, A. A. … Ferrie, J. E. (2009). Associations of C-reactive protein and interleukin-6 with cognitive symptoms of depression: 12-year follow-up of the Whitehall II study. *Psychological Medicine*, *39*(3), 413–423. doi: 10.1017/S0033291708003723

Goethals, G. R., Messick, D. M., & Allison, S. T. (1991). The uniqueness bias: Studies of constructive social comparison. In J. Suls & T. Wills (Eds.), *Social comparison: Contemporary theory and research* (pp. 149–176). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Gottman, J. M. (1983). How children become friends. *Monographs of the Society for Research in Child Development*, *48*(3). doi: 10.2307/1165860

Graham, M. A., Eich, C., Kephart, B., & Peterson, D. (2000). Relationship among body image, sex, and popularity of high school students. *Perceptual and Motor Skills*, *90*(3,Pt2), 1187–1193. doi: 10.2466/PMS.90.3.1187-1193

Graham, S., & Juvonen, J. (1998). Self-blame and peer victimization in middle school: An attributional analysis. *Developmental Psychology*, *34*(3), 587–538. doi: 10.1037/0012-1649.34.3.587

Granic, I., & Dishion, T. J. (2003). Deviant talk in adolescent friendships: A step toward measuring a pathogenic attractor process. *Social Development*, *12*(3), 314–334. doi: 10.1111/1467-9507.00236

Guo, G. (2006). Genetic similarity shared by best friends among adolescents. *Twin Research and Human Genetics*, *9*(1), 113–121. doi: 10.1375/twin.9.1.113

Guo, G., Elder, G. H., Cai, T., & Hamilton, N. (2009). Gene–environment interactions: Peers' alcohol use moderates genetic contribution to adolescent drinking behavior. *Social Science Research*, *38*(1), 213–224. doi: 10.1016/j.ssresearch.2008.04.002

Hafen, C. A., Laursen, B., Burk, W. J., Kerr, M., & Stattin, H. (2011). Homophily in stable and unstable adolescent friendships: Similarity breeds constancy. *Personality and Individual Differences*, *51*(5), 607–612. doi: 10.1016/j.paid.2011.05.027

Hall, J. A., & Valente, T. W. (2007). Adolescent smoking networks: The effects of influence and selection on future smoking. *Addictive Behaviors*, *32*(12), 3054–3059. doi: 10.1016/j.addbeh.2007.04.008

Hanish, L. D., & Guerra, N. G. (2002). A longitudinal analysis of patterns of adjustment following peer victimization. *Development and Psychopathology*, *14*(1), 69–89. doi: 10.1017/S0954579402001049

Hankin, B. L., Stone, L., & Wright, P. (2010). Corumination, interpersonal stress generation, and internalizing symptoms: Accumulating effects and transactional influences in a multiwave study of adolescents. *Development and Psychopathology*, *22*(1), 217–235. doi: 10.1017/S0954579409990368

Harden, K., Hill, J. E., Turkheimer, E., & Emery, R. E. (2008). Gene–environment correlation and interaction in peer effects on adolescent alcohol and tobacco use. *Behavior Genetics*, *38*(4), 339–347. doi: 10.1007/s10519-008-9202-7

Harrist, A. W., Zaia, A. F., Bates, J. E., Dodge, K. A., & Pettit, G. S. (1997). Subtypes of social withdrawal in early childhood: Sociometric status and social-cognitive differences across four years. *Child Development*, *68*(2), 278–294. doi: 10.2307/1131850

Harter, S., Stocker, C., & Robinson, N. S. (1996). The perceived directionality of the link between approval and self-worth: The liabilities of a looking glass self-orientation among young adolescents. *Journal of Research on Adolescence*, *6*(3), 285–308.

Hartup, W. W. (1996). The company they keep: Friendships and their developmental significance. *Child Development*, *67(1)*, 1–13. doi: 10.2307/1131681

Hartup, W. W. (2003). Toward understanding mutual antipathies in childhood and adolescence. *New Directions for Child and Adolescent Development*, *111–123*. doi: 10.1002/cd.92

Haselager, G. T., Hartup, W. W., van Lieshout, C. M., & Riksen-Walraven, J. A. (1998). Similarities between friends and nonfriends in middle childhood. *Child Development*, *69*(4), 1198–1208. doi: 10.2307/1132369

Hawker, D. J., & Boulton, M. J. (2000). Twenty years' research on peer victimization and psychosocial maladjustment: A meta-analytic review of cross-sectional studies. *Journal of Child Psychology and Psychiatry*, *41*(4), 441–455. doi: 10.1111/1469-7610.00629

Heilbron, N., & Prinstein, M. J. (2010). Adolescent peer victimization, peer status, suicidal ideation, and nonsuicidal self-injury. *Merrill-Palmer Quarterly*, *56*(3), 388–419. doi: 10.1353/mpq.0.0049

Higgins, E. (1987). Self-discrepancy: A theory relating self and affect. *Psychological Review*, *94*(3), 319–340. doi: 10.1037/0033-295X.94.3.319

Hodges, E. E., Boivin, M., Vitaro, F., & Bukowski, W. M. (1999). The power of friendship: Protection against an escalating cycle of peer victimization. *Developmental Psychology*, *35*(1), 94–101. doi: 10.1037/0012-1649.35.1.94

Homel, R., Burns, A., & Goodnow, J. (1987). Parental social networks and child development. *Journal of Social and Personal Relationships*, *4*(2), 159–177. doi: 10.1177/0265407587042004

Hoover, J. H., Oliver, R., & Hazler, R. J. (1992). Bullying: Perceptions of adolescent victims in the midwestern USA. *School Psychology International*, *13*(1), 5–16. doi: 10.1177/0143034392131001

Hostinar, C. E., Sullivan, R. M., & Gunnar, M. R. (2014). Psychobiological mechanisms underlying the social buffering of the hypothalamic–pituitary–adrenocortical axis: A review of animal models and human studies across development. *Psychological Bulletin*, *140*(1), 256–282. doi: 10.1037/a0032671

Hoza, B., Mrug, S., Gerdes, A. C., Hinshaw, S. P., Bukowski, W. M., Gold, J. A. … Arnold, L. E. (2005). What aspects of peer relationships are impaired in children with attention-deficit/hyperactivity disorder? *Journal of Consulting and Clinical Psychology*, *73*(3), 411.

Hoza, B., Murray-Close, D., Arnold, L. E., Hinshaw, S. P., & Hechtman, L. (2010). Time-dependent changes in positively biased self-perceptions of children with ADHD: A developmental psychopathology perspective. *Development and Psychopathology*, *22*(2), 375.

Hoza, B., Pelham, W. E., Milich, R., Pillow, D., & McBride, K. (1993). The self-perceptions and attributions of attention deficit hyperactivity disordered and nonreferred boys. *Journal of Abnormal Child Psychology*, *21*(3), 271–286. doi: 10.1007/BF00917535

Hoza, B., Waschbusch, D. A., Pelham, W. E., Molina, B. G., & Milich, R. (2000). Attention-deficit/hyperactivity disordered and control boys' responses to social success and failure. *Child Development*, *71*(2), 432–446. doi: 10.1111/1467-8624.00155

Hubbard, J. A., Smithmyer, C. M., Ramsden, S. R., Parker, E. H., Flanagan, K. D., Dearing, K. F., … Simons, R. F. (2002). Observational, physiological, and self-report measures of children's anger: Relations to reactive versus proactive aggression. *Child Development*, *73*(4), 1101–1118. doi: 10.1111/1467-8624.00460

Hussong, A. M. (2000). Distinguishing mean and structural sex differences in adolescent friendship quality. *Journal of Social and Personal Relationships*, *17*(2), 223–243. doi: 10.1177/0265407500172004

Hymel, S., Rubin, K. H., Rowden, L., & LeMare, L. (1990). Children's peer relationships: Longitudinal prediction of internalizing and externalizing problems from middle to late childhood. *Child Development*, *61*(6), 2004–2021.

Irwin, M. R., & Cole, S. W. (2011). Reciprocal regulation of the neural and innate immune systems. *Nature Reviews Immunology*, *11*(9), 625–632.

Jiang, X., & Cillessen, A. N. (2005). Stability of continuous measures of sociometric status: A meta-analysis. *Developmental Review*, *25*(1), 1–25. doi: 10.1016/j.dr.2004.08.008

Joiner, T. E., Jr., & Katz, J. (1999). Contagion of depressive symptoms and mood: Meta-analytic review and explanations from cognitive, behavioral, and interpersonal viewpoints. *Clinical Psychology: Science and Practice*, *6*(2), 149–164. doi: 10.1093/clipsy/6.2.149

Juvonen, J., & Galván, A. (2008). Peer influence in involuntary social groups: Lessons from research on bullying. In M. J. Prinstein & K. A. Dodge (Eds.), *Understanding peer influence in children and adolescents* (pp. 225–244). New York, NY: Guilford Press.

Juvonen, J., & Ho, A. Y. (2008). Social motives underlying antisocial behavior across middle school grades. *Journal of Youth and Adolescence*, *37*(6), 747–756.

Juvonen, J., & Murdock, T. B. (1995). Grade-level differences in the social value of effort: Implications for self-presentation tactics of early adolescents. *Child Development*, *66*(6), 1694–1705. doi: 10.2307/1131904.

Kärnä, A., Voeten, M., Little, T. D., Poskiparta, E., Alanen, E., & Salmivalli, C. (2011). Going to scale: A nonrandomized nationwide trial of the KiVa antibullying program for grades 1–9. *Journal of Consulting and Clinical Psychology*, *79*(6), 796.

Kärnä, A., Voeten, M., Little, T. D., Poskiparta, E., Kaljonen, A., & Salmivalli, C. (2011). A Large-scale evaluation of the KiVa antibullying program: Grades 4–6. *Child Development*, *82*(1), 311–330.

Kagan, J., Reznick, J. S., Clarke, C., Snidman, N., & Garcia-Coll, C. (1984). Behavioral inhibition to the unfamiliar. *Child Development*, *2212–2225*.

Kandel, D. B. (1978). Similarity in real-life adolescent friendship pairs. *Journal of Personality and Social Psychology*, *36*(3), 306–312.

Katz, S. J., Conway, C. C., Hammen, C. L., Brennan, P. A., & Najman, J. M. (2011). Childhood social withdrawal, interpersonal impairment, and young adult depression: A mediational model. *Journal of Abnormal Child Psychology*, *39*(8), 1227–1238.

Keijsers, L., Branje, S., Hawk, S. T., Schwartz, S. J., Frijns, T., Koot, H. M., . . . Meeus, W. (2012). Forbidden friends as forbidden fruit: Parental supervision of friendships, contact with deviant peers, and adolescent delinquency. *Child Development*, *83*(2), 651–666.

Kennedy, E., Spence, S. H., & Hensley, R. (1989). An examination of the relationship between childhood depression and social competence amongst primary school children. *Journal of Child Psychology and Psychiatry*, *30*(4), 561–573. doi: 10.1111/j.1469-7610.1989.tb00268.x

Kiesner, J. (2002). Depressive symptoms in early adolescence: Their relations with classroom problem behavior and peer status. *Journal of Research on Adolescence*, *12*(4), 463–478. doi: 10.1111/1532-7795.00042

Kim, J., & Cicchetti, D. (2010). Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology. *Journal of Child Psychology and Psychiatry*, *51*(6), 706–716. doi: 10.1111/j.1469-7610.2009.02202.x

Kistner, J., Balthazor, M., Risi, S., & Burton, C. (1999). Predicting dysphoria in adolescence from actual and perceived peer acceptance in childhood. *Journal of Clinical Child Psychology*, *28*(1), 94–104. doi: 10.1207/s15374424jccp2801_8

Knack, J. M., Jensen-Campbell, L. A., & Baum, A. (2011). Worse than sticks and stones? Bullying is associated with altered HPA axis functioning and poorer health. *Brain and Cognition*, *77*(2), 183–190.

Kochel, K. P., Ladd, G. W., & Rudolph, K. D. (2012). Longitudinal associations among youth depressive symptoms, peer victimization, and low peer acceptance: An interpersonal process perspective. *Child Development*, *83*(2), 637–650.

Kochenderfer-Ladd, B. (2004). Peer victimization: The role of emotions in adaptive and maladaptive coping. *Social Development*, *13*(3), 329–349. doi: 10.1111/j.1467-9507.2004.00271.x

Kupersmidt, J. B., & Patterson, C. J. (1991). Childhood peer rejection, aggression, withdrawal, and perceived competence as predictors of self-reported behavior problems in preadolescence. *Journal of Abnormal Child Psychology*, *19*(4), 427–449. doi: 10.1007/BF00919087

La Greca, A. M., & Harrison, H. (2005). Adolescent peer relations, friendships, and romantic relationships: Do they predict social anxiety and depression? *Journal of Clinical Child and Adolescent Psychology*, *34*(1), 49–61. doi: 10.1207/s15374424jccp3401_5

La Greca, A. M., Prinstein, M. J., & Fetter, M. D. (2001). Adolescent peer crowd affiliation: Linkages with health-risk behaviors and close friendships. *Journal of Pediatric Psychology*, *26*(3), 131–143. doi: 10.1093/jpepsy/26.3.131

Ladd, G. W., & Burgess, K. B. (1999). Charting the relationship trajectories of aggressive, withdrawn, and aggressive/withdrawn children during early grade school. *Child Development*, *70*(4), 910–929. doi: 10.1111/1467-8624.00066

Ladd, G. W., & Troop-Gordon, W. (2003). The role of chronic peer difficulties in the development of children's psychological adjustment problems. *Child Development*, *74*(5), 1344–1367.

Ladd, G. W., Kochenderfer, B. J., & Coleman, C. C. (1996). Friendship quality as a predictor of young children's early school adjustment. *Child Development*, *67*(3), 1103–1118.

Ladd, G. W., Kochenderfer-Ladd, B., Eggum, N. D., Kochel, K. P., & McConnell, E. M. (2011). Characterizing and comparing the friendships of anxious-solitary and unsociable preadolescents. *Child Development*, *82*(5), 1434–1453. doi: 10.1111/j.1467-8624.2011.01632.x

Lagerspetz, K. M., Björkqvist, K., & Peltonen, T. (1988). Is indirect aggression typical of females? Gender differences in aggressiveness in 11- to 12-year-old children. *Aggressive Behavior*, *14*(6), 403–414. doi: 10.1002/1098-2337

Laird, R. D., Pettit, G. S., Dodge, K. A., & Bates, J. E. (2005). Peer relationship antecedents of delinquent behavior in late adolescence: Is there evidence of demographic group differences in developmental processes? *Development and Psychopathology*, *17*(1), 127–144. doi: 10.1017/S0954579405050078

Landau, S., & Moore, L. A. (1991). Social skill deficits in children with attention-deficit hyperactivity disorder. *School Psychology Review*, *20*(2), 235–251.

Lansford, J. E., Costanzo, P. R., Grimes, C., Putallaz, M., Miller, S., & Malone, P. S. (2009). Social network centrality and leadership status: Links with problem behaviors and tests of gender differences. *Merrill-Palmer Quarterly*, *55*(1), 1–25. doi: 10.1353/mpq.0.0014

Lansford, J. E., Malone, P. S., Dodge, K. A., Pettit, G. S., & Bates, J. E. (2010). Developmental cascades of peer rejection, social information processing biases, and aggression during middle childhood. *Development and Psychopathology*, *22*(3), 593–602. doi: 10.1017/S0954579410000301

Larsen, H., Overbeek, G., Vermulst, A. A., Granic, I., & Engels, R. E. (2010). Initiation and continuation of best friends and adolescents' alcohol consumption: Do self-esteem and self-control function as moderators?. *International Journal of Behavioral Development*, *34*(5), 406–416. doi: 10.1177/0165025409350363

Larsen, H., van der Zwaluw, C. S., Overbeek, G., Granic, I., Franke, B., & Engels, R. E. (2010). A variable-number-of-tandem-repeats polymorphism in the dopamine D4 receptor gene affects social adaptation of alcohol use: Investigation of a gene–environment interaction. *Psychological Science*, *21*(8), 1064–1068.

Latendresse, S. J., Bates, J. E., Goodnight, J. A., Lansford, J. E., Budde, J. P., Goate, A. . . . Dick, D. M. (2011). Differential susceptibility to adolescent externalizing trajectories: Examining the interplay between CHRM2 and peer group antisocial behavior. *Child Development*, *82*(6), 1797–1814. doi: 10.1111/j.1467-8624.2011.01640.x

Laursen, B., & Hartup, W. W. (2002). The origins of reciprocity and social exchange in friendships. In B. Laursen & W. G. Graziano (Eds.), *Social exchange in development* (pp. 27–40). San Francisco, CA: Jossey-Bass.

Laursen, B., Bukowski, W. M., Aunola, K., & Nurmi, J. (2007). Friendship moderates prospective associations between social isolation and adjustment problems in young children. *Child Development*, *78*(4), 1395–1404. doi: 10.1111/j.1467-8624.2007.01072.x

Laursen, B., Hafen, C. A., Kerr, M., & Stattin, H. (2012). Friend influence over adolescent problem behaviors as a function of relative peer acceptance: To be liked is to be emulated. *Journal of Abnormal Psychology*, *121*(1), 88–94. doi: 10.1037/a0024707

Leadbeater, B. J., & Hoglund, W. L. (2009). The effects of peer victimization and physical aggression on changes in internalizing from first to third grade. *Child Development*, *80*(3), 843–859.

Lee, S. S. (2011). Deviant peer affiliation and antisocial behavior: Interaction with monoamine oxidase A (MAOA) genotype. *Journal of Abnormal Child Psychology*, *39*(3), 321–332. doi: 10.1007/s10802-010-9474-2

Lemerise, E. A., & Arsenio, W. F. (2000). An integrated model of emotion processes and cognition in social information processing. *Child Development*, *71*(1), 107–118. doi: 10.1111/1467-8624.00124

Lewin, L. M., Davis, B., & Hops, H. (1999). Childhood social predictors of adolescent antisocial behavior: Gender differences in predictive accuracy and efficacy. *Journal of Abnormal Child Psychology*, *27*(4), 277–292. doi: 10.1023/A:1022606608840

Lieberman, M., Gauvin, L., Bukowski, W. M., & White, D. R. (2001). Interpersonal influence and disordered eating behaviors in adolescent girls: The role of peer modeling, social reinforcement, and body-related teasing. *Eating Behaviors*, *2*(3), 215–236. doi: 10.1016/S1471-0153(01)00030-7

Lochman, J. E., & Wayland, K. K. (1994). Aggression, social acceptance, and race as predictors of negative adolescent outcomes. *Journal of the American Academy of Child & Adolescent Psychiatry*, *33*(7), 1026–1035. doi: 10.1097/00004583-199409000-00014

Logue, S., Chein, J., Gould, T., Holliday, E., & Steinberg, L. (2014). Adolescent mice, unlike adults, consume more alcohol in the presence of peers than alone. *Developmental Science*, *17*(1), 79–85. doi: 10.1111/desc.12101

Lynne-Landsman, S. D., Bradshaw, C. P., & Ialongo, N. S. (2010). Testing a developmental cascade model of adolescent substance use trajectories and young adult adjustment. *Development and Psychopathology*, *22*(4), 933–948. doi: 10.1017/S0954579410000556

MacDonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin*, *131*(2), 202–223. doi: 10.1037/0033-2909.131.2.202

Magnusson, D., & Stattin, H. (2006). The person in context: A holistic-interactionistic approach. In R. M. Lerner & W. Damon (Eds.), *Handbook of child psychology: Vol. 1, Theoretical models of human development* (6th ed., pp. 400–464). Hoboken, NJ: John Wiley & Sons Inc.

Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology*, *38*, 299–337. doi: 10.1146/annurev.ps.38.020187.001503

Masten, C. L., Eisenberger, N. I., Borofsky, L. A., McNealy, K., Pfeifer, J. H., & Dapretto, M. (2011). Subgenual anterior cingulate responses to peer rejection: A marker of adolescents' risk for depression. *Development and Psychopathology*, *23*(1), 283–292. doi: 10.1017/S0954579410000799

Masten, C. L., Eisenberger, N. I., Borofsky, L. A., Pfeifer, J. H., McNealy, K., Mazziotta, J. C., & Dapretto, M. (2009). Neural correlates of social exclusion during adolescence: Understanding the distress of peer rejection. *Social Cognitive and Affective Neuroscience*, *4*(2), 143–157. doi: 10.1093/scan/nsp007

Masten, C. L., Telzer, E. H., Fuligni, A. J., Lieberman, M. D., & Eisenberger, N. I. (2012). Time spent with friends in adolescence relates to less neural sensitivity to later peer rejection. *Social Cognitive and Affective Neuroscience*, *7*(1), 106–114. doi: 10.1093/scan/nsq098

Mathieson, L. C., Murray-Close, D., Crick, N. R., Woods, K. E., Zimmer-Gembeck, M., Geiger, T. C., & Morales, J. R. (2011). Hostile intent attributions and relational aggression: The moderating roles of emotional sensitivity, gender, and victimization. *Journal of Abnormal Child Psychology*, *39*(7), 977–987. doi: 10.1007/s10802-011-9515-5

Maxwell, K. A. (2002). Friends: The role of peer influence across adolescent risk behaviors. *Journal of Youth and Adolescence*, *31*(4), 267–277. doi: 10.1023/A:1015493316865

Mayeux, L., Sandstrom, M. J., & Cillessen, A. N. (2008). Is being popular a risky proposition? *Journal of Research on Adolescence*, *18*(1), 49–74. doi: 10.1111/j.1532-7795.2008.00550.x

McCord, J. (1992). The Cambridge-Somerville study: A pioneering longitudinal experimental study of delinquency prevention. In J. McCord & R. Tremblay (Eds.), *Preventing antisocial behavior: Interventions from birth through adolescence* (pp. 196–206). New York, NY: Guilford Press.

McLaughlin, K. A., Hatzenbuehler, M. L., & Hilt, L. M. (2009). Emotion dysregulation as a mechanism linking peer victimization to internalizing symptoms in adolescents. *Journal of Consulting and Clinical Psychology*, *77*(5), 894–904. doi: 10.1037/a0015760

Meaney, M. J. (2010). Epigenetics and the biological definition of gene × environment interactions. *Child Development*, *81*(1), 41–79.

Mercer, S. H., & DeRosier, M. E. (2010). Selection and socialization of internalizing problems in middle childhood. *Journal of Social and Clinical Psychology*, *29*(9), 1031–1056. doi: 10.1521/jscp.2010.29.9.1031

Mercken, L., Candel, M., Willems, P., & de Vries, H. (2007). Disentangling social selection and social influence effects on adolescent smoking: The importance of reciprocity in friendships. *Addiction*, *102*(9), 1483–1492. doi: 10.1111/j.1360-0443.2007.01905.x

Miller, A. H., Maletic, V., & Raison, C. L. (2009). Inflammation and its discontents: The role of cytokines in the pathophysiology of major depression. *Biological Psychiatry*, *65*(9), 732–741. doi: 10.1016/j.biopsych.2008.11.029

Miller, G. E., & Chen, E. (2010). Harsh family climate in early life presages the emergence of a proinflammatory phenotype in adolescence. *Psychological Science*, *21*(6), 848–856. doi: 10.1177/0956797610370161

Miller, G. E., & Cole, S. W. (2012). Clustering of depression and inflammation in adolescents previously exposed to childhood adversity. *Biological Psychiatry*, *72*(1), 34–40. doi: 10.1016/j.biopsych.2012.02.034

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*(6), 959–997. doi: 10.1037/a0024768

Miller, G. E., Chen, E., Fok, A. K., Walker, H., Lim, A., Nicholls, E. F., … Kobor, M. S. (2009). Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proceedings of the National Academy of Sciences*, *106*(34), 14716-14721.

Miller, G. E., Rohleder, N., & Cole, S. W. (2009). Chronic interpersonal stress predicts activation of pro- and anti-inflammatory signaling pathways 6 months later. *Psychosomatic Medicine*, *71*(1), 57–62. doi: 10.1097/PSY.0b013e318190d7de

Miller-Johnson, S., Winn, D.-M., Coie, J., Maumary–Gremaud, M., Hyman, C., Terry, R., & Lochman, J. (1999). Motherhood during the teen years: A developmental perspective on risk factors for childbearing. *Development and Psychopathology*, *11*(1), 85–100.

Monahan, K. C., Steinberg, L., & Cauffman, E. (2009). Affiliation with antisocial peers, susceptibility to peer influence, and antisocial behavior during the transition to adulthood. *Developmental Psychology*, *45*(6), 1520–1530. doi: 10.1037/a0017417

Moor, B., Güroğlu, B., Op de Macks, Z. A., Rombouts, S. B., Van der Molen, M. W., & Crone, E. A. (2012). Social exclusion and punishment of excluders: Neural correlates and developmental trajectories. *Neuroimage*, *59*(1), 708–717. doi: 10.1016/j.neuroimage.2011.07.028

Mrug, S., Molina, B. S., Hoza, B., Gerdes, A. C., Hinshaw, S. P., Hechtman, L., & Arnold, L. E. (2012). Peer rejection and friendships in children with attention-deficit/hyperactivity disorder: contributions to long-term outcomes. *Journal of Abnormal Child Psychology*, *40*(6), 1013–1026.

Mueller, E., & Brenner, J. (1977). The origins of social skills and interaction among playgroup toddlers. *Child Development*, *48*(3), 854–861. doi: 10.2307/1128334

Murphy, M. L., Slavich, G. M., Rohleder, N., & Miller, G. E. (2013). Targeted rejection triggers differential pro-and anti-inflammatory gene expression in adolescents as a function of social status. *Clinical Psychological Science*, *1*(1), 30–40.

Murray-Close, D. (2013). Psychophysiology of adolescent peer relations I: Theory and research findings. *Journal of Research on Adolescence*, *23*(2), 236–259. doi: 10.1111/j.1532-7795.2012.00828.x

Murray-Close, D., Hoza, B., Hinshaw, S. P., Arnold, L., Swanson, J., Jensen, P. S., & … Wells, K. (2010). Developmental processes in peer problems of children with attention-deficit/hyperactivity disorder in the multimodal treatment study of children with ADHD: Developmental cascades and vicious cycles. *Development and Psychopathology*, *22*(4), 785–802. doi: 10.1017/S0954579410000465

Nelson, S. E., & Dishion, T. J. (2004). From boys to men: Predicting adult adaptation from middle childhood sociometric status. *Development and Psychopathology*, *16*(2), 441–459.

Newcomb, A. F., & Bagwell, C. L. (1995). Children's friendship relations: A meta-analytic review. *Psychological Bulletin*, *117*(2), 306–347. doi: 10.1037/0033-2909.117.2.306

Newcomb, A. F., Bukowski, W. M., & Pattee, L. (1993). Children's peer relations: A meta-analytic review of popular, rejected, neglected, controversial, and average sociometric status. *Psychological Bulletin*, *113*(1), 99–128. doi: 10.1037/0033-2909.113.1.99

Nylund, K., Bellmore, A., Nishina, A., & Graham, S. (2007). Subtypes, severity, and structural stability of peer victimization: What does latent class analysis say? *Child Development*, *78*(6), 1706–1722.

O'Brien, L., Albert, D., Chein, J., & Steinberg, L. (2011). Adolescents prefer more immediate rewards when in the presence of their peers. *Journal of Research on Adolescence*, *21*(4), 747–753. doi: 10.1111/j.1532-7795.2011.00738.x

Oh, W., Rubin, K. H., Bowker, J. C., Booth-LaForce, C., Rose-Krasno, L., & Laursen, B. (2008). Trajectories of social withdrawal from middle childhood to early adolescence. *Journal of Abnormal Child Psychology*, *36*(4), 553–566. doi: 10.1007/s10802-007-9199-z

Ouellet-Morin, I., Danese, A., Bowes, L., Shakoor, S., Ambler, A., Pariante, C. M., … Arseneault, L. (2011). A discordant monozygotic twin design shows blunted cortisol reactivity among bullied children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *50*(6), 574–582.

Panak, W. F., & Garber, J. (1992). Role of aggression, rejection, and attributions in the prediction of depression in children. *Development and Psychopathology*, *4*(1), 145–165. doi: 10.1017/S0954579400005617

Parke, R. D., & Ladd, G. W. (1992). *Family–peer relationships: Modes of linkage*. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Parker, J. G., & Asher, S. R. (1987). Peer relations and later personal adjustment: Are low-accepted children at risk? *Psychological Bulletin*, *102*(3), 357–389. doi: 10.1037/0033-2909.102.3.357

Parker, J. G., & Asher, S. R. (1993). Friendship and friendship quality in middle childhood: Links with peer group acceptance and feelings of loneliness and social dissatisfaction. *Developmental Psychology*, *29*(4), 611–621. doi: 10.1037/0012-1649.29.4.611

Parker, J. G., Rubin, K. H., Erath, S. A., Wojslawowicz, J. C., & Buskirk, A. A. (2006). Peer relationships, child development, and adjustment: A developmental psychopathology perspective. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, Vol. 1: Theory and method* (2nd ed., pp. 419–493). Hoboken, NJ: John Wiley & Sons Inc.

Parkhurst, J. T., & Hopmeyer, A. (1998). Sociometric popularity and peer-perceived popularity two distinct dimensions of peer status. *Journal of Early Adolescence*, *18*(2), 125–144.

Patterson, G. R., Dishion, T. J., & Yoerger, K. (2000). Adolescent growth in new forms of problem behavior: Macro- and micro-peer dynamics. *Prevention Science*, *1*(1), 3–13. doi: 10.1023/A:1010019915400

Pedersen, S., Vitaro, F., Barker, E. D., & Borge, A. H. (2007). The timing of middle-childhood peer rejection and friendship: Linking early behavior to early-adolescent adjustment. *Child Development*, *78*(4), 1037–1051. doi: 10.1111/j.1467-8624.2007.01051.x

Perren, S., Ettekal, I., & Ladd, G. (2013). The impact of peer victimization on later maladjustment: mediating and moderating effects of hostile and self-blaming attributions. *Journal of Child Psychology and Psychiatry*, *54*(1), 46–55.

Perry, D. G., Hodges, E. E., & Egan, S. K. (2001). Determinants of chronic victimization by peers: A review and new model of family influence. In J. Juvonen & S. Graham (Eds.), *Peer harassment in school: The plight of the vulnerable and victimized* (pp. 73–104). New York, NY: Guilford Press.

Peters, E., Riksen-Walraven, J. M., Cillessen, A. H., & de Weerth, C. (2011). Peer rejection and HPA activity in middle childhood: Friendship makes a difference. *Child Development*, *82*(6), 1906–1920.

Pfeifer, J. H., & Allen, N. B. (2012). Arrested development? Reconsidering dual-systems models of brain function in adolescence and disorders. *Trends in Cognitive Sciences*, *16*(6), 322–329. doi: 10.1016/j.tics.2012.04.011

Piaget, J. (1932). *The moral judgment of children*. London, UK: Routledge & Kegan-Paul.

Piehler, T. F., & Dishion, T. J. (2007). Interpersonal dynamics within adolescent friendships: Dyadic mutuality, deviant talk, and patterns of antisocial behavior. *Child Development*, *78*(5), 1611–1624. doi: 10.1111/j.1467-8624.2007.01086.x

Pike, A., & Atzaba-Poria, N. (2003). Do sibling and friend relationships share the same temperamental origins? A twin study. *Journal of Child Psychology and Psychiatry*, *44*(4), 598–611. doi: 10.1111/1469-7610.00148

Poulin, F., & Chan, A. (2010). Friendship stability and change in childhood and adolescence. *Developmental Review*, *30*(3), 257–272. doi: 10.1016/j.dr.2009.01.001

Preciado, P., Snijders, T. B., Burk, W. J., Stattin, H., & Kerr, M. (2012). Does proximity matter? Distance dependence of adolescent friendships. *Social Networks*, *34*(1), 18–31.

Prentice, D. A. (2008). Mobilizing and weakening peer influence as mechanisms for changing behavior: Implications for alcohol intervention programs. In M. J. Prinstein & K. A. Dodge (Eds.), *Understanding peer influence in children and adolescents* (pp. 161–180). New York, NY: Guilford Press.

Prinstein, M. J. (2007). Moderators of peer contagion: A longitudinal examination of depression socialization between adolescents and their best friends. *Journal of Clinical Child and Adolescent Psychology*, *36*(2), 159–170. doi: 10.1080/15374410701274934

Prinstein, M. J., & Aikins, J. (2004). Cognitive moderators of the longitudinal association between peer rejection and adolescent depressive symptoms. *Journal of Abnormal Child Psychology*, *32*(2), 147–158. doi: 10.1023/B:JACP.0000019767.55592.63

Prinstein, M. J., & Cillessen, A. N. (2003). Forms and functions of adolescent peer aggression associated with high levels of peer status. *Merrill-Palmer Quarterly*, *49*(3), 310–342. doi: 10.1353/mpq.2003.0015

Prinstein, M. J., & Dodge, K. A. (2008). *Understanding peer influence in children and adolescents*. New York, NY: Guilford Press.

Prinstein, M. J., & La Greca, A. M. (2004). Childhood peer rejection and aggression as predictors of adolescent girls' externalizing and health risk behaviors: A 6-year longitudinal study. *Journal of Consulting and Clinical Psychology*, *72*(1), 103–112. doi: 10.1037/0022-006X.72.1.103

Prinstein, M. J., Borelli, J. L., Cheah, C. S. L., Simon, V. A., & Aikins, J. W. (2005). Adolescent girls' interpersonal vulnerability to depressive symptoms: A longitudinal examination of reassurance-seeking and peer relationships. *Journal of Abnormal Psychology*, *114*, 676–688.

Prinstein, M. J., Brechwald, W. A., & Cohen, G. L. (2011). Susceptibility to peer influence: Using a performance-based measure to identify adolescent males at heightened risk for deviant peer socialization. *Developmental Psychology*, *47*(4), 1167–1172. doi: 10.1037/a0023274

Prinstein, M. J., Cheah, C. S., & Guyer, A. E. (2005). Peer victimization, cue interpretation, and internalizing symptoms: Preliminary concurrent and longitudinal findings for children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, *34*(1), 11–24.

Prinstein, M. J., Choukas-Bradley, S. C., Helms, S. W., Brechwald, W. A., & Rancourt, D. (2011). High peer popularity longitudinally predicts adolescent health risk behavior, or does it? An examination of linear and quadratic associations. *Journal of Pediatric Psychology*, *36*(9), 980–990. doi: 10.1093/jpepsy/jsr053

Prinstein, M. J., Heilbron, N., Guerry, J. D., Franklin, J. C., Rancourt, D., Simon, V., & Spirito, A. (2010). Peer influence and nonsuicidal self injury: Longitudinal results in community and clinically-referred adolescent samples. *Journal of Abnormal Child Psychology*, *38*(5), 669–682. doi: 10.1007/s10802-010-9423-0

Prior, M., Smart, D., Sanson, A., & Oberklaid, F. (2000). Does shy-inhibited temperament in childhood lead to anxiety problems in adolescence? *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*(4), 461–468. doi: 10.1097/00004583-200004000-00015

Rabiner, D. L., Coie, J. D., Miller-Johnson, S., Boykin, A. S. M., & Lochman, J. E. (2005). Predicting the persistence of aggressive offending of African American males from adolescence into young adulthood. The importance of peer relations, aggressive behavior, and ADHD symptoms. *Journal of Emotional and Behavioral Disorders*, *13*(3), 131–140.

Rambaran, A. J., Dijkstra, J., & Stark, T. H. (2013). Status-based influence processes: The role of norm salience in contagion of adolescent risk attitudes. *Journal of Research on Adolescence*, *23*(3), 574–585. doi: 10.1111/jora.12032

Rancourt, D., & Prinstein, M. J. (2010). Peer status and victimization as possible reinforcements of adolescent girls' and boys' weight-related behaviors and cognitions. *Journal of Pediatric Psychology*, *35*(4), 354–367. doi: 10.1093/jpepsy/jsp067

Rancourt, D., Conway, C. C., Burk, W. J., & Prinstein, M. J. (2013). Gender composition of preadolescents' friendship groups moderates peer socialization of body change behaviors. *Health Psychology*, *32*(3), 283–292. doi: 10.1037/a0027980

Reijntjes, A., Kamphuis, J. H., Prinzie, P., & Telch, M. J. (2010). Peer victimization and internalizing problems in children: A meta-analysis of longitudinal studies. *Child Abuse & Neglect*, *34*(4), 244–252. doi: 10.1016/j.chiabu.2009.07.009

Reijntjes, A., Kamphuis, J. H., Prinzie, P., Boelen, P. A., van der Schoot, M., & Telch, M. J. (2011). Prospective linkages between peer victimization and externalizing problems in children: A meta-analysis. *Aggressive Behavior*, *37*(3), 215–222. doi: 10.1002/ab.20374

Roff, M. (1961). Childhood social interactions and young adult bad conduct. *Journal of Abnormal Social Psychology*, *63*, 333–337.

Rogosch, F. A., Cicchetti, D., & Aber, J. L. (1995). The role of child maltreatment in early deviations in cognitive and affective processing abilities and later peer relationship problems. *Development and Psychopathology*, *7*(4), 591–609. doi: 10.1017/S0954579400006738

Rose, A. J. (2002). Co-rumination in the friendships of girls and boys. *Child Development*, *73*(6), 1830–1843. doi: 10.1111/1467-8624.00509

Rose, A. J., Carlson, W., & Waller, E. M. (2007). Prospective associations of co-rumination with friendship and emotional adjustment: Considering the socioemotional trade-offs of co-rumination. *Developmental Psychology*, *43*(4), 1019–1031. doi: 10.1037/0012-1649.43.4.1019

Ross, L., Greene, D., & House, P. (1977). The false consensus effect: An egocentric bias in social perception and attribution processes. *Journal of Experimental Social Psychology*, *13*(3), 279–301. doi: 10.1016/0022-1031(77)90049-X

Rubin, K. H., Bowker, J. C., & Kennedy, A. E. (2009a). Avoiding and withdrawing from the peer group. In K. H. Rubin, W. M. Bukowski & B. Laursen (Eds.), *Handbook of peer interactions, relationships, and groups* (pp. 303–321). New York, NY: Guilford Press.

Rubin, K. H., Bowker, J., & Gazelle, H. (2010). Social withdrawal in childhood and adolescence: Peer relationships and social competence. In K. H. Rubin & R. J. Coplan (Eds.), *The development of shyness and social withdrawal* (pp. 131–156). New York, NY: Guilford Press.

Rubin, K. H., Burgess, K. B., & Hastings, P. D. (2002). Stability and social-behavioral consequences of toddlers' inhibited temperament and parenting behaviors. *Child Development*, *73*(2), 483–495. doi: 10.1111/1467-8624.00419

Rubin, K. H., Chen, X., & Hymel, S. (1993). Socioemotional characteristics of withdrawn and aggressive children. *Merrill-Palmer Quarterly*, *39*(4), 518–534.

Rubin, K. H., Coplan, R. J., & Bowker, J. C. (2009). Social withdrawal in childhood. *Annual Review of Psychology*, *60*, 141–171. doi: 10.1146/annurev.psych.60.110707.163642

Rubin, K. H., Wojslawowicz, J. C., Rose-Krasnor, L., Booth-LaForce, C., & Burgess, K. B. (2006). The best friendships of shy/withdrawn children: Prevalence, stability, and relationship quality. *Journal of Abnormal Child Psychology*, *34*(2), 143–157. doi: 10.1007/s10802-005-9017-4

Rudolph, K. D., & Clark, A. G. (2001). Conceptions of relationships in children with depressive and aggressive symptoms: Social-cognitive distortion or reality? *Journal of Abnormal Child Psychology*, *29*(1), 41–56. doi: 10.1023/A:1005299429060

Rudolph, K. D., Hammen, C., Burge, D., Lindberg, N., Herzberg, D., & Daley, S. E. (2000). Toward an interpersonal life-stress model of depression: The developmental context of stress generation. *Development and Psychopathology*, *12*(2), 215–234. doi: 10.1017/S0954579400002066

Rudolph, K. D., Lansford, J. E., Agoston, A. M., Sugimura, N., Schwartz, D., Dodge, K. A., … Bates, J. E. (2014). Peer victimization and social alienation: Predicting deviant peer affiliation in middle school. *Child Development*, *85*(1), 124–139. doi: 10.1111/cdev.12112

Rudolph, K. D., Troop-Gordon, W., & Granger, D. A. (2011). Individual differences in biological stress responses moderate the contribution of early peer victimization to subsequent depressive symptoms. *Psychopharmacology*, *214*(1), 209–219. doi: 10.1007/s00213-010-1879-7

Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene–environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry*, *47*(3–4), 226–261. doi: 10.1111/j.1469-7610.2005.01557.x

Salmivalli, C., & Peets, K. (2009). Bullies, victims, and bully-victim relationships in middle childhood and early adolescence. In K. H. Rubin, W. M. Bukowski, & B. Laursen (Eds.), *Handbook of peer interactions, relationships, and groups* (pp. 322–340). New York, NY: Guilford Press.

Sameroff, A. J. (2000). Developmental systems and psychopathology. *Development and Psychopathology*, *12*(3), 297–312. doi: 10.1017/S0954579400003035

Schwartz, C. E., Kunwar, P. S., Greve, D. N., Kagan, J. J., Snidman, N. C., & Bloch, R. B. (2012). A phenotype of early infancy predicts reactivity of the amygdala in male adults. *Molecular Psychiatry*, *17*(10), 1042–1050. doi: 10.1038/mp.2011.96

Schwartz, D., Lansford, J. E., Dodge, K. A., Pettit, G. S., & Bates, J. E. (2013). The link between harsh home environments and negative academic trajectories is exacerbated by victimization in the elementary school peer group. *Developmental Psychology*, *49*(2), 305–316. doi: 10.1037/a0028249

Schwartz, D. (2000). Subtypes of victims and aggressors in children's peer groups. *Journal of Abnormal Child Psychology*, *28*(2), 181–192. doi: 10.1023/A:1005174831561

Schwartz, D., McFadyen-Ketchum, S. A., Dodge, K. A., Pettit, G. S., & Bates, J. E. (1998). Peer group victimization as a predictor of children's behavior problems at home and in school. *Development and Psychopathology*, *10*(1), 87–99. doi: 10.1017/S095457949800131X

Simon, V. A., Aikins, J., & Prinstein, M. J. (2008). Romantic partner selection and socialization during early adolescence. *Child Development*, *79*(6), 1676–1692. doi: 10.1111/j.1467-8624.2008.01218.x

Slavich, G. M., & Cole, S. W. (2013). The emerging field of human social genomics. *Clinical Psychological Science*, *1*, 331–348. doi: 10.1177/2167702613478594

Slavich, G. M., & Irwin, M. R. (2014). From stress to inflammation and major depressive disorder: A social signal transduction theory of depression. *Psychological Bulletin*, *140*(3), 774–815. doi: 10.1037/a0035302

Slavich, G. M., O'Donovan, A., Epel, E. S., & Kemeny, M. E. (2010). Black sheep get the blues: A psychobiological model of social rejection and depression. *Neuroscience & Biobehavioral Reviews*, *35*(1), 39–45.

Slavich, G. M., Way, B. M., Eisenberger, N. I., & Taylor, S. E. (2010). Neural sensitivity to social rejection is associated with inflammatory responses to social stress. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, *107*(33), 14817–14822. doi: 10.1073/pnas.1009164107

Smith, P. K., Mahdavi, J., Carvalho, M., Fisher, S., Russell, S., & Tippett, N. (2008). Cyberbullying: Its nature and impact in secondary school pupils. *Journal of Child Psychology and Psychiatry*, *49*(4), 376–385.

Snijders, T. B., van de Bunt, G. G., & Steglich, C. G. (2010). Introduction to stochastic actor-based models for network dynamics. *Social Networks*, *32*(1), 44–60. doi: 10.1016/j.socnet.2009.02.004

Snijders, T., Steglich, C., & Schweinberger, M. (2007). Modeling the Coevolution of Networks and Behavior. In K. van Montfort, J. Oud, & A. Satorra (Eds.), *Longitudinal models in the behavioral and related sciences* (pp. 41–71). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Snyder, J. J., Schrepferman, L. P., Bullard, L., McEachern, A. D., & Patterson, G. R. (2012). Covert antisocial behavior, peer deviancy training, parenting processes, and sex differences in the development of antisocial behavior during childhood. *Development and Psychopathology*, *24*(3), 1117–1138. doi: 10.1017/S0954579412000570

Snyder, J., Brooker, M., Patrick, M., Snyder, A., Schrepferman, L., & Stoolmiller, M. (2003). Observed peer victimization during early elementary school: Continuity, growth, and relation to risk for child antisocial and depressive behavior. *Child Development*, *74*(6), 1881–1898. doi: 10.1046/j.1467-8624.2003.00644.x

Soenens, B., Vansteenkiste, M., & Niemiec, C. P. (2009). Should parental prohibition of adolescents' peer relationships be prohibited? *Personal Relationships*, *16*(4), 507–530. doi: 10.1111/j.1475-6811.2009.01237.x

Solberg, M. E., & Olweus, D. (2003). Prevalence estimation of school bullying with the Olweus Bully/Victim Questionnaire. *Aggressive Behavior*, *29*(3), 239–268. doi: 10.1002/ab.10047

Somerville, L. H. (2013). The teenage brain: Sensitivity to social evaluation. *Current Directions in Psychological Science*, *22*(2), 121–127. doi: 10.1177/0963721413476512

Sroufe, L. (1997). Psychopathology as an outcome of development. *Development and Psychopathology*, *9*(02), 251–268.

Steglich, C., Snijders, T. A., & Pearson, M. (2010). Dynamic networks and behavior: Separating selection from influence. *Sociological Methodology*, *40*(1), 329–393.

Steinberg, L., & Monahan, K. C. (2007). Age differences in resistance to peer influence. *Developmental Psychology*, *43*, 1531–1543. doi: 10.1037/0012-1649.43.6.1531

Stevens, E. A., & Prinstein, M. J. (2005). Peer contagion of depressogenic attributional styles among adolescents: A longitudinal study. *Journal of Abnormal Child Psychology*, *33*(1), 25–37. doi: 10.1007/s10802-005-0931-2

Stewart, S. L., & Rubin, K. H. (1995). The social problem-solving skills of anxious-withdrawn children. *Development and Psychopathology*, *7*(2), 323–336. doi: 10.1017/S0954579400006532

Stone, L. B., Hankin, B. L., Gibb, B. E., & Abela, J. Z. (2011). Co-rumination predicts the onset of depressive disorders during adolescence. *Journal of Abnormal Psychology*, *120*(3), 752–757. doi: 10.1037/a0023384

Stone, L. B., Uhrlass, D. J., & Gibb, B. E. (2010). Co-rumination and lifetime history of depressive disorders in children. *Journal of Clinical Child and Adolescent Psychology*, *39*(4), 597–602. doi: 10.1080/15374416.2010.486323

Strauss, C. C., Forehand, R., Smith, K., & Frame, C. L. (1986). The association between social withdrawal and internalizing problems of children. *Journal of Abnormal Child Psychology*, *14*(4), 525–535. doi: 10.1007/BF01260521

Sturaro, C., van Lier, P. C., Cuijpers, P., & Koot, H. M. (2011). The role of peer relationships in the development of early school-age externalizing problems. *Child Development*, *82*(3), 758–765. doi: 10.1111/j.1467-8624.2010.01532.x

Sullivan, H. (1953). *The interpersonal theory of psychiatry*. New York, NY: W.W. Norton & Co.

Sumter, S. R., Baumgartner, S. E., Valkenburg, P. M., & Peter, J. (2012). Developmental trajectories of peer victimization: Off-line and online experiences during adolescence. *Journal of Adolescent Health*, *50*(6), 607–613. doi: 10.1016/j.jadohealth.2011.10.251

Szyf, M., & Bick, J. (2013). DNA methylation: A mechanism for embedding early life experiences in the genome. *Child Development*, *84*(1), 49–57. doi: 10.1111/j.1467-8624.2012.01793.x

Tilton-Weaver, L. C., Burk, W. J., Kerr, M., & Stattin, H. (2013). Can parental monitoring and peer management reduce the selection or influence of delinquent peers? Testing the question using a dynamic social network approach. *Developmental Psychology*, *49*(11), 2057. doi: 10.1037/a0031854

Troop-Gordon, W., & Ladd, G. W. (2005). Trajectories of peer victimization and perceptions of the self and schoolmates: Precursors to internalizing and externalizing problems. *Child Development*, *76*(5), 1072–1091.

Underwood, M. K., Kupersmidt, J. B., & Coie, J. D. (1996). Childhood peer sociometric status and aggression as predictors of adolescent childbearing. *Journal of Research on Adolescence*, *6*, 201–23.

Urberg, K. A., Luo, Q., Pilgrim, C., & Degirmencioglu, S. M. (2003). A two-stage model of peer influence in adolescent substance use: Individual and relationship-specific differences in susceptibility to influence. *Addictive Behaviors*, *28*(7), 1243–1256. doi: 10.1016/S0306-4603(02)00256-3

Vaillancourt, T., Brittain, H. L., McDougall, P., & Duku, E. (2013). Longitudinal links between childhood peer victimization, internalizing and externalizing problems, and academic functioning: Developmental cascades. *Journal of Abnormal Child Psychology*, *41*(8), 1203–1215. doi: 10.1007/s10802-013-9781-5

van Lier, P., Boivin, M., Dionne, G., Vitaro, F., Brendgen, M., Koot, H., . . . Perusse, D. (2007). Kindergarten children's genetic vulnerabilities interact with friends' aggression to promote children's own aggression. *Journal of the American Academy of Child & Adolescent Psychiatry*, *46*(8), 1080–1087.

van Lier, P. A., Vitaro, F., Barker, E. D., Brendgen, M., Tremblay, R. E., & Boivin, M. (2012). Peer victimization, poor academic achievement, and the link between childhood externalizing and internalizing problems. *Child Development*, *83*(5), 1775–1788.

van Lier, P. C., & Koot, H. M. (2010). Developmental cascades of peer relations and symptoms of externalizing and internalizing problems from kindergarten to fourth-grade elementary school. *Development and Psychopathology*, *22*(3), 569–582. doi: 10.1017/S0954579410000283

van Workum, N., Scholte, R. J., Cillessen, A. N., Lodder, G. A., & Giletta, M. (2013). Selection, deselection, and socialization processes of happiness in adolescent friendship networks. *Journal of Research on Adolescence*, *23*(3), 563–573. doi: 10.1111/jora.12035

Van Zalk, M., Kerr, M., Branje, S. T., Stattin, H., & Meeus, W. J. (2010). It takes three: Selection, influence, and de-selection processes of depression in adolescent friendship networks. *Developmental Psychology*, *46*(4), 927–938. doi: 10.1037/a0019661

Vandell, D. L., Wilson, K. S., & Buchanan, N. R. (1980). Peer interaction in the first year of life: An examination of its structure, content, and sensitivity to toys. *Child Development*, *51*(2), 481–488. doi: 10.2307/1129282

Vanhalst, J., Luyckx, K., & Goossens, L. (2014). Experiencing loneliness in adolescence: A matter of individual characteristics, negative peer experiences, or both? *Social Development*, *23*, 100–118. doi: 10.1111/sode.12019

Vaughn, B. E., & Waters, E. (1981). Attention structure, sociometric status, and dominance: Interrelations, behavioral correlates, and relationships to social competence. *Developmental Psychology*, *17*(3), 275–288. doi: 10.1037/0012-1649.17.3.275

Veenstra, R., Dijkstra, J., Steglich, C., & Van Zalk, M. W. (2013). Network–behavior dynamics. *Journal of Research on Adolescence*, *23*(3), 399–412. doi: 10.1111/jora.12070

Vitaro, F., Brendgen, M., & Tremblay, R. E. (2000). Influence of deviant friends on delinquency: Searching for moderator variables. *Journal of Abnormal Child Psychology*, *28*(4), 313–325. doi: 10.1023/A:1005188108461

Vitaro, F., Gendreau, P. L., Tremblay, R. E., & Oligny, P. (1998). Reactive and proactive aggression differentially predict later conduct problems. *Journal of Child Psychology and Psychiatry*, *39*(3), 377–385. doi: 10.1017/S0021963097002102

Vygotsky, L. S. (1981). The genesis of higher mental functions. *Concept of Activity in Soviet Psychology*, 144–188.

Wasserstein, S. B., & La Greca, A. M. (1996). Can peer support buffer against behavioral consequences of parental discord? *Journal of Clinical Child Psychology*, *25*(2), 177–182. doi: 10.1207/s15374424jccp2502_6

Weaver, I. G., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., . . . Meaney, M. J. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience*, *7*(8), 847–854. doi: 10.1038/nn1276

Weigard, A., Chein, J., Albert, D., Smith, A., & Steinberg, L. (2014). Effects of anonymous peer observation on adolescents' preference for immediate rewards. *Developmental Science*, *17*(1), 71–78. doi: 10.1111/desc.12099

Werner, N. E., & Crick, N. R. (2004). Maladaptive peer relationships and the development of relational and physical aggression during middle childhood. *Social Development*, *13*(4), 495–514. doi: 10.1111/j.1467-9507.2004.00280.x

Woodward, L. J., & Fergusson, D. M. (1999). Childhood peer relationship problems and psychosocial adjustment in late adolescence. *Journal of Abnormal Child Psychology*, *27*(1), 87–104. doi: 10.1023/A:1022618608802

Zettergren, P., Bergman, L. R., & Wångby, M. (2006). Girls' stable peer status and their adulthood adjustment: A longitudinal study from age 10 to age 43. *International Journal of Behavioral Development*, *30*(4), 315–325. doi: 10.1177/0165025406072793

Zwierzynska, K., Wolke, D., & Lereya, T. S. (2013). Peer victimization in childhood and internalizing problems in adolescence: A prospective longitudinal study. *Journal of Abnormal Child Psychology*, *41*(2), 309–323. doi: 10.1007/s10802-012-9678-8

CHAPTER 13

Family Systems from a Developmental Psychopathology Perspective

PATRICIA K. KERIG

INVITING FAMILY SYSTEMS THEORY TO THE TABLE 580
HISTORICAL ORIGINS AND THEORETICAL UNDERPINNINGS OF FAMILY SYSTEMS THEORIES 581
Progenitors of Family Systems Theory 581
Bowen Family System Theory 582
The MRI Group 582
Borzormenyi-Nagy's Contextual Approach 583
Minuchin's Structural Family Theory 583
The Functional Family Perspective 584
Social Learning Perspectives on the Family 585
Summary 585
WHAT MAKES A FAMILY THEORY SYSTEMIC? 586
Holism 586
The Effects of Relationships on Relationships 587
Circularity 588
Homeostasis 588
Families as Organized Systems 588
Boundaries and Subsystems 589
Families as Open Systems 590
Change Processes in Family Systems 591
PSYCHOPATHOLOGY AND ITS CLASSIFICATION FROM A FAMILY SYSTEMS PERSPECTIVE 593
Inclusion of Family Factors in Existing Diagnostic Systems 593
Alternative Approaches to Diagnosis from a Systemic Perspective 595
CONVERGENCE AND DIVERGENCE BETWEEN FAMILY SYSTEMS AND DEVELOPMENTAL PSYCHOPATHOLOGY 597
Points of Divergence 597
Points of Convergence 597
Toward an Emergent Synthesis: What Can Family Systems Theory Offer Developmental Psychopathology, and Vice Versa? 597
CHALLENGES TO FAMILY SYSTEMS THEORY 601
Bringing Fathers into the Fold 601
Feminist Critiques of Family Systems Theory 601
Cultural and Ethnic Diversity in Family Systems 601
Implications of Cultural Differences in Family Dynamics for Psychopathology 603
Historical Changes Affecting the Family System 603
What Is a Family? Diversity in Family Structures and Composition 604
FAMILY SYSTEMIC RESEARCH METHODS 606
Self-Reports of Family Processes 606
Observational Coding 607
Analytic Strategies in Family Observational Research 610
EMPIRICAL INVESTIGATIONS OF FAMILY SYSTEMS CONSTRUCTS 613
Global Family Qualities 613
Spillover of Marriage onto Parenting and Family Process 615
Triangulation 615
Coparenting Conflict and Family Process 617
Boundary Dissolution 618
GENDER DYNAMICS IN FAMILY PROCESS 619
Fathering and Spillover 619
Variations in Family Processes Related to Child Gender 620
TOWARD THE FUTURE: GROWTH POINTS FOR DEVELOPMENTAL PSYCHOPATHOLOGY FROM A FAMILY SYSTEMS PERSPECTIVE 621
Incorporation of Biological Processes into Research on Family Systems 621
Methodological Advances with Relevance for Family Research 621
CONCLUDING THOUGHTS 622
REFERENCES 622

INVITING FAMILY SYSTEMS THEORY TO THE TABLE

Almost two decades have passed since, in the first edition of this volume, Wagner and Reiss (1995) pondered whether the relationship between family systems theory and developmental psychopathology could best be characterized as a "courtship, marriage, or divorce." Despite some thoughtful steps toward fostering integration between these fields (e.g., Cicchetti & Howes, 1991; Cox & Paley, 2003; Cummings, Davies, & Campbell, 2000; Davies & Cicchetti, 2004; Howes & Cicchetti, 1993), on the whole, it seems fair

to say that systemic thinking has not been fully embraced within the developmental psychopathology literature. The current relationship between the two bodies of work might be better captured by the metaphor of the estranged relative who, although invited to the feast and even heartily welcomed by some, nevertheless has no place-marker at the table and stands slightly outside the family circle.

Although certainly research in developmental psychopathology in the present day shows no lack of appreciation for family-related variables, and many if not most interventions for childhood psychopathology include attention to the family, relatively little of this work can be said to derive from family *systemic* constructs. In that regard, in the second edition of this volume, Philip and Carolyn Cowan (2006) made a helpful distinction to which we would offer the friendly amendment of rephrasing as a *family variables* approach versus a *family systems* perspective. The family variables approach is a long-standing and well-integrated aspect of traditional research in abnormal child psychology as well as in developmental psychopathology. This approach focuses on identifying risk and protective factors within the family that play a role in the etiology of psychological problems, protection against them, or their amelioration in treatment. For example, poverty, domestic violence, and harsh parenting are all family-relevant variables that increase the likelihood of maladaptation, just as interventions that promote positive development typically go beyond the individual child to include the family. However, as Cowan and Cowan (2006) pointed out, inherent in the family variables approach is that each of these family influences on childhood psychopathology, whether viewed individually or in combination, is conceptualized as having a unidirectional effect on the individual. In contrast, a *family systems* approach considers not the individual but the entire system of interrelationships within the family to be the object of study or the target of treatment. Moreover, it is in the structure, the patterns, and the dynamic and reciprocal transactions in that system of relationships that maladaptation and adaptation arise. Given that the inclusion of family variables permeates virtually all research in childhood psychopathology, a comprehensive review of family variables research would entail an unwieldy compilation of studies on the topics spanning all three volumes of the present anthology. By the same token, the majority of families are composed of parents and children (although there are notable exceptions, such as sibling-headed households), and thus any and all of the voluminous research regarding parent–child relations could said to be relevant to the study of the *family*. Consequently, to maintain a sharper focus for the present chapter, this review will be specific to a discussion of the historical underpinnings of and current state of research regarding family systems perspectives as here defined.

HISTORICAL ORIGINS AND THEORETICAL UNDERPINNINGS OF FAMILY SYSTEMS THEORIES

Progenitors of Family Systems Theory

In the United States of the mid-1950s and early 1960s, a number of groups of researchers and clinicians independently began to raise objections to the prevailing psychoanalytic perspective and its emphasis on psychopathology as an individual phenomenon, promoting instead the idea that dysfunction might occur at the level of the family system. Nathan Ackerman (1962), head of the institute in New York later to bear his name, was an early proponent of working conjointly with parents and children in treatment, although his approach continued to show its psychoanalytic roots in focusing on the individual psychopathology of each of the family members in the room. Around the same time, Theodore Lidz and his colleagues (Lidz, Cornelison, Fleck, & Terry, 1957) at the Yale Psychiatric Institute took on as their target a particularly hurtful legacy of psychoanalytic dogma, that psychosis originated with the *schizophrenic mother* (Fromm-Reichmann, 1948). Novel in their approach was bringing fathers into the room and examining ways disorder was demonstrated in multiple relationships in the family—not just between mother and child but also between father and child as well as the members of the couple. This team made the important observation that in many cases the source of the problem was in the marital relationship, whether as a function of high levels of unresolved conflict (*marital schism*) or conflict that was denied and subterranean, expressing itself in covert ways such as overt agreement followed by invalidation or undermining of the partner (*marital skew*). Another suggestion that these investigators offered that has ramifications for developmental psychopathology is that these problematic family patterns could be discerned across three generations, from parents to children to grandchildren.

Schizophrenia also was the focus of work going on simultaneously in the lab of Lyman Wynne and his colleagues (Singer & Wynne, 1965; Wynne, Ryckoff, Day, & Hirsch, 1958) at the National Institutes of Mental Health in Washington, DC. Their seminal study involved a demonstration that disordered psychological processing was more evident among individuals with schizophrenia when they were involved in interaction with family

members than when they were alone. Therefore, the disorder was seen as residing not in the individual but in the family system. The innovative method the researchers used to reveal these processes (asking family members to come to a consensus regarding responses to the Rorschach inkblot test) provided a model for many subsequent studies utilizing family observational methods in which family members have been challenged to come to a mutual agreement on a task (e.g., completing a questionnaire, planning a meal, designing an ideal vacation; see Kerig & Lindahl, 2001).

Bowen Family System Theory

Also at the National Institute of Mental Health during this time period was Murray Bowen (1961), whose initial observations emphasized the importance of poor psychological boundaries among family members of patients with schizophrenia, whom he described as evidencing an undifferentiated ego mass or fusion. Consequently, the *differentiation of self* and its extrication from the family groupthink became central to Bowen's notions of psychological health. Another of Bowen's contributions that has become pivotal in family systems theory is his articulation of the idea of the *triangle* as a basic building block of family interaction (Bowen, 1978; Kerr & Bowen, 1988). Bowen (1978) offered the insight that a relational system comprised of two persons was inherently unstable and "easily disturbed by emotional forces within it and from outside" (p. 135, particularly when the participants in that relationship were poorly differentiated from one another. To reduce this anxiety, therefore, the two-person constellation naturally gravitates toward including a third person in its orbit. The resulting triangular system is a more stable one, and one that serves the function of diffusing anxiety and preventing any particular relationship from becoming overheated.

> There are several ways a twosome can incorporate a third person into its tension. The uncomfortable insider (A) can pull the outsider (C) into the situation through complaints to him about the other insider (B). If C responds sympathetically, taking sides with A, a comfortable closeness (based on undifferentiation) is established between A and C. B is the new outsider ... Or a third person may play a very active role in getting himself in the middle of a problem between two others. Through years of "training," such a person has learned to gravitate toward the disharmony he senses in others, regardless of whether an "invitation" is actually extended. A poorly differentiated child often occupies this type of position with his parents. He predictably makes himself a problem whenever tension reaches a certain level between his parents. This draws one or both parents' focus to him, thus reducing tension between them (pp. 136–137).

Intriguingly, Bowen (1978) also offered the observation that a live person need not be available for the establishment of a triangle—fantasied relationships, objects, even pets can be mustered to serve the purpose of creating a third leg to maintain the emotional equilibrium of the family stool. Further, although the term triangulation currently tends to be used to refer exclusively to pathological processes in families, Bowen abjured such value judgments, instead construing triangles as natural. The third party's participation in the relationship was seen not as inherently problematic but as having the potential to be helpful, so long as the third member of the triangle remained simultaneously emotionally connected to both parties while also emotionally detached from their conflict. His observations of the processes involved in detriangling—"probably the most important technique in family systems therapy" (p. 150)—likewise emphasized a therapeutic stance of nonjudgmental emotional neutrality along with the capacity to see relationship problems from multiple points of view. "To observe triangles, it is necessary to see 'past' the symptoms to the underlying emotional process: the interplay of individuality and togetherness and the impact of anxiety on that interplay. To observe triangles, it is also necessary to recognize the influence on one's perceptions of one's own emotional reactivity to the system" (p. 151).

Nevertheless, in Bowen's theory triangles are implicated in the development of psychopathology, particularly because symptoms may serve a function within the triangular system of relationships. For example, becoming symptomatic may serve to bring family members closer when one has been made an outsider in the family system. Bowen also suggested that parents may attempt to triangulate children in their conflicts by projecting the problem on to them. This *family projection process,* he proposed, is precipitated by marital fusion. When a lack of differentiation between parents evokes anxiety, they may seek to alleviate this tension by turning to the child.

The MRI Group

Another of the seminal teams of family systems thinkers of this period was based at the Mental Research Institute (MRI) in Palo Alto and included experts from the fields of psychiatry (Don Jackson), anthropology (Gregory Bateson), social work (Virginia Satir), and communications theory (Jay Haley, John Weakland, and Paul Watzlawick). Again focusing on schizophrenia, the MRI group proposed the idea that distorted communication processes in the family were responsible for the disorder. In particular, they originated the idea of the *double bind*, in which family

members (and here, unfortunately, the theory tended to backslide to targeting *schizophrenogenic* mothers) confronted the patient with irreconcilably contradictory messages or commands such that following either would constitute an act of disobedience or a violation of family rules. Moreover, one of the family rules that ensnared children in those binds was the rule that the bind was not to be acknowledged or spoken about openly, just as another was that the child was not allowed to exit the scene. Therefore, the child was left in an emotionally and cognitively impossible situation that was, literally, crazy-making. Although highly influential, the theory was not able to be empirically validated because the binds referred to were so subtle (e.g., a lack of correspondence between content of speech and tone of voice) that observers were not able to agree on their presence or code them reliably (Ringuette & Kennedy, 1966).

Perhaps the most significant legacy of the MRI group to modern family systems theory is the overarching conceptual framework that Bateson contributed, based on the philosopher Bertalanffy's (1973) general systems theory, which he integrated with game theory and cybernetics. From general systems theory came the idea of organic systems as organized and open to change, but also as self-regulating and striving toward balance by the use of internal mechanisms that serve to achieve a self-righting function when the organism undergoes an excess of change or stasis. From cybernetics and the study of nonorganic systems, Bateson derived the idea of homeostasis, which suggests that systems are in fact resistant to change, with internal mechanisms that act like thermostats which regulate a return to a prior state after disruption has occurred. This theoretical groundwork subsequently inspired the development of a number of approaches to family therapy that can be understood as offshoots of the MRI approach, including Haley's (1976) strategic approach and the therapeutic formulations of the Milan group in Italy (Palazzoli, Boscolo, Cecchin, & Prata, 1978).

Borzormenyi-Nagy's Contextual Approach

Another influential school of thought in the family systems pantheon was housed at the Family Institute of Philadelphia, led by Ivan Borzormenyi-Nagy (1962). Although maintaining a psychodynamically informed focus on the individual within the family system, Borzormenyi-Nagy brought to the forefront the ways family systemic processes comprised a legacy spanning multiple generations. In their seminal text, *Invisible Loyalties,* Borzormenyi-Nagy and Spark (1973) introduced a dynamic that has become a centerpiece of a significant amount of research in family systems theory, that of *parentification*, also termed role-reversal, between parent and child. "By definition, parentification implies the subjective distortion of a relationship as if one's partner or even children were his parent" (p. 151). Although such dynamics might reflect or precipitate problems in the family, Borzormenyi-Nagy and Spark emphasized that they were normative relational processes that weren't necessarily associated with maladaptation. In fact, they proposed, parentification might play a function within the family relational system that was associated with adaptivity. Although it might seem improper and unethical for a parent to abdicate responsibilities or seek caregiving from a child, the young person who successfully takes on those roles serves an important function by normalizing the family and enabling it to remain intact. Thus, although perhaps at a personal sacrifice of the nurturance and care usually provided a child, the parentified youth helps to meet the needs of the family and its members, therefore fulfilling an overarching fundamental need of his or her own for an intact and operational family system. In this view, parentification only becomes problematic when children's contributions to bolstering parental well-being are unfair, unacknowledged, unappreciated, or unreciprocated. Nonetheless, Borzormenyi-Nagy and Spark also clearly associated parentification with pathological processes in families, processes that were passed on across generations. Adults who had been leaned upon too heavily by their own parents, they suggested, were those most likely turn to their own children in order to fulfill those unmet needs for nurturance and care. In effect, this account-due or debit perspective suggests that a parent who was deprived of nurturance in childhood will feel entitled to balance the ledger by extracting those provisions from the emotional stores of their own offspring.

Minuchin's Structural Family Theory

Another major architect of family systems theory was Salvador Minuchin, who established the Philadelphia Child Guidance Clinic (PCGC) for the training of family therapists and wrote some of the most influential conceptual and clinical works on understanding and treating psychopathology from a family systems perspective. Minuchin and colleagues' (1967) first book, *Families of the Slums,* was a groundbreaking description of clinical work with a population of disadvantaged families who were rarely the subject of more traditional psychoanalytic treatment. His subsequent book, *Families and Family Therapy* (Minuchin, 1974) became one of the most widely cited

textbooks in family systems theory and intervention techniques. In this work the PCGC team literally drew maps of family dynamic processes to reveal the ways problems in family structure contributed to psychological dysfunction.

Minuchin's structural family perspective built upon the earlier groundwork laid by Bowen (1978) and Haley (1977) regarding the importance of maintaining appropriate boundaries around the subsystems in the family. As Minuchin (1974) suggests, "The family is the matrix of its members' psychosocial development" (p. 48), and an essential part of that matrix is the opportunity the family provides its members to experience simultaneously a sense of belonging as well as of separateness. This occurs naturally through the subsystems that comprise the family, connecting some members to one another while differentiating them from others. In particular, clear boundaries should separate the *parental subsystem*, which defines the executive and complementary roles played by the parents, from the *parent–child subsystems*, which are defined by hierarchical and nonreciprocal caregiving roles, and the *sibling subsystem*, in which brothers and sisters share the same status as children but also are differentiated by age. What allows these subsystems to function adaptively is the presence of clear boundaries between them. "For proper family functioning, the boundaries of subsystems must be clear. Boundaries must be defined well enough to allow subsystem members to carry out their functions without undue interference, but they must allow contact between the members of the subsystem and others" (p. 54). Thus, in Minuchin's perspective, boundaries could be problematic at either extreme; they could be overly *diffuse*, resulting in enmeshed and poorly differentiated family relationships, or they could be overly *rigid*, resulting in family disengagement and a loss of connection.

Particularly important in the structural perspective is the clarity of the boundary around the parental subsystem. When this boundary is not maintained and children become inappropriately involved in the interparental relationship, neither the family system nor its individual members are able to function adaptively. Minuchin went on to describe specific ways this boundary might be violated, which he referred to as *rigid triangles* in the family. First, one parent may form a *coalition* with the child in which an overly close relationship develop that excludes the other parent and compromises the ability of the couple to achieve intimacy or effectively carry out their executive functions in the family. In the second scenario, termed *triangulation,* each parent attempts to enlist the exclusive support or loyalty of the child, essentially placing the child in a form of double bind in which there is no way to achieve emotional closeness with one parent without alienating or angering the other. The third triangular form, *detouring*, is reminiscent of Bowen's family projection process in that parents circumvent their conflicts or marital unhappiness by focusing on the joint project of caring for their ostensibly troubled or needy child. Detouring may be further defined as *supportive* (e.g., with the parents uniting around the task of providing nurturance for a child that is perceived as fragile, special, or delicate) or as *attacking*, in which the child is scapegoated, ostracized, or blamed. Essential to the concept of detouring is that having a symptomatic child serves a function for the larger family system, which therefore leads all family members—including the emotionally disturbed child—to be resistant to changing this family pattern. Thus, to have a child who is an identified patient serves to maintain homeostasis in the family.

The Functional Family Perspective

Although ultimately evolving into a technique of family therapy rather than a research-generating school of family theory, as is true of so many of the foregoing frameworks, the Functional Family Therapy (FFT) perspective (Alexander & Parsons, 1982; Alexander, Robbins, Waldron, & Neeb, 2013) also contributed a number of seminal ideas that warrant consideration. One of these is the idea that family relationships can best be understood by attending to the *relational functions* that family members are striving to achieve with one another. The influence of interpersonal theory can be seen in the definition of these relational functions as comprising the two orthogonal dimensions of hierarchy and connection. *Relational hierarchy* refers to the relative balance of power in the relationship, such that family members strive toward roles that are one-up, one-down, or symmetrical in relation to one another. In describing the role of hierarchy in parent–child relationships, FFT is unusual among family systems models for not being prescriptive that this necessarily should comprise a dynamic in which the parent is "in charge" and the child "in tow" (Kerig & Alexander, 2012). Instead, the FFT model proposes that there is diversity in what comprises an adaptive family structure, and no form is inherently maladaptive; rather maladaptation arises when family members are not able to successfully meet their relational functional needs. The second dimension of relational functions concerns *relational connection*, the balance between family members' needs for autonomy versus connection. Here again, the presumptions are made that there are individual differences in the extent to which family members

have needs for autonomy or connection and that neither of these extremes is inherently problematic. Dysfunction instead arises when there is a mismatch between family members' relational functions (e.g., a connection-seeking adolescent with an autonomy-seeking father) that prevents one or both from getting their relational needs met.

Another way in which the FFT perspective differs from most family systems theories is that, rather than typologizing the family as a whole—as, for example, hierarchically organized or autonomous—FFT's conceptualization of relational functions is particular to an *individual* in the context of a particular *dyad*. For each dyad in the family, the assessor evaluates the extent to which each partner seeks closeness versus distance in that specific relationship and considers the ways the individual's behavior (e.g., acting out on the part of the connection-seeking adolecent) might serve a function in that relationship (e.g., to bring an autonomy-seeking father closer). Once the underlying functions of the behavior are understood in the light of family members' relational needs, the clinician can foster change by encouraging the development of more adaptive ways of getting those needs met, ways that are respectful of and matched to the relational needs of each dyad. For example, rather than risking resistance, or merely awkwardness, by proposing that an autonomy-seeking father spend his day off in intimate togetherness with his contact-seeking son, the FFT therapist might suggest the judicious use of text messaging to allow the father to communicate more comfortably that his son is in his thoughts during the day. Furthermore, because relational functions operate at the level of the dyad, each family member may exhibit different relational functions in those dyads; for example, a youth may be contact seeking toward his father yet distancing with his mother. By the same token, parents develop different relational functions with individual children in the family and may seek closeness with one while maintaining distance from another.

Because of its focus on clinical implementation, FFT has tended to organize its thinking around the implications of family relational processes for the individual child as the identified patient, and therefore less attention in the theoretical model has been paid to the intersections of these various relational functions among the dyads in the family and how they might affect one another. And, as with most of the family models discussed so far, the priority of FFT-related research has tended to focus on establishing the effectiveness of the intervention rather than testing hypotheses about the underlying assumptions of the theory.

Social Learning Perspectives on the Family

Subsequent to the 1980s, little groundbreaking new work emerged in the elaboration of these earlier seminal family systems theories, perhaps due to a larger change in the zeitgeist in psychology that increasingly favored behavioral approaches. The most influential figure in this school of thought is Gerald Patterson (1986), whose studies of the families of antisocial youth at the Oregon Social Learning Center were groundbreaking not only for astuteness of their observations but also for the sophistication of their methods. In addition to developing a highly influential model of parent management training, Patterson's work has contributed many important conceptual and methodological components to modern family systems research. One of these is their recognition of the importance of reciprocal processes in the family and their construction of observational methods to capture and quantify those processes. Another is the consistent inclusion of fathers in the research (at least among those families with two-parent households) and the recognition that each parent may have a specific parenting style and relational quality with the child that has unique effects on development.

Summary

This brief outline of the early historical origins of family systems theory suggests a couple of observations. One is that a likely reason for the limited influence of family theory on research in developmental psychopathology may be that so many of the influential figures in the field conducted their work at independent institutes rather than in mainstream academia, where they might have had more influence on shaping the thinking of the next generation of clinical scientists and scholars. In addition, many of the seminal family systems theorists were more invested in clinical work than in research; therefore, the focus of the training offered was not conducive to the development of a program of empirical inquiry. Moreover, this review of the origins of the field brings home the point that the family systems label comprises a canopy for a quite diverse set of perspectives that derived as offshoots from different theoretical branches. Some show their roots in psychodynamic thinking, whether overtly labeled as such (Slipp, 1991) or more covertly displayed through their attention to unconscious dynamics and the construal of the family system as internal conflict made manifest (e.g., Boszormenyi-Nagy & Spark, 1973). Others have firm roots in behavioral and social learning theories that hold no truck with unconscious notions (e.g., Patterson, 1982), and still others rely

on constructs that were quite novel and groundbreaking at their time, such as communications theory and semiotics (e.g., Watzlawick, Weakland, & Fisch, 1974). Nonetheless, despite these differences in theoretical origins, a number of shared features characterize systemic theories, and these we turn to next.

WHAT MAKES A FAMILY THEORY SYSTEMIC?

Holism

Central to family systems theory is the construct of holism, the idea that "the whole is more than the sum of its parts" (Bertallanffy, 1968, p. 55):

> Systems theory begins with the observation that nature can be viewed in a hierarchically arranged continuum, with the more complex, larger units superordinate to the less complex, smaller units. Each level needs to be regarded as an organized whole, with distinctive properties and characteristics that are altered by interchange with other levels, but with no level reducible to simpler levels. Thus, organs are more than simple aggregates of cells, the person is more than an aggregate of organs, and the family is more than an aggregate of persons. In this hierarchy of organizations, each level has distinctive and unique qualities (Wynne, 1984/2004, p. 2).

Rather than a collection of individuals, each with his or her own pathology, systemic thinking involves viewing the family as an entity that has unique properties not merely reducible to the individual constituents of its members. The source of pathology or well-being therefore derives from the system and the interrelations among its members rather than the individual members themselves. By the same token, the study of individual dyads within the family does not act as a proxy for understanding the family as a systemic whole (Kerig, 2001). Family members' roles and behaviors change as they move between dyadic and triadic relationships; for example, a child interacting together with both mother and father involves the simultaneous dynamics of three different relationships—mother–child, father–child, and the interparental dyad—as well as the repercussions of each of those for the others (e.g., the father's reaction to a daughter's expression of disrespect for her mother). Similarly, for the adults in the family, moving from the dyad to the triad involves balancing the role demands of parent and partner—and "thus the parent–child dyad is transformed into a family system" (Gjerde, 1986, p. 297). Observation at the whole-family level also has the potential for conferring a possible higher order of meaning to dyadic interactions such that, for example, a parent may affect a child indirectly through the ways he or she affects the relationship of the other parent with that child (Parke, 1990) (e.g., the father who encourages disrespect by finding humor in his daughter's sassiness toward her mother).

Further, being part of a system has both constraining and potentiating influences on the behavior of its members, pulling them toward participation in the family's typical dynamics in ways that bring to the fore qualities that cohere with family patterns while at the same time dampening latent tendencies that might otherwise be expressed in different contexts. Thus, something new arises in the chemistry of the family crucible that could not have been predicted from knowledge of its individual members on their own.

However, one point of contention in the family systems literature has been whether families can be accurately described in holistic ways—to be typologized as an enmeshed or disengaged family, for example—versus whether the family system is better construed as a matrix of dyads, each of which might be represented by a different point along these continua and each of which has an impact on the other dyads as well as the family process as a whole. For example, although there certainly may be families in which all relationships can be described equally as enmeshed, clinical descriptions of pathological families are more likely to involve those in which one enmeshed relationship (e.g., between a parent and an eldest child) contributes to problems in rest of the system, such as increased friction (e.g., in the interparental dyad) or imbalance (e.g., among the siblings). Therefore, as Philip and Carolyn Cowan (1990) pointed out, if one enmeshed dyad is counterbalanced by another one that is disengaged, "How then, are we to describe the *family*?" (p. 42, italics in original).

McHale, Kuersten-Hogan, & Lauretti (2001) offered a helpful distinction between *whole-family* constructs, which refer to abstract characteristics of the family gestalt, and *family-level* constructs, which comprise observable behaviors that arise when the family members are considered as a group. Notably, however, these processes may arise even when one member of the system is not present, such as when parents engage in covert coparenting conflict behaviors such as denigrating the absent spouse to the child (McHale, 1997) or, on the positive side, when there is good coordination and synchrony between parents in the absence of one another. "The ability of mothers and fathers to parent in ways that are consistently supportive of and coordinated with one another—*especially* when apart from one another—may provide the child the most convincing demonstration of the firmness and security

of the marital alliance" (Kerig, 2001, p. 9). Therefore, we can add to this definition a third category, *family dynamic* processes, "referring to those interactions that have implications for family-level functioning even when they do not take place in the presence of all family members" (Kerig, 2001, p. 10).

In sum, the study of family-level processes requires neither a group of people to be in the same room nor the typologizing of a family into a particular category. As Belsky, Putnam, & Crnic (1986) asserted, "[Family-level] dynamics include events and processes that involve all family members together or a family subsystem (parent–child or husband–wife) that affects and is affected by the other subsystems in the family" (p. 46). Whereas many of the early conceptual attempts to bring family systems constructs to bear in the study of psychopathology imposed this whole-family typological approach, research more often has assessed either family-level constructs or, even more often, dyadic relationships within the triad.

The Effects of Relationships on Relationships

A middle-way position between the extremes of holism and individualism is the broader systemic idea of interdependency—in other words, that relationships have effects on other relationships (Emde, 1988). As Rutter (1988) argued, despite the fact that the "functioning of the whole is qualitatively different from the sum of its parts . . . the properties of the family as a whole derive from the properties of the relationships between the individuals in the family" (p. 333). This is a way family systems thinking most clearly differentiates itself from individualistic or unidirectional approaches to psychopathology: rather than relationships affecting the person, systems theory proposes that relationships affect other relationships in the family system (Gjerde, 1986). Thus, the family is viewed as an interrelated web in which perturbations in one strand send reverberations along the threads interconnecting other parts of the system. "The most important clues to the significance and meaning of an interaction to the participants may be the context of other interactions in which it lies" (Hinde, 1976, p. 4).

The relationships that affect and are affected by others in the family may be dyadic, or triadic, or may involve intersections among more complex combinations of subsystems. A prototypical example of such interrelatedness of relationships is provided by the idea of triangulation, in which an imbalance in the closeness and distance between members of one family dyad has implications for each of the others. For instance, an overly close relationship between a parent and child might inappropriately involve that child in the executive functioning of the marital subsystem and thus exacerbate a riff in the interparental relationship. By the same token, diffuse boundaries between a parent and child might contribute to conflict among the siblings, such as when a parentified child blocks the other children's access to emotional closeness and instrumental assistance from that parent.

A second and related example of the effects of relationships on relationships is provided by the concepts of spillover and *compensation* (Engfer, 1988; Kerig, Cowan, & Cowan, 1993). In the former process, conflict in the interparental relationship may spill over onto parent–child relationships in negative ways by increasing parental negativity (Katz & Gottman, 1996; Margolin, Gordis, & Oliver, 2004), decreasing parental emotional availability (Cox, Paley, & Harter, 2001), or compromising the child's felt security in the family system (Cummings & Davies, 2011). In the latter process, dissatisfaction in the couple relationship might instead lead to a compensatory closeness between a parent and child that, although ostensibly positive in tone, may have negative repercussions through imbalancing the family system (Kerig, 2005).

One of the important implications of this systemic view of the interdependencies of relationships is that the overarching context might confer very different meanings upon the qualities of the dyadic relationships within the family. For example, a close relationship with one parent might be associated with positive benefits for a child in a cohesive family system but have negative implications for the child whose alliance with one parent occurs at the expense of his or her relationship with the other (Kerig, 1995). As Grych, Raynor, and Fosco (2004) stated, "Whereas a strong parent–child alliance generally would be expected [to] have a protective effect, parent–child alliances formed as a reaction to discord in the marital relationship may not be protective if they lead to cross-generational coalitions that exclude the other parent [and] cause tension in the child's relationship with the other parent or lead to loyalty conflicts for the child" (p. 652).

Another related implication of this systemic view of interdependencies is that associations among family processes may vary depending on other properties of the larger family system (Davies, Cummings, & Winter, 2004). For example, a number of family context variables might moderate the strength of the association between interparental conflict and parent–child dysfunction, such as the presence of spousal violence (e.g., Margolin, Gordis, & Oliver, 2004), family hierarchy (e.g., Lindahl, Malik, Kaczynski, & Simons, 2004), or the presence of clear boundaries

(e.g., Kerig & Swanson, 2010), thus leading to heterogeneity and multifinality among families experiencing the same risk factors. Whereas some of these moderators might operate in ways that dilute the impact of family adversities, others impart vulnerability and do so particularly by increasing the interdependencies among family subsystems and the consequent spillover of negativity from one to the other.

Circularity

The term circularity in family systems theory refers to the notion that cause-and-effect in systems is not unidirectional but rather that family members influence one another reciprocally, mutually, and bidirectionally. Although it might be possible to observe family patterns in which unidirectional patterns of influence appear to be operating (e.g., a father criticizes his daughter, who then expresses negative affect), in the systems perspective these assumptions are viewed as merely deriving from a point of punctuation in an ongoing process. An observation of the same family commencing at a different time point might reveal the cause and effect to be explainable just as well from the other direction (e.g., a daughter repeatedly expresses negative affect toward her father, to which he finally responds with exasperation). The close correspondence between the construct of circularity and that of *transactions* (Sameroff, 1995) in developmental psychopathology is evident, and the link is made explicitly by some systems theorists, such as Wynne (1968): "In transactional processes, persons undergo internal change during the course of interchange with one another. All parts of a transactional field are interdependent, each modifying the rest through recursive ('circular') feedback; the whole exhibits the functional relation between the parts" (p. 4). However, as used by many systems theories, the construct of circularity lacks the important developmental dimension involving an evolving process in which actors and environments successively shape, influence, and even transform one another over time in ways that have important implications for the developing person (Kerig, Ludlow, & Wenar, 2012; Sameroff, 2010).

Research supports the idea that there are reciprocal effects among family processes. For example, sequential analyses have shown that episodes of interparental conflict are followed by increased negativity in whole-family (Christensen & Margolin, 1988; Kitzmann, 2000) and parent–child interactions (Almeida, Wethington, & Chandler, 1999); however, tensions in the father–child relationship also are predictive of an increased likelihood of subsequent interparental conflict (Almeida et al., 1999).

Homeostasis

The construct of *homeostasis* represents a contribution of the MRI group that has continued to have a long-lasting influence on family systems theory (Jackson, 1965). Homeostasis is an often-cited construct in the clinical literature that is used to explain the conundrum of why family systems seem so readily to fall back into pathological patterns and to resist therapeutic change. Consequently, homeostasis has gained the status of a kind of invisible adversary in the room that inspires family therapists to work actively and vigorously to countervail this dark force that pulls the family inexorably backward, in the direction of entropy. But, as Wagner and Reiss (1995) remind us, homeostasis needn't necessarily have only negative connotations in that "homeostasis can also be understood to be a way in which normal families maintain constancy and predictability in the face of constantly changing environmental demands … [and] maintain a sense of regularity in the presence of everyday stresses and changes" (p. 697). Thus, homeostasis has pathogenic potential for stubbornly returning the system to its set-point in ways that don't allow for adaptive change but also has an adaptive function in self-righting or balancing a system that is in danger of becoming destabilized. As Wagner and Reiss phrased it, "Threats to family stability, in the organization, power, or affective realm, tend to be counteracted by family members in a manner that will retain or restore their balance" (p. 697). In this way, homeostatic regulation may allow the family to act as a secure base from which its members can more freely explore their environment.

Families as Organized Systems

Another corollary to family members' interconnectedness in the systemic perspective is that these interrelationships are *organized* by the larger system in ways that confer coherence and lawful continuity across contexts as well as across developmental transformations (Sroufe & Fleeson, 1988). This larger organizational framework has implications for the functioning of individual members as well as the system as a whole:

> At the family level, coherence means that there is a total organization that may not be reduced to the qualities of the relationship units within the family and that relationships within the family are constrained by the total organization. Moreover, given certain qualities in one relationship, other relationships in the family are further constrained in the service of maintaining the larger organization. This view is distinct from emphases on 'influences' of one relationship upon another.

> Rather, in this organizational perspective interdependency among relationships in the family is emphasized (Sroufe & Fleeson, 1988, p. 45).

An important implication of this dynamic organizational perspective in systemic theory is that dysfunctional family patterns not only increase the risk for maladaptation in the individual, but in the family system as a whole. By the same token, adaptive family functioning confirms positive benefits that radiate outward from the individual and enhance the capacity for all family members, and the system itself, to thrive. As Diamond, Allen, and Butterworth (2012, p. 262) suggested:

> These adaptive processes are just as important for the *family's* eventual well-being as for the *adolescent's* well-being. Accordingly, perhaps the most valuable potential contribution of a family systems approach is that it prompts us to ask broader, more complex questions about how different family members' trajectories of adjustment are fundamentally interconnected, and the ways each member's adaptation (or the lack thereof) "pulls" on the system as a whole.

Boundaries and Subsystems

Also inherent in family systems theory is the idea that the larger whole is composed of subsystems that are both interconnected and demarcated from one another. Although most centrally associated with structural family theory, a common thread across perspectives is the idea that adaptive family functioning requires the maintenance of appropriate psychological boundaries between these subsystems and among the members of each subsystem. Like the membrane of a cell, boundaries need to be permeable enough to allow communication and emotional contact to traverse the divide while at the same time being firm enough to ensure the integrity of each separate unit (Kameguchi, 1996). In particular, however, the maintenance of boundaries around the couple relationship is seen as being at the heart of adaptive family functioning, and violations of that boundary create cross generational alliances and coalitions that inappropriately involve children in the marital subsystem and are associated with maladaptation. Family theory proposes a number of ways boundaries might be violated in the family system.

Enmeshment

The term enmeshment is used in a variety of ways in the family systems literature. Consistent with Minuchin's (1974) structural perspective, as a whole-family phenomenon, enmeshment refers to those families in which all members are highly overinvolved with and emotionally overreactive to one another. However, following psychodynamic models, enmeshment often is studied as a quality of dyadic relationships, which may have implications for the other relationships in the family, such as when enmeshment with one parent is counterbalanced by disengagement in the child's relationship with the other parent (Cowan & Cowan, 1990; Jacobvitz, Riggs, & Johnson, 1999). Enmeshment also is used as a superordinate construct within which other all other forms of boundary dissolution and triangulation are subsumed (e.g., Jacobvitz, Hazen, Curran, & Hitchens, 2004). Research has linked enmeshed relationships within family systems and parent–child relationships to a number of negative child outcomes, particularly those involving internalizing disorders (e.g., Jacobvitz et al., 2004; Jewell & Stark, 2003; Kerig, 2005) and attachment insecurity (e.g., Allen & Hauser, 1996). Moreover, despite the tendency in some perspectives to conflate enmeshment with other family-level constructs, such as cohesion (Olson, Russell, & Sprenkle, 1983), research has demonstrated that enmeshment is a unique construct with discriminable effects; whereas overall family cohesion is associated with positive child adjustment, enmeshed relationships are not (Barber & Buehler, 1996).

Some research suggests that enmeshment may be a culturally bounded phenomenon, however. Whereas among adolescents in the United Kingdom similar results are found to those in U.S. samples, with enmeshment associated with depression, anxiety, and perceived threats to identity, such findings have not emerged in samples drawn from cultures that more highly prioritize family members' interdependence, such as Italy (Manzi, Vignoles, Regalia, & Scabini, 2006) and India (Jackson, Raval, Bendikas, Raval, & Trivedi, in press).

Parentification

Boszomenyi-Nagy and Sparks's (1973) construct of parentification has spurred a significant body of research in developmental psychopathology (Jacobvitz & Bush, 1996; Jurkovic, 1997; Kerig, 2005; Kerig & Swanson, 2010; MacFie, Houts, McElwain, & Cox, 2005). Also termed role reversal or compulsive caregiving (Bowlby, 1977; Crittenden, 1992), this is a form of boundary dissolution in which parents turn to children for nurturance, comradeship, or instrumental assistance. Among clinical samples, role reversals and their accompanying developmentally inappropriate expectations for children are associated with maltreating parenting (Macfie et al., 1999) and are predictive of severe forms of psychopathology in youth such as borderline personality traits (Lyons-Ruth, Bureau,

Holmes, Easterbrooks, & Brooks, 2012). Research also evidences that children placed in a parental role are at increased risk for maladaptation across toddlerhood, preschool, middle childhood, adolescence, and emerging adulthood; moreover, consistent with family systems theory, problems in the marriage appear to create an emotional ground that is fertile for the development of boundary violations involving parentification (Kerig, 2005).

Some research suggests, however, that the construct of parentification conflates processes that are not equally pathological for children's development and thus should be discriminated. One such process, termed *adultification* (Kerig, 2005) or role diffusion (Jacobvitz & Sroufe, 1987), involves egalitarian relationships in which adults treat the child not as a parent but as an agemate. This might involve a parent coming down to the child's level and behaving like a childhood peer (the child-like parent) or the child being promoted to the status of an adult peer (the adult-like child).

Moreover, cross-cultural research points to the importance of differentiating between destructive forms of parentification/adultification and normative expectations for childhood and adolescence that differ across cultures, particularly among those that do not sanction the long period of childhood dependency characteristic of middle-class European American society (Jurkovic, 1997). Cultural roles valuing familism (Gibbs & Huang, 2003), parent–child mutuality (Anderson, 1999), and interdependence (Kağıtçıbaşı, 2005) as well as the practical demands incumbent upon family economic disadvantage (Boyd-Franklin, 2003) increase the likelihood that children will be assigned instrumental responsibilities in the home, such as helping to raise younger siblings, or emotional responsibilities, such as soothing others' hurt feelings, that could appear to an outside observer as involving precocious elevation to adult status. However, these *filial responsibilities* have been shown not only to vary widely across cultures but also to be associated with child distress or dysfunction only when they are perceived as unfair, unacknowledged, or unappreciated (Jurkovic, Kuperminc, Sarac, & Weisshaar, 2005).

Spousification

The construct of spousification originates from observations by Sroufe and Ward (1980) of seductive behavior demonstrated by mothers who were effusively affectionate and flirtatious toward their young sons, relating to them more as though they were adult intimate partners than children. Most often studied in the context of mother–child interactions, spousification has been found to predict child inattention in preschool (Jacobvitz & Sroufe, 1987), hyperactivity in middle childhood (Carlson, Jacobvitz, & Sroufe, 1995), and emotional and behavioral undercontrol in adolescence (Sroufe, 1991). Using data from the Minnesota Longitudinal Study of Parents and Children, Shaffer and Sroufe (2005) found that maternal seductive parenting demonstrated continuity from toddlerhood into adolescence, particularly for boys, and was associated with youth problem behavior above and beyond other measures of family dysfunction and parent–child negativity. Moreover, maternal spousification in infancy forecast the adolescent's own behavior during observed parent–child interactions, including role equalization (e.g., treating the parent as a peer) and sexualized behavior (e.g., flirting with or excessively caressing the parent; Shaffer & Egeland, 2011). Among the rare studies to include measures of paternal spousification, Rowa, Kerig, and Geller (2001) found that young women with eating disorders reported higher levels of such boundary problems with their fathers than did community women and, in a sample of emerging adults, Kerig and Swanson (2010) found that both maternal and paternal spousification mediated the association between interparental conflict and maladaptation, but paternal spousification was linked uniquely to relational aggression against romantic partners.

Intrusiveness

Also termed psychological control, intrusiveness refers to "parental behaviors that are intrusive and manipulative of children's thoughts, feelings, and attachments to parents" (Barber, 2002, p. 15), such as guilt induction or withdrawal of love. Again, a large body of research has emerged to show that intrusive parenting is associated with maladaptation across development, particularly in regard to children's capacities for autonomy and self-directedness (Kerig, 2005; Kerig & Swanson, 2010; Kerig, Swanson, & Ward, 2012). These results have emerged in widely diverse samples in the United States (e.g., Barber, Bean, & Crane, 2006; Bean & Northrup, 2009) and cross-culturally (Barber, Stolz, & Olson, 2005).

Families as Open Systems

Finally, according to systems theory, a characteristic of the family is that it is an open system, which, although self-regulating and governed by coherence and organizational structure that confer stability, is one that is open to change in response to demands from both within and without the system (Cox & Paley, 2003). As Davies et al. (2004) phrased it: "According to family systems theory,

the family unit is an open system in which family perturbations and stressors (e.g., parent psychopathology, relationship disturbances, disruptive family events) may provoke subsequent reorganizations in family functioning and function as architects of interdependencies between family subsystems" (p. 775). Although this is a conceptually important underpinning to family systems theory that provides a point of convergence with the developmental psychopathology perspective, in fact a closer look at systemic conceptualizations of change raises a number of important questions, to which we turn next.

Change Processes in Family Systems

Less a matter of consistency among family systems theories than an issue with which they each have to contend, systemic perspectives must also offer a theory of change. As noted already, the family system is conceptualized as an open system and thus one that is open to change; therefore, families cannot be understood by homeostasis alone but rather theories also must account for the processes that allow for family growth and adaptation. Systems theories also make a distinction between *first-order* change, which refers to fluctuations and minor alterations in family functioning that leave the essential structure of the system unchanged, and *second-order* change, which involves a transformation in the family structure that is discontinuous with what has gone before (Watzlawick, Weakland, & Fisch, 1974).

Three primary models of family change can be found infused throughout the family literature. The first proposes that stress precipitates change through disequilibrating the family system and requiring it to abandon ineffectual strategies to reorganize in a more adaptive way to meet the challenge (Wagner & Reiss, 1996). This model of change has inspired the development of therapeutic techniques that evoke a crisis in the family to inspire transformation, such as strategic family therapists' use of *paradoxical interventions* that throw the system off balance, including prescribing the symptom (Papp, 1983).

A second perspective on change is the family life cycle approach, modeled after Erikson's (1950) psychosocial stages of development, in which each successive stage gives rise to specific developmental tasks that must be mastered for optimal development and thus calls for adaptations in the family role structure to accommodate. McGoldrick and Carter (2003) are the most well-known proponents of this approach, which outlines the stage-salient tasks associated with each successive stage in the formation of the family system. However, this is less a model of change than of normative development in families, and thus is discussed in greater detail in a later section of this chapter.

A more process- than content-oriented approach to family development was proposed by Steinglass, Bennett, Wolin, and Reiss (1987), which they termed the *Family Life History* model. Their view of family development was organized around three sequential tasks that they viewed to be universal across family forms, with each task emerging as most salient at particular points in the family's life span. In the earliest stage of family formation, the critical task is the establishment of an identity of the family as a distinctive entity, achievement of which involves the establishment of *boundaries*, including those within the members of the newly forming family and between each member and his or her family of origin. The second stage of family life involves *commitment* of the family system to a structure, organization, and set of values. The later phase is focused on consolidation of family identity and generativity regarding *the future*, particularly the preservation and transmission of family identity, values, practices, and rituals to generations to come. Inherent in this model also is a theory of dysfunction. Steinglass and colleagues proposed that the key sources of difficulties within each of these stages of family life are asynchronies that arise between the overarching tasks and developmental status of the system and those of any of its members or subsystems. For example, a clash might be seen between a mature family form seeking to consolidate its identity into the future (e.g., an immigrant family invested in maintaining a history of strong cultural values) and the developmental tasks of members who are striving toward forging independent identities (e.g., an adult child who is starting her own family with a partner of a different cultural background).

A third approach to understanding family change—the model most truly systemic as well as the one most consonant with the developmental psychopathology approach—is the *Epigenetic Relational Systems* model proposed by Wynne (1984/2004). In this innovative work, Wynne set out to apply the principles of epigenesis to relational systems in ways that explicitly incorporated general systems theory (Bertalanffy, 1968) and its tenets, including the construct of transactional processes underlying relationships in the family. Wynne was concerned to delineate not merely changes in the content or structure of roles such as those outlined in family life cycle theories but rather changes in relational processes that occur over the course of family development.

> What is crucial for my present formulation is the point that most of these life cycle changes in family role structure proceed more or less inexorably regardless of the quality of relating of the participants. To be sure, some relational changes facilitate and are complementary to changes in family composition, but other changes in family roles and structure take place unexpectedly or precipitously or are delayed. Meanwhile, the epigenesis of relational processes, I shall contend, proceeds at a rate and in a sequence that has its own inner "logic," sometimes unfolding smoothly and continuously and sometimes destabilizing discontinuously ... as new ingredients are randomly introduced or created within each variety of relational process. This means that "structural" family change and relational development are expectably and frequently going to be out of synchrony. Thus, the traditional approach to the family life cycle, defined primarily in terms of *structural* role change by exits and entries, is in a sense orthogonal to the unfolding of *relational* processes (Wynne, 1984/2004, p. 4).

In this light, Wynne identified five processes that unfold epigenetically in the development of relational systems: (1) *attachment* and affectional bonding; (2) *communicating* and sharing meanings; (3) *joint problem solving* and shared engagement in tasks; (4) *mutuality*, which involves reengaging, reevaluating, and renegotiating the preceding relational processes in ways that accommodate shifts in the internal states of family members and external demands of the environment; and (5) *intimacy*, a deeper and more elaborated form of relatedness that emerges as an outgrowth of the other processes but that Wynne considered more of an aspirational goal than a developmental necessity. Each relational process in Wynne's model can be conceptualized as having a positive or negative pole (Figure 13.1), with the latter comprising relational failures.

Whereas the positive poles of each relational process are viewed as nodal points around which mild fluctuations occur, representing first-order change, second-order change is seen when the family moves to a different predominant relational mode. Consistent with a crisis model of change, Wynne (1984/2004) proposed that failures in navigating these processes can "serve as challenges leading to mastery ... Although prolonged, intense instability or divergence will surely impair or disrupt relatedness, divergence that is contextually and developmentally appropriate is necessary as a decisive stimulus for moving on to the next level of relating" (p. 3). In addition, like Steinglass and colleagues (1987), Wynne also noted that problems may result from asynchronies in stages of the family life cycle, the developmental status of individual members, and the epigenetic unfolding of these relational patterns. "What is 'negative' at the relational system level may, of course be 'positive' for individual participants, for example in achieving differentiation" (p. 3).

Although Wynne's model has been used to good effect in clinical descriptions of the family systemic origins of certain disorders, such as in Guttman's (2002) discussion of adolescent borderline personality, the absence of a method for assessing these relational processes has precluded their incorporation into the empirical literature (Wagner & Reiss, 1996). Moreover, until recently, the lack of statistical methods sophisticated enough for modeling such dynamic change processes also has proven an impediment, one that future research may be able to overcome.

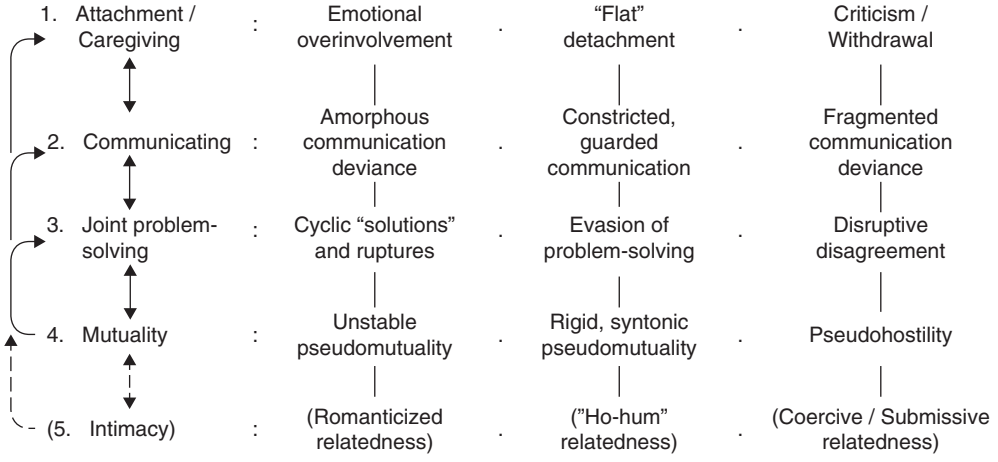


Figure 13.1 Wynne's model of epigenetic processes in relational systems. *Source:* Wynne L. C. (1984/2004). The epigenesis of relational systems: a model for understanding family development. *Family Process, 23*(3), 297–318; http://onlinelibrary.wiley.com/doi/10.1111/j.1545-5300.1984.00297.x/pdf. Copyright 2004, John Wiley & Sons. Reproduced with permission.

PSYCHOPATHOLOGY AND ITS CLASSIFICATION FROM A FAMILY SYSTEMS PERSPECTIVE

A long source of frustration for family systems thinkers, and another reason that systemic thinking has tended to not have a place at the table, is the lack of attention paid to family systemic factors in the major nosological and diagnostic systems used to study childhood psychopathology (Beach, Wamboldt, Kaslow, Heyman, & Reiss, 2006; Denton, 2007; First, 2006; First et al., 2002; Wamboldt & Wamboldt, 2000). Given that much of the literature in childhood psychopathology is organized around discrete disorders—the etiology and developmental course of anorexia, conduct disorder, and the like—where is the place for a system of thought that instead construes the subject of study as the anorexic or antisocial *family*?

Inclusion of Family Factors in Existing Diagnostic Systems

DSM

In the current version of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*; American Psychiatric Association, 2013), the only diagnostic category whose etiology is explicitly related to relationships within the family is reactive attachment disorder, a syndrome which arises as a function of an extreme absence of caregiving that is rarely seen outside of dramatically physically and emotionally depriving institutions. Instead, in the *DSM-5*, clinicians are given the opportunity to take family relational factors into account in two ways. One is to allow the clinician to note significant psychosocial and contextual features, such as extent to which the child's disorder is complicated by a milieu that is stressful or fails to offer necessary support and that might compromise the child's relative capacity to function with the disorder with which she or he is being diagnosed. The second way family factors come into play in the *DSM* is in the category of Other Conditions That May Be a Focus of Clinical Attention, or V codes, which are applied when the main focus of clinical concern is not considered to comprise a mental disorder per se. Among these V codes are *relational problems* in the dyadic relationships between parent and child (e.g., impaired communication, overprotection, inadequate discipline), partners (e.g., negative, distorted, or absent communication), or siblings, with a separate category indexing problems related to physical abuse, sexual abuse, and neglect. The family system of relationships is nowhere in sight.

In a major critique of the *DSM* framework and its sequestering of relational problems into a V code ghetto, Heyman et al. (2009) argued that compelling research exists to confirm that interpersonal problems in the family constitute bona fide clinically relevant syndromes that warrant our full attention. Relational problems meet the full *DSM* definition of disorder by virtue of being associated with subjective distress and functional impairment as well as predicting poor mental, physical, and interpersonal outcomes; thus, their neglect by *DSM* is unjustified. Their concerns echo those of the Group for the Advancement of Psychiatry Committee on the Family (1989) two decades earlier:

> Compared with the distress and disability they incur, these syndromes remain neglected—a situation reinforced by the lack of a place where such problems are meaningfully identified in our official diagnostic and statistical manual. The *DSM* approach requires individual-based diagnoses for relational disorders and thereby misdirects assessment and focused treatment away from the couple, family, or other relational systems in which dysfunction is most pivotally present (pp. 1492–1493).

Moreover, given the centrality of the family context for child development, family relational dysfunctions might loom particularly large in the life of children and thus constitute the kinds of failure in the expectable environment that are associated with the highest risk of maladaptation, a point of argument that has long informed criticism of the *DSM* framework by developmental psychopathologists (Bemporad & Schwab, 1986; Garber, 1984). "Consequently, some of the most significant concerns that bring children to the attention of mental health professionals—abuse, family conflict, bereavement, family dissolution—are placed in . . . a somewhat vaguely defined category of V codes, which may not be seen as priorities for treatment" (Kerig, Ludlow, & Wenar, 2012, p. 94).

Diagnostic and Statistical Manual for Primary Care

One attempt to revisit the *DSM* and to reconstrue the diagnostic system in ways that are more sensitive to developmental and family processes is the *Diagnostic and Statistical Manual for Primary Care—Child and Adolescent Version* (*DSM-PC*; Wolraich, Felice, & Drotar, 1996). The *DSM-PC* grew out of a collaboration among pediatricians, child psychologists, and child psychiatrists concerned with providing primary care physicians with tools for evaluating the extent to which behavioral and emotional problems presented by child patients might reflect deviations from normal development serious enough to warrant intervention. Notable is that the *DSM-PC* is a diagnostic system that is particularly closely aligned with

a developmental psychopathology perspective. Not only are symptoms placed explicitly in a developmental context and understanding of normative development, and not only is the family context given priority in understanding the etiology of childhood disorders, but the manual also is explicit in referencing the transactional nature of the child's interface with the social environment:

> A critical influence on children's general well-being—including behavioural and cognitive functioning and physical health—is the quality of the environment in which the child lives. A basic tenet of this manual is the central importance of the transaction over time between the child and the environment in which the child lives and grows (p. 31).

Despite this overarching transactional conceptualization, and despite the way in which the *DSM-PC* encourages the clinician to begin diagnosis by first considering familial and broader environmental, social, and interpersonal sources of risk and protection, the manual ultimately conforms to the *DSM* strategy of locating the clinically significant developmental variation, problem, or disorder within the child rather than within the family system. Moreover, despite its strengths, psychometric data are not available and, to date, the *DSM-PC* has not gained wide usage in clinical contexts nor in the research literature (Friedman, Sadhu, & Jellinek, 2012).

International Classification of Mental and Behavioral Disorders in Children and Adolescents

The International Classification of Mental and Behavioral Disorders in Children and Adolescents (*ICD-10*; World Health Organization, 2007) is the diagnostic system more widely used outside of the United States. The *ICD* also is undergoing revision at the time of this writing; in fact, the publication of the *ICD-11* has been delayed due to attempts to better align the system with the forthcoming *DSM-5* (First, 2009). Like the *DSM*, the *ICD* includes a separate axis for Associated Abnormal Psychosocial Situations in which family factors and other contextual variables can be taken into account. However, the *ICD* is more explicit than the *DSM* in suggesting that these situations might have implications for understanding the etiology of the problem and not only in qualifying its presentation or informing treatment. Moreover, the *ICD*'s much more extensive list of such situations includes a number that go beyond static family variables and appear to reflect interrelational difficulties that are more consonant with a family systems framework than are any in the *DSM*. These include disturbed intrafamilial relationships such as discord among adults or scapegoating of a child, inadequate or distorted intrafamilial communication, abnormal qualities of parenting such as overprotection, and acute life events that negatively alter patterns of family relationships.

Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood, Revised

The *Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood, Revised* (*DC:0-3R*; Zero to Three, 2005) was developed specifically as a supplement to fill gaps in the *DSM* and *ICD* systems related to their presentation of clinical syndromes in young children. Consistent with the centrality of attachment relationships for the adaptive development of children in the first years of life, the *DC:0-3R* includes not only an axis for coding psychosocial stressors similar to the *DSM* and *ICD* systems but also requires that for each child a specification be given on a separate axis for Relationship Classification. However, this relationship is explicitly defined as comprising that between the child and the primary caregiver; the larger family system of relationships affecting this dyad does not play a part. In fact, the criteria for this axis expressly preclude the consideration of family systemic processes:

> Axis II is intended to diagnose the presence of a clinical problem in the child's relationship with the primary caregiver, usually the parent. When a disorder exists, it is specific to a relationship, meaning that the child's behavioral disturbances while interacting with *one* caregiver are not observed in the child's relationships with *other* caregivers. If the relationship disorder does not affect the child's functioning in other domains, then a diagnosis would be given only on Axis II.

RDoC

Although currently an early work in progress, the National Institute of Mental Health has spawned an initiative to develop new ways of classifying mental disorder that may have a significant impact on diagnostic theory and practice in times to come. The Research Domain Criteria (RDoC; National Institute of Mental Health, 2011) propose to develop classifications that are based upon neuroscience related to major systems underlying human behavior, including negative valence, positive valence, cognitive, social process, and arousal/regulatory systems (Cuthbert & Insel, 2013). Although feared by some to comprise an attempt to reduce all of psychopathology to a matter of biology, there is potential within the proposal to incorporate family relational dimensions. As the authors of

the RDoC proposal state, "The central nervous system is exquisitely sensitive to interaction with various elements of its environment . . . environmental influences may thus be considered as another critical dimension of the RDoC matrix" (National Institute of Mental Health, 2011). In this regard, as Denton and Bell (2013) pointed out, "For humans, an important environmental influence is interaction with the important people in our life" (p. 152)—that is, the family.

Alternative Approaches to Diagnosis from a Systemic Perspective

Global Assessment of Relational Functioning

One explicit attempt to integrate family systems perspectives into the *DSM* was the development of the Global Assessment of Relational Functioning (GARF). Spearheaded by a taskforce initiated by Lyman Wynne and the GAP (1996), the GARF was designed to provide an assessment of the quality of adaptive family functioning parallel to the existing individual-based global assessment of functioning in the *DSM*. Using the GARF, family functioning is rated along three dimensions presumed to reflect the ability of the family system to meet the instrumental or emotional needs of its members: (1) *problem-solving/interactional skills* (e.g., negotiating family rules, adapting to stress, communicating, resolving conflict); (2) *organizational structure* (e.g., maintenance of boundaries generally and developmentally specific boundaries such as those between children and the marital subsystem; hierarchical structure with parents retaining an executive role in the family; and an appropriate distribution of power and responsibility); and (3) *emotional climate* (e.g., openness to expression of feelings; involvement, caring, and commitment to one another; sharing of values; affection; and appropriate sexual functioning; Yingling, Miller, McDonald, & Galewaler, 1998). Although the GARF was included in the *DSM-IV* as an Axis Provided for Further Study, a body of empirical support for its use does not appear to have been established. Moreover, the GARF still may not satisfy the systems theory mandate to view the heart of the problem as located in the family system rather than viewing qualities of the family relationships as adjunctive to the individual's mental disorder.

Relational Diagnoses

A more thoroughgoing challenge to the individualistic perspective on diagnosis was launched by a consortium of family systems thinkers who represented some 15 different organizations (Kaslow & Patterson, 2006). Throwing down the gauntlet, this coalition argued, "In our view, a defect of the current diagnostic manual is the explicit restriction to syndromes that occur 'in a person.' We recommend recognition of syndromes that are best defined and identified as occurring between persons, as relational or interpersonal syndromes. These relational syndromes often entail serious personal dysfunction and warrant inclusion as mental disorders" (GAP, 1989, p. 1492). Although not completed in time to be considered for the *DSM-IV*, this group proposed a Classification and Diagnosis of Relational Diagnoses that reformulated diagnoses to take into account the relational network that both contributes to the etiology of disorders and is as essential contributor to their treatment (GAP, 1995).

The approach taken by the GAP was to base their scheme for the categorization of families on their structure and function and to include two major divisions. The first consists of disorders within one generation, including couples (i.e., conflict with or without physical aggression; sexual dysfunction; sexual abuse; divorce dysfunction; and induced psychotic disorder, aka folie à deux) or siblings (conflictual disorder, physical or sexual abuse, and folie à deux). The second division includes disorders spanning multiple generations, whether focusing on children and youth (e.g., intrafamilial child sexual or physical abuse, under- or overinvolvement, under- or overcontrol, communication deviance) or adult offspring and their parents (e.g., physical abuse or neglect, under- or overinvolvement, cutoff or conflictual communication). Paralleling the *DSM*, for each diagnosis the scheme outlines the diagnostic and associated features, predisposing features, prevalence, course, sequelae, and guidelines for differential diagnosis.

Proposed Classification of Relational Disorders. Subsequent work revealed a wide diversity of perspectives among relationship- and family systems-oriented scholars, which suggested that a consensus on the content—as well as the utility and even the ethics—of relational diagnosis would be difficult to achieve (Kaslow, 1996). Nonetheless, some intriguing specific proposals were laid out for how disorders might be placed into a relational paradigm. For example, Kaslow, Deering, and Ash (1996) put forward a set of criteria for a Relational Diagnosis of Child and Adolescent Depression to be applied when the child met *DSM-IV* not only for a diagnosis in the depressive spectrum but also in a context in which maladaptive family patterns of relating were associated with the development

or maintenance of the disorder. Problematic patterns of relationship in the family that might cause or exacerbate depression were specified as insecure attachment, low cohesion and support, child maltreatment, excessive parental controllingness, high levels of conflict accompanied by ineffective conflict resolution, poor affect regulation, impaired communication, transmission of depressogenic cognitions, and poor fit between the child's temperament and the family's style of relating.

Subsequently revisiting this issue, Beach, Wamboldt, Kaslow, Heyman, and Reiss (2006) again made the case for the inclusion of relational diagnoses in the forthcoming *DSM-5*, citing the burgeoning research evidencing the powerful associations between relational dysfunctions and both child and adult mental health. Helpfully, these authors propose a distinction between *relational disorders*, which are recognizable as clinically distinct constellations of symptoms that interfere with functioning or cause distress, and *relational risk factors*, which might exacerbate a disorder but are not themselves the focus of clinical attention. Beach and colleagues' scheme also differentiates between problematic relationship processes that are overarching and negatively affect functioning across domains (e.g., domestic violence) and those that are specific to the etiology of a specific disorder (e.g., intrusive feeding in the case of infant eating disorders or coercive parenting in the case of conduct disorder). Whereas the *DSM*'s V codes might accommodate the former, a relational diagnosis framework is necessary to elucidate the latter. Consequently, Beach et al. (2006, p. 366) made five concrete and pragmatic suggestions for strategies that would allow future diagnostic manuals to address relational factors:

> (a) devise clear criteria for the relational disorders currently depicted in V codes, perhaps creating a relational disorder category on Axis I; (b) develop an axis devoted to relational risk factors with clear criteria for assessing empirically supported contextual processes that might be the target of clinical intervention, including both family and multisystem contexts; (c) use relational specifier codes to indicate important disorder-specific relational processes; (d) elaborate relationship criteria for existing disorders where appropriate; and (e) describe relevant relational processes in the text associated with the disorder. In addition to these five approaches, it might also be useful to provide guidelines for relational formulations in an appendix or a companion volume to guide clinical use of the new information regarding relational processes. For each option, enhancing the current system could stimulate new research, guide the integration of applied and basic research findings, and inform services.

Relational Syndromes

Another attempt to give a relational focus to diagnosis was Heyman and colleagues' (2009) proposal for the creation of a series of new *relational syndromes*. These authors identified 11 distinct relational patterns or syndromes within families that, they argued, could be identified and for which reliable and valid diagnostic criteria could be developed. The specific relational syndromes include partner relational problems, parenting problems, parent–child relational problems, partner maltreatment (physical abuse, emotional abuse, sexual abuse, and neglect), and child maltreatment (physical abuse, emotional abuse, sexual abuse, and neglect). To demonstrate their point, the authors devised formal *DSM*-style criteria and empirically tested the validity of three of their proposed disorders: partner relational problems, child physical abuse, and parenting problems. Parallel to the process used in the validation of the *DSM*, Heyman and colleagues engaged in a series of iterative steps involving developing, field testing, and refining their criteria, assessments, and decision-making rubrics, using a sample of families in the U.S. military referred for child maltreatment allegations. Although the strong evidence presented in support of the validity, reliability, and utility of these relational diagnoses mustered by these investigators offers a valuable step forward, a limitation of this work in the current context is that the diagnoses offered do not derive from a family systemic perspective in that the relations involved are all dyadic (e.g., partner–partner, parent–child) and that they do not so much capture the interpersonal dynamics of family relational problems as identify negative sequelae of those problems.

Relational Diagnosis in DSM-5 and ICD-11

Family systems thinkers have continued their efforts to bring relational diagnoses into the forefront of the newest diagnostic systems. For example, *DSM-5* had a promising start when members of the *DSM-5* task force acknowledged the value of the construct of relational disorders (First et al., 2002) and invited workgroups devoted to relational assessment and relational diagnosis to participate in discussions leading up to the development of the new diagnostic compendium (Wamboldt, Kaslow, & Reiss, 2015). The workgroups even progressed to the point of conducting field trials demonstrating the reliability and clinical utility of relational diagnoses, such as parent-child relational problem (Wamboldt, Cordaro, & Clarke, 2015). However, as *DSM-5*'s developers acknowledged, including relational disorders would necessitate "a conceptual shift in the *DSM*'s exclusive focus on the diagnosis of individual

patients" (Kupfer, First, & Regier, 2002, p. 157), a shift they ultimately were not willing to make. In the final version of *DSM-5*, the relational diagnosis workgroups' recommendations were overlooked. Relational problems were again relegated to the V code section of the manual which, although providing expanded and clarified definitions that improve on *DSM-IV*, does not include reference to the specific impacts that help raters to diagnose them reliably (Foran, Whisman, & Beach, 2015; Smith Slep, Heyman, & Foran, 2015; Heyman, Smith Slep, & Foran, 2015; Wamboldt, Cordaro, & Clarke, 2015).

In contrast, the concurrent effort to develop the new *ICD-11* criteria has appeared much more receptive to the inclusion of relational problems, albeit with due concern regarding their cross-cultural appropriateness (Foran et al., 2013). As detailed in Wamboldt, Kaslow et al.'s (2015) overview, at the time of this writing field trials are ongoing to test the reliability and clinical utility of seven relational disorder diagnoses: intimate partner relationship distress, intimate partner physical abuse, intimate partner psychological abuse, parent–child relationship problem, child physical abuse, child neglect, and child psychological abuse. Although a promising step, as noted earlier, these relational diagnoses still largely represent dyadic (i.e., couple, parent-child) problems and are unidirectional (i.e., abuse of one family member by another) rather than representing whole-family processes nor the transactional dynamics inherent to family systems thinking.

CONVERGENCE AND DIVERGENCE BETWEEN FAMILY SYSTEMS AND DEVELOPMENTAL PSYCHOPATHOLOGY

Points of Divergence

As Davies and Cicchetti (2004) noted, a number of points of divergence are apparent between developmental psychopathology and family systems models. One is the unit of analysis; whereas developmental psychopathology generally has been concerned with patterns of adaptation or pathology within the individual, family systems theory has focused on maladaptation or positive adjustment as a function of the whole family or the interrelations among those comprising it. Second, whereas much of the early family systems work emerged in the context of attempts to understand adult psychopathology, developmental psychopathology has mostly been the purview of childhood research. Third, consistent with their clinical origins, many family systems theorists have utilized case studies and qualitative methods to validate their ideas, whereas the epistemological tradition in developmental psychopathology has leaned decisively toward quantitative methods and large-sample research. Fourth, whereas developmental psychopathology has been dedicated to understanding the continuum between normal and abnormal development (Cicchetti, 2006; Sroufe, 1990), with notable exceptions (e.g., Walsh, 2003), the focus of family systems theory generally has been on the study of dysfunction among clinical populations.

Points of Convergence

Despite these differences, some points of convergence promise to contribute to a rapprochement between the fields. One of these is a shared appreciation for systemic principles, including the individual and family context as open systems involved in ongoing dynamic transactional interchange with one another as well as the larger environment (Davies & Cicchetti, 2004). Holism also represents a shared assumption, in regard to both individual organisms and families, such that dimensions of individual development and the behaviors of family members cannot be understood in isolation but rather must be viewed within the context of the greater gestalt that gives them form and meaning. Appreciation for multideterminism and multifinality also can be found in either perspective, in that both developmental psychopathology and family systems acknowledge that a variety of child outcomes may ensue from the various risk and protective factors that impinge on development. Last, both systems of thought share a belief that systems follow lawful processes of successive hierarchical organization and integration, whether in the context of individual or family development. Therefore, the two perspectives might increasingly begin to complement one another in new theoretical work that strives for a true integration of the two, particularly given recent methodological advances in the tools available to test dynamic, systemic, multidimensional, interactional, and developmental models.

Toward an Emergent Synthesis: What Can Family Systems Theory Offer Developmental Psychopathology, and Vice Versa?

Focus on Process

One of the ways family systems theory might benefit from a better integration with the developmental psychopathology perspective would be to focus more on underlying processes and mechanisms rather than on the content of family variables. In other words, rather than judging

families as enmeshed versus disengaged or balanced versus triangulated, better questions might be: What are the *functions* that different kinds of family systems might serve at successive stages of development? What are the *developmental capacities* that can be supported in different ways by different family types for optimal adaptation at those stages? How do systemic-level *interrelations, transactions,* and *dynamic processes* provide a context in which those developmental attainments are achieved or thwarted? How can the developmental needs of various family members be *coordinated, balanced, negotiated,* or *synchronized* to provide a context conducive to optimal functioning for all subsystems—interparental, parent–child, sibling—and the interrelations among those subsystems?

Multiple Levels of Analysis. A valuable contribution of research from the developmental psychopathology perspective is an appreciation of interdisciplinarity and the importance of studying phenomena from multiple levels of analysis, including intrapersonal and interpersonal, observed and perceived, and psychological as well as biological processes in addition to the transactions among these sources of influence on the development of individuals and systems (Cicchetti, 2008; Cicchetti & Dawson, 2002). Although such efforts would not be not be incompatible with a family systems point of view—and certainly there is a growing body of research that includes family variables among the levels of analysis considered in multilevel investigations (e.g., Burnett & Cicchetti, 2012)—little research from a family *systemic* perspective to date has taken full advantage of the opportunity to explore the intersections among these sources of variance.

Family Resilience

Another important developmental psychopathology construct that is reflected—yet too often neglected—in the family systems literature is that of resilience, the capacity of individuals and families to function adaptively in the face of adversity (Cowan, Cowan, & Schulz, 1996). As Minuchin, Colapinto, and Minuchin (2007) noted, the focus on much of the research attention to families has been on the dark side and the ways family systems act as sources of risk and psychopathology. However, these authors point out, there is indeed a bright side involving the ways that families serve protective functions and foster resilience. Even among the most disadvantaged and stressed families, they argue, those who look closely will see characteristics such as loyalty, connection, affection, and shared identification that serve as sources of strength.

One early model of family resilience from a stress and coping perspective was offered by McCubbin and McCubbin (1989). They proposed that family success in overcoming adversity involved the intersection of several factors: the balance between a family's *vulnerabilities* (e.g., the presence of ongoing stressors or pressures associated with stage of life challenges) versus its *strengths* (e.g., resources for resisting stress such as the ability to adjust to change), its *structural patterns* (e.g., cohesiveness, organization, communication, and balance), the availability of *coping strategies* (e.g., the presence of good problem-solving skills), and the family's *appraisals* (e.g., subjective meanings given to the stressful event that are non-toxic and allow for hopefulness in the face of adversity).

A more recent well-articulated conceptualization of resilience from a family systems perspective has been developed by Walsh (2003), who proposes that familial adaptation in the face of stress or challenge involves three key domains of resilience. The first domain involves the family's *organizational patterns,* which include the family's clarity of structure, capacity for flexibility, and ability to mobilize necessary social and economic resources. The second domain, *communication and problem solving,* includes family members' ability to communicate clearly with one another, express emotions openly, and collaboratively work toward shared goals in ways that reflect resourcefulness, conflict resolution, fairness, and reciprocity. Finally, echoing the McCubbins, the third comprises the *belief systems* that influence how family members make meaning of adversity, preserve hope, and harness spirituality to serve as a source of strength and even to achieve posttraumatic growth and transcendence.

Although valuable and insightful, these models of family resilience have been relatively unintegrated into the literature on developmental psychopathology to date. Moreover, just as has the risk literature, the research on resilience has tended to focus on family variables rather than family systemic processes. Well-researched sources of resilience include a wide range of family factors, such as economic advantage, cohesion, and cultural engagement (Masten & Powell, 2003; see Kerig, Ludlow, & Wenar, 2012). But less well-heeded is Rutter's (1990) call for resilience research to turn from a focus on static factors to the investigation of *protective processes*—the underlying mechanisms by which such family characteristics as affluence, education, organization, and the like come to have beneficial effects for the developing person. Rutter's

four proposed protective processes are readily translated into a family systemic framework. For example, Rutter's protective process of *reduction of risk impact* may be conferred by membership in a family system that provides safety, warmth, and structure and buffers its members from external stressors; for example, a family with a strong sense of ethnic identity may serve to protect children from the harmful effects of racism. In turn, *interruption of negative chain reactions* may occur through engagement in positive transactions in which family members reciprocally influence one another's behavior in beneficial ways; an example of this is Wynne's concept of the differentiated individual who helps to diffuse tensions between two family members rather than becoming inveigled or triangulated in their quarrels. *Self-efficacy and coping* in the family context is captured well by Walsh's (2006) description of family strategies for making meaning of adversity in ways that confer a shared sense of optimism and affirmation of the family's strengths. Finally, *opening of opportunities* may take the form of family receptivity to individuality, differentiation, and even risk-taking among its members as they explore identities, lifestyles, or extrafamilial goals; an example here might be the family that not only passively accepts but also actively supports and validates the life choices of its sexual minority status child.

An intriguing process-oriented view of the family's contributions to the development of resilience is offered by Fivush, Bohanek, and Duke's (2005) studies of family narratives. The investigators first developed a measure assessing the extent to which children were knowledgeable about their family history (e.g., Do you know where your grandparents grew up; where your parents met; the story of your birth?). Their investigations showed that awareness of their family's history was one of the best predictors of children's self-esteem, perceived control, and positive view of their family. Subsequently, the investigators taped family conversations at the dinner table and distinguished among three types of family narratives: ascending (stories of unbridled family success), descending (stories involving family misfortune), and oscillating (stories in which the family was depicted as experiencing but overcoming adversities). Taking advantage of a natural experiment, the investigators were able to assess youth before and after 9/11 and found that growing up in a family characterized by oscillating narratives was associated with youths' resilience and the ability to modulate the effects of stress. Fivush and colleagues refer to the capacity that is fostered by these family narratives as an intergenerational self—the perception that one is part of something bigger with a place in history and the greater scheme of things (Fivush, Bohanek, & Zaman, 2011).

Infusing Development into Family Systems Theory

Another of the salient characteristics of the developmental psychopathology approach is the importance it places on understanding the emergence, organization, and integration of psychological, interpersonal, and biological processes over time in the ontogeny of the developing person. In contrast, with the notable exceptions described already, work in family systems theory has not consistently been attentive to developmental factors. In fact, many of the early systemic theory builders based their observations on families of adult children and did not explicitly give consideration to how those processes might relate differently to families with children at different developmental stages. Even those whose work focuses on younger families tend make reference to children without discussing ways family processes might play out differentially with infants, toddlers, preschoolers, school-age youth, and adolescents.

As Cicchetti and Toth (2005) pointed out, however, considerations of developmental differences are essential to our understanding of how family processes might contribute to psychopathology or adaptation. What comprises an adaptive family process at one stage of development may in fact be maladaptive at another (Barnett, Manly, & Cicchetti, 1993). For example, close emotional synchronization among family members plays an important role in fostering secure attachment during infancy, whereas autonomy promotion is conducive to healthy functioning in adolescence. In addition, the function and meaning of family processes might change over the course of development in several ways (Kerig, 2005). First, the relative impact of particular family processes might vary as a function not only of the stage-salient issues the child is navigating at each stage (Sroufe & Rutter, 1984) but also of how the family system is organized so as to support or conflict with those developmental tasks. For example, given the salience of differentiation of self as a stage-salient task for the toddler years, family enmeshment might be particularly injurious to adaptive functioning during that developmental period. In turn, given the salience of autonomy strivings in the teenage years, child triangulation in interparental disputes might come to the fore as the family process most disruptive to adolescent development (Davies et al., 2004).

A second way development comes into play concerns changes in how each successive stage-salient task of childhood might challenge or interact with family systemic

processes. For example, the terrible twos, or practicing phase of separation–individuation, is characterized by an *ambitendent* (Lieberman, 1992) alternation between proximity and distance seeking that may be particularly difficult to accommodate by those family systems with less adaptable structures and less tolerance for negative emotionality. By the same token, parenting school-age children requires a delicate balance between the provision of a secure base and the simultaneous promotion of extrafamilial competencies (Cummings et al., 2000); therefore, the school-age child's increasing shift toward prioritizing peer relations and achievements outside the home might present a particular challenge to those family systems that are organized around family-centeredness.

A third developmental dimension involves how, over time, children's emerging developmental capacities allow them to become increasingly active agents and architects of family processes (Becker-Blease & Kerig, 2015; Cummings & Schermerhorn, 2003). This is seen most clearly in the developmental psychopathology construct of transactions, in which family members influence one another's behavior in ways that foster the emergence of new qualities in one another and in the relationship between them. With the increasing differentiation and sophistication of their cognitive, emotional, and interpersonal capacities across development, children have a wider range of capacities with which to influence parents, siblings, and the nature of the interrelations among the various subsystems in the family.

By the same token, one way children increasingly can exercise their agency with age is to extricate themselves from problematic family processes. Age opens up expanding opportunities for children to become engaged with extrafamilial activities and peer associations that emotionally and physically separate them from and potentially create a buffer against family stress. Thus, it may be that, over the course of development, the impact of maladaptive family system processes is weakened for those resilient children who enjoy prosocial activities and relationships outside the home. However, in keeping with the concepts of developmental pathways and canalization, it also is possible that there are constraining effects of a bad beginning such that, at least for children without such extrafamilial protective factors, "spending one's early years within the context of a negative or hostile family system sets them on a [negative] trajectory" (Leary & Katz, 2004, p. 596) from which it is difficult to deviate.

A fourth way development comes to play concerns age-differentiated expressions of family systems processes. Related to the idea of *transformations* in symptom expression across development (Sroufe, 1990), the manner in which family systemic processes such as triangulation, communication, or cohesiveness manifest themselves might be expressed differently among families that are focused on the tasks of raising infant, school-age, or emerging adult children. An excellent example of this is Shaffer and Sroufe's (2005) longitudinal work investigating boundary dissolution in the family system across developmental periods spanning early childhood to adulthood, which required the creation of unique coding systems to capture these processes as they were played out between parents and children in the toddler, preschool, school-age, and adolescent years. A reflection of the power of the underlying theory is its ability to inform the investigators with strategies for coding different manifest behaviors while detecting consistencies in their latent purpose and meaning in the family system. Despite differences in the specific behavioral signs indicative of spousification, for example, the investigators were able to identify lawful continuities and coherence in the underlying patterns seen among family members.

Another intriguing developmental line concerns the child's ability to engage in and coordinate the complexities of family process. Fivaz-Depeursinge and Corboz-Warnery (1999) use the term *triangular capacity* to describe the child's ability to track the exchanges between two parents and to enter their interaction in ways that influence—and at times improve—its flow. Although this is a capacity that is observed in infants as young as 3 months of age and to date has been investigated only in relation to the child–mother–father triad (e.g., McHale, Fivaz-Depeursinge, Dickstein, Robertson, & Daley, 2008), it seems reasonable to expect that, over the course of development, children's triangular capacities become more sophisticated and they are increasingly able to comprehend, mediate, or avoid becoming entangled within problematic interactions among increasingly complex combinations involving other members of their family, including quarreling siblings.

A fifth way development might impact family processes concerns the other side of the coin, namely, changes in how family members respond to children across different age periods in ways that impact the entire system. For example, enmeshed families that perceive differentiation of their members from the collective to be a source of threat might be characterized by overtly intrusive processes when children are young but by subtler and sophisticated autonomy-limiting processes, such as psychological control and guilt induction, when children reach the adolescent years. Sixth, there may be lawful developmental progressions in family systems processes such that some emerge as developmental antecedents of others (Kerig, 2005). Again,

boundary dissolution constructs provide an example here, whereby psychologically controlling processes might foster the development of enmeshed relationships in the families of young children, which in turn might set the stage for the later emergence of a role-reversed family system in which adolescents become preoccupied with caring for their parents' emotional needs.

Seventh, as was noted already, McGoldrick and Carter (2003) pointed out that it is not only children who develop over the life span. Parents also navigate different stage-salient issues over the life course and family systems themselves must change in order to accommodate these developmental processes. In the first stage of these authors' family lifecycle framework, the salient tasks involve members of a couple redefining their relationships with their families of origin and establishing a new partnership and level of intimacy with one another. The birth of a child requires a further reorganization of the system to accommodate this new family member and often involves a significant renegotiation of roles within the marital subsystem (Cowan & Cowan, 1992). Another major shift in family structure and functions occurs as children enter late adolescence, when the system must become more flexible to allow youth to more freely move in and out of the intimate hold of the family. The next stage involves launching the emerging adult into the world and realigning the family to encompass new members, including children's own intimate partners, in-laws, and offspring. The final stage identified in McGoldrick and Carter's (2003) family life cycle is a poignant one, in that parents in midadulthood are confronted with the new freedoms (or fears) represented by their now-adult children's independent status while often simultaneously taking on new caregiving responsibilities involving their own aging parents. Although the literature reflects appreciation of this model, it has not been subject to empirical validation, nor has it been incorporated into family systems research in a systematic way. In addition, criticisms of this model have included its limited applicability to diverse family forms as well as its focus on the tasks of family members rather than on changes related to the structure or function of the family as a system more broadly (Wagner & Reiss, 1995).

CHALLENGES TO FAMILY SYSTEMS THEORY

Bringing Fathers into the Fold

If we are truly to view family dynamics from a systemic rather than a dyadic parent–child perspective, it is necessary to acknowledge the unique role that fathers play in two-parent families. Family systemic thinkers can be credited with stimulating our thinking in this regard, and particularly with calling our attention to the long neglect of fathers in the literature on parent–child relations. Just as Phares (1992) queried humorously in the title of her seminal articles, "Where's Poppa?" and "Still Looking for Poppa" (Phares, Fields, Kamboukos, & Lopez, 2005), Minuchin (1985) quipped, "Psychological researchers created the single-parent family long before it was a characteristic of American society" (p. 296). In addition, however, family systems requires not viewing fathers as just a second parent and father–child relations as just a second dyad in the family but rather understanding the influence of father–child and father–mother relationships on the dynamics of the family as a whole (Lamb, 2013).

Feminist Critiques of Family Systems Theory

A pointed challenge to the prevailing wisdom in family systems theories in the 1970s and 1980s was raised by feminist scholars who noted the highly gender-stereotyped assumptions made about men's and women's "proper" roles in the family system (Hare-Mustin, 1978; Walters, Carter, Papp, & Silverstein, 1988). These were particularly noticeable given how strikingly at odds they were the zeitgeist of the emerging women's movement and evolution of gender roles in U.S. families and society more broadly during that era. In addition, feminist scholars have continued to raise questions about some of the unarticulated assumptions underlying systems theory that inform its practice in the therapy room, particularly in regard to dynamics of power, control, and hierarchy, both among family members and between the family and the therapist (Luepnitz, 1988). Instead, feminists have suggested that construals of family relationships and the relationships between family members and therapists can be based on assumptions of cooperation, mutuality, and egalitarianism more consistent with feminist perspectives (Hare-Mustin, 1994; Walters et al., 1988). In addition, these writers have argued, family systems also must be understood within the interconnecting contexts of race and class as well as gender (Silverstein & Goodrich, 2003).

Cultural and Ethnic Diversity in Family Systems

Attempts to integrate developmental psychopathology and family systems perspectives (e.g., Cox & Paley, 2003; Lynch & Cicchetti, 1998) have pointed toward the importance of transactional processes involving the larger ecological contexts that " . . . may shape, affect, and be affected by that which happens in families and children's lives" (Lindahl, Malik, Kaczynski, & Simons, 2004, p. 610). Consequently,

another challenge to family systems theories has been to demonstrate that models of family development, adaptation, and psychopathology are relevant across a range of cultural and ethnic groups (McGoldrick, Giordano, & Garcia-Preto, 2005).

Cross-Cultural Perspectives on the Family

As Kağıtçıbaşı (2005, 2007) argued, the family as a system must be viewed within the contexts of the larger cultural and social structures in which it is embedded. These contexts inform the sense of purpose that guides the family in all respects, including family structure, parents' socialization goals, family practices and rituals, and the emotional tone of family relations. Similarly, Chao (2000) suggested that the socialization goals imparted to the family vary across cultures, and those socialization goals guide parenting practices and family interactions in systematic ways. Culturally shared understandings of the purpose and meaning of family relatedness are further seen as embedded deeply in historical culture and as representing "accumulated knowledge of prior generations within that environment" (Keller & Otto, 2009, p. 997). Moreover, Bornstein and Cheah (2006) implied the family itself represents a microcosm of the overarching culture. The larger society imparts information about what is expected of families and their members (e.g., norms, customs, and shared values), but parents then "select, edit, and refashion" (p. 15)—in other words, assimilate—which information to communicate to their children. In essence, then, each family can be construed as its own culture in which parents and caregivers pass on their attitudes, behaviors, and values to their children. Although children take in and process the information received from their parents, this also should be seen as an active, bidirectional process in which both parents and children contribute to the making of meaning. In sum, culturally constructed family beliefs and behaviors directly influence the development of children and contribute to the transmission of cultural information across generations.

Although much of the theory on cross-cultural differences among families has focused on a distinction between collectivism and individualism (e.g., Markus & Kitayama, 1991), Kağıtçıbaşı (2005, 2007) offered a critique that suggests this duality does not reflect the complexity of family forms associated with culture. In Kağıtçıbaşı's model, underlying the cultural expectations that influence family functioning are two dimensions: *agency*, defined by the poles of autonomy and heteronomy; and *interpersonal distance*, defined by the poles of separateness and relatedness. Using the matrix of these two dimensions, Kağıtçıbaşı proposed that cultures may promote each of four family interaction patterns that influence parenting styles, and in turn, the development of the child's sense of self. The first of these is a family pattern of *interdependence*, defined as high in relatedness and low in autonomy. Interdependence arises as an adaptive family form among rural, farming communities in which family members must rely heavily on one another for meeting both emotional and material needs, often seen in developing societies. Such families are typically hierarchically arranged with parents adopting an authoritarian parenting style related to the instrumental functions of the family, as it strives to ensure the material well-being of its members. In particular, because children are needed to provide economic and practical support to the family, especially as parents age, their independence from the family is not functional and may even represent a threat to family well-being. Consequently, the emotional functioning of the family is one that promotes permeable boundaries in interpersonal relationships, thereby fostering in children the development of self-constructs that are profoundly relational.

A second family pattern, *independence*, is characterized as high in autonomy and low in relatedness and is more typically seen among families in Western urbanized, industrialized societies such as the United States, at least among those in the majority White subculture. Such families are organized in ways that help their members to function well in the context of cultural expectations for autonomy and self-reliance. Parents in independent families tend toward an egalitarian or even permissive mode of interaction with their children whereas the family structure is defined by clear interpersonal boundaries that foster the development among children of a separate sense of self. The third family pattern proposed by Kağıtçıbaşı (2007) is labeled as *hierarchically neglecting*, composed of families that are high in separation and low in relatedness.

The fourth family model Kağıtçıbaşı (2007) proposes, which has been overlooked in other cross-cultural family models, is that of *psychological interdependence*, described as a dialectical synthesis of the interdependent and individualistic forms combining high levels of relatedness with high promotion of autonomy. This family mode is most likely to be seen among the rising class of suburban families in modernizing societies that have traditionally embraced interdependence. Such families function in a cultural context in which members no longer remain dependent on the family system for providing material needs throughout the lifespan and yet continue to look toward one another as important sources of emotional

connection. Parents in these families tend to engage in authoritative parenting, combining structure and direction with warmth and emotional responsiveness, which fosters in children the development of a sense of self that is termed autonomous-related; that is, simultaneously both relational and independent.

> The weakening of intergenerational material interdependencies allows autonomy to enter child rearing. This is because the child's autonomy is not any longer perceived as a threat when his or her material contribution is not required for family livelihood. Nevertheless, because psychological interdependence continues to be valued, the connectedness of the growing child is desired, rather than separateness. Therefore, together with autonomy, there continues to be control rather than permissiveness in child rearing, and control may function as a centripetal rather than a centrifugal force (Kağıtçıbaşı, 2007, p. 411).

Intriguing support for this model is suggested by research emerging from a project spanning 10 countries in which changes in family attitudes have been assessed over the course of three decades and three generations (Bond et al., 2004; Kağıtçıbaşı & Ataca, 2005). With increasing modernization, all cultures studied have evidenced a decrease in the view of the child as a utilitarian entity and economic asset and a concomitant rise in the appreciation of the child's status as a psychological contributor to the family. "These results provide evidence for a shift from the family model of interdependence to one of psychological interdependence over three decades of socioeconomic development and urbanization. In line with these changes are changes in desired qualities of children" (Kağıtçıbaşı, 2005, p. 414).

Implications of Cultural Differences in Family Dynamics for Psychopathology

One of the implications of a cross-cultural perspective is that particular family processes may affect children differently depending on the larger context which lends them social meaning. For example, culturally derived values of *familism* may confer quite different meanings on family processes such as rigid boundaries or role reversal. Among cultures valuing familism, role complementarity between parents and children and tightly structured family systems with unbalanced power differentials may be conducive to positive child functioning in ways they are not in other cultural contexts (e.g., Lindahl & Malik, 1999). Similarly, the expectations for children's ongoing familial obligations their parents and siblings in such cultures may contribute to benign associations between processes such as parentification and youth functioning (Jurkovic, Kuperminc, Sarac, & Weisshar, 2005).

Historical Changes Affecting the Family System

Yet another challenge to family systems theory is its capacity to reflect how families operate and are construed across historical epochs. For example, in contrast to the supposition inherent in much of the early family systems literature that the prototypical family takes a nuclear form, one of the most evident changes in family systems over the past few decades in the United States has been the rising rates of divorce and resulting single-parent families. According to the most recent U.S. census, only 67% of children in the United States currently are living with both biological parents. Single-parent families provide a context for development that is rife with potential vulnerabilities and sources of resilience (Murry et al., 2001). Moreover, the alternatives involving one biological parent are numerous and include households encompassing three generations, extended family members, nonmarried cohabitating partners, and shared living arrangements among multiple families. In addition, although perhaps more slowly and more slightly than some would wish, there also is evidence for changing norms in U.S. majority culture regarding the role of the father in child development, with parents increasingly indicating an expectation that fathers will be actively involved in the lives of their children (Lamb, 2013).

Another phenomenon affecting what is perceived as normative in U.S. families is the increasing length of the adolescent period into age ranges (e.g., the mid-20s), which in other eras would have been considered to be part of the adult phase. Current U.S. culture's lack of clarity about when the adolescent period ends and adulthood begins seems to be reflected in the wide range of ways different developmental theorists discuss the stage-salient tasks involved in this transition. For example, as Kerig and Schulz (2012) observed, developmental tasks such achieving individuation and psychological independence from parents are listed variously by stage theorists as emerging during the early, mid, or late adolescent period, just as leaving the family home and assuming responsibility for independent decision making are variously associated with late adolescence and emerging adulthood (e.g., Arnett & Tanner, 2006; Micucci, 2009). Studies of emerging adulthood among middle-class majority families in the United States in fact suggest that such leave taking is for many youth only metaphoric in that declining economic

prospects force them to continue living in the family home late into their 20s or beyond (Kaplan, 2012).

These observations suggest a number of research questions that deserve further exploration. What are the effects on the family system of changing norms regarding the roles of fathers and the developmental status of adult children, particularly when those children continue to cohabit with their parents and thus the organization of the system retains the form of a traditional nuclear family? Even more to the point may be the need to ask questions about the effects on the family system of a lack of overall clarity among all family members, or a lack of consensus among its members, about these changing role expectations of parents and their adult children. How do these changes in family structure and function interact with larger societal definitions and expectations for the role of parents in the lives of their adult children, and vice versa?

What Is a Family? Diversity in Family Structures and Composition

As noted already, a frequent criticism of classic family systems theories is that they are based on a highly stereotypical model of what constituted a *family*. However, in U.S. culture as well as around the world, it is clear that not all families are composed of the nuclear pattern of a mother, father, and child. To be relevant across cultures and subcultures, family systems theory must rise to the challenge of recognizing and understanding the wide range of diverse family forms. These include single-parent (e.g., Murry et al., 2001) and same-sex parent (e.g., Farr & Patterson, 2013) families, multigenerational households (Jones & Lindahl, 2011), never-married, fragile families (Carlson & Högnäs, 2011), blended/step-families with complex combinations of coparenting arrangements (Burton & Hardaway, 2012), sibling-headed households (Richter & Desmond, 2008), and families that are emotionally connected yet physically separated by such challenges as parental military deployment (e.g., Bowen, Martin, & Mancini, 2013) or incarceration (e.g., Loper, Phillips, Nichols, & Dallaire, 2014). Each of these represents a legitimate family form, and in virtually all of these kinds of families the same sorts of processes arise as do amongst two-parent families, requiring the coordination of relationships across multiple boundaries (e.g., parent–child, child–sibling, sibling–parent) as well as synchronization among those involved in coparenting the child. Even when a coparent is completely absent from the picture, such as might be the case when a biological father maintains no relationship with the child, there are important but virtually unexplored family dynamics involved in how the resident parent permits or disallows his emotional presence in the life of the child and in the defining parameters of who is family. "Thus, it may be important to take into account family members who are absent as well as those who are present" (Cicchetti & Howes, 1991, p. 274).

Extended Family and Fictive Kin

Most typically mentioned in regard to diverse family structures are extended family members who, although perhaps relegated to the periphery in the stereotypic middle-class European American family, are viewed as core members of the unit in other cultural and ethnic groups (Jones & Lindahl, 2011). For example, among African American single mothers, 97% identify another adult as centrally involved in the parenting of their child: these include not only biological fathers, grandmothers, aunts, and other relatives but also friends (Jones, Shaffer, Forehand, Brody, & Armistead, 2003). The meaning of the term family in and of itself also is a subjective and diversely defined one, in that among some subcultures and individuals, the term refers not to blood relations only—or even at all—but rather to those who are perceived to be one's core source of support, emotional intimacy, protection, and identity. Thus families may be created on the basis of choice, with members defined by the instrumental and emotional roles they play rather than by bloodline. McHale and Irace (2011) expressed this nicely from a child's-eye point of view:

> Children see their families as a collection of individuals who loves and cares for them. And children are right: in the end, their welfare in the world will ultimately be protected and assured … by the collection of individuals who step up to take responsibility for their care and upbringing (p. 15).

In this regard, fictive kin, who are rarely included in family research, may play an unexamined role in the family processes that influence child development. This may be true particularly among certain cultures, such as Mexican American families in the United States, in which the system of *compadrazgo* ensures that a significant place in family life is reserved for non–blood-related members such as godparents (Luna et al., 1996). The significance of fictive kin in a child's life also may increase with age, as adolescents become increasingly active agents in niche picking and selecting their own interpersonal environments (Becker-Blease & Kerig, 2015; Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Scarr, 1992). The relative risk or protective influence of these fictive familial relations likely is complex. Although having "someone to turn to" may

provide youth with a sense of felt security that promotes adaptation, evidence also suggests that troubled youth tend to gravitate toward others who share their attitudes, behaviors, and lifestyles and thus reinforce maladaptation (van der Zwaluw et al., 2009). A particularly compelling example is provided by disaffected gang-involved youth, for whom the gang is explicitly identified as a substitute family and fellow gang members are perceived quite literally as brothers and sisters (Miller, Winn, Taylor, & Wiki, 2012).

Three-Generational Families

At the same time that many young people in the United States continue (or return to) living in their parents' home after reaching their majority, an increasing number do so accompanied by children of their own. The phenomenon of three-generational families is not new in U.S. society, having long been a family form common among low-income and minority communities, for whom cohabitation is both a means of survival and a culturally sanctioned choice (Goodman & Silverstein, 2006). Even when cohabitation is not involved, grandparents play a large role in childrearing in ethnically diverse families; for example, among single mothers in Jones et al.'s (2003) African American sample, a full 31% reported that the maternal grandmother was their key source of coparenting support. Further, research shows that the quality of the coparenting relationship between mothers and their mothers makes an important contribution to children's adjustment (see Jones & Lindahl, 2011).

In an in-depth look at this family form, Pittman, Wakschlag, Chase-Lansdale, and Brooks-Gunn (2012) discuss the intersecting developmental, family, and sociodemographic factors impacting three-generational families. Whereas individuation-promoting family interactional styles generally are found to provide a good fit for the stage-salient issues of the late adolescent period and to be associated with positive outcomes for youth, this assumption is complicated by issues of race, socioeconomic status, and the unique developmental challenges of becoming a young mother, or of becoming a young grandmother, among coresiding three-generational families. The ideal balance between the provision of adequate support and autonomy may be difficult for mother and grandmother alike to discern and to create. Further, although it can be expected that "grandmothers who support the young mothers' independence while remaining emotionally available facilitate their daughters' assumption of adult roles, . . . the specific dimensions of a supportive relationship between mothers and grandmothers that are linked to positive parenting have not been well-delineated" (p. 183). Pittman and colleagues resolved this dilemma by proposing that the interpersonal skills that allow young mothers to successfully navigate the stage-salient issues of individuation in adolescence, and the qualities of their own parents' behavior that support that individuation, are precisely the qualities that undergird young mothers' success in parenting their own children. These qualities include self-assertion, which evolves into firm limit-setting with children; perspective taking, which supports the capacity to accommodate to a child's point of view; separateness, which contributes to the ability to support children's autonomy; and mutuality, which takes the form of emotional connection and availability in parenting. As they suggest, "mothers who can maintain a sense of closeness with their own mothers, despite their differences, may also be more likely to feel emotionally connected to their children as their children become increasingly independent" (p. 184). In sum, the authors propose a lawful intergenerational continuity in relational functioning between the adolescent mother, her adult mother, and the toddler-age grandchild.

These ideas were confirmed in Brooks-Gunn's (1990) study of 96 African American mothers, grandmothers, and 3-year-old children, almost half of whom were graduates of an earlier study that followed teen mothers and their families over the course of 17 years. Thus, these were multigenerational families including teenage mothers. Results showed that, as expected, individuation in the mother–grandmother relationship was linked to the mother's own parenting practices and ability to demonstrate a balance between emotional responsiveness and appropriate parental control with their children. Moreover, these effects extended into the next generation, with children of mothers whose grandmothers supported individuation showing a greater ability to work independently on a challenging problem-solving task while still being responsive to maternal direction.

Teen Fathers

Compared with teenage mothers, adolescent fathers have been largely absent from studies of multigenerational families. This is likely because young fathers are more rarely coresident with the families of their child's mother but also because these couple relationships are highly vulnerable to dissolution and the father's subsequent disengagement from parenting (Carlson, McLanahan, & Brooks-Gunn, 2008). Nonetheless, as Florsheim and Moore (2012) pointed out, "Not all young fathers fail as relationship partners nor as parents; some are in fact able to manage the frustration, anxiety, and demands that accompany the monumental responsibility of parenthood and maintain

(or establish for the first time) positive relations with both their partners and children" (p. 201). Valuable research has been conducted examining the factors that promote successful coparenting between adolescent fathers and their partners (Carlson & McLanahan, 2006; Florsheim & Moore, 2012) and positive engagement between teen fathers and their children (Fagan, Palkovitz, Roy, & Farrie, 2009), as well as the intersection of these interpersonal processes (Florsheim et al., 2003; Moore & Florsheim, 2008). Nonetheless, an interesting set of questions remains to be answered from a family systemic perspective. For example, what are the adaptations that must be made in the young man's family of origin to accommodate his attainment of this new status of father? How do systemic processes in the paternal and maternal families of origin serve to support, foster, or interfere with the integration of this new relationship, and these new family members, into the fold? What are the three-generational family dynamics that potentiate teen father's motivation, and competence, to engage with the caregiving tasks of new parenthood? Some research has emerged to suggest that grandparents are important mediators of the relationships between teen fathers and their offspring. For example, positive relationships with the child's mother are one of the best predictors of teen fathers' involvement with their offspring (Gavin et al., 2002), and Krishnakumar and Black (2003) found that adolescent mothers enjoyed higher quality relationships with their baby's father if the maternal grandmother reported more positive affiliations with both teenagers.

Grandparent-Headed Households

In addition to three-generational families in which parents, children, and grandparents coreside, family systems theory also needs to accommodate family forms in which the middle generation is absent. Grandparents who are raising children on their own might easily be considered as caregivers interchangeable with biological parents in terms of the roles they play in the family system, but there is some reason to suspect that this is an assumption that should be questioned. Grandparents may not feel fully enfranchised to take an executive role and make decisions, either within the family or in the context of their interactions with extrafamilial contexts such as schools and social welfare agencies. This ambiguity may provide a source of tension that reverberates throughout the family system, one that is further complicated when the permanence of the custody arrangement also is not secure (Boyd-Frankin, 2003). Further, the demands of parenting a second generation may not coalesce well with the life stage of aging grandparents, particularly those in failing health, who may be oriented toward relinquishing caregiving responsibilities and moving into the phase of fulfilling their own personal late-life existential tasks. The strain that this added parenting role confers may spill over in negative ways onto the family dynamics and compromise the ability of the system to most favorably support the development of its members. On the other hand, grandparent-headed households are not a new family form, particularly among nonmajority culture communities, and a long history suggests that they also can be adaptive, overcome challenges, and provide positive contexts for children's development (Jones & Lindahl, 2011).

FAMILY SYSTEMIC RESEARCH METHODS

Before turning to a review of the current state of knowledge regarding developmental psychopathology from a family systems perspective, it is necessary to give consideration to the methods that inform that body of research. As with family systems theory itself, those research methods have been various. The diversity of methods used in family systemic research necessarily reflects the diversity of underlying theoretical models and purposes of the research.

Self-Reports of Family Processes

A number of self-report measures have been developed to assess individual family members' perspectives on the family system, with such subjective self-reports providing valuable information about how each member of the family perceives, understands, or is affected by family system processes. Among those developed to reflect a specific theoretical conceptualization of families, a premiere example is the Family Adaptability and Cohesion Evaluation Scale (FACES IV; Olson, 2011), which is designed to capture family members' perceptions of the dimensions of cohesion, flexibility, and communication encompassed by Olson's circumplex model.

In turn, widely used empirically derived omnibus measures of family characteristics include the McMaster Family Assessment Device (Epstein, Baldwin, & Bishop, 1983), which assesses family members' perceptions of a wide variety of traits, including problem solving, communication, clarity of roles, affective responsiveness, affective involvement, behavioral control, and general family functioning. Similarly, the Family Assessment Measure (Skinner, Steinhauer, & Santa-Barbara, 1983) casts a wide net, assessing characteristics such as task accomplishment,

role performance, communication, affective expression, involvement, control, and values and norms in the context of the family as a whole, dyadic relationships in the family, and one's perception of oneself as a family member.

One of the interesting but challenging outcomes of gathering multiple family members' perspectives is that they often fail to agree to one another; for example, in a study comparing ratings from multiple members of more than 26,000 families, Georgiades, Boyle, Jenkins, Sanford, and Lipman (2008) found that 54% of the variance in ratings was not shared among members of the same family. If family members' ratings are discrepant, what then is the data point to be used as the indicator of family warmth, cohesion, problem solving, or communication? Some research also suggests that disagreement about the quality of family relationships is higher in families that are functioning more poorly (Georgiades et al., 2008; Kerig, 1995); thus, the level of discrepancy among family members is a valuable data point in and of itself.

Observational Coding

Perhaps the richest source of information about systemic family processes derives from observations of family members as they interact with one another in the home or in the laboratory. Observational methods provide a unique source of data in that they are able to capture aspects of family interactions of which the participants themselves are not directly aware. "People behave in ways that are discrepant from their self-perceptions, and only direct observation can capture their behavior independently of their appraisals of it" (Kerig, 2001, p. 2). However, whereas the perspective of an outsider is useful, it is not necessarily more objective or more accurate (Weiss & Heyman, 2004). Following in the footsteps of the clinicians who founded the field, researchers devise systems for categorizing observed behaviors in ways that are lent meaning through the lenses of the investigator's underlying theoretical framework. As Margolin (1990) stated, "The coding systems themselves are derived out of researchers' a priori assumptions as to what constitutes important communication differences . . . Even when such differences are empirically validated, we have no way of knowing whether the differences discovered [are] the only ones, or are the most significant ones" (p. 210). Thus, observational methods present a number of challenges.

The Role of Theory

One of the implications of the diverse theoretical assumptions underlying family observational coding systems is that the meanings of coded behaviors are construed differently and thus even coding systems that appear to be tapping the same constructs in fact may not be. Thus, for example, a specific interactional behavior, such as a positive statement, may be interpreted as a sign of enmeshment in one coding system versus positive affect in another. In addition, similar behaviors may have different functions in the family system when they are exhibited in the context of individual, dyadic, or triadic episodes. Therefore, it is difficult to extrapolate with confidence across studies and compare findings related to specific constructs when those were obtained using different observational coding systems (Kerig, 2001).

Level of Analysis

Another limit to comparability concerns differences in the level of analysis at which coding of family processes take place. Constructs that involve a high order of inference are most likely to require molar or *macrolevel* codes that attempt to extract overall patterns of meaning or coherence. An example is Fiese et al.'s (2001) system for coding family narratives, in which observers are trained to make judgments about overarching constructs such as narrative coherence, narrative style, and relationship beliefs. Such coding systems confer a number of advantages, including the capacity to take into account the context in which behavior occurs, to synthesize complex individual data points into an organized whole, and to capture complex relational patterns. Disadvantages of macrolevel coding, on the other hand, include the cognitive complexity involved that may require extensive training of coders in order to attain reliability, the necessity to make the right a priori choices of what observations to bring to the foreground and which to leave in the background, and the possibility of lumping together categories of behavior that are conceptually distinct from one another.

At the other end of the continuum, constructs that involve more discrete or concrete behaviors may lend themselves best to molecular or *microanalytic* coding systems. Microanalysis is particularly valuable for the study of contingent responding and the investigation of the direction of influence in family processes. For example, Kerig, Cowan, and Cowan (1993) used a microanalytic coding system to investigate ways marital discord was associated with gender-differentiated patterns of interaction between parents and children. Whereas overall levels of negativity were not observably higher in the interactions of maritally dissatisfied fathers and their daughters, analyses of contingent responding showed that fathers in unhappy marriages were more likely than other parents to respond negatively

to their daughters' assertiveness. Although such contingent patterns are discernible only through microanalytic coding, a distinct disadvantage of microanalytic systems is that coding such fine-tuned behaviors may be highly time-consuming; in some cases, as many as 20 hours are needed to code a single hour of family interaction.

A third level of analysis involves analyzing constructs at the midrange or *mesoanalytic* level, which involves an integration of both of the aforementioned approaches. Rather than coding discrete behaviors during each speaking turn or brief (e.g., 10-second) time interval as in microanalytic systems or providing a single rating for the entire period of family interaction as do macroanalytic codes, mesoanalytic systems involve applying a summative evaluation of each participant's behavior during a set period of interaction. Thus, such systems allow for the analysis of transactional patterns in exchanges between parents and children while organizing those observations into higher order constructs. An example of a mesoanalytic system is Mahoney, Coffield, Lewis, and Lashley's (2001) Meso-Analytic Behavioral Rating System for Family Interactions, which was designed to assess both parent and child contributions to triadic interactions. Parent rating scales include hostility, involvement, and coercive control, whereas child rating scales include overt noncompliance and prosocial behavior.

Dimensions of Family Interaction

Coding systems also differ in the strategic choices they make regarding the underlying dimensions of family relationships to be assessed. Copeland and White (1991) offered a musical metaphor for these dimensions. The tune is represented by the structure of the interaction (e.g., timing, sequencing, order of influence), the lyrics involve the content of what is said (e.g., commands versus questions), and the sentiment refers to the affective tone of the interaction (e.g., warmth, playfulness, tension). Assaying each of these dimensions requires a different kind of coding scheme and each provides information that would be difficult to infer from the others. Another dimension on which coding systems differ might be referred to as bandwidth, that is, how narrowly or broadly defined are the set of constructs they attempt to capture. Some are quite wide-ranging and attempt to assess a wide array of family behaviors, such as the Iowa Family Interaction Rating Scales (Melby & Conger, 2001), which include 60 behavioral rating scales divided into three categories (general interaction, parenting, and problem solving) each of which includes one or more measurements across four levels of the family (individual, dyadic interaction, dyadic relationship, and group interaction). In contrast, other coding systems are very specific to measuring a single construct, such as the Repair Attempts Observational Coding System (Tabares, Driver, & Gottman, 2004) designed to capture intimate partners' efforts to heal riffs in their relationship during an interaction.

Although numerous coding systems have been developed for assessing family interactions, one particularly well-validated and widely used measure that is specifically designed to measure dimensions associated with family systems theory is the System for Coding Interactions and Family Functioning (SCIFF; Lindahl & Malik, 2001). Global whole-family codes are provided for Cohesiveness (e.g., mutual appreciation of one another while working toward a common goal versus interpersonal distance and awkwardness). In addition, each parent is rated on a scale of Triangulation, consistent with Minuchin's (1974) description of the parent who attempts to engage the child in providing support, sympathy, or to side with him or her against the other parent. Boundary formation comprises a separate rating of the extent to which families appear to have dysfunctional cross-generational alliances, assessing as to whether relationships are balanced (e.g., all family members are equally close or affectionate with one another) or demonstrate dyadic alliances (e.g., detouring, in which parents band together to control or blame the child; parent–child coalitions, in which one parent and child evidence an overly close relationship that excludes the other parent; or else children actively side with one parent against the other). Last, a lack of any strong boundaries or alliances in the family is coded as evidence of disengagement.

Setting, Task, and Participant Reactivity

One potential limit to the generalizability and even validity of observations is that family members may react in various ways to being observed. Although this may be less of a concern in the present day, when many young adults grew up with video cameras ubiquitous in their daily lives, family observations nonetheless typically are conducted in contexts in which family members know they are being watched and evaluated by the investigators. Family members may react generally, such as by becoming self-conscious or stilted, or may react in specific ways, such as by attempting to disguise their typical interactional styles to make a favorable impression. To the somewhat morbid advantage of researchers, however, it has been suggested that families are better at faking bad than at faking good; for example, those who do not know how to resolve conflicts successfully find it difficult to simulate doing so (Copeland & White, 1991). A number of strategies

can be implemented to reduce family members' reactivity, including conducting observations under more naturalistic conditions, such as in the home, assigning them tasks that are engaging as well as realistic (e.g., to talk about an actual episode of conflict they recently experienced rather than attempting to generate a mock conflict in the lab), and to sample behavior over the course of multiple interactions.

The setting and task chosen by the investigator also bound generalizability. For example, perhaps as a function of participant reactivity, some family investigators suggest that there may be systematic differences between family interactions elicited in the home versus the laboratory. In particular, family members may be more likely to display conflict and negativity in the home setting than in the lab; however, little empirical attention has been given to this issue, and, as Brody and Stoneman (1990) cautioned, we are in danger of "confus[ing] the occurrence of behavior with its representativeness" (p. 208). Similarly, the choice of task assigned to the family during the interaction is a crucial one given the capacity of various kinds of tasks to elicit specific family behaviors and interactional patterns. Structured tasks offer the advantage of directing the family to engage in the kinds of interactions most likely to relate to the investigator's constructs of interest; for example, asking parents to discuss a parenting disagreement in the presence of the child might be an effective strategy to elicit triangulation, whereas assigning the family the task of planning a vacation may better draw out themes of overall warmth and cohesion, and challenging the family to solve a puzzle together might be a preferred way to assess decision making and cooperativeness. Consequently, the range of tasks assigned in family observational studies is vast, including free play, storytelling, problem solving, sharing a meal, resolving a conflict, jointly completing a questionnaire, and others. Guided by the purposes of their research, investigators may choose markedly different kinds of interactions to code, and thus the results of these research projects may be difficult to compare or summarize across.

The Contribution of the Child to Family Interaction

An important feature of the transactional perspective underlying the family systems model is the idea that children not only receive or react to parental behaviors but also are active agents in driving the family process (Cummings & Schermerhorn, 2003; Kerig, 2001b). However, first we must consider the question of who is considered to be a child in family observational research. As noted earlier, the study of some constructs in the family research literature has been accompanied by careful attention to how they are evidenced differently across different ages; for example, the developmentally attuned measures developed by Shaffer and Sroufe (2005) to assess patterns of boundary dissolution across the lifecycle. In addition, some observational coding systems are thoughtful about providing coders with guidance about how to rate behaviors in the context of their different expressions across different ages (e.g., Florsheim & Benjamin, 2001; Robbins, Hervis, Mitrani, & Szapocznik, 2001). Others include separate coding categories for adults and children, designed to be sensitive to the different ways they contribute to family process (e.g., Lindahl & Malik, 2001). But, in the wider pantheon of family coding schemes, it is surprising the extent to which this kind of attention to development is the exception rather than the rule. Often, the child is not contextualized by age but rather is construed as an actor whose behavior has the same range and thus can be coded with the same categories, across ages and even into adulthood. As suggested previously, with increasing cognitive, emotional, and interpersonal skills and capacities across age, we would expect that children are able to become increasingly agentic and impactful contributors to the tone, lyrics, and rhythms of the family music.

Moreover, a valuable distinction may be that between family processes that are *child-driven* versus *parent-driven* (Kerig, 2001a, 2001b). For example, triangulation of the child in interparental conflict that derives from parents' efforts to place their child in the middle might have distinctly different ramifications for the family system and child outcomes compared with the family in which the child volunteers for that role. Similarly, there may be different contexts that promote, and different meanings for family members regarding, boundary violations such as parentification that arise from adults turning to their children for care as opposed to children attempting to care for their parents (Kerig, 2005). The boundaries between these processes may be difficult to discern, however, given that one may well grow out of the other (i.e., children who have been trained to respond to parents' bids for nurturing may begin anticipating those needs and offering such care unbidden), and role complementarity may be such that, in some families, the child-like parent and the parent-like child transact with apparent seamlessness.

There are a number of other ways children can play a role in the choreography of the family system. One is through characteristics they bring to the interaction that are sources of either resilience or vulnerability for the child, the marriage, the sibling subsystem, or the family as a whole. For example, there is some research to suggest that parents of temperamentally difficult infants are more

susceptible to the negative effects of a strained coparenting relationship (McHale et al., 2004). In contrast, children with a higher capacity for physiological self-regulation might be relatively buffered from problematic family processes. In this regard, Leary and Katz's (2004) observations showed that hostile-withdrawn coparenting was not predictive of poor peer relations for those children with high vagal tone who were able to regulate their own emotional state. Considered transactionally, physiologically well-regulated children may be less likely than their peers to engage in negative interactions with others outside the home that, in turn, might exacerbate family conflict (e.g., if parents are called on to respond—or conflict with one another over how best to respond—to a complaint from school about a child's fighting with peers). In contrast, children with characteristics that represent valued family attributes (e.g., academic prowess, social skill, athleticism) might bring sources of esteem back to the family from extrafamilial contexts (e.g., positive reports from teachers, sporting or academic accolades, social contacts with prosocial peers and their parents), which buffer not only the child but also the family itself from discord, thereby conferring upon all family members a more harmonious home life (Kerig et al., 2012; Werner & Smith, 2001).

Analytic Strategies in Family Observational Research

As outlined by Sayers and McGrath (2004), the questions researchers seek to answer with their data, as well as the kinds of data they gather, lend themselves to different analytic strategies. For example, some questions may rest on the frequencies with which behaviors are demonstrated, either in terms of base rates, proportions, or durations. An example might be a study examining whether families living in violent communities evidence higher levels of interactional strategies designed to constrain autonomy among their members than do those in more affluent areas. A second kind of question concerns simple sequences in behavior; an example here might be a study investigating, in distressed couples characterized by a demand–withdraw pattern of behavior, whether children are most likely to reciprocate the affect of the demanding parent. Analyses of these kinds of lag-sequential interactions require the use of strategies designed to test conditional probabilities: given that X has just occurred, what is the likelihood of Y rather than Z? Yule's Q is a commonly used statistic to estimate such probabilities based on an odds ratio. For example, in a study of parent–child discourse mentioned earlier, Kerig, Cowan, and Cowan (1993) used Yule's Q to estimate the likelihood that, given a daughter had made an assertion, her maritally distressed father would respond with a positive or negative statement.

A third question requires examining more complex series of events and analyzing data gathered in series over a longer time frame, such as a study examining whether repeated detriangulating maneuvers by a third-party shift in the family structure over time to one with more balanced alliances. This kind of question requires the use of log-linear data analytic methods that enlarge upon the logic of the simpler transitional probability represented by Yule's Q. One such log-linear analysis is a logit model which allows for the prediction of a particular event of interest from other events during a family interaction; for example, whether children's attempts to intervene in their parents' conflicts are met with encouragement or discouragement depending on whether the member of the dyad raising the conflictual issue is the mother or the father. In turn, time series analyses are used to analyze observations involving many points of data collected virtually continuously over time. Time series also can be used to test models regarding who is driving the interaction by looking for asymmetries in the probabilities of one partner's behavior predicting the other—for example, whether family interactions become increasingly negative as fathers, as opposed to mothers, attempt to triangulate children in interparental conflicts. In addition, an interrupted time-series analysis can be used to investigate the impact on the interaction of a low-frequency but notable event, such as a threat of violence by a family member, or a therapeutic intervention by a therapist.

Kashy and Kenny's (2000) actor–partner interdependence model (APIM) also can be extended to the study of family groups beyond the dyad. APIM allows the investigator to assess the extent to which each actor in the interaction is affected by, and in turn affects, the partner. In addition, the model accounts for a group effect beyond the individual or dyadic, which in family research would be represented by the effects on all members of being member of the family. For example, a dyadic actor effect would indicate that an individual's score changes depending on whether a given family member participates in the interaction (e.g., does a son's contact-seeking behavior toward his mother decrease when his father is present in the room). In turn, a dyadic partner effect would refer to whether the presence of a given dyad in the family influences the behavior of others in the group (e.g., when both parents in a maritally distressed couple are present, do all family members demonstrate a decrease in proximity seeking).

Although the study of sequences and transactional patterns in family process data has generally relied on the

use of lagged sequential models, underlying such models is an assumption of linearity which might not accurately describe the functioning of more complex systems.

> A challenging alternative might be to assume that the behaviors of interest are part of complex systems and are not linear (i.e., although topographically similar, the same behaviors function differently as the interaction progresses). What if coded elements are not additive (i.e., unfold linearly as assumed in sequential analysis) but rather follow properties of complex adaptive systems ... Chief among the defining properties of dynamic systems is that change can occur not only not linearly but catastrophically; a small input can disrupt an ongoing process in a major (disproportionate), nonlinear way (Weiss & Heyman, 2004, pp. 22–23).

Dynamic Systems Approaches

One of the most exciting developments in the integration of family systems research with developmental psychopathology is the contribution of dynamic systems (DS) theory. As defined by Granic and Hollenstein (2003), dynamic systems models in developmental psychopathology provide a metatheoretical framework for understanding how systems emerge, stabilize, form, and change as a function of their own internal feedback processes. Not only has DS theory been conceptually elaborated in ways that have increased its dissemination across fields of psychological research, but new statistical methods also have been developed that enable researchers to create and test dynamic models in family research. Thus, analyses of systems change require mathematical modeling of nonlinear parameters that reflect patterns of change. As outlined in Fischer, van Geert, and Mascolo's chapter in this volume, dynamic systems models allow for the analysis of complex patterns of behavior as these emerge and are shaped by each partner, and the relationship between them, over time. An essential construct in DS is that of *attractors*, patterns of behavior that have a pulling effect on the system, restraining it and limiting the degrees of freedom available for variation. Family members "influence each other in transactional fashion: the daily interactive experiences of mother and child lead, over time, to the emergence of relatively few but enduring behavioral patterns, or order parameters, that are characteristic of their relationship. In turn, these patterns modulate their ongoing interactions, channeling them to approximate what they have been in the past, thereby maintaining the relationship across time" (Dumas, Lemay, & Dauwalder, 2001, p. 318). One of the ways these patterns coalesce is through the *coupling* of interactional partners' emotions, appraisals, and the behavior by which affect and cognition are linked; thus, established patterns become intricately interwoven into the thoughts, feelings, and actions of each participant in the relationship. The result is *rigidity* that results in family members falling into a repetitive and narrowly defined set of behavioral repertoires with one another, as well as others outside the system (Granic & Patterson, 2006). Moreover, these patterns tend toward increasing *escalation* over time. For example, past experience with negative transactions leads family members to become sensitized to even subtle signs that the cycle is beginning (e.g., a huff of exasperation, a roll of the eyes) and to respond to these mild provocations with a level of intensity that matches the increasingly hostile exchange they anticipate coming.

Empirical evidence in support of this dynamic analytic framework is offered by Dumas et al. (2001) in their study comparing mother–child dyadic interactions in clinically distressed and normative families. Coders rated each minute of mother–child interaction to assess for maternal controlling, positive, and aversive behaviors as well as child compliance, noncompliance, positive, and aversive behaviors. Using both quantitative and graphical analyses, the investigators examined phase transitions in the interactions between dyads in the two groups. The patterns that emerged evidenced that, amongst clinical families only, mothers and children repeatedly ended up in aversive cycles in which mothers commanded and children refused to comply, even when mothers initiated the interaction in positive ways. Thus, the coercive family processes that had come to organize the system of relationships wove a sticky web from which it was difficult for mothers and children to extricate themselves. Figure 13.2 displays these findings in a four-variable Karnaugh map, which represents the frequency with which interactional partners engage in each of the various possible transitions in behavior. This map indicates that, among clinically referred families only, mothers and children tended to engage in repeated cycles involving maternal control attempts that were followed by child noncompliance (indicated by the cell labeled configuration number 8). "In other words, in average dyads, the major attractor, besides Configuration 0, was one in which children tended to respond positively to their mothers' requests and instructions, but in referred dyads, it was one in which mothers tended to 'spin their wheels' in fruitless attempts to get their children to comply" (p. 328).

To date, the majority of the work utilizing DS in family systems research has focused on dyadic exchanges between parents and children. However, an important contribution to our conceptualization of dynamic systems from a whole-family perspective is offered by Schermerhorn

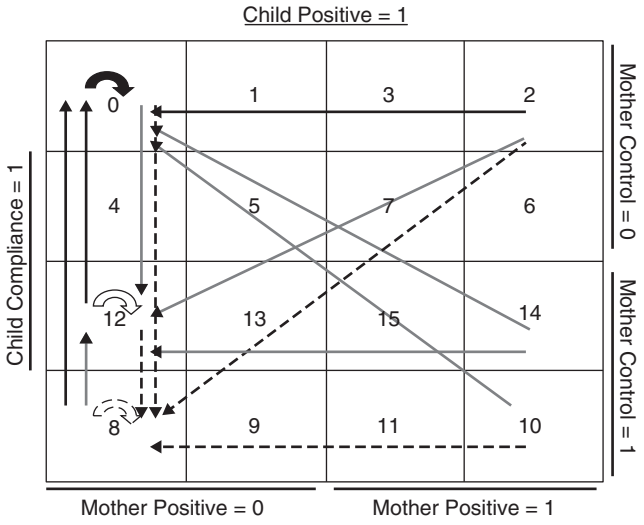


Figure 13.2 Karnaugh map displaying mother–child transactions in typical and clinically referred groups. *Source:* Dumas, J. E., Lemay, P., & Dauwalder, J.-P., *Journal of Abnormal Child Psychology,* Dynamic analyses of mother-child interactions in functional and dysfunctional dyads: A synergetic approach, *29*(4), 2001, p. 327, Figure 6. With kind permission from Springer Science+Business Media.

and Cummings (2008) in their proposal for a model of *transactional family dynamics* (Figure 13.3).

> Transactional family dynamics refers to the myriad ways in which family members influence one another, that is, mutual influence processes within families. For example, these processes may include complex patterns of influence in interparental, father–child, mother–child, and sibling relationships. Notably, our interest is in transactional processes—not unidirectional processes—that is, influence processes continuously moving in both directions over time (Schermerhorn & Cummings, 2008, p. 188).

Using this model, Schermerhorn, Cummings, and Davies (2008) demonstrated dynamic links among children's representations of interparental, mother–child, and father–child relationships over time in a three-year longitudinal investigation of kindergarten-age children in a community sample. Structural equation modeling with autoregressive paths showed that children's representations of the destructiveness of interparental conflict predicted increased sensitization and reactivity to those conflicts, which in turn were predictive of children's representations of the security of their relationships with mothers and fathers. Moreover, evidence of transactional processes involving children's impact on the family system emerged from this laboratory's investigation of the interrelations among interparental conflict, child behavior, and child adjustment. Over a three-year period, Schermerhorn, Cummings, DeCarlo, and Davies (2007) found that interparental discord was predictive of child negative emotional reactivity, which in turn predicted behavioral reactivity. Moreover, when children's behavioral reactions took the form of agentic behavior (e.g., trying to distract, comfort,

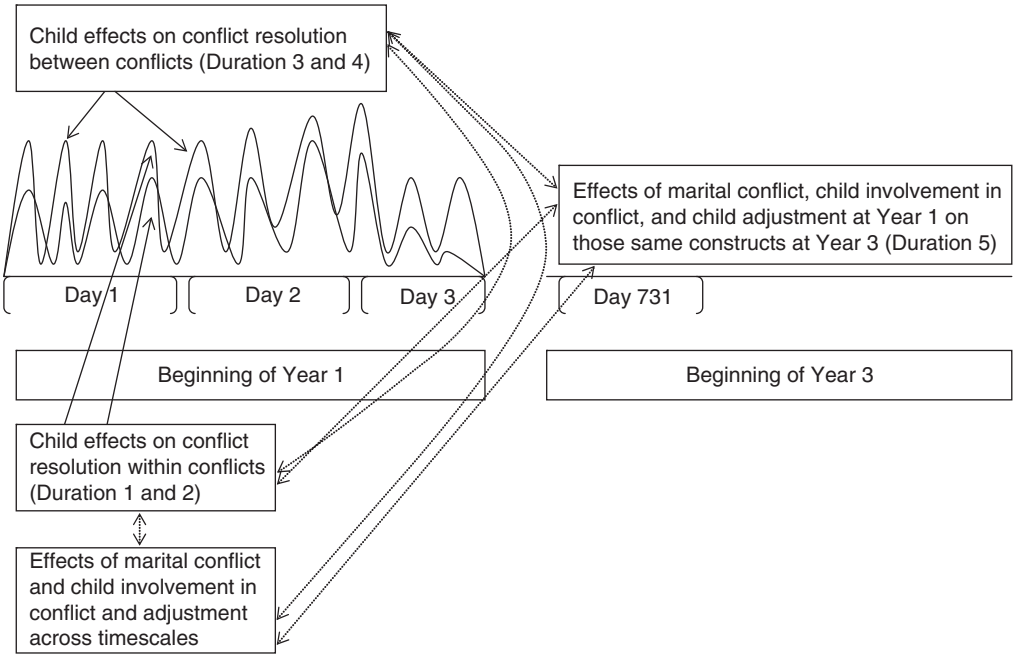


Figure 13.3 Schermerhorn and Cummings's model of transactional family dynamics. *Source:* Reprinted from Schermerhorn, A. C., & Cummings, M. E., *Advances in Child Development and Behavior*, 36, Transactional family dynamics: A new framework for conceptualizing family influence processes, p. 223. Copyright 2008, with permission from Elsevier.

or help parents resolve their dispute), interparental conflict tended to decrease whereas, when children's behavioral reactions took the form of dysregulated behavior, both interparental conflict and child maladjustment tended to increase.

EMPIRICAL INVESTIGATIONS OF FAMILY SYSTEMS CONSTRUCTS

Global Family Qualities

Cohesion

One of the most well-studied whole-family constructs is that of family cohesion, the idea that family members experience a positive sense of emotional bond and unity. Along with flexibility and communication, cohesion is one of the key dimensions of the Circumplex model of marital and family systems (Olson, Sprenkle, & Russell, 1979). Implicit in the construct is that cohesion is uniform across the dyads and triads in the family and, therefore, that there is an absence of the kinds of unbalanced coalitions or triangles that are associated with pathological family structure in structural family theory (Minuchin, 1974). A large body of research has confirmed the positive benefits of living in a cohesive family system for both adults and children (Olson, 2011). For example, Favez et al. (2012) found that observed family cohesion, defined by consistently balanced alliances and an absence of triangulation, was associated with more positive adaptation of children over the course of five years, from infancy to early school age. Leary and Katz (2004) similarly found that observational ratings of family cohesion (defined as adaptability, cohesiveness, and playfulness) when children were in preschool were predictive of more positive relationships with peers in middle childhood.

Moreover, family cohesion appears to provide a buffer for children against the negative impact of other family stressors, such as interparental conflict (Davies, Harold, Goeke-Morey, & Cummings, 2002). Further refining our understanding of these interrelations among family processes in a multiethnic framework, Lindahl and Malik (1999) found that family cohesion was a more powerful buffer against the negative effects of exposure to interparental conflict for children in Hispanic American versus European American families. Subsequently, Lindahl, Malik, Kaczynski, and Simons (2004) again showed that observed family cohesion and intergenerational coalition formation mediated the association between interparental conflict and child adjustment in ways that were moderated by ethnicity. Low family cohesion and cross-generational coalitions were more strongly associated with internalizing problems for children in European families than those in Hispanic families, perhaps as a function of the extended family networks that might have helped to buffer Hispanic children from the stresses of their parents' marriage. In one of the few studies to investigate the interrelationship among these family processes in a sequential manner, Kitzmann (2000) demonstrated that, following a marital conflict discussion, families displayed observable reductions in cohesion and concomitant increases in unbalanced alliances during triadic interactions.

Hierarchical Organization

Also consistent with both structural and strategic family theory is the idea that adaptive family structure requires a hierarchical organization in which the generational boundaries are respected in ways that place parents clearly in an executive position in the family (Haley, 1976; Minuchin & Fishman, 1975). In turn, lack of role clarity and organization is characteristic of clinically distressed families, such as those in which children are maltreated (Howes, Cicchetti, Toth, & Rogosch, 2000). Although a number of prior cross-sectional studies had found that violations of hierarchy are associated with childhood externalizing problems, Shaw, Criss, Schonberg, and Beck (2004) made an important contribution to this literature by examining the developmental unfolding of hierarchical structure and child behavior problems from infancy to middle childhood. The investigators used a combination of maternal report (e.g., "If you ask your child to do something and he does not do it, how often do you give up trying to get him to do it?"), observational coding (e.g., a 9-point rating of level of intergenerational boundaries), and interviewer judgment (e.g., "Parent has good control of the child") to create an index of family hierarchy. Structural equation modeling showed that lower levels of family hierarchical organization were associated with higher levels of child conduct problems over the course of development. Again, however, these associations may be tempered by culture. Lindahl and Malik (1999) found that a strongly hierarchical family structure was associated with externalizing among European American youth, whereas its opposite (a lax, permissive style) was predictive of externalizing among Hispanic youth.

Family Structural Types

Although many studies have examined triangulating processes in the family, fewer have attempted to validate the structural family (Minuchin, 1974) typology specifying the distinct and discriminable forms that rigid triangles might

take in the family system. One such investigation (Kerig, 1995) elicited self-reports of perceived family structure from mothers, fathers, and children using a revised version of Cooper, Holman, and Braithwaite's (1983) Family Cohesion Index (Figure 13.4). Although family members agreed only modestly with one another, each individual's depiction of the family structure was meaningfully related to his or her ratings of other aspects of family functioning. Specifically, parents and children in triangulated relationships gave the highest ratings of interparental conflict and negative affect in family interactions. In contrast, children in families typologized as detouring were highest in self-blame and internalizing problems, whereas children in disengaged families were rated by their parents as highest in externalizing.

Subsequently, Davies, Cummings, and Winter (2004) tested whether this typology could be confirmed in a study of school-age children and their parents followed over a one-year period. The investigators used cluster analyses to create four family profiles, based on observed and self-report measures of interparental, coparenting, and parenting behaviors. *Cohesive* families were defined as those high in interparental affection and acceptance of children and low in interparental hostility, coparenting disagreements, inconsistent discipline, and psychological control of children. *Enmeshed* families, in contrast, combined moderate levels of interparental affection and parental acceptance with very high levels of interparental hostility, coparenting disagreements, inconsistent discipline, and psychological control. *Disengaged* families, in turn, evidenced very low levels of interparental affection and parental acceptance along with moderately high levels of the other variables. Finally, a group labeled *adequate* was identified that was like the cohesive families in all respects except for showing high levels of psychologically controlling behaviors. Structural equation modeling demonstrated that family disengagement was linked to children's emotional insecurity in the family system, which in turn predicted elevations in child internalizing symptoms over the course of one year. In contrast, family enmeshment was predictive of increased child internalizing and externalizing, an association that also was mediated by children's emotional insecurity. This study provides a nice illustration of the idea that dysfunction in one dyad in the family system—such as interparental conflict—may have different implications depending on the larger context of the organization of the family system. In particular, the diffuse boundaries of enmeshed families appear to create a context in which the spillover of family tensions is exacerbated and potentiates their negative impact on child development.

In turn, using observational and qualitative methods, Fivaz-Depeursinge, Lopes, Python, and Favez (2009) also found evidence for three of Minuchin's rigid triangular types with families of toddlers: *detouring*, characterized by parents shifting their tension onto the child, to which children tended to respond by adopting an overly engaged role-reversed style; *triangulation*, in which parents each vied for the child's affection and the child attempted to cope with the strain by either withdrawing or playing the role of go-between; or the binding of the child into a *coalition* with one parent. Finally, focusing specifically

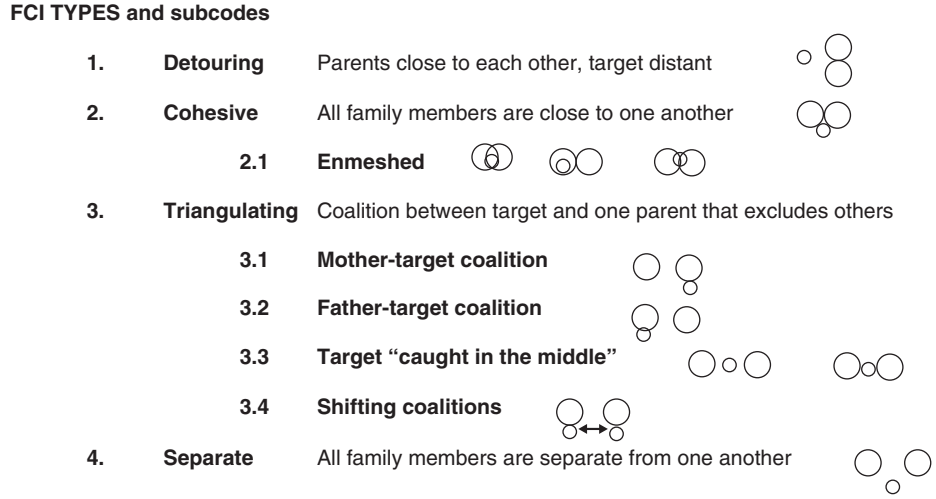


Figure 13.4 The Revised Family Cohesion Index. *Source:* Kerig, P. K., Triangles in the family circle: Effects of family structure on marriage, parenting, and child adjustment, 1995, *Journal of Family Psychology, 9*(1), 28–43. Reprinted with permission from Patricia K. Kerig.

on the latter family process, Lindahl and Malik (1999) also used observational coding to show that coalitions between one parent and the child that excluded the other parent were associated with externalizing problems in an ethnically diverse sample of families.

Spillover of Marriage onto Parenting and Family Process

Although a rich literature points to ways marital conflict spills over onto dyadic parent–child relationships, a smaller body of work has been devoted to the interactive contributions of interparental conflict, parenting, and whole-family processes (Buehler & Gerard, 2002). The majority of these studies have examined parent–child dyadic interaction within the triad, however, rather than whole-family processes. Among the studies that have led the field in this regard, Katz and Gottman (1996) demonstrated that observed marital hostility contributed to boys' peer aggression through the mediating factor of parental rejection during triadic family interactions. In turn, Gordis, Margolin, and John (1997) and Margolin, Gordis, and John (2001) found that marital hostility and aggression were associated with increased parenting hostility during observed family interactions, which in turn was associated with boys' withdrawal, anxiety, and behavior problems.

Siblings

A rich literature has emerged suggesting that sibling relationships play a significant role in child development (Buist, Deković, & Prinzie, 2012), one that can be disrupted by other family processes such as interparental conflict (Dunn & Davies, 2001; Rinaldi & Howe, 2003). However, little of the research to date on family systems theory has included siblings, an unfortunate yet understandable omission given the difficulty of coordinating so many different data points and sources of influence in family interactional research. In one suggestive study, Grych, Raynor, and Fosco (2004) found that sibling empathy and similarity did not help to buffer children from perceived threat regarding interparental conflict; a possible explanation for this finding is that sibling closeness reflects an overall climate of diffuse boundaries in the family, which fosters carryover of emotional reactivity from one relationship to the other.

Triangulation

One of the challenges to integrating the research that has been carried out on family systemic constructs is that they have been defined and operationalized in diverse ways. For example, as we just reviewed, the term triangulation in Minuchin's (1974) original usage referred to only one of several rigid triadic forms in the family, whereas in current parlance it tends to be used as the superordinate construct under which various pathological structural processes are subsumed. As originally defined by Minuchin (1974), triangulation referred to a family form in which parents competed with one another to establish an alliance with the child, consistent with the construct of competitive coparenting (McHale, 1997) or, in its extreme form, parental alienation when the other parent is actively excluded, estranged, or vilified (Johnston, Walters, & Olesen, 2005).

In turn, a related but distinguishable use of the term triangulation focuses on the child's perspective of feeling caught in the middle or being entangled in a loyalty conflict (e.g., Buchanan & Heiges, 2001; Fosco & Grych, 2008; Grych, Seid, & Fincham, 1992). Another related but not identical construct is that of child involvement in parents' quarrels, whether observed (e.g., Davies & Cummings, 1998), reported by parents (e.g., Kerig, 1996) or reported by children (e.g., O'Brien, Bahadur, Gee, Balto, & Erber, 1997). Again, although not identical constructs, these are related in that children who feel caught in a loyalty conflict might well feel compelled to intervene in their parents' conflicts, as evidenced empirically (Grych & Fincham, 1993). Nonetheless, from a systemic perspective, the behavior of intervening also may arise from a different family process, such as the formation of a parent–child coalition that leads a child to feel obligated to step into the fray on behalf of the parent with whom she or he has aligned. As noted previously, child involvement also might be initiated either by parents (e.g., parents who enlist the child as a "co-combatant") or by children (e.g., children who attempt to intercede in their parents' quarrels, even if unbidden) (Kerig, 2001a, 2001b). Triangulation also has been defined as inappropriate child elevation to the role of fellow-adult and decision-maker in the family which might, but might not, take the form of intervening in their conflicts, and thus which might blend into other boundary dissolution-related constructs such as parentification or adultification (Kerig, 2005).

The interrelatednesss of these constructs is illustrated by the literature that has emerged investigating children's involvement in interparental conflict, as variously defined. In studies deriving from the cognitive contextual model (Grych & Fincham, 1990), children's perceptions that they are implicated in their parents' quarrels are associated with distress and maladaptation (e.g., Grych, Harold, & Miles, 2003; Grych, Raynor, & Fosco, 2004; Kerig, 1998). By the same token, emotional security theory (Davies & Cummings, 1994) accurately predicts that children's

anxiety about threats to the security of the family system will motivate them to intervene when their parents argue (Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006). Studies also have shown that interparental conflict increases the likelihood that parents will form cross-generational coalitions with children and involve them in discussion of marital issues (Christensen & Margolin, 1988; Lindahl, Clements, & Markman, 1997). In turn, children who become involved in coalitions characterized by siding with the father against the mother during observed family interaction are at increased risk for the development of externalizing problems (Davis, Hops, Alpert, & Sheeber, 1998).

In an important contribution to this literature, Lindahl and colleagues (2004) demonstrated the interrelations among interparental discord, whole-family processes, and child adjustment. In particular, these authors found in a multi-ethnic sample that a latent construct of family functioning (composed of observed cohesion, triangulation, and cross-generational boundary violations), fully mediated the association between couple conflict and child behavior problems. This study points to the value of understanding the family as an organized system in which the parts are inextricably interrelated with one another. As the authors note, "In the language of structural family systems theory, conflicts that erode the solidity of the marital subsystem can create fault lines in the family structure, affecting dyadic, triadic, and whole-family subsystem relationships as well as child functioning" (p. 625).

Dimensions Underlying Triangulation

As noted already, however, the various ways triangulation has been defined and operationalized complicate our ability to integrate and learn from this literature. As a way of untangling these skeins, we might consider five underlying dimensions along which these various definitions of triangulation can be distinguished. One helpful way of thinking about the different ways these constructs have been construed across studies is a distinction between *process* (e.g., a parent attempts to enlist the child as an ally against the other parent) and *outcome* (e.g., a parent and child have formed an entrenched alliance that excludes the other parent). For example, some studies focus on capturing the ongoing dynamics in the family (e.g., parents vying with one another to establish a special relationship with the child, as in competitive coparenting), whereas others assess the family structures, or outcomes, which may arise from those processes (e.g., the establishment of a parent–child coalition as a fait accompli). A good reason for differentiating between process and outcome is that the same process might lead to different outcomes, either across families or within the same family. Consider, for example, the possibility that in a family characterized by marital conflict, a mother succeeds in forming a cross-generational alliance with her adolescent son, perhaps fueled by concomitant negativity or disengagement in the father-son relationship, whereas the preschool-age daughter's intensified security needs lead her to experience a loyalty conflict upon feeling herself to be forced to choose between her parents. We can complicate the picture even further by proposing a school-age son who acts out in ways that serve to detour around the parents' conflicts and, at least briefly and transiently, unite them in the task of responding to his misbehavior. By the same token, parent- and child-driven processes may lead to the same or to different outcomes. Knowing that the adolescent son in the family above intervenes in interparental quarrels in order to defend his mother's point of view does not inherently tell us whether his mother has encouraged him to take such a role, versus whether he has mistakenly construed protecting her as his duty. Similarly, the daughter's feeling of being caught in the middle might arise from a misplaced belief that it is her responsibility to mediate between her parents even though her parents actively discourage this.

A second dimension discriminating the ways these constructs have been construed is whether they are measures of *behavior* (e.g., a child attempts to intervene in her parents' quarrels) versus *perceptions* (e.g., a child feels caught in the middle between her parents). For example, some operationalizations of triangulation involve observed or reported actions (e.g., "When my parents argue, my mother tries to get me to side with my father") whereas others focus on the child's felt experience (e.g., "I feel caught in the middle when my parents argue; I feel it is my job to try to help my parents get along with each other when they fight"). These have been shown to be correlated in that children whose parents entangle them in their conflicts are those most likely to feel responsible for solving them (Grych et al., 2004), yet one cannot be presumed to be caused by the other. Children's egocentric perspective may inflate their sense of responsibility for, and beliefs that they have control over interparental conflicts even when parents do not encourage them to believe so (Kerig, 1998).

In this regard, as noted previously, a third dimension underlying triangulation is whether it is *child-driven* (e.g., a child steps in to defends her mother against her father) or *parent-driven* (e.g., a mother invites the child to join her in criticizing the father) (Kerig, 2001a, 2002b). As Grych et al. (2004) noted, however, there likely are transactional associations between these two processes. Although their study

established that children involved in cross-generational coalitions were vulnerable to feeling caught in the middle of their parents' conflicts, it also may well be the case that children's triangulation affects parent–child relationships, such as by drawing the child closer to one parent than the other. Thus, parents who engage children in unbalanced alliances may increase the likelihood that children become involved in interparental conflicts, which in turn may further disrupt family cohesion.

A fourth distinction worth considering is whether child involvement is *overt* (e.g., a child walks into the room and asks her quarreling parents to stop; parents ask a child to pick sides during their argument) or *covert* (e.g., a child knocks over the CD tower in the next room as a way of distracting her parents from their quarrel; parents express disparaging views about the other parent when engaging in one-to-one interactions with the child). A higher level of inference is needed to determine that the child's purpose in engaging in misbehavior is to intervene in the conflict, and this has seldom been queried directly (Kerig, 2001b).

Last, a little-studied dimension underlying these various forms of boundary violations is their *valence* (Kerig, Swanson, & Ward, 2012)—that is, whether they are conveyed in the context of a positive or negative emotional tone in the family. Sometimes the message is mixed; for example, Jacobvitz and colleagues (2004) point out that sweetness and coyness of seductive parenting often is accompanied by harsh and punitive behavior. But in other cases, a clearer discrimination can be made: for example, between hostile versus seductive spousification; between detouring processes that scapegoat a child versus parent–child coalitions that make that child special; or between psychological control that is demanding and critical versus warm and appreciative. For example, Rohner (1986) proposed that perceived parental warmth might potentiate the effects of intrusiveness by pulling the child even closer to the parent emotionally. Some evidence in support of this idea has emerged in studies showing that psychological control is most strongly associated with youth depression when perceived parental acceptance is high (Barber, Stolz, & Olson, 2005). Further, Kerig, Swanson, and Ward (2012) found specificity in the ways that emotional valence modified the association between psychological control and youths' mutuality of autonomy in intimate peer relationships. Youth whose intrusive parents were perceived as accepting were those most likely to develop imbalanced relationships in which they put others' needs before their own, whereas youth whose intrusive parents were perceived as rejecting were most likely to develop nonmutual relationships in which they put their own needs before those of others.

Again, a major shortcoming of the investigations of these constructs to date is how little research has investigated the larger family systems of interrelationships in order to examine how process or outcome, behavior or perceptions, child driven or parent driven, overt or covert, and positively or negatively valenced boundary violations between members of one subsystem of the family are influenced by and influence those among the other dyads and triads. Sibling relations have been neglected here too, in that siblings might provide support that helps to buffer a brother or sister who is triangulated between parents and yet also might play the role of active contributors who collude in the exclusion, scapegoating, or differential treatment of one another.

Coparenting Conflict and Family Process

The coparenting literature provides a number of examples of studies encompassing the interrelations among whole-family, interparental, and parent–child processes. The construct of coparenting follows clearly from the structural family model in which healthy family functioning requires that parents be united with one another, with clearly demarcated boundaries between their executive unit and those of the child subsystems. However, coparenting brings additional clarity to this picture by distinguishing the alliance that partners maintain related to the tasks of *parenting*, as distinct from their adult intimacy or marital relations (McHale, 1997). Thus, coparenting is inherently a triadic process in that it concerns how parents coordinate with one another in relation to the child. Further, through observing their parents interacting together as coparents, children glean important information about the quality of each parent's relationship with the child and with one another, about how relational partners coordinate with one another within the family system, and, fundamentally, about the intactness of the family and their security within it.

The evidence that the coparenting relationship has significant implications for child development is clear. Research has shown that coparenting warmth and cooperation are associated with positive marital and parent–child relationships as well as child well-being (McHale, Kuersten, & Lauretti, 1996). In contrast, hostile-competitive coparenting, as we have seen, represents a form of triangulation that has demonstrated negative effects for children, including internalizing and externalizing symptoms as assessed both contemporaneously (McConnell & Kerig, 2002) and longitudinally over the transition from preschool to schoolage (Leary & Katz, 2004; McHale & Rasmussen, 1998). Hostile-detached coparenting, in which parents

exhibit a combination of anger toward and withdrawal from one another, also has been associated with child maladaptation (Katz & Low, 2004; Katz & Woodin, 2002).

Moreover, McHale and Rasmussen (1998) explicitly investigated the links among interparental, coparenting, parent–child, and family-level processes in a study of mother-father-infant triads followed over the course of three years. In addition to obtaining self-reports of coparenting, family processes during triadic interactions were observed, including measures of coparenting, parent-to-child behavior, and total family warmth. These measures were summarized to create three factors: hostility-competitiveness (i.e., parent- vs. child-centeredness combined with a competitive coparenting relationships), family harmony (i.e., high warmth and coparenting support), and parenting discrepancy (i.e., high levels of discrepancy between ratings of each parents' warmth and investment with the infant). The results showed not only that these family process variables forecast child behavior problems over and above measures associated with marital quality, but that the coparental relationship was associated with specific aspects of family process for both husbands and wives. For example, men's reports of engaging in positive coparenting behaviors (e.g., showing affection to the spouse and child when the three are together) were related both to observed family warmth in infancy and fewer child behavior problems in preschool. In turn, mothers in families with observed negative processes in infancy were those most likely to disparage their husbands to their preschool-age children.

In turn, Katz and Low (2004) utilized a multimethod approach to investigate the combined and unique contributions of marital violence, coparenting, and whole-family processes as predictors of child adjustment. Their results showed that hostile-withdrawn coparenting behavior during triadic interactions accounted for the relationship between marital violence and children's internalizing problems while marital violence, coparenting and family-level processes also made independent contributions to predicting child maladjustment. Specifically, children in families whose interactions were characterized as fragmented (i.e., high in disengagement, parent-centered vs. child-centered balance of power, and expression of negative affect) were those most likely to demonstrate externalizing problems in peer and parental relationships.

A valuable next step in this research will be to more clearly explicate the interrelations among these dyadic, triadic, and family-level processes as they unfold over time. It will be of interest to discern to what extent, for example, whole-family dynamics such as warmth and child-centeredness might buffer children from negative effects of coparenting discrepancies. Similarly, it would be important to better understand how coparenting conflict might differentially impact the quality of mother-child and father-child relations in ways that, in turn, are reflected in changes in the overarching emotional quality of family life.

Boundary Dissolution

The literature on boundary dissolution as a concept in family systems theory dates back to its progenitors, and originates with the construct of *enmeshment*. The term enmeshment is used in two contexts regarding family systems: as a description of a form of dysfunction in the relationship between two family members, or as a description of the family system as a whole. The first derives from Bowen's ideas of the undifferentiated ego mass in which poor psychological boundaries between two family members creates a sense of psychological fusion that interferes with either one achieving individuation. The second usage refers back to Minuchin's (1974) structural perspective, in which families are characterized as having diffuse boundaries among all the members and thus as being, as a whole, enmeshed. Despite this distinction, either form of boundary dissolution—between members of a dyad or within in the entire family system—is perceived as increasing the likelihood of the involvement of third parties. In Bowen's model, such triangulation might serve a beneficial function for the dyad and the whole family system, whereas in Minuchin's perspective, third-party involvement is likely to be dysfunctional, particularly when the triangulation of a third family member requires her or him to cross generational boundaries. Thus, the boundaries between the concepts of triangulation and boundary dissolution are necessarily fuzzy, given their interrelatedness, while at the same time they have tended to spur separate programs of research and to be defined and operationalized differently by various researchers. For our present purposes here, of particular interest is the small but valuable body of research that has investigated boundary dissolution not only as a dyadic variable in the parent–child relationship, but as a dynamic in the context of the larger family system of relationships. In this regard, the literature on the interrelations of boundary dissolution and triangulation is particularly rich.

Boundary Dissolution from a Family Systems Perspective

Jacobvitz and colleagues (e.g., Hazen, Jacobvitz, & McFarland, 2005; Jacobvitz & Bush, 1996; Jacobvitz, Riggs, & Johnson, 1999; Jacobvitz & Sroufe, 1987) have

carried out a significant series of studies examining boundary dissolution in the context of both dyadic and triadic family interactions, in which they interweave the constructs of triangulation and boundaries by construing triangular processes as subsumed under the broader construct of boundary dissolution. For parents to engage in triangulating strategies, such as enlisting their child in a coalition, suggests a breakdown of intergenerational boundaries that also might emerge in other ways. For example, Jacobvitz, Hazen, Curran, and Hitchins (2004) observed mother–father–child triads when children were 2 years of age and coded the interactions for indications of patterns they subsumed under the rubric *enmeshment* in each parent–child dyad, operationalized as one parent turning to the child for caregiving (e.g., parentification of child) or intimacy (e.g., spousification) or to exclude the other parent (e.g., parent–child coalition), as well as one spouse's attempts to parentify the other. In addition, family interactions were coded for hostile (e.g., criticism, sarcasm), controlling (e.g., one family member dominating the others), disengaged (e.g., a lack of emotional connection between any two family members), and balanced patterns (e.g., families in which there is promotion of autonomy and emotional openness among all three members). Results of the study showed distinct patterns of child maladaptation associated longitudinally with each of these dimensions from ages 2 to 7. Blurred boundaries were associated with child depression, whereas controllingness and disengagement were predictive of child anxiety and depression; in turn, hostility predicted child attentional problems and somatic complaints. Gender differences also emerged, with enmeshed patterns differentially related to inattentiveness among boys and depression among girls.

The Effects of Relationships on Relationships: Parent–Child Boundary Dissolution in the Context of Interparental Conflict

As Margolin, Gordis, and Oliver (2004) pointed out, the maintenance of clear boundaries helps to protect the family from other negative processes, such as the spillover of marital tensions onto children. "When clear boundaries exist between the marital and the parent–child subsystem, spouses have their own separate adult relationship, and also are available to meet the needs of their children. Without clear boundaries, conflict from the marital relationship may intrude on the parent–child relationship and alter the nature of that relationship" (p. 754). Through the maintenance of boundaries, the diffusion of negative emotions in the family is constrained to one subsystem rather than upwelling into the "cloud of unpleasantness" (Patterson & Forgatch, 1987, p. 8) that often overshadows distressed families. In their observational research, as Margolin and colleagues (2004) hypothesized, families in which husbands were violent toward their wives were especially susceptible to the leakage of hostilities from the marital to the parent–child relationships, which was expressed in the form of reduced empathy by fathers and increased negative affect by mothers.

In the spirit of further investigating the interdependencies and interconnectedness of these constructs, Kerig and Swanson (2010) attempted to explicitly integrate triangulation and boundary dissolution in parent–child, interparental, and triadic family processes among a sample of emerging adults. A broader perspective on family boundaries suggests that interparental conflict may increase the likelihood that parents will turn toward, against, or away from children in a variety of ways, each of which involves the violation of emotional, psychological, or generational boundaries. In other words, although one way marital stress may spill over onto children is to involve children in the conflict, as triangulation is often defined, other family processes might arise which we would discern better by looking through the lenses of the boundary dissolution construct. Drawing upon a multidimensional model of boundary dissolution (Kerig, 2005), we focused on three facets of the construct for which evidence suggests the risk is greatest among families characterized by marital unhappiness or conflict: intrusiveness, hostile spousification, and parentification. Structural equation modeling showed that, among emerging adults, maternal intrusiveness, hostile spousification, and parentification all acted as mediators of the association between exposure to interparental conflict and poor adjustment. In turn, only paternal hostile spousification played such a mediational role, and its effects were specific to the development of externalizing problems, including relational aggression toward intimate partners.

GENDER DYNAMICS IN FAMILY PROCESS

Fathering and Spillover

As noted before, although long neglected in psychological research, fathering provides a unique source of influence on children's development that deserves recognition (Lamb, 2010). Some research on the spillover of marital conflict has suggested that fathering behavior is particularly vulnerable to the disruptive effects of an unhappy marriage. For example, similar to Katz and Gottman (1996), Lindahl

and Malik (1999) also found that marital conflict was more strongly associated with fathers' than with mothers' behavior during triadic family interactions, including observed withdrawal, rejection, and lack of support. One explanation proposed for these gender differences in parental behavior is that mothering is a more well-defined role in U.S. culture than is fathering, and therefore fathers are more likely than mothers to look toward their relationships with their spouses for models about how to engage emotionally with their children (Cowan & Cowan, 1992; McHale et al., 2004).

Variations in Family Processes Related to Child Gender

As noted by Cowan, Cowan, and Kerig (1993), in majority-culture heterosexual families, parents and children each come in two genders, as mothers, fathers, sons, and daughters. Attention therefore is warranted to how family dynamics might play out differently in the intersections between parent and children. However, the literature on gender differences in the effects of family process on parent–daughter and parent–son relationships is highly inconsistent (Davies & Lindsay, 2004). For example, among studies of gender differences associated with the effects of interparental conflict, some studies show differential impacts on mother and sons (e.g., Osborne & Fincham, 1996), fathers and daughters (e.g., Kerig, Cowan, & Cowan, 1993), or none of the above (Erel & Burman, 1995). Other studies have indicated that gender effects in discordant families are not specific to one gender but rather tend to affect other-sex dyads in general (Cox, Paley, & Harter, 2001). For example, Kerig and Swanson (2010) showed via path analysis that interparental discord predicted denigration of the spouse's sex role characteristics, which was associated with denigration of those characteristics in the child of the same sex as the spouse and, in turn, with child internalizing problems. However, it is likely that these dynamics do not emerge as gross main effects associated with gender across all families but rather are qualified by a number of other variables, including child age and cultural variability in family attitudes and practices related to gender roles. Therefore, the more apt question may be not whether, for example, mother–son relationships are more sensitive to the negative effects of interparental conflict in all families but rather when mother–son relationships are disrupted what are the larger family dynamics that account for disruption to that subsystem, and what then are the specific consequences for child development?

One suggestion that has received some support is that daughters may be more likely than sons to feel, and to succumb to, the pull to become involved in intergenerational boundary violations, whether overtly (e.g., as mediators) or covertly (e.g., as worriers who feel responsible for caring for their emotionally distressed parents; Kerig, Fedorowicz, Brown, Patenaude, & Warren, 1998). Gender socialization theory would predict that girls' interpersonal orientation and propensity to tend and befriend would increase their sense of responsibility for maintaining the emotional harmony in the home generally, but as Davies and Lindsay (2004) pointed out, these concerns are intensified during adolescence when gender role consolidation emerges as an important stage-salient task. Adolescence also may be a sensitive period for boundary violations specifically in the mother–daughter relationship. The literature on depression in particular points toward a special sensitivity to their mothers' affect that is exhibited by girls, and this may well intensify in the teenage years (Sheeber, Davis, & Hops, 2002). Not only may adolescent daughters' increasing cognitive and interpersonal capacities lead them to feel obligated or even equipped to take on the burden of caring for mothers who are depressed or maritally distressed, but unhappy mothers also may tend to perceive their nearly adult daughters as potential sources of companionship, solace, and emotional support (e.g., Jacobvitz & Bush, 1996). As has been pointed out, although daughters and mothers may both report finding such special bonds to be gratifying, and they even may be associated with high levels of apparent competence, there may be hidden costs related to the burden of providing friendship or parenting to a parent (Hetherington, 2003) and being a pleasing child at the expense of one's own childhood (Koslowska, 2001).

In turn, although again not entirely consistent across the literature, is the suggestion that family processes might differ when the child under study is a boy. For example, McHale (1995) found that, in maritally conflictual families, parents of sons were more likely to engage in hostile-competitive coparenting, each vying for an alliance with the child, whereas fathers of daughters were more likely to withdraw from the interaction. In contrast, Jouriles and Norwood (1995) found evidence that sons of maritally distressed parents were more likely than daughters to be involved in scapegoating or detouring patterns in which they were subject to hostility from both parents, which may have been accounted for by boys' tendency to demonstrate externalizing behavior problems in reaction to interparental conflict.

TOWARD THE FUTURE: GROWTH POINTS FOR DEVELOPMENTAL PSYCHOPATHOLOGY FROM A FAMILY SYSTEMS PERSPECTIVE

Incorporation of Biological Processes into Research on Family Systems

Although not reflected in classical family systems theory, modern systemic perspectives increasingly derive from the overarching biopsychosocial perspective shared by developmental psychopathology. In the biopsychosocial perspective, childhood adaptation and maladaptation are viewed as being a function of complex interactions among the individual's psychological and biological functioning, her or his stage of cognitive, interpersonal, and emotional development, the psychological and biological contributions of the family system in which that development takes place, and the larger societal context that shapes and constrains family members' responses to one another. "The biopsychosocial model assumes that illness in one family member affects the psychological health and relationships of other members, and, in turn, that the reactions of these members affect the functioning of the person with the illness" (Miklowitz, 2004, p. 669). The same might also be said for individual characteristics that bring to the family table sources of protection, resilience, and psychological health.

A number of different biological approaches are beginning to be integrated into research on family systems, including work from behavioral genetic, molecular genetic, and epigenetic perspectives (Beach & Whisman, 2013). Gene–environment correlations (passive rGEs) have been found to be helpful in understanding the development of externalizing disorders (Harold et al., 2013) whereas evocative gene–environment associations (evocative rGEs) have been found to account for the associations between interparental conflict and parenting dysfunctions (Ulbright et al., 2013) as well as the effects of marital strain on children's behavior problems. Moreover, additional research in molecular genetics suggests that individual differences in specific genes, such as the oxytocin receptor and serotonin transporter genes, may increase the vulnerability of family members to the spillover of interparental conflict (Sturge-Apple, Cicchetti, Davies, & Suor, 2012). Gene by environment interactions also have emerged, such as among youth carrying sensitivity alleles who are exposed to nonsupportive family environments (Brody et al., 2013). Epigenetic approaches, in turn, have shown how exposure to psychosocial toxins, such as child sexual abuse, affects gene methylation in ways that "are attributable to environmental reprogramming of genetic machinery" (Beach & Whisman, 2013, p. 2) and thus has subsequent profound and distinct impacts on developmental psychopathology (Beach et al., 2013).

Particularly exciting for the further development and testing of family systemic ideas is work that incorporates biological processes into the study of transactional processes in the family. For example, Mills-Koonce and colleagues (2007) found evidence suggestive that child polymorphism in the dopamine receptor gene had an evocative effect on parents' behavior, eliciting lower levels of sensitivity, and also moderated the association between maternal sensitivity and the emergence of child depressive symptoms.

Methodological Advances with Relevance for Family Research

In addition to new advances in available methods to test dynamic systems models, a number of methodological and statistical advances hold promise for advancing family systems research (Davies & Cicchetti, 2004). These include pattern-based approaches such as latent class analysis (e.g., Belsky & Fearon, 2004); multilevel modeling allowing for the comparison and integration of multiple sources of information within and about the family (e.g., Meunier, Boyle, O'Connor, & Jenkins, 2013); multivariate models encompassing complex interactions of moderation, mediation, moderated mediation, and mediated moderation effects (e.g., Auerbach & Ho, 2012; Lou, Lalonde, & Giguère, 2012); and growth curve modeling to investigate complex patterns and variables influencing change over time (e.g., Herrenkohl, Kosterman, Hawkins, & Mason, 2009).

Translational Research

As noted previously, from its inception the family systems perspective has demonstrated an investment in designing treatments to intervene in troubled family processes. Therefore, there is no shortage of research that is translational in that it brings family systems out of the laboratory and into the clinical arena (Spoth, 2008). Most of this family therapy research, however, has been focused on answering the question of whether these interventions work and on examining mediators and moderators of how and why they work, rather striving to use these findings to advance our theoretical understanding of family processes. A highly desirable goal of translational research is that it be transactional

and bidirectional—that is, that findings gleaned from the implementation of real-world applications of systemic constructs be used to reflect back on, refine, and enhance the development of family theory (Cicchetti & Gunnar, 2009; O'Connor, 2013).

Although there is no shortage of translational family therapy outcome research that could be cited here, far more than could be done justice in the page limits of the present chapter, a premier example comes from the multiple generations of work testing and refining the Parent Management Training Oregon (PMTO) model originally sired by Gerald Patterson's (1982) research on coercive family processes. This work has spawned a set of highly effective family systems interventions and has provided a key ingredient that has been folded with good effect into other innovative and integrative evidence-based interventions across a wide variety of presenting problems, including aggressive children (e.g., Bank, Rains, & Forgatch, 2004; Patterson, Chamberlain, & Reid, 1982); adolescents at risk for substance use (Dishion, Patterson, & Kavanagh, 1992); foster families (Eddy, Whaley, & Chamberlain, 2004); stepparents (Forgatch, Patterson, DeGarmo, & Beldavs, 2009); and families in diverse cultures (e.g., Domenech Rodríguez, Baumann, & Schwartz, 2011; Ogden & Hagen, 2008; Tremblay et al., 1991). One of the ways the PTMO model exemplifies the ideals of translational research is the attention paid to testing the underlying theory regarding the purported mechanisms of the treatment's effects (e.g., Chamberlain, Fisher, & Moore, 2002; DeGarmo, Patterson, & Forgatch, 2004; Patterson, Forgatch, & DeGarmo, 2010) and, in an iterative process, to using the findings of this research to further refine the underlying theory and, in turn, to modify and improve the interventions themselves (Patterson, 2005). Such growth edges include findings suggesting the possible benefits of incorporating components into the treatment that address parental attributions for child behavior (Snyder, Cramer, Afrank, & Patterson, 2005), that increase emotion regulation capacities (Stoolmiller & Snyder, 2004), that address sibling influences (Bank, Rains, & Forgatch, 2004), and that reduce associations with deviant peers (DeGarmo & Forgatch, 2002). In addition, moreover, this research team continues to use the insights gathered from their observations of family process to advance both research and theory, particularly in regard to developing models and methods for understanding family processes as dynamical systems (Granic & Dishion, 2003; Granic & Patterson, 2006).

CONCLUDING THOUGHTS

As this review of the historical and contemporary literature suggests, the richness of the feast that developmental psychopathology offers us can only be enhanced by inviting family systems theory to the table. The fact that so many family systems models evolved into therapy training institutes divorced from the academic, science-driven enterprise likely contributed to some estrangement, but a growing interest in translational research may help to provide a basis for rapprochement. At its heart, the family systems perspective embraces many of the fundamental principles of developmental psychopathology, including holism, bidirectionality, and the organizational perspective, and expands them further by situating each child and caregiver in a broader context of interconnected, dynamic, and developing relationships. The ongoing integration of these models promises to provide a mutually nourishing and sustaining repast.

REFERENCES

Ackerman, N. W. (1958). *The psychodynamics of family life*. New York, NY: Basic Books.

Alexander, J. F., & Parsons, B. V. (1982). *Functional family therapy*. Monterey, CA: Brooks/Cole.

Alexander, J. F., Robbins, M., Waldron, H., & Neeb, A. (2013). *Functional family therapy*. Washington, DC: American Psychological Association.

Allen, J. P., & Hauser, S. T. (1996). Autonomy and relatedness in adolescent-family interactions as predictors of young adults' states of mind regarding attachment. *Development and Psychopathology*, *8*(4), 793–809. doi: http://10.1017/S0954579400007434

Almeida, D. M., Wethington, E., & Chandler, A. L. (1999). Daily transmission of tensions between marital dyads and parent–child dyads. *Journal of Marriage and the Family*, *61*, 49–61.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders, (DSM-5)*. Washington, DC: American Psychiatric Press.

Anderson, L. P. (1999). Parentification in the context of the African American family. In N. D. Chase (Ed.), *Burdened children* (pp. 154–170). Thousand Oaks, CA: Sage.

Arnett, J. J., & Tanner, J. L. (2006). *Emergent adults in America: Coming of age in the 21st century*. Washington, DC: American Psychological Association.

Auerbach, R. P., & Ho, M. H. R. (2012). A cognitive-interpersonal model of adolescent depression: The impact of family conflict and depressogenic cognitive styles. *Journal of Clinical Child & Adolescent Psychology*, *41*(6), 792–802.

Bank, L., Burraston, B., & Snyder, J. (2004). Sibling conflict and ineffective parenting as predictors of adolescent boys' antisocial behavior and peer difficulties: Additive and interactional effects. *Journal of Research on Adolescence*, *14*, 99–125.

Barber, B. K. (2002). *Intrusive parenting: How psychological control affects children and adolescents*. Washington, DC: American Psychological Association.

Barber, B. K., & Buehler, C. (1996). Family cohesion and enmeshment: Different constructs, different effects. *Journal of Marriage and the Family*, *58*, 433–441.

Barber, B. K., Stolz, H. E., & Olson, J. A. (2005). *Parental support, psychological control, and behavioral control*. Boston, MA: Blackwell Publishing.

Barnett, D., Manly, J. T., & Cicchetti, D. (1993). Defining child maltreatment: The interface between policy and research. In D. Cicchetti, S. L. Toth, & I. E. Sigel (Eds.), *Child abuse, child development, and social policy* (pp. 7–73). Norwood, NJ: Ablex.

Beach, S. R. H., Brody, G. H., Lei, M. K., Gibbons, F. X., Gerrard, M., Simons, R. L., … Philibert, R. A. (2013). Impact of child sex abuse on adult psychopathology: A genetically and epigenetically informed investigation. *Journal of Family Psychology*, *27*, 3–11.

Beach, S. R. H., Wamboldt, M. Z., Kaslow, N. J., Heyman, R. E., & Reiss, D. (2006). Describing relationship problems in DSM-V: Toward better guidance for research and clinical practice. *Journal of Family Psychology*, *20*(3), 359–368.

Beach, S. R. H., & Whisman, M. A. (2013). Genetics and epigenetics in family context. *Journal of Family Psychology*, *27*, 1–2.

Bean, R. A., Barber, B. K., & Crane, D. R. (2006). Parental support, behavioral control, and psychological control among African American youth the relationships to academic grades, delinquency, and depression. *Journal of Family Issues*, *27*(10), 1335–1355.

Bean, R. A., & Northrup, J. C. (2009). Parental psychological control, psychological autonomy, and acceptance as predictors of self-esteem in Latino adolescents. *Journal of Family Issues*, *30*(11), 1486–1504.

Becker-Blease, K., & Kerig, P. K. (2015). *Child maltreatment: A developmental psychopathology perspective*. Washington, DC: American Psychological Association Press.

Belsky, J., & Fearon, R. P. (2004). Exploring marriage-parenting typologies and their contextual antecedents and developmental sequelae. *Development and Psychopathology*, *16*(3), 501–523.

Belsky, J., Putnam, S., & Crnic, K. (1996). Coparenting, parenting, and early emotional development. *New Directions in Child Development*, *7*, 45–56.

Bemporad, J. R., & Schwab, M. E. (1986). The DSM-III and clinical child psychiatry. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 135–151). New York, NY: Guilford.

Bond, M. H., Leung, K., Au, A., Tong, K., de Carrasquel, S. R., Murakami, F., …, Lewis, J. R. (2004). Culture-level dimensions of social axioms and their correlates across 41 cultures. *Journal of Cross-Cultural Psychology*, *35*, 548–570.

Bornstein, M. H., & Cheah, C. S. L. (2006). The place of "culture and parenting" in the ecological contextual perspective on developmental science. In K. H. Rubin & O. B. Chung (Eds.), *Parenting beliefs, behaviors, and parent–child relations: A cross-cultural perspective* (pp. 3–34). New York, NY: Taylor and Francis.

Boszormenyi-Nagy, I., & Framo, J. (Eds.). (1965). *Intensive family therapy: Theoretical and practical aspects*. New York, NY: Harper & Row.

Boszormenyi-Nagy, I., & Spark, G. (1973). *Invisible loyalties: Reciprocity in intergenerational family therapy*. New York, NY: Harper & Row.

Bowen, G. L., Martin, J. A., & Mancini, J. A. (2013). The resilience of military families: Theoretical perspectives. In M. A. Fine & F. D. Fincham (Eds.), *Handbook of family theories: A content-based approach* (pp. 417–436). New York, NY: Routledge Academic.

Bowen, M. (1961). Family psychotherapy. *American Journal of Orthopsychiatry*, *31*, 40–60.

Bowen, M. (1978). *Family therapy in clinical practice*. New York, NY: Jason Aronson.

Bowlby, J. (1977). The making and breaking of affectional bonds. I. Aetiology and psychopathology in the light of attachment theory. *British Journal of Psychiatry*, *130*(3), 201–210.

Boyd-Franklin, N. (2003). *Black families in therapy*. New York, NY: Guilford.

Brody, G. H., & Stoneman, Z. (1990). Sibling relationships. In I. E. Sigel & G. H. Brody (Eds.), *Methods of family research: Biographies of research projects* (pp. 189–212). Hillsdale, NJ: Erlbaum.

Brody, G. H., Yu, T., Chen, Y. F., Kogan, S. M., Evans, G. W., Windle, M., … Philibert, R. A. (2013). Supportive family environments, genes that confer sensitivity, and allostatic load among rural African American emerging adults: A prospective analysis. *Journal of Family Psychology*, *27*, 22–29.

Brooks-Gunn, J. (1990). Adolescents as daughters and as mothers: A developmental perspective. In I. Sigel & G. Brody (Eds.), *Methods of family research: Biographies of research projects: Vol. 1: Normal families* (pp. 213–248). Hillsdale, NJ: Erlbaum.

Buchanan, C. M., & Heiges, K. L. (2001). When conflict continues after marriage ends: Effects of postdivorce conflict on children. In J. H. Grych & F. D. Fincham (Eds.), *Interparental conflict and child development* (pp. 337–362). New York, NY: Cambridge University Press.

Buehler, C., & Gerard, J. M. (2002). Marital conflict, ineffective parenting, and children's and adolescents' maladjustment. *Journal of Marriage and Family*, *64*(1), 78–92.

Buist, K. L., Deković, M., & Prinzie, P. (2013). Sibling relationship quality and psychopathology of children and adolescents: A meta-analysis. *Clinical Psychology Review*, *33*, 97–106.

Burnette, M. KL., & Cicchetti, D. (2012). Multilevel approaches toward understanding antisocial behavior: Current research and future directions. *Development and Psychopathology*, *24*(3), 703–704.

Burton, L. M., & Hardaway, C. R. (2012). Low-income mothers as "other-mothers" to their romantic partners' children: Women's coparenting in multiple partner fertility relationships. *Family Process*, *51*(3), 343–359.

Carlson, E., & Sroufe, L. A. (1995). Contribution of attachment theory to developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. I: Theory and methods* (pp. 581–617). New York, NY: Wiley.

Carlson, M. J., & Högnäs, R. S. (2011). Coparenting in fragile families: Understanding how parents work together after a nonmarital birth. In J. McHale & K. M. Lindahl (Eds.), *Coparenting: A conceptual and clinical examination of family systems* (pp. 81–103). Washington, DC: American Psychological Association.

Carlson, M. J., & McLanahan, S. S. (2006). Strengthening unmarried families: Could enhancing couple relationships also improve parenting? *Social Service Review*, *80*(2), 297–321.

Carlson, M. J., McLanahan, S. S., & Brooks-Gunn, J. (2008). Coparenting and nonresident fathers' involvement with young children after a nonmarital birth. *Demography*, *45*(2), 461–488.

Chamberlain, P., Fisher, P. A., & Moore, K. J. (2002). Multidimensional treatment foster care: Applications of the OSLC intervention model to high-risk youth and their families. In J. B. Reid, G. R. Patterson, & J. Snyder (Eds.), *Antisocial behavior in children: Developmental theories*

and models for intervention (pp. 203–218). Washington, DC: American Psychological Association.

Chao, R. K. (2000). The parenting of immigrant Chinese and European American mothers: Relations between parenting styles, socialization goals, and parental practices. *Journal of Applied Developmental Psychology*, *21*, 233–248.

Christensen, A., & Margolin, G. (1988). Conflict and alliance in distressed and nondistressed families. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Relationships within families* (pp. 263–282). Oxford, UK: Clarendon.

Cicchetti, D. (2006). Development and psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Volume I: Theory and method* (2nd ed., pp. 1–23). New York, NY: Wiley.

Cicchetti, D. (2008). A multiple-levels-of-analysis perspective on research in development and psychopathology. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (pp. 27–57). New York, NY: Wiley.

Cicchetti, D., & Dawson, G. (Eds.). (2002). Multiple levels of analysis [Special Issue]. *Development and Psychopathology*, *14*, 417–666.

Cicchetti, D., & Gunnar, M. (2009). *Minnesota symposia on child psychology: Meeting the challenge of translational research in child psychology* (Vol. 35). New York, NY: Wiley.

Cicchetti, D., & Howes, P. W. (1991). Developmental psychopathology in the context of the family: Illustrations from the study of child maltreatment. *Canadian Journal of Behavioural Science*, *23*(3), 257–281.

Cicchetti, D., & Toth, S. L. (2005). Child maltreatment. *Annual Review of Clinical Psychology*, *1*(1), 409–438.

Cooper, J. E., Holman, J., & Braithwaite, V. A. (1983). Self-esteem and family cohesion: The child's perspective and adjustment. *Journal of Marriage and the Family*, 153–159.

Copeland, A. P., & White, K. M. (1991). *Studying families*. Newbury Park, CA: Sage.

Cowan, P. A., & Cowan, C. P. (1990). Becoming a family: Research and intervention. In I. E. Sigel & G. H. Brody (Eds.), *Methods of family research: Biographies of research projects. Vol. I: Normal families* (pp. 1–51). Hillsdale, NJ: Erlbaum.

Cowan, P. A., & Cowan, C. P. (2006). Developmental psychopathology from family systems and family risk factors perspectives: Implications for family research, practice, and policy. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Volume I: Theory and method* (2nd ed., pp. 530–587). New York, NY: Wiley.

Cowan, P. A., Cowan, C. P., & Kerig, P. K. (1993). Mothers, fathers, sons, and daughters: Gender differences in family formation and parenting style. In P. A. Cowan, D. Field, D. Hansen, A. Skolnick, & G. Swanson (Eds.), *Family, self and society: Toward a new agenda for family research* (pp. 165–195). Hillsdale, NJ: Erlbaum.

Cowan, P. A., Cowan, C. P., & Schulz, M. S. (1996). Thinking about risk and resilience in families. In E. M. Hetherington & E. A. Blechman (Eds.), *Advances in family research, Vol. 5: Stress, coping and resiliency in children and families* (pp. 1–37). Mahwah, NJ: Erlbaum.

Cox, M. J., & Paley, B. (2003). Understanding families as systems. *Current Directions in Psychological Science*, *12*(5), 193–196.

Cox, M. J., Paley, B., & Harter, K. (2001). Interparental conflict and parent–child relationships. In J. H. Grych & F. D. Fincham (Eds.), *Child development and interparental conflict* (pp. 249–272). Cambridge, UK: Cambridge University Press.

Crittenden, P. M. (1992). Quality of attachment in the preschool years. *Development and Psychopathology*, *4*(2), 209–241. doi: http://10.1017/S0954579400000110

Cummings, E. M., & Davies, P. T. (2011). *Marital conflict and children: An emotional security perspective:* New York, NY: Guilford.

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2000). *Developmental psychopathology and family process: Theory, research, and clinical implications*. New York, NY: Guilford.

Cummings, E. M., & Schermerhorn, A. C. (2003). A developmental perspective on children as agents in the family. In L. Kuczynski (Ed.), *Handbook of dynamics of parent–child relations* (pp. 91–108). Thousand Oaks, CA: Sage.

Cummings, E. M., Schermerhorn, A. C., Davies, P. T., Goeke-Morey, M. C., & Cummings, J. S. (2006). Interparental discord and child adjustment: Prospective investigations of emotional security as an explanatory mechanism. *Child Development*, *77*(1), 132–152.

Cuthbert, B., & Insel, T. (2013). Toward the future of psychiatric diagnosis: The seven pillars of RDoC. *BMC Medicine*, *11*(1), 126.

Davies, P. T., & Cicchetti, D. (2004). Toward an integration of family systems and developmental psychopathology approaches. *Development and Psychopathology*, *16*(3), 477–482.

Davies, P. T., & Cummings, E. M. (1994). Marital conflict and child adjustment: An emotional security hypothesis. *Psychological Bulletin*, *116*(3), 387.

Davies, P. T., & Cummings, E. M. (1998). Exploring children's emotional security as a mediator of the link between marital relations and child adjustment. *Child Development*, *69*(1), 124–139.

Davies, P. T., Cummings, E. M., & Winter, M. A. (2004). Pathways between profiles of family functioning, child security in the interparental subsysten, and child psychological problems. *Development and Psychopathology*, *16*(3), 525–550.

Davies, P. T., Harold, G. T., Goeke-Morey, M. C., & Cummings, E. M. (2002). Child emotional security and interparental conflict. *Monographs of the Society for Research in Child Development*, *67*, Serial No. 270, 1–127.

Davies, P. T., & Lindsay, L. L. (2004). Interparental conflict and adolescent adjustment: Why does gender moderate early adolescent vulnerability? *Journal of Family Psychology*, *18*(1), 160–170.

Davis, B. T., Hops, H., Alpert, A., & Sheeber, L. (1998). Child responses to parental conflict and their effect on adjustment: A study of triadic relations. *Journal of Family Psychology*, *12*, 163–177.

DeGarmo, D. S., & Forgatch, M. S. (2004). Putting problem solving to the test: Replicating experimental interventions for preventing youngsters' problem behaviors. In R. D. Conger, F. O. Lorenz, & K. A. S. Wickrama (Eds.), *Continuity and change in family relations: Theory, methods, and empirical findings* (pp. 189–223). Mahwah, NJ: Lawrence Erlbaum.

Denton, W. (2007). Issues for *DSM-V*: Relational diagnosis: An essential component of biopsychosocial assessment. *American Journal of Psychiatry*, *164*(8), 1146–1147.

Denton, W. H., & Bell, C. (2013). *DSM-5* and the family therapist: First-order change in a new millennium. *Australian and New Zealand Journal of Family Therapy*, *34*(2), 147–155.

Diamond, L. M., Butterworth, M. R., & Allen, K. (2012). Sexual minority development in the family context. In P. K. Kerig, M. S. Schulz, & S. T. Hauser (Eds.), *Adolescence and beyond: Family processes and development* (pp. 249–266). New York, NY: Oxford University Press.

Dishion, T. J., Patterson, G. R., & Kavanagh, K. A. (1992). An experimental test of the coercion model: Linking theory, measurement, and intervention. In J. McCord & R. Tremblay (Eds.), *The interaction of theory and practice: Experimental studies of intervention* (pp. 253–282). New York, NY: Guilford.

Domenech Rodríguez, M., Baumann, A., & Schwartz, A. (2011). Cultural adaptation of an evidence based intervention: From theory to practice in a Latino/a community context. *American Journal of Community Psychology*, *47*(1–2), 170–186.

Dumas, J. E., Lemay, P., & Dauwalder, J.-P. (2001). Dynamic analyses of mother–child interactions in functional and dysfunctional dyads: A synergetic approach. *Journal of Abnormal Child Psychology*, *29*(4), 317–329.

Dunn, J., & Davies, L. (2001). Sibling relationships and interparental conflict. In J. Grych & F. Fincham (Eds.), *Child development and interparental conflict* (pp. 273–290). New York, NY: Cambridge University Press.

Eddy, J. M., Whaley, R. B., & Chamberlain, P. (2004). The prevention of violent behavior by chronic and serious male juvenile offenders: A 2-year follow-up of a randomized clinical trial. *Journal of Emotional and Behavioral Disorders*, *12*(1), 2–8.

Emde, R. N. (1988). The effect of relationships on relationships: A developmental approach to clinical intervention. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Relationships within families: Mutual influences* (pp. 354–364). Oxford, UK: Clarendon Press.

Engfer, A. (1988). The interrelatedness of marriage and the mother–child relationship. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Relationships within families: Mutual influences* (pp. 104–118). Oxford, UK: Clarendon Press.

Epstein, N. B., Baldwin, L. M., & Bishop, D. S. (1983). The McMaster Family Assessment Device. *Journal of Marital and Family Therapy*, *9*(2), 171–180.

Erel, O., & Burman, B. (1995). Interrelatedness of marital relations and parent–child relations: A meta-analytic review. *Psychological Bulletin*, *118*(1), 108–132.

Fagan, J., Palkovitz, R., Roy, K., & Farrie, D. (2009). Pathways to paternal engagement: Longitudinal effects of risk and resilience on nonresident fathers. *Developmental Psychology*, *45*(5), 1389–1405.

Farr, R. H., & Patterson, C. J. (2013). Coparenting among lesbian, gay, and heterosexual couples: Associations with adopted children's outcomes. *Child Development*, *84*(4), 126–1240.

Favez, N., Lopes, F., Bernard, M., Frascarolo, F., Lavanchy Scaiola, C., Corboz-Warnery, A., & Fivaz-Depeursinge, E. (2012). The development of family alliance from pregnancy to toddlerhood and child outcomes at 5 years. *Family Process*, *51*(4), 542–556.

Fiese, B. H., Sameroff, A. J., Grotevant, H. D., Wamboldt, F. S., Dickstein, S., & Fravel, D. L. (2001). Observing families through the stories that they tell: A multidimensional approach. In P. K. Kerig & K. M. Lindahl (Eds.), *Family observational coding systems: Resources for systemic research* (pp. 259–271). Mahwah, NJ: Erlbaum.

First, M. B. (2006). Relational processes in the *DSM-V* revision process: Comment on the special section. *Journal of Family Psychology*, *20*(3), 359–368.

First, M. B. (2012). A practical prototypic system for psychiatric diagnosis: The ICD-11 Clinical Descriptions and Diagnostic Guidelines. *World Psychiatry*, *11*(1), 24–25.

First, M. B., Bell, C. C., Cuthbert, B., Krystal, J. H., Malison, R., Offord, D. R., . . . Wisner, K. L. (2002). Personality disorders and relational disorders. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM–V* (pp. 123–199). Arlington, VA: American Psychiatric Association.

Fivaz-Depeursinge, E., & Corboz-Warnery, A. (1999). *The primary triangle: A developmental systems view of mothers, fathers, and infants*. New York, NY: Basic Books.

Fivaz–Depeursinge, E., Lopes, F., Python, M., & Favez, N. (2009). Coparenting and toddler's interactive styles in family coalitions. *Family Process*, *48*(4), 500–516.

Fivush, R., Bohanek, J. G., & Duke, M. (2005). *The intergenerational self: Subjective perspective and family history*. Mahwah, NJ: Erlbaum.

Fivush, R., Bohanek, J. G., & Marin, K. (2010). Patterns of family narrative co-construction in relation to adolescent identity and well-being. In K. C. McLean & M. Pasupathi (Eds)., *Narrative development in adolescence* (pp. 45–63). New York: Springer

Fivush, R., Bohanek, J. G., & Zaman, W. (2011). Personal and intergenerational narratives in relation to adolescents' well-being. *New Directions for Child and Adolescent Development*, *131*, 45–57.

Florsheim, P., & Moore, D. R. (2012). Young fathers and the transition to parenthood: An interpersonal analysis of paternal outcomes. In P. K. Kerig, M. S. Schulz, & S. T. Hauser (Eds.), *Adolescence and beyond: family processes and development: family processes and development* (pp. 200–223). New York, NY: Oxford University Press.

Florsheim, P., Sumida, E., McCann, C., Winstanley, M., Fukui, R., Seefeldt, T., & Moore, D. (2003). The transition to parenthood among young African American and Latino couples: relational predictors of risk for parental dysfunction. *Journal of Family Psychology*, *17*(1), 65–79.

Foran, H., Beach, S., Smith Slep, A., Heyman, R., & Wamboldt, M. (2013). *Family problems and family violence: Reliable assessment and the ICD-11*. New York: Springer.

Foran, H., Whisman, M. A., Beach, S. R. H. (2015). Intimate partner relationship distress in the DSM-5. *Family Process*, *54*(1), 48–63.

Forgatch, M. S., Patterson, G. R., Degarmo, D. S., & Beldavs, Z. G. (2009). Testing the Oregon delinquency model with 9-year follow-up of the Oregon Divorce Study. *Development and Psychopathology*, *21*(2), 637.

Fosco, G. M., & Grych, J. H. (2008). Emotional, cognitive, and family systems mediators of children's adjustment to interparental conflict. *Journal of Family Psychology*, *22*(6), 843–854.

Friedman, N., Sadhu, J., & Jellinek, M. (2012). *DSM-5*: Implications for pediatric mental health care. *Journal of Developmental & Behavioral Pediatrics*, *33*(2), 163–178.

Fromm-Reichmann, F. (1948). Notes on the development of treatment of schizophrenics by psychoanalysis and psychotherapy. *Psychiatry*, *11*, 263–273.

Garber, J. (1984). Classification of childhood psychopathology: A developmental perspective. *Child Development*, *55*, 30–48.

Gavin, L. E., Black, M. M., Minor, S., Abel, Y., Papas, M. A., & Bentley, M. E. (2002). Young, disadvantaged fathers' involvement with their infants: An ecological perspective. *Journal of Adolescent Health*, *31*(3), 266–276.

Georgiades, K., Boyle, M. H., Jenkins, J. M., Sanford, M., & Lipman, E. (2008). A multilevel analysis of whole family functioning using the McMaster Family Assessment Device. *Journal of Family Psychology*, *22*(3), 344–354.

Gibbs, J. T., & Huang, L. N. (2003). *Children of color: Psychological interventions with culturally diverse youth* (2nd ed.). San Francisco, CA: Jossey-Bass.

Gjerde, P. F. (1986). The interpersonal structure of family interaction settings: Parent-adolescent relations in dyads and triads. *Developmental Psychology*, *22*(3), 297–304.

Goodman, C. C., & Silverstein, M. (2006). Grandmothers raising grandchildren: Ethnic and racial differences in well-being among custodial and coparenting families. *Journal of Family Issues*, *27*(11), 1605–1626.

Gordis, E. B., Margolin, G., & John, R. S. (1997). Marital aggression, observed parental hostility, and child behavior during triadic family interaction. *Journal of Family Psychology*, *11*, 76–89.

Granic, I., & Dishion, T. J. (2003). Deviant talk in adolescent friendships: A step toward measuring a pathogenic attractor process. *Social Development*, *12*(3), 314–334.

Granic, I., & Hollenstein, T. (2003). Dynamic systems methods for models of developmental psychopathology. *Development and Psychopathology*, *15*(3), 641–669.

Granic, I., Hollenstein, T., Dishion, T. J., & Patterson, G. R. (2003). Longitudinal analysis of flexibility and reorganization in early adolescence: A dynamic systems study of family interactions. *Developmental Psychology*, *39*(3), 606–617.

Granic, I., & Patterson, G. R. (2006). Toward a comprehensive model of antisocial development: A dynamic systems approach. *Psychological Review*, *113*(1), 101–131.

Gribble, P. A., Cowen, E. L., Wyman, P. A., Work, W. C., Wanon, M., & Raoof, A. (1993). Parent and child views of parent–child relationship qualities and resilient outcomes among urban children. *Journal of Child Psychology and Psychiatry*, *34*, 507–519.

Group for the Advancement of Psychiatry Committee on the Family. (1989). The challenge to relational diagnoses: Applying the biopsychosocial model in *DSM-IV*. *American Journal of Psychiatry*, *146*, 1492–1494.

Group for the Advancement of Psychiatry Committee on the Family. (1996). Global assessment of relational functioning scale (GARF): I. Background and rationale. *Family Process*, *35*(2), 155–190.

Grych, J. H., & Fincham, F. D. (1990). Marital conflict and children's adjustment: A cognitive-contextual framework. *Psychological Bulletin*, *108*(2), 267–290.

Grych, J. H., & Fincham, F. D. (1993). Children's appraisals of marital conflict: initial investigations of the cognitive-contextual framework. *Child Development*, *64*(1), 215–230.

Grych, J. H., Harold, G. T., & Miles, C. J. (2003). A prospective investigation of appraisals as mediators of the link between interparental conflict and child adjustment. *Child Development*, *74*(4), 1176–1193.

Grych, J. H., Raynor, S. R., & Fosco, G. M. (2004). Family processes that shape the impact of interparental conflict on adolescents. *Development and Psychopathology*, *16*(3), 649–665.

Grych, J. H., Seid, M., & Fincham, F. D. (1992). Assessing marital conflict from the child's perspective: The children's perceptions of interparental conflict scale. *Child Development*, *63*, 558–572.

Guttman, H. A. (2002). The epigenesis of the family system as a context for individual development. *Family Process*, *41*(3), 533–545.

Haley, J. (1963). *Strategies of psychotherapy*. New York, NY: Grune & Stratton.

Haley, J. (1976). *Double bind*. New York, NY: Grune & Stratton.

Hare-Mustin, R. (1978). A feminist approach to family therapy. *Family Process*, *17*, 181–194.

Hare-Mustin, R. T. (1994). Discourses in the mirrored room: A postmodern analysis of therapy. *Family Process*, *33*(1), 19–35.

Harold, G. T., Leve, L. D., Elam, K. K., Thapar, A., Neiderhiser, J. M., Natsuaki, M. N., . . . Reiss, D. (2013). The nature of nurture: Disentangling passive genotype-environment correlation from family relationship influences on children's externalizing problems. *Journal of Family Psychology*, *27*, 12–21.

Hazen, N., Jacobvitz, D., & McFarland, L. (2005). Antecedents of boundary disturbances in families with young children: Intergenerational transmission and parent-infant caregiving patterns. In P. K. Kerig (Ed.), *Implications of parent–child boundary dissolution for developmental psychopathology* (pp. 85–110). Binghamton, NY: Haworth.

Herrenkohl, T. I., Kosterman, R., Hawkins, J. D., & Mason, W. A. (2009). Effects of growth in family conflict in adolescence on adult depressive symptoms: Mediating and moderating effects of stress and school bonding. *Journal of Adolescent Health*, *44*(2), 146–152.

Hetherington, E. M. (2003). Social support and the adjustment of children in divorced and remarried families. *Childhood*, *10*(2), 217–236.

Heyman, R. E., & Smith Slep, A. M. (2009). Reliability of family maltreatment diagnostic criteria: 41 site dissemination field trial. *Journal of Family Psychology*, *23*(6), 905–910.

Heyman, R. E., Smith Slep, A. M., & Foran, H. M. (2015). Enhanced definitions of intimate partner violence for DSM-5 and iCD-11 may promote improved screening and treatment. *Family Process*, *54*(1), 64–81.

Hinde, R. A. (1976). On describing relationships. *Journal of Child Psychology and Psychiatry*, *17*, 1–19.

Howes, P. W., & Cicchetti, D. (1993). A family/relational perspective on maltreating families: Parallel processes across systems and social policy implications. In D. Cicchetti & S. L. Toth (Eds.), *Child abuse, child development and social policy* (pp. 249–300). Norwood, NJ: Ablex.

Howes, P. W., Cicchetti, D., Toth, S. L., & Rogosch, F. A. (2000). Affective, organizational, and relational characteristics of maltreating families: A systems perspective. *Journal of Family Psychology*, *14*(1), 95–110.

Jackson, D. D. (1965). The study of the family. *Family Process*, *4*(1), 1–20.

Jackson, E. F., Raval, V. V., Bendikas, E. A., Raval, P. H., & Trivedi, S. S. (in press). Cultural variations in reports of subjective experience of parent–child boundary dissolution among emerging adults. *Journal of Family Issues*.

Jacobvitz, D., Hazen, N., Curran, M., & Hitchens, K. (2004). Observations of early triadic family interactions: Boundary disturbances in the family predict depressive and anxious symptoms in middle childhood. *Development and Psychopathology*, *16*(3), 577–592.

Jacobvitz, D., Riggs, S., & Johnson, E. (1999). Cross-sex and same-sex family alliances: Immediate and long-term effects on sons and daughters. In N. D. Chase (Ed.), *Burdened children* (pp. 34–55). Thousand Oaks, CA: Sage.

Jacobvitz, D. B., & Bush, N. F. (1996). Reconstructions of family relationships: Parent–child alliances, personal distress, and self-esteem. *Developmental Psychology*, *32*(4), 732–743.

Jacobvitz, D. B., & Sroufe, L. A. (1987). The early caregiver–child relationship and attention-deficit disorder with hyperactivity in kindergarten: A prospective study. *Child Development*, *58*(6), 1496–1504.

Jewell, J. D., & Stark, K. D. (2003). Comparing the family environments of adolescents with conduct disorder or depression. *Journal of Child and Family Studies*, *12*(1), 77–89.

Johnston, J. R., Walters, M. G., & Olesen, N. W. (2005). Is it alienating parenting, role reversal or child abuse? In P. K. Kerig (Ed.), *Implications of parent–child boundary dissolution for developmental psychopathology* (pp. 191–218). Binghamton, NY: Haworth.

Jones, D. J., & Lindahl, K. M. (2011). Coparenting in extended kinship systems: African American, Hispanic, Asian heritage, and Native American families. In J. P. McHale & K. M. Lindahl (Eds.), *Coparenting: A conceptual and clinical examination of family systems* (pp. 61–80). Washington, DC: American Psychological Association.

Jones, D. J., Shaffer, A., Forehand, R., Brody, G., & Armistead, L. P. (2003). Coparent conflict in single mother-headed African American families. *Behavior Therapy*, *34*(2), 259–272.

Jouriles, E. N., & Norwood, W. D. (1995). Physical aggression toward boys and girls in families characterized by the battering of women. *Journal of Family Psychology*, *9*(1), 69–78.

Jurkovic, G. J. (1997). *Lost childhoods: The plight of the parentified child*. New York, NY: Brunner/Mazel.

Jurkovic, G. J., Kuperminc, G. P., Sarac, T., & Weisshaar, D. (2005). Role of filial responsibility in the post-war adjustment of Bosnian young adolescents. *Journal of Emotional Abuse*, *5*(4), 219–235.

Kağıtçıbaşı, C. (2005). Autonomy and relatedness in cultural context implications for self and family. *Journal of Cross-Cultural Psychology*, *36*(4), 403–422.

Kağıtçıbaşı, C. (2007). *Family, self, and human development across cultures: Theory and applications* (2nd ed.). Mahwah, NJ: Erlbaum.

Kağıtçıbaşı, C., & Ataca, B. (2005). Value of children and family change: A three-decade portrait from Turkey. *Applied Psychology*, *54*(3), 317–337.

Kameguchi, K. (1996). Chaotic states of generational boundaries in contemporary Japanese families. In M. Cusinato (Ed.), *Research on family*

resources and needs across the world (pp. 235–242). Milan, Italy: Edizioni Universitarie di Lettere Economia Diritto.

Kaplan, G. (2012). Moving back home: Insurance against labor market risk. *Journal of Political Economy*, *120*(3), 446–512.

Kashy, D. A., & Kenny, D. A. (2000). The analysis of data from dyads and groups. In H. T. Reis & C. M. Judd (Eds.), *Handbook of research methods in social and personality psychology* (pp. 451–477). New York, NY: Cambridge University Press

Kaslow, F. (1996). History, rationale and philosophic overview of issues and assumptions. In F. Kaslow (Ed.), *Handbook of relational diagnosis and dysfunctional family patterns* (pp. 3–28). New York, NY: Wiley.

Kaslow, N. J., Deering, C. G., & Ash, P. (1996). Relational diagnosis of child and adolescent depression. In F. Kaslow (Ed.), *Handbook of relational diagnosis and dysfunctional family patterns* (pp. 171–185). New York, NY: Wiley.

Katz, L. F., & Gottman, J. M. (1996). Spillover effects of marital conflict: In search of parenting and coparenting mechanisms. *New Directions for Child and Adolescent Development*, *74*, 57–76.

Katz, L. F., & Low, S. M. (2004). Marital violence, co-parenting and family-level processes, and children's adjustment. *Journal of Family Psychology*, *18*, 372–382.

Katz, L. F., & Woodin, E. M. (2002). Hostility, hostile detachment, and conflict engagement in marriages: Effects on child and family functioning. *Child Development*, *73*(2), 636–652.

Keller, H., & Otto, H. (2009). The cultural socialization of emotion regulation during infancy. *Journal of Cross-Cultural Psychology*, *40*(6), 996–1011.

Kerig, P. K. (1995). Triangles in the family circle: Effects of family structure on marriage, parenting, and child adjustment. *Journal of Family Psychology*, *9*(1), 28–43.

Kerig, P. K. (1996). Assessing the links between marital conflict and child development: The Conflicts and Problem-Solving Scales. *Journal of Family Psychology*, *10*, 454–473.

Kerig, P. K. (1998). Gender and appraisals as mediators of adjustment in children exposed to interparental violence. *Journal of Family Violence*, *15*, 345–363.

Kerig, P. K. (2001a). Children's coping with interparental conflict. In J. H. Grych & F. D. Fincham (Eds.), *Interparental conflict and child development* (pp. 213–245). Cambridge, UK: Cambridge University Press.

Kerig, P. K. (2001b). Conceptual issues in family observational research. In P. K. Kerig & K. M. Lindahl (Eds.), *Family observational coding systems: Resources for systemic research* (pp. 1–22). Hillsdale, NJ: Erlbaum.

Kerig, P. K. (2005). Revisiting the construct of boundary dissolution: A multidimensional perspective. In P. K. Kerig (Ed.), *Implications of parent–child boundary dissolution for developmental psychopathology* (pp. 5–42). New York, NY: Haworth.

Kerig, P. K. & Alexander, J. F. (2012). Family matters: Integrating trauma treatment into Functional Family Therapy with delinquent youth. *Journal of Child and Adolescent Trauma*, *5(3)*, 205–223.

Kerig, P. K., Cowan, P. A., & Cowan, C. P. (1993). Marital quality and gender differences in parent–child interaction. *Developmental Psychology*, *29*, 931–939.

Kerig, P. K., Fedorowicz, A. E., Brown, C. A., Patenaude, R. L. and Warren, M. (1999). When warriors are worriers: Gender, appraisals, and children's strategies for coping with interparental violence. *Journal of Emotional Abuse*, *1*(2), 89–114.

Kerig, P. K., & Lindahl, K. M. (2001). *Family observational coding systems: Resources for systemic research*. Hillsdale, NJ: Erlbaum.

Kerig, P. K., Ludlow, A., & Wenar, C. (2012). *Developmental psychopathology: From infancy through adolescence* (6th ed.). Maidenhead, UK: McGraw-Hill.

Kerig, P. K., & Schulz, M. S. (2012). The transition from adolescence to adulthood: What lies beneath and what lies beyond. In P. K. Kerig, M. S. Schulz, & S. T. Hauser (Eds.), *Adolescence and beyond: Family processes in development* (pp. 3–12). New York, NY: Oxford University Press.

Kerig, P. K. & Swanson, J. A. (2010). Ties that bind: Triangulation, boundary dissolution, and the effects of interparental conflict on child development. In M. S. Schulz, M. K. Pruett, P. K. Kerig, & R. Parke (Eds.), *Strengthening couple relationships for optimal child development* (pp. 59–76). Washington, DC: American Psychological Association.

Kerig, P. K., Swanson, J. A., & Ward, R. M. (2012). Autonomy with connection: Influences of parental psychological control on mutuality in emerging adults' intimate relationships. In P. K. Kerig, M. S. Schulz, & S. T. Hauser (Eds.), *Adolescence and beyond: Family processes in development* (pp. 134–153). New York, NY: Oxford University Press.

Kerr, M. E., & Bowen, M. (1988). *Family evaluation*. New York, NY: W.W. Norton.

Kitzmann, K. M. (2000). Effects of marital conflict on subsequent triadic family interactions and parenting. *Developmental Psychology*, *36*, 3–13.

Koslowska, K. (2001). Good children presenting with conversion disorder. *Clinical Child Psychology and Psychiatry*, *4*, 575–591.

Krishnakumar, A., & Black, M. M. (2003). Family processes within three-generation households and adolescent mothers' satisfaction with father involvement. *Journal of Family Psychology*, *17*(4), 488–498.

Kupfer, M. D. J., First, M. B., & Regier, D. A. (2008). *A research agenda for DSM V*. Arlington, VA: American Psychiatric Press.

Lamb, M. E. (2010). *The role of the father in child development* (5th ed.). Hoboken, NJ: Wiley.

Lamb, M. E. (2013). The changing faces of fatherhood and father–child relationships: From fatherhood as status to father as Dad. In M. A. Fine & F. D. Fincham (Eds.), *Handbook of family theories: A content-based approach* (pp. 87–103). New York, NY: Routledge.

Leary, A., & Katz, L. F. (2004). Coparenting, family-level processes, and peer outcomes: The moderating role of vagal tone. *Development and Psychopathology*, *16*(3), 593–608.

Lidz, T., Cornelison, A., Fleck, S., & Terry, D. (1957). The intrafamilial environment of the schizophrenic patient. II: Marital schism and marital skew. *American Journal of Psychiatry*, *114*(3), 241–248.

Lieberman, A. F. (1992). Infant–parent psychotherapy with toddlers. *Development and Psychopathology*, *4*(4), 559–574.

Lindahl, K. M., Clements, M., & Markman, H. (1997). Predicting marital and parent functioning in dyads and triads: A longitudinal investigation of marital processes. *Journal of Family Psychology*, *11*(2), 139–151.

Lindahl, K. M., & Malik, N. M. (1999). Observations of marital conflict and power: Relations with parenting in the triad. *Journal of Marriage and the Family*, *61*(2), 320–330.

Lindahl, K. M., & Malik, N. M. (2001). The system for coding interactions and family functioning. In P. K. Kerig & K. M. Lindahl (Eds.), *Family observational coding systems* (pp. 77–92). Hillsdale, NJ: Erlbaum.

Lindahl, K. M., Malik, N. M., Kaczynski, K., & Simons, J. S. (2004). Couple power dynamics, systemic family functioning, and child adjustment: A test of a mediational model in a multiethnic sample. *Development and Psychopathology*, *16*(3), 609–630.

Loper, A. B., Phillips, V., Nichols, E. B., & Dallaire, D. H. (2014). Characteristics and effects of the co-parenting alliance between incarcerated parents and child caregivers. *Journal of Child and Family Studies*, *23*(2), 225–241.

Lou, E., Lalonde, R. N., & Giguère, B. (2012). Making the decision to move out: Bicultural young adults and the negotiation of cultural demands and family relationships. *Journal of Cross-Cultural Psychology*, *43*(5), 663–670.

Luepnitz, D. A. (1988). *The family interpreted: Feminist theory in clinical practice*. New York, NY: Basic Books.

Luna, I., de Ardon, E. T., Lim, Y. M., Cromwell, S. L., Phillips, L. R., & Russell, C. K. (1996). The relevance of familism in cross-cultural studies of family caregiving. *Western Journal of Nursing Research*, *18*(3), 267–283.

Lynch, M., & Cicchetti, D. (1998). An ecological-transactional analysis of children and contexts: The longitudinal interplay among child maltreatment, community violence, and children's symptomatology. *Development and Psychopathology*, *10*(2), 235–257.

Lyons-Ruth, K., Bureau, J.-F., Holmes, B., Easterbrooks, A., & Brooks, N. H. (2013). Borderline symptoms and suicidality/self-injury in late adolescence: Prospectively observed relationship correlates in infancy and childhood. *Psychiatry Research*, *206*(2–3), 273–281.

MacFie, J., Houts, R. M., McElwain, N. L., & Cox, M. J. (2005). The effect of father-toddler and mother-toddler role reversal on the development of behavior problems in kindergarten. *Social Development*, *14*(3), 514–531.

MacFie, J., Toth, S. L., Rogosch, F. A., Robinson, J., Emde, R. N., & Cicchetti, D. (1999). Effect of maltreatment on preshoolers' narrative representations of responses to relieve distress and of role reversal. *Developmental Psychology*, *35*(2), 460–465.

Mahoney, A., Coffield, A., Lewis, T., & Lashley, S. (2001). Meso-Analytic Behavioral Rating System for Family Interactions: Usefulness for observing play and forced-compliance tasks with young children. In P. Kerig & K. Lindahl (Eds.), *Family observation coding systems* (pp. 225–241). Mahwah, NJ: Erlbaum.

Melby, J. N., & Conger, R. D. (2001). The Iowa Family Interaction Rating Scales: Instrument summary. In P. K. Kerig & K. M. Lindahl (Eds.), *Family observational coding systems* (pp. 33–58). Mahwah, NJ: Erlbaum.

Manzi, C., Vignoles, V. L., Regalia, C., & Scabini, E. (2006). Cohesion and enmeshment revisited: Differentiation, identity, and well-being in two European cultures. *Journal of Marriage and Family*, *68*(3), 673–689.

Margolin, G. (1990). Marital conflict. In G. H. Brody & I. E. Sigel (Eds.), *Methods of family research: Biographies of research projects. Vol. II: Clinical populations* (pp. 191–225). Hillsdale, NJ: Erlbaum.

Margolin, G., Gordis, E. B., & John, R. S. (2001). Coparenting: A link between marital conflict and parenting in two-parent families. *Journal of Family Psychology*, *15*(1), 3–21.

Margolin, G., Gordis, E. B., & Oliver, P. H. (2004). Links between marital and parent–child interactions: Moderating role of husband-to-wife aggression. *Development and Psychopathology*, *16*(3), 753–771.

Markus, H. R., & Kitayama, S. (1991). Culture and the self: Implications for cognition, emotion, and motivation. *Psychological Review*, *98*(2), 224–253.

Masten, A. S., & Powell, J. L. (2003). A resilience framework for research, policy, and practice. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 1–25). New York, NY: Cambridge University Press.

McConnell, M. C., & Kerig, P. K. (2002). Assessing coparenting in families of school-age children: Validation of the coparenting and family rating system. *Canadian Journal of Behavioural Science*, *34*(1), 44–58.

McCubbin, H. I., & McCubbin, M. A. (1988). Typologies of resilient families: Emerging roles of social class and ethnicity. *Family Relations*, *37*(3)–254.

McGoldrick, M., & Carter, B. (2003). The family life cycle. In F. Walsh (Ed.), *Normal family processes* (pp. 460–489). New York, NY: Guilford.

McGoldrick, M., Giordano, J., & Garcia-Preto, N. (2005). *Ethnicity and family therapy* (3rd ed.). New York, NY: Guilford.

McHale, J. P. (1995). Coparenting and triadic interactions during infancy: The roles of marital distress and child gender. *Developmental Psychology*, *31*(6), 985–996.

McHale, J. P. (1997). Overt and covert coparenting processes in the family. *Family Process*, *36*(2), 183–210.

McHale, J., Fivaz-Depeursinge, E., Dickstein, S., Robertson, J., & Daley, M. (2008). New evidence for the social embeddedness of infants' early triangular capacities. *Family Process*, *47*(4), 445–463.

McHale, J., & Irace, K. (2011). Coparenting in diverse family systems. In J. McHale & K. M. Lindahl (Eds.), *Coparenting: A conceptual and clinical examination of family systems* (pp. 15–37). Washington, DC: American Psychological Association.

McHale, J. P., Kazali, C., Rotman, T., Talbot, J., Carleton, M., & Lieberson, R. (2004). The transition to coparenthood: Parents' prebirth expectations and early coparental adjustment at 3 months postpartum. *Development and Psychopathology*, *16*(3), 711–733.

McHale, J. P., Kuersten, R., & Lauretti, A. (1996). New directions in the study of family-level dynamics during infancy and early childhood. *New Directions for Child and Adolescent Development*, *74*, 5–26.

McHale J. P., Kuersten-Hogan, R., & Lauretti, A. (2001). Evaluating coparenting and family-level dynamics during infancy and early childhood: The Coparenting and Family Rating System. In P. K. Kerig & K. M. Lindahl (Eds.), *Family observational coding systems* (pp. 151–170). Mahwah, NJ: Erlbaum.

McHale, J. P., & Rasmussen, J. L. (1998). Coparental and family group-level dynamics during infancy: Early family precursors of child and family functioning during preschool. *Development and Psychopathology*, *10*(1), 39–59.

Meunier, J. C., Boyle, M., O'Connor, T. G., & Jenkins, J. M. (2013). Multilevel mediation: Cumulative contextual risk, maternal differential treatment, and children's behavior within families. *Child Development*, *84*(5), 1594–1615.

Micucci, J. (2009). *Adolescents in family therapy: Harnessing the power of relationships*. New York, NY: Guilford.

Miklowitz, D. J. (2004). The role of family systems in severe and recurrent psychiatric disorders: A developmental psychopathology view. *Development and Psychopathology*, *16*(03), 667–688.

Miller, S., Winn, D. M., Taylor, J., & Wiki, T. (2012). Girls' relational orientation and interpersonal dynamics of delinquency delinquent girls. In S. Miller, L. D. Leve, & P. K. Kerig (Eds.), *Delinquent girls: Contexts, relationships, and adaptation* (pp. 85–101). New York, NY: Springer.

Mills-Koonce, W. R., Propper, C. B., Gariepy, J., Blair, C., Garrett-Peters, P., & Cox, M. J. (2007). Bidirectional genetic and environmental influences of mother and child behavior: The family system as the unit of analyses. *Development and Psychopathology*, *19*(4), 1073–1087.

Minuchin, P. (1985). Families and individual development: Provocations from the field of family therapy. *Child Development*, *56*, 289–302. doi: http://www.jstor.org/stable/1129720

Minuchin, P., Colapinto, J., & Minuchin, S. (2007). *Working with families of the poor* (2nd ed.). New York, NY: Guilford.

Minuchin, S. (1967). *Families of the slums*. New York, NY: Basic Books.

Minuchin, S. (1974). *Families and family therapy*. Cambridge, MA: Harvard University Press.

Minuchin, S., & Fishman, H. C. (1975). *Family therapy techniques*. Boston, MA: Harvard University Press.

Murry, V. M., Bynum, M. S., Brody, G. H., Willert, A., & Stephens, D. (2001). African American single mothers and children in context: A review of studies of risk and resilience. *Clinical Child and Family Psychology Review*, *4*(2), 133–155.

National Institute of Mental Health. (2011). NIMH Research Domain Criteria (RDoC). Rockville, MD: National Institute of Mental Health. Retrieved from http://www.nimh.nih.gov/research-funding/rdoc/nimh-research-domain-criteria-rdoc.shtml

O'Brien, M., Bahadur, M. A., Gee, C., Balto, K., & Erber, S. (1997). Child exposure to marital conflict and child coping responses as predictors of child adjustment. *Cognitive Therapy and Research*, *21*(1), 39–59.

O'Connor, T. (2013). Editorial: Translational research in practice. *Journal of Child Psychology and Psychiatry*, *54*(11), 1153–1154.

Ogden, T., & Hagen, K. A. (2008). Treatment effectiveness of parent management training in Norway: a randomized controlled trial of children with conduct problems. *Journal of Consulting and Clinical Psychology*, *76*(4), 607.

Olson, D. H., Russell, C. S., & Sprenkle, D. H. (1983). Circumplex model of marital and family systems: Vl. Theoretical update. *Family Process*, *22*(1), 69–83.

Olson, D. H., Sprenkle, D. H., & Russell, C. S. (1979). Circumplex model of marital and family systems: I. Cohesion and adaptability dimensions, family types, and clinical applications. *Family Process*, *18*(1), 3–28.

Osborne, L. N., & Fincham, F. D. (1996). Marital conflict, parent–child relationships, and child adjustment: Does gender matter? *Merrill-Palmer Quarterly*, *42*(1), 48–75.

Palazzoli, M. S., Cecchin, G., Prata, G., & Boscolo, L. (1978). *Paradox and counterparadox: A new model of the family in schizophrenic transaction*. New York, NY: Aronson.

Papp, P. (1983). *The process of change*. New York, NY: Guilford.

Parke, R. D. (1990). In search of fathers: A narrative of an empirical journey. In I. E. Sigel & G. H. Brody (Eds.), *Methods of family research: Biographies of research projects. Vol. I: Normal families* (pp. 153–188). Hillsdale, NJ: Erlbaum.

Patrick, M. R., Snyder, J., Schrepferman, L. M., & Snyder, J. (2005). The joint contribution of early parental warmth, communication and tracking, and early child conduct problems on monitoring in late childhood. *Child Development*, *76*(5), 999–1014.

Patterson, G. R. (1975). *Families: Applications of social learning to family life*. Champaign, IL: Research Press.

Patterson, G. R. (1982). *Coercive family process: A social learning approach*. Eugene, OR: Castalia.

Patterson, G. R. (2005). The next generation of PMTO models. *Behavior Therapist*, *28*(2), 27–33.

Patterson, G. R., Chamberlain, P., & Reid, J. B. (1982). A comparative evaluation of parent training procedures. *Behavior Therapy*, *13*, 638–651.

Patterson, G. R., & Forgatch, M. (1987). *Parents and adolescents living together: Part 1: The basics*. Eugene, OR: Castalia.

Patterson, G. R., Forgatch, M. S., & DeGarmo, D. S. (2010). Cascading effects following intervention. *Development and Psychopathology*, *22*(4), 949–970.

Phares, V. (1992). Where's Poppa? The relative lack of attention to the role of fathers in child and adolescent psychopathology. *American Psychologist*, *47*(5), 656–664.

Phares, V., Fields, S., Kamboukos, D., & Lopez, E. (2005). Still looking for Poppa. *American Psychologist*, *60*(7), 735–736.

Pittman, L. D., Wakschlag, L. S., Chase-Lansdale, P. L., & Brooks-Gunn, J. (2012). "Mama, I'm a person, too!" Individuation and young African-American mothers' parenting competence. In P. K. Kerig, M. S. Schulz, & S. T. Hauser (Eds.), *Adolescence and beyond: Family processes in development* (pp. 177–199). New York, NY: Oxford University Press.

Reiss, D., Neiderhiser, J. M., Hetherington, E. M., & Plomin, R. (2000). *The relationship code: Deciphering genetic and social influences on adolescent development*. New York, NY: Cambridge University Press.

Richter, L. M., & Desmond, C. (2008). Targeting AIDS orphans and child-headed households: A perspective from national surveys in South Africa, 1995–2005. *AIDS Care*, *20*(9), 1019–1028.

Rinaldi, C. M., & Howe, N. (2003). Perceptions of constructive and destructive conflict within and across family subsystems. *Infant and Child Development*, *12*, 441–459.

Ringuette, E. L., & Kennedy, T. (1966). An experimental study of the double bind hypothesis. *Journal of Abnormal Psychology*, *71*(2), 136–141.

Robbins, M. S., Hervis, O., Mitrani, V. B., & Szapocznik, J. (2001). Assessing changes in family interaction: The structural family systems ratings. In P. K. Kerig & K. M. Lindahl (Eds.), *Family observational coding systems* (pp. 207–224). Mahwah, NJ: Erlbaum.

Rohner, R. P. (1986). *The warmth dimension: Foundations of parental acceptance-rejection theory*. Thousand Oaks, CA: Sage Publications.

Rowa, K., Kerig, P. K., & Geller, J. (2001). The family and anorexia nervosa: examining parent-child boundary problems[†]. *European Eating Disorders Review*, *9*(2), 97–114.

Rutter, M. (1988). Functions and consequences of relationships: Some psychopathological considerations. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Relationships within families: Mutual influences* (pp. 332–353). Oxford, UK: Clarendon Press.

Rutter, M. (1990). Psychosocial resilience and protective mechanisms. In J. Rolf, A. S. Masten, D. Cicchetti, K. H. Nuechterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 181–214). New York, NY: Cambridge University Press.

Sameroff, A. J. (1995). General systems theories and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. I: Theory and methods* (pp. 659–695). New York, NY: Wiley.

Sayers, S. L., & McGrath, K. (2004). Data analytic strategies for couple observational coding systems. In P. K Kerig & D. H. Baucom (Eds.), *Couple observational coding systems* (pp. 43–66). Mahwah, NJ: Erlbaum.

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development*, *63*(1), 1–19.

Schermerhorn, A. C., & Cummings, M. E. (2008). Transactional family dynamics: A new framework for conceptualizing family influence processes. *Advances in Child Development and Behavior*, *36*, 187–250.

Schermerhorn, A. C., Cummings, E. M., & Davies, P. T. (2008). Children's representations of multiple family relationships: Organizational structure and development in early childhood. *Journal of Family Psychology*, *22*, 89–101.

Schermerhorn, A. C., Cummings, E. M., DeCarlo, C. A., & Davies, P. T. (2007). Children's influence in the marital relationship. *Journal of Family Psychology*, *21*(2), 259–269.

Shaffer, A., & Egeland, B. (2011). Intergenerational transmission of familial boundary dissolution: Observations and psychosocial outcomes in adolescence. *Family Relations*, *60*(3), 290–302.

Shaffer, A., & Sroufe, L. A. (2005). The developmental and adaptational implications of generational boundary dissolution: Findings from a prospective, longitudinal study. In P. K. Kerig (Ed.), *Implications of parent–child boundary dissolution for developmental psychopathology* (pp. 67–84). Binghamton, NY: Haworth.

Shaw, D. S., Criss, M. M., Schonberg, M. A., & Beck, J. E. (2004). The development of family hierarchies and their relation to

children's conduct problems. *Development and Psychopathology*, *16*(3), 483–500.

Sheeber, L., Davis, B., & Hops, H. (2002). Gender specific vulnerability to depression in children of depressed mothers. In S. H. Goodman & I. H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment* (pp. 253–274). Washington, DC: American Psychological Association.

Shortt, J. W., Stoolmiller, M., Smith-Shine, J. N., Mark Eddy, J., & Sheeber, L. (2010). Maternal emotion coaching, adolescent anger regulation, and siblings' externalizing symptoms. *Journal of Child Psychology and Psychiatry*, *51*(7), 799–808.

Silverstein, L. B., & Goodrich, T. J. (2003). *Feminist family therapy: Empowerment in social context*. Washington, DC: American Psychological Association.

Singer, M. T., & Wynne, L. C. (1965). Thought disorder and family relations of schizophrenics: IV. Results and implications. *Archives of General Psychiatry*, *12*(2), 201–212.

Skinner, H. A., Steinhauer, P. D., & Santa-Barbara, J. (1983). The family assessment measure. *Canadian Journal of Community Mental Health*, *2*(2), 91–103.

Slipp, S. (1991). *The technique and practice of object relations family therapy*. New York, NY: Aronson.

Smith Slep, A. M. , Heyman, R. E., & Foran, H. M. (2015). Child maltreatment in DSM-5 and ICD-11. *Family Process*, *54*(1), 17–32.

Snyder, J., Cramer, A., Afrank, J., & Patterson, G. R. (2005). The contributions of ineffective discipline and parental hostile attributions of child misbehavior to the development of conduct problems at home and school. *Developmental Psychology*, *41*(1), 30–41.

Spoth, R. (2008). Translating family-focused prevention science into effective practice: Toward a translational impact paradigm. *Current Directions in Psychological Science*, *17*(6), 415–421.

Sroufe, J. W. (1991). Assessment of parent–adolescent relationships: Implications for adolescent development. *Journal of Family Psychology*, *5*(1), 21–45.

Sroufe, L. A. (1990). Considering normal and abnormal together: The essence of developmental psychopathology. *Development and Psychopathology*, *2*(4), 335–348.

Sroufe, L. A., & Fleeson, J. (1988). The coherence of family relationships. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Relationships within families: Mutual influences* (pp. 27–47). Oxford, UK: Oxford University Press.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, *55*, 17–29.

Sroufe, L. A., & Ward, M. J. (1980). Seductive behavior of mothers of toddlers: Occurrence, correlates and family origins. *Child Development*, *51*, 1222–1229.

Steinglass, P. (1987). *The alcoholic family: Drinking problems in a family context*. New York, NY: Taylor & Francis.

Sturge-Apple, M. L., Cicchetti, D., Davies, P. T., & Suor, J. H. (2012). Differential susceptibility in spillover between interparental conflict and maternal parenting practices: Evidence for OXTR and 5-HTT genes. *Journal of Family Psychology*, *26*, 431–442.

Tabares, A. A., Driver, J. L., & Gottman, J. M. (2004). Repair attempts observational coding system: Measuring de-escalation of negative affect during marital conflict. In P. K Kerig & D. H. Baucom (Eds.), *Couple observational coding systems* (pp. 227–242). Mahwah, NJ: Erlbaum.

Tremblay, R. E., McCord, J., Boileau, H., & Charlebois, P. (1991). Can disruptive boys be helped to become competent? *Psychiatry: Interpersonal and Biological Processes*, *54*(2), 148–161.

Ulbricht, J. A., Ganiban, J. M., Button, T. M. M., Feinberg, M., Reiss, D., & Neiderhiser, J. M. (2013). Marital adjustment as a moderator for genetic and environmental influences on parenting. *Journal of Family Psychology*, *27*, 42–52.

van der Zwaluw, C. S., & Engels, R. C. (2009). Gene–environment interactions and alcohol use and dependence: current status and future challenges. *Addiction*, *104*(6), 907–914.

van der Zwaluw, C. S., Scholte, R. H. J., Vermulst, A. A., Buitelaar, J., Verkes, R. J., & Engels, R. C. M. E. (2009). The crown of love: Intimate relations and alcohol use in adolescence. *European Child & Adolescent Psychiatry*, *18*(7), 407–417.

Von Bertalanffy, L. (1973). *General system theory: Foundations, development, applications*. New York, NY: George Braziller, Inc.

Wagner, B. M., & Reiss, D. (1995). Family systems and developmental psychopathology: Courtship, marriage, or divorce? In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. I: Theory and methods* (pp. 696–730). New York, NY: Wiley.

Walsh, F. (2003). Family resilience: A framework for clinical practice. *Family Process*, *42*(1), 1–18.

Walsh, F. (2006). *Strengthening family resilience:* New York, NY: Guilford Press.

Walsh, F. (Ed.). (2011). *Normal family processes* (4th ed.). New York, NY: Guilford.

Walters, M., Carter, B., Papp, P., & Silverstein, O. (1988). *The invisible web: Gender patterns in family relationships*. New York, NY: Guilford.

Wamboldt, M., Cordaro, A., & Clarke, D. (2015). Parent–child relational problem: Field trial results, changes in DSM-5, and proposed changes for ICD-11. *Family Process*, *54*(1), 33–47.

Wamboldt, M., Kaslow, N., & Reiss, D. (2015). Description of relational processes: Recent changes in DSM-5 and proposals for ICD-11. *Family Process*, *54*(1), 6–16.

Wamboldt, M. Z., & Wamboldt, F. S. (2000). Role of the family in the onset and outcome of childhood disorders: Selected research findings. *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*(10), 1212–1219.

Watzlawick, P., Weakland, J. H., & Fisch, R. (1974). *Change: Principles of problem formation and problem resolution*. New York, NY: W.W. Norton.

Weiss, R. L., & Heyman, R. E. (2004). Couples observational research: An impertinent, critical overview. In P. K Kerig & D. H. Baucom (Eds.), *Couple observational coding systems* (pp. 11–26). Mahwah, NJ: Erlbaum.

Werner, E. E., & Smith, R. S. (2001). *Journeys from childhood to midlife: Risk, resilience, and recovery*. New York, NY: Cornell University Press.

Wolraich, M. L., Felice, M. E., & Drotar, D. (1997). *The classification of child and adolescent mental diagnoses in primary care: Diagnostic and statistical manual for primary care (DSM-PC) child and adolescent version*. Elkgrove Village, IL: American Academy of Pediatrics.

World Health Organization. (2007). *Multiaxial classification of child and adolescent psychiatric disorders: The ICD-10 classification of mental and behavioural disorders in children and adolescents*. Cambridge, UK: World Health Organization.

Wynne, L. C. (1968). Methodologic and conceptual issues in the study of schizophrenics and their families. *Journal of Psychiatric Research*, *6*, 185–199.

Wynne, L. C. (1984/2004). The epigenesis of relational systems: A model for understanding family development. *Family Process*, *23*(3), 297–318.

Wynne, L. C., Ryckoff, I. M., Day, J., & Hirsch, S. I. (1958). Pseudo-mutuality in the family relations of schizophrenics. *Psychiatry: Journal for the Study of Interpersonal Processes*, *21*, 205–220.

Yingling, L. C., Miller, W. E., McDonald, A. L., & Galewaler, S. T. (1998). *The GARF assessment sourcebook*. New York, NY: Brunner/Mazel.

Zero to Three. (2005). The DC:0-3R multi-axial system. Retrieved from http://www.zerotothree.org/child-development/early-childhood-mental-health/dc-0-3r-multi-axial-system.html

CHAPTER 14

Adolescent/Young Adult Romantic Relationships and Psychopathology

JOANNE DAVILA, DEBORAH M. CAPALDI, and ANNETTE M. LA GRECA

INTRODUCTION 631
ADOLESCENT/YOUNG ADULT ROMANTIC RELATIONSHIPS 631
INTERNALIZING PROBLEMS AND DISORDERS 633
Depression and Depressive Symptoms 633
Anxiety Symptoms and Disorders 635
EXTERNALIZING PROBLEMS AND DISORDERS 637
The Development of Externalizing Behaviors 638
The Oregon Youth Study and the OYS-Couples Study 638
Mixed-Sex Friendship Groups 639
Romantic Involvement 639
Intimate Partner Violence 640
Behavior and Experiences in Relationships 641
EATING DISORDERS 642
ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND OTHER DISORDERS 643
COMORBIDITY 644
ADOLESCENT PARENTHOOD AND ITS ASSOCIATION WITH PSYCHOPATHOLOGY 645
SAME-SEX RELATIONSHIPS 646
THE PEER CONTEXT 647
Number and Quality of Friendships 647
Peer Selection and Socialization Processes in Romantic Relationships 648
Involvement with Antisocial Peers/Peer Victimization 649
Online Communication with Peers and Partners 649
THE FAMILY CONTEXT: INTERGENERATIONAL TRANSMISSION AND IMPLICATIONS FOR LONG-TERM OUTCOMES 650
CONCLUSIONS AND FUTURE DIRECTIONS 651
Issues in Theory Development 651
Methodological Issues 652
Translational Implications 653
REFERENCES 655

INTRODUCTION

The initiation of romantic involvement in adolescence is an emotionally charged event that can bring with it both pleasure and pain. Navigating the world of romance can be challenging for youth, and success at this critical developmental task has important implications for psychological well-being both in the moment and in the future. In addition, adolescents' psychological well-being has implications for how well they navigate their romantic lives. This chapter reviews the literature on the associations between psychopathology and youth romantic relationships and experiences.

To set the stage for understanding these associations, we first focus on developmental aspects of dating and romantic relationships during the period from early to late adolescence (young adulthood), giving attention to some of the positive and negative features of dating and romantic relationships. The remainder of the chapter focuses on associations with psychopathology, particularly internalizing and externalizing disorders on which the bulk of the research has been conducted, as well as key peer and family contexts in which psychopathology and skills for romantic relationships are developing. The chapter closes with discussions of methodological issues and implications for prevention and intervention.

ADOLESCENT/YOUNG ADULT ROMANTIC RELATIONSHIPS

Romantic relationships in adolescence/young adulthood are a normative aspect of development. The prevalence of romantic involvement in the United States rises across adolescence, with 36% of 13-year-olds, 53% of 15-year-olds, and 70% of 17-year-olds having had a romantic relationship (Carver, Joyner, & Udry, 2003). These relationships are generally short-lived but vary in length and get longer over time, with median durations being 5 months for ages 14 years and under, 8 months for ages 15 to 16 years,

and 21 months for ages 16 years and over (Carver et al., 2003). Both prevalence and length of involvement continue to grow into early adulthood, and over 70% of men and women in the United States marry for the first time between ages 25 to 40 years (Goodwin, McGill, & Chandra, 2009).

The rates of engagement in sexual intercourse among never-married U.S. adolescents show a similar developmental progression, although at a lower prevalence. Approximately 13% of girls and 15% of boys have engaged in sexual intercourse before age 15 years; by ages 15 to 17 years, approximately 30% of girls and 31% of boys have done so; and by ages 18 to 19 years the rates of sexual intercourse reach 69% of females and 64% of males (Abma, Martinez, Mosher, & Dawson, 2004). Not surprisingly for most adolescents, dating activities and romantic relationships provide the context for engaging in sexual activities (Kuttler & La Greca, 2004), although the rates of engaging in sexual activities outside a committed relationship, including hooking up, are high. Among sexually active adolescents, 60 to 70% have engaged in a hookup (Grello, Welsh, Harper, & Dickson, 2003; Manning, Giordano, & Longmore, 2006). Clearly, engaging in romantic relationships and sexual intercourse is normative, and typically these activities occur first in adolescence.

Initial research on heterosexual adolescent romantic development indicated that it progressed in a series of relatively predictable stages (e.g., Collins, Welsh, & Furman, 2009; Connolly & Goldberg, 1999; Furman & Wehner, 1994). In early adolescence, teens primarily engage in mixed-gender group interactions or group dating, which is characterized by affiliation and companionship rather than intimacy (Shulman & Scharf, 2000). As adolescence progresses, teens are more likely to engage in dyadic dating that involves higher levels of intimacy and closeness, deeper mutual feelings, and more extensive sexual activity (Connolly & Goldberg, 1999; Shulman & Scharf, 2000). By late adolescence, teens' romantic relationships also include caretaking and caregiving functions and begin to resemble what we typically think of as adult dyadic relationships (Furman & Wehner, 1994). Over time, youth spend more time with romantic partners and, by late adolescence, report greater closeness with their romantic partners than with their best friends or their parents (Kuttler & La Greca, 2004; Laursen, 1996). Of course, the specific timing of all of these behaviors is related to hormonal, social, and cultural conditions (Collins et al., 2009; Connolly, Craig, Goldberg, & Pepler, 2004; Laursen & Williams, 1997; Richards, Crowe, Larson, & Swarr, 1998), and given recent sociocultural shifts in adolescent romantic and sexual activity (e.g., Blais, Craig, Pepler, & Connolly, 2008), further examination of normative development will be warranted.

Adolescent romantic relationships are adaptive in numerous ways that may benefit development and emotional functioning. It is generally believed that romantic and sexual experiences in adolescence serve important functions with regard to identity development and the capacity for intimacy (see Collins, 2003; Furman & Shaffer, 2003). Indeed, the data support these ideas. Being in a romantic relationship of high quality is associated with positive affect, positive self-worth, and a sense of social competence (Connolly & Konarski, 1994; Harter, 1999; Larson & Richards, 1998; Masten, Coatsworth, Neemann, & Gest, 1995; Zimmer-Gembeck, Siebenbruner, & Collin, 2001, 2004). In addition, lighter sexual behaviors (e.g., kissing, hugging, holding hands) are associated with positive family relationships, romantic relationship satisfaction, and commitment (e.g., Welsh, Haugen, Widman, Darling, & Grello, 2005; Williams, Connolly, & Cribbie, 2008).

Adolescent romantic relationships also provide learning experiences that can set the stage for romantic functioning in adulthood. For example, less romantic competence among early adolescent girls is associated with girls making stronger predictions that they will be unlikely to marry and also with greater engagement in potentially risky sexual activity (Davila, Steinberg, et al., 2009). Higher quality romantic relationships in middle adolescence are associated with positive relationships and commitment in early adult relationships (Seiffge-Krenke & Lang 2002) as well as with more adaptive relationship processes (e.g., better conflict resolution and caregiving) in young adulthood (Madsen & Collins, 2011). Overall, adolescent romantic relationships can provide support, enhance self-esteem, and serve important socialization functions that prepare adolescents for adult relationships, and also may have positive long-term effects (Collins, 2003; Collins et al., 2009; Connolly & Goldberg, 1999).

Adolescent romantic relationships can, however, be a significant challenge or stressor that contributes to psychosocial distress. Romantic relationships explain 25 to 34% of the strong emotions that high school students' experience, and about 42% of these strong emotions are negative feelings such as anxiety, anger, jealousy, and depression (Larson, Clore, & Wood, 1999). In addition, most adolescents will face specific relationship stressors, including breakups. Interactions with a romantic partner also may involve criticism, conflict, and pressure; negative interactions may be greater in adolescents' romantic relationships than in their close friendships, especially for girls (Kuttler & La Greca, 2004; La Greca & Harrison, 2005;

Laursen, 1995). Some adolescents will engage in risky sexual activity (e.g., failure to use condoms, multiple sexual partners) that results in negative outcomes (e.g., unplanned pregnancy, sexually transmitted infections), and they will face or engage in relationship aggression. Romantic experiences also can teach young people dysfunctional beliefs about relationships and maladaptive interpersonal behaviors, which they may repeat over time (Waldinger et al., 2002). Not surprisingly, then, adolescent romantic relationships and what happens in them are associated with numerous types of psychopathology.

INTERNALIZING PROBLEMS AND DISORDERS

Depression and Depressive Symptoms

Adolescent depression is common. Lifetime prevalence rates in adolescence range from 9 to 24%, with rates of subclinical depression even higher (for a review see Merikangas & Knight, 2009). Peak age of onset is at ages 13 to15 years, and as many as 40% of adolescents experience a recurrence within 2 to 5 years (for a review see Rudolph, 2009). Therefore, many adolescents will experience depression or depressive symptoms during the very time that they become engaged in romantic activities. This is particularly true for girls. Adolescent girls are twice as likely to be depressed than are boys, and this gender difference emerges in adolescence around the time of puberty and persists through adulthood (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003; Nolen-Hoeksema & Girgus, 1994). Not surprisingly, then, the literature on adolescent romantic relationships and depression has largely focused on girls. Although research in this area is still limited, a number of key findings have begun to emerge with regard to depression and relationship involvement as well as behavior and experiences in relationships.

Romantic Involvement

One of the most consistent findings has been that involvement in dating activities and romantic relationships during adolescence is associated with depressive symptoms, particularly for girls. To our knowledge, the first study to demonstrate this was Joyner and Udry (2000) in their analyses of ADD Health data—a large, nationally representative, longitudinal sample of adolescents. They found that adolescents, particularly girls, who became involved in a romantic relationship reported increased depressive symptoms over time. Davila, Steinberg, Kachadourian, Cobb, and Fincham (2004) replicated these findings in two samples. In a cross-sectional study of early adolescent girls, those who reported current or past involvement in a romantic relationship reported greater depressive symptoms than girls with no romantic involvement. In a longitudinal study of late adolescents (girls and boys), those in romantic relationships reported greater increases in depressive symptoms 6 months later than those not in relationships, even controlling for whether they experienced a relationship break up during that time. Vujeva and Furman (2011) also found a concurrent association between depressive symptoms and relationship involvement among tenth-grade girls and boys. The association extends to romantic activities as well. For example, Compian, Gowen, and Hayward (2004) and Quatman, Sampson, Robinson, and Watson (2001) found that adolescents who were steady or frequent daters reported greater depressive symptoms. Similarly, Davila, Stroud et al. (2009) found, in a sample of early adolescent girls, that engaging in more romantic activities predicted increases in depressive symptoms over one year.

The opposite direction of effect also has been demonstrated. Depressive symptoms among girls in early adolescence are associated with entry into romantic relationships 1 year later (Davila, Stroud et al., 2009), and a number of studies suggest that early onset depression is associated with early marriage (e.g., Gotlib, Lewinsohn, & Seeley, 1998; Kessler, Walters, & Forthofer, 1998). Therefore, there appears to be a bidirectional association between depressive symptoms and romantic involvement in adolescence, particularly among girls.

However, a recent study by Mendle, Ferrero, Moore, and Harden (2013) failed to replicate the association between romantic involvement and depression. Using a behavioral genetics approach, Mendle et al. analyzed sibling pairs in the ADD Health data to examine whether associations between dating and depression held when controlling for familial factors. They examined the concurrent association between relationship involvement and depressive symptoms and failed to find a significant association. Instead, they found a significant association—independent of familial factors—between sexual activity with a nonromantic partner and depressive symptoms and suggested that prior research may have found associations between romantic involvement and depressive symptoms because it did not control for sexual activity. Although that was not true of the Davila, Stroud, et al. (2009) study, the findings of the Mendle et al. study are important for a number of reasons.

First, Mendle et al. (2013) provide more evidence for the association between sexual activity and depression,

another finding that has begun to emerge. For example, both Compian et al. (2004) and Quatman et al. (2001) found that adolescents who engaged in more sexual activity reported greater depressive symptoms. Furthermore, in line with Mendle et al., Grello et al. (2003) found that engaging in sex outside of a committed relationship, in particular, was associated with depressive symptoms. One might take these findings to mean that adolescent sexual activity leads to depression—and it might in the case of casual sexual activity (Grello et al., 2003). Although no research has been done on adolescent hookups, among college students, penetrative hookups are associated with increases in depressive symptoms and psychological distress, particularly for girls (Fielder & Carey, 2010; Owen, Fincham, & Moore, 2011). Given that younger adolescents likely have fewer resources to manage potentially challenging sexual experiences, hookups in adolescence may also predict depressive symptoms. In addition, research shows that involvement with an older partner is associated with increases in depressive symptoms among adolescent girls (Haydon & Halpern, 2010; Loftus, Kelly, & Mustillo, 2011). Given that such involvement also is associated with early sexual initiation (e.g., Marín, Kirby, Hudes, Coyle, & Gómez, 2006), one might speculate that the association between involvement with an older partner and depressive symptoms may be a function of early sexual involvement.

Research also clearly supports the opposite direction of effect, such that depression or dysphoria leads to sexual activity. For example, Davila, Stroud, et al. (2009) found that, among early adolescent girls, depressive symptoms were associated with increased engagement in sexual activity (intercourse and nonintercourse) 1 year later. Similarly, Lehrer, Shrier, Gortmaker, and Buka (2006) found that greater depressive symptoms predicted engagement in sexual risk taking over time. In addition, adolescents who have intercourse with nonromantic partners have elevated levels of depressive symptoms even prior to their first sexual experience (Grello et al., 2003; Monahan & Lee, 2008; Welsh, Grello, & Harper, 2003), suggesting that dysphoria may be involved in the onset of sexual activity—an issue which we will return to later.

Mendle et al. (2013) also draw our attention to the need to consider sexual activity in models of adolescent romantic involvement and depression. As noted earlier, both romantic involvement and sexual activity are normative in adolescence and are intertwined; future research in this area must consider both when examining associations with depression. Furthermore, research needs to focus on mechanisms of their association, including questions of why depression might lead to romantic and sexual activity and what it is about such activities that can lead to depression.

Davila (2008) described a number of conceptual models that might explain why romantic activity predicts increases in depression. Although research is sparse at present, findings are in line with the notion that because adolescent romantic experiences are inherently challenging, they may put youth at risk for depression—particularly youth with poor coping, support, or personal resources that impair their ability to manage such challenges. Consistent with this, the association between romantic activities and depressive symptoms among early adolescent girls has been found to be most strong among those with emotionally unavailable parents (Steinberg & Davila, 2008), those who engage in co-rumination (a maladaptive emotion regulation strategy; Starr & Davila, 2009), and those with a preoccupied style of relating (i.e., who are needy and fearful of rejection; Davila et al., 2004).

Studies are even sparser regarding effects of depression on romantic and sexual activity. Researchers have posited that depressed or dysphoric youth may seek out romantic experiences to meet dependence/support needs, to compensate for poor family or peer relations, or as a search for mood-regulating experiences (Davila, Stroud, et al., 2009; see also Gotlib et al., 1998). In line with these ideas, Davila, Stroud, et al. (2009)—in their sample of early adolescent girls described earlier—found that depressive symptoms predicted increased engagement in sexual intercourse most strongly in two instances. The first instance was among girls experiencing high levels of stress in the parent–adolescent relationship (as reported by the parent and objectively coded by the interviewer). Hence, dysphoric adolescent girls who have a more stressful relationship with their parents may turn to other relationships to meet their needs. Alternatively, dysphoric adolescents with stressful relationships with parents may be rebelling through sexual activity, or such adolescents may not have learned from their parent how to make healthy decisions about sexual activity. The second instance was among girls reporting higher levels of avoidance of intimacy (Hershenberg & Davila, 2010), suggesting that dysphoric girls who are uncomfortable with being emotionally intimate may be attempting to meet their needs for support or regulate their mood through physical rather than emotional intimacy. Alternatively, it may be that dysphoric girls are reticent to communicate and, hence, agree or acquiesce to sexual intercourse. The specific function of these behaviors is an important topic for future research.

Behavior and Experiences in Relationships

Similar to the literature on adult romantic relationships and depression, the research on adolescents, although still

quite limited, points to a number of maladaptive relational processes. In general, depressive symptoms are associated with poorer romantic competence among adolescent girls as assessed via an objectively coded interview conducted with the girls (Stroud & Davila, 2008). In line with this, depressive symptoms also are associated with romantic partners' ratings of adolescent girls as less interpersonally competent (Daley & Hammen, 2002).

A number of specific relational experiences also are associated with depressive symptoms. For example, self-silencing (suppressing self-expression) is associated with depressive symptoms among adolescents in romantic relationships (Harper & Welsh, 2007); this is particularly true for adolescents engaging in more frequent sex (Little, Welsh, Darling, & Holmes, 2011). Less intimacy is associated with greater depressive mood reactivity among adolescent girls (Williams, Connolly, & Segal, 2001). Lower emotional support and higher relationship stress are associated with depressive symptoms among adolescent girls (Daley & Hammen, 2002). Moreover, romantic relationship stress predicted increases in depressive symptoms over 6 months for a sample of tenth-grade Latina and African American girls; this was most true for girls high on interpersonal sensitivity (Rizzo, Daley, & Gunderson, 2006). Depressive symptoms in adolescence also have been shown to predict increases in stressful romantic events (Hankin, Mermelstein, & Roesch, 2007), suggesting bidirectional associations between stress and depression consistent with a romantic stress generation model of depression (Davila, Bradbury, Cohan, & Tochluk, 1997).

Depressive symptoms also are associated with maladaptive interaction patterns in adolescent romantic relationships. Galliher, Rostosky, Welsh, and Kawaguchi (1999) found that reports of inequality in the contribution of emotional resources and in decision making are associated with depressive symptoms, especially for adolescent girls. La Greca and Harrison (2005) found an association between depressive symptoms and reports of more negative interactions with romantic partners among girls and boys ages 14 to 19 years. Two recent longitudinal studies provide further evidence for these associations. In a sample of 80 Dutch couples between ages 13 and 19 years, both girls' and boys' depressive symptoms were associated with their own reports of greater negative conflict resolution styles and less positive problem-solving styles (Ha, Overbeek, Cillessen, & Engels, 2012). Additionally in a 5-year longitudinal study of late adolescent boys and girls, Vujeva and Furman (2011) found that greater depressive symptoms at the initial assessment predicted increases in reported conflict and less positive problem solving in romantic relationships over time. Such findings suggest that the effects of early depressive symptoms on romantic functioning are long-lasting.

Ha et al. (2012) also found that girls' depressive symptoms were associated with time to breakup, in that greater symptoms were associated with shorter relationships. Importantly, the breakup of a relationship is associated with first onset of depression in adolescence (Monroe, Rohde, Seeley, & Lewinsohn, 1999). Again, these findings point to the bidirectional nature of depression and romantic relationship problems in adolescence.

Anxiety Symptoms and Disorders

Anxiety disorders are the most prevalent type of mental disorders among adults and are also common among children and adolescents (Merikangas, Nakamura, & Kessler, 2009). In community studies, prevalence rates for anxiety disorders in youth have ranged from 2.2 to 9.5% (see Canino et al., 2004; Costello, Egger, & Angold, 2005; Merikangas, Avenevoli, Costello, Koretz, & Kessler, 2009), with general anxiety disorder (GAD) and social anxiety disorder (SAD) being the most common. As reviewed by Merikangas et al. (2009), across all ages, girls have greater rates of anxiety disorders (of all types) than do boys, and for both sexes the typical peak period of onset is middle adolescence for SAD and young adulthood for GAD. Moreover, comorbidity between anxiety disorders and other disorders is common; in fact, the comorbidity between anxiety and mood disorders in youth is so sufficiently high that anxiety is considered a potential developmental pathway in the etiology of mood disorders (e.g., Merikangas & Avenevoli, 2002; Merikangas et al., 2009). This is not surprising in that interpersonal processes are considered to be important to the development and maintenance of both anxiety (especially social anxiety) and depression (see Davila, La Greca, Starr, & Landoll, 2010; La Greca, Davila, & Siegel, 2008).

Social anxiety, in particular, has been associated with problems in adolescents' and young adults' romantic relationships. Social anxiety is characterized by fear of negative evaluation by others as well as social avoidance and distress in social situations (La Greca, 1999; La Greca & Lopez, 1998). Although some degree of social anxiety is normative (La Greca, 1999), when socially anxious feelings and behaviors are sufficiently extreme, persist over time, and interfere with functioning they may be consistent with the diagnosis of SAD (American Psychiatric Association, 2000, 2013).

On a related note, some research with adolescents and young adults also has focused on dating anxiety. Dating anxiety refers to worry, distress, and inhibition in heterosocial situations involving dating partners or potential

dating partners (e. g., Chorney & Morris, 2008; Glickman & La Greca, 2004). Social anxiety and dating anxiety are strongly related but appear to be distinct constructs (Glickman & La Greca, 2004). Similar to social anxiety, dating anxiety is conceptualized as being composed of fear of negative evaluation (in dating situations or with other-sex individuals) and social inhibition and distress (either with a dating partner, a potential dating partner, or during heterosocial group situations; Glickman & La Greca, 2004).

We now focus on social and dating anxiety; specifically, how they may affect and be affected by romantic relationships. Although romantic relationships have many positive aspects (e.g., provide social support, enhance self-esteem, contribute to intimacy), romantic relationships also can be stressful, especially for adolescents who are just learning how to navigate them (Downey, Bonica, & Rincon, 1999). Thus, the presence of romantic relationships (and even their *absence*) might contribute to feelings of social or dating anxiety; in turn, social and dating anxiety may influence the presence and quality of romantic relationships.

Romantic Involvement

Social anxiety can interfere with youths' close relationships (La Greca & Lai, 2014; La Greca & Landoll, 2011). Socially anxious adolescents have fewer close friendships, have lower quality friendships, and are the recipients of more peer rejection and negative peer experiences than other adolescents (Greco & Morris, 2005; La Greca & Harrison, 2005; La Greca & Lopez, 1998; Siegel, La Greca, & Harrison, 2009). In particular, inhibition among socially anxious youth may limit their frequency of social interactions, and thus their ability to form close, supportive social ties (Beidel, Turner, & Morris, 1999; La Greca & Lopez, 1998). Socially anxious youth and young adults also make more negative appraisals of social situations (Flanagan, Erath, & Bierman, 2008) and display interpersonal avoidance (Darcy, Davila, & Beck, 2005; Grant, Beck, Farrow, & Davila, 2007). The inhibition, avoidance, and negative appraisals of socially anxious adolescents and young adults present challenges for forming romantic relationships. In fact, among youth followed longitudinally, shy and socially inhibited boys are more likely to marry later in life than comparison youth (Caspi, Elder, & Bem, 1988). Large-scale epidemiological studies (Lampe, Slade, Issakidis, & Andrews, 2003; Wittchen, Fuetsch, Sonntag, Muller, & Liebowitz, 2000) also reveal that socially anxious adults are more likely to have been never married or to be separated or divorced than nonanxious adults.

Evidence supports the view that socially anxious adolescents are less likely to be involved in a dating or romantic relationship than their less-anxious peers (La Greca & Harrison, 2005), perhaps because they are uncomfortable in or avoid social situations that might lead to opportunities for dating or romantic relationships (Glickman & La Greca, 2004). For example, La Greca and Harrison (2005) found that, among high school students (mean age of 16.5 years), the *absence* of a current romantic relationship was associated with greater social anxiety; in contrast, romantic relationship involvement was unrelated to adolescents' depressive symptoms. These findings are intriguing as they suggest that nondating adolescents are more socially anxious (or potentially that romantic involvement reduces feelings of social anxiety). The findings also contrast with those reported above for depression, which indicated that romantic involvement is associated with greater depressive affect, especially for early to midadolescents. Perhaps because romantic involvement is normative among older adolescents (Carver et al., 2003), such involvement is not as likely to be an indicator of depressive mood but instead might be a source of anxiety for adolescents who are not romantically involved by their late teens. Also as noted already, associations between romantic involvement and depressive symptoms may be because of youths' involvement in sexual activity, rather than youths' involvement in a relationship per se.

Findings regarding the romantic involvement of adolescents with dating anxiety parallel those obtained for socially anxious individuals. For example, compared with their less anxious peers, adolescents who report greater dating anxiety are less likely to be dating (Glickman & La Greca, 2004) and more likely to report that they never have had a romantic relationship (La Greca & Mackey, 2007). Adolescents high on dating anxiety also report having fewer close friends of the other sex (La Greca & Mackey, 2007). In the normal course of adolescent development, such friendships provide opportunities to develop social skills specific to other-sex relationships and to meet potential romantic partners (Herbert, Fales, Nangle, Papadakis, & Grover, 2013). Thus, adolescents with social or dating anxiety may miss out on key socialization experiences that then delay or preclude the development of romantic relationships (Connolly & Goldberg, 1999; Dunphy, 1963; Herbert et al., 2013). By their avoidance or lack of participation in romantic relationships, socially or dating anxious individuals also miss out on the potential psychological benefits of romantic relationships.

Behavior and Experiences in Relationships

Once dating or romantic relationships have been established, the qualities of such relationships appear to be less

adequate or skillful among socially anxious individuals. Findings reveal that social anxiety is associated with lower levels of intimacy and companionship in adolescents' close friendships (Vernberg, Abwender, Ewell, & Beery, 1992), especially for adolescent girls (La Greca & Lopez, 1998). Similarly, young adults who are socially anxious display less emotional expression and interpersonal assertiveness in their social interactions (Davila & Beck, 2002; Melfsen, Osterlow, & Florin, 2000). Such findings suggest that inhibition in close relationships might limit positive interactions within the romantic relationships of socially anxious adolescents and young adults. Although research is scant, it does appear that socially anxious adults show less emotion, fewer positive behaviors, less self-disclosure, and decreased intimacy in their romantic relationships (Sparrevohn & Rapee, 2009; Wenzel, Graff-Dolezal, Macho, & Brendle, 2005).

Along with fewer positive behaviors, socially anxious individuals also appear to engage in more negative behaviors in their romantic relationships. Kashdan, Volkmann, Breen, and Han (2007) found that for individuals low in social anxiety, the ability to express negative emotions in an open and uninhibited way fostered a good-quality romantic relationship; however, for individuals high in social anxiety, the expression of negative emotions in the context of a romantic relationship was associated with deteriorating relationship quality. Socially anxious young adults also have been found to demonstrate more negative and fewer positive interactions with their romantic partner during an observed communication task (Wenzel et al., 2005). Interestingly, research by Beck, Davila, Farrow, and Grant (2006) found that socially anxious women demonstrated more negative behavior in the context of relationships that were viewed as more satisfying. It is possible that socially anxious women might view high-quality relationships as a secure base for expressing negative feelings (Beck et al., 2006). Overall, it appears that negative emotional expression is common in the romantic relationships of socially anxious individuals.

For the most part, this area of research does not clarify the direction of causal pathways between social anxiety and relationship qualities. So, for example, it is possible that social anxiety leads to inhibited social behavior that limits the intimacy and quality of a romantic relationship. However, it is also plausible that negative interactions in romantic relationships, which are fairly common, contribute to or exacerbate adolescents' and young adults' feelings of distress and, specifically, of social anxiety (La Greca & Mackey, 2007). Moreover, once engaged in a romantic relationship, socially anxious individuals may have difficulties with social appraisal and evaluation; these difficulties may, in turn, increase symptoms of social anxiety.

In addition to research linking social anxiety with difficulties in romantic relationships, associations between romantic relationship issues and dating anxiety have been noted. Dating anxiety is associated with romantic relationship difficulties, even when controlling for other indicators of social competence (e.g., number of close friends, qualities of close friendships). For example, among adolescents who were currently dating (and thus less likely to be socially or dating anxious to begin with), fewer positive and more negative qualities in the romantic relationship were associated with higher levels of dating anxiety (La Greca & Mackey, 2007).

The previously discussed findings suggest that youth who are anxious about dating might avoid heterosocial situations, thus making it difficult to develop a romantic relationship. It is possible—once adolescents become romantically involved—that fears and worries about dating may become less salient, thereby reducing feelings of social or dating anxiety. Consistent with this latter perspective, Nieder and Seiffge-Krenke (2001) tracked the romantic relationships of adolescents from ages 14 to 17 years. As participants progressed through adolescence, their reported stress in romantic situations decreased and their romantic relationship quality increased. Thus, increased exposure to dating relationships during adolescence may decrease anxiety in dating situations over time (Nieder & Seiffge-Krenke, 2001).

Although it has not been studied directly, greater experience with romantic relationships might lead to reductions in dating or social anxiety over time, especially if the qualities of the romantic relationships are generally positive. In contrast, the presence of a high level of negative interactions with a romantic partner (e.g., criticism, pressure, conflict) might well exacerbate feelings of social anxiety and contribute to significant distress and potentially to GAD or SAD.

EXTERNALIZING PROBLEMS AND DISORDERS

The cluster of externalizing behaviors at adolescence—particularly involving delinquency, substance use, and sexual-risk behaviors—have long been sources of anguish to parents and major foci of adolescent researchers seeking to understand the emergence and consequences of these behaviors. Conduct problems—including impulsive, defiant, and aggressive behaviors—are among the most common reason for referrals to child mental health

clinics in the western hemisphere (Frick, 1998) and have a poor prognosis for adult adjustment (Kohlberg, Ricks, & Snarey, 1984). The study of romantic relationships at adolescence generally was a separate area of research; thus, the influence of externalizing behavior on adolescent romantic relationship formation and functioning and the ways in which such relationships may influence externalizing behaviors are more recent areas of research.

In consideration of these issues, we draw heavily on two sources. First, on research conducted by Capaldi and colleagues (e.g., Capaldi, Shortt, & Kim, 2005) using the Oregon Youth Study (OYS)-Couples Study sample that examined problems in romantic relationships in late adolescence and young adulthood—notably intimate partner violence (IPV, including here both physical and psychological aggression)—and second, on a recent systematic review of risk factors for IPV (Capaldi, Knoble, Shortt, & Kim, 2012). As discussed by Capaldi, Knoble, et al. (2012), IPV includes physical, psychological, and sexual abuse by men and by women toward romantic partners of the same or opposite sex. Physical violence involves forceful physical contact ranging from low force pushes and slaps to severe beatings and lethal violence. Sexual abuse includes coercive and physical behaviors varying from trying to persuade someone to perform a sexual act against their will, ignoring "no" responses, to physically forced sex acts (Teten, Hall, & Capaldi, 2009; Tjaden & Thoennes, 2000). Psychological aggression (or emotional abuse) involves acting in an offensive or degrading manner toward a partner—including threats, ridicule, withholding affection, and restrictions (e.g., social isolation, financial control; O'Leary & Maiuro, 2001).

Minor or occasional psychological aggression is very common (Shortt et al., 2012). Psychological and physical aggression are significantly associated. Capaldi and Crosby (1997) found that at age 18 years the two types of abuse perpetration were significantly associated for young men and young women ($r = .60$ for men, $r = .55$ for women, both $p < .001$), and psychological abuse has been found to be predictive of physical abuse and to have severe impacts (O'Leary & Maiuro, 2001).

The Development of Externalizing Behaviors

Antisocial behaviors or conduct problems refer to a cluster of impulsive or under-controlled and societal rule-breaking behaviors, including delinquent and aggressive behaviors. Aggression is normative in the first few years of life and then decreases (Tremblay et al., 2004). Tremblay, Masse, Pagani-Kurtz, and Vitaro (1996) examined developmental trends in frequent versus occasional physical aggression (hitting, biting, and kicking) from ages 24 months to 12 years for boys and girls. The highest levels occurred at age 2 years and declined over time. Similarly, in a study examining externalizing behaviors from ages 3 to 9 years, both boys and girls decreased or improved across this period (Capaldi, Pears, Kerr, Owen, & Kim, 2012). Thus, there is evidence that the normative pattern is for children to improve in inhibitory control with age. Despite this improvement overall, some children remain high in externalizing behaviors—thus not showing the normative developmental improvement—and some increase in externalizing behaviors in childhood.

In contrast, delinquent behaviors peak between ages 14 and 17 years (Blumstein, Cohen, Roth, & Visher, 1986) for the key symptoms of adolescent conduct disorder—namely behaviors that would be illegal in adulthood (e. g., theft, assault) and status offenses or behaviors that are illegal due to the youth being younger than age 18 years (e.g., minor in possession of alcohol). Peak ages at police arrest for the life span are typically at ages 18 and 19 years for both boys/men and girls/women in the United States (e.g., Uniform Crime Reports, 2009). In addition, substance use beyond very light experimentation (e.g., trying a sip of alcohol) mostly emerges in adolescence, with accelerated onset and use particularly in the early high school years (Capaldi, Stoolmiller, Kim, & Yoerger, 2009; Washburn & Capaldi, 2013). Thus, the adolescent years of dating onset are also the years of high levels of externalizing behaviors (including aggression) and essentially an explosion of substance use. Although symptoms of externalizing and internalizing disorders differ considerably, there is a significant association between externalizing and internalizing (e.g., sadness, anxiety) problems at adolescence (Angold, Costello, & Erkanli, 1999). Thus, important associations between conduct problems and particular aspects of romantic relationships may also apply to youth with internalizing symptoms (and vice versa).

The Oregon Youth Study and the OYS-Couples Study

Before going further, and because much of the following review draws heavily on the OYS, we describe the design of the OYS-Couples Study. The OYS is a community-based longitudinal study of males who were considered at increased risk for delinquency due to the neighborhoods where they lived in childhood (within a medium-sized town). The sample is predominantly White and lower or working class. The boys were followed from childhood (Grade 4, ages 9 to 10 years), and the study had very strong recruitment and retention rates (Capaldi, Chamberlain, Fetrow, & Wilson, 1997). The sample proved to be at

risk for delinquency, despite the fact that most were not showing high levels of conduct problems in childhood. In fact, in the period prior to age 18 years, about one half of the boys were arrested one or more times, with 20% having five or more arrests. Only 49% graduated from high school with their class.

In late adolescence (ages 17–18 years), the young men were invited to participate in an ancillary study with a romantic partner; a further assessment was conducted at about age 21 years. The assessments with the partner included interviews for the young men and women (few participated with male partners) and an observed discussion of issues in their relationships. The OYS-Couples Study continued with data collection waves about every other year through the 20s, and arrest records for IPV were later collected. In late adolescence, at an average age of around 18 years, 31% of the young men with partners and 36% of their female partners had engaged in physical aggression toward a partner in the prior year (Capaldi & Crosby, 1997), a typical prevalence of physical aggression for couples of this age (Langhinrichsen-Rohling, Misra, Selwyn, & Rohling, 2012). Observed aggression was found to be significantly associated with reported aggression, and the correlation between physical and psychological aggression toward a partner in late adolescence was .60 for the young men and .55 for the young women ($p < .001$).

Mixed-Sex Friendship Groups

As noted earlier, other-sex friendships increase in frequency in early adolescence (Feiring, 1999; Poulin & Pedersen, 2007), setting the stage for mixed-sex friendship groups from which dating and romantic relationships often emerge. Whereas it might be expected that adolescents who enter mixed-gender friendship groups (and date) relatively early might be more mature; in fact, there is evidence of higher levels of conduct-problem behaviors among adolescents who transition from same-gender to mixed-gender groups earlier than their peers. For example, Poulin and Pedersen (2007) found that girls (but not boys) who were rated as more antisocial than their peers by their sixth-grade teachers had more male friends in Grade 7. Further, higher growth in mixed-sex friendships from Grades 6 to 10 was related to earlier onset of puberty for the girls. Stattin and Magnusson (1990) found associations between other-sex friendships and delinquency that were associated with early maturing girls developing friendships with or dating older more deviant boys. However, a large longitudinal study of sixth to ninth graders, Molly, Gest, Feinberg, & Osgood, (2014) found consistent evidence that mixed-sex groups were composed of the most at-risk youth in the sample, with such groups showing relatively high levels of substance use and delinquency. Thus, both boys and girls in mixed-sex groups may be more at risk, particularly during early adolescence. Among older adolescents, other-sex friendships tend to develop among high status and more socially skilled youth (see Herbert et al., 2013; La Greca & Harrison, 2005).

One study found interesting evidence that adolescent romantic partners may be network bridges, connecting daters to new peer contexts (Kreager & Haynie, 2011). Using couples (average ages 14 to 15 years) from the AddHealth project, Kreager and Haynie found unique contributions of partners' drinking, friends' drinking, and friends-of-partners' drinking to daters' own later drinking frequency and heavy episodic drinking. Thus, other-sex network influences may be intimately involved in the accelerated growth in alcohol use, which occurs in the high school years.

Romantic Involvement

Consistent with findings regarding mixed-sex networks, there is evidence that youth with higher levels of conduct problems begin dating earlier than their peers. Capaldi and Crosby (1997) found for the OYS-Couples Study at ages 17 to 18 years that the boys who participated with a partner showed higher levels of problems than those who did not participate (usually because they did not have a partner). The boys who participated were more likely to have been arrested and had more than twice as many arrests on average than those who did not. They also had twice the prevalence of marijuana use and were much more likely to have already fathered a child. Similarly, Connolly, Pepler, Craig, and Taradash (2000) found that young adolescents (Grades 5 to 8) who bullied their peers started dating earlier and engaged in more advanced dyadic dating than did their peers. Bullies were found to be strongly relationship oriented, yet their views of their boy/girlfriends were less positive than those of other adolescents. The bullies were more developed pubertally, but this did not fully account for the dating precocity. Finally, they were more likely to report dating violence.

The association of conduct problems with early dating may be part of a pattern whereby conduct-problem youth follow a more accelerated pathway to adult behaviors (Capaldi & Shortt, 2003; Newcomb, 1987). High school dropout is a large factor in such acceleration, as it is strongly associated with conduct problems and other early risk factors (Eliott & Voss, 1974) and because adolescents are then likely to leave the family-of-origin home prior to age 18 years (Capaldi & Stoolmiller, 1999). Also, conduct

problems are associated with an early age at initiation of sexual intercourse (Capaldi, Crosby, & Stoolmiller, 1996).

Intimate Partner Violence

In a recent review of risk factors for IPV, Capaldi, Knoble, et al. (2012) examined risk from deviant peer association, antisocial behavior, and alcohol and other drug use.

Conduct Problems/Antisocial Behavior, Anger, and Hostility

Surprisingly, given that aggression and violence are part of the definition of conduct problems, they have been a focus of study as developmental risk factors for IPV only in the past 10 to 15 years. In fact, a review by Schumacher, Feldbau-Kohn, Slep, and Heyman (2001) of risk factors for male perpetration of IPV did not mention antisocial behavior as a risk factor. This is partly because of the history of romantic relationships and externalizing problems being separate fields of research. Fortunately, there have now been a relatively large number of prospective longitudinal studies examining these issues.

As discussed by Capaldi, Knoble, et al. (2012), one study (Magdol, Moffitt, Caspi, & Silva; 1998) found that early problem behaviors were the most consistent predictor of IPV of a number of early risk factors, and others have found similar strong associations with different samples (Ehrensaft et al., 2003; Lussier, Farrington, & Moffitt, 2009). There is evidence that earlier onset of conduct problems (i.e., in childhood versus adolescence) is predictive of a more severe course of antisocial behavior (Patterson, Capaldi, & Bank, 1991). Consistent with this, Woodward, Fergusson, and Horwood (2002) found that individuals with younger onset antisocial behavior were more likely than those with later or no onset of antisocial behavior to be perpetrators or victims of IPV, and there was no gender difference in IPV perpetration. Also for the Dunedin Study, Moffitt, Krueger, Caspi, and Fagan (2000) found that IPV and general crime shared risk from negative emotionality (e.g., anxiety, anger, hostility). In a third Dunedin Study related to IPV, Ehrensaft, Moffitt, and Caspi (2004) found that women in relationships resulting in injury or police contact had childhood family adversity, adolescent conduct problems, and aggressive personality, and that men had disinhibitory psychopathology since childhood and pervasive personality deviance. Capaldi and Clark (1998) found for the OYS-Couples Study that boys' antisocial behavior in midadolescence was significantly associated with their IPV perpetration in late adolescence (at age 18 years); for the same sample, Capaldi, Dishion, Stoolmiller, and Yoerger (2001) found that antisocial behavior at ages 17 to 18 years was predictive of IPV perpetration at ages 20 to 23 years. In a study including young men and women, Andrews, Foster, Capaldi, and Hops (2000) found, after controlling for a number of risk factors and their interactions, that family-aversive communication and participant antisocial behavior predicted IPV in couples. Herrenkohl, Kosterman, Mason, and Hawkins (2007) found for young men and women that the odds of moderately severe IPV perpetration were double for chronic youth offenders compared with nonoffenders.

Findings from these and other predominantly fully prospective longitudinal studies indicate that conduct problems and associated hostility are a robust and substantial risk factor for later IPV perpetration and victimization, and that it is a developmental risk factor for both perpetration and victimization for both girls/young women and boys/young men. This is consistent with the finding that much IPV both in adolescence and young adulthood is bidirectional (i.e., that both partners are engaging in physical aggression), estimated to be about 50 to 60% of couples where there is any IPV present (Langhinrichsen-Rohling et al., 2012). Also, Capaldi, Knoble, et al. (2012) concluded from their review that antisocial behavior is frequently found to be a mediator for earlier risk factors such as harsh parental treatment.

An important issue is the extent to which antisocial behavior and IPV relate to risk for injuries in young couples. Capaldi and Owen (2001) examined levels of antisocial behavior for young men and women, aged 21 years, who showed frequent physical aggression toward a partner—at about the frequency reported by shelter samples. They found that both men and women showed elevated antisocial behavior levels if they perpetrated such frequent physical aggression toward partners, and the highest levels for both men and women were found for couples where the physical aggression was both bidirectional and frequent. Higher levels of injuries were reported when the men and women were frequently physically aggressive.

Alcohol and Drug Use

Alcohol and drug use are closely associated with antisocial behavior (Donovan, Jessor, & Costa, 1988) and are a key aspect of externalizing behaviors in adolescence and adulthood. Whereas alcohol use is thought to be a proximal predictor of IPV because of its hypothesized disinhibitory effect on aggression (Flanzer, 2005), drug use has been less frequently examined as a predictor until more recently. In fact, there are relatively few prospective longitudinal studies that have examined prediction to IPV from either alcohol or drug use for adolescents and young adults. A problem with studies in this area is that when this association is

examined, antisocial behavior—which would be expected to account for an association of substance use and IPV—is often not controlled in the analyses.

As discussed by Capaldi, Knoble, et al. (2012), Reyes, Foshee, Bauer, and Ennett (2011) found for a rural school-based sample that heavy alcohol use was significantly associated with the overall cohort trajectory of physical dating aggression perpetration with no evidence of sex differences. Whereas adolescents with heavy alcohol use initially reported higher levels of dating aggression perpetration during early and middle adolescence, by late adolescence there were no differences in perpetration levels between heavy alcohol users and nonusers. In within-person analyses, a negative interaction between heavy alcohol use and grade was found—indicating that the effect of heavy alcohol use on dating violence perpetration diminished as grade level increased, possibly suggesting that as early heavy use is associated with antisocial behavior that the effect might be explained by antisocial behavior (i.e., later onset heavy alcohol users showed less antisocial behavior). In a study of victimization, Buzy et al. (2004) found that higher levels of alcohol use predicted physical and sexual victimization months later. Counter to common wisdom, for a predominantly African American and Hispanic sample, Schnurr and Lohman (2008) found that drug and alcohol use were predictive of perpetration of dating violence for girls but not for boys. In a study of dating violence victimization, Maas, Fleming, Herrenkohl, and Catalano (2010) found that externalizing behavioral problems predicted IPV victimization for boys, whereas internalizing, externalizing, and alcohol consumption predicted IPV victimization for girls.

The association of drug use with IPV has more frequently been examined in cross-sectional than in longitudinal work. In a study of IPV victimization for a high school sample, Temple and Freeman (2011) found that, after controlling for demographic variables and alcohol use, lifetime use of any controlled substance significantly increased the likelihood of dating violence victimization. A second study of victimization using a national sample of ninth to 12th graders (Eaton, Davis, Barrios, Brener, & Noonan, 2007) found that dating violence victimization was associated with alcohol use and marijuana use for girls but not for boys. For a school-based sample of students in Grades 11 and 12, O'Keefe (1997) found that alcohol and drug use were associated with dating violence perpetration for both boys and girls.

Although there is evidence for an association of indicators of alcohol use with IPV perpetration and victimization, it is not as strong or as consistent as has generally been supposed, likely partly because of alcohol and IPV sharing a common risk factor of antisocial behavior. Furthermore, the association may be stronger for girls and young women than for boys and young men. There are fewer studies on the use of drugs and IPV, but some suggest that there could be a stronger association between such use and IPV. Findings in the area of substance use and IPV therefore suggest that some prevailing assumptions regarding both causal and gender associations may need modification and further study to understand how to tackle prevention in this important area of health risk.

One important question regarding dating violence or IPV in very young couples is whether it is predictive of IPV in the future or whether IPV shows stability over time. This question has rarely been examined; however, Shortt et al. (2012) found that IPV in young adulthood (at average age 21 years) predicted IPV into the late 20s, and this was true for both physical and psychological aggression. Thus, IPV in young adulthood is predictive of longer-term problems with aggression toward a partner. Capaldi, Kim, and Shortt (2007) examined observed (during a problem-solving discussion) initiation and reciprocity of physical aggression. They found that the likelihood of injury over about an 8-year period was about twice as high for those couples where both young man and woman were observed to use some physical aggression, compared with couples for whom either no physical aggression or aggression by only one partner was observed.

On a positive note, and contrary to early reports that IPV increases over time (e.g., Walker, 1984), there is evidence that IPV peaks in young adulthood and then declines with age (Fritz & O'Leary, 2004; Kim, Laurent, Capaldi, & Feingold, 2008). In this respect, it shows a similar pattern to crime more generally, which peaks at young ages—with peak ages at arrest for males and females in the United States being ages 18 to 19 years (Uniform Crime Reports, 2009)—and then shows a sharp decline.

Behavior and Experiences in Relationships

Whereas there is now a substantial body of work on the association of conduct problems and IPV, much less work has been conducted regarding the association of conduct problems with other aspects of romantic relationship functioning at adolescence, such as relationship satisfaction and relationship stability or longevity. It is well established in the adult couples' literature that couples' conflict is related to aggression or antisocial behavior and that crime and antisocial behavior predicts divorce or relationship breakdown (Farrington, 2003). Whereas couples' conflict may not necessarily involve psychological aggression, it is rare to have substantial conflicts that avoid such behaviors.

In fact, some degree of psychological aggression is ubiquitous in young couples, with 80% of the young men and 86% of the young women in the OYS-Couples Study either reporting or being observed to engage in such behaviors (Capaldi & Crosby, 1997).

Shortt, Capaldi, Kim, and Owen (2006) examined prediction to relationship separation over the course of 6 years from dyadic physical aggression assessed at age 21 years for the OYS-Couples Study. Over this period, 62% of couples separated. Physical aggression significantly increased the likelihood of relationship dissolution—controlling for psychological aggression, prior relationship satisfaction, and relationship contextual factors including length of relationship, relationship type, and children in the household. In the models, including all these factors, psychological aggression was not a significant predictor of relationship breakdown. Both physical and psychological aggression were associated with low relationship satisfaction. Thus, aggression toward a partner, associated with externalizing behaviors, is predictive of additional problems in the relationship including greater likelihood of relationship breakdown.

In another study with the OYS-Couples Study and using the Specific Affect Coding System (SPAFF; Gottman, McCoy, Coan, & Collier, 1996), Shortt, Capaldi, Kim, and Laurent (2010) examined the association between three categories of observed affect—including positive affect, externalizing negative affect (criticism, domineering, contempt, belligerence, threats, defensiveness, anger), and internalizing negative affect (sadness, fear/tension, stonewalling, or refusing to respond)—and both IPV and relationship satisfaction from ages 21 to 25–26 years. Externalizing negative affect showed a robust negative association with relationship satisfaction for both the young men and young women, being associated at each of the three study time points. Internalizing negative affect showed some significant associations with relationship satisfaction but not as consistently as for externalizing negative affect.

Problems in the family of origin that are related to externalizing problems are related to conflictual and poor quality of relationships in young adults. Andrews et al. (2000) examined the association of antisocial behavior and family functioning at ages 16 to 17 years, and romantic relationship functioning 6 years later at around age 23 years. They found that low relationship satisfaction, higher observed aversive communication, and higher physical aggression were predicted by observed family aversive communication in adolescence, and adolescent antisocial behavior was associated with lower relationship satisfaction and higher physical aggression. Parent–adolescent conflict predicted lower levels of observed couple facilitative communication. Thus, there is evidence that conduct problems are associated with pervasive relationship problems over time, and prevention of such problems could have a strong beneficial impact on romantic relationship functioning.

EATING DISORDERS

Eating disorders are prominent in adolescence, particularly among girls (Hudson, Hiripi, Pope, & Kessler, 2007; Lewinsohn et al., 1993). However, there are relatively few studies of eating pathology and romantic functioning among this age group. These relatively rare studies point to a few key findings. First, romantic and sexual involvement may attune youth more to their physical appearance and lead to insecurities about their bodies. Cauffman and Steinberg (1996) found, among adolescents, an association between high levels of involvement in heterosexual social and sexual activities and reports of greater disordered eating and dieting (also see Compian et al., 2004). Moreover, the belief that thinness is important for dating success is associated with dietary restraint, body dissatisfaction, and bulimic symptoms among adolescents (Paxton et al., 1991). Longitudinal research demonstrates that adolescent girls and female college students become less satisfied with their appearance after engaging in sexual intercourse for the first time, although the opposite is true for males (Valle, Røysamb, Sundby, & Klepp, 2009; Vasilenko, Ram, & Lefkowitz, 2011). Therefore, interest and experience in romantic and sexual activities may increase body dissatisfaction—leading to subsequent eating pathology, at least for women.

Second, there also is evidence that eating pathology affects romantic and sexual involvement; in some cases, it might inhibit involvement. Compared with non–eating disorder youth, youth (ages 14 to 21 years) with anorexia had fewer dating experiences and more negative attitudes about sexuality (Ruuska, Kaltiala-Heino, Koivisto, & Rantanen, 2003). However, in other studies, eating pathology and body image issues are associated with greater or more risky sexual involvement. Among girls, a number of studies show that poorer body image (e.g., negative body evaluation, lower body satisfaction, greater body objectification) predicts earlier onset of sexual intercourse (Kvalem, von Soest, Træen, & Singsaas, 2011; Pearson, Kholodkov, Henson, & Impett, 2012) and less condom use (Schooler, 2013). In addition, compared with girls (ages 13 to 25 years) without eating disorders, those with eating disorders are less knowledgeable about sexual-health risks and the prevention of HIV and pregnancy (Hicks et al., 2013).

It appears that male youth with eating pathology may be similarly at risk. Ackard, Fedio, Neumark-Sztainer, and Britt (2008) found that, compared with males without disordered eating, those with such disorders reported a greater number of sex partners. Conversely, among adolescent boys, body satisfaction is associated with sexual agency and clarity in sexual values (Schooler, Impett, Hirschman, & Bonem, 2008).

Among adolescents, it is unclear how eating and body image issues play out within relationships. One study found that perceived pressure from dating partners to be thin was not associated with increases in body dissatisfaction among adolescent girls and boys (Presnell, Bearman, & Stice, 2004). However, there is a somewhat larger literature on young adults, typically college students, indicating that eating pathology/body image issues and relationship functioning affect one another. Bidirectional associations exist between relationship functioning and young women's eating, weight, and shape concerns (Morrison, Doss, & Perez, 2009). Goins, Markey, and Gillen (2012) found that young men were more likely to be satisfied with their body when their partners were and when they experienced high levels of sexual intimacy in the relationship. Greater body dissatisfaction is associated with lower perceived trust among young women and lower perceived support among young men in romantic relationships (Juarez & Pritchard, 2012). Sanchez and Kwang (2007) found that the more young women's self-worth is dependent on a romantic relationship, the more they report body shame and bulimic symptoms. In addition, factors such as anxious romantic attachment and low perceived romantic support are associated with body image disturbance (Cash, Theriault, & Annis, 2004; Weller & Dziegielewski, 2004). Therefore, although one study failed to find associations between females' body satisfaction and reports of relationship quality (Markey & Markey, 2006), it does appear that varied aspects of young adult relationships, for both young men and women, are related to their body image and eating behavior.

One aspect of relationship functioning that has been consistently related to eating and body image pathology in adolescents and young adults is aggression in relationships. Youth who experience dating violence, date rape, unwanted sexual contact in dating situations, physical aggression, or psychological aggression report greater eating pathology (e.g., Ackard & Neumark-Sztainer, 2002; Skomorovsky, Matheson, & Anisman, 2006; Thompson, Wonderlich, Crosby, & Mitchell, 2001).

It should be noted that, as the previous review shows, although some of the literature has focused on actual eating disorders or symptoms, much of the research has focused on body image issues. Whether these studies capture associations and processes that also pertain to diagnosable eating disorders is unknown.

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND OTHER DISORDERS

There is very little research on associations between romantic relationships and other disorders in adolescence. The few available studies have focused on different aspects of romantic relationships.

With respect to youth or adults with attention-deficit/hyperactivity disorder (ADHD), one study found that girls diagnosed with ADHD in childhood were involved in fewer romantic relationships in late adolescence compared with their non-ADHD peers (Babinski et al., 2011). In contrast, however, another study (Siegel & La Greca, 2009) found that, among adolescents in a community sample, those with higher levels of ADHD symptoms were more likely to be romantically involved than those with lower levels of such symptoms. Barkley, Fischer, Smallish, and Fletcher (2006) similarly found that young adults with ADHD had begun dating earlier than those without ADHD and were younger during their first sexual experience. It is possible that the impulsivity that accompanies ADHD, or associated conduct problems, leads to earlier romantic experiences for such adolescents. Furthermore in the Siegel and La Greca (2009) study, although adolescents with symptoms of ADHD had more romantic partners, these relationships were characterized by higher rates of negative interactions. This is troubling, as negative experiences in romantic relationships during adolescence predict poor intimate relationship quality in adulthood (Connolly, Furman, & Konarski, 2000). Indeed, a recent study of young adult men (ages 18 to 25 years) found that those with childhood ADHD, especially those with conduct problems, were more likely to be verbally aggressive and violent with their romantic partners than were young men without histories of ADHD or conduct problems (Wymbs et al., 2012). Others have found that the marital functioning of adults with ADHD is significantly poorer than that for matched non-ADHD controls (Eakin et al., 2004). Thus, emerging evidence suggests that adolescents and young adults with ADHD, and especially those with concurrent conduct problems, represent an at-risk population for romantic relationship difficulties.

Other studies have focused on personality pathology, finding that such pathology was associated with romantic dysfunction over 4 years among late-adolescent women (Daley, Burge, & Hammen, 2000). In addition,

Kim, Miklowitz, Biuckians, and Mullen (2007) found that, among adolescents diagnosed with bipolar disorder, greater chronic stress in intimate relationships was associated with less improvement in mood symptoms over the following year. However, the measure of stress in intimate relationships was a composite of romantic and family relationships, so whether this association is specific to romantic relationships is unclear. Given the scarcity of research on other disorders, it is hard to draw meaningful conclusions.

COMORBIDITY

Rates of comorbidity of psychopathology (or of co-occurrence at the symptom level) are high in adolescence and young adulthood (Kessler, Chiu, Demler, Merikangas, Walters, 2005; Lewinsohn et al., 1993; Merikangas et al., 2010). Yet, up to this point, we reviewed associations between psychopathology and youths' romantic relationships by key disorders, with limited attention to the issue of comorbidity. The neglect of comorbidity primarily reflects the way that research on developmental psychopathology has evolved, but this neglect also underscores the need for more complex models that incorporate issues of comorbidity.

To emphasize the importance of the issue, consider recent findings from the National Comorbidity-Adolescent Supplement (NCS-A). Among adolescents (ages 13 to 18 years), data from the NCS-A (Merikangas et al., 2010) revealed that the most common disorders were anxiety disorders (31.9%), behavior disorders (19. 1%, including 8.7% for ADHD and 6.8% for CD), mood disorders (14.3%), and substance-use disorders (11.4%). Strikingly, almost 40% of those with one class of disorder also met criteria for another class of disorder (lifetime prevalence). Specifically, 25% met criteria for two classes of disorder, 11% for three classes, and 3% for four classes; anxiety disorders were the most likely to occur alone. Similar findings emerged from the National Comorbidity Survey Replication (Kessler et al., 2005), conducted with adults age 18 years and older, in that 45% of adults evidenced comorbid disorders (22% with two diagnoses, 23% with three or more diagnoses).

Not only is comorbidity high across classes of disorders, but it also appears that youth with comorbid psychopathology are likely to be from high-risk family backgrounds. Copeland, Shanahan, Costello, and Angold (2009) examined psychosocial risk factors in two community samples using latent class analysis. Their findings identified a latent class of 8.6% of youth who had experienced high levels of parental mental illness, parental crime, interparental problems (e.g., conflict, poor communication, dissatisfaction), and parent–child conflict. These high-risk youth were uniquely at risk for comorbidity, being both the most likely youth in the samples to be diagnosed with a disruptive behavior disorder and also one of the two classes at high risk for emotional disorders.

Furthermore, youth with comorbid disorders appear to be at high risk for social dysfunction. For example, in a review of the comorbidity of depression and conduct disorder, Capaldi and Kim (2014) found that youth experiencing comorbidity (or co-occurrence of high levels of symptoms) were at risk for a broad range of problem outcomes and experienced difficulties in their social relationships. Given the family risk factors experienced by these comorbid/co-occurring youth and the problematic outcomes associated with comorbidity or co-occurrence, such youth would likely be at high risk for problematic romantic relationships.

Others have recently reviewed issues of comorbidity among adolescents and young adults with social anxiety and depressive disorders (Starr, Davila, La Greca, & Landoll, 2011), suggesting that such comorbidity is related to impairments in youths' social functioning. Moreover, impaired social functioning with peers is common among youth with anxiety, depression, and disruptive behavior problems (La Greca & Lai, 2014) and thus is likely to be impaired among youth with comorbid conditions.

There is, however, very little research that specifically speaks to the issue of comorbidity or co-occurrence of psychopathology in relation to adolescent romantic relationships. Most research, as reviewed in the earlier sections, has examined specific disorders or symptoms without considering or controlling for comorbid conditions. However, one study of early adolescent girls (ages 13 to 15 years) did examine the unique associations between different types of psychopathology (depressive, anxiety, externalizing, and eating symptoms) and romantic and sexual experiences (Starr et al., 2012). In models including all four types of symptoms, both depressive and externalizing symptoms uniquely predicted increases in sexual activity over 1 year, and eating pathology was marginally associated with decreases in dating activities. In models predicting symptoms from dating and romantic activities, greater dating activities predicted increases in depressive, externalizing, and anxiety symptoms, and greater sexual activity predicted increases in externalizing and eating pathology over 1 year. These findings suggest bidirectional associations between dating/sexual involvement and psychopathology among early adolescent girls. In addition, the study found that girls with more comorbid disorders reported more dating and sexual activities, suggesting that comorbidity may

put adolescent girls at greatest risk for potential negative outcomes. Overall, these findings provide evidence of equifinality in that different types of symptoms may lead to problematic romantic and sexual outcomes, as well as multifinality, in that romantic and sexual experiences each lead to multiple types of symptomatic outcomes.

Another study examined the association of antisocial behavior and depressive symptoms of both partners with risk for IPV in young adults (ages approximately 20 to 24 years) in a lower socioeconomic sample (Kim & Capaldi, 2004). In multivariate models predicting to IPV from both partners' antisocial behavior and depressive symptoms, it was found that young women's antisocial behavior and depressive symptoms were significantly related to concurrent levels of men's physical and psychological IPV, and women's depressive symptoms remained significant in predicting men's psychological IPV over time. Overall, the men's risk factors had little effect on their partner's IPV. These findings indicate increased risk for IPV if individuals have high levels of co-occurring symptoms of psychopathology in the externalizing and internalizing domains, and that risk also is increased because of assortative partnering by such symptoms.

ADOLESCENT PARENTHOOD AND ITS ASSOCIATION WITH PSYCHOPATHOLOGY

There is clear evidence that adolescent parenthood is associated with various types of psychopathology. High rates of depressive symptoms are found among adolescent mothers (e.g., Brown, Harris, Woods, Buman, & Cox, 2012), even higher than those among adult mothers (e.g., Deal & Holt, 1998). Furthermore, reduced support may play a role in depression among adolescent mothers. A study of low-income, single Latina and African American adolescent mothers indicated particularly high rates of prenatal depressive symptoms among those reporting low-maternal availability (Milan et al., 2007). Another study with a similar population found that increases in depressive symptoms 3 years postpartum were predicted by stressors and low levels of support (Leadbetter & Linares, 1992). Similar findings exist for young men. Not only do expectant fathers report more internalizing disorders than nonexpectant fathers (Moore & Florshein, 2001), but Lee, Fagan, and Chen (2012) also found that late adolescent fatherhood was associated with depressive symptoms, particularly among those reporting low levels of support.

Early onset depression is associated with increased risk for adolescent parenthood, particularly for females (Kessler et al., 1997). This also is true for anxiety, substance use, and conduct disorders (the latter particularly for males; Kessler et al., 1997). In line with this, a number of additional longitudinal studies have found that both male and female adolescents who in childhood demonstrate aggressive behavior, substance use, or association with deviant peers are at greater risk of adolescent parenthood (Fagot, Pears, Capaldi, Crosby, & Leve, 1998; Miller-Johnson, Winn, Coie, Malone, & Lochman, 2004; Miller-Johnson et al., 1999, Underwood, Kupersmidt, & Coie, 1996). Similarly, compared with nonexpectant males, expectant males report higher rates of behavioral disorders and substance-use disorders (Moore & Florsheim, 2001).

Psychopathology puts youth at risk for adolescent parenthood, but parenthood can increase risk for psychopathology. As noted earlier, depressive symptoms increase over time among adolescent mothers with high stress and low support (Leadbeater & Linares, 1992). In addition, adolescent males show a rise in alcohol consumption after becoming parents, whereas young-adult men do not (Little, Handley, Leuthe, & Chassin, 2009). Taken together, the data suggest that there may be bidirectional associations between adolescent parenthood and psychopathology. This is consistent with a study by Oxford et al. (2005), who categorized unmarried, pregnant 17-year-old females into three groups and followed them over time: problem prone (those who demonstrated mental health and conduct problems), psychologically vulnerable (those who demonstrated mental health problems but not conduct problems), and normative. Compared with the normative group, the psychologically vulnerable group continued to show mental health problems over time, and the problem-prone group had the worst long-term outcomes across a variety of dimensions. This may suggest that being pregnant in adolescence is primarily associated with negative outcomes among those already demonstrating conduct and mental health problems.

Psychopathology among adolescent parents is associated with relationship problems. Moore, Florsheim, and Butner (2007) found, for a sample of adolescent co-parenting couples, that mothers with a history of substance abuse and fathers with a history of conduct disorders were more likely to engage in IPV. The same research team also found that adolescent co-parenting couples in which both partners had a substance-use disorder displayed (during interaction) more hostile and less warm behaviors with one another compared to couples in which neither partner had a substance-use disorder (Florsheim & Moore, 2008).

Not surprisingly, relationship problems among adolescent parents also are associated with psychopathology. Two domains of functioning have been implicated across

a number of studies. First, IPV experienced by adolescent mothers is associated with depressive symptoms (Gavin, Lindhorst, & Lohr, 2011; Lindhorst & Oxford, 2008). Second, adolescent parents, both females and males, who are less satisfied with their partners' provision of support or involvement in caregiving report greater depressive symptoms.

Adolescent parenthood is part of a pattern of premature adulthood among conduct problem and comorbid youth (Capaldi & Stoolmiller, 1999). Because of the peak of delinquency in mid to late adolescence and high rates of substance use, becoming a parent during these years is problematic for the adolescent parent and also for the child. These young parents have not yet reached the age for the improvements associated with development and maturation.

SAME-SEX RELATIONSHIPS

There is ample evidence that sexual-minority youth experience greater psychological distress (especially depression, suicidality, and substance-use problems) than heterosexual youth (Mays & Cochran 2001; Meyer, Dietrich, & Schwartz, 2008; Ueno, 2010). These disparities are in place by early adolescence (Jager & Davis-Kean, 2011). There is some evidence that they are particularly large for youth who are attracted to both sexes (Russell, Driscoll, & Truong, 2002), youth who have greater changes in sexual orientation over time (Ott et al., 2013), and youth who identify as heterosexual but report same-sex attractions (Jager & Davis-Kean, 2011). There is evidence that these disparities remain largely stable over the transition from adolescence to adulthood (Needham, 2012) or narrow over time (Jager & Davis-Kean, 2011). Thus, although there is ongoing risk for sexual minority youth, the lack of evidence that the disparities increase over time is a positive aspect.

Compared to the research on mental health disparities among sexual-minority youth, there is very little research that examines romantic and sexual functioning and its association with psychopathology for these youth. As Russell and Consolacion (2003) describe, initiation of romantic relationships in adolescence for sexual-minority youth does not tend to follow the progression identified for heterosexual youth (see D'Augelli, 1988; Koch, 1993; Savin-Williams, 1994). The typical progression of romantic development in adolescence in which youth move from friendships to intimate dyadic relationships may not apply to sexual-minority youth who may not feel comfortable initiating, or may not be able to safely initiate, romantic or sexual involvements. As such, sexual-minority youth likely face challenges that may impact their well-being and mental health. Indeed, Glover, Galliher, and Lamere (2009) found that gay, lesbian, bisexual, and transgender (GLBT) youth (ages 14 to 21 years), who described themselves as isolated from intimate relationships, reported lower levels of relational self-esteem and higher levels of relational depression, which included beliefs about not being able to be a good partner and being disappointed in relationships.

The few additional existing studies provide a mix of good news and bad news. On the positive side, there is some evidence that sexual-minority youth are as likely to be in a romantic relationship as heterosexual youth (Russell & Consolacion, 2003), suggesting that sexual-minority youth are pursuing relationships, hence providing them the opportunity to reap the benefits of relationships. In line with this, gay male youth (ages 15 to 19 years), who reported being in a same-sex relationship, reported greater self-esteem than gay male youth not in a relationship (Bauermeister et al., 2010). Bauermeister et al. (2010) also found no differences in anxiety and depression among GLBT youth (males and females) who were in versus not in a same-sex romantic relationship. Unfortunately, comparisons with heterosexual youth could not be made in this study because it included only GLBT youth. In addition, the sample was from drop-in centers in New York City, which may not be representative of youth from other areas of the country and which may represent a sample of youth who already have better coping skills.

On the less positive side, Russell and Consolacion (2003) found that same-sex daters reported more suicidal thoughts than heterosexual daters. Interestingly, same-sex daters did not report greater depression (although they did so in comparison to heterosexual singles). The researchers suggest that this may be because being in an open same-sex relationship may increase stigma and bullying, resulting in suicidal ideation. Russell and Consolacion (2003) also found that, compared to all heterosexual youth (single and dating), sexual-minority youth who are single or who are in an opposite-sex relationship report greater anxiety. This finding has a number of implications. First, it may be that, for sexual-minority youth, although being in a same-sex relationship has some potentially negative consequences (e.g., stigma and suicidal ideation), not being in a same-sex relationship may deprive them of the benefits of intimate relationships. In addition, being single or in an opposite-sex relationship may increase feelings of isolation, loneliness, or a sense of false identity or identity confusion that may result in anxiety. Similarly, being in an opposite-sex relationship may reflect internalized homophobia, which also is associated with heightened levels of anxiety (and depression) (Bos, Sandfort, Bruyn, & Hakvoort, 2008;

Igartua, Gill, & Montoro, 2003; Szymanski, Chung, & Balsam, 2001).

Cleary, the existing literature shows that the associations between romantic and sexual functioning and psychopathology among sexual-minority youth are not simple. Although mental health disparities exist between sexual-minority and heterosexual youth, when and how they do so in the context of actual relationship and sexual functioning is not entirely clear. Furthermore, there are likely to be complex issues, revolving around issues like identity, stress, and environmental support that bear on and complicate developmental trajectories. Further research designed to examine these potentially complex associations is needed.

One area that is problematic for sexual-minority youth is that of increased risk of sexually transmitted infections associated with anal intercourse. According to CDC surveillance statistics, in 2010 the majority of new HIV infections was attributed to male-to-male sexual contact (63% overall and 78% among males). The results of a meta-analysis conducted by Herrick, Marshal, Smith, Sucato, and Stall (2011) indicated that sexual-minority youth are almost twice as likely as heterosexual youth to report engaging in sex while intoxicated. These high-risk behaviors have implications for mental as well as for physical health.

Further research regarding romantic relationship development for sexual-minority youth, using new samples, is greatly needed. The majority of research comes from the Add Health data set. Although this is a large and nationally representative data set, repeated analyses of the same sample will, eventually, fail to yield new knowledge. Furthermore, there are limitations to the data available from any one data set. Moreover, changes in sociocultural and sociopolitical contexts in the United States may result in important changes in how sexual minorities are viewed and the types of stressors they experience, which may eventually bear upon associations between romantic functioning and psychopathology.

THE PEER CONTEXT

In earlier sections, we reviewed the ways in which problematic romantic relationships (or lack of romantic relationships) have been linked with psychopathology, and particularly with depression, anxiety, and antisocial behavior. Here we take a step back to examine key antecedents of adolescents' romantic relationships—specifically, the presence and quality of their friendships—and to also consider other peer contextual issues.

There is no doubt that peer relations play an instrumental role in the development of youths' romantic relationships. As noted earlier, during early to midadolescence, other-sex close friendships become common (Collins et al., 2009; Kuttler, La Greca, & Prinstein, 1999), and youth engage in mixed-sex group interactions or group dating that is characterized by affiliation and companionship (Collins et al., 2009; Schulman & Scharf, 2000). These mixed-sex peer groups set the stage for the more intimate dyadic romantic relationships that occur in later adolescence (Collins et al., 2009; Connolly & Goldberg, 1999) and that involve higher levels of intimacy and closeness and more extensive sexual activity (Connolly & Goldberg, 1999; Shulman & Scharf, 2000).

Here we briefly describe ways that the peer context contributes to problems within romantic relationships. As Furman, Low, and Ho (2009) emphasized, adolescent romantic relationships have both positive and negative correlates; they are associated not only with social competence but also with aggressive and risky behaviors that emerge in peer contexts.

Number and Quality of Friendships

The number and quality of adolescents' same- and other-sex friendships facilitate (or hinder) romantic relationships (Connolly & Goldberg, 1999; Feiring, 1999: Furman, Simon, Shaffer, & Bouchey, 2002; Kuttler & La Greca, 2004; La Greca & Mackey, 2007; Shulman & Scharf, 2000). For example, Feiring (1999) followed youth from ages 9 to 18 years, finding that the frequency of other-sex friendships increased markedly during early adolescence and that adolescents with fewer other-sex friendships began dating later than those with more other-sex friends. Adolescents (especially girls) with more peer friendships are more likely to be involved in romantic relationships (Kuttler & La Greca, 2004).

Furthermore, across community samples, findings indicate that less socially competent youth have poorer quality romantic relationships than those who are more socially competent (e.g., Connolly et al., 2000; Herbert et al., 2013; Kuttler & La Greca, 2004; Kuttler et al., 1999; La Greca & Harrison, 2005). For example, Connolly et al. (2000) found that adolescents' reports of low friend support and negative friendship interactions predicted similar problems in their romantic relationships. Additionally, adolescents with few or no close friends or with limited interpersonal skills appear to have delayed or problematic romantic relationships (La Greca & Harrison, 2005; La Greca & Lopez, 1998).

The above developmental findings suggest that adolescents with limited close friendships, or with poor quality friendships, are at risk for problems with or delays in developing romantic relationships. In turn, absent, delayed, or problematic romantic relationships can contribute to adjustment problems and psychopathology. A recent study by Herbert and colleagues (2013) examined some of these processes in detail. Specifically, adolescents (aged 14 to 19 years) with a prior or current history of romantic relationship involvement were evaluated with respect to the number and quality of their same- and other-sex friendships and their functioning in romantic relationships. Using structural equation modeling to evaluate a developmental psychopathology model, the authors found that adolescents with greater social anxiety had poorer functioning in their same- and other-sex friendships (i.e., fewer friends, less satisfaction with friends, and less social support) that, in turn, was associated with poorer romantic relationship functioning. Impairments in same-sex friendships appeared to mark the initial deviation from normative social development for socially anxious adolescents.

Peer Selection and Socialization Processes in Romantic Relationships

Peer selection and socialization processes, also known as homophily (Kandel, 1978a, 1978b), play a key role in peer friendships and also are important for adolescents' romantic relationships (e.g., Furman & Simon, 2008; Furman et al., 2009; Simon, Aikens, & Prinstein, 2008; Wargo Aikins, Simon, & Prinstein, 2010). *Selection* refers to the process of individuals forming relationships with others (friends, romantic partners) who are similar to themselves; whereas *socialization* refers to the process of being influenced or socialized by one's friends and partners.

In the context of romantic relationships, selection processes (also referred to as "assortative partnering") may lead adolescents to select partners who share similar behaviors, attitudes, or personal characteristics. For example, Simon and colleagues (2008) examined selection processes among middle school youth, finding that even *before* developing a romantic relationship, adolescents and their romantic partners were similar on variables such as popularity, attractiveness, and depressive symptoms.

Selection and socialization processes have been most widely studied in the context of adolescent conduct problems and substance use (e.g., Dishion & Patterson, 2006; Kupersmidt, DeRosier, & Patterson, 1995). Specifically, adolescents who are high in conduct problems and substance-use behaviors often choose to associate with peers like themselves. Similarly, as discussed by Rhule-Louie and McMahon (2007), there is a significant degree of similarity between romantic partners for conduct-problem behaviors and substance use (e. g., Capaldi & Crosby, 1997; Haynie, Giordano, Manning, & Longmore, 2005; Kim & Capaldi, 2004) as well as for association with deviant peers (Krueger, Moffitt, Caspi, Bleske, & Silva, 1998); both selection and socialization processes may come into play in explaining these findings.

As discussed by Capaldi et al. (2005), selective or assortative partnering tends to occur because of at least two individual-environment interaction effects. The first effect is by active selection of environments (e.g., a young woman who likes to "party" and use substances is likely to meet young men with the same preferences). Second, engagement in conduct problems and related adjustment failures (Capaldi & Shortt, 2003) leads to unintended restriction of environmental options (e.g., an adolescent who drops out of high school may not attend a 4-year college and is less likely to date a young woman attending such a college).

In addition to selection processes, peers may influence future deviant behaviors through socialization (Dishion & Owen, 2002); and there is evidence of such socialization processes within young couples. For example, Moffitt, Caspi, Rutter, and Silva (2001) showed that controlling for the individual's prior levels of antisocial behavior, partners' antisocial behavior contributed to and encouraged antisocial behavior during the transition to adulthood.

Moreover, given higher levels of conduct-problem behaviors and delinquency in boys than in girls, such socialization influence within couples appear to be more detrimental to girls. Moffitt and colleagues' (2001) findings suggest a social amplification effect (i.e., an effect of an interaction between the individual's and partner's antisocial behavior) in predicting the persistence and growth in antisocial behavior for the girls who already showed higher levels of antisocial behavior than their female peers in adolescence.

On the other hand, young women are often considered to have a steadying or reforming effect on young men who show problem behaviors, and an association between marriage and desistence or decreases in problem behaviors in men has been found in numerous studies (Sampson & Laub, 2005). However, there is considerable heterogeneity in antisocial behavior in both girls/women and boys/men, and partner influences are likely to vary depending on their levels of antisocial behavior. Capaldi, Kim, and Owen (2008) examined influences of romantic partner's and peers' antisocial behavior on crime persistence from late adolescence through the late 20s (as indexed by official arrest records). Controlling for men's prior arrests, substance use, and depressive symptoms, findings indicated

that the young women's antisocial behavior predicted both arrest persistence and arrest onset for young men who had no previous history of arrest, whereas romantic relationship stability was protective against desistance.

Other studies, such as those by Wargo Aikins and colleagues (2010), illustrate romantic partners' socialization effects for youths' cigarette use and behavior problems. Also, in a study of high school aged adolescents, La Greca and colleagues (La Greca & Landoll, 2010) demonstrated that a romantic partner's use of cigarettes and alcohol predicted adolescents' substance use, even when controlling for friends' substance use.

Involvement with Antisocial Peers/Peer Victimization

As noted above, youth with peer/friendship problems are at risk for problems in their romantic relationships. It also appears that associations between the negative aspects of friendships and romantic relationships are *even more striking among aggressive or antisocial youth*. For example, in a rare observational study of late adolescent at-risk boys (aged about 18 years), Shortt, Capaldi, Dishion, Bank, and Owen (2003) examined the role of adolescent friends, romantic partners, and siblings in the emergence of the adult antisocial lifestyle. Across relationships, observations were coded for two behavioral domains: interpersonal negative engagement (e.g., criticism, name calling) and antisocial talk involving societal rule-breaking (e.g., suggesting the two go out and get drunk, swearing). Significant associations across relationships were seen for both negative engagement and antisocial talk, providing evidence that processes related to continuance of psychopathology in adolescence—in this case externalizing or problem behaviors—show some similarities across key close and influential relationships, including friends and romantic partners.

Moreover, there is relatively robust evidence that *involvement with antisocial and aggressive peers* is predictive of involvement in adolescent dating aggression. For example, Vézina et al. (2011) examined deviant-peer affiliation and dating violence victimization among adolescent girls. Girls with a higher level of association with antisocial peers were more likely to be involved in a risky lifestyle and report higher levels of dating violence victimization. The authors concluded that dating violence prevention efforts should target antisocial peer groups and girls displaying problem behaviors. For both boys and girls, Schnurr and Lohman (2008) found that early involvement and an increase in involvement with antisocial peers were linked to perpetrating dating violence. In addition, Foshee and colleagues (2011) found that having friends who were perpetrators or victims of dating violence was strongly associated with an adolescent's own experiences as both a perpetrator and a victim of dating violence (Arriaga & Foshee, 2004).

Being victimized by peers has also been directly linked to trajectories of dating violence victimization in adolescence. In a recent study, Brooks-Russell, Foshee, and Ennett (2013) examined heterogeneity in physical dating violence in adolescents (Grades 8 through 12) using growth mixture modeling. In multivariate models predicting to class membership, being victimized by peers distinguished the high-dating violence victimization from the noninvolved class for girls and was the only factor distinguishing the victimized from the noninvolved class for boys.

Associations have also been found for peer relational aggression and dating aggression. In a longitudinal study involving midadolescents and peer nominations, peer-group relational aggression at Time 1 positively predicted dating abuse, victimization and perpetration, and negatively predicted romantic relationship quality 6 months later—over and above individual predictions (Ellis, Chung-Hall, & Dumas, 2013). However, membership in physically aggressive peer groups at Time 1 was positively related to romantic relationship quality 6 months later.

In an unusual comparison of peer and family variables, Arriaga and Foshee (2004) compared the contributions of a much-researched predictor of dating violence, namely interparental violence, to that of having friends in violent romantic relationships. Among midadolescents, both friend-dating violence and interparental violence showed unique concurrent prediction to dating violence perpetration and victimization, but only friend violence consistently predicted dating violence 6 months later.

Peer violence has also been related to sexual aggression across partners. In a longitudinal study, Ozer, Tschann, Pasch, and Flores (2004) found that adolescent boys who engaged in peer violence were also at risk for perpetrating sexual aggression and dating violence. Results for girls were less consistent.

Overall, adolescents' involvement with antisocial and aggressive peers predicts their involvement in dating aggression, although higher friendship quality appears to be a protective factor. Peer behavior is a relatively strong and proximal predictor of adolescents' behavior in romantic relationships.

Online Communication with Peers and Partners

Technological advances that allow for immediate, rapid, and readily available communication represent another important peer context that affects adolescents' romantic relations. According to Lenhart, Ling, Campbell and

Purdell (2010), 72% of teens report using text messaging, and those who do send about 3000 messages per month. The use of social networking sites has also increased exponentially in recent years, especially among adolescents and young adults (Lenhart, Purcell, Smith, & Zickuhr, 2010).

Online communication tools, such as instant messaging, social networking sites, email, smartphone applications, and text messaging have the potential to enhance existing relationships with friends and romantic partners (e.g., Ellison, Steinfield, & Lampe, 2007; Subrahmanyam & Greenfield, 2008), enabling them to stay in touch. Indeed, Blais et al. (2008) found that greater instant messaging use was associated with greater romantic relationship quality in adolescence.

Although often a benefit to close relationships, cell phones, social networking sites, and the Internet also have the potential to complicate and even undermine romantic relationships. Adolescents and young adults report that cell phones and the Internet have been used by their romantic partners to stalk, harass, and "spy on" them (Magid, 2009). In recent work on cyber-related peer experiences, Landoll, La Greca, and Lai (2013) found that 26% of the adolescents and 8% of the young adults in the sample reported that their boyfriend/girlfriend broke up with them via a social networking site or other communication tool, and over 30% of both the adolescents and young adults reported that a peer made them feel jealous by "messing with" their boyfriend/girlfriend on a social networking site (such as by posting pictures together or writing on their Facebook wall).

Also concerning is a recent national survey indicating that about 20% of teens (aged 13 to 19 years) participate in "sexting" (i.e., sending sexually explicit picture messages) and that such activities increase among young adults ages 20 to 26 years (National Campaign to Prevent Teen and Unplanned Pregnancy, 2008); rates of sending suggestive messages via text, email, or instant messaging are even more prevalent, with 39% of teens and 59% of young adults reporting such activities. Sexting often occurs in the context of a romantic relationship (either current or desired) (Drouin & Landgraff, 2012), but when such images are distributed after a break up with a romantic partner or released with malicious intent, sexting can be associated with high levels of psychological distress (Willard, 2010).

In fact, studies of the psychological ramifications of such cyber experiences are relatively few but recently emerging. For example, negative social networking experiences have been associated with adolescents' and young adults' reports of social anxiety and depressive symptoms (Landoll et al., 2013), even when controlling for other forms of peer victimization. In another recent study, Temple and colleagues (2014) surveyed over 900 ethnically diverse high school students, finding that sexting was associated with symptoms of depression, impulsivity, and substance use. Drouin and Landgraff (2012) also found that sexting was more common among older adolescents/young adults with insecure attachments in their romantic relationships. Although further study is needed, it is reasonable to expect that these examples of cyber victimization and sexting, in particular, could undermine romantic relationships and contribute to substantial psychological distress.

THE FAMILY CONTEXT: INTERGENERATIONAL TRANSMISSION AND IMPLICATIONS FOR LONG-TERM OUTCOMES

The association between adolescent romantic functioning and psychopathology can develop in the context of the family of origin and, once developed, has implications for the adolescent's future mental health and relational well-being. A number of studies have examined the evidence for intergenerational transmission of relationship conflict and problems and the pathways or processes that may underlie such associations. Indeed, as reviewed by Capaldi, Knoble, et al. (2012), many researchers hypothesize that the primary cause of IPV perpetration is having witnessed IPV in the family of origin.

Although numerous studies have supported this association, most have involved retrospective data and did not address mechanisms. In addition, a number of studies point to a variety of additional factors beyond simply witnessing IPV. For the OYS described earlier, intergenerational transmission of IPV was examined; evidence was found for indirect rather than direct effects. Parental antisocial behavior was found to predict both IPV in the parents and poor parenting of the son, and the poor parenting was predictive of antisocial behavior in the sons, which in turn was predictive of their IPV (Capaldi & Clark, 1998). In addition, there is substantial evidence from other studies that child maltreatment and IPV are associated (for reviews see Capaldi, Kim, & Pears, 2009; Slep & O'Leary, 2001). Furthermore, in a recent study, Feiring, Simons, Cleland, and Barrett (2013) studied 160 youth with confirmed childhood sexual abuse (CSA). They found that greater shame and self-blame following the abuse were associated with dating aggression 6 years later; this was mediated through reports of greater anger. In addition, two longitudinal studies (Ehrensaft et al., 2003; White & Widom, 2003) found that conduct problems mediated between childhood physical/sexual abuse and adult IPV

perpetration. Intergenerational transmission, therefore, is a more complex issue than modeling (in adulthood) IPV that was witnessed in childhood, although this may be a part of the transmission pattern.

In addition to evidence of transmission of relationship problems via antisocial behavior, there also is evidence that depression and emotional dysregulation play a role in the intergenerational transmission of relationship dysfunction. There is growing evidence that maternal depression sets the stage for poor relational functioning and depression among offspring. The most direct evidence of this comes from a study by Katz, Hammen, and Brennan (2013). They followed 182 youth from birth to age 20 years; at age 20 years all were in committed romantic relationships. Katz et al. found support for a series of mediational models showing that maternal depression predicted greater mother-child discord at age 15 years, which then predicted poorer romantic relationship quality at age 20 years (as reported by both partners) that then predicted increases in depressive symptoms from ages 15 to 20 years. Therefore, youth growing up with depressed mothers, with whom they have poor relationships, are at increased risk for poor quality romantic relationships and greater depression. There is ample evidence for a bidirectional association between poor romantic functioning and depression in adulthood (see Davila, Stroud, & Starr, 2014). The Katz et al. study suggests that this association begins early and can have roots in the family of origin.

Kim, Pears, Capaldi, and Owen (2009) examined the role of emotional dysregulation in the intergenerational transmission of conflict with partner for the OYS sample. Emotional dysregulation was defined as intense negative affect and emotional lability. Conflict included arguments and behaviors such as shouting, but short of psychological or physical aggression. Associations were modeled prospectively across a 20-year period into early adulthood for the male offspring. No direct association of relationship conflict across generations was found. Emotion dysregulation was associated across generations, however, and there was evidence for an indirect association of conflict across generations via both emotion dysregulation and poor parental discipline.

A number of other studies, although not as direct as the Katz et al. (2013) study, are relevant for understanding and theorizing about how maternal depression and interpersonal dysfunction can lead to similar outcomes for youth. For example, maternal mood disorders increase risk of earlier puberty among girls; this is particularly true in the absence of a biological father in the house, in the presence of a stepfather, or in the presence of a stressful home environment (Ellis & Garber, 2000). This is important because girls with early pubertal timing are at greater risk for depression and risky sex (Galvao et al., 2014; Marceau, Ram, Houts, Grimm, & Susman, 2011) than their on-time or late-developing counterparts. Keenan-Miller, Hammen, and Brennan (2007) found that maternal depression predicted IPV victimization at age 20 years for young women, and this association was mediated by poorer social functioning at age 15 years. This suggests that growing up with a depressed mother may put girls at risk for more problematic, and ultimately violent, relationships in the future.

CONCLUSIONS AND FUTURE DIRECTIONS

The literature on adolescent/young adult romantic relationships has grown dramatically in the last 20 years with the increased recognition that these relationships matter, both in the moment and over time as they set the stage for subsequent relationship development and individual well-being. In line with this recognition, researchers have begun to demonstrate the ways in which adolescent romantic functioning both contributes to and is affected by psychopathology, including depression, anxiety, and externalizing problems. There have been numerous gains in knowledge in these areas, although much more research is needed. In this concluding section, we review theoretical and methodological issues in need of continued attention, and we discuss the implications for prevention and intervention.

Issues in Theory Development

The main point we want to make here is that theory development will be critical for the research on adolescent romantic relationships and psychopathology to progress. Researchers need to develop conceptually grounded hypotheses not only about whether romantic functioning will be associated with psychopathology, but about how, why, for whom, and for which specific types of romantic experiences. Theory needs to be developmentally informed to answer, for example, questions about how early experiences relate to or shape later ones and the likelihood of associations, as well as their nature, between different types of romantic functioning and psychopathology at different ages. Theory also needs to be sensitive to potential gender differences in associations over time and their differing implications for functioning. We see advances in specific models of associations as characterizing the next generation of research in this area. The section below contains further suggestions for specific variables on which to focus.

Methodological Issues

In this section we briefly highlight some methodological issues that will be important considerations for the continuing growth and development of this critical area of research.

Study Designs

There is an ongoing need for prospective studies, particularly long-term ones, that evaluate temporal associations and bidirectional influences between romantic relationships and psychopathology. Although a number of important, methodologically strong studies exist, such as the OYS, most of the existing research was not designed to well evaluate temporal associations. For example, much of the research linking social anxiety with problems in romantic relationships has focused on concurrent or short-term prospective research designs. Multi-wave prospective studies that extend over longer time periods would be useful, as well as studies that use theoretically based and conceptually driven structural models (e.g., Herbert et al., 2013) to test temporal associations among the key variables of interest.

Sample Issues/Study Populations

Much of the available research has been conducted with community samples of adolescents and college samples of young adults. Although such studies have been invaluable for examining the role of romantic relationships as both risk and protective factors for psychological disorder, they do not reveal the full story. For example, studies of adolescents' or young adults' romantic relationships that are conducted with community samples are likely to have an underrepresentation of socially anxious individuals, as such individuals are less likely to date or be romantically involved than their peers (as noted earlier in this chapter).

Future studies are needed that evaluate the romantic relationships of adolescents and young adults with clinical levels of problems as well as those with a known risk factor for clinical disorder (e.g., offspring of depressed parents). Moreover, studies of clinical populations should address a broad array of psychological problems. As is apparent from this chapter, much of the available evidence on romantic relationships and psychopathology comes from studies of youth with conduct problems, substance-use problems, depression, or social anxiety. Considerably less is known about those with eating disorders, ADHD, bipolar disorder, or other clinical and adjustment difficulties.

The diversity of study samples is also an issue for consideration. Future studies might recruit samples that are ethnically and culturally diverse, or diverse with respect to other important variables such as sexual orientation, religion, or socioeconomic status.

In particular, the current literature has focused predominantly on heterosexual relationships; thus, the literature on sexual-minority youths' romantic relationships is sparse. Most of the available research on sexual-minority youth comes from the large and nationally representative Add Health data set. However, further research examining romantic relationship development and its psychological benefits and consequences for sexual-minority youth is greatly needed.

Measurement Issues

Many studies rely on self-reports from adolescents or young adults and focus on questionnaires and survey measures. Studies that incorporate observational data (e.g., Shortt et al., 2010), reports from romantic partners (e.g., Daley & Hamman, 2002), daily diaries (e.g., Starr & Davila, 2012), or objectively coded structured interviews (e.g., Davila, Steinberg et al., 2009; Shulman, Davila, & Shachar-Shapira, 2011) would help advance the field. Furthermore, studies that incorporate data from the dyadic romantic relationship would be especially informative.

A big question concerns which types of relationship behaviors should be the focus of study and how best to define or measure them. At present, many of the relationship variables studied are disorder specific. Thus, for example, romantic relationship variables—such as self-disclosure, emotional expression, and levels of intimacy (Sparrevohn & Rapee, 2009), as well as negative emotional expression (Kashdan et al,, 2007)—have been examined among socially anxious individuals; negative interactions, aggression, and antisocial talk have been examined among youth at risk for conduct problems (e.g., Shortt et al., 2003); and excessive reassurance seeking (Starr & Davila, 2008) and self-silencing (Harper & Welsh, 2007) have been examined among depressed adolescents and young adults. Given the high co-morbidity across psychological disorders, efforts to measure romantic relationship variables comprehensively, and to capture both positive and negative aspects of such interactions, would be important and desirable.

In addition to the relationship qualities that may be linked to psychological difficulties, other descriptive aspects of romantic relationships are important to evaluate. Especially with adolescents, such variables might include the length of the romantic relationship, its intensity (e.g., casual vs. committed), whether it includes sexual intimacy, and the frequency and duration of break ups with partners.

Finally, future studies need to consider technological and cultural trends. For example, studies are needed that

examine the impact of technology on the course and outcomes of romantic relationships, as well as studies that keep abreast of emerging cultural trends in dating and romantic relationships (e.g., hooking up, oral sex, friends with benefits).

Psychological Outcomes and Issues of Comorbidity

For the most part, investigators have approached psychological outcomes as if they were discrete. However, studies are needed that measure multiple psychological outcomes and examine co-morbid psychological variables in a more consistent and sophisticated manner. In general, externalizing problems (e.g., conduct disorders, substance use) have been associated with greater problems in romantic relationships (e.g., more negative qualities, more IPV); whereas internalizing problems, and especially anxiety, have been more closely associated with the absence or delay of romantic relationships or poor communication skills within such relationships.

As noted earlier in the chapter, psychological comorbidity is high. In particular, studies are needed that examine co-morbid depression and externalizing disorders such as conduct disorder. For example, several problematic romantic relationship issues (e.g., early dating, early sexual activity, early transition from same-sex to mixed-sex groups) have been associated both with depression and conduct problems.

The neglect of comorbidity primarily reflects the way that research on developmental psychopathology has evolved. However, this neglect also underscores the need for more complex models that incorporate issues of comorbidity.

Important Mediating and Moderating Variables

As reflected in our comments on theory development, in building conceptually driven models of the impact of romantic relationships, investigators would also do well to focus on mechanisms of association. The literature on depression provides a model for doing so. For example, researchers have begun to question why depression might lead to romantic and sexual activity and what it is about such activities that can lead to depression; specific theory-driven predictions are now being tested. In general, it has been suggested that romantic experiences are inherently challenging and consequently may put youth at risk for depression, anxiety, aggressive behavior, or substance use—particularly youth with poor coping, social support, or personal resources that impair their ability to manage such challenges. Alternatively, certain youth (depressed or aggressive youth) may seek out romantic experiences to meet their support needs, compensate for poor family or peer relations, or as a means of mood regulation. These hypotheses would now be important to examine using study designs that allow for their testing.

In developing more sophisticated, conceptually driven models to understand the linkages between romantic relationships and psychopathology, concerted efforts are needed to examine variables that moderate these associations. Consistent with our earlier suggestions, several variables warrant serious consideration in this regard. In particular, *age/developmental level* may be incredibly important for understanding what is healthy or dysfunctional (e.g., sexual activity may be related to psychopathology at certain ages but not at other ages). Similarly, the *timing* of transitions to dating or to mixed-sex friendship groups may be an important consideration for mental health, as appears to be the case for youth with conduct problems. *Gender* is another key variable for understanding the connection between romantic relationships and psychological functioning. Among the findings reviewed in this chapter, it appears that girls date earlier than boys but are also more likely to report depression in romantic relations; girls also appear to be more adversely affected by sexting and IPV. Systematic attention to gender as a moderating variable is needed.

Other variables of interest include those that may be important links or mediators between romantic relationship difficulties and psychological dysfunction. Potentially important candidates for mediating variables include *interpersonal and cognitive processes* that could affect the relationship/adjustment linkages, such as co-rumination, problems with emotion regulation, communication deficits, or poor problem-solving and conflict-resolution skills.

Public Health Implications and Translational Efforts

Finally, future studies are needed that address outcomes that are clinically and practically relevant. Investigators need to translate their findings into practical implications for assessment, intervention, and prevention. In the next section, we discuss some of the clinical implications that can be drawn from the findings presented in this chapter.

Translational Implications

As this chapter demonstrates, not only are youth romantic relationships significant for normative development, they play a particularly significant role in the development and course of psychopathology. That their joint courses start early, continue into adulthood, and are transmitted across generations suggests that intervention and prevention

efforts that target their association are needed. At present, few exist.

As our review shows, the association between depression/depressive symptoms and romantic relationships is one of the most well-studied and consistently demonstrated. Yet, for the most part, there are no depression prevention or treatment programs that are explicitly aimed at or include components designed to address navigating romantic relationships. Perhaps the one exception is Interpersonal Psychotherapy for Adolescents (IPT-A; Mufson et al., 2004), a clinical treatment for depressed adolescents, and a related depression-prevention program, Interpersonal Psychotherapy - Adolescent Skills Training (IPT-AST; Young, Mufson, & Davies, 2006; Young, Mufson, & Gallop, 2010). IPT-A and IPT-AST both focus on enhancing adolescents' interpersonal relationships and teaching effective communication strategies, particularly for relationships that contribute to depressed mood. Thus, although IPT-A and IPT-AST were not designed to focus explicitly on issues of romantic relationships, in practice, they may do so for depressed adolescents who are concerned about their romantic relationships and want help with navigating them successfully. Notably, IPT-AST has shown positive effects in improving adolescents' functioning in social and dating relationships (e.g., Young, Kranzler, Gallop, & Mufson, 2012).

As discussed earlier, adolescent social anxiety can also contribute to problematic or delayed romantic relationships. At present, no evidence-based programs exist for the prevention of adolescent social anxiety. However, current work is underway to extend the IPT-AST prevention model to adolescents with elevated levels of social anxiety with or without depressive symptoms (La Greca, Ehrenreich-May, & Mufson, 2013), so data on the utility of the IPT-AST model for socially anxious adolescents will be forthcoming.

There also are no intervention or prevention programs for externalizing youth that specifically include components designed to address navigating romantic relationships. This is even true for programs that target prevention of IPV, which our review has shown is strongly associated with externalizing pathology. Whitaker, Murphy, Eckhardt, Hodges, and Cowart (2013) recently reviewed IPV prevention programs conducted with adolescents. Most studies that met design-quality criteria for inclusion in the review were universal interventions (16 of 19 or 84%) and thus did not involve selection for level of IPV risk (e.g., by higher levels of conduct problems). Nine rigorous studies were identified, although none had been replicated. Four were based in middle and high school and only one of these, a universal intervention, had an unqualified positive impact on IPV behavior, including long-term effects (Foshee et al., 1996, 2005). This program has been adapted to an intervention mailing informative booklets to parents to review with their adolescent, which has promising initial results and could potentially reach many more adolescents and families. There have been only a few successful indicated IPV prevention programs with adolescents. Wolfe et al. (2003) conducted a preventive intervention with youth involved in child protective services, with positive effects on IPV perpetration and victimization. Florsheim, McArthur, Hudak, Heavin, and Burrow-Sánchez (2011) conducted a program with pregnant teens and the babies' fathers, with beneficial effects on IPV. A note of caution regarding prevention is introduced by the fact that one universal prevention program tested with early middle school students actually had iatrogenic effects, such that the intervention conditions showed greater perpetration of physical and sexual aggression.

As discussed by Capaldi and Kim (2014), despite the fact that effective behavior-based prevention and treatment programs are available for children who exhibit externalizing problems (e.g., Reid, Eddy, Fetrow, & Stoolmiller, 1999) and internalizing problems (e.g., Stice, Shaw, Bohon, Marti, & Rohde, 2009), prevention and intervention programs targeting co-occurring symptoms are limited. This is unfortunate given the high rates of comorbidity that exist. A new treatment approach involving flexible modules suitable to addressing depression, conduct disorders, and anxiety—the MATCH approach—shows promising evidence of effectiveness in a randomized control trial (Weisz et al., 2012). Such a modular approach could be a strong direction for addressing relationship issues with multiproblem youth, and could contain a module addressing positive romantic relationship skills and violence-free relationships. Adding such a component to youth prevention and intervention programs currently in place—which are designed to address factors such as deviant peer association, conduct problems, and substance use—would also be valuable because youth at risk for such problems are also at risk for romantic relationship problems and IPV. Adding a component to ongoing programs could be a relatively simple and cost-effective way to increase prevention related to romantic relationship issues (Capaldi, Knoble et al., 2012).

Related to this, developing romantic relationship education (RE) programs that are targeted to at-risk or symptomatic youth would also be an important direction for prevention efforts. At present, there are two youth RE programs that have been shown to be efficacious (see Davila and Rhoades, in preparation) and both were

designed as universal programs to be delivered in a school context. Adapting these programs and/or developing new RE programs that are uniquely suited to the needs of different at-risk/symptomatic youth, and that are based on existing empirical data such as that reviewed in this chapter, may also be an effective way to increase romantic competence and, ultimately, decrease psychopathology among high risk adolescents.

An important conclusion to be drawn from findings on the association between romantic relationships and psychopathology in youth is that associations exist for both boys and girls. Therefore, psychopathology prevention efforts should alleviate relationship problems and enhance relationship functioning for both boys and girls and RE efforts should reduce risk for psychopathology for both boys and girls. This is particularly true for the association of externalizing problems and relationship functioning. For internalizing problems, including depressive symptoms and anxiety, associations between psychopathology and relationship functioning may in some cases be worse for adolescent girls than boys. However, associations are also found for boys, especially boys who are socially anxious, and research has not yet sufficiently studied associations among boys. Furthermore, for externalizing problems, evidence of higher risk for relationship problems because of assortative partnering by problem behaviors (i.e., that adolescents with conduct problems are likely to date adolescents with conduct problems) indicates that prevention or alleviation of psychopathology may be especially beneficial.

Considering the magnitude of public health issues caused by dysfunctional or violent romantic relationships, the state of the field regarding rigorously assessed prevention programs is fledgling. Part of the reason for this has been a relative lack of federal investment in the research necessary to develop and test such programs at an adequately rigorous level. It is also the case that, in the past, programs related to romantic relationship problems have not addressed proven risk factors such as psychopathology. Greater understanding of the role that both boys' and girls' psychopathology and related risk factors play in the development of romantic relationship problems should lead to stronger intervention programs.

In conclusion, given that dating relationships form a foundation for future romantic relationships that themselves are the foundation for the family of procreation, and that adolescent dating relationships themselves may involve the birth of children, the costs of very dysfunctional and even violent relationships for the adolescents, their offspring, and society can be very high. Thus investment in programs around relationships may reap multiple benefits.

REFERENCES

Abma, J. C., Martinez, G. M., Mosher, W. D., & Dawson, B. S. (2004). Teenagers in the United States: Sexual activity, contraceptive use, and childbearing, 2002. *Vital and Health Statistics*, *23*(24). Hyattsville, MD: National Center for Health Statistics.

Ackard, D. M., Fedio, G., Neumark-Sztainer, D., & Britt, H. R. (2008). Factors associated with disordered eating among sexually active adolescent males: Gender and number of sexual partners. *Psychosomatic Medicine*, *70*(2), 232–238. doi: 10.1097/PSY.0b013e318164230c

Ackard, D. M., & Neumark-Sztainer, D. (2002). Date violence and date rape among adolescents: Associations with disordered eating behaviors and psychological health. *Child Abuse & Neglect*, *26*, 455–473. doi: 10.1177/1524838006297029

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision). Arlington, VA: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Association.

Andrews, J. A., Foster, S. L., Capaldi, D. M., & Hops, H. (2000). Adolescent and family predictors of physical aggression, communication, and satisfaction in young adult couples: A prospective analysis. *Journal of Consulting and Clinical Psychology*, *68*, 195–208. doi: 10.1037/0022-006X.68.2.195

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, *40*, 57–87. doi: 10.1111/1469-7610.00424

Arriaga, X. B., & Foshee, V. A. (2004). Dating violence: Do adolescents follow in their friends', or their parents', footsteps? *Journal of Interpersonal Violence*, *19*, 162–184**.** doi: 10.1177/0886260503260247

Babinski, D. E., Pelham, W. R., Molina, B. G., Gnagy, E. M., Waschbusch, D. A., Yu, J., . . . Karch, K. M. (2011). Late adolescent and young adult outcomes of girls diagnosed with ADHD in childhood: An exploratory investigation. *Journal of Attention Disorders*, *15*, 204–214. doi: 10.1177/1087054710361586

Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2006). Young adult outcome of hyperactive children: Adaptive functioning in major life activities. *Journal of American Academy of Child and Adolescent Psychiatry*, *45*, 192–209. doi: 10.1097/01.chi.0000189134.97436.e2

Bauermeister, J. A., Johns, M., Sandfort, T. M., Eisenberg, A., Grossman, A. H., & D'Augelli, A. R. (2010). Relationship trajectories and psychological well-being among sexual minority youth. *Journal of Youth and Adolescence*, *39*, 1148–1163. doi: 10.1007/s10964-010-9557-y

Beck, J. G., Davila, J., Farrow, S., & Grant, D. (2006). When the heat is on: Romantic partner responses influence distress in socially anxious women. *Behaviour Research and Therapy*, *44*, 737–748. doi: 10.1016/j.brat.2005.05.004

Beidel, D. C., Turner, S. M., & Morris, T. L. (1999). Psychopathology of childhood social phobia. *Journal of American Academy of Child and Adolescent Psychiatry*, *38*, 643–650. doi: 10.1097/00004583-199906000-00010

Blais, J. G., Craig, W. M., Pepler, D. J., & Connolly, J. (2008). Adolescents online: The importance of Internet activity choices to salient relationship. *Journal of Youth and Adolescence*, *37*, 49–58. doi: 10.1007/s10964-007-9262-7

Blumstein, A., Cohen, J., Roth, J. A., & Visher, C. A. (Eds.). (1986). *Criminal careers and career criminals* (Vol. 1). Washington, DC: National Academy Press.

Bos, H. M. W., Sandfort, T. G. M., Bruyn, E. H., & Hakvoort, E, M. (2008).Same-sex attraction, social relationships, psychosocial functioning, and school performance in early adolescence. *Developmental Psychology*, *44*, 59–68. doi: 10.1037/0012-1649.44.1.59

Brooks-Russell, A., Foshee, V. A., & Ennett, S. T. (2013). Predictors of latent trajectory classes of physical dating violence victimization. *Journal of Youth and Adolescence*, *42*, 566–580. doi: 10.1007/s10964-012-9876-2

Brown, J. D., Harris, S., Woods, E. R., Buman, M. P., & Cox, J. E. (2012). Longitudinal study of depressive symptoms and social support in adolescent mothers. *Maternal and Child Health Journal*, *16*(4), 894–901. doi: 10.1007/s10995-011-0814-9

Buzy, W. W., McDonald, R., Jouriles, E., Swank, P. R., Rosenfield, D., Shimek, J. S., & Corbittt-Shindler, D. (2004). Adolescents girls' alcohol use as a risk factor for relationship violence. *Journal of Research on Adolescence*, *14*, 449–470. doi: 10.1111/j.1532-7795.2004.00082.x

Canino, G., Shrout, P. E., Rubio-Stipec, M., Bird, H. R., Bravo, M., Ramirez, R., . . . Martinez-Taboas, A. (2004). The *DSM-IV* rates of child and adolescent disorders in Puerto Rico: Prevalence, correlates, service use, and the effects of impairment. *Archives of General Psychiatry*, *61*, 85–93. doi: 10.1001/archpsyc.61.1.85

Capaldi, D. M., Chamberlain, P., Fetrow, R. A., & Wilson, J. (1997). Conducting ecologically valid prevention research: Recruiting and retaining a "whole village" in multimethod, multiagent studies. *American Journal of Community Psychology*, *25*, 471–492. doi: 10.1023/A:1024607605690

Capaldi, D. M., & Clark, S. (1998). Prospective family predictors of aggression toward female partners for at-risk young men. *Developmental Psychology*, *34*, 1175–1188. doi: 10.1037/0012-1649.34.6.1175

Capaldi, D. M., & Crosby, L. (1997). Observed and reported psychological and physical aggression in young, at-risk couples. *Social Development*, *6*, 184–206. doi: 10.1111/j.1467-9507.1997.tb00101.x

Capaldi, D. M., Crosby, L., & Stoolmiller, M. (1996). Predicting the timing of first sexual intercourse for at-risk adolescent males. *Child Development*, *67*, 344–359. doi: 10.2307/1131818

Capaldi, D. M., Dishion, T. J., Stoolmiller, M., & Yoerger, K. (2001). Aggression toward female partners by at-risk young men: The contribution of male adolescent friendships. *Developmental Psychology*, *37*, 61–73. doi: 10.1037/0012-1649.37.1.61

Capaldi, D. M., & Kim, H. K. (2014). Comorbidity of depression and conduct disorder. In C. S. Richards & M. W. O'Hara (Eds.), *The Oxford handbook of depression and comorbidity* (pp. 186–199). New York, NY: Oxford University Press.

Capaldi, D. M., Kim, H. K., & Owen, L. D. (2008). Romantic partners' influence on men's likelihood of arrest in early adulthood. *Criminology*, *46*, 401–433. DOI: 10.1111/j.1745-9125.2008.00110.x

Capaldi, D. M., Kim, H. K., & Pears, K. C. (2009). The association between partner violence and child maltreatment: A common conceptual framework. In D. Whitaker & J. Lutzker (Eds.), *Preventing partner violence: Research and evidence-based intervention strategies* (pp. 93–111). Washington, DC: American Psychological Association.

Capaldi, D. M., Kim, H. K., & Shortt, J. W. (2004). Women's involvement in aggression in young adult romantic relationships: A developmental systems model. In M. Putallaz & K. L. Bierman (Eds.), *Aggression, antisocial behavior, and violence among girls: A developmental perspective* (pp. 223–241). New York, NY: Guilford.

Capaldi, D. M., Kim, H. K., & Shortt, J. W. (2007). Observed initiation and reciprocity of physical aggression in young, at-risk couples. *Journal of Family Violence*, *22*, 101–111. doi: 10.1007/s10896-007-9067-1

Capaldi, D. M., Knoble, N. B., Shortt, J. W., & Kim, H. K. (2012). A systematic review of risk factors for intimate partner violence. *Partner Abuse*, *3*, 231–280. doi: 10.1891/1946-6560.3.2.231

Capaldi, D. M., & Owen, L. D. (2001). Physical aggression in a community sample of at-risk, young couples: Gender comparisons for high frequency, injury, and fear. *Journal of Family Psychology*, *15*, 425–440. doi: 10.1037/0893-3200.15.3.425

Capaldi, D. M., Pears, K. C., Kerr, D. C. R., Owen, L. D., & Kim, H. K. (2012). Growth in externalizing and internalizing problems in childhood: A prospective study of psychopathology across three generations. *Child Development*, *833*, 1945–1959. doi: 10.1111/j.1467-8624.2012.01821.x

Capaldi, D. M., & Shortt, J. W. (2003). Understanding conduct problems in adolescence from a lifespan perspective. In G. R. Adams & M. D. Berzonsky (Eds.), *Blackwell handbook of adolescence* (pp. 470–493). Oxford, UK: Blackwell Publishing.

Capaldi, D. M., Shortt, J. W., & Kim, H. K. (2005). A life span developmental systems perspective on aggression toward a partner. In W. M. Pinsof & J. L. Lebow (Eds.), *Family psychology: The art of the science* (pp. 141–167). New York, NY: Oxford University Press.

Capaldi, D. M., & Stoolmiller, M. (1999). Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: III. Prediction to young-adult adjustment. *Development and Psychopathology*, *11*, 59–84. doi: 10.1017/S0954579499001959

Capaldi, D. M., Stoolmiller, M., Kim, H. K., & Yoerger, K. (2009). Growth in alcohol use in at-risk adolescent boys: Two-part random effects prediction models drug and alcohol dependence. *Drug and Alcohol Dependence*, *105*, 109–117. doi: 10.1016/j.drugalcdep.2009.06.013

Carver, K., Joyner, K., & Udry, J. R. (2003). National estimates of adolescent romantic relationships. In P. Florsheim (Ed.), *Adolescent romantic relations and sexual behavior: Theory, research, and practical implications* (pp. 23–56). Mahwah, NJ: Lawrence Erlbaum.

Cash, T. F., Theriault, J., & Annis, N. M. (2004). Body Image in an interpersonal context: Adult attachment, fear of intimacy, and social anxiety. *Journal of Social and Clinical Psychology*, *23*, 89–103. doi: 10.1521/jscp.23.1.89.26987

Caspi, A., Elder, G. H., & Bem, D. J. (1988). Moving away from the world: Life-course patterns of shy children. *Developmental Psychology*, *24*, 824–831. doi: 10.1037/0012-1649.24.6.824

Cauffman, E., & Steinberg, L. (1996). Interactive effects of menarche, dating, and heterosocial involvement on dieting behavior in early adolescence. *Developmental Psychology*, *32*, 631–635. doi: 10.1037/0012-1649.32.4.631

Chorney, D. B., & Morris, T. L. (2008). The changing face of dating anxiety: Issues in assessment with special populations. *Clinical Psychology: Science and Practice*, *15*, 224–238. doi: 10.1111/j.1468-2850.2008.00132.x

Collins, W. (2003). More than myth: The developmental significance of romantic relationships during adolescence. *Journal of Research on Adolescence*, *13*, 1–24. doi: 10.1111/1532-7795.1301001

Collins, W. A., Welsh, D. P., & Furman, W. (2009). Adolescent romantic relationships. *Annual Review of Psychology*, *60*, 631–652. doi: 10.1146/annurev.psych.60.110707.163459

Compian, L., Gowen, L. K., & Hayward, C. (2004). Peripubertal girls' romantic and platonic involvement with boys: Associations with body image and depression symptoms. *Journal of Research on Adolescence*, *14*, 23–47. doi: 10.1111/j.1532-7795.2004.01401002.x

Connolly, J. A., Craig, W., Goldberg, A., & Pepler, D. (2004). Mixed gender groups, dating, and romantic relationships in early adolescence. *Journal of Research on Adolescence*, *14*, 185–207. doi: 10.1111/j.1532-7795.2004.01402003.x

Connolly, J., Furman, W., & Konarski, R. (2000). The role of peers in the emergence of heterosexual romantic relationships in adolescence. *Child Development*, *71*, 1395–1408. doi: 10.1111/1467-8624.00235

Connolly, J. A., & Goldberg, A. (1999). Romantic relationships in adolescence: The role of friends and peers in their emergence and development. In W. Furman, B. B. Brown, & C. Feiring (Eds.),

The development of romantic relationships in adolescence (pp. 266–290). New York, NY: Cambridge University Press.

Connolly, J. A., & Konarski, R. (1994). Peer self-concept in adolescence: Analysis of factor structure and of associations with peer experience. *Journal of Research on Adolescence*, *4*, 385–403. doi: 10.1207/s15327795jra0403_3

Connolly, J. A., Pepler, D. J., Craig, W., & Taradash, A. (2000). Dating experiences of bullies in early adolescence. *Child Maltreatment: Journal of the American Professional Society on the Abuse of Children*, *5*, 299–310. doi: 10.1177/1077559500005004002

Copeland, W. E., Shanahan, L., Costello, E. J., & Angold, A. (2009). Configurations of common childhood psychosocial risk factors. *Journal of Child Psychology and Psychiatry*, *50*, 451–459. doi: 10.1111/j.1469-7610.2008.02005.x

Costello, E. J., Egger, H. L., & Angold, A. (2005). The developmental epidemiology of anxiety disorders: Phenomenology, prevalence, and comorbidity. *Child and Adolescent Psychiatric Clinics of North America*, *14*, 631–648. doi: 10.1016/j.chc.2005.06.003

Daley, S. E., Burge, D., & Hammen, C. (2000). Borderline personality disorder symptoms as predictors of 4-year romantic relationship dysfunction in young women: Addressing issues of specificity. *Journal of Abnormal Psychology*, *109*, 451–460. doi: 10.1037/0021-843X.109.3.451

Daley, S. E., & Hammen, C. (2002). Depressive symptoms and close relationships during the transition to adulthood: Perspectives from dysphoric women, their best friends, and their romantic partners. *Journal of Consulting and Clinical Psychology*, *70*, 129–141. doi: 10.1037/0022-006X.70.1.129

Darcy, K., Davila, J., & Beck, J. G. (2005). Is social anxiety associated with both interpersonal avoidance and dependence? *Cognitive Therapy and Research*, *29*, 171–186. doi: 10.1007/s10608-005-3163-4

D'Augelli, A. R. (1988). The adolescent closet: Promoting the development of the lesbian or gay male teenager. *School Psychologist*, *42*, 2–3.

Davila, J. (2008). Depressive symptoms and adolescent romance: Theory, research, and implications. *Child Development Perspectives*, *2*, 26–31. doi: 10.1111/j.1750-8606.2008.00037.x

Davila, J., & Beck, J. G. (2002). Is social anxiety associated with impairment in close relationships? A preliminary investigation. *Behavior Therapy*, *33*, 427–446. doi: 10.1016/S0005-7894(02)80037-5

Davila, J., Bradbury, T. N., Cohan, C. L., & Tochluk, S. (1997). Marital functioning and depressive symptoms: Evidence for a stress generation model. *Journal of Personality and Social Psychology*, *73*, 849–861. doi: 10.1037/0022-3514.73.4.849

Davila, J., La Greca, A. M., Starr, L. R., & Landoll, R. (2010). Anxiety disorders in adolescence. In J. G. Beck (Ed.), *Interpersonal processes in the anxiety disorders: Implications for understanding psychopathology and treatment*, (pp. 97–124). Washington, DC: American Psychological Association.

Davila, J., & Rhoades, G. K. (in preparation). *Future directions for adolescent romantic relationship education*. Manuscript in preparation.

Davila, J., Steinberg, S. J., Kachadourian, L., Cobb, R., & Fincham, F. (2004). Romantic involvement and depressive symptoms in early and late adolescence: The role of preoccupied relational style. *Personal Relationships*, *11*, 161–178. doi: 10.1111/j.1475-6811.2004.00076.x

Davila, J., Steinberg, S. J., Ramsay, M., Stroud, C. B., Starr, L., & Yoneda, A. (2009). Assessing romantic competence in adolescence: The Romantic Competence Interview. *Journal of Adolescence*, *32*, 55–75. doi: 10.1016/j.adolescence.2007.12.001

Davila, J., Stroud, C. B., & Starr, L. (2014). Depression in couples and families. In I. Gotlib & C. Hammen (Eds.), *Handbook of Depression* (3rd ed.). (pp. 410–428). New York, NY: Guilford Press.

Davila, J., Stroud, C. B., Starr, L. R., Ramsay Miller, M., Yoneda, A., & Hershenberg, R. (2009). Romantic and sexual activities, parent-adolescent stress, and depressive symptoms among early adolescent girls. *Journal of Adolescence*, *32*, 909–924. doi: 10.1016/j.adolescence.2008.10.004

Deal, L. W., & Holt, V. L. (1998). Young maternal age and depressive symptoms: Results from the 1988 National Maternal and Infant Health Survey. *American Journal of Public Health*, *88*, 266–270. doi: 10.2105/AJPH.88.2.266

Dishion, T. J., & Owen, L. D. (2002). A longitudinal analysis of friendships and substance use: Bidirectional influence from adolescence to adulthood. *Developmental Psychology*, *38*, 480–491. doi: 10.1037/0012-1649.38.4.480

Dishion, T. J., & Patterson, G. R. (2006). The development and ecology of antisocial behavior in children and adolescents. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Risk, disorder, and adaptation* (Vol. 3, pp. 503–541). New York, NY: Wiley.

Donovan, J. E., Jessor, R., & Costa, F. M. (1988). Syndrome of problem behavior in adolescence: A replication. *Journal of Consulting and Clinical Psychology*, *56*, 762–765. doi: 10.1037/0022-006X.56.5.762

Downey, G., Bonica, C., & Rincon, C. (1999). Rejection sensitivity and adolescent romantic relationships. In W. Furman, B. B. Brown, & C. Feiring (Eds.), *The development of romantic relationships in adolescence* (pp. 148–174). New York, NY: Cambridge University Press.

Drouin, M., & Landgraff, C. (2012). Texting, sexting, and attachment in college students' romantic relationships. *Computers in Human Behavior*, *28*(2), 444–449. doi: 10.1016/j.chb.2011.10.015

Dunphy, D. (1963). The social structure of urban adolescent peer groups. *Sociometry*, *26*, 230–246. doi: 10.2307/2785909

Eakin, L., Minde, K., Hechtman, L., Ochs, E., Krane, E., Bouffard, R., Greenfield, B., & Looper, K. (2004). The marital and family functioning of adults with ADHD and their spouses. *Journal of Attention Disorders*, *8*, 1–10. doi: 10.1177/108705470400800101

Eaton, D. K., Davis, K. S., Barrios, L., Brener, N. D., & Noonan, R. K. (2007). Associations of dating violence victimization with lifetime participation, co-occurrence, and early initiation of risk behaviors among U.S. high school students. *Journal of Interpersonal Violence*, *22*, 585–602. doi: 10.1177/0886260506298831

Ehrensaft, M. K., Cohen, P., Brown, J., Smailes, E., Chen, H., & Johnson, J. (2003). Intergenerational transmission of partner violence: A 20-year prospective study. *Journal of Consulting and Clinical Psychology*, *71*, 741–753. doi: 10.1037/0022-006X.71.4.741

Ehrensaft, M. K., Moffitt, T. E., & Caspi, A. (2004). Clinically abusive relationships in an unselected birth cohort: Men's and women's participation and developmental antecedents. *Journal of Abnormal Psychology*, *113*, 258–270. doi: 10.1037/0021-843X.113.2.258

Ellison, N., Steinfield, C., & Lampe, C. (2007). The benefits of Facebook "friends": Social capital and college students' use of online social network sites. *Journal of Computer-Mediated Communication*, *12*, Article 1. Retrieved October 25, 2012, from http://jcmc.indiana.edu/vol12/issue4/ellison.html

Elliott, D. S., & Voss, H. L. (1974). *Delinquency and dropout*. Lexington, MA: Lexington Books.

Ellis, W. E., Chung-Hall, J., & Dumas, T. M. (2013). The role of peer group aggression in predicting adolescent dating violence and relationship quality. *Journal of Youth and Adolescence*, *42*, 487–499. doi: 10.1007/s10964-012-9797-0

Ellis, B. J., & Garber, J. (2000). Psychosocial antecedents of variation in girls' pubertal timing: Maternal depression, stepfather presence, and marital and family stress. *Child Development*, *71*, 485–501. doi: 10.1111/1467-8624.00159

Fagot, B. I., Pears, K. C., Capaldi, D. M., Crosby, L., & Leve, C. S. (1998). Becoming an adolescent father: Precursors and parenting. *Developmental Psychology*, *34*, 1209–1212. doi: 10.1037/0012-1649.34.6.1209

Farrington, D. P. (2003). Key results from the first forty years of the Cambridge Study in Delinquency Development. In T. P. Thornberry & M. D. Krohn (Eds.), *Taking stock of delinquency: An overview of findings from contemporary longitudinal studies* (pp. 137–183). New York, NY: Springer.

Feiring, C. (1999). Other-sex friendship networks and the development of romantic relationships in adolescence. *Journal of Youth and Adolescence*, *28*, 495–512. doi: 10.1023/A:1021621108890

Feiring, C., Simon, V. A., Cleland, C. M., & Barrett, E. P. (2013). Potential pathways from stigmatization and externalizing behavior to anger and dating aggression in sexually abused youth. *Journal of Clinical Child and Adolescent Psychology*, *42*, 309–322. doi: 10.1080/15374416.2012.736083

Fielder, R. L., & Carey, M. P. (2010). Predictors and consequences of sexual "hookups" among college students: A short-term prospective study. *Archives of Sexual Behavior*, *39*, 1105–1119. doi: 10.1007/s10508-008-9448-4

Flanagan, K. S., Erath, S. A., & Bierman, K. L. (2008). Unique associations between peer relations and social anxiety in early adolescence. *Journal of Clinical Child and Adolescent Psychology*, *37*, 759–769. doi: 10.1080/15374410802359700

Flanzer, J. P. (2005). Alcohol and other drugs are key causal agents of violence. In D. R. Loseke, R. J. Gelles, & M. M. Cavanaugh (Eds.), *Current controversies on family violence* (pp. 163–189). Thousand Oaks, CA: Sage.

Florsheim, P., McArthur, L., Hudak, C., Heavin, S., & Burrow-Sánchez, J. (2011). The Young Parenthood Program: Preventing violence between adolescent mothers and young fathers. *Journal of Couple and Relationship Therapy*, *10*, 117–134. doi: 10.1080/15332691.2011.562823

Florsheim, P., & Moore, D. R. (2008). Observing differences between healthy and unhealthy adolescent romantic relationships: Substance abuse and interpersonal process. *Journal of Adolescence*, *31*, 795–814. doi: 10.1016/j.adolescence.2007.09.005

Foshee, V. A., Bauman, K. E., Ennett, S. T., Suchindran, C., Benefield, T., & Linder, G. F. (2005). Assessing the effects of dating violence prevention program "Safe Dates" using random coefficient regression modeling. *Prevention Science*, *6*, 245–258. doi: 10.1007/s11121-005-0007-0

Foshee, V. A., Linder, G. F., Bauman, K. E., Langwick, S. A., Arriaga, X. B., Heath, J. L., . . . Bangdiwala, S. (1996). The Safe Dates Project: Theoretical basis, evaluation design, and selected baseline findings. *American Journal of Preventive Medicine*, *12*(Suppl. 5), 39–47.

Foshee, V. A., Reyes, H. L. M., Ennett, S. T., Suchindran, C., Mathias, J. P., Karriker-Jaffe, K., Bauman K. E., & Benefield, T. S. (2011). Risk and protective factors distinguishing profiles of adolescent peer and dating violence perpetration. *Journal of Adolescent Health*, *48*, 344–350. doi: 10.1016/j.jadohealth.2010.07.030

Frick, P. J. (1998). *Conduct disorders and severe antisocial behaviour*. New York, NY: Plenum.

Fritz, P. A., & O'Leary, K. D. (2004). Physical and psychological partner aggression across a decade: A growth curve analysis. *Violence and Victims*, *19*, 3–16. doi: 10.1891/088667004780842886

Furman, W., Low, S., & Ho, M. (2009). Romantic experience and psychosocial adjustment in middle adolescence. *Journal of Clinical Child and Adolescent Psychology*, *38*, 1–16. doi: 10.1080/15374410802575347

Furman, W., & Shaffer, L. (2003). The role of romantic relationships in adolescent development. In P. Florsheim (Ed.), *Adolescent romantic relations and sexual behavior: Theory, research, and practical implications* (pp. 3–22). Mahwah, NJ: Lawrence Erlbaum.

Furman W., & Simon V.A. (2008). Homophily and influence in adolescent romantic relationships. In M. Prinstein & K. A. Dodge (Eds.), *Understanding peer influence in children and adolescents* (pp. 203–24). New York, NY: Guilford.

Furman, W., Simon, V. A., Shaffer, L., & Bouchey, H. A. (2002). Adolescents' working models and styles for relationships with parents, friends, and romantic partners. *Child Development*, *73*, 241–255. doi: 10.1111/1467-8624.00403

Furman, W., & Wehner, E. A. (1994). Romantic views: Toward a theory of adolescent romantic relationships. In R. Montmayer, G. R. Adams, & G. P. Gullota (Eds.), *Advances in adolescent development: Personal relationships during adolescence* (Vol. 6, pp. 168–175). Thousand Oaks, CA: Sage.

Galliher, R. V., Rostosky, S. S., Welsh, D. P., & Kawaguchi, M. C. (1999). Power and psychological well-being in late adolescent romantic relationships. *Sex Roles*, *40*, 689–710. doi: 10.1023/A:1018804617443

Galvao, T. F., Silva, M. T., Zimmermann, I. R., Souza, K. M., Martins, S. S., & Pereira, M. G. (2014). Pubertal timing in girls and depression: A systematic review. *Journal of Affective Disorders*, *155*, 13–19. doi: 10.1016/j.jad.2013.10.034

Gavin, A. R., Lindhorst, T., & Lohr, M. (2011). The prevalence and correlates of depressive symptoms among adolescent mothers: Results from a 17-year longitudinal study. *Women and Health*, *51*, 525–545. doi: 10.1080/03630242.2011.606355

Glickman, A. R., & La Greca, A. M. (2004). The Dating Anxiety Scale for Adolescents: Scale development and associations with adolescent functioning. *Journal of Clinical Child and Adolescent Psychology*, *33*, 566–578. doi: 10.1207/s15374424jccp3303_14

Glover, J. A., Galliher, R. V., & Lamere, T. G. (2009). Identity development and exploration among sexual minority adolescents: Examination of a multidimensional model. *Journal of Homosexuality*, *56*, 77–101. doi: 10.1080/00918360802551555

Goins, L. B., Markey, C. N., & Gillen, M. M. (2012). Understanding men's body image in the context of their romantic relationships. *American Journal of Men's Health*, *6*(3), 240–248. doi: 10.1177/1557988311431007

Goodwin, P., McGill, B., & Chandra, A. (2009). *Who marries and when? Age at first marriage in the United States: 2002*. NCHS Data Brief, No. 19. Hyattsville, MD: National Center for Health Statistics.

Gotlib, I. H., Lewinsohn, P. M., & Seeley, J. R. (1998). Consequences of depression during adolescence: Marital status and marital functioning in early adulthood. *Journal of Abnormal Psychology*, *107*, 686–690. doi: 10.1037/0021-843X.107.4.686

Gottman, J. M., McCoy, K., Coan, J., & Collier, H. (1996). The Specific Affect Coding System (SPAFF). In J. M. Gottman (Ed.), *What predicts divorce? The measures*. Hillsdale, NJ: Lawrence Erlbaum.

Grant, D. M., Beck, J. G., Farrow, S. M., & Davila, J. (2007). Do interpersonal features of social anxiety influence the development of depressive symptoms? *Cognition and Emotion*, *21*, 646–663. doi: 10.1080/02699930600713036

Greco, L. A., & Morris, T. L. (2005). Factors influencing the link between social anxiety and peer Acceptance: Contributions of social skills and close friendships during middle childhood. *Behavior Therapy*, *36*, 197–205. doi: 10.1016/S0005-7894(05)80068-1

Grello, C. M., Welsh, D. P., Harper, M. S., & Dickson, J. W. (2003). Dating and sexual relationship trajectories and adolescent functioning. *Adolescent and Family Health*, *3*, 103–112.

Ha, T., Overbeek, G., Cillessen, A. H. N., & Engels, R. C. M. E., (2012). A longitudinal study of the associations among adolescent conflict resolution styles, depressive symptoms, and romantic relationship longevity. *Journal of Adolescence*, *35*, 1247–1254. doi: 10.1016/j.adolescence.2012.04.009

Hankin, B. L., Mermelstein, R., & Roesch, L. (2007). Sex differences in adolescent depression: Stress exposure and reactivity models. *Child Development*, *78*, 279–295. doi: 10.1111/j.1467-8624.2007.00997.x

Harper, M. S., & Welsh, D. P. (2007). Keeping quiet: Self-silencing and its association with relational and individual functioning among adolescent romantic couples. *Journal of Social and Personal Relationships*, *24*, 99–116. doi: 10.1177/0265407507072601

Harter, S. (1999). *The construction of the self: A developmental perspective*. New York, NY: Guilford Press.

Haydon, A. A., & Halpern, C. (2010). Older romantic partners and depressive symptoms during adolescence. *Journal of Youth and Adolescence*, *39*, 1240–1251. doi: 10.1007/s10964-010-9539-0

Haynie, D. L., Giordano, P. C., Manning, W. D., & Longmore, M. A. (2005). Adolescent romantic relationships and delinquency involvement. *Criminology*, *43*, 177–210. doi: 10.1111/j.0011-1348.2005.00006.x

Herbert, K. R., Fales, J., Nangle, D. W., Papadakis, A. A., & Grover, R. L. (2013). Linking social anxiety and adolescent romantic relationship functioning: Indirect effects and the importance of peers. *Journal of Youth and Adolescence*, *42*, 1708–1720. doi: 10.1007/s10964-012-9878-0

Herrenkohl, T. I., Kosterman, R., Mason, W. A., & Hawkins, J. D. (2007). Youth violence trajectories and proximal characteristics of intimate partner violence. *Violence and Victims*, *22*, 259–274. doi: 10.1891/088667007780842793

Herrick, A. L., Marshal, M. P., Smith, H. A., Sucato, G., & Stall, R. D. (2011). Sex while intoxicated: A meta-analysis comparing heterosexual and sexual minority youth. *Journal of Adolescent Health*, *48*, 306–309. doi: 10.1016/j.jadohealth.2010.07.008

Hershenberg, R., & Davila, J. (2010). Depressive symptoms and sexual experiences among early adolescent girls: Interpersonal avoidance as a moderator. *Journal of Youth and Adolescence*, *39*, 967–976. doi: 10.1007/s10964-009-9446-4

Hicks, C. W., DeMarsh, S., Singh, H., Gillespie, L. A., Worley, S., & Rome, E. S. (2013). Knowledge about various contraceptive methods in young women with and without eating disorders. *International Journal of Eating Disorders*, *46*, 171–176. doi: 10.1002/eat.22048

Hudson, J. I., Hiripi, E., Pope Jr.,, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *1*, 348–358. doi: 10.1016/j.biopsych.2006.03.040

Igartua, K. J., Gill, K., & Montoro, R. (2003). Internalized homophobia: a factor in depression, anxiety, and suicide in the gay and lesbian population. *Canadian Journal of Community Mental Health*, *22*(2), 15–30.

Jager, J., & Davis-Kean, P. (2011). Same-sex sexuality and adolescent psychological well-being: The influence of sexual orientation, early reports of same-sex attraction, and gender. *Self and Identity*, *10*(4), 417–444. doi: 10.1080/15298861003771155

Joyner, K., & Udry, J. R. (2000). You don't bring me anything but down: Adolescent romance and depression. *Journal of Health and Social Behavior*, *41*, 369–391. doi: 10.2307/2676292

Juarez, L., & Pritchard, M. (2012). Body dissatisfaction: Commitment, support, and trust in romantic relationships. *Journal of Human Behavior in the Social Environment*, *22*(2), 188–200. doi: 10.1080/10911359.2012.647478

Kandel, D. (1978a). Homophily, selection, and socialization in adolescent friendships. *American Journal of Sociology*, *84*, 427–436. doi: 10.1086/226792

Kandel, D. B. (1978b). Similarity in real-life adolescent friendship pairs. *Journal of Personality and Social Psychology*, *36*(3), 306–312. doi: 10.1037/0022-3514.36.3.306

Kashdan, T. B., Volkmann, J. R., Breen, W. E., & Han, S. (2007). Social anxiety and romantic relationships: The costs and benefits of negative emotion expression are context-dependent. *Journal of Anxiety Disorders*, *21*, 475–492. doi: 10.1016/j.janxdis.2006.08.007

Katz, S. J., Hammen, C. L., & Brennan, P. A. (2013). Maternal depression and the intergenerational transmission of relational impairment. *Journal of Family Psychology*, *27*, 86–95. doi: 10.1037/a0031411

Keenan-Miller, D., Hammen, C., & Brennan, P. (2007). Adolescent psychosocial risk factors for severe intimate partner violence in young adulthood. *Journal of Consulting and Clinical Psychology*, *75*, 456–463. doi: 10.1037/0022-006X.75.3.456

Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 617–627. doi: 10.1001/archpsyc.62.6.617

Kessler, R. C., Crum, R. M., Warner, L. A., Nelson, C. B., Schulenberg, J., & Anthony, J. C. (1997). Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Archives of General Psychiatry*, *54*, 313–321. doi: 10.1001/archpsyc.1997.01830160031005

Kessler, R. C., Walters, E. E., & Forthofer, M. S. (1998). The social consequences of psychiatric disorders, III: Probability of marital stability. *American Journal of Psychiatry*, *155*, 1092–1096.

Kim, E. Y., Miklowitz, D. J., Biuckians, A., & Mullen, K. (2007). Life stress and the course of early-onset bipolar disorder. *Journal of Affective Disorders*, *99*, 37–44. doi: 10.1016/j.jad.2006.08.022

Kim, H. K., & Capaldi, D. M. (2004). The association of antisocial behavior and depressive symptoms between partners and risk for aggression in romantic relationships. *Journal of Family Psychology*, *18*, 82–96. doi: 10.1037/0893-3200.18.1.82

Kim, H. K., Capaldi, D. M., & Crosby, L. (2007). Generalizability of Gottman and colleagues' affective process models of couples' relationship outcomes. *Journal of Marriage and Family*, *69*, 55–72. doi: 10.1111/j.1741-3737.2006.00343.x

Kim, H. K., Laurent, H. K., Capaldi, D. M., & Feingold, A. (2008). Men's aggression toward women: A 10-year panel study. *Journal of Marriage and Family*, *70*, 1169–1187. doi: 10.1111/j.1741-3737.2008.00558.x

Kim, H. K., Pears, K. C., Capaldi, D. M., & Owen, L. D. (2009). Emotion dysregulation in the intergenerational transmission of romantic relationship conflict. *Journal of Family Psychology*, *23*, 585–595. doi: 10.1037/a0015935

Koch, P. B. (1993). Promoting healthy sexual development during early adolescence. In R. Lerner (Ed.), *Early adolescence: Perspectives on research, policy, and intervention* (pp. 293–307). Hillsdale, NJ: Lawrence Erlbaum.

Kohlberg, L., Ricks, D., & Snarey, J. (1984). Childhood development as a predictor of adaptation in adulthood. *Genetic Psychology Monographs*, *110*, 91–172.

Kreager, D. A., & Haynie, D. L. (2011). Dangerous liaisons? Dating and drinking diffusion in adolescent peer networks. *American Sociological Review*, *76*, 737–763. doi: 10.1177/0003122411416934

Krueger, R. F., Moffitt, T. E., Caspi, A., Bleske, A., & Silva, P. A. (1998). Assortative mating for antisocial behavior: Developmental and methodological implications. *Behavior Genetics*, *28*, 173–186. doi: 10.1023/A:1021419013124

Kupersmidt, J. B., DeRosier, M. E., & Patterson, C. P. (1995). Similarity as the basis of children's friendships: The roles of sociometric status, aggressive and withdrawn behavior, academic achievement, and demographic characteristics. *Journal of Social and Personal Relationships*, *12*, 439–457. doi: 10.1177/0265407595123007

Kuttler, A., & La Greca, A. M. (2004). Linkages among adolescent girls' romantic relationships, best friendships, and peer networks. *Journal of Adolescence*, *27*, 395–414. doi: 10.1016/j.adolescence.2004.05.002

Kuttler, A. F., La Greca, A. M., & Prinstein, M. J. (1999). Adolescents' close friendships: Same- versus cross-sex friends. *Journal of Research in Adolescence*, *9*, 339–366. doi: 10.1207/s15327795jra0903_5

Kvalem, I. L., von Soest, T., Træen, B., & Singsaas, K. (2011). Body evaluation and coital onset: A population-based longitudinal study. *Body Image*, *8*(2), 110–118. doi: 10.1016/j.bodyim.2011.02.001

La Greca, A. M. (1999). The Social Anxiety Scales for Children and Adolescents. *Behavior Therapist*, *22*, 133–136.

La Greca, A. M., Davila, J., & Siegel, R. (2008). Friendships, romantic relationships, and depression. In N. Allen & L. Sheeber (Eds.), *Adolescent emotional development and the emergence of depressive disorders* (pp. 318–336). New York, NY: Cambridge University Press.

La Greca, A. M., Ehrenreich-May, J., & Mufson, L. (2013, November). *Peer victimization processes contributing to adolescents' internalizing symptoms: Current findings and intervention development*. Paper presented at the symposium Association for Behavioral and Cognitive Therapies symposium, Nashville, TN.

La Greca, A. M., & Harrison, H. M. (2005). Adolescent peer relations, friendships, and romantic relationships: Do they predict social anxiety and depression? *Journal of Clinical Child and Adolescent Psychology*, *34*, 49–61. doi: 10.1207/s15374424jccp3401_5

La Greca, A. M., & Lai, B. S. (2014). The role of peer relationships in youth psychopathology. In B. C. Chu & J. Ehrenreich-May (Eds.), *Transdiagnostic treatments for children and adolescents* (pp. 111–137). New York, NY: Guilford Press.

La Greca, A. M., & Landoll, R. R. (2010, November). *Is dating hazardous to health?* Paper presented at the Association for Behavioral and Cognitive Therapies, San Francisco, CA.

La Greca, A. M., & Landoll, R. R. (2011). Peer influences in the development and maintenance of anxiety disorders. In W. K. Silverman & A. Field (Eds.), *Anxiety disorders in children and adolescents: Research, assessment, and intervention* (2nd ed.). London, UK: Cambridge University Press.

La Greca, A. M., & Lopez, N. (1998). Social anxiety among adolescents: Linkages with peer relations and friendships. *Journal of Abnormal Child Psychology*, *26*, 83–94. doi: 10.1023/A:1022684520514

La Greca, A. M., & Mackey, E. R. (2007). Adolescents' anxiety in dating situations: Do friends and romantic partners contribute? *Journal of Clinical Child and Adolescent Psychology*, *34*, 522–533. doi: 10.1080/15374410701662097

Lampe, L., Slade, T., Issakidis, C., & Andrews, G. (2003). Social phobia in the Australian National Survey of Mental Health and Well-Being (NSMHWB). *Psychological Medicine*, *33*, 637–646. doi: 10.1017/S0033291703007621

Landoll, R. R., La Greca, A. M., & Lai, B. (2013). Aversive peer experiences on social networking sites: Development of the Social Networking-Peer Experiences Questionnaire. *Journal of Research on Adolescence*, *23*, 695–705. doi: 10.1111/jora.12022

Langhinrichsen-Rohling, J., Misra, T. A., Selwyn, C., & Rohling, M. L. (2012). Rates of bidirectional versus unidirectional intimate partner violence across samples, sexual orientations, and race/ethnicities: A comprehensive review. *Partner Abuse*, *3*, 199–230. doi: 10.1891/1946-6560.3.2.199

Larson, R. W., Clore, G. L., & Wood, G. A. (1999). The emotions of romantic relationships: Do they wreak havoc on adolescents? In W. Furman, B. Brown, & C. Feiring (Eds.), *The development of romantic relationships in adolescence* (pp. 19–49). New York, NY: Cambridge Press.

Larson, R. W., & Richards, M. H. (1998). Waiting for the weekend: Friday and Saturday nights as the emotional climax of the week. In A. C. Crouter & R. W. rLarson (Eds.) *Temporal rhythms in adolescence: Clocks, calendars, and the coordination of daily life: New Directions for Child Development* (No. 82, pp. 37–51). San Francisco, CA: Jossey-Bass.

Laursen, B. (1995). Conflict and social interaction in adolescent relationships. *Journal of Research on Adolescence*, *5*, 55–70. doi: 10.1207/s15327795jra0501_3

Laursen, B. (1996). Closeness and conflict in adolescent peer relationships: Interdependence with friends and romantic partners. In W. M. Bukowski, A. F. Newcomb, & W. W. Hartup (Eds.), *The company they keep: Friendships in childhood and adolescence* (pp. 186–210). New York, NY: Cambridge University Press.

Laursen, B., & Williams, V. (1997). Perceptions of interdependence and closeness in family and peer relationships among adolescents with and without romantic partners. In S. Shulman & W. A. Collins (Eds.), *Romantic relationships in adolescence: New directions for child development* (No. 78, pp. 3–20). San Francisco, CA: Jossey-Bass.

Leadbeater, B. J., & Linares, O. (1992). Depressive symptoms in Black and Puerto Rican adolescent mothers in the first 3 years postpartum. *Development and Psychopathology*, *4*, 451–468. doi: 10.1017/S0954579400000894

Lee, Y., Fagan, J., & Chen, W. (2012). Do late adolescent fathers have more depressive symptoms than older fathers? *Journal of Youth and Adolescence*, *41*, 1366–1381. doi: 10.1007/s10964-011-9717-8

Lehrer, J. A., Shrier, L. A., Gortmaker, S., & Buka, S. (2006). Depressive symptoms as a longitudinal predictor of sexual risk behaviors among U.S. middle and high school students. *Pediatrics*, *118*, 189–200. doi: 10.1542/peds.2005-1320

Lenhart, A., Ling, R., Campbell, S., & Purdell, K. (2010, April). *Teens and mobile phones*. Pew Research Center's Internet and American Life Project. Retrieved from http://pewinternet.org/Reports/2010/Teens-and-Mobile-Phones.aspx

Lenhart, A., Purcell, K., Smith, A., & Zickuhr, K. (2010). Social media and mobile internet use among teens and young adults. *Pew Internet and American Life Project*, 1–51. Retrieved from http://www.pewinternet.org/~/media//Files/Reports/2010/PIP_Social_Media_and_Young_Adults_Report_Final_with_toplines.pdf

Lewinsohn, P. M., Hops, H., Roberts, R. E., Seeley, J. R., & Andrews, J. A. (1993). Adolescent psychopathology: I. Prevalence and incidence of depression and other *DSM-III-R* disorders in high school students. *Journal of Abnormal Psychology*, *102*, 133–144. doi: 10.1037/0021-843X.102.4.517

Lewinsohn, P. M., Rohde, P., Seeley, J. R., Klein, D. J., & Gotlib, I. H. (2003). Psychosocial functioning of young adults who have experienced and recovered from major depressive disorder during adolescence. *Journal of Abnormal Psychology*, *112*, 353–363. doi: 10.1037/0021-843X.112.3.353

Lindhorst, T., & Oxford, M. (2008). The long-term effects of intimate partner violence on adolescent mothers' depressive symptoms. *Social Science and Medicine*, *66*, 1322–1333. doi: 10.1016/j.socscimed.2007.11.045

Little, K. C., Welsh, D. P., Darling, N., & Holmes, R. M. (2011). Brief report: "I can't talk about it:" Sexuality and self-silencing as interactive predictors of depressive symptoms in adolescent dating couples. *Journal of Adolescence*, *34*, 789–794. doi: 10.1016/j.adolescence.2010.04.006

Little, M., Handley, E., Leuthe, E., & Chassin, L. (2009). The impact of parenthood on alcohol consumption trajectories: Variations as a function of timing of parenthood, familial alcoholism, and gender. *Development and Psychopathology*, *21*, 661–682. doi: 10.1017/S0954579409000352

Loftus, J., Kelly, B. C., & Mustillo, S. A. (2011). Depressive symptoms among adolescent girls in relationships with older partners: Causes and lasting effects? *Journal of Youth and Adolescence*, *40*, 800–813. doi: 10.1007/s10964-010-9589-3

Lussier, P., Farrington, D. P., & Moffitt, T. E. (2009). Is the antisocial child father of the abusive man? A 40-year prospective longitudinal study on the developmental antecedents of intimate partner violence. *Criminology*, *47*, 471–780. doi: 10.1111/j.1745-9125.2009.00160.x

Maas, C. D., Fleming, C. B., Herrenkohl, T. I., & Catalano, R. F. (2010). Childhood predictors of teen dating violence victimization. *Violence and Victims*, *25*, 131–149. doi: 10.1891/0886-6708.25.2.131

Madsen, S. D., & Collins, W. (2011). The salience of adolescent romantic experiences for romantic relationship qualities in young adulthood. *Journal of Research on Adolescence*, *21*, 789–801. doi: 10.1111/j.1532-7795.2011.00737.x

Magdol, L., Moffitt, T. E., Caspi, A., & Silva, P. A. (1998). Developmental antecedents of partner abuse: A prospective longitudinal study. *Journal of Abnormal Psychology*, *107*, 375–389. doi: 10.1037/0021-843X.107.3.375

Magid, L. (2009). Youth cell phones for "dating abuse." Safekids.com. Retrieved from http://www.safekids.com/2009/12/08/youth-use-cell-phones-for-dating-abuse/

Manning, W. D., Giordano, P. C., & Longmore, M. A. (2006). Hooking up: The relationship contexts of 'nonrelationship' sex. *Journal of Adolescent Research*, *21*, 459–483. doi: 10.1177/0743558406291692

Marceau, K., Ram, N., Houts, R. M., Grimm, K. J., & Susman, E. J. (2011). Individual differences in boys' and girls' timing and tempo of puberty: Modeling development with nonlinear growth models. *Developmental Psychology*, *47*, 1389–1409. doi: 10.1037/a0023838

Marín, B. V., Kirby, D., Hudes, E. S., Coyle, K. K., & Gómez, C. A. (2006), Boyfriends, girlfriends and teenagers' risk of sexual involvement. *Perspectives on Sexual and Reproductive Health*, *38*, 76–83. doi: 10.1363/3807606

Markey, C. N., & Markey, P. M. (2006). Romantic relationships and body satisfaction among young women. *Journal of Youth and Adolescence (Special Issue on Body Image)*, *35*, 256–264. doi: 10.1007/s10964-005-9013-6

Masten, A. S., Coatsworth, J. D., Neemann, J., & Gest, S. D. (1995). The structure and coherence of competence from childhood through adolescence. *Child Development*, *66*, 1635–1659. doi: 10.2307/1131901

Mays, V. M., & Cochran, S. D. (2001). Mental health correlates of perceived discrimination among lesbian, gay, and bisexual adults in the United States. *American Journal of Public Health*, *91*(11), 1869–1876. doi: 10.2105/AJPH.91.11.1869

Melfsen, S., Osterlow, J., & Florin, I. (2000). Deliberate emotional expression of socially anxious children and their mothers. *Journal of Anxiety Disorders*, *14*, 249–261. doi: 10.1016/S0887-6185(99)00037-7

Mendle, J., Ferrero, J., Moore, S. R., & Harden, K. (2013). Depression and adolescent sexual activity in romantic and nonromantic relational contexts: A genetically-informative sibling comparison. *Journal of Abnormal Psychology*, *122*, 51–63. doi: 10.1037/a0029816

Merikangas, K. R., & Avenevoli, S. (2002). Epidemiology of mood and anxiety disorders in children and adolescents. In M. T. Tsaung & M. Tohen (Eds.), *Textbook in psychiatric epidemiology* (2nd ed., pp. 657–704). New York, NY: Wiley-Liss.

Merikangas, K. R., Avenevoli, S., Costello, E. J., Koretz, D., & Kessler, R. C. (2009). National Comorbidity Survey Replication Adolescent Supplement (NCS-A): I. *Background and measures. Journal of the American Academy of Child and Adolescent Psychiatry*, *48*, 367–379. doi: 10.1097/CHI.0b013e31819996f1

Merikangas, K. R., He, J., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L. . . . Swendsen, J. (2010). Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Survey Replication—Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*, 980–989. doi: 10.1016/j.jaac.2010.05.017

Merikangas, K. R., & Knight, E. (2009). The epidemiology of depression in adolescents. In S. Nolen-Hoeksema & L. M. Hilt (Eds.), *Handbook of depression in adolescents* (pp. 53–74). New York, NY: Taylor and Francis Group.

Merikangas, K. R., Nakamura, E. F., & Kessler, R. C. (2009). Epidemiology of mental disorders in children and adolescents. *Dialogues in Clinical Neuroscience*, *11*, 7–20.

Meyer, I. H., Dietrich, J., & Schwartz, S. (2008). Lifetime prevalence of mental disorders and suicide attempts in diverse lesbian, gay, and bisexual populations. *American Journal of Public Health*, *98*(6), 1004–1006. doi: 10.2105/AJPH.2006.096826

Milan, S., Kershaw, T. S., Lewis, J., Westdahl, C., Rising, S., Patrikios, M., & Ickovics, J. R. (2007). Cargiving history and prenatal depressive symptoms in low-income adolescent and young adult women: Moderating and mediating effects. *Psychology of Women Quarterly*, *31*, 241–251. doi: 10.1111/j.1471-6402.2007.00367.x

Miller-Johnson, S., Winn, D. C., Coie, J. D., Malone, P. S., & Lochman, J. (2004). Risk factors for adolescent pregnancy reports among African American males. *Journal of Research on Adolescence*, *14*, 471–495. doi: 10.1111/j.1532-7795.2004.00083.x

Miller-Johnson, S., Winn, D. M., Coie, J., Maumary-Gremaud, A., Hyman, C., Terry, R., & Lochman, J. (1999). Motherhood during the teen years: A developmental perspective on risk factors for childbearing. *Development and Psychopathology*, *11*, 85–100. doi: 10.1017/S0954579499001960

Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. A. (2001). Sex, antisocial behavior, and mating: Mate selection and early childbearing. In T. E. Moffitt, A. Caspi, M. Rutter, & P. A. Silva (Eds.), *Sex differences in antisocial behavior: Conduct disorder, delinquency, and violence in the Dunedin Longitudinal Study* (pp. 184–197). New York, NY: Cambridge University Press.

Moffitt, T. E., Krueger, R. F., Caspi, A., & Fagan, J. (2000). Partner abuse and general crime: How are they the same? How are they different? *Criminology*, *38*, 199–232. doi: 10.1111/**j**.1745-9125.2000.tb00888.x

Molloy, Lauren E., Gest, Scott D., Feinberg, Mark E., & Osgood, D. Wayne (2014). Emergence of Mixed-Sex Friendship Groups During Adolescence: Developmental Associations with Substance Use and Delinquency. In *Developmental Psychology*, Vol. 50(11), Nov, pp. 2449–2461.

Monahan, K. C., & Lee, J. M. (2008). Adolescent sexual activity: Links between relational context and depressive symptoms. *Journal of Youth and Adolescence*, *37*, 917–927. doi: 10.1007/s10964-007-9256-5

Monroe, S. M., Rohde, P., Seeley, J. R., & Lewinsohn, P. M. (1999). Life events and depression in adolescence: Relationship loss as a prospective risk factor for first onset of major depressive disorder. *Journal of Abnormal Psychology*, *108*, 606–614. doi: 10.1037/0021-843X.108.4.606

Moore, D. R., & Florsheim, P. (2001). Interpersonal processes and psychopathology among expectant and nonexpectant adolescent couples. *Journal of Consulting and Clinical Psychology*, *69*, 101–113. doi: 10.1037/0022-006X.69.1.101

Moore, D. R., Florsheim, P., & Butner, J. (2007). Interpersonal behavior, psychopathology, and relationship outcomes among adolescent mothers and their partners. *Journal of Clinical Child and Adolescent Psychology*, *26*, 541–556. doi: 10.1080/15374410701662709

Morrison, K., Doss, B. D., & Perez, M. (2009). Body image and disordered eating in romantic relationships. *Journal of Social and Clinical Psychology*, *28*, 281–306. doi: 10.1521/jscp.2009.28.3.281

Mufson, L., Dorta, K.P., Wickramaratne, P., Nomura, Y., Olfson, M., & Weissman, M. M. (2004). A randomized effectiveness trial of interpersonal psychotherapy for depressed adolescents. *Archives of General Psychiatry*, *61*, 577–584. doi: 10.1001/archpsyc.61.6.577

National Campaign to Prevent Teen and Unplanned Pregnancy. (2008). *Sex and tech: Results from a survey of teens and young adults*. Washington, DC: National Campaign to Prevent Teen and Unplanned Pregnancy.

Needham, B. L. (2012). Sexual attraction and trajectories of mental health and substance use during the transition from adolescence to adulthood. *Journal of Youth and Adolescence*, *41*, 179–190. doi: 10.1007/s10964-011-9729-4

Nieder, T., & Seiffge-Krenke, I. (2001). Coping with stress in different phases of romantic development. *Journal of Adolescence*, *24*, 297–311. doi: 10.1006/jado.2001.0407

Newcomb, M. D. (1987). Consequences of teenage drug use: The transition from adolescence to young adulthood. *Drugs and Society*, *1*(4), 25–60. doi: 10.1300/J023v01n04_03

Nolen-Hoeksema, S., & Girgus, J. S. (1994). The emergence of gender differences in depression during adolescence. *Psychological Bulletin*, *115*(3), 424–443. doi: 10.1037/0033-2909.115.3.424

O'Keefe, M. (1997). Predictors of dating violence among high school students. *Journal of Interpersonal Violence*, *12*, 546–568. doi: 10.1177/088626097012004005

O'Leary, K. D., & Maiuro, R. D. (2001). *Psychological abuse in violent domestic relations*. New York, NY: Springer.

Ott, M. Q., Wypij, D., Corliss, H. L., Rosario, M., Reisner, S. L., Gordon, A. R., & Austin, S. (2013). Repeated changes in reported sexual orientation identity linked to substance use behaviors in youth. *Journal of Adolescent Health*, *52*, 465–472. doi: 10.1016/j.jadohealth.2012.08.004

Owen, J., Fincham, F. D., & Moore, J. (2011). Short-term prospective study of hooking up among college students. *Archives of Sexual Behavior*, *40*, 331–341. doi: 10.1007/s10508-010-9697-x

Oxford, M. L., Gilchrist, L. D., Lohr, M., Gillmore, M., Morrison, D. M., & Spieker, S. J. (2005). Life course heterogeneity in the transition from adolescence to adulthood among adolescent mothers. *Journal of Research on Adolescence*, *15*, 479–504. doi: 10.1111/j.1532-7795.2005.00108.x

Ozer, E. L., Tschann, J. M., Pasch, L. A., & Flores, E. (2004). Violence perpetration across peer and partner relationships: Co-occurrence and longitudinal patterns among adolescents. *Journal of Adolescent Health*, *34*, 64–71. doi: 10.1016/j.jadohealth.2002.12.001

Patterson, G. R., Capaldi, D. M., & Bank, L. (1991). An early starter model predicting delinquency. In D. J. Pepler & K. H. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 139–168). Hillsdale, NJ: Lawrence Erlbaum.

Paxton, S. J., Wertheim, E. H., Gibbons, K., Szmukler, G. I., Hillier, L., & Petrovich, J. L. (1991). Body image satisfaction, dieting beliefs and weight loss behaviours in adolescent girls and boys. *Journal of Youth and Adolescence*, *20*, 361–379.

Pearson, M. R., Kholodkov, T., Henson, J. M., & Impett, E. A. (2012). Pathways to early coital debut for adolescent girls: A recursive partitioning analysis. *Journal of Sex Research*, *49*(1), 13–26. doi: 10.1080/00224499.2011.565428

Poulin, F., & Pedersen, S. (2007). Developmental changes in gender composition of friendship networks in adolescent girls and boys. *Developmental Psychology*, *43*, 1484–1496. doi: 10.1037/0012-1649.43.6.1484

Presnell, K., Bearman, S., & Stice, E. (2004). Risk factors for body dissatisfaction in adolescent boys and girls: A prospective study. *International Journal of Eating Disorders*, *36*(4), 389–401. doi: 10.1002/eat.20045

Quatman, T., Sampson, K., Robinson, C., & Watson, C. M. (2001). Academic, motivational, and emotional correlates of adolescent dating. *Genetic, Social, and General Psychology Monographs*, *127*(2), 211–234.

Reid, J. B., Eddy, J. M., Fetrow, R. A., & Stoolmiller, M. (1999). Description and immediate impacts of a preventative intervention for conduct problems. *American Journal of Community Psychology*, *24*, 483–517.

Reyes, H. L. M., Foshee, V. A., Bauer, D. J., & Ennett, S. T. (2011). The role of heavy alcohol use in the developmental process of desistance in dating aggression during adolescence. *Journal of Abnormal Child Psychology*, *39*, 239–250. doi: 10.1007/s10802-010-9456-4

Rhule-Louie, D. M., & McMahon, R. J. (2007). Problem behavior and romantic relationships: Assortative mating, behavior contagion, and desistance. *Clinical Child and Family Psychology Review*, *10*, 53–100. doi: 10.1007/s10567-006-0016-y

Richards, M. H., Crowe, P. A., Larson, R., & Swarr, A. (1998). Developmental patterns and gender differences in the experience of peer companionship during adolescence. *Child Development*, *69*, 154–163. doi: 10.2307/1132077

Rizzo, C. J., Daley, S. E., & Gunderson, B. H. (2006). Interpersonal sensitivity, romantic stress, and the prediction of depression: A study of inner-city, minority adolescent girls. *Journal of Youth and Adolescence*, *35*, 469–478. doi: 10.1007/s10964-006-9047-4

Rudolph, K. D. (2009). Adolescent depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed., pp. 444–466). New York, NY: Guilford Press.

Russell, S. T., & Consolacion, T. B. (2003). Adolescent romance and emotional health in the United States: Beyond binaries. *Journal of Clinical Child and Adolescent Psychology*, *32*, 499–508. doi: 10.1207/S15374424JCCP3204_2

Russell, S. T., Driscoll, A. K., & Truong, N. (2002). Adolescent same-sex romantic attractions and relationships: Implications for substance use and abuse. *American Journal of Public Health*, *92*, 198–202. doi: 10.2105/AJPH.92.2.198

Rutter, M. (1990). *Psychosocial resilience and protective mechanisms*. New York, NY: Cambridge University Press.

Ruuska, J., Kaltiala-Heino, R., Koivisto, A. M., & Rantanen, P. (2003). Puberty, sexual development, and eating disorders in adolescent outpatients. *European Child and Adolescent Psychiatry*, *12*, 114–120. doi: 10.1007/s00787-003-0340-4

Sampson, R. J., & Laub, J. H. (2005). A life-course view of the development of crime. *The ANNALS of the American Academy of Political and Social Science*, *602*, 12–45. doi: 10.1177/0002716205280075

Sanchez, D. T., & Kwang, T. (2007). When the relationship becomes her: Revisiting women's body concerns from a relationship contingency perspective. *Psychology of Women Quarterly*, *31*(4), 401–414. doi: 10.1111/j.1471-6402.2007.00389.x

Savin-Williams, R. C. (1994). Dating those you can't love and loving those you can't date. In R. Montemayor, G. R. Adams, & T. P. Gullatta (Eds.), *Advances in adolescent development Personal relationships during adolescence* (Vol. 6, pp. 168–195). Thousand Oaks, CA: Sage.

Schnurr, M. P., & Lohman, B. J. (2008). How much does school matter? An examination of adolescent dating violence perpetration. *Journal of Youth and Adolescence*, *37*, 266–283. doi: 10.1007/s10964-007-9246-7

Schooler, D. (2013). Early adolescent body image predicts subsequent condom use behavior among girls. *Sexuality Research and Social Policy: A Journal of the NSRC*, *10*(1), 52–61. doi: 10.1007/s13178-012-0099-9

Schooler, D., Impett, E. A., Hirschman, C., & Bonem, L. (2008). A mixed-method exploration of body image and sexual health among adolescent boys. *American Journal of Men's Health*, *2*(4), 322–339. doi: 10.1177/1557988308318508

Schumacher, J. A., Feldbau-Kohn, S., Slep, A. M. S., & Heyman, R. E. (2001). Risk factors for male-to-female partner physical abuse. *Aggression and Violent Behavior*, *6*, 281–352. doi: 10.1016/S1359-1789(00)00027-6

Seiffge-Krenke, I., & Lang, J. (2002, April). *Forming and maintaining romantic relations from early adolescence to young adulthood: Evidence of a developmental sequence*. Paper presented at the conference of the Society for Research on Adolescence, New Orleans, LA.

Shortt, J. W., Capaldi, D. M., Dishion, T. J., Bank, L., & Owen, L. D. (2003). The role of adolescent peers, romantic partners, and siblings in the emergence of the adult antisocial lifestyle. *Journal of Family Psychology*, *17*, 521–533. doi: 10.1037/0893-3200.17.4.521

Shortt, J. W., Capaldi, D. M., Kim, H. K., Kerr, D. C. R., Owen, L. D., & Feingold, A. (2012). Stability of intimate partner violence by men across 12 years in young adulthood: Effects of relationship transitions. *Prevention Science*, *13*, 360–369. doi: 10.1007/s11121-011-0202-0

Shortt, J. W., Capaldi, D. M., Kim, H. K., & Laurent, H. K. (2010). The effects of intimate partner violence on relationship satisfaction over time for young at risk couples: The moderating role of observed negative and positive affect. *Partner Abuse*, *1*, 131–151. doi: 10.1891/1946-6560.1.2.131

Shortt, J. W., Capaldi, D. M., Kim, H. K., & Owen, L. D. (2006). Relationship separation for young, at-risk couples: Prediction from dyadic aggression. *Journal of Family Psychology*, *20*, 624–631. doi: 10.1037/0893-3200.20.4.624

Shulman, S., Davila, J., & Shachar-Shapira, L. (2011). Assessing romantic competence among older adolescents. *Journal of Adolescence*, *34*, 397–406. doi: 10.1016/j.adolescence.2010.08.002

Shulman, S., & Scharf, M. (2000). Adolescent romantic behaviors and perceptions: Age- and gender-related differences, and links with family and peer relationships. *Journal of Research on Adolescence*, *10*, 99–118. doi: 10.1207/SJRA1001_5

Siegel, R. S., & La Greca, A. M., (2009, November). *Symptoms of ADHD in Adolescents: Are they related to difficulties in friendships and romantic relationships*. Paper presented at the Association for Behavioral and Cognitive Therapies, New York, NY.

Siegel, R. S., La Greca, A. M., & Harrison, H. M. (2009). Peer victimization and social anxiety in adolescents: Prospective and reciprocal relationships. *Journal of Youth and Adolescence*, *38*, 1096–1109. doi: 10.1007/s10964-009-9392-1

Simon, V. A., Aikins, J. W., & Prinstein, M. J. (2008). Romantic partner selection and socialization during early adolescence. *Child Development*, *79*, 1676–1692. doi: 10.1111/j.1467-8624.2008.01218.x

Skomorovsky, A., Matheson, K., & Anisman, H. (2006). The buffering role of social support perceptions in relation to eating disturbances among women in abusive dating relationships. *Sex Roles*, *54*(9–10), 627–638. doi: 10.1007/s11199-006-9030-2

Slep, A. M. S., & O'Leary, S. G. (2001). Examining partner and child maltreatment: Are we ready for a more integrated approach to family violence. *Clinical Child and Family Psychology Review*, *4*, 87–107. doi: 10.1023/A:1011319213874

Sparrevohn, R. M., & Rapee, R. M. (2009). Self-disclosure, emotional expression and intimacy within romantic relationships of people with social phobia. *Behaviour Research and Therapy*, *47*, 1074–1078. doi: 10.1016/j.brat.2009.07.016

Starr, L. R., & Davila, J. (2008). Excessive reassurance seeking, depression, and interpersonal rejection: A meta-analytic review. *Journal of Abnormal Psychology*, *117*, 762–775. doi: 10.1037/a0013866

Starr, L. R., & Davila, J. (2009). Clarifying co-rumination: Associations with internalizing symptoms and romantic involvement among adolescent girls. *Journal of Adolescence*, *32*, 19–37. doi: 10.1016/j.adolescence.2007.12.005

Starr, L. R., & Davila, J. (2012). Temporal patterns of anxious and depressed mood in generalized anxiety disorder: A daily diary study. *Behaviour Research and Therapy*, *50*, 131–141. doi: 10.1016/j.brat.2011.11.005

Starr, L. R., Davila, J., La Greca, A.M., & Landoll, R. R. (2011). Social anxiety and depression: The teenage and early adult years in youth people. In C. A. Alfano & D. C. Beidel (Eds.), *Social anxiety disorder in adolescents and young adults: Translating developmental research into practice* (pp. 75–92). Washington, DC: American Psychological Association.

Starr, L. R., Davila, J., Stroud, C. B., Li, P. C. C., Yoneda, A., Hershenberg, R., & Miller, M. R. (2012). Love hurts (in more ways than one): Specificity of psychological symptoms as predictors and consequences of romantic activity among early adolescent girls. *Journal of Clinical Psychology*, *68*, 403–420. doi: 10.1002/jclp.20862

Stattin, H., & Magnusson, D. (1990). *Pubertal maturation in female development*. Hillsdale, NJ: Lawrence Erlbaum.

Steinberg, S. J., & Davila, J. (2008). Romantic functioning and depressive symptoms among early adolescent girls: The moderating role of parental emotional availability. *Journal of Clinical Child and Adolescent Psychology*, *37*, 350–362. doi: 10.1080/15374410801955847

Stice, E., Shaw, H., Bohon, C., Marti, C. N., & Rohde, P. (2009). A meta-analytic review of depression prevention programs for children and adolescents: Factors that predict magnitude of intervention effects. *Journal of Consulting and Clinical Psychology*, *77*, 486–503. doi: 10.1037/a0015168

Stroud, C. B., & Davila, J. (2008). Pubertal timing and depressive symptoms in early adolescents: The roles of romantic competence and romantic experiences. *Journal of Youth and Adolescence*, *37*, 953–966. doi: 10.1007/s10964-008-9292-9

Subrahmanyam, K., & Greenfield, P. (2008). Online communication and adolescent relationships. *The future of children*, *18*(1), 119–146.

Swahn, M. H., Simon, T. R., Hertz, M. F., Arias, I., Bossarte, R. M., Ross, J. G., . . . Hamburger, M. E. (2008). Linking dating violence, peer violence, and suicidal behaviors among high-risk youth. *American Journal of Preventative Medicine*, *34*, 30–38. doi: 10.1016/j.amepre.2007.09.020

Szymanski, D. M., Chung, Y. B., & Balsam, K. F. (2001). Psychosocial correlates of internalized homophobia in lesbians. *Measurement and Evaluation in Counseling and Development*, *34*, 27–38.

Temple, J. R., & Freeman, D. H. (2011). Dating violence and substance use among ethnically diverse adolescents. *Journal of Interpersonal Violence*, *26*, 701–718. doi: 10.1177/0886260510365858

Temple, J. R. Le, V. D., van den Berg, P., Ling, Y. Paul, J. A., & Temple, B. W. (2014). Brief report: Teen sexting and psychosocial health. *Journal of Adolescence*, *37*, 33–36. doi: 10.1016/j.adolescence.2013.10.008

Teten, A. L., Hall, G. C. N., & Capaldi, D. M. (2009). Use of coercive sexual tactics across 10 years in at-risk young men: Developmental patterns and co-occurring problematic dating behaviors. *Archives of Sexual Behavior*, *38*, 574–582. doi: 10.1007/s10508-007-9309-6

Thompson, K. M., Wonderlich, S. A., Crosby, R. D., Mitchell, J. E. (2001). Sexual violence and weight control techniques among adolescent girls. *International Journal of Eating Disorders*, *29*, 166–176. doi: 10.1002/1098-108X(200103)29:2<166::AID-EAT1006>3.0.CO;2-3

Tjaden, P., & Thoennes, N. (2000). *Full report of the prevalence, incidence, and consequences of violence against women*. Washington, DC: U.S. Department of Justice, Office of Justice Programs, National Institute of Justice.

Tremblay, R. E., Masse, L. C., Pagani-Kurtz, L., & Vitaro, F. (1996). From childhood physical aggression to adolescent maladjustment: The Montreal Prevention Experiment. In R. D. V. Peters & R. J.

McMahon (Eds.), *Preventing childhood disorders, substance use, and delinquency* (pp. 268–298). Thousand Oaks, CA: Sage.

Tremblay, R. E., Nagin, D. S., Séguin, J. R., Zoccolillo, M., Zelazo, P. D., Boivin, M., . . . Japel, C. (2004). Physical aggression during early childhood: Trajectories and predictors. *Pediatrics*, *114*, e43–e50. doi: 10.1542/peds.114.1.e43

Ueno, K. (2010). Same-sex experience and mental health during the transition between adolescence and young adulthood. *Sociological Quarterly*, *51*, 484–510. doi: 10.1111/j.1533-8525.2010.01179.x

Underwood, M. K., Kupersmidt, J. B., & Coie, J. D. (1996). Childhood peer sociometric status and aggression as predictors of adolescent childbearing. *Journal of Research on Adolescence*, *6*, 201–223.

Uniform Crime Reports. (2009). *Crime in the United States 2008*. Washington, DC: Federal Bureau of Investigation, U.S. Department of Justice.

Valle, A. K., Røysamb, E., Sundby, J., & Klepp, K. I. (2009). Parental social position, body image, and other psychosocial determinants and first sexual intercourse among 15- and 16-year olds. *Adolescence*, *44*, 479–498.

Vasilenko, S. A., Ram, N., & Lefkowitz, E. S. (2011). Body image and first sexual intercourse in late adolescence. *Journal of Adolescence*, *34*, 327–335. doi: 10.1016/j.adolescence.2010.04.005

Vernberg, E. M., Abwender, D. A., Ewell, K. K., & Beery, S. H. (1992). Social anxiety and peer relationships in early adolescence: A prospective analysis. *Journal of Clinical Child Psychology*, *21*, 189–196. doi: 10.1207/s15374424jccp2102_11

Vézina, J., Hébert, M., Poulin, F., Lavoie, F., Vitaro, F., & Tremblay, R. E. (2011). Risky lifestyle as a mediator of the relationship between deviant peer affiliation and dating violence victimization among adolescent girls. *Journal of Youth and Adolescence*, *40*, 814–824. doi: 10.1007/s10964-010-9602-x

Vujeva, H. M., & Furman, W. (2011). Depressive symptoms and romantic relationship qualities from adolescence through emerging adulthood: A longitudinal examination of influences. *Journal of Clinical Child and Adolescent Psychology*, *40*, 123–135. doi: 10.1080/15374416.2011.533414

Waldinger, R. J., Diguer, L., Guastella, F., Lefebvre, R., Allen, J. P., Luborsky, L., & Hauser, S. T. (2002). The same old song?—Stability and change in relationship schemas from adolescence to young adulthood. *Journal of Youth and Adolescence*, *31*, 17–29. doi: 10.1023/A:1014080915602

Walker, L. E. (1984). *The battered women syndrome*. New York, NY: Springer Publishing Company.

Wargo Aikins, J., Simon, V., & Prinstein, M. (2010). Romantic partner selection and socialization of young adolescents' behavior problems and substance use. *Journal of Adolescence*, *33*, 813–826. doi: 10.1016/j.adolescence.2010.07.007

Washburn, I. J., & Capaldi, D. M. (2014). Influences on boys' marijuana use in high school: A two-part random intercept growth model. *Journal of Research on Adolescence*, *24*, 117–130. doi: 10.1111/jora.12030

Weisz, J. R., Chorpita, B. F., Palinkas, L. A., Schoenwald, S. K., Miranda, J., Bearman, S. K., . . Research Network on Youth Mental Health. (2012). Testing standard and modular designs for psychotherapy treating depression, anxiety, and conduct problems in youth: A randomized effectiveness trial. *Archives of General Psychiatry*, *69*, 274–282. doi: 10.1001/archgenpsychiatry.2011.147

Weller, J. E., & Dziegielewski, S .F. (2005). The relationship between romantic partner support styles and body image disturbance. *Journal of Human Behavior and the Social Environment*, *10*(2), 71–92. doi: 10.1300/J137v10n02_04

Welsh, D. P., Grello, C. M., & Harper, M. S. (2003). When love hurts: Depression and adolescent romantic relationships. In P. Florsheim (Ed.), *Adolescent romantic relations and sexual behavior: Theory, research, and practical implications* (pp. 185–211). Mahwah, NJ: Lawrence Erlbaum.

Welsh, D. P., Haugen, P. T., Widman, L., Darling, N., & Grello, C. M. (2005). Kissing is good: A developmental investigation of sexuality in adolescent romantic couples. *Sexuality Research and Social Policy: A Journal of the NSRC*, *2*(4), 32–41. doi: 10.1525/srsp.2005.2.4.32

Wenzel, A., Graff-Dolezal, J., Macho, M., & Brendle, J. R. (2005). Communication and social skills in socially anxious and nonanxious individuals in the contest of romantic relationships. *Behavior Research and Therapy*, *43*, 505–519. doi: 10.1016/j.brat.2004.03.010

Whitaker, D. J., Murphy, C. M., Eckhardt, C. I., Hodges, A. E., & Cowart, M. (2013). Effectiveness of primary prevention efforts for intimate partner violence. *Partner Abuse*, *4*, 175–195. doiss 10.1891/1946-6560.4.2.175

White, H. R., & Widom, C. S. (2003). Intimate partner violence among abused and neglected children in young adulthood: The mediating effects of early aggression, antisocial personality, hostility and alcohol problems. *Aggressive Behavior*, *29*, 332–345.

Williams, T., Connolly, J., & Cribbie, R. (2008). Light and heavy heterosexual activities of young Canadian adolescents: Normative patterns and differential predictors. *Journal of Research on Adolescence*, *18*, 145–172. doi: 10.1111/j.1532-7795.2008.00554.x

Williams, S., Connolly, J., & Segal, Z. V. (2001). Intimacy in relationships and cognitive vulnerability to depression in adolescent girls. *Cognitive Therapy and Research*, *25*, 477–496. doi: 10.1023/A:1005590720895

Willard, N. E. (2010). Sexting and youth: Achieving a rational response. *Journal of Social Science*, *6*, 542–562. doi: 10.3844/jssp.2010.542.562

Wittchen, H. U., Fuetsch, M., Sonntag, H., Muller, N., & Liebowitz, M. (2000). Disability and quality of life in pure and comorbid social phobia: Findings from a controlled study. *European Psychiatry*, *15*, 46–58. doi: 10.1016/S0924-9338(00)00211-X

Wolfe, D. A., Wekerle, C., Scott, K., Staatman, A.-L., Grasley, C., & Reitzel-Jaffe, D. (2003). Dating violence prevention with at-risk youth: A controlled outcome evaluation. *Journal of Consulting and Clinical Psychology*, *71*, 279–291. doi: 10.1037/0022-006X.71.2.279

Woodward, L. J., Fergusson, D. M., & Horwood, L. J. (2002). Romantic relationships of young people with childhood and adolescent onset antisocial behavior problems. *Journal of Abnormal Child Psychology*, *30*, 231–243. doi: 10.1023/A:1015150728887

Wymbs, B., Molina, B., Pelham, W., Cheong, J., Gnagy, E., Belendiuk, K., . . . Waschbusch, D. (2012). Risk of intimate partner violence among young adult males with childhood ADHD. *Journal of Attention Disorders*, *16*, 373–383. doi: 10.1177/1087054710389987

Young, J. F., Kranzler, A., Gallop, R., & Mufson, L. (2012). Interpersonal Psychotherapy Adolescent Skills Training: Effects on school and social functioning. *School Mental Health. 4*(4): 254–264. doi: 10.1007/s12310-012-9078-9

Young, J., Mufson, L., & Davies, M. (2006). Impact of comorbid anxiety in an effectiveness study of interpersonal psychotherapy for depressed adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*, 904–912. doi: 10.1097/01.chi.0000222791.23927.5f

Young, J. F., Mufson, L., & Gallop, R. (2010). Preventing depression: A randomized trial of interpersonal psychotherapy-adolescent skills training. *Depression and Anxiety*, *27*, 426–433. doi: 10.1002/da.20664

Zimmer-Gembeck, M. J., Siebenbruner, J., & Collins, W. A. (2001). Diverse aspects of dating: Associations with psychosocial functioning from early to middle adolescence. *Journal of Adolescence*, *24*, 313–336. doi: 10.1006/jado.2001.0410

Zimmer-Gembeck, M. J., Siebenbruner, J., & Collins, W. A. (2004). A prospective study of intraindividual and peer influences. *Archives of Sexual Behavior*, *33*, 381–394. doi: 10.1023/B:ASEB.0000028891.16654.2c

CHAPTER 15

What Can Dynamic Systems Models of Development Offer to the Study of Developmental Psychopathology?

MICHAEL F. MASCOLO, PAUL VAN GEERT, HENDERIEN STEENBEEK, and KURT W. FISCHER

WHAT CAN DYNAMIC SYSTEMS THEORIES OF DEVELOPMENT OFFER TO THE STUDY OF DEVELOPMENTAL PSYCHOPATHOLOGY? 665
RETHINKING PSYCHOPATHOLOGY: FROM FIXED FORMS TO DYNAMIC TRAJECTORIES 667
The Psychopathology of Fixed Forms 667
The Problem of Averages and Aggregates 667
DYNAMIC SYSTEMS THEORY AND DEVELOPMENTAL PSYCHOPATHOLOGY 669
The Dynamics of the Person–Environment System 669
The Self-Organization of Adaptive and Maladaptive Forms of Action 671
The Epigenesis of Structure of Adaptive and Maladaptive Behavior 672
THE DEVELOPMENT OF AFFECTIVE DISSOCIATION IN MALTREATED CHILDREN 673
Developmental Changes in the Structure of Skilled Action 673
Webs of Development 675
Natural Fractionation of Psychological Activity 675
Passive and Active Splitting and Dissociation 677
Growing Up Abused 678
Abusive Relationships 679
Hidden Family Violence and Isolating Dissociation 679
Psychopathology Is Not Developmental Immaturity 683
Modeling the Growth of Dissociation and Coordination 684
SELF-SUSTAINING LEARNING TRAJECTORIES IN CHILDREN WITH ATTENTION AND EMOTIONAL-BEHAVIORAL DIFFICULTIES 686
Context Sensitivity of Attention Problems 686
The Strengths and Limitations of Neurobiological Approaches 687
Setting the Stage: The Dynamics of Teaching and Learning 687
Scaffolding Dynamics During Arithmetic Lessons 689
Arithmetic Learning Among EBD and Typically Developing Students 691
Self-Sustaining Trajectories in Suboptimal Learning Dynamics 692
Modeling School Performance in EBD Students 694
THE DEVELOPMENT SOCIOEMOTIONAL ADJUSTMENT IN CHILDREN WITH EMOTIONAL AND BEHAVIORAL DIFFICULTIES 695
The Dynamics of Adaptive and Maladaptive Socioemotional Adjustment 696
Pathways in the Development of Adaptive and Maladaptive Patterns of Socioemotional Adjustment 696
Trajectories of Development: Making a Path by Walking 701
BEYOND BEST PRACTICES AND EVIDENCE-BASED INTERVENTION: TRANSLATING DYNAMIC SYSTEMS INTO PREVENTION AND PRACTICE 701
CONCLUSIONS: THE SELF-ORGANIZATION OF ORDER, VARIABILITY, AND DISORDER IN STRUCTURES OF THINKING, FEELING, AND ACTING 704
From Fixed Forms to Emergent Structures 704
Conceptualizing Dysfunction: From Pathology to Adaptive Conflict 705
From Aggregates to Individual Trajectories 706
The Challenges Ahead 706
REFERENCES 707

WHAT CAN DYNAMIC SYSTEMS THEORIES OF DEVELOPMENT OFFER TO THE STUDY OF DEVELOPMENTAL PSYCHOPATHOLOGY?

The field of developmental psychopathology evolved from dissatisfaction with static models of pathology developed for the study and treatment of adults. Developmentally oriented theorists, researchers, and practitioners observed that profiles of psychological dysfunction in children tended to differ dramatically from those found in adults. Models of psychopathology that were grounded in the study of adult psychopathology contained few provisions for understanding the origins and development of psychopathology over the course of ontogenesis. Since its inception, the term *developmental psychopathology* has served a dual function. It both names a field of study within psychology as well as a particular theoretical approach within that field (Cicchetti, 2006; Cicchetti &

Toth, 2009). In inaugurating the field, Sroufe and Rutter (1984) defined developmental psychopathology as "*the study of the origins and course of individual patterns of behavioral maladaptation*, whatever the age of onset, whatever the cause, whatever the transformations in behavioral manifestation, and however complex the course of the developmental pattern may be" (p. 18, italics in original). This definition differs in important ways from the presuppositions of the medical model that informs the study of adult psychopathology. First, it identifies the subject matter of developmental psychopathology as individual patterns of behavioral maladaptation rather than as psychological disorders. As such, the definition implies that psychopathology must be understood as a phenomenon that occurs at the *psychological* level of functioning. Second, the definition proceeds from the premise that patterns of behavioral maladaptation have their origins in diverse causal processes. The phrase "whatever the cause" implies that maladaptive psychological dysfunction is to be understood not as a linear outcome of a single underlying pathology but as an emergent product of relations among multiple processes that occur over time. Third, the definition is based on the proposition that maladaptive behavior patterns, like all psychological structures, undergo developmental change and thus are processes—sequences of events that occur over time—rather than conditions. This conception stands in opposition to the idea that psychological disorders consist of fixed categories of more-or-less static and homogeneous forms. If maladaptive behavior patterns undergo developmental change, then the form of psychopathology cannot be fixed or static.

The concept of *developmental psychopathology* has its origins in organismic-developmental and (Cairns, 1983; Cicchetti, 1984; Sameroff, 1983) general systems theory (Sameroff, 1995; von Bertalanffy, 1968). From an organismic perspective, human behavior is composed of open-ended systems of representational, emotional, and behavioral processes whose functioning is sensitive to social context. Development occurs when psychological structures undergo reorganization in the direction of increasing differentiation, integration and hierarchic integration (Cicchetti, 2010). Both adaptive and maladaptive patterns of psychological activity develop through complex interactions among biological (Gogtay & Thompson, 2010; Petersen et al., 2012), psychological (Broeren, Muris, Bouwmeester, Heijden, & Abee, 2011), and sociocultural (Tronick & Beeghly, 2011) processes over time. At any given point in time, interplay multiple among risk (Muris, Brakel, Arntz, & Schouten, 2012), compensatory (Yates, 2004), vulnerability (Goodman & Brand, 2009), and protective (Ivanova & Israel, 2006; Lansford et al., 2006; Pearce, Jones, Schwab-Stone, & Ruchkin, 2003) factors canalizes development toward or away from a broad range of adaptive or maladaptive outcomes (Muris, 2006; Steinhausen, 2006). Because human development is both dynamic and context sensitive, individuals can fluctuate between adaptive and maladaptive forms of behavior as a function of time and context (Myin-Germeys, Marcelis, Krabbendam, Delespaul, & van Os, 2005). Further, forms of action that may be maladaptive in one context may be adaptive in another (Andersen, 2003; Coifman & Bonanno, 2010).

In the past 20 years, there has been an explosion of theory and research directed toward elaborating dynamic systems models of both normative and nonnormative development (Dishion, Nelson, Winter, & Bullock, 2004; Fischer, 2008; Granic & Patterson, 2006; Gottlieb & Lickliter, 2007; Kunnen, 2012; Spencer, Austin, & Schutte, 2012; Steenbeek & van Geert, 2007; Thelen & Smith, 2006). Given their joint lineage in systems principles, developmental psychopathology and dynamic systems theory have much in common (Witherington & Margett, 2009). At their most basic level, dynamic systems models maintain that order and variability *emerge* in development through the process of *self-organization* (Kelso, 1995). To say that psychological structures self-organize is to reject the idea that any single process, module, or system is primary in the developmental formation of psychological activity. Dynamic systems invoke the importance of mutual regulation among component processes (Gottlieb & Halpern, 2002); multiple emergent pathways of psychological development (Fischer & Bidell, 2006); nonlinear change (van Geert, 2009); and shifting dynamics between stability and change (Howe & Lewis, 2005).

In what follows, we examine what dynamic systems approaches can offer to the study of developmental psychopathology. We begin with an examination of theoretical and methodological problems that arise in the study of psychopathology and its development. We examine the problems that arise from the traditional approach of viewing patterns of psychological dysfunction as fixed forms rather than as emergent processes. Thereafter, after outlining their basic principles, we explore the implications of dynamic systems models for the field of developmental psychopathology (Cramer, Waldorp, van der Maas, & Borsboom, 2010a, 2010b). We illustrate these ideas through analyses of (1) the dynamic development of affective splitting and dissociation among maltreated children; (2) learning trajectories in children who exhibit difficulties

in attention; and (3) the development of adaptive and maladaptive patterns of socioemotional adjustment over time. Throughout the discussion, we explore how individual developmental pathways self-organize over time. Finally, we discuss how dynamic systems thinking can provide alternatives to the prevailing medical model approach to understanding psychological dysfunction and its development.

RETHINKING PSYCHOPATHOLOGY: FROM FIXED FORMS TO DYNAMIC TRAJECTORIES

As is well understood, the study of psychological dysfunction in adults and children is heavily influenced by the medical model of psychopathology (Kawa & Giordano, 2012; Zachar & Kendler, 2007). A series of issues arise from the application of the medical model to the study of psychological dysfunction. First, in conceptualizing patterns of maladaptive psychological activity as symptoms of underlying pathology, such patterns are treated as fixed structures or static forms. Such an approach fails to account for the massive heterogeneity that occurs within and between classes of maladaptive behavior. Second, while research is intended to understand maladaptive activity that operates within individuals, most research is organized around the study of groups. The focus on group averages tends to obscure an understanding of how maladaptive behavior develops within individuals. This leads to the related problem of *ergodicity*, which concerns the question of whether relationships established between variables at the group level are the same as those that operate at the level of processes occurring within individuals. We examine the implications of each of these problems for the study of developmental psychopathology.

The Psychopathology of Fixed Forms

The view that maladaptive behaviors are symptoms of underlying pathologies or disorders immediately frames such behaviors as fixed forms or static entities. In so doing, the pathology model rests on three basic assumptions. First, for any given category of disorder, there should be a degree of homogeneity among behaviors taken to be expressions of any given pathology. Second, it should be possible to identify the pathology or pathologies that give rise to the said symptoms. Third, there should be some degree of coherence between the so-called symptoms and the underlying pathology. However, research on psychopathology has consistently revealed the problems of heterogeneity and comorbidity (Beauchaine, 2003; Lilienfeld, 2007; Spencer, Biederman, & Mick, 2007). Within particular categories of disorder, there is heterogeneity in the forms of behavior named by the category (Waschbusch et al., 2004), the causal underpinnings of those patterns (Barry, Golmaryami, & Rivera-Hudson, 2013; Nandi, Beard, & Galia, 2009); and the effects of intervention (Carter et al., 2012; Peer, Strachan, & Spaulding, 2008). A common approach to addressing such heterogeneity is to search for subtypes within particular diagnostic categories (Herman et al., 2012). Even here, however, the various subcategories are often defined in terms of overlapping behavioral criteria (Blackburn, Logan, Donnelly, & Renwick, 2008; Hastings, Fortier, Utendale, Simard, & Robaey 2009; Martel, Goth-Owens, Martinez-Torteya, & Nigg, 2010). A related problem concerns comorbidity, or the tendency of different categories of psychological disorder to co-occur within single individuals (Boylan, Vaillancourt, Boyle, & Szatmari, 2007; Hechtman, 2009; Weismer, 2006; Wichstrøm et al., 2012). Viewed from the standpoint of the medical model, the co-occurrence of clinical diagnoses within individuals makes it difficult to discriminate the properties of one disorder from another.

However, heterogeneity, comorbidity, and other sources of variation are problems only under the presupposition that patterns of maladaptive behavior exist as coherent expressions of fixed underlying pathologies. However, there is no reason to accept these suppositions. If we relax these assumptions, questions about the forms that maladaptive behavior assume in particular individuals, groups, contexts or periods of time become open ones (Boorsboom, Cramer, Schmittman, Epskamp, & Waldorp, 2011; Cramer, Waldorp, van der Maas, & Borsboom, 2010a; Dirks, De Los, Briggs-Gowan, Cella, & Wakschlag, 2012). Instead of presuming the existence of fixed forms of psychopathology, it is better to explore how patterns of maladaptive activity arise dynamically over time within particular individuals and contexts (Kagan, 1997, 2007). Actualization of this goal requires attention to a series of additional issues.

The Problem of Averages and Aggregates

Although the focus of developmental science is on the nature of individuals, most psychological research focuses primarily on establishing relations at the level of the group or population. However, it is often unclear how relations established at the level of the group relate to the development of individuals. Over the past decades, evidence

has accumulated that demonstrates the difficulty of drawing inferences about individual behavior from research conducted on groups. Molenaar (2004) argued that such inferences are possible only under certain conditions (called ergodic assumptions) that are virtually never met in psychology. Evidence suggests that more often than not, population insights represent only a small subset of individuals at best; at worst, they are statistical artifacts that represent no one (Estes, 1956; von Eye, 2009). In the study of developmental psychopathology, these problems arise in research that focuses on the analysis of group means that are tabulated by collapsing across observations of individuals. In addition, researchers tend to base assessments of individuals on aggregate estimates of individual behavior across multiple contexts (e.g., the number of times a child exhibits a certain behavior in a given time period). Thus, research in developmental psychopathology often represents children's behavior in terms of group means of a series of decontextualized variables.

The data depicted in Figure 15.1 help illustrate this problem. Figure 15.1 identifies a series of age-related trajectories for various conduct problems (Bongers, Koot, van der Ende, & Verhulst, 2003). As indicated in Figure 15.1, not only does each assessed behavior propensity undergo different age-related trajectories, but also within each class of behavior different subgroups of children are identified that exhibit different age-related profiles of change. Thus, not only do conduct problems assume different forms, but also each class of behavior develops through diverse pathways. If one were to focus merely on group means within each category of behavior, the resulting curve would obscure the massive variability that exists among individuals and subgroups.

The Problem of Ergodicity and the Study of Change

It is commonly assumed that if a theory holds for a representative set of all possible observations of a particular phenomenon, then it must also hold for a specific subset of those observations. Although this reasoning is true in an abstract sense, its application in psychological science relies upon an unproven and likely false corollary. This is the assumption of *ergodicity*—the idea that the statistical structure of relations among variables that holds *between subjects* is statistically similar to the structure of relations among those variables that holds *within* individual subjects. To illustrate the problem of ergodicity, consider the results of studies assessing the contributions of genes and environment to the formation of psychological activity. *Heritability* coefficients refer to the percentage of variation in a given psychological disposition that can be

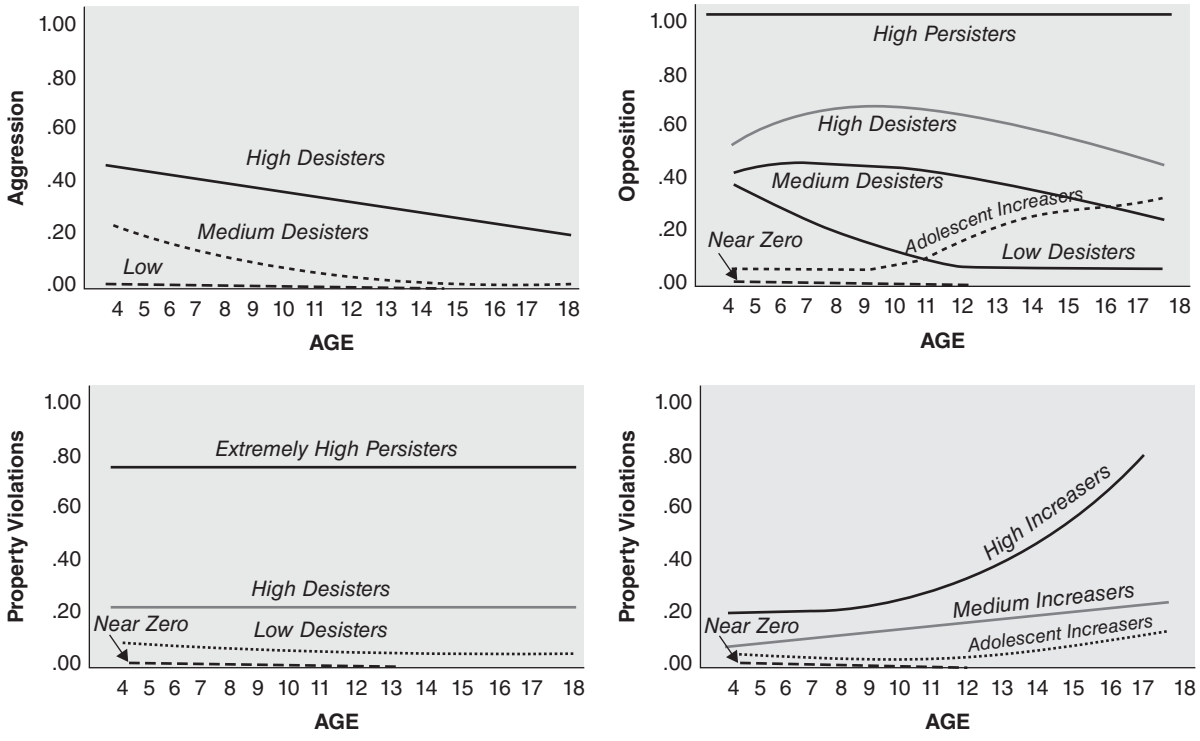


Figure 15.1 Trajectories in the development of diverse conduct problems from 4 to 18 years of age.

accounted for by differences between people in their genetics; conversely, *environmentality* refers to the percentage of variation that can be explained by differences between people in their environments. Heritability and environmentality are statistics that are derived by partitioning variation that occurs *between individuals* within a population. As such, they are concepts related to the origins of *differences between people* within populations. A heritability coefficient of .50 for IQ means that 50% of the *variation* between individuals within a population on IQ scores can be accounted for by *differences* between them in their genetics, and the remaining 50% by *differences* between them in their environments.

Problems arise, however, when concepts like heritability and environmentality—statistics that apply *only* to populations—are applied to individuals. A finding that 50% of the *variation* in IQ scores within a population can be accounted for by *differences* between those individuals in their genetics tells us nothing about the roles of genes and environment in the development of *individuals*. Individual behavior cannot be parsed into separate genetic and environmental components. To say that a disposition is *heritable* is not the same as saying that it is *inherited*. For example, adoption studies show that correlations between the IQs of adopted children and their biological parents are higher than those between adopted children and their adoptive parents. It would be a mistake to infer, however, that experience plays a less important role in the formation of intelligence than genetics. Such correlations simply indicate that the *relative rankings* of the IQs of children and their biological parents are more stable than those for children and adoptive parents. In fact, research shows that the IQ scores of adopted twins tend to be *higher* than those of nonadopted twins. Adoption tends to increase a child's IQ proportional to the socioeconomic status (SES) of the adoptive family (Duyme, Dumaret, & Tomkiewicz, 1999). When statistical relationships that hold at the level of the group fail to hold at the level of the individual, the assumption of ergodicty is violated. The assumption of ergodicity is rarely met in psychological research.

DYNAMIC SYSTEMS THEORY AND DEVELOPMENTAL PSYCHOPATHOLOGY

Dynamic systems approaches provide a framework for addressing many conceptual and empirical problems that arise in the study of developmental psychopathology. In its most basic form, *dynamic systems theory provides tools for describing how one state (of a system) develops or changes into another state over the course of time* (Weisstein, 1999). Any theory of development, learning, or intervention should be formulated as a way of describing how a particular state of a system (an early developmental state) changes into another state (a more fully developed state; improved performance) over time. In the context of developmental psychopathology, fundamental questions concern how forms of maladapative psychological functioning emerge and undergo transformation. In this paper, we describe three major principles as they relate to the dynamic development of adaptive and maladaptive behavior: (1) *dynamic systems* and *mutual regulation*; (2) *emergence* and *self-organization*; and (3) the epigenesis of developmental processes.

The Dynamics of the Person–Environment System

The word *system*, from the Greek *systema*, refers to an organized whole, or a series of things standing together. A system is not simply a set of elements or variables, or even a set of relationships among variables. Instead, a system is a collection of interrelated elements that operate as a whole. As complex dynamic systems, persons are composed of multiply nested components (e.g., organ systems, tissues, cells) whose functioning is embedded in the ecology of broader sociocultural systems. The elements of a dynamic system do not operate independently of one another. Instead, they *coact* (Gottlieb, 2002; Mascolo, 2013) and *mutually affect* (Witherington, 2011) each other over time. Novel forms of behavior emerge over time and within particular contexts as a product of local coactions among system components.

Figure 15.2 contains a model of the dynamic person–environment system (Mascolo, 2013; Mascolo & Fischer, 2010). The person–environment system is composed of five basic categories of processes: (1) individual *action* (i.e., what a person does); (2) the *objects* of action (i.e., what one's actions are performed *on*); (3) the *actions* of others; (4) *mediational means* (i.e., the tools that persons act with, e.g., language, computers); and (5) the *physical and sociocultural context*. It is essential to understand that contexts function as an integral part of the person–environment system itself, and cannot be regarded as independent variables to which persons merely react. To illustrate the functioning of the person–environment system, consider a child who experiences difficulty regulating attention. For example, when being tutored on a math problem, the child (1) performs an *act* of addition (2) *on* the math problem (e.g., 5+7). The child adjusts his actions to the ongoing and anticipated (3) guidance of his tutor. In so

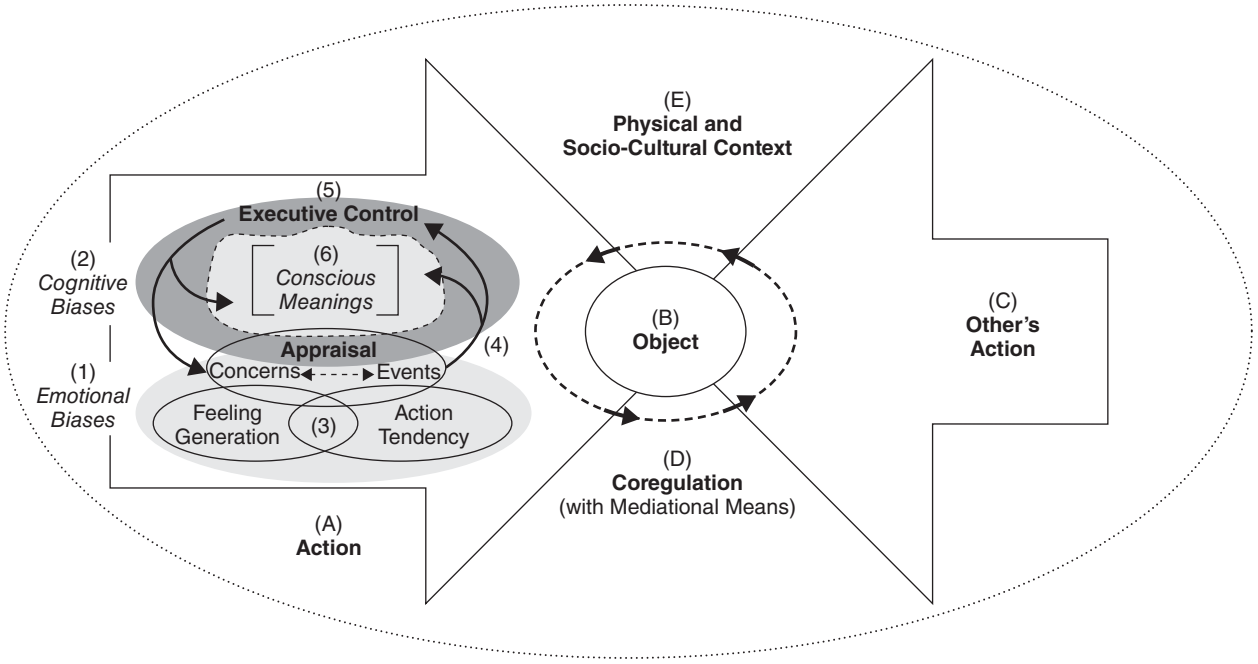


Figure 15.2 The coactive person–environment system.

doing, both the child and the tutor alike use (4) cultural tools (e.g., computer, blackboard, self-regulation techniques) to *mediate* their actions, the most important of which is *language*. Finally, the dyad's activity necessarily occurs within a (5) physical and sociocultural context (e.g., a classroom filled with desks and chairs). As a multinested system, the person–environment system contains all of the processes and dispositions that compose and mediate action, including the biological substrata that mediates a child's impulsiveness; the specific properties of the materials at hand; the representational and physical components of the problem at hand, and so forth. Within the person–environment system, there are no fundamental differences between the constraints on action that originate within individuals (e.g., executive functions), between individuals (e.g., the nature of the relationship), or from the objects on which persons act (e.g., a computer).

A system consists of sets of components (variables) and relationships among these components. The notion of relationship has various meanings. In traditional research, it means that some property of one component (e.g., its level) is associated with some property of another component. For example, if we find a component with a high level (e.g., a high level of anxiety) we can also expect to find a high (or low) level in the associated component (e.g., a high level of regulatory control). However, in a dynamic system, a relationship means that some property of one component (e.g., its level) *affects* some property of another component (e.g., its level) over the course of time. Instead of referring to relations between abstract variables, in dynamic systems, relationships among component processes are played out in the actual behavior of individuals in real time.

Figure 15.2 provides a representation of the coacting systems that operate *within* individual persons (as a part of the broader person–environment system). As indicated by the two shaded ellipses, individual action is composed of two broad classes of systems: *emotional* and *representational*. Emotion generation—which is largely nonconscious—is itself composed of three classes of mutually influencing processes: Nonconscious *appraisals* continuously monitor the relevance of event-related input for one's goals, motives, and concerns (e.g., This is a difficult math problem). Motive-relevant changes in a person's circumstances continuously modify *emotional experience* (e.g., anxiety), which thereupon provides feedback that (3) *selects* and *organizes* ongoing appraisal (4) in working memory for conscious awareness (e.g., I don't think I can do this problem!). Affective processes simultaneously activate *action tendencies* (e.g., fidgeting, withdrawal from the situation) that function in the service of an individual's broader goals, motives and concerns (Shen & Bigsby, 2010). Once conscious, (5) higher order goals (e.g., avoid being seen as incompetent) exert (6) downward control over the process of regulating thinking, feeling,

and acting (Carver & Scheier, 2013; Frijda 2009; Lewis, 1996; Mascolo, Li, Finke, & Fischer, 2003; Steenbeek & van Geert, 2007, 2008).

The Self-Organization of Adaptive and Maladaptive Forms of Action

The functioning of a dynamic system is emergent rather than predetermined. To say that psychological structures *emerge* implies that they do not exist as structures prior to their construction in development. Within dynamic systems, novel structures emerge through the process of *self-organization* (Kelso, 1995). In self-organizing systems, emergence occurs in the absence of pre-determined plans or fixed blueprints. Instead, self-organization refers to the idea that order and variability in the production patterns of adaptive and maladaptive behavior arise through the ways elements of the person–environment system mutually constrain and regulate each other over time. Dynamic systems models provide tools for tracking the ways the current state of a system self-organizes into another state.

The elements or components of dynamic systems are characterized by the fact that they can vary. Elements are properties that vary not only between systems of the same kind but also as within a system from moment to moment. Figure 15.3 depicts changes in the psychological functioning of an attention-deficit/hyperactivity disorder (ADHD)–classified child over the course of an intervention program. The figure identifies the relationship between task performance and motivation in a single child. The technical term for such a descriptive space is *state space*, and the combination of a particular system's current values on the dimensions of the space forms the system's current state. Thus, dynamic systems models assess change in a system by tracing a particular trajectory (i.e., a sequence of states) over time in a state space. As indicated in Figure 15.3, over a period of 54 days, the boy engaged in 24 sessions of computer play aimed at improving executive functioning and working memory. Before every computer game, the child was asked how motivated he was to play the game (i.e., How do you feel like playing the next game?). Total playing time was recorded, which was used as a simple measure of performance. As indicated in Figure 15.3, the changes that occur before Day 40, operating in the lower half of the state space, correspond to low levels of performance and oscillating levels of motivation. The negative covariation and oscillation observed prior to Day 40 suggests that a competitive relationship exists between these two variables. Around Day 40, this

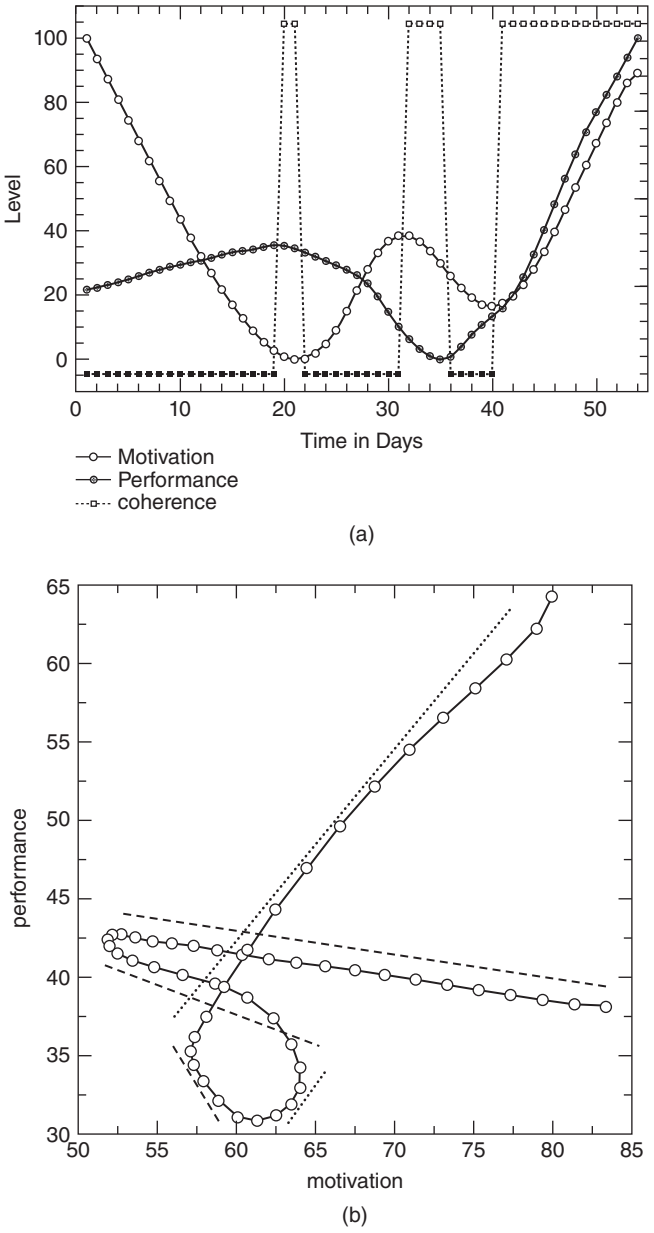


Figure 15.3 State space model of shifting relations between motivation and performance over time in a child with attention difficulties.

relationship shifted. It was replaced by a supportive relationship characterized by an increase in both motivation and in performance. Day 40 thus marked a change in an attractor—a shift from a self-sustaining conflict between motivation and performance to a pattern of self-sustaining cooperation. While these data do not indicate reasons for the shifting relation, they nonetheless indicate a change from less adaptive to a more adaptive relation between motivation and performance over time. In so doing, they

show how movement from maladaptive to adaptive action self-organizes over time.

The Epigenesis of Structure of Adaptive and Maladaptive Behavior

Over the past decades, an *epigenetic* (i.e., beyond genetic) conception of development has become increasingly prominent in the developmental sciences. At its most general level, epigenesis refers to the idea that anatomical and psychological structures emerge over time in development and are neither preformed nor predetermined (Gottlieb, 2002; Tavory, Jablonka, & Ginsburg, 2012; Wolf & Linden, 2012). In its strongest form, it refers to the idea that processes beyond the level of the genes (e.g., RNA, cytoplasm, DNA methylation) play a role activating gene expression at points of development (Danchin et al., 2011; Ferguson-Smith, 2011; Nugent & McCarty, 2011). The strong form of epigenesis challenges the central dogma of the modern synthesis (Huxley, 1942) in evolutionary theory. This central dogma states that the direction of causality between genetic and nongenetic processes in the creation of proteins is unidirectional (DNA → RNA → mRNA . . . → protein). Genes orchestrate the building of proteins but are themselves unaffected by the processes involved in protein synthesis. However, recent research has shown that nongenetic biological processes, particularly DNA methylation and histone modification, function to activate and inhibit gene expression during protein synthesis. As such, the epigenetic model embraces the idea of bidirectional rather than unidirectional causality between genetic and extragenetic processes (DNA ↔ RNA ↔ mRNA . . . ↔ protein).

Processes that regulate gene expression are sensitive to changes that occur at multiple levels of an organism's functioning in the world. Figure 15.4 contains a schematic representation (adapted from Gottlieb et al., 2006) of the hierarchical and temporal organization of the epigenetic system. As indicated in Figure 15.2, no single element of the developmental system (e.g., genes, extragenetic biological processes, individual action, or social experience) is autonomous or primary in human psychological development. Psychological structures are emergent products of coactions among both horizontal (gene–gene; cell–cell; organism–organism) and vertical (gene–cytoplasm; cell–organ; organism–ecosystem) aspects of the developmental system. For example, the offspring of rat mothers who show high levels of maternal care during a pup's first week of life (i.e., grooming, pup licking, nursing) show lower levels of stress as adults than offspring of less nurturing mothers. These maternal behaviors produce a suite of biological changes in rat pups, some of which are involved in the silencing and expressing of genes (Weaver et al., 2005). Research has identified epigenetic processes involved in the construction of psychological structures in a variety of domains in humans as well. These include the development of attachment behavior (Lickliter, 2008; Schore & Schore, 2008); emotionality and stress regulation (Champange, 2010); suicidality among adult survivors of maltreatment (Weaver et al., 2005); and other domains (Jablonka & Jones, 2005).

In the remainder of this chapter, we examine how dynamic systems models can be used to illuminate the study of the development of adaptive and maladaptive behavior in a variety of different domains. We begin with an analysis of the developmental course of affective dissociation as a strategy for managing emotional distress in maltreated children. We then examine the social dynamics of teaching and learning with children who exhibit attentional, emotional, and behavioral problems. We then turn

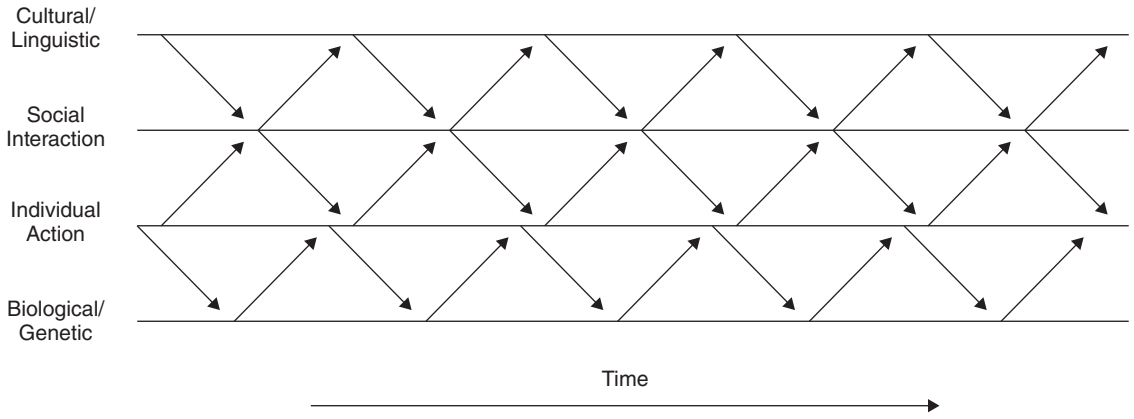


Figure 15.4 The epigenetic nature of development. Source: Adapted from Gottlieb (2002).

to an analysis of the epigenesis of adaptive and maladaptive patterns of socioemotional adjustment from infancy through adulthood.

THE DEVELOPMENT OF AFFECTIVE DISSOCIATION IN MALTREATED CHILDREN

We organize our worlds and ourselves into positive and negative splits involving right and wrong, good and bad, friends and enemies, heaven and hell, good guys and bad guys, virtue and sin (Freud, 1961/1963, 1966 Osgood et al., 1957; Sullivan, 1953; Wundt, 1905/1907). These fundamental splits identify the organizing dimension along which we structure our experiences and conceptions. We argue that positive and negative valence are the fundamental, background organizers of both normal development and psychopathology. Biases and constraints growing from positive and negative experiences organize action and shape development from birth (Fischer, Shaver, & Carnochan, 1990; Li, Wang, & Fischer, 2004). One example is affective splitting, where a person splits events, people, or aspects of a situation into positive and negative, even though to another observer the things are not simply positive and negative (Fischer & Pipp, 1984; Harter & Buddin, 1987). Examples of splitting occur naturally in young children. A 2-year-old boy had been left with his grandparents for a few days, and he split his mother into separate representations of "Good Mom" and "Bad Mom." When she called him on the telephone, he responded with affection. At the same time, he told her about the "bad Mommy" who had left him at his grandparents. A similar process occurred in a 4-year-old girl: Asked if she can be happy and sad simultaneously, she said, "No way! I've only got one head."

While these examples show normal emotional splitting, splitting can permeate a person and play a central role in psychopathology, especially in cases of abuse. Research in the area of trauma and psychopathology shows a connection between the repeated trauma of abuse and pervasive changes in relations between thinking and emotion (Figure 15.2**,** Point 3) (Cole & Putnam, 1992; Feiring et al., 2010; Hulette et al., 2011; Kenardy et al., 2007; Kim & Cicchetti, 2006; Li et al., 2004; Terr, 1991; Valentino, Cicchetti, Rogosch, & Toth, 2008). Consequences include fragmentations in the sense of self and feelings of inner badness at the core. An illustration is the case of Marilyn van Derbur.

Living in a seemingly proper middle-class family, van Derbur's father sexually abused her at night for many years through grade school and adolescence (Van Derbur Atler, 1991). She organized her experience and action into a day child and a night child. She experienced feeling quite different in her two selves:

> In order to survive, I split into a day child, who giggled and smiled, and a night child, who lay awake in a fetal position, only to be pried apart by my father. Until I was 24, the day child had no conscious knowledge of the night child. During the day, no embarrassing or angry glances ever passed between my father and me . . . because I had no conscious knowledge of what he was doing to me . . . The more degraded the night child became, the more the day child needed to excel . . . from skiing on the University of Colorado's ski team, to being a debutante, to graduating with Phi Beta Kappa Honors, to being named Miss America. I believed I was the happiest person who ever lived. I truly believed that.

The day child was bright, earned strong grades, and had many friends. The night child was afraid, ashamed, and full of night terrors. After she left home to go to a private school, her father stopped abusing her, and it took her several years to remember the nights. After she turned 18, she began to wake up with upsetting dreams and anxiety attacks that seemed to come from nowhere, particularly at night or on dates. It took several more years to remember the night child and to begin to make sense of her experience. Gradually she joined together the ways she had split her experiences.

Developmental Changes in the Structure of Skilled Action

We employ a set of tools called dynamic skills theory to analyze how splitting develops based on the intense emotions that shape learning and development (Fischer, 1980; Fischer & Bidell, 2006; Fischer, Bullock, Rotenberg, & Raya, 1993; Fischer & Rose, 1994). People develop along specific pathways, building ever more complex control systems called skills. A skill is defined not as a property of a person but instead as a property of a person in a situation. A skill comprises the joining of the person's activities with the context in which the activities happen. This principle forms one of the basics of skill theory. As indicated in Figure 15.2, the coupling between person and context (e.g., continuous perception-context-action loops) is a fundamental feature of dynamic systems models of human behavior and development. Emotion and cognition coact in the development of control systems. Over time, people build successively more complex skills—control structures that integrate increasingly complex systems of emotion, cognition, and action, both adaptive and maladaptive.

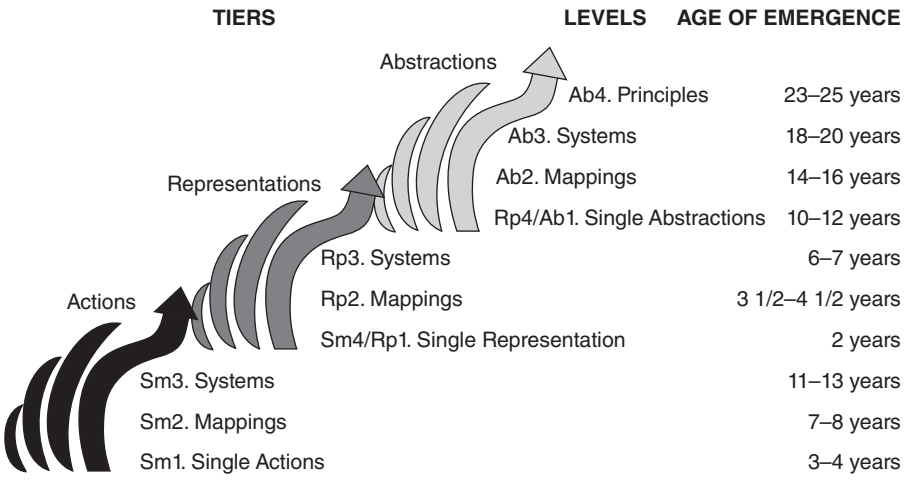


Figure 15.5 Hierarchical levels of skill development. *Source:* Fischer, 1980.

Dynamic skill theory identifies a series of 13 different levels of complexity in the development of skills from infancy through adulthood. As indicated in Figure 15.5, skills develop from the differentiation and integration of lower order skill components into increasingly higher order structures. Skills develop through four broad tiers (*reflexes*, *sensorimotor acts*, *representations*, and *abstractions*). Within each tier, skills develop through a series of four iterative steps. For example, in the *representational* tier, beginning around 18–24 months, children become able to construct concrete symbolic representations—concrete images and meanings that go beyond the information given in sensorimotor experience. Development in the representational tier begins with the ability to construct single representations (e.g., simple concrete ideas, images) at around 18–24 months of age. As shown in (1) in Figure 15.6, using single representations, a child can create simple concrete meanings like "Mommy is nice," "Daddy is mean," or "I'm a good/bad boy/girl." Around 3.5 months to 4 years of age, children gain the capacity to map one representation onto another. Such representations are called representational mappings. At this level, a child can represent the relation between two concrete ideas or representations. For example, as indicated in (2) in Figure 15.6, children can understand mappings such as "Mommy was nice because I was good" or "Daddy was mean because I was bad."

Around 6–7 years of age, children can begin to map one representational mapping onto another; this results in the formation of a single representational system. At this level, a child can relate two concrete relationships into a single integrated understanding. For example, as indicated in (3) in Figure 15.6, bringing together two mappings, a child can

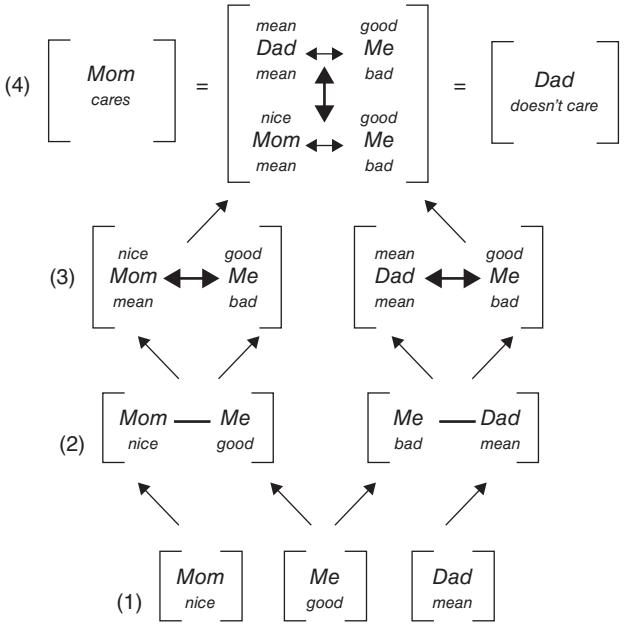


Figure 15.6 Levels within the representational tier of development.

connect "Mommy is nice when I'm good, but mean when I'm bad" or "Daddy is mean no matter if I'm good or bad." The final iterative level within a tier consists of a system of systems, which results from the intercoordination of two or more systems; a system of systems is both the fourth level of a lower order developmental tier as well as the first level of the next broad tier of development. As indicated in (4) in Figure 15.6, a system of representational systems results from the coordination of two representational systems to form a single abstraction—the first level of the abstract tier of development. At this level, abstracting across multiple

concrete interactions with her mother (e.g., Mom is angry when I'm bad but nice when I'm good), a preteen can construct a single abstract conception like "Mom has my best interests at heart." Alternatively, by abstracting across multiple interactions with her father (e.g., Dad is mean no matter if I'm good or bad), a youth can construct an abstraction like "Dad cares only about himself."

Webs of Development

We build skills for specific contexts in which we live, with no simple progression through any ladder of stages. As a result, we build diverse pathways shaped powerfully by emotions and by contexts. Thus, rather than thinking of developing skills as moving in a single direction up a ladder or staircase, it is more helpful to think of development as organized like a web of pathways, such as that shown in Figure 15.7. Automatized areas of pathways refer to behavior patterns that have been routinized and are automatically activated without effortful coordination. Functional areas refer to developmental levels operative in everyday contexts without contextual support. Optimal levels of performance refer to effortfully executed activities under the effortful control of individuals, but operating under conditions of high contextual support. Scaffolded levels of activity are those that are coconstructed between individual and in which more accomplished others support, guide task performance, or perform part of a given task.

Standard analyses of psychopathology emphasize fixation at or regression to an early stage, but we see psychopathology as development in unusual or strange contexts, such as children growing up in an abusive environment. The result of these unusual contexts is that people develop webs of skill that are adaptive in their specific world, based in their experience. Sometimes they grow up strangely. The mismatch comes with shifts outside that world, where the context-appropriate skills no longer match the particular context. People develop simultaneously along many diverse strands, building different stands and webs for acting in different contexts. Although this developmental diversity is common in both adaptive and maladaptive development, prior cognitive developmental research has used methods that made it impossible to detect such diversity in developmental pathways (Block & Block, 1980; Case & Edelstein, 1993; Rose, Rouhani, & Fischer, 2013; Stein, Dawson, & Fischer, 2010).

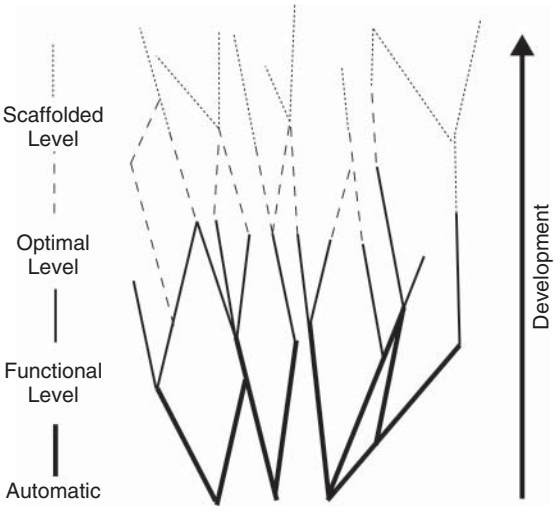


Figure 15.7 The developmental web.

At any given time in development, children (and adults) do not function at any single *point* along a pathway or strand. Instead, depending upon a wide range of variables (e.g., social context, level of support, emotional state, domain of activity), they function along a segment or range of points along a pathway (Brown & Reeve, 1987; Fischer et al., 1993). An individual's *optimal level* of performance consists of the highest level of skill that she is capable of sustaining in situations that provide high levels of contextual support. A person's *functional level* of performance refers to the level of skill at which she typically performs spontaneously in the absence of high levels of contextual support. We define the *developmental range* as the difference between optimal and functional levels of an individual's skilled activity at any given point in development. For example, suppose that a child wants to tell a story about people being mean. Often a teacher or other adult will offer support by explaining a story at the upper limits of the individual's capacity to understand. This support helps children to act at their optimal level, the upper limit on their performance. Trying to tell a story without support, children act at their functional level, which defines the limit on spontaneous performance. A person's functional level is generally lower than his or her optimal level. Further, optimal and functional levels of performance show different growth functions. While changes in one's functional level typically show continuous and slow growth, optimal level development shows abrupt changes and periods of discontinuity.

Natural Fractionation of Psychological Activity

The diverse strands in the web of development show that people's minds are naturally fractionated according to context. The control systems are distinct and independent, not coordinated or integrated (Fischer & Pipp, 1984; Stein et al., 2010). Although Piaget (1957, 1975) posited

logic as the shape of intelligence, real people do not have fully integrated or logical minds. We are driven instead by control systems that are naturally separate, but we can potentially coordinate and integrate diverse strands. Although the various control systems that drive human action are naturally fractionated rather than integrated, any particular control system (or skill) is itself an integral structure in the sense that it is composed of some sort of configuration of representation, affect and motor action that operate in relation to each other within the larger person–environment system. Real human behavior is the result of the ways separate control systems act—that is, how they cooperate, work independently, or even work against each other. For example, when learning to ride a bicycle, control systems for steering, pedaling and balancing tend to operate separate from one another. Directing attention toward balancing draws resources away from steering and pedaling, and vice-versa. Over time, as these control systems become intercoordinated, they operate together as a single seamless skill, freeing resources for the development of new skills (e.g., manipulating the gears). Although these skills start off separate, each skill nonetheless consists of a structured integration of representation, affect and motor action dynamically operating within particular physical and social environments.

One well-documented source of difference is tasks, with hundreds of studies showing that the same capacity across tasks produces behaviors at different developmental levels. The conclusion, to use Piaget's terminology, is that décalage (unevenness) is the rule in development in most domains (Bavelier et al., 2012; Block & Block, 1980; Flavell, 1982). For instance, most 7-year-olds who go to school understand that when orange juice is transferred into a container with a different shape, the total amount stays the same. But when these same 7-year-olds are asked about conservation of area (for example, the amount of area covered by an array of farm buildings in diverse locations), they do not generalize to this new task. Children need to construct each type of conservation anew. It is not a general concept but is tied to the task where it was learned. In the developmental web, this fact is represented by separate strands for the two types of conservation.

So many tasks create separate strands, creating an inevitable, pervasive fractionation. In a classroom, children learn how to do addition, but playing with marbles on the playground they learn a different addition skill. Emotions organize experience into distinct strands, as with emotional splitting. Indeed domain specificity is everywhere, and generalization takes special effort. Application and generalization of skills across contexts is not at all automatic. Instead, application of skills from one context or developmental domain to another typically takes time, effort, and even training. Skills can coordinate across contexts and domains in many ways depending upon task demands, social context, and the nature of the skills themselves. For example, distinct skills can (1) operate *independent* of one another (e.g., watching basketball while eating popcorn) or (2) *work together* (e.g., running while dribbling a basketball). Further, they can (3) *mutually support* each other (e.g., naming the ink color of words that name the same ink color in which the word is printed) or (4) *compete* with each another (e.g., naming the ink color of words that name colors that are inconsistent with the ink color in which the word is printed; the Stroop effect). While development involves the effortful coordination of skills across contexts or skills, the coactive coordination of skills also leads to new forms of learning and poses novel learning challenges for the developing person.

With fractionation so pervasive, people commonly fail to recognize what goes together in the world. We routinely simplify tasks by splitting components as if they are separate tasks. A 2-year-old who was distressed at being left by his mother emotionally split his experience of her into separate representations—one good and one bad. Alternatively, a 5-year-old split a story that his father had told him so that it left out the ways that his Dad had been nice to him and only included a mean interaction. Dealing with conservation of liquid, for instance, children commonly split amount into two separate quantities—height and width—and they ignore the other one, focusing on just height or just width (Piaget & Szeminska, 1941/1952). Thus, people routinely oversimplify challenging tasks and situations based on their level of understanding or their feelings about the events around them.

On a more positive note, with development people move *toward integration*, constructing specific coordinations of previously separate skills. Coordination is a fundamental part of development, laying a groundwork for more complex control systems (Fischer & Bidell, 2006; Fischer & Rose, 1994; Fischer & Watson, 1981; Mascolo & Fischer, 1998). Indeed vacillation between skills can be a sign of progress, marking a step toward integration. When a child vacillates between two skills, a process called shift of focus, the vacillation marks transition to a new coordination. The dual activation marks transition to a new understanding based on a novel coordination (Goldin-Meadow, Alibali, & Church, 1993). In this way, retelling an integrated story involving nice and mean themes as a sequence of two separate stories (one about nice interactions and then one about mean ones) demonstrates the shift-of-focus capacity,

which marks progress toward eventual integration of fractionated skill components.

Passive and Active Splitting and Dissociation

In splitting, elements of an action, task, or situation are separated despite the obvious need for them to be coordinated. Affective splitting happens when such elements are separated into positive and negative classes. The more general concept of dissociation happens when mental/affective elements are separated despite the fact that they should be coordinated in the everyday world. In psychopathology, the words *dissociation* and *splitting* are used to designate *motivated* separation, such as dissociating a feeling from the thought where it originated, dissociating the self into multiple personalities, or splitting family and friends into good and bad people (Breuer & Freud, 1895/1955; Putnam, 1989, 1997; Putnam, Helmers, & Trickett, 1993). Typically, clinical analyses, especially when based in Freudian thought (Freud, 1917/1961/1963, 1923/1961), presume active control of dissociation and splitting in some unconscious way. Yet if psychological activity is naturally separated into domains (naturally fractionated), then splitting and dissociation happen routinely every day, simply because a person has not coordinated skills or experiences (Feffer, 1982). There is no specific motive required to motivate splitting or dissociation.

At the same time, splitting and dissociation can be motivated, usually through unconscious processes—special cases involving a split or dissociation that is functional albeit unconscious. Analyzing the development of splitting and dissociation requires distinguishing passive separation from the types of controlled separation that permeate motivated dissociation and splitting. A vivid example comes from a case of repeated sexual abuse of 8-year-old Shirley, whose father repeatedly raped her in her bed in the basement of her home (Canadian Broadcasting Corporation, 1990). This case shows how the trauma of abuse can be partially controlled unconsciously, and how a child can survive such horrendous abuse. Whenever she resisted his efforts, he beat her up. As a coping mechanism, she focused on a little hole on the wall near her bed. She concentrated so intensely that she could experience herself as hiding in the hole. From inside the hole, she could dissociate herself from the ongoing rape, and in that way, she could survive the trauma without disruptive distress.

Coping was always difficult, but it was especially hard when her father changed what he did. When her mother went out to shop, her father committed the rape upstairs, not in the usual basement room. The context had allowed her to control her reaction, but without that context, especially the hole where she put her mind, she was unable to dissociate. She screamed and fought with her father, and he became viciously angry, knocking her unconscious and continuing with the rape. With this sort of situation involving abuse and trauma, children need skills of dissociation to survive (Cole & Putnam, 1992; Famularo et al., 1994; Feiring et al., 2010; Terr, 1991).

In this way, people can develop controlled dissociation and splitting, which vary along a dimension from actively controlled to passive. In *active dissociation*, people purposely (consciously or unconsciously) keep two experiences separated. Some psychological disorders such as multiple personality are grounded in active dissociation. Many Freudian defense mechanisms involve active dissociation, as with repression, in which one part of the mind works to prevent a thought or feeling from entering awareness. Experimental research has established that repression and active dissociation take place not only in pathology but also in normal functioning, especially in hypnosis, dreaming, and in experiences of religious spirit possession (Cepeda et al., 2013; Erdelyi, 1985; Foulkes, 1982; Hencke, 1996; Hilgard, 1977; Meares, 2012; Mischel & Mischel, 1958).

When viewed from a dynamic skills perspective, active dissociation is a developmental accomplishment: people need to create a coordination for active dissociation so that they can purposely keep components separated. The groundwork for this capacity begins to develop at about 4 to 6 years of age, when children can for the first time actively dissociate components. Shirley used this capacity when she mentally put her mind in a hole in the wall to separate her personal experience from what her father was doing to her body during the rape. As people develop ever more complex mental skills, they can construct more complex types of dissociative coordination (Freud, 1936/1966; Haan, 1977; Vaillant, 1977). We hypothesize that the strong forms of active dissociation that are often described in psychopathology begin at 4 to 6 years with the first dissociative capacity but reach full fruition during adolescence. Youth develop complex abstract skills that bring together many components of action, thought, and feeling, and this capacity gives them great potential for complex thinking, even when the complexities are not healthy.

In this chapter, we will describe a developmental pathway that produces divided consciousness. We call it *hidden family violence*, in which people live two rigidly separate worlds at home and in public. At home, they act in physically and emotionally abusive ways with their children, but they keep that pattern hidden from public view. In public, they show a world of good behavior, acting as the

perfect family. We call this pattern *isolating dissociation*, with development moving along two dissociated pathways: One is the public, "good" self, which is displayed in conventional interactions in the public world of school, community, and church. The other is the private, "bad" self, which is displayed in intimate interactions in the family. This dissociation develops originally from environmentally supported dissociations tied to children's experiences in public and private contexts. Over time, development moves toward active dissociation of private and public worlds, with the private world hidden from public view.

It is important to note the idiosyncratic nature of such developmental processes, even as they point to more universal developmental processes. The patterns of active dissociation exhibited in hidden family violence (and other forms of maltreatment) involve the construction of specific developmental pathways. Such idiosyncrasies are the result of the fact that development processes operate as a complex, dynamic system. Although the developmental pathways through which individuals develop are idiosyncratic, they nonetheless point to more universal developmental processes. However, what we conceive to be a universal process is not the average or typical trajectory abstracted across many individuals, but instead the set of underlying processes and principles that describe how such pathways are constructed. In this section, these include the emergent principles of dissociation and integration.

Growing Up Abused

Recurrent abuse radically shifts the shape of development. Our thesis is that recurring, extreme emotional experiences caused by maltreatment fundamentally alter social-emotional development, changing how people organize themselves along the positive–negative dimension and inducing dissociation based on the type of abuse. One result is predominant negativity in self-representation, spreading to many social relationships and accompanied by splitting and dissociation of both self and other. Continuing maltreatment produces repeated traumas and fundamental changes in personality, which include an affect of inner badness and self-fragmentation (Breuer & Freud, 1895/1955; Famularo et al., 1994; Hencke, 1996; Putnam, 1993; Terr, 1991; Westen & Cohen, 1992; Westen et al., 2012). Of all the patterns of maltreatment, the most destructive appear to be repeated sexual abuse and the combination of physical abuse with emotional abuse. When children try to deal with the dangers of repeated abuse, they organize themselves in ways that aid survival. These patterns often become integrated into a child's personality, and they can turn out to be maladaptive, even in benign, day-to-day interactions. Pervasive outcomes include a negativity bias and frequent dissociation/splitting, although outcomes vary widely with the type and severity of abuse. That is, abusive experiences create diverse developmental pathways, including multiple personality, hidden family violence as well as borderline, narcissistic personality disorder, and psychopathy (Meares, 2012).

Two Characteristics: Negativity Bias and Dissociation

Findings suggest a sketch of the pathways produced by abuse in children's constructions of positive and negative interactions as well as self-representations (Ayoub, Willett, & Robinson, 1993; Cicchetti & Toth, 2005; Feiring et al., 2010; Fischer & Ayoub, 1994; Fischer & Bidell, 2006; Fischer & Pipp, 1984; Fischer & Rose, 1994). Research shows that in severely maltreated children and adolescents, the positivity bias in their approach to events evaporates early on and is replaced by a negativity bias. The emotions of maltreated children tend to be dominated by negativity. Among maltreated individuals, representations about negative interactions are rich, dynamic, involving, and often frightening for people witnessing such interactions, and even for the children themselves. As the children grow older, they typically try to inhibit negative expression in certain contexts, particularly when they sense danger from a powerful adult or older child (Ayoub et al., 2006) or when other people are listening.

In addition to this transformation to a negativity bias, abuse seems to produce increased splitting and dissociation in representations of self and others. Patients with dissociative patterns such as hidden family violence and multiple personality show a remarkably high incidence of severe maltreatment (Ayoub et al., 2003, 2006; Putnam, 1989; Westen & Cohen, 1992). Maltreatment leads to prominent dissociation and splitting early in development (Calverley et al., 1994). Children show unrealistic distortions such as false positive affect (Crittenden, 1988) and overevaluations of their own competence (Vondra et al., 1989). Other findings include using fewer words about internal states, attributing those states to fewer people, and limiting their attributions to narrower contexts (Cicchetti & Beeghly, 1987). Affective splitting increases unrealistic views of others and self, which eventually leads to maladaptive patterns among adults such as narcissism and borderline personality (Meares, 2012).

In reacting to maltreatment, children struggle to see themselves as competent and in control. Abuse delays development of self and the ability to get along with others

(Aber et al., 1989; Ayoub et al., 2006). But even worse is the lack of trust and love that goes with maltreatment, often producing not simply delay but a shift to a different developmental pathway dominated by negativity and the use of splitting and dissociation to organize self in relationships. These results do not mean that only abused children show emotional splitting. Remember that splitting and dissociation occur normally in all children and adults. However, with maltreatment come new kinds of splitting and dissociation, starting with a strong negativity bias toward both self and others. Trust is a deep, broad issue for maltreated children, who typically show a marked dissociation including lack of trust in close relationships.

Abusive Relationships

The organization of action and thought adapts to fit the close relationships that children experience in the family. Child and adult come to relate to other people in ways shaped by the relationships in their family (Sullivan, 1953). Close relationships form the foundation for a child's personal development (Ainsworth et al., 1978; Bretherton & Waters, 1985; Crittenden, 1988; Fischer & Bidell, 2006; Fischer et al., 1990; Mascolo & Fischer, 2010; Vondra et al., 1990) and are candidates for the primary source of environmental influences on that development (Waller & Shaver, 1993; Westen et al., 2012). As we sketched above, a child's initial organization of these interactions naturally starts out as split into positive and negative, including good and bad self, good and bad parent, nice and mean peers, and good and evil forces in the world.

Continued maltreatment in close relationships alters how children organize their experience as positive and negative, and these consequences transform their developmental pathways. They start at a young age with normal affective splitting into good and bad. Children who are not maltreated typically develop skills for integrating opposing experiences while simultaneously focusing on the positive (although young children can enjoy playing aggressively). In contrast, maltreated children split or dissociate their experiences of relationships while focusing on negative emotions. When they grow up, many of these children have created what Westen (Westen & Cohen, 1992; Westen et al., 2012) has called malevolent rather than benevolent self-concepts. Simultaneously, they develop sophisticated strategies for dissociating their experiences of people, events, and emotions. Happily, some fortunate children discover the healing power of healthy relationships, and move away from abusive pathways, while often still showing effects of abuse (Cicchetti, 1989, 2005, 2013). Such children illustrate the idiosyncratic nature of individual pathways and suggest that the conditions that give rise to active and passive dissociation are probabilistic rather than deterministic.

Hidden Family Violence and Isolating Dissociation

One common type of maltreatment we call *hidden family violence*. It produces a specific kind of dissociation, which is grounded in sharply isolating different domains of social life. Families hiding violence sharply split their private and public worlds so as to encapsulate violence in their private relationships and maintain a positive public image. Children in such families build a dissociation between a benevolent public self and a malevolent (abusive or abused) self in private, with little integration between the two.

The pattern starts with passive dissociation of public and private, which becomes more active as children internalize the separation and strive to sustain it. The basic mechanism is isolating dissociation, shifting from public to private or vice versa, maintaining a rigid distinction. Adults developing along this pathway have great skill at sustaining the separation, keeping their experienced worlds distinct. In public settings, such as shopping or church, they are well behaved, seemingly outstanding citizens. They hide all signs of violence at home. They often champion moral causes and criticize others who do not conform, showing devout religious belief or intense commitment to an ethnic group. Asked about maltreatment, they vehemently reject it. They seem to believe that they are perfect parents. They sustain such a powerful split between the public and the private that their malevolent private world seems to disappear.

Of course, the distinction between public and private is pervasive in human cultures. Families everywhere clearly separate what is appropriate behavior in public and in private (Heider, 1991; Wallbott & Scherer, 1993). What distinguishes hidden family violence is the way the distinction is defined emotionally, centering on isolating dissociation. The private world is marked by tyranny, maltreatment, and emphasis on power. The public world in contrast shows a prosocial stance, with stereotyped, positive images and inability to talk about the private world.

Skills for Isolating Dissociation

Hidden family violence starts with dissociation arising from emotional experiences related to trauma, usually in important relationships. Hidden family violence is built around a rigid situational structure with strong dissociation between private and public domains. As a result, children

growing up in this situation develop *isolating dissociation*, separating public and private worlds with only unconscious coordination. Children learn to shift from one domain to the other, not coordinating the domains except unconsciously. They employ shift of focus, a simple shifting between skills, requiring no evident coordination but requiring decisions about which world is focal—public or private (Fischer & Bidell, 2006; Fischer, 1980; Mascolo & Fischer, 2010). In our research with young children, many have demonstrated affective splitting by the first half of the second year. Recall the 2-year-old boy who split his mother into Good Mom and Bad Mom when he talked with her on the phone.

The splitting is especially pronounced in children who have been both physically threatened and emotionally berated. These toddlers display conventional but faked positive emotion, or they take on a caretaking, parentified stance showing both a negativity bias, an attempt to control the interaction (Ayoub et al., 2006; Cicchetti & Toth, 2005; Cicchetti & Beeghly, 1987; Crittenden, 1988) as well as sequelae in adults (Main & Hesse, 1990). These children also tend to inhibit negative emotion with adults but act aggressively with peers and others they perceive as less powerful. Parents in hidden-violence families reinforce this separation by being secretive about their home life and forbidding their children from talking about it, especially with outsiders. Outside the home, the parents are polite and overtly kind to their children, while inside the home they act as abusive tyrants. The type of hidden family violence we have most frequently encountered involves physical and emotional abuse in private.

Our focus will be on this pattern, illustrated by the case of Mr. M, a 37-year-old church elder, a successful businessman, and respected leader in a New England town. He owned a fleet of ice cream trucks, which drove through neighborhoods ringing their bells to gather children and adults to buy ice cream. Mr. M indicated that he had no close friends. What mattered to him was his family, which he said was widely respected in the community, close-knit, and "good." He focused on his successful business, his family's church attendance, his children's regular school attendance, and his position in the church. He reported that his marriage was wonderful and that his wife was the perfect woman for him. In public, he acted as a model husband and father. At home, he was dominant and in constant need of being correct. He would routinely degrade and abuse his wife. He read selected passages from the Bible, and according to his interpretation, admonished her as a whore who should repent. He would then physically assault her, telling her that she must obey him as scripture demands. He was often sadistic in his punishment of the children. Under great stress, his wife eventually reported his abuse of the children and thus brought him to the attention of the Department of Social Services.

Mr. M described his own childhood in idyllic terms, but with no recollection of details. He said that his father was a good provider and a respected citizen. Evidence from family friends indicated that his father was a stern disciplinarian who was harsh with his children. Mr. M consistently denied being violent at home. His children denied that he abused them and claimed that their injuries came from accidents at home, on the playground, or in sports. When Mr. M's wife and children were able to escape to the safety of a shelter, they related information about their abuse in private. Even after allegations were confirmed with physical evidence, Mr. M denied them. Mr. M showed isolating dissociation of his public and private worlds.

Drawing on the tools provided by dynamic skill theory (Figure 15.5), we analyze the development of dissociation through a series of levels, from representations of concrete agents in social interactions to abstractions of intangible personality characteristics. In so doing, we describe developmental changes in the structure of children's dissociations in terms of a series of skill diagrams of the sort provided in Figure 15.6. Skill diagrams identify the structure and developmental level of integrative structures of thinking, feeling and acting in terms of the levels of skill identified by skill theory. Levels of dissociative skill develop over time in terms of patterns of differentiation and coordination that are typical of developmental change in general. The primary difference is that instead of acts of coordination that result in relating one skill element to another, dissociative skills involve coordinative acts of separating elements of meaning along positive and negative emotions and evaluations.

First Tier of Dissociation: Isolating Agents

Hidden family violence develops through a series of levels of isolating dissociation. First, children develop *isolating agents*, which are coordinated within each domain, public or private, but not across domains. Gradually the isolating agents of children become the *isolating personalities* of adolescents and adults, and the dissociation becomes highly skilled.

Fluent control of representations of individual agents first develops at 2 to 2.5 years, and at that time isolating dissociation starts with the shift-of-focus process. Children become nervous when their father acts angry, reminding them of previous abuse. They typically see themselves as negative $\mathbf{ME_{MEAN}}$, and so they try to shift to what

they believe their father wants, $\mathbf{ME_{NICE}}$. This kind of shift takes place normally in most children if their family becomes upset. For example, operating at the level of single representations (Level Rp1 in Figure 15.5), 2.5-year-old Jonathan heard his mother sounding angry, and he began to jump around, trying to get her to laugh and play. He tried to shift her emotion from negative to positive:

$$\left[\mathbf{ME_{MEAN}}\right] > \left[\mathbf{ME_{NICE}}\right] \tag{15.1}$$

In abusive families, parental upset often leads to abuse, and children watch vigilantly for a hint of negativity and then try to shift to the positive, such as $\mathbf{ME_{NICE}}$, and so avoid the negative, $\mathbf{ME_{BAD}}$ or $\mathbf{ME_{MEAN}}$. Through emotional splitting they try to avoid the bad.

$$\underline{\mathbf{not}}\ \left[\mathbf{ME_{BAD}}\right] \not> \left[\mathbf{ME_{GOOD}}\right] \tag{15.2}$$

In this first crude dissociation skill a child tries to suppress $\mathbf{ME_{BAD}}$ and works unconsciously to shift to $\mathbf{ME_{GOOD}}$, provided that the context supports it. If the context supports the opposite representation, $\mathbf{ME_{BAD}}$, children will have great difficulty sustaining the shift at this level, but they will gain more control as they develop.

Children of this age also start to distinguish between how to act in private (e.g., dealing with their family at home) and in public (e.g., climbing on the town's play structure). In this way, they shift between representations of private and public and thus begin to internalize differences in their public and private worlds. Children in public work to keep a focus on $\mathbf{ME_{GOOD}}$, reserving $\mathbf{ME_{BAD}}$ mostly for private settings (as parents can attest). That is, they avoid $\mathbf{ME_{BAD}}$ in public.

$$\left[\mathbf{ME\text{-}PRIVATE_{BAD}}\right] > \left[\mathbf{ME\text{-}PUBLIC_{GOOD}}\right] \tag{15.3}$$

When maltreated children differentiate public and private, they try to dissociate the two.

On the playground, they strive to keep in mind their private world, acting good and proper. They avoid acting out the private **ME**s that connect to their abuse at home. We represent this affective splitting with a bar through the symbol for shift of focus.

$$\underline{\mathbf{not}}\ \left[\mathbf{ME\text{-}PRIVATE_{BAD}}\right] \not> \left[\mathbf{ME\text{-}PUBLIC_{GOOD}}\right] \tag{15.4}$$

Isolating dissociation allows **ME-PUBLIC** to suppress and control **ME-PRIVATE** in contexts that support focus on the public sphere. The same limit was present for the isolating shift from $\mathbf{ME_{BAD}}$ to $\mathbf{ME_{GOOD}}$. Single representations have little power for overcoming context effects and so are not generally effective. The ability to relate representations using representational mappings (Level Rp2) gives children a stronger capacity to direct their own activities. That means that more stable dissociation requires the stronger structures that derive from coordination of representations into mappings, because they provide a mechanism for children to control that mapping, shifting **MOM** from mean to nice.

$$\left[\mathbf{MOM_{MEAN}} \text{ —— } \mathbf{ME_{NICE}}\right] \tag{15.5}$$

Normally the capacity to sustain mappings develops at about 4 years and creates many new skills involving relations with other people, such as mother to father or mother to child (Fischer & Pipp, 1984). Children living in a situation of hidden family violence manipulate these skills frequently in their life in private. When 4-year-old Jason's mom scolded his father sitting at dinner, Jason drew attention intensely to the drawing he did earlier that day (taped on the wall near the table). This abrupt switch led his parents to recognize immediately that he was trying to change their interaction. Jason was attempting to direct his mother's attention to something happy.

At this age children show a surge in understanding role relationships, which gives children in abusive families a capacity to separate public and private roles, thus facilitating strong isolating dissociation of public and private. For example, a child can maintain a public pattern of interaction, such as acting out **ME-PUBLIC** playing with their father, even though the primary private relationship with him is abusive boss interacting with bad child:

$$\underline{\mathbf{not}}\ \left[\mathbf{ME_{BAD}} \overset{\text{private}}{\text{——}} \mathbf{DAD_{BOSS}}\right] \not> \left[\mathbf{ME_{GOOD}} \overset{\text{public}}{\text{——}} \mathbf{DAD_{GOOD}}\right] \tag{15.6}$$

By age 6 or 7 children can build representational systems (Level Rp3), giving them more reliable control over their public and private worlds. At school or on the playground they can shift from public to private modes depending on who is more powerful. Roger shows this movement to a higher developmental step, sharply distinguishing public versus private, as for example when he tried to impose his private mode on other children whom he thought he could dominate. At age 9 Roger made a conscious effort to please his teacher and earn her approval. Being watched by adults Roger was a model student, even helping teachers to

monitor class when they had to leave the room. However, out of sight of the teacher he was a ruthless bully, threatening other children to take their possessions or make them help him with homework, based on his assessment of their power. Approached by an older, stronger boy, he would yield. Threatened by another boy, he would cry and beg for mercy. Roger bragged that he had a good family, including his father's business, money, and community respect. He did not say that his father, Mr. S, was strict and unpredictable and his mother was visibly sad and passive. Mr. S expected the children to respond quickly when he made a demand, and he often grew angry. In private, he told his children that they were worthless misfits, and he routinely degraded his wife, even physically abusing her. She often cried in silence. At home, Roger was subjected to the hidden family violence pattern and dissociated public and private, as evident in his relationships at school. In public (with teachers watching) he was a good boy, seeking the teacher's approval. At home, he was submissive (a follower or victim) and obeyed his father's commands and was frequently punished. In school with no teachers watching, the private domain dominated: Roger would take on the role of either follower or bully, based on his judgment of the other child's power. Now social relationships were becoming increasingly complex involving (1) both public and private representational systems (Level Rp3) with multiple roles and (2) an isolating shift of focus between public and private systems:

$$\left[\begin{array}{c}\text{OBEY}\\ \text{ME-FOLL'R}\\ \text{BAD}\end{array}\ \overset{\text{private}}{\longleftrightarrow}\ \begin{array}{c}\text{DEMAND}\\ \text{YOU-BOSS}\\ \text{PUNISH}\end{array}\right] \gtrdot \left[\begin{array}{c}\text{GOOD}\\ \text{ME-STUD'T}\\ \text{OBEY}\end{array}\ \overset{\text{public}}{\longleftrightarrow}\ \begin{array}{c}\text{LIKE}\\ \text{YOU-TEAC'R}\\ \text{COMMAND}\end{array}\right] \tag{15.7}$$

Here Roger acted out the roles of follower and student, **ME-FOLLOWER** and **ME-STUDENT**. In appropriate contexts he could switch his role, acting as boss or teacher (**ME-BOSS** or **ME-TEACHER**) to someone else as follower or student (**YOU-FOLLOWER** or **YOU-STUDENT**). He could shift between follower or boss, student or teacher based on his judgment of context. By Roger's age, children work hard to skillfully deceive and lie (Lamborn et al., 1994; Saarni & Lewis, 1993). Deception is at the center of hidden family violence, with individuals lying to both others and themselves. Detecting lies requires relating representations to each other, what is true and what is a lie. Understanding a lie requires coordinating two representations in at least a mapping:

$$\left[\textbf{TRUTH} \text{ —— } \textbf{LIE}\right] \tag{15.8}$$

Children develop and detect lies with representational mappings and they master them with the next level of skills, representational systems. (Consolidation of skills often requires at least two skill levels.) For instance, Roger stole a key from James and then "helped" James by blaming the robbery on the least popular child in the class, Colin. To skillfully lie required coordinating several states of James' knowledge (Roger "helping" and Colin "stealing") with claims or actions by Roger (stealing the toy and blaming Colin) in a representational system:

$$\left[\begin{array}{c}\text{STOLE}\\ \text{ROGER}\\ \text{BLAME COLIN}\end{array}\ \overset{\text{lie}}{\longleftrightarrow}\ \begin{array}{c}\text{ROGER HELP}\\ \text{JAMES}\\ \text{COLIN STOLE}\end{array}\right] \tag{15.9}$$

Second Tier of Dissociation: Isolating Personalities

As Roger grew cognitively he transitioned to the next developmental tier, moving from representational systems to abstractions for people as personalities. As a result, his isolating dissociation became more skilled, as he improved his capacity for deceiving people and for skillfully separating public and private. By coordinating representational systems into abstractions (Level A1), he could use abstractions to coordinate concrete interactions across his public versus private domains. In his private world he coordinated two representational systems for abusive bossiness, one as boss to some other victim and a second one as victim to some other boss. We name the general personality characteristic "tyranny" to reflect his goal of being a tyrant in his family:

$$\left[\begin{array}{ccc}\text{OBEY} & & \text{DEMAND}\\ \text{ME-FOLL'R} & \longleftrightarrow & \text{YOU1-BOSS}\\ \text{BAD} & & \text{PUNISH}\\ \Updownarrow & & \\ \text{DEMAND} & & \text{OBEY}\\ \text{ME-BOSS} & \longleftrightarrow & \text{YOU2-FOLL'R}\\ \text{PUNISH} & & \text{BAD}\end{array}\right] \equiv \left[\begin{array}{c}\text{ROGER}\\ \text{TYRANT}\end{array}\right] \tag{15.10}$$

Roger elaborated these personalities using isolating dissociation to keep them separate as in this formula:

$$\left[\textbf{ROGER-PRIV}_{\textbf{TYRANT}}\right] \gtrdot \left[\textbf{ROGER-PUBL}_{\textbf{COMPETENT}}\right] \tag{15.11}$$

By age 16 he had firmly established his capacity for single abstractions and started to construct abstract mappings (Level A2), establishing his public personality clearly at school, and using it skillfully:

> At 16 years Roger was a successful sophomore in high school—on the student council and the track team. But he did not have any close friends and spent lots of time seeking attention from older student council members. He helped

the vice principal, whom he believed had the power to set the rules. Roger was influential in school politics, presenting himself as knowing the answers and handling issues better than others. He was directly cruel to younger students, and especially condescending to girls. Roger was quiet and withdrawn at home with his father, who remained rigid and cold. His father often confronted Roger, who whined and hung his head. With his mother he insulted her when his father was not at home, and one time he pushed her into a door. He bullied his younger brother to do many of his chores.

Roger is building abstract mappings that connect the characteristics of victim and tyrant in the private domain, and in the public he is building the image of competent student leader and satisfied authority, as in the following isolating dissociation skill:

$$\left[\mathbf{ROGER}_{\mathbf{VICTIM}} \overset{\text{private}}{\text{———}} \mathbf{FATHER}_{\mathbf{TYRANT}}\right] \succ \left[\mathbf{ROGER}_{\mathbf{COMPETENT}} \overset{\text{public}}{\text{———}} \mathbf{AUTH'TY}_{\mathbf{IMPRESSED}}\right] \quad (15.12)$$

With this skill Roger focuses on roles of both victim and competent leader. At the same time he can reverse his stance to take the role of tyrant to someone else's victim, as shown by his treatment of girls, his mother, and younger students:

$$\left[\mathbf{ROGER}_{\mathbf{TYRANT}} \overset{\text{private}}{\text{———}} \mathbf{MOTHER}_{\mathbf{VICTIM}}\right] \quad (15.13)$$

As Roger became an adult, he used his developing skills to create the isolating dissociative roles that he has sought all along. As he searched for a wife, he soon showed affective splitting in his treatment of the young woman he fancied.

At age 24 Roger demonstrated his competence and apparent leadership in his public self. He joined the local church and became a member of the Young Republican Club. He offered to volunteer to promote his town's business for the Chamber of Commerce, and he founded his own real-estate business. He started to date a 17-year-old named June, who had recently finished high school, having met her when she worked for him as a temporary secretary. Roger was highly attentive to her, buying expensive gifts and sending flowers almost daily. He said repeatedly how special she was and that she did not have a single fault. Roger grew increasingly controlling and possessive. He accompanied her to the hair stylist to tell the stylist how to do her hair. Telling June to throw out all the clothes in her closet, he insisted on picking her new clothes. He began to complain that June was a poor cook, had no sense of style, and was too naive to go out alone.

Roger was constructing more complex skills to control June in both private and public domains, continuing to dissociate his positive public competence and negative private tyranny. He was attempting to construct his own family with an unfortunate joining of love with tyranny. He strived to bring June into the vice of his isolating dissociation, letting her be part of his public one only as a perfect stereotype. In public, in contrast, he connected his general competence with doing good for his community, limited by his rigid control of his private tyranny. Thus, he built abstract systems (Level A3) to continue his isolating dissociation of the two worlds called Public and Private:

$$\left[\begin{matrix}\text{LOVER} & \text{private} & \text{LOVED}\\ \mathbf{ROGER} & \longleftrightarrow & \mathbf{JUNE}\\ \text{TYRANT} & & \text{VICTIM}\end{matrix}\right] \succ \left[\begin{matrix}\text{PROSOCIAL} & \text{public} & \text{GRATIFIED}\\ \mathbf{ROGER} & \longleftrightarrow & \mathbf{AUTH'Y}\\ \text{COMPETENT} & & \text{IMPRESSED}\end{matrix}\right] \quad (15.14)$$

Over time, Roger will build ever more complex skills of isolating dissociation that separate his public and private worlds. These are complex, sophisticated skills, and at the same time they are clearly dangerous and pathological. Unfortunately, Roger is creating hidden violence in his family as evidenced by his actions with June. For *hidden family violence*, the pattern starts with a maltreating family that dissociates their proper public world from their private, tyrannical one. Children growing up in this family are abused physically and emotionally, and sometimes sexually. From this contextual and affective dissociation, the children isolate public from private, and eventually their maltreatment promotes active dissociation, separating the public and private worlds.

Psychopathology Is Not Developmental Immaturity

This dynamic analysis of development of isolating dissociation questions the traditional view that psychopathology comes from developmental immaturity or regression. Whereas traditional approaches tend to presume that psychopathology reflects a relative lack of skills or some form of deficiency, a developmental perspective brings forth the possibility that maladaptive behavioral development involves advances in skill development, not simply immature development. In the case of maltreated children, such advances take the form of increasingly powerful and more complex types of dissociation. Most scholars and practitioners who study maladaptive development have not detected this complexity because they have not taken the viewpoint of their patients. Moreover, patients are

commonly observed under minimizing conditions because of absence of contextual support and presence of stress. People subjected to stress and abuse follow strange pathways that organize their worlds in radically different ways, and which are characterized by increasing sophistication and complexity—and not regression or simplicity.

Modeling the Growth of Dissociation and Coordination

Dynamic mathematical modeling, based on general principles of dynamic interactions between variables, can provide deductive explanations of empirical observations. They are deductive explanations in the sense that they are derived from basic principles of developmental change and are expressed as mathematical rules generating descriptions of developmental trajectories. The strength of dynamic modeling lies in their power to deductively explain—on the basis of a small set of general dynamic principles of change—a broad range of qualitative properties that are typical of the phenomena that we can empirically observe in a particular domain such as developmental psychopathology.

According to Weisstein (1991), a dynamic system is a formal way of describing how one state (of a system) develops or changes into another state over the course of time. A highly simplified mathematical representation of a dynamic system is as follows

$$x_{t+1} = f(x_t)$$

which can be read as: the next state—at time $t+1$—of a particular variable x is a mathematical function, f, of the current state, namely the state at time t.[1] If the function f is a multiplication by a fraction such as 0.02, the dynamic model would represent stepwise exponential growth, such as money in a savings account. We have seen that most dynamic systems take the form of interactions among many components or variables. Such interactions can be modeled through the coupling of change equations.

In order to dynamically model the developmental process occurring under traumatic conditions, we shall assume that skills may vary between 0, meaning that the skill is totally absent, and some value around 1, meaning that the skill is optimally or maximally developed. Hence, for a psychologically healthy person, the value of the coordination skill may be about 1, and that of the dissociation skill about 0, meaning that there are hardly any contexts that cause dissociation in this healthy person. For young children, coordination and dissociation may still be a developmental issue, and they may still be sensitive to transient (nontraumatic) contexts in which they tend to solve the situation by dissociation (e.g., by lying about things, while suppressing the fact that they know they are lying[2]). In the discussion on dissociation, we have seen that people who suffered from serious traumas may develop sophisticated and adaptive dissociation skills. In principle, these people may still have reasonable coordination skills, which means that there are still contexts in which the person's personality is not dissociated. However, it is likely that such skills would not reach the higher levels of abstractions or principles that are typical of coordination in psychologically healthy people. We can mathematically represent this situation by values of skill levels between 0 and 1, for example, around 0.5 (for the modeling, the exact details of these numerical values do not really matter as long as they express observable differences in skill levels).

In previous work, the authors have shown that there exists a powerful and yet relatively simple mathematical expression for defining the growth of dynamic skills (Fischer & Bidell, 2006; Van Geert, 1991, 1994; Van Geert & Steenbeek, 2005). This equation is the coupled logistic growth equation, which takes the following form. Let the value C represent the level of the coordination skill of a particular person at a particular moment in the development, and D the level of the dissociation skill. For simplicity, let us focus on the growth of coordination, which can be expressed as follows:

$$C_{t+1} = C_t + \underline{C_t * r_C * (1 - C_t/K_C)} - \underline{\underline{a * D_t * C_t}}$$

The equation says that the next level of coordination (e.g., after a day or after a week, dependent on the chosen step size) is equal to the current level of coordination plus a certain amount of change. This amount of change is the result of the growth of coordination on the one hand (the underlined part of the equation), and the competition with other skills, in this particular case the competition with the dissociation skill (the double underlined part of the equation). The growth part of the equation specifies

[1] The step size +1 can be given any temporal value, such as a second, a day, or a month, but it can also be given a value that approaches an infinitely small time step, in which case we shall call the above equation a differential equation, instead of a difference equation.

[2] See figure 15.8, illustrating the changing levels of coordination and dissociation as a consequence of childhood trauma.

change in coordination as a function of the current level of the coordination, a growth rate r and a carrying capacity K (Van Geert, 1991, 1994). Both the growth rate and the carrying capacity are an expression of the total underlying dynamic system, consisting of internal properties such as memory capacity, intelligence, information processing skills and external properties such as the family environment and experienced events. The carrying capacity is an important parameter since it is the summary of the entire underlying system, in terms of the total support it can give to this particular skill, namely coordination. In a well-functioning and caring family, this total support and hence the carrying capacity for personality coordination is likely to be much higher than in a badly functioning family characterized by many coercive interactions. The competition part of the equation describes the change of coordination as the multiplication of a competition parameter, a, the level of the competing skill, which in this particular case is the dissociation skill, and the level of the coordination skill itself. The equation says that the higher the level of the dissociation skill, the more the coordination skill will suffer from it, implying that level of the coordination skill will diminish over time, unless the competition can be counteracted by the growth of coordination, as it is influenced by all the personal and family factors that support it.

We can now present a similar equation for the growth of the dissociation skill, but with parameter values that relate to dissociation this time (for instance, in a well-functioning family the total of factors supporting the growth of dissociation will be very low compared with the total of factors supporting the growth of coordination).

$$D_{t+1} = D_t + D_t * r_D * (1 - D_t/K_D) - b * C_t * D_t$$

We shall now assume that if a serious trauma occurs in the life of a particular young person—for instance sexual abuse by the father as described in the earlier example—the total set of factors supporting dissociation as an adaptive skill is considerably increased. Hence, the carrying capacity for the dissociation skill, K_D, strongly increases as a consequence of traumatic events. In addition, the growth rate r_D of dissociation skill is increasing, because the child now feels the need for developing dissociation as a way of coping with the traumatic situation. A very simple way of mathematically expressing the effect of trauma is the following:

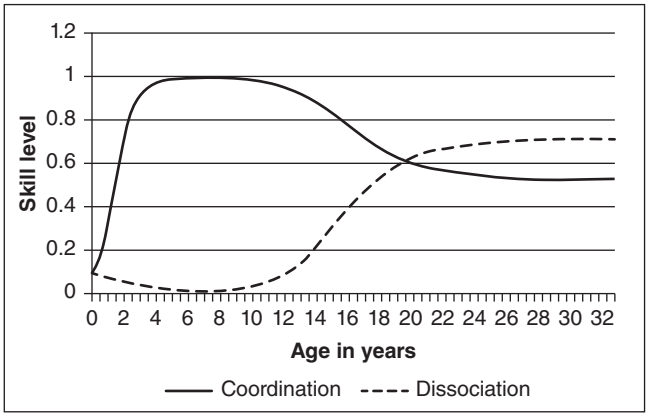
$$K_{Dt+1} = K_{Dt} + d * T$$

$$r_{Dt+1} = r_{Dt} * e * T$$

The factor T, which stands for the occurrence of traumatic events, is a parameter that is either 0 (when no traumatic events occur) or 1 (when traumatic events occur). The parameters d and e moderate the effect of trauma, and, like all other parameters, are specific for a particular individual child in a particular environment. For instance, children with a high level of resilience will be less affected by a particular trauma than children with a low level. In a family, where one of the parents is highly supportive of the child, a trauma caused by the other parent might eventually have less effect on the growth of dissociation of the child in a family where the mother for instance tacitly supports the abuse by the father. Since all parameters are individual and environment specific, we obtain an entire space of possible parameter values that represent possible individual cases.

We can now use this dynamic systems model of coordination and dissociation skill growth to study the variety of individual trajectories that results from various combinations of parameters, including the various family support levels, growth rates that are dependent on individual personality characteristics, competition factors and factors influencing the effect of a particular traumatic experience.

Figure 15.8 shows the simulation of two trajectories of coordination and dissociation skills in a child who suffers from serious traumatic abuse from the age of about eight years on (the models step size is one month). The top graph shows a trajectory of dissociation following trauma at around the age of eight years which stabilizes well into adulthood and becomes the sole way of coping for the person in question. The bottom graph shows a trajectory after a similar trauma that shows a pattern of stabilization between coordination and dissociation, with coordination still active, but with dissociation as the dominant mode of adaptation. This latter pattern implies that there still exist enough contexts in which the person can use personality coordination skills, for instance in the context of therapy where the person is able to deal with the consequences of trauma in a healing way. Surprisingly enough, the only difference between these two scenarios is the value of the growth rate parameter for coordination in the second case, which is slightly bigger than in the first case. That is to say, the person's slightly bigger ability to build up coordination skills—which is represented by the value of the growth rate parameter—functions as a strongly protective factor, in that the situation is still maladaptive, but less so than the complete dissociation of the first example. This provides but one of the many illustrations of the nonlinear character of dynamic systems, where the effect



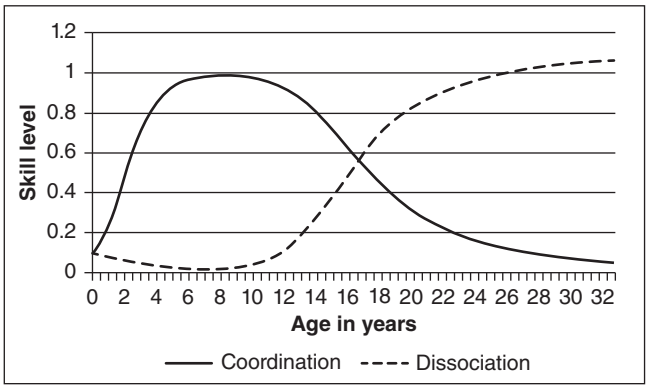


Figure 15.8 Modeling of two scenarios of dissociation-coordination after trauma occurring at 8 years of age; trajectories are determined by dynamic interactions between coordination and dissociation.

of changes in certain parameters strongly depends on the total constellation of the system including the values of the parameters.[3]

SELF-SUSTAINING LEARNING TRAJECTORIES IN CHILDREN WITH ATTENTION AND EMOTIONAL-BEHAVIORAL DIFFICULTIES

What is true for maltreated children is also true for other children who exhibit maladaptive patterns of thinking, feeling, and acting. Both adaptive and maladaptive patterns of psychological activity undergo development and assume different forms over time. Dynamic systems models contribute to understanding the ways adaptive and maladaptive patterns of thinking, feeling, and acting develop in children who exhibit attention and emotional-behavioral difficulties (EBD). In this section, we examine how dynamic systems models can be used to understand the processes by which learning fostered and thwarted in children with attention and emotional-behavioral difficulties.

Context Sensitivity of Attention Problems

Drawing upon the medical model, the concept of ADHD is typically understood as a fixed and trait-like property of individuals. Traditional models maintain that attention difficulties are products of primary deficits in the capacity to sustain attention, inhibit behavior, executive functioning, regulation of motor behavior, and related central deficits (Nigg, 2006). However, from a dynamic systems perspective (see Figure 15.2), all behavior must be analyzed in the context in which it occurs. Behavior—whether adaptive or maladaptive—is not a *thing* that a person *has*, but instead is an ongoing *process* that emerges from interactions between individuals and their contexts. Further, research supporting the idea that ADHD classified behavior is the direct product of central cognitive deficits has been mixed. Research suggests that primary deficits in the capacity to sustain attention, inhibit impulses, regulate motor action, etc., apply only to subsets of children exhibiting ADHD classified behavior (Sonuga-Barke, Wiersema, van der Meere & Roeyers, 2009).

Recently, theorists have elaborated alternatives to fixed deficit models of ADHD. For example, *delay aversion* (Castellanos et al., 2006) and *state regulation* deficit (van de Meere, 2005) models suggest that both adaptive and maladaptive behavior in children exhibiting ADHD behaviors are context-dependent products of relations between motivational resources and task demands. Among such children, overactivation of motivational resources occurs in the context of challenging tasks, whereas underactivation occurs in less challenging tasks. In structured contexts, underactivation can produce attentional drift and motivate stimulus seeking (e.g., fidgeting, heighted motor movement) accompanied by a preference for immediate rather than delayed forms of gratification, thus evoking impulsive behavior. Overactivation of arousal under high task demands can result in failure to inhibit prepotent behavior patterns (Sonuga-Barke, Wiersema, van der Meere, & Roeyers, 2009). Support for these ideas comes from studies in which children are asked to produce or inhibit simple responses (e.g., pressing or inhibiting a button press) under conditions that vary the speed with which children are required to respond. In comparison to controls, children with ADHD classifications make more errors under both

[3]To enable the reader to experiment with the complex interactions between the parameter values and the resulting effects on the developmental pattern, we shall provide a simple Excel model that can be downloaded at (http://www.paulvangeert.nl/articles_appendices.htm).

high (i.e., short deadline to respond) and low (i.e., long deadline to respond) task demands but not in response to moderate demands (Benikos, Johnstone, & Roodenrys, 2013a & b; van der Meere & Stemerdink, 1999; van der Meere et al., 1995).

Such findings suggest that task performance in children who exhibit ADHD behavior arises not simply as a product of general, trait-like deficits, but instead from the dynamic coupling of child and task demands. Children performed as well as controls in tasks that fall within an *optimal range* of difficulty; beyond that range, performance dissipated beyond that observed in controls. These studies suggest that inattentive, hyperactive and impulsive behavior are not linear results of central deficits but instead emerge through the self-organization motivational, affective and cognitive processes as they adjust to each other in response to local task demands.

The Strengths and Limitations of Neurobiological Approaches

Students with emotional and behavioral difficulties (EBD) are mainly known for problems involving so-called internalizing and externalizing behavior (Lane, Wehby, & Barton-Arwood, 2005). Many children with emotional and behavioral difficulties also exhibit difficulties in attention (e.g., ADHD). In recent years, however, increasing evidence also points to the negative academic outcomes typical for these students (Lane, Barton-Arwood, Nelson, & Wehby, 2008). Students with EBD continue to display significant academic delays across all placements (Reid, Gonzalez, Nordness, Trout, & Epstein, 2004) and do not seem to improve over time (Lane et al., 2008). These students thus require extra attention to support the development of academic performance. Unfortunately, the academic needs of this population continue to be unmet (Hayling, Cook, Gresham, State, & Kern, 2008).

Research assessing the academic needs of EBD children has focused on the neurobiological shortcomings that mediate, among other problems, lags and delays in their academic performances. In addition, research also examines how to ameliorate these shortcomings using pharmacological (and sometimes nonpharmacological) treatments (e.g., Hoekzema et al., 2010). Overall, neurobiological research about ADHD has yielded important insights (Castellanos & Tannock, 2002; de Zeeuw, Weusten, van Dijk, van Belle, & Durson, 2012; Tomasi & Volkow, 2012). Cortese (2012) provided an overview of anatomical, functional, neurophysiological, neurochemical, and genetic processes in the development of ADHD behavior. A primary conclusion consists of the finding that the brains of many children with ADHD differ from those of controls. Further, the processes that mediate ADHD behavior are both complex and heterogeneous, and are unlikely to be accounted for using simple pathophysiological models. As a result, there is a need to develop models that assess patterns of altered connectivity among multiple brain areas in individuals who exhibit ADHD behavior, rather than in terms of the localization of functional deficits in specific brain sites (Cortese, 2012).

Despite the importance of research assessing the neurobiologial contributions to attentional and emotional-behavioral problems, there are limitations to the neurobiological approach. First, until recently, findings from neurobiological research have produced few nonpharmaceutical interventions to address the behavioral and academic performance of ADHD and EBD classified children (Cortese, 2012). Teachers and practitioners face the task of interacting with whole children, and not simply their brains. As a result, there is a need to understand how neurobiological processes operate as but one important layer of functioning within the multinested epigenetic person–environment system. Second, within the neurobiological approach, neuro-biological deficits are often depicted as the primary causes of children's maladaptive actions. As a result, the complex dynamics that operate between child and context are excluded from theoretical and empirical consideration. This neglect is a problem, as child and social context (i.e., teachers, cultural tools, learning and motivation systems) are inseparable as causal processes in the development of children's attention and learning (Fabiano et al., 2013; Raver et al., 2008). An exception to this rule involves increasingly rich analysis of gene–environment interactions (G x E interactions) in the development of attentional and socioemotional problems (Nigg, Nilolas, & Burt, 2010; Pennington et al., 2009). A full accounting of the origins and development of attentional and emotional problems must build on to show how specific genes coact epigenetically with multinested environments to produce variation in attentional and behavioral problems.

Setting the Stage: The Dynamics of Teaching and Learning

Learning processes operate as socially situated, transactional processes (Steenbeek & Van Geert, 2013). To understand the process of teaching and learning involving children with attention and emotional difficulties, it is necessary to examine how learning is fostered or thwarted as a product of coactions that occur over time among aspects of the person–environment system. Such analyses

require examining the (1) structure of a student's attention and action as it arises (2) in coregulated exchanges that occur between (3) teachers and students (4) using cultural tools and practices as they operate (5) within particular sociocultural contexts. In this section, we elaborate a framework for understanding how teaching and learning operate as socially situated, transactional processes.

The Nature and Importance of Coactive Scaffolding

Students learn through their participation in sociocultural activities. Most often, successful learning occurs under the guidance and direction of more competent others who organize instruction, guidance, motivation, and emotion within a child's zones of optimal learning (Mascolo, 2013; Van Geert, 2008). An important means for teachers to help students to learn is to make use of coactive *scaffolding*. Scaffolding consists of the process of providing a student with external support and assistance, and optimally, discarding this support after learning has taken place (Granott & Parziale, 2002; Wood, Bruner, & Ross, 1976). Scaffolding is an intrinsically dynamic notion. We use the term to refer to any process that operates outside of the direct control of the individual over a given unit of behavior that functions to raise an individual's level of function beyond that which he or she is capable of performing him or herself (Mascolo, 2005). This principle is similar to the distinction made above between optimal and functional levels of skill that arise in contexts that provide high and low levels of contextual support. As discussed already, optimal level performance is that which a child can sustain under conditions of high contextual support; a child's functional level is the level at which he functions in everyday contexts in the absence of such support.

However, the process of *scaffolding* is different from the provision of *high support*. In contexts involving high support, a child performs a task on his own *with* contextual support but *without instruction or assistance during the actual execution of the task*. For example, a teacher may provide high support by *first* showing a child how to perform an addition problem that is beyond the child's ability, and *then* asking the child to perform the addition problem *on his own* without additional instruction or support. In contrast, when a teacher or adult scaffolds a child's performance, he or she often *performs part of the task for the child* (so the child can complete the remainder), or otherwise provides directives and cues to organize the child's actions as the child is actually carrying out the task. Scaffolding occurs, for example, when a teacher provides ongoing instruction (e.g., Now carry the one) or cues (e.g., What's next?) without which the child would be unable to complete the task himself.

The level of functioning that children can sustain under conditions of scaffolding tends to be higher than their optimal level performance under conditions of high support. Social scaffolding functions by breaking down a child's unmanageable task into more manageable units, thus allowing a child to coordinate novel aspects of a task in vivo—during the process of learning by doing. Scaffolding thus has the effect of holding up novel configurations of a child's actions so that the child can coordinate those actions for himself even though he could not coordinate them by himself previously. Empirical studies have shown that the process of scaffolding (1) occurs on a widespread scale, (2) is effective in that it advances learning, and (3) has different forms (i.e., that it differs among individuals and contexts; see, e.g., an overview of scaffolding in the context of learning disabilities; Stone, 1998). In special education, the quality of scaffolding can be hampered by the ways behavioral problems interfere with the teacher's actions (Wehby et al. 2003). This tends to occur because teachers are insufficiently trained to cope with problems in their scaffolding activities.

Embodiment, Coaction, and the Initial Conditions of Learning

An important feature of our approach is that we conceive of learning as an embodied process. Representational development occurs within the medium of the brain and body and is organized by context sensitive interactions between cognition and affect (Figure 15.2, Point 3). In any given context, the development of higher order representational activities can occur only under conditions of optimal affective engagement (see Yun-Dai, 2012). Affective engagement, in turn, is maintained when representational content remains rich, varied and accessible over the course of the teaching process. Secondly, in our view, the quality of teacher-student interactions cannot simply be perceived as having a mere *influence* on the ADHD child's learning process but instead functions as a changing indicator of the types of dynamic influences that shape an ADHD child's learning—through both desired and undesired pathways. Over time, iterations in teacher-student interactions consolidate into mutually responsive relationships. In this way, interaction dynamics (direct, immediate, short-term processes) then become a proximal measure of many distant factors.

While research indicates that brain-related constraints on learning are present in children who exhibit ADHD behavior (Cortese, 2012), from a dynamic systems

perspective, such constraints should be viewed as the *initial conditions* for the learning process, and not necessarily fixed, stable, or deterministic factors. In principle, these starting conditions can be changed by designing interactions that focus on the neuropsychological strengths and weaknesses of particular children, and by providing cognitive and affective experience that falls within the optimal range of learning of individual children (Immordino-Yang & Damasio, 2007; Vinogradov, Fisher, & de Villers-Sidani, 2012). Over time, the consolidation of repeated short-term representational and affective experience yields long-term changes in the neural networks of the brain and corresponding changes in the child's skill repertoire (John, Bullock, Zikopoulos, & Barbas, 2013; Winkielman, Niedenthal, & Oberman, 2009). It is only through analysis of sequences of learning over time that we can understand the circumstances under which neurological and brain-based constraints remain stable, or eventually change for the better or the worse. To trace the developmental consolidation of such processes, it is necessary to have access to densely sampled serial indicators of learning and action that occurs between EBD children and their teachers.

Learning as an Interactive, Microdevelopmental Process. From our perspective, learning, which is often defined as a macrolevel variable operating over the long-term—is best studied as a microlevel process. That is, it must be studied during individual instruction sessions that occur within the learning triangle of teacher–material–learner (Figure 15.2). By tracking moment-by-moment teaching and learning over time, it is possible to identify how short-term triangular dynamics are consolidated into long-term learning trajectories. Toward this end, microgenetic methods have proved their value (Basseches & Mascolo, 2010; Siegler, 2006; Van der Aalsvoort, Van Geert, & Steenbeek, 2008) and are now broadly recognized as particularly suitable for studying learning as a transactional, coregulated process (Granott & Parziale, 2002; Mascolo, 2013; Siegler, 1995; Siegler & Stern, 1998).

An important mechanism of change in this microgenetic process is *iteration*, that is, the fact that learning takes place in the form of iterative steps, which can be modeled as a series of successive operations, in which the output of the preceding operation provides the input of the next one (Van Geert, 1991, 1994, 2008). In an iterative sequence, each previous action of the student has an influence on the subsequent action of the teacher, and vice versa, indicating the cyclic character of iterative causality. Teacher–student relationships evolve as successive iterations and tend to consolidate into stable structures (Bierman, 2011; Luckner & Pianta, 2011). The formation of mutually responsive teacher-student relations has a direct impact on student attitudes, which thereupon foster enhanced peer acceptance and positive peer reputations, which in turn promote increased student engagement. Especially at-risk students may have particular benefits from sensitively structured classroom climates (Bierman, 2011; Gazelle, 2006; Hughes & Chen, 2011).

When studying learning-in-interaction as a dynamic, complex process, it is fundamental to capture it by means of a variable that reflects the important characteristics of the dynamics of that process. This variable is called an *indicator variable*, in that it captures some of the most characteristic qualitative properties of the underlying process. Indicator variables are means of representing the dynamics of a system without aiming to approximate a complete or exhaustive description, which, in the case of complex processes often hampers our understanding. Instead of examining the roles of the interaction partners separately, it is important to take the iterative action–reaction responses of both partners together as defining variable (see also Fogel, 1993, 2009). One way to assess such dynamics is by focusing on scaffolding. By way of illustration, we present two studies on scaffolding dynamics in special education, involving individual math instruction with children exhibiting ADHD and related problems.

Scaffolding Dynamics During Arithmetic Lessons

We examined the dynamics of short-term interaction between teachers and students during arithmetic lessons. In particular, we focused upon the responsiveness of teachers and students over the course of one-to-one math instruction. Within any single initiation-response interaction, we defined responsiveness in terms of whether the responding utterance followed meaningfully from the content of the initiating utterance. Responses that connected meaningfully to initiating utterances (responsive utterances) were classified as matches, and those that were unrelated to initiating statements (unresponsive statements) were identified as mismatches. Matching and mismatching utterances could occur in either direction between teacher and student (i.e., teacher → student, student → teacher). Thus, response matches functioned as the indicator variable in this study. We also identified teacher and student self-iterations. Teacher self-iterations refer to statements made by the teacher subsequent to a self-initiated utterance (e.g., after a short wait, answering

a question that she had asked the student). We assumed that variations in scaffolding quality would be represented by variations in responsiveness over the course of teaching and learning. As it is difficult for even experienced teachers to provide clear instructions that maximize student self-regulation, we expected that the interaction dynamics between teachers and students would show evidence of disruption and mismatch over time. Participants included four primary school students exhibiting emotional and behavioral problems (mean age was 9.7 years) in a small town in northern Netherlands. The case focuses on one of these students (W) who exhibited behavioral problems as well as problems with attention, concentration, and impulse regulation. The data took the form of videotaped arithmetic lessons delivered biweekly over a 2-year period (32 lessons).

Figure 15.9 shows the number of matches, mismatches, student self-iterations, and teacher self-iterations expressed in the scaffolding dynamics of student W and his teacher over a 2-year period. As indicated in the figure, each variable showed substantial fluctuations over time. These patterns do not substantially differ from fluctuations that appear in the data of the other three EBD students. (This finding is based on an overall variability measure and Monte Carlo permutation analysis, $p = 0.66$.) Focusing on changes in the percentage of matches over time, two basic phases can be distinguished. Matches occurred more frequently in the first half of the observational period than in the remaining half. A change point analysis revealed that lesson 12 marked a discontinuity in the mean number of matches observed. A Monte Carlo permutation test shows that the difference is statistically significant ($p < 0.01$). In addition, the standard deviation of the matches became significantly reduced after fragment 17 ($p = 0.02$), marking a second transition point. Thus, the variable M showed a nonstationary pattern, with discontinuities around fragments 12 and 17. These discontinuities suggest changes in the attractor state of this student–teacher system.

Figure 15.10 represents the scaffolding dynamics between W and his teacher as reflected in changes in matches, mismatches, teacher self-iterations, and student negative responses over the 2-year period. Figure 15.10 shows that the drop in matches around Lesson 12 coincides with a gradual increase of mismatches and in the level and variability of the teacher's self-iterations. Thus, over the long term, relations among matches, mismatches, and teacher and student statements shifted from one attractor pattern to another. The shift is one from responsive, coherent, and contingent interactions prior to Lesson 12 to increasingly less responsive, coherent, and contingent interactions thereafter (see Steenbeek, Jansen, & van Geert, 2012).

Figure 15.11 identifies the number of math assignments that the child was successfully completing over the course of time. The figure provides a graphical illustration of the complexity of the relationships among response matches and self-iterations on one hand and the amount of productivity on the other. The patterns demonstrate a *decrease* in the quality of the scaffolding between child and teacher coincided with *progress* in math performance, as indicated by the number of assignments completed.

Our main question concerned what the short-term scaffolding dynamics would reveal about the different phases identified in the previous section. Figure 15.12 shows the sequences of the variables match, mismatch, teacher self-iteration, and student self-iteration in two prototypical instruction sessions between W and his teacher. The first session (left panel) occurred before the first change

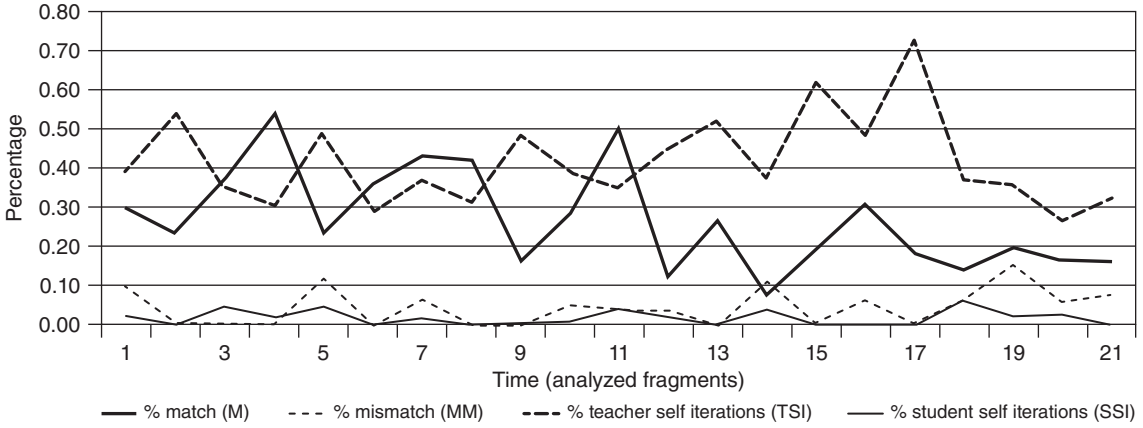


Figure 15.9 Changes in matches, mismatches and self-iterative utterances between a child and tutor in mathematics instruction.

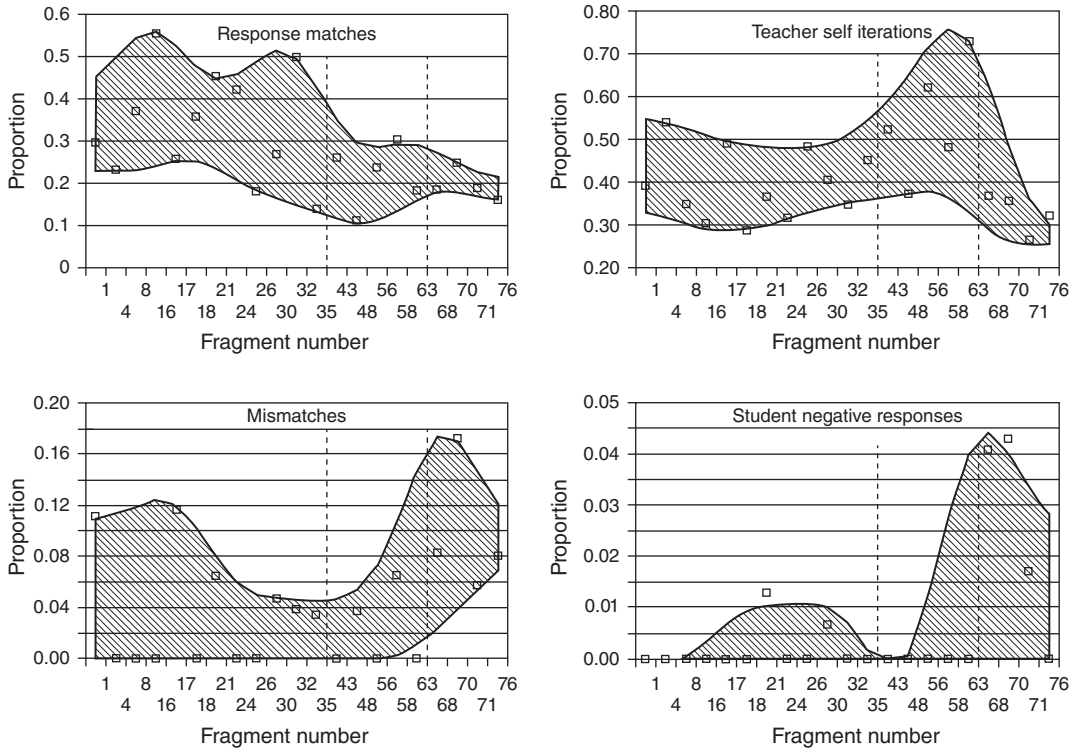


Figure 15.10 Independent analysis of growth for matches, mismatches, teacher self-iterations and student self-iterations.

point and represents a typical responsive session, the second session (right panel) occurred after the second change point, and represents a prototypical nonresponsive session. Figure 15.12 shows transition diagrams for both sessions. As indicated in Figure 15.12 (Session 4, upper left), in a prototypical responsive session, most of the self-iterations were produced by the teacher. This pattern was repeated in the prototypical nonresponsive session (Session 19, upper right). As indicated in Session 4 (Lower Left Corner), in the prototypical responsive session, when a match occurred (91% of the sequences), it was almost always followed by another match (93% of the sequences). In addition, the prototypical responsive session contained no mismatches. In contrast, in the proto-typical nonresponsive session (Session 19, lower right), when matches occur (41% of sequences), they were followed by a match in only 41% of the sequences, a mismatch in 41%, and by a neutral match in 15%. Thus, there were more vacillations among match, mismatch, and neutral sequences in the nonresponsive than in the responsive session ($p < 0.01$).

Thus, although the match–mismatch ratio differs substantially across the two lessons, the ratio of teacher self-iteration and student self-iteration stayed the same. This latter observation points to the stable and inactive role of the student during the instruction sessions. These findings support the idea that the interaction dynamics in EBD student–teacher dyads make it difficult, even for highly trained teachers, to motivate and enhance the use of self-regulatory strategies in EBD students.

Arithmetic Learning Among EBD and Typically Developing Students

Many studies suggest that EBD students show lags and delays in their academic performances, and that instructional practices can play an important role in reducing these delays. Several types of instructional behavior are effective for EBD students. These include providing clear and explicit instructions, enhancing opportunities to respond to questions, teaching the effective use of self-talk and self-regulation strategies (Kroesbergen & Van Luit, 2003; Sutherland, Alder, & Gunter, 2003). In addition, error correction, consistent feedback, individualized instruction, enhanced levels of instructional talk, and prompts for responding help increase student engagement and learning (Conroy, Sutherland, Snyder, & Marsch, 2008).

Steenbeek, Jansen, and van Geert (2012) examined the arithmetic performance of a group of five EBD students in comparison with that of a group of five typically developing students. Student performance on arithmetic

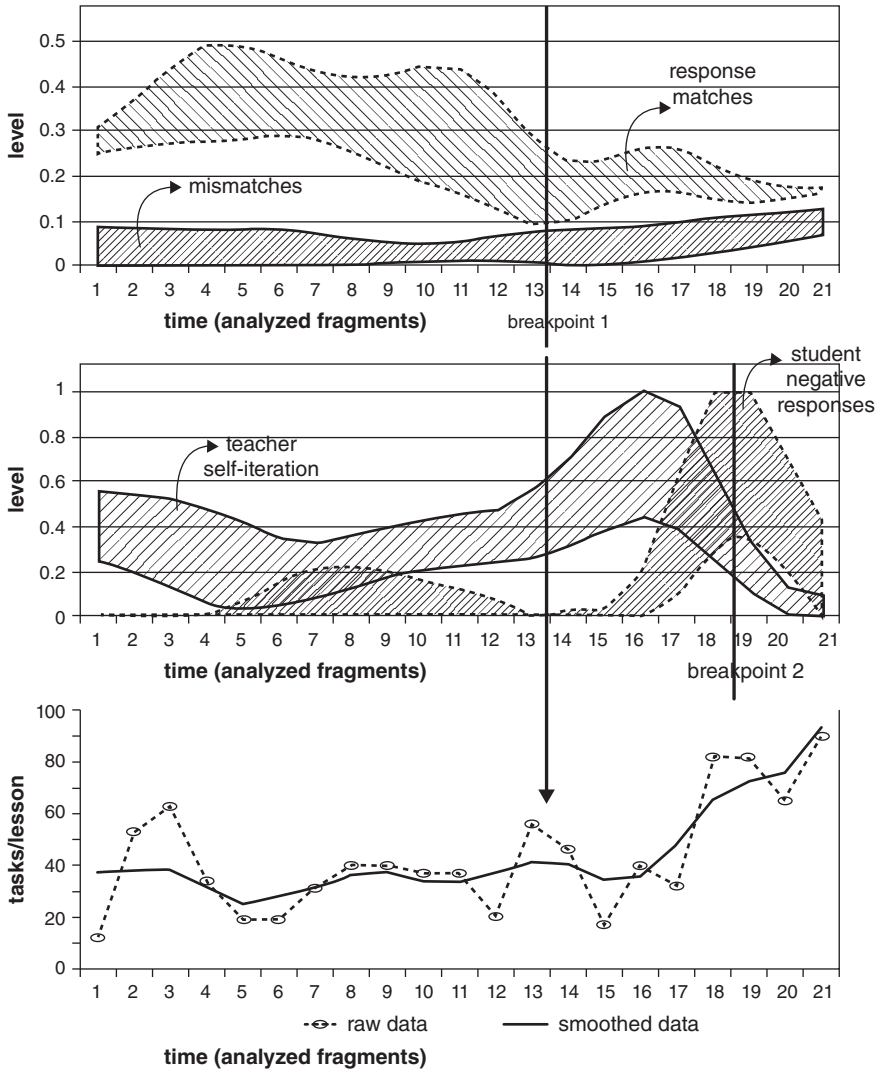


Figure 15.11 Relation between changes in matches, mismatches and self-iterations and the growth of mathematical performance.

assignments ordered with respect to increasing complexity over a 2-year period was assessed. We expected that EBD would exhibit lower levels of performance but higher levels of inter-individual variability than typically developing students. We also expected that typically developing students would make progress at a uniform pace consistent with the class-based nature of instruction. Figure 15.13 shows changes in arithmetic performance for each group over a one-year period. As expected, typically developing students successfully completed more assignments than the EBD students.

Further, as indicated in Figure 15.13, the growth curves for EBD children show greater inter-individual variability than those for the typically developing group. Although the growth curves for the typically developing group began at the same intercept, the pace of growth, as indicated by the slope of the curves, differed from individual to individual. In contrast, the growth curves for the four EBD students differed in terms of their intercepts (i.e., the level of complexity at which students began the instruction) but were similar in terms of their slopes (i.e., learning pace). Similar slopes for EBD students occurred even though these students followed individualized learning courses in the context of individualized scaffolding situations.

Self-Sustaining Trajectories in Suboptimal Learning Dynamics

These patterns of results point to the development of a self-sustaining dynamic of suboptimal educational

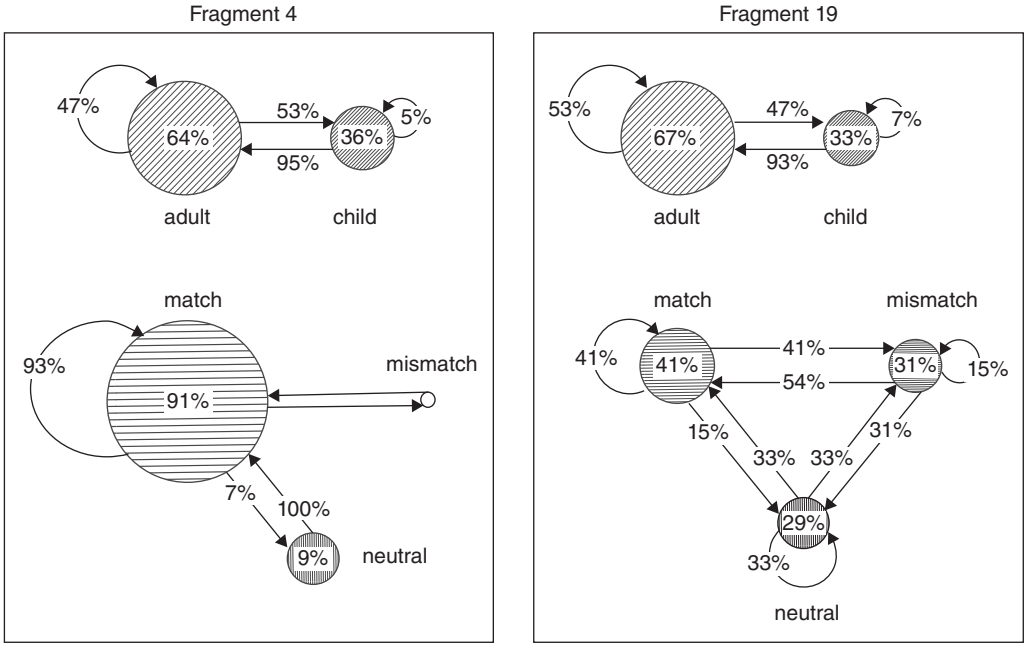


Figure 15.12 Interaction dynamics in prototypical responsive and nonresponsive learning sessions with a boy with emotional and behavioral difficulties.

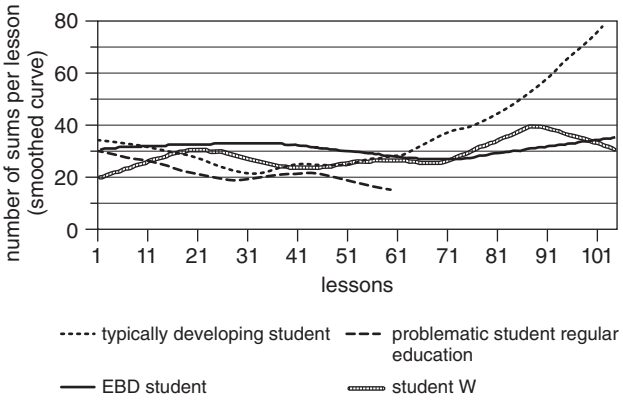


Figure 15.13 Growth of mathematical performance in EBD children.

functioning in which teacher's overinvestment and the student underinvestment support and maintain each other over time. Teacher overinvestment comes in the form of sustained patterns of scaffolding over time and through her willingness to bridge the gap between a series of assignments missed by the student. The student's underinvestment is indicated by poor effort and a lack of interest and motivation. This pattern is supported by a highly unbalanced surplus of self-iterations produced by the teacher in comparison to the student. The teacher continuously produced long utterances and tended to answer most of the questions she posed to the student. In contrast, the student maintained the strategy of responding with short answers (e.g., yes, no, or I don't know). Response matches—a measure of responsivity—had their origins in the teacher's effort to match the student, and not vice versa, and declined during the second part of the 2-year period.

This self-sustaining suboptimal pattern of teacher overinvestment likely has its origins in the teacher's desire to ensure *short-term* continuity in instruction, which, in the long term, proves problematic. Teachers know that students are easily distracted and tend to react to frustration in terms of negative emotion. Sensitive to the emotional reactions of their peers, negative emotions, and disorder easily spread through the class. Hence, there was a strong desire of the teacher to orient students toward their work without too much frustration in order to create room for another student to receive individual instruction. Teachers were well aware of this self-sustaining dynamic. In this way, suboptimal teaching and learning processes had their origins in the ways children's difficulties influenced the classroom dynamics, and not by a lack of quality among the teachers. These suboptimal interactions reflect a dynamic process in which student difficulties reciprocally affect teacher activities in ways that perpetuate and amplify a downward spiral in teaching and learning (Van Geert & Steenbeek, 2005).

Modeling School Performance in EBD Students

It is possible to describe the performance of EBD students using a dynamic model of learning and teaching called the dynamic scaffolding model (Van Geert & Steenbeek, 2005). The model starts with the assumption that a student's learning and a teacher's teaching can be represented by means of the same ruler. For instance, a particular student's current level in math can be represented on a measurement scale that specifies mathematical ability. The student's teacher provides instruction and scaffolding in the form of assignments, explanations, questions and so forth. The teacher's instruction will target a particular level of mathematical ability from the student. The level of teaching can, in principle, be represented on the same developmental ruler, (e.g., a dynamic skill ruler) as the child's mathematical ability.

While the student's progress strongly depends on the teacher's instruction, a teacher's instruction can be effective only if it operates on a level that is slightly higher than the level currently achieved by the student. As the student learns, the teacher should adapt his level of instruction to the new skill level of the student. Learning and teaching are dynamically coupled processes. Thus, changes in learning depend, among other factors, on the level of teaching provided, whereas changes in teaching depend on the amount of learning accrued from preceding teaching activities. For the teacher to adapt, on the one hand, his teaching to the level of the student, and, on the other hand, to the progress made by the student, the teacher must be able to assess the student's level and progress in real-time. Thus, if the student's progress is observed as being low, the teacher will hardly, if at all, modify the teaching level.

Students differ in terms of the distance between their actual levels of functioning and the levels of teaching under which they optimally learn. In comparison to intellectually advanced students, students with learning difficulties will exhibit smaller distances between their actual levels of functioning and the levels of teaching under which optimal learning occurs. Hence, while teaching, a teacher must not only assess the students' levels of functioning and their degree of progress, she must also estimate the optimal distance between their levels of functioning and the level of teaching under which students can make optimal progress.

The general mathematical properties of this teaching-learning model rely on the logistic change model. In so doing, they also rely on couplings between a student's level of skill, a student's progress in learning, and the level and progress in the teacher's instruction (see van Geert & Steenbeek, 2005; see also Van Dijk et al., 2013). As far as EBD students are concerned, we may assume EBD students profit more from a relatively small distance between their actual level of functioning and the level of instruction. In the studies described above, we have also seen that the teachers of EBD tend to avoid strong challenges and in general stay rather close to the students' actual levels of functioning. As a result, student learning remains suboptimal. EBD students are "under-challenged," as student and teacher get stuck in a mutually influencing process of underachievement and under-challenge.

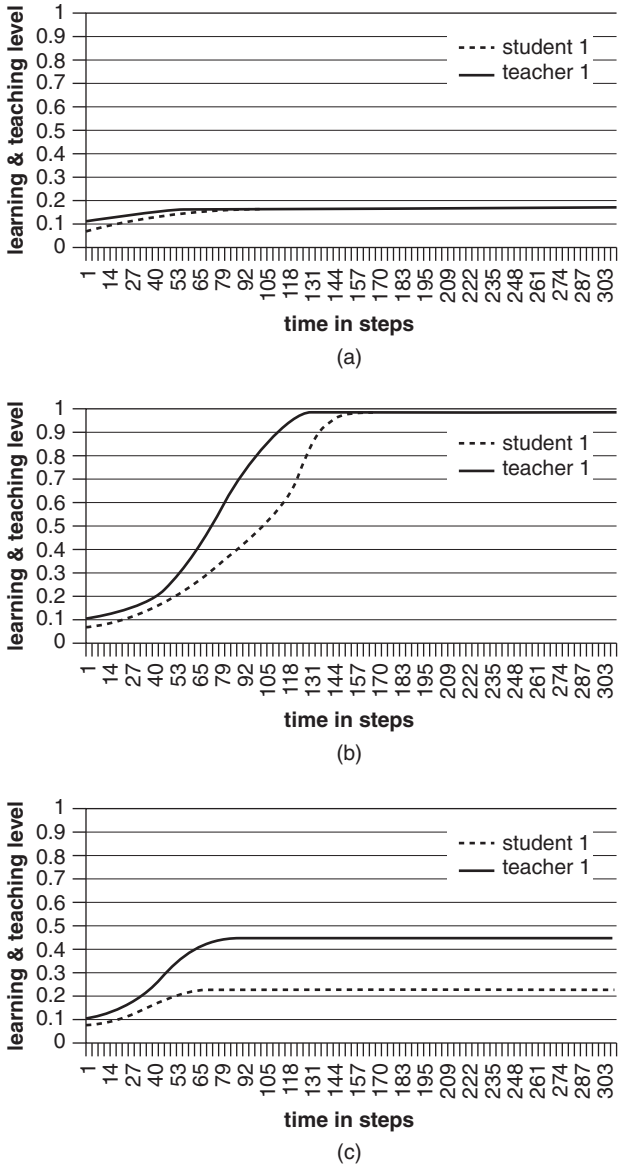


Figure 15.14 Modeling growth in teaching and learning with children with attentional and emotional problems.

The dynamic model predicts that if teachers tend to stay too close to the student's actual levels of performance, the process will lock itself into a pattern where no real learning progress is made (Figure 15.14a). If the teacher attempts to force learning by presenting challenges above a student's optimal level, there will be a similar self-sustaining process of suboptimal learning (Figure 15.14c). However, the model also predicts an intermediary window where the level of teaching and challenge provides an opportunity for optimal learning (see Figure 15.14b). The simulations based on the model show that the rate of student learning (which, along with other factors, is a function of the student's effort and motivation) is also a critical factor. Lower rates of learning destroy the window of possible opportunities and create self-sustaining processes of underachievement that are difficult to change. In short, what the model deductively demonstrates is that among EBD students, learning opportunities are fragile and dependent on the teacher's capacity for sensitive adaptation. The fragility of the teaching-learning dynamics, which the dynamic model clearly demonstrates, can explain why so many children in special education perform at lower intellectual levels than might be expected on the basis of their actual intellectual abilities.

THE DEVELOPMENT SOCIOEMOTIONAL ADJUSTMENT IN CHILDREN WITH EMOTIONAL AND BEHAVIORAL DIFFICULTIES

Thus far, we have examined idiosyncratic pathways in the macro-development of dissociative structures among maltreated individuals as well as microdevelopmental trajectories of teaching and learning involving children with attention difficulties. In this section, we extend our discussion of the dynamics of development to an analysis of developmental pathways taken by different groups of individuals from childhood through adulthood. In particular, we examine the development of socioemotional functioning among children who exhibit emotional and behavioral difficulties. A common way to understand emotional and behavioral-regulation difficulties involves the *internalizing–externalizing* dichotomy. According to Merrell and Walker (2004):

> Externalizing problems include behavioral characteristics that are considered to be undercontrolled and other-directed, such as antisocial and aggressive behavior, conduct problems and delinquency, destructive and harmful behavior, and the hyperactive impulsive manifestations of ADHD. Internalizing problems include "overcontrolled" or self directed behavioral and emotional characteristics such as depression, anxiety, social withdrawal, and somatic problems. It is important to recognize that in some cases, a child's behavioral characteristics can be of a 'mixed' variety, manifesting important aspects of the both the internalizing and externalizing broad bands (p. 907).

Despite its prominence in developmental research, the internalizing–externalizing dimension raises several problems. First, the dimension is broad. As a result, there is considerable heterogeneity in the behaviors to which the terms *externalizing* and *internalizing* apply. Second, given its breadth, assessments of internalization and externalization tend to involve aggregations across a broad range of behavior collapsed across context, time, and domain of functioning. Third, the practice of representing externalization and internalization as polar opposites implies that the co-occurrence of behaviors named by these terms should be low. However, research, shows dramatic comorbidity between so-called internalizing and externalizing behavior.

Most important, the internalizing–externalizing dimension seems to have limited descriptive value. While it is clear how everyday concepts like *acting out* or *aggression* can reflect forms of externalizing behavior, it is not clear why anxiety or depression reflect internalization. The externalizing–internalizing contrast has its origins in the distinction between over- and undercontrol (Block & Block, 1980). From this view, problems of aggression, attention regulation and impulsivity reflect an underdeveloped capacity for regulation (undercontrol), whereas anxiety, depression, and related states reflect a tendency toward overcontrol (Block & Block, 1980). While a large literature suggests that many (but not all) externalizing problems are linked to poor behavior regulation (Rubin, Burgess, Dwyer, & Hastings, 2003), relations between overregulation and internalization are less clear. Kagan's (2003, 2008) work on behavioral inhibition is an exception. Kagan and his colleagues have demonstrated a degree of stability in the emotional development of children who exhibit a bias toward *behavioral inhibition*. They have also shown that a tendency toward behavioral inhibition is related to the development of social anxiety, alcoholism, and related conditions (Smoller et al., 2003). The concept of *behavioral inhibition*, however, is different from the concept of *overcontrol*. Behavioral inhibition reflects an emotional disposition to withdraw from novelty, whereas overcontrol implies a more controlled and self-directed process.

The Dynamics of Adaptive and Maladaptive Socioemotional Adjustment

Instead of representing maladaptive emotional processes along a single externalizing-internalizing dimension, Figure 15.15 depicts a multidimensional space-state representation of socioemotional adjustment in motive-inconsistent contexts. The horizontal dimension reflects variation in the *direction* of emotional action from *moving toward* to *moving away* from motive-inconsistent events. The vertical dimension reflects degree of activation, ranging from *activation* to *immobilization*. In any given motive-inconsistent situation, a person confronts a series of *affective choices*—alternative modes of emotional responsivity within or between situations (de Rivera, 1991). Figure 15.15 shows affective channels that avail themselves in motive-inconsistent situations. These include (1) anxiety under uncertainty; (2) moving against others in response to moral violations (e.g., anger, rage); (3) moving away from others under conditions of danger or powerlessness (e.g., fleeing in fear; hiding in shame); and (4) immobilization under conditions of hopelessness or helplessness (e.g., depression; freezing in fear).

Within and between contexts, emotional functioning fluctuates dynamically throughout this state-space. Within this state-space, some affective movements may be more likely than others. For example, theorists have long postulated dynamic relations between *shame* and *anger* (Hejdenberg & Andrews, 2011; Scheff & Retzinger, 1991). Research suggests that angry aggression (moving against) is often motivated by the invocation of shame-related challenges to identity (e.g., insults, criticism; Ferguson, Eyre, & Ashbaker, 2000; Scheff, 2010; Shanahan, Jones, & Thomas-Peter, 2011; Tangney, Wagner, Fletcher, & Gramzow, 1992; Wright, Gudjonsson, & Young, 2008). Conversely, children who exhibit reactive aggression (moving against others) are more likely to experience peer rejection (Morrow, Hubbard, McAuliffe, Rubin, & Dearing 2006) and shame-related feelings that result from rejection (Ji, Wei, Chen, & Zhang, 2012). Movements between anger and shame can thus foment a mutually sustaining anger ←→ shame dynamic.

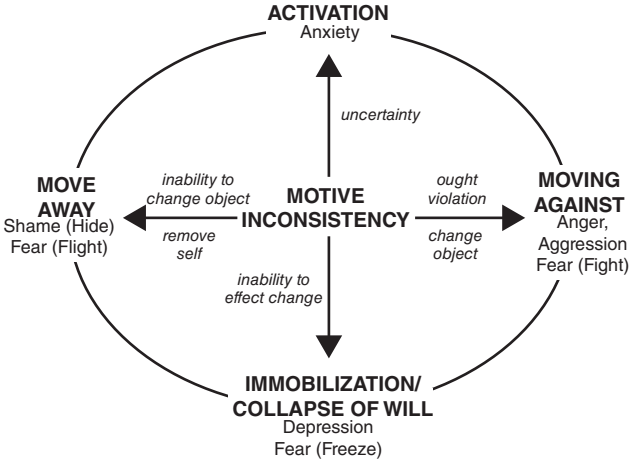


Figure 15.15 The dynamics of socioemotional adjustment for negative emotions.

Several points arise from this example. First, emotional life is rarely organized around single emotional states. As a result, it is not helpful to think of socioemotional development as organized around any single class of emotions or around *any single pole* of a bipolar dimension. Instead, emotional life involves dynamic movements between and among different emotional states throughout the state space. Second, some emotional dynamics may be more probable than others (e.g., shame ←→ anger vs. fear ←→ shame). Third, some emotional dynamics (e.g., shame ←→ anger) may involve movement between opposite poles within the multi-dimensional state space. As a result, thinking of emotional life in terms of dynamic movements within a state space provides a framework for understanding how emotional functions self-organize into heterogeneous, fluctuating, and comorbid patterns over time.

Pathways in the Development of Adaptive and Maladaptive Patterns of Socioemotional Adjustment

Patterns of socioemotional adjustment are epigenetic products of the mutual interplay among (not always obvious) nested processes that orient development in particular directions (Gottlieb, Lickliter, & Wabash, 2005; Mascolo, 2013). Given a set of initial conditions (e.g., a child's genotype and temperament), variations in local conditions can move development along a variety of different pathways. Development, however, is an iterative process; as development occurs, developmental changes at one moment create the conditions that canalize movement in the next moment (Cox, Mills-Koonce, Propper, & Gariepy, 2010; Witherington, 2011; Van Geert, 1994). As conditions of development organize into stable, mutually strengthening patterns, developmental changes can cascade in particular directions. As development advances, deviations from particular pathways are possible, but tend to require more systemic changes in developmental conditions. Such changes can often be initiated by modifying the functioning of key developmental variables—sometimes called control parameters (Fogel & Thelen, 1987)—which then

iterate and ripple over time, creating novel opportunities for development in alternative directions.

Figure 15.16 displays a developmental web describing pathways in the development of adaptive and maladaptive patterns of socioemotional adjustment. The figure identifies patterns of converging, diverging, and overlapping trajectories in patterns of socioemotional adjustment within groups of individuals. The bottom portion identifies the temperamental starting points of development in terms of patterns of affectivity and attentional control (Evans & Rothbart, 2009). Each pathway is defined in terms of a series of movements toward or away from the varied socioemotional outcomes identified at the top of the figure. Within the figure, statements in shaded boxes refer to the influences of socialization agents (caregivers and peers); unshaded statements refer to developmental steps along a socioemotional trajectory.

There are multiple pathways to the development of adaptive, prosocial, behavioral outcomes. Three socially adaptive pathways are identified in Figure 15.16. The empathic–prosocial pathway (1) involves development toward adaptive, empathy-based prosocial action. Prosocial outcomes are most probable among children who (1) exhibit an emotional bias toward positive affect and

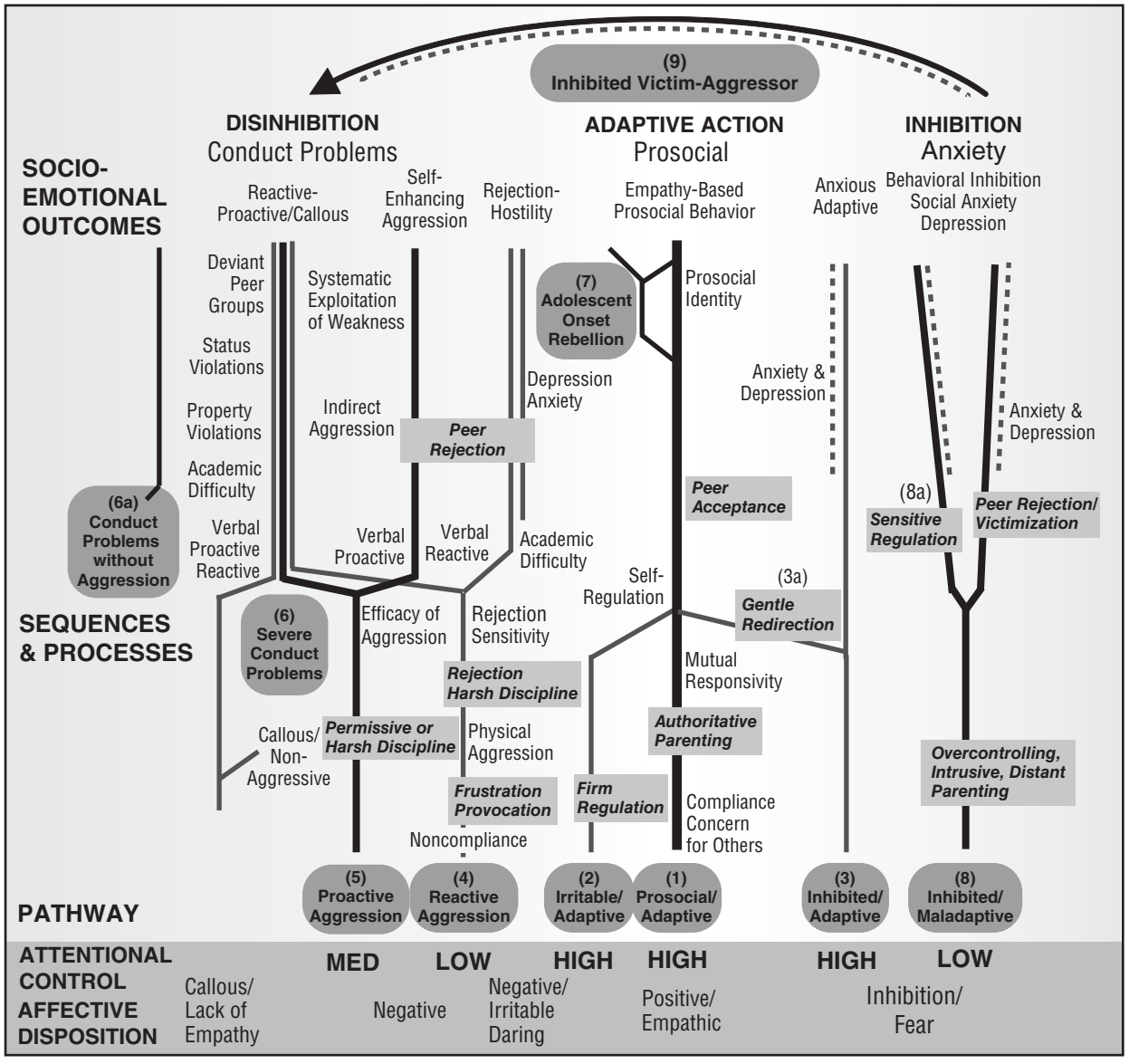


Figure 15.16 Pathways Toward Adaptive and Maladaptive Socioemotional Adjustment. Phrases along individual trajectories indicate order of developing skills or deficits; gray boxes indicate social effects on developmental pathways; dashed lines indicate co-morbidity of anxiety and depression.

concern for others; (2) exert high dispositional levels of effortful control over action and emotion (Eisenberg, Hofer, & Vaughan, 2007); and (3) are cared for by sensitive and authoritative parents (Kochanska & Akzan, 2006; Kochanska & Kim, 2013). Such infants and their caregivers mutually influence each other's actions over time, and quickly self-organize into mutually responsive and emotionally attuned interactions (Kochanska & Aksan, 2004). Children who develop along the adaptive–prosocial path tend to demonstrate affective concern for others from an early age (Roth-Hanania, Davida, & Zahn-Waxler, 2011) and begin to regulate their actions in terms of parental rules during the second year of life (Kochanska & Aksan, 2006). The form of intersubjectivity established within mutually responsive relationships mediates the joint production of positive affective affect (Bornstein, 2013), the development of emotional-regulation skills (Ursache, Blair, Stifter, & Voegtline, 2013), a concern for others (Roth-Hanania, Davida, & Zahn-Waxler, 2011), and the appropriation of sociomoral standards to regulate social conduct (Kochanska & Kim, 2013). The development of prosocial action in children both fosters and is reciprocally facilitated by peer acceptance and the formation of increasingly stable peer relationships (Clark & Ladd, 2000).

Figure 15.16 identifies two additional pathways toward adaptive prosocial behavior. These occur among children who exhibit emotional biases toward negative or irritable (Pathway 2, irritable–adaptive) and inhibited (Pathway 3, inhibited–adaptive) affect. These children are protected from maladaptive development by virtue of their capacity for high levels of behavioral and emotional control, coupled with parenting styles that are sensitive to children's emotional needs. The prosocial development of children with irritable temperaments is facilitated by the use of firm but affectively positive behavioral regulation. In contrast, because less arousal is needed to motivate inhibited children, they benefit from gentle redirection in regulatory contexts (Kochanska & Aksan, 2006).

Pathways in the Development of Aggression and Conduct Problems

Figure 15.16 identifies four pathways in the development of conduct problems. Conduct problems encompass a broad range of rule violating behavior, including noncompliance, aggression, property violations (e.g., stealing, vandalism), status violations (e.g., running away, truancy, substance abuse), and delinquency (e.g., arrest; incarceration; Barry, Golmaryami, Rivera-Hudson, & Frick, 2013; Wakschlag, Tolan, & Leventhal, 2010). Aggression is among the more important and widely studied conduct problems. Figure 15.16 identifies four pathways in the development of problems involving physical aggression. Researchers have differentiated between adolescent-onset and child-onset pathways in the development of aggression (Barry et al., 2013; McMahon & Frick, 2005). Figure 15.16 identifies three pathways in the development of child-onset aggression: reactive aggression (Pathway 4), proactive aggression (Pathway 5), and severe conduct problems and aggression (Pathway 6). Adolescent-onset aggression (Pathway 7) tends to be limited to adolescence and arises as the product of comparatively few risk factors. It tends to arise as an amplification of the normative process of autonomy seeking (Moffitt, 2003).

Reactive aggression refers to defensive or retaliatory attempts to harm others in response to real or perceived provocation; proactive aggression involves deliberate, nonemotional, and unprovoked attempts to harm others for the purpose of instrumental gain (Kempes, Matthys, de Vries, & van Engeland, 2005). Research assessing the reactive-proactive dimension has produced considerable debate. On the one hand, research shows that reactive and proactive aggression have different origins and developmental courses (Hubbard, McAuliffe, Morrow & Romano, 2010). Reactive aggression is associated with irritable/angry infant temperament (Vitaro, Barker, Boivin, Brendgen, & Tremblay, 2006), lower dispositional capacities for effortful and attentional control (Connor, Chartier, Preen, & Kaplan, 2010), and poor impulse regulation (Connor, Chartier, Preen, & Kaplan, 2010). Children and adults who exhibit reactive aggression are more likely to develop a hostile attribution bias—a tendency to interpret ambiguous emotion in others as indicating hostile intent (Bailey & Ostrov, 2008)—and heighted sensitivity to rejection (Jacobs & Harper, 2013). Such biases may arise from a history of reciprocal provocation by parents and peers, maltreatment or other forms of threat (Dodge, Lochman, Harnish, Bates, & Pettit, 1997). These biases also tend to feed forward into later romantic relationships (Canyas, Downey, Berenson, Ayduk, & Kang, 2010) and play a role in dating violence (Brendgen, Vitaro, Tremblay, & Lavoie, 2001).

In contrast, as a form of deliberate provocation, proactive anger is less likely to be accompanied by anger (Hubbard et al., 2002). It tends to be associated with lower basal physiological arousal and may be associated with high levels of dispositional sensation seeking (Wilson & Scarpa, 2011). Children who display proactive anger tend to hold beliefs about the social legitimacy of aggression and its efficacy as a means to instrumental ends (Arsenio, Adams & Gold, 2009). Teens who exhibit high levels of proactive aggression show higher levels of delinquency

(Brendgen, Vitaro, Tremblay, & Lavoie, 2001) and lower levels of psychosocial adjustment than those who exhibit high levels of reactive aggression (Card & Little, 2006).

On the other hand, researchers have noted that reactive and proactive aggression tend to co-occur (Vitaro & Brendgen, 2005) with correlations that range between .40 and .70 (Poulin & Boivin, 2000). In an important study, Barker, Tremblay, Nagin, Vitaro, and Lacourse (2006) identified three trajectories of aggressive behavior between the ages of 13 and 17. The vast majority of teens (.93) were classified as either low aggressors or moderate aggressors. A small minority (.06) exhibited high levels of aggression that peaked at age 15. However, regardless of group membership, children who aggressed exhibited similar levels of *both* proactive and reactive aggression. Given the massive co-occurrence of reactive and proactive aggression, some theorists have suggested abandoning the distinction (Bushman & Anderson, 2001).

Recent research has produced findings that may help account for these seemingly contradictory findings. Researchers have demonstrated that early-onset aggression is often associated with the presence of callous and unemotional affective dispositions in children (Frick & Viding, 2009; McMahon & Frick, 2005). Callous and unemotional behavior includes a lack of empathy and concern for others, an absence of guilt upon wrongdoing, and the intentional manipulation of others for personal gain (Frick & Viding, 2009; Muñoz, Qualter, Padgett, 2011). Children who show early-onset aggression tend to score high on measures of callous and unemotional social engagement (Dandreaux & Frick, 2009). Frick and White (2008) reviewed research showing that children exhibiting callous emotionality are more likely to show fearless and thrill-seeking behaviors, deficits in processing emotional information, less sensitivity to punishment cues, and positive beliefs about the use of aggression. Children who exhibit callous and unemotional dispositions are more likely to exhibit conduct problems, bullying (Fanti & Kimonis, 2012) and delinquent behavior (Edens, Campbell, & Weir, 2007) that carries forward into adulthood (Frick & Viding, 2009).

Aggression and conduct problems are not the results of single causes or risk factors. Risk and protective processes combine in complex ways in the probabalisitc self-organization of developmental trajectories. Fanti, Frick, and Georgio (2009) found that adolescents showing higher levels of callous and unemotional dispositions were more likely to exhibit combined proactive and reactive aggression and not pure forms of either proactive or reactive aggression (see also Thorton, Frick, Crapazono, & Terranova, 2013). Similarly, in a large sample of Greek-Cypriot adolescents, Fanti (2013) found that teens who exhibited both high levels of conduct problems and high levels of callousness exhibited higher levels of bullying, substance abuse; inattentiveness, impulsivity, and narcissism, as well as less robust family support than teens rated as high in either conduct problems or callousness.

Together, these findings support the proposition for the three child-onset pathways identified in Figure 15.16. While research suggests two distinct but overlapping pathways involving reactive (4) and proactive (5) aggression, children who exhibit these forms of aggression in the absence of callous and unemotional dispositions are less likely to be involved in severe conduct problems. Thus, severe antisocial outcomes (6) involve the co-occurrence of callous and unemotional affective disposition with a combination of both proactive and reactive aggression. As indicated in Figure 15.16, in addition primary pathways toward conduct problems, research suggests an additional path toward conduct problems without aggression (Burt, 2009; Eley, Lichtenstein, & Moffitt, 2003).

Pathways in the Development of Inhibition and Social Anxiety

Inhibited patterns of socioemotional adjustment are those that are organized around avoidance as a style of affective coping (Rapee, Schniering, & Hudson, 2009). Such individuals tend to approach events with a disposition toward fearful or anxious affect, show avoidance toward unfamiliar people and events, and exhibit heightened vigilance in novel contexts (Lahat, Hong, & Fox, 2011). Research suggests that children exhibiting fearful/inhibited biases early in life are more likely to develop problems related to anxiety in adolescence and adulthood (Lahat, Hong, & Fox, 2011; Muris, Brakel, Arntz, & Schouten, 2011; Schwartz, Snidman, & Kagan, 1999; Sportel, Nauta, Hullu, Jong, & Hartman, 2011). For example, Prior, Smart, Sanson, and Oberklaid (2000) found that 42% of children who were rated as shy across multiple observations in childhood exhibited anxiety problems in adolescence. In contrast, only 11% of the children not previously classified as shy exhibited anxiety problems in their large sample. Thus, while not all children classified as inhibited develop anxiety problems later in development, a sizable proportion do.

Figure 15.16 identifies four pathways in the development of inhibited patterns of socioemotional adjustment. Severe social anxiety problems develop through the inhibited/maladaptive pathway (8). Children who pass through

this pathway tend to experience fearful/inhibited temperament and a diminished capacity for attentional control (Sportel et al., 2011) coupled with parenting styles that can be classified as overprotective, overcontrolling, or intrusive or distant (Lahat et al., 2011; Lindhout, Markus, Hoogendijk, & Boer, 2009). Such parenting styles are unlikely to operate as static properties of individual parents. Instead, it is most likely such parenting styles are emergent, coregulated products of a parent's attempt to respond to an inhibited child (Evans & Porter, 2009, 2012). While over-protective parenting fails to promote strategies regulating emotional arousal in the context of novel events, over-controlling or intrusive parenting is likely to amplify inhibited emotion and restrict a child's capacity to master novel situations. Emotionally distant parents may foster the development of anxious attachment relationships, which increase the probability of anxiety problems in children (Warren, Huston, Egeland, & Sroufe, 1997).

Anxiety problems in children who display inhibited temperaments and diminished capacities for attentional control can be mitigated by parents who introduce children to novel events with persistence and sensitivity (Pathway 8a). Conversely, although the capacity for high levels of attention control acts as a protective factor in the development of anxiety problems in children with inhibited temperament (Lahat et al., 2011; Sportel et al., 2011) insensitive or intrusive parenting can channel development away from a prosocial path and in the direction of social anxiety (Pathway 3a). While inhibited temperament biases socioemotional development toward the direction of social anxiety, it is important not to view inhibited temperament simply as a risk factor for maladaptive behavior. Shyness and behavioral inhibition is often viewed negatively, at least among Westerners (Aho, 2011). The work of Kochanksa, Aksan, and colleagues suggest that behavioral inhibition may serve important developmental functions. For example, children with inhibited temperament do not require strong arousal induction in moral socialization (Kochanska & Aksan, 2006). In an insightful study, Aksan and Kochanska (2004) showed that children who exhibited inhibited temperament and inhibition to novelty in infancy (9, 14, and 22 months) showed an increased capacity for voluntary inhibitive control at 45 months). Aksan and Kochanska (2004) suggested that the involuntary reactive inhibition (inhibited temperament) functions to decreases the speed of approach to novel situations early in life. This decrease, in turn, provides the opportunity for additional attentional focus on novel events, thus facilitating the development of voluntary effortful control. These findings illustrate the ways nonobvious relations between developing systems promote psychological development. In the case of Aksan and Kochanska's (2004) findings, involuntary relations between inhibition and novelty earlier in development operate iteratively to set up conditions for the development of the capacity for voluntary control. The resulting developing of voluntary control then buffers against developmental movement in the direction of social anxiety.

As is the case with the development of aggression, the development of social anxiety is also accompanied by experiences of depression (Epkins & Heckler, 2011; Sportel et al., 2013). Peer rejection, victimization, social isolation, and loneliness play important and cumulative roles in the development of depression and anxiety in children, adolescents, and adults (Dempsey & Storch, 2008; Epkins & Heckler, 2011). Briggs, Nelson, and Sampino (2010) reported evidence that depression among adolescents who experience social anxiety was mediated over time by experiences of peer rejection and victimization. Silk, Davis, McMakin, Daahl, and Forbes (2012) reviewed evidence suggesting that the codevelopment of anxiety and depression in adolescence is mediated by a heightened sensitivity to self-evaluative social threat (fear of negative evaluations of the self by others) coupled with modifications in the proclivity to seek out wanted outcomes. Research indicates, for example, that anxious adolescents show a heightened attentional bias to social threat, and are more likely to interpret socially ambiguous situations in terms of threats to one's self-evaluation (Silk et al., 2012). These same processes are also implicated in the development of depression. Silk et al. (2012) suggest that in an attempt to adapt to social threat, adolescents revise their expectations for social acceptance, ultimately resulting in increasing depression. These findings suggest that experiences of rejection, exclusion, loneliness, and related social threats to self-evaluation play cumulative roles as risk factors in the codevelopment of anxiety and depression (Epkins & Heckler, 2011). The codevelopment of anxiety and depression as a product of peer exclusion and rejection is represented in terms of the double lines associated with the inhibited pathways depicted in Figure 15.16. For some children, the anxiety-rejection link not only results in depression, but also evokes aggressive reactive and proactive attempts to lash out against rejection. The *inhibited-victor aggressor* pathway (Pathway 9) occurs in children with inhibited temperaments who are recipients of harsh discipline or consistent patterns of humiliation by peers. Research shows that in some circumstances (e.g., when children are humiliated or raised in violent homes) temperamentally

inhibited children become highly aggressive in later childhood and adulthood (Watson, Fischer, Andreas, & Smith, 2004).

Trajectories of Development: Making a Path by Walking

Developmental pathways are the outcomes of constructive processes. Pathways should not be viewed as inherent, preformed, or predestined roads that anyone with a particular precondition must take. Unlike highways that have been planned and pre-structured by a central authority, developmental paths are more like trails that that are formed through the active process of walking (Thompson, 2007). A developmental pathway emerges slowly as if one were walking through a thicket. As each step is taken, part of the path is formed. Each step provides the conditions that orient the next step. Each movement is constrained and channelized by the obstacles and clearings that one encounters along the way. Obstacles, however, can turn into clearings with the thrash of a sickle, while clearings can invite movement away from desired destinations. Thus, pathway representations of development are like prototypical images that researchers have imposed upon an enormous variety of individual trajectories. In this way, the notion of pathway provides a particular way of summarizing large numbers of individual, self-organized trajectories that show sufficient similarity to be treated as members of common idealized class.

BEYOND BEST PRACTICES AND EVIDENCE-BASED INTERVENTION: TRANSLATING DYNAMIC SYSTEMS INTO PREVENTION AND PRACTICE

How can dynamic systems approach inform the goals of preventing and redirecting maladaptive behavior? Perhaps the most important—and most difficult—task is a conceptual one—namely the task of helping practitioners and policy makers think systemically about the origins and treatment of maladaptive behavior. Many current approaches to intervention and prevention continue to adopt a static, discrete state approach to the problem of maladaptive behavior. From this view, maladaptive behavior is understood as a reflection of more or less fixed pathologies located within individual persons; interventions (e.g., medication, therapy, skill development) are focused on effecting change within the individual child. With exceptions (e.g., family systems therapy; coordinated intervention teams), interventions tend to be focused on the individual child viewed as separate from the contexts in which she acts. However, treatments (e.g., drugs, psychotherapy) are often administered in an unsystematic (or loosely connected) fashion, with different treatments administered and assessed independent of one another. Still further, therapeutic interventions—psychotherapy, social skills training and so forth—tend to occur in contexts that are isolated from everyday life (e.g., the four walls of a therapist's office). If one thinks of psychological maladaption as a kind of pathology that resides within individuals, it makes sense to focus interventions on processes that occur *within* persons. However, if one understands patterns of maladaptive behavior as *emergent* products of individuals in social contexts, this argument begins to lose its force.

In the past decades, the quest for evidence-based interventions and prevention programs based on best practices has occupied the attention of policy makers, practitioners and clinically oriented researchers. Evidence-based interventions are those that can cite scientific support for their effectiveness (Gullotta & Blau, 2008). The call for evidence-based therapies had its origins in dissatisfaction with therapeutic approaches that had long been untested, that had been based primarily on anecdotal testing, or that were judged impervious to testing. Interest in evidence-based therapies is not simply a product of scientific motives; insurance companies make evidence of the effectiveness of an intervention a requisite for the compensation of practitioners; federal agencies make funding of mental health research and programs contingent upon empirical demonstrations of therapeutic effectiveness. Evidence-based research has demonstrated the efficacy of different modes of therapy for various broad classes of maladaptive behavior. The requirement that practitioners garner empirical evidence supporting the effectiveness of psychological interventions has positive value. However, calls for evidence-based prevention and treatment tend to be based on the assumption that certain intervention and prevention strategies for particular classes of problems have universal application across contexts. That is, they tend to assume that (1) patterns of maladaptive behavior reflect more or less fixed types of problems (i.e., pathologies) that (2) lend themselves to standardized, generalized and even manualized modes of treatment. They also assume (3) that change is the result of a causal force—a property of the treatment as such, exerting an influence on every individual client similar to that causal force—plus random individual influences—that may increase or decrease the observed effect. This view reflects a continued reliance on models of science developed in the early twentieth century that seek to identify universal laws and relationships that transcend the context and content of

target phenomena. Prevention and intervention programs that fail to acknowledge the dynamic, heterogeneous and nonlinear origins of maladaptive behavior are unlikely to have high levels of effectiveness. Further, they may mistakenly identify group-based average effects with real effects on individuals, without taking into account that some interventions might also have a causal negative effect on some clients who are compensated in the group averages by positive effects in other clients.

Dynamic systems approaches seek to move beyond the limits imposed by linear ways of thinking about prevention, intervention, and assessment. Dynamic systems approaches maintain that effective prevention and intervention must be organized around a foundational appreciation of the nature of complexity (Gorman et al., 2006; Schensul, 2009). One cannot seek to understand the contextualized dynamics of maladaptive activity using principles and methodologies designed to assess relations among decontextualized variables. Systems thinking requires nothing less than a shift in mind-set:

- The unit of prevention and intervention should not be the individual child separate from its social context; instead, it should focus on the multiply nested child–environment system as a whole (Antonishak & Reppucci, 2008). Maladaptive patterns of action are not fixed forms or linear expressions of inner pathologies; instead, they are emergent products of individuals (with their dispositions and histories) operating within a complex person–environment system.
- A complex system is not defined as one that is composed of a simple set of multiple processes acting in isolation; instead, a complex system is one in which component processes mutually influence each other over time. If this is so, then prevention and intervention programs organized around discrete sets of uncoordinated interventions that are insensitive to local context are unlikely to be effective. Instead, prevention and intervention should focus on seeking transformations in the system as a whole. This can be accomplished by exerting control over central variables (sometimes called *control variables*) that, through their coactions with other variables in the system, modify the functioning of the whole.
- Complex systems are not steady states. They are sensitive to the initial conditions of their component processes (Kelso, 1995; Thelen & Smith, 2006). This means that minor variations in the initial conditions of two different complex systems can, over time, produce dramatic variations in the operations of those systems. If follows that efforts at prevention and intervention that are based on generalized or standardized procedures that are insensitive to the dynamics of local systems are unlikely to be effective. Instead, interventions must be organized around particular dynamics of local child–environment systems. Further, because small changes in local variables within a system can modify system functioning, changes introduced by prevention and intervention programs are often unforeseen and unpredictable. As a result, prevention and intervention programs must be sensitive to concrete changes in target systems as they evolve dynamically over time.

Instead of seeking to produce standardized remedies intended to have broad application across diverse contexts, dynamic systems approaches maintain that the tasks of prevention and intervention become ones of seeking to exert control over the dynamics of local complex systems. In the language of dynamic systems theory, the goal of prevention and intervention would be to produce phase shifts in functioning of the system—namely, a series of cascading or rippling effects that transform relations among variables throughout the child–environment system.

To illustrate, consider the simple child–environment system shown in Figure 15.17. This system describes the structure of a particular social interaction (Tseng & Seidman, 2007) between a child and a staff member in a group home for children who exhibit emotional and behavioral problems. At this school, staff members are trained in the use of time-out techniques for managing rule violations among children (8–11-year-olds) in the unit. A product of behaviorist thinking, the time-out technique was initially proposed as a way to provide a child with time out from the reinforcing contingencies assumed to be operative during the production of rule breaking behavior (Bostow & Bailey, 1969). Many practitioners, educators, and parents, however, misuse the time-out technique as a form of punishment (Kemp, 1996). Instead of operating as a break from reinforcing contingencies, the technique is used as an unpleasant consequence for a child's misdeed. As a result, time-out procedure often has effects

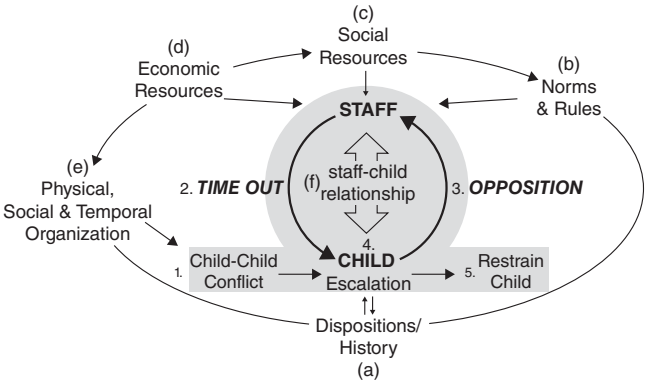


Figure 15.17 Dynamics of staff–child interaction in a group home.

that are opposite to their intended ones. For example, in an interview study with children enrolled in a special education program, Visser, Singer, van Geert, and Kunnen (2009) found that some children experience a time-out as a moment in which they can better reflect on and prepare a negative reaction to the behavior of the other child. In the interaction depicted in Figure 15.17, two boys were engaged in a conflict while watching television together (Point 1). As a result, (2) a staff member ordered one of the boys to a time-out. The boy responded by (3) mouthing off to the staff member, who then (4) increased the amount of time that the boy was asked to spend in time-out. Over time, the boy's oppositional behavior escalated out of control, and (5) the boy was physically restrained.

In this example, the child's unwanted behavior emerged as a product of coactive processes that operated within a complex child–environment system. The components of this simple system affect and constrain each other over time. In Figure 15.17, the shaded area indicates coregulated activity between a staff member and a child over time. The nonshaded elements indicate processes and resources that constrain child–staff interaction. The most obvious example of the mutual interplay among system components involves the positive feedback cycle that arose between the staff member and the child over the course of the encounter. In this interaction, the child's unwanted behavioral escalation was the emergent product of coregulated processes that occurred between the child and the staff member. While this was the most proximal source of the child's behavioral escalation, other elements of the system played an important if less obvious role. The (1) rules and norms of the group home stipulated that staff were to use the time-out technique to regulate misbehavior. Interactions between students and staff were constrained by the (2) social and (3) economic resources available to the group home—namely limits on the availability of funds and the capacity to recruit and retain qualified staff members. Further, the (4) physical (i.e., close quarters), social (i.e., the particular children involved in the conflict; the relationship between the target child and the staff member) and temporal (i.e., the placement of quiet TV time after homework and before bedtime) place constraints on the ways children can respond to each other and to the staff member.

However, changes in one or more core components of this child–environment system would likely produce dramatic changes in the capacity of staff to regulate the behavior of the children who lived there. The most obvious source of transformation would involve implementing appropriate uses of the time-out procedure. This would involve training staff to use time-outs as an opportunity to break behavior sustaining contingencies and to provide an opportunity for reflection and problem-solving, and also to talk with the children about how they experience the meaning of time-out, what it does to them emotionally, physically and rationally (Visser, Singer, van Geert, & Kunnen 2009). Success in the use of the time-out procedure—especially if coupled with sensitive emotional regulation and scaffolded behavioral redirection—would likely create a cascade of reciprocally influencing transformations. These could include the emergence of a shared ethos of improved intervention strategies among staff; increased capacity for behavioral and emotional regulation among children; increased cooperativeness between and among staff and children; and improved staff–client relationships. Over time, such subtle changes would settle into a stable and organized system of functioning. Participation in the system would have the effect of organizing children's capacity for effective social engagement and emotional regulation within the context of the unit.

It is also possible that similar results could be achieved by seeking to influence less central components of the child–environment system. For example, it is possible that misuse of time-outs in this particular organization was a product of difficulty in recruiting and retaining staff with suitable levels of experience and education. It is possible that the misuse of the time-out procedure arises from difficulties in training less qualified staff, or in retaining qualified or trained staff. If this were the case, it might be possible to transform the child–environment system by enhancing economic resources and using those resources to improve the quality of the social resources available to the organization, perhaps by recruiting and retaining more highly qualified staff who would require less intensive training in basic procedures. It is also possible that the aggressive behavior within a group of children prevails as a product of reciprocal imitation or retaliation that originates its in the aggressive activity of a minority of students. Simply changing the context often improves the behavior quite substantially (Visser, Kunnen, & Van Geert 2010).

We propose a series of general principles that can guide efforts at prevention and intervention at multiple levels of functioning. Efforts at prevention and intervention can be organized around several loosely organized steps:

1. Understand the dynamics of the local nonlinear systems in question. Efforts at prevention and intervention must begin with contextualized understandings of the nature of the local systems of psychological adaptation or maladaptation under consideration (Gorman et al., 2006; Schensul, 2009). This may require access to local data related to the functioning of particular child–environment systems of interest. Efforts at prevention and intervention can be focused on systems that operate at multiple levels. These may include the level of the individual child at home or at school; of multiple children interacting with each other and

with adults within organizations; or groups of children functioning within larger communities (Tricket & Schensul, 2009; Visser, Kunnen, & Van Geert 2010; Yoshikawa & Hsueh, 2001). Success in assessing the nature of nonlinear systems is facilitated by cooperation among interdisciplinary teams of practitioners who can coordinate their expertise relative to understanding a problem (Aiken & Hanges, 2012). For example, to assess a problem of aggression within a school system, it is important to bring together teams that can assess a broad range of variables related to the problem of aggression. These may include identifying (1) forms of observed aggression and the contexts in which they occur; (2) students at risk of being perpetrators or targets of aggression; (3) student culture and student-student relationships; (4) teacher–student interaction that arise during aggressive encounters; (5) demographics and values and of the families; (6) school culture; and (7) school–home–community relations. Varieties of methods are available to collect and analyze data for conducting such assessments, from dynamic mathematical modeling to ethnographic observation (Aiken & Hanges, 2012; Schiepek, 2003; Steenbeek & van Geert, 2013; Tricket & Schensul, 2009).

2. Identify core control variables. Having gained an understanding of the dynamics of the local system of interest, identify major control variables whose manipulation can have effects on the operation of other components in the system. For example, studies suggest that parental involvement and moral climate and children's personal interpretive socioemotional biases are important variables in mediating the level of aggression observed in middle schools (Farrell, Henry, Mays, & Schoeny, 2011; Foà, Brugman, & Mancini, 2012). Such variables may function as important control variables that, when modified, can produce cascading changes in a multiple variables related to the production of aggression within a given school system.
3. Coordinate attempts to regulate core control variables in relation to each other. It is not enough simply to seek to modify parameters of a dynamic system in isolation of one another. Instead, research suggests that prevention and intervention programs that seek to coordinate interventions and resources in relation to each other are more able to produce systemic change than those that do not (Eron et al., 2002; Farmer, Farmer, Estell, & Hutchins, 2007). The process of coordinating possible control variables during prevention and intervention increases the likelihood of fostering synergy among the coacting variables that compose a dynamic system.
4. Monitor and respond to changes in the system continuously over time. Traditional approaches to intervention tend to assess target functioning of individuals both prior to and subsequent to an intervention. Pre- and posttest designs yield insufficient data to allow assessment of the change dynamics in a complex system. To gain sufficient information to provide a clear understanding of the developmental course of intervention, it is necessary to assess the status of an evolving system at multiple points over the course of an intervention. A mindset of continuous assessment provides an important corrective to static pre- and posttest designs. Without multiple assessments, it is not possible to identify the types of nonlinear changes that result from coactions among system variables. Further, nonlinear systems often exhibit instabilities and fluctuations that result from minor changes in component processes. As a result, modifications in any given control variable can produce both wanted and unwanted effects (Gorman et al., 2006). Continuous monitoring of changes in system dynamics is necessary to know when and how to regulate the fluctuation of system parameters to produce optimal effects. Still further, understanding how manipulation of control parameters produces patterns of wanted and unwanted change over time is important for the development of prevention programs. The capacity to identify core control parameters provides practitioners with the ability to know where and how to allocate limited resources to produce optimal gain.

CONCLUSIONS: THE SELF-ORGANIZATION OF ORDER, VARIABILITY, AND DISORDER IN STRUCTURES OF THINKING, FEELING, AND ACTING

The way we conceptualize a problem affects how we study it. In the case of deviant behavior, our conceptualizations affect how we approach people who exhibit patterns of action that we classify as maladaptive. The study of deviant behavior raises difficult issues that have long captured the attention of psychologists, sociologists, philosophers, physicians, and practitioners. Although the medical model of psychopathology remains dominant, recently, it has weathered its share of controversy (Frances, 2013). From our perspective, the medical model influences theory and research in developmental psychopathology by representing psychological dysfunction as fixed structures that reflect an underlying pathology. In what follows, we discuss how dynamic systems thinking can address limitations of the psychopathology of fixed forms.

From Fixed Forms to Emergent Structures

When we think of psychological problems in terms of the concept of disorder, we run the risk of treating behavior and psychological problems as if they were entities or

things. The psychopathology of fixed forms leads theorists and researchers to think of psychological problems as trait-like entities that exist within individuals. When this occurs, researchers seek to investigate the nature of the presumed trait or form: What is the nature of the trait? Where is it located? What causes it to come into being? However, from a dynamic systems perspective, behavior is not a thing that a person has; behavior is a process that occurs over time within particular social contexts. It is necessary, therefore, to study how behavior emerges over time in particular contexts and social interactions. An inhibited child is not shy all the time; a child who withdraws at school is often noisy and rambunctious at home (Kagan & Snidman, 1990).

Further, when we treat psychological problems as trait-like entities, we tend to direct our attention to behavior that fits our a priori conception of the nature of the entity, and ignore behavior that does not comport with those preconceptions. For example, by focusing on the so-called trait rather than on the dynamic production of behavior in context, we fail to observe how patterns of both maladaptive and adaptive behavior arise as individuals adjust to the demands of local contexts. By studying individuals in contexts, systems approaches seek to understand the origins of order and variability, adaptive and maladaptive action, consistency and inconsistency. Similarly, if we think of patterns of maladaptive activity as discrete and trait-like forms, we are likely to be surprised when we find evidence that presumably fixed categories of disorder in fact fade into one another and assume a variety of diverse and overlapping forms. In contrast, if we seek to understand how patterns of psychological adaptation and maladaptation develop over time in individual children and contexts, it would not be surprising to find adaptive and maladaptive variation in behavior as individuals with different psychological resources and histories adjust to the contextual demands.

Conceptualizing Dysfunction: From Pathology to Adaptive Conflict

In medicine, pathology refers to a discernible deviation in the structure and functioning of the body. Drawing on this concept, the term psychopathology is typically employed in two ways. The first uses the concept of pathology as a metaphor for deviant behavior. From this view, psychopathology is to healthy behavior as a disease is to a healthy organ. Problems arise when one treats this metaphorical construction as a literal statement. Unlike the body, psychological functioning (or mind) is not an entity or thing. As a result, it is not the kind of thing that can be diseased. Treating psychopathology as a disease obscures the social, scientific, and pragmatic processes through which psychological processes are identified as deviant or maladaptive. The standards we use to identify psychological dysfunction are different from those used to determine physical pathology (Banner, 2013). Judgments that any given set of psychological processes is dysfunctional or disordered can be made only against the backdrop of some framework for identifying what is functional or ordered. Such judgments are ultimately normative and evaluative in nature. While many seek to define psychological dysfunction in neutral or nonevaluative terms, this is not possible. Judgments of human action necessarily operate against the backdrop of inescapable evaluative frameworks (Boysen, 2007; Taylor, 1989).

The second use of the term psychopathology is arguably less metaphorical, but nonetheless troublesome. From this view, psychological problems are regarded as symptoms of an underlying pathology located outside of the sphere of behavior itself (e.g., in the brain or body). This view is less metaphorical in the sense that it holds that psychological problems are actually products of bodily (brain) pathology. Several problems arise from this view (Banner, 2013). First, this approach suggests that there should be some correspondence between an underlying biological pathology and psychological symptoms. However, as indicated throughout this chapter, research shows patterns of psychological dysfunction are highly variegated and multiply determined. Patterns of psychological dysfunction emerge over time and function as dynamic systems. However, the proposition that psychological problems are caused by brain disorders begs the question of how to discriminate psychological dysfunction from psychological health. Whatever the role of biological disorder in psychological dysfunction, judgments of psychological dysfunction can only made by invoking standards for defining psychological functioning as optimal or nonoptimal. The identification of biological dysfunction does not in and of imply that any concomitant psychological processes are dysfunctional.

From a dynamic systems perspective, persons—like all organisms—are complex adaptive systems (Lickliter & Honeycutt, 2013; Masten, 2007; van Geert, 1994). A person's psychological processes operate as modes of adapting to their environs. Adaptation reflects the capacity of an organism to adjust its behavior to environmental demands. From this view, it might be better to think of modes of psychological dysfunction as systemic difficulties in adaptation, rather than as distinct forms of pathology, disorder, disease, or illness. The concepts of adaptation and maladaptation are relational concepts; they make reference to the goodness of fit between the resources of the organism and the demands of its environment (Gottlieb & Halpern, 2002; Lerner, Hess, & Nitz, 1990). Thus, maladaptive behavior is behavior that causes problems for an

individual, dyad, or community. The concept of dysfunction as adaptive conflict reflects a more person-centered rather than pathology-centered understanding psychological dysfunction (von Eye, 2009). Instead of seeking to understand the essence of a given category of psychological disorder, emphasis is placed upon the processes by which individual develop adaptive and maladaptive ways of relating to their worlds over time.

From Aggregates to Individual Trajectories

From a dynamic systems perspective, human behavior self-organizes through the dynamic coupling of person-in-context. Thus to understand the process of psychological development, it becomes necessary to examine how novel psychological structures arise through the moment-by-moment coactions among aspects of the person–environment system. This requires moving beyond methods based primarily on aggregate judgments collapsed across time and context and toward direct observation of integrative patterns of behavior during the process of their developmental formation. Traditionally, much research in developmental psychopathology has been variable centered rather than person centered (von Eye & Bergman, 2003). Research directed toward identifying relations among abstract variables using aggregated data tends to be nonergodic and thus fails to capture the nature of processes that occur at the individual level of functioning (Molenaar & Ram, 2010). Further, much research is conducted using participant judgments on rating scales and checklists without actually observing the activity of individual persons. Such methods fail to examine the data that is most essential to understand the process by which order and variability develop over time: the dynamic coupling of individual and social context over time. The use of direct observation of individual-context interaction allows researchers to track how complex patterns of adaptive and maladaptive functioning codevelop in single individuals (Granic & Hollenstein, 2006; Kagan, Snidman, McManis, Woodward, & Hardway, 2002).

The Challenges Ahead

Over the past 20 years, dynamic systems approaches to maladaptive development have themselves undergone considerable development. Nonetheless, dynamic systems models of development are only beginning to show their promise. There remain important challenges to be overcome.

The Importance of Psychological Theory in Dynamic Systems Analyses of Development

Dynamic systems theory has its origins in the fields of physics, mathematics, biology, and chemistry. Since then, it has proliferated in virtually all areas of scientific inquiry. The principles of dynamic systems theory are broadly applicable to the study of any system that demonstrates complexity. Over the past 20 years, there has been an astonishing proliferation of work in many areas of psychology and developmental science. While the principles of *dynamic systems theory* are broadly applicable, it is perhaps misleading to speak of dynamic systems thinking as a theory. Dynamic systems theory operates more as a meta-theory that provides principles and methods for understanding and modeling dynamic phenomena (Witherington, 2007). If this is so, meaningful dynamic systems modeling—mathematical or otherwise—requires a solid foundation of psychological theorizing (Cowan, 2003; Spencer, Austin, & Schutte, 2012; Van Geert, 1994). This is not to say that dynamic systems approaches should necessarily work from a hypothetico-deductive framework. Dynamic systems models of development are still in their infancy. Much work remains in developing dynamic system methods that are capable of representing the overwhelming complexity of human psychological functioning as it unfolds over time. As this work ensues, researchers are producing extraordinarily rich analyses of the dynamic coupling of local psychological processes as they unfold and develop in a wide variety of psychological domains (e.g., Ferrer & Helm, 2013; McAssey, Helm, Hsieh, Sbarra, & Ferrer, 2013). Such studies, even when they do not proceed from a specific theoretical model, are providing an extremely rich matrix of findings that can inform the development of psychological theory. Thus, while dynamic systems theory provides meta-theoretical and meta-methodological tools for developmental science, to exploit the promise of dynamic systems thinking, it is important to wed systems meta-theory more closely with genuinely psychological theorizing.

Elaborating Quantitative and Qualitative Methods for Assessing Complexity in Normative and Nonnormative Development

The task of studying dynamic complexity in development raises obvious methodological questions. Traditional methods that focus on parsing variance at the level of populations typically fail to shed light on the dynamic processes that operate at the level of the development individual action (Molenaar & Campbell, 2009; Ong & Zautra, 2009). Dynamic systems researchers have developed a multiplicity of methods that track dynamic change in ways that are sensitive to the individual development. These methods include mathematical modeling (Ferrer & Steele, 1982; Kunnen, 2012); state space grids (Hollenstein, 2013); microdevelopment of individual and dyadic activity (Mascolo & Fischer, 2015; Thomas, Hopwood, Woody, Ethier, & Sadler, 2013); analyses of alternative developmental pathways (Mascolo & Fischer, 2015); self-report

and diary methods (Navarro, Arrieta, & Ballén, 2007); thick description (Fogel, Garvey, Hsu, & West-Stroming, 2006); and qualitative analyses (Haggis, 2008; Sommerfeld & Hollenstein, 2011). Traditional methods—including experimental design (Gottlieb, 2001) and factor analysis (Ram, Brose, & Molenaar, 2013)—are also applicable to the dynamic analysis of complex systems. These approaches are sensitive to issues relating to developmental timing (Granic, 2005); synchronicity of unfolding psychological processes (McAssey, Helm, Hsieh, Sbarra, & Ferrer, 2013); multiple levels of analysis (Van Geert & Lichtwarck-Aschoff, 2005); sequence (Molenaar & Goode, 2013); and structural transformation (Mascolo & Fisher, 2015).

While there has been enormous progress made in the elaboration of dynamic systems methodology, a series of challenges remain. First, most of this work has been performed with normative populations. There is a need to apply and develop these methods further for the study of the dynamic development of maladaptive behavior patterns. Second, despite the impressive progress in the development of dynamic systems methods, the issue of representing developmental complexity remains. For example, in pursuing ergodic methods that are sensitive to individual development, researchers are increasingly performing longitudinal analyses of multiple single individuals. This raises the question of the extent to which it is desirable and possible to use idiographic methods to make nomothetic generalizations (Fisher, Newman, & Molenaar, 2011). Further, given the enormous complexity of normative and nonnormative development, the question of how to differentiate core control variables from other variables relevant to the development of any given system or systems of activity remains a central challenge. Third, as research comes to address increasingly complex units of behavior, there will be a need to find ways to assess developmental complexity at multiple levels of analysis in ways that are accessible, meaningful and intelligible but not overly simplistic. Toward this end, there is a need to explore ways to coordinate quantitative and qualitative research methods. Similarly, it is essential to keep in mind that the study of developmental complexity does not always require complex designs. We should never lose sight of the principle that *methods* are driven by problems (Bergman & Vargha, 2013). Simple (but not simplistic) methods can often reveal vast complexity. Examples of these include but are not limited to the use of experimental designs to study the effect of variations in rearing conditions on behavioral development (Gottlieb, 1991, 2002; Jablonka & Lamb, 2006); thick description of historical-relational trajectories of development (Fogel, Garvey, Hsu, & West-Stroming, 2006; Piaget, 1952); and the microdevelopmental tracking of developmental complexity in real time (Mascolo & Fischer, 2015).

Accounting for Structural Change in Dynamic Assessments of Development

While dynamic systems approaches have produced powerful methods for the quantitative analysis of nonlinear trajectories of development, it is equally important to understand the structural aspects of developmental change. Many attempts to identify trajectories of maladaptive development focus on quantitative changes in the frequency of or rate of maladaptive activity over time. While useful, such analyses fail to account for the patterns of differentiation and integration that define a sequence as a genuinely developmental one. The concept of development is not simply a synonym for change over time or age-related change. Development is a directional and transformational concept (Bibace & Kharlamov, 2013; Raeff, 2011; Van Geert, 1986). To say that a psychological structure is developing implies that it is undergoing successive structural changes defined with reference to some actual, hypothetical, or idealized developmental outcome (see Basseches & Mascolo, 2010; Sroufe, 2007; Van Geert, 1986, 1987). For example, the structure of indirect aggression in adolescence is different from the reactive aggression of a preschooler (Arnocky, Sunderani, Miller, & Vaillancourt, 2012; Card & Little, 2006); the dynamics of shame, worthlessness and rage experienced among adult victims of early sexual abuse differ from the raw unarticulated experiences of victimization in childhood (Isely, Isely, Freiburger, & McMackin, 2008; Rahm, Renck, & Ringsberg, 2013). To measure structural change, there is a need to invoke some type of developmental yardstick to measure the transformations in the structure of normative and nonnormative patterns of thinking, feeling and acting over time. Dynamic skill theory (Fischer, 1980; Mascolo & Fischer, 2015) provides one such yardstick. Fischer's analysis of developmental transformations in dissociative splitting (described earlier in this chapter) provides but one example of how researchers can account for structural transformations in nonnormative patterns of thinking, feeling, and acting over the course of development.

REFERENCES

Aber, J. L., Allen, J. P., Carlson, V., & Cicchetti, D. (1989). The effects of maltreatment on development during early childhood: Recent studies and their theoretical, clinical, and policy implications. In D. Cicchetti & V. Carlson (Ed.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 579–619). Cambridge, UK: Cambridge University Press. doi: 10.1017/CBO9780511665707.019

Aho, K. (2010). The psychopathology of American shyness: A hermeneutic reading. *Journal for the Theory of Social Behaviour*, *40*, 190–2.

Aiken, J. R., & Hanges, P. J. (2012). Research methodology for studying dynamic multiteam systems: Application of complexity science. In S. J. Zaccaro, M. A. Marks, & L. A. DeChurch (Eds.), *Multiteam systems: An organization form for dynamic and complex environments* (pp. 431–458). New York, NY: Routledge/Taylor & Francis Group.

Ainsworth, M. D. S., Blehar, M., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale, NJ: Erlbaum.

Aksan, N., & Kochanska, G. (2004). Links between systems of inhibition from infancy to preschool years. *Child Development*, *75*, 1477–1490. doi: 10.1111/j.1467-8624.2004.00752.x

Antonishak, J., & Reppucci, N. (2008). Ecological and community level influences on child development. In T. P. Gullotta & G. M. Blau (Eds.), *Handbook of childhood behavioral issues: Evidence-based approaches to prevention and treatment* (pp. 69–86). New York, NY: Routledge/Taylor & Francis Group.

Arnocky, S., Sunderani, S., Miller, J. L., & Vaillancourt, T. (2012). Jealousy mediates the relationship between attractiveness comparison and females' indirect aggression. *Personal Relationships*, *19*(2), 290–303. doi: 10.1111/j.1475-6811.2011.01362.x

Arsenio, W. F., Adams, E., & Gold, J. (2009). Social information processing, moral reasoning, and emotion attributions: Relations with adolescents reactive and proactive aggression. *Child Development*, *80*, 1739–1755. doi: 10.1111/j.1467-8624.2009.01365.x

Ayoub, C. C., Fischer, K. W., & O'Connor, E. E. (2003). Analyzing development of working models for disrupted attachments: The case of family violence. *Attachment and Human Development*, *5*, 97–119. doi: 10.1080/1461673031000108478

Ayoub, C. C., Rogosh, F., Toth, S. L., O'Connor, E., Cicchetti, D., Rappolt-Schlichtmann, G., & Fischer, K. W. (2006). Cognitive and emotional differences in young maltreated children: A translational application of dynamic skill theory. *Development and Psychopathology 18*, 670–706. doi: 10.1017/S0954579406060342

Ayoub, C., Willett, J. B., & Robinson, D. (1993b). Families at risk of child maltreatment: Entry-level characteristics and growth in family functioning. *Child Abuse and Neglect*, *16*, 495–511.

Badcock, P. B. (2012). Evolutionary systems theory: A unifying meta-theory of psychological science. *Review of General Psychology*, *16*(1), 10–23. doi: 10.1037/a0026381

Bailey, C., & Ostrov, J. M. (2008). Differentiating Forms and Functions of Aggression in Emerging Adults: Associations with Hostile Attribution Biases and Normative Beliefs. *Journal of Youth and Adolescence*, *37*(6), 713–722. doi: 10.1007/s10964-007-9211-5

Banner, N. F. (2013). Mental disorders are not brain disorders. *Journal of Evaluation in Clinical Practice*, *19*, 509–513.

Barker, E. D., Tremblay, R. E., Nagin, D. S., Vitaro, F., & Lacourse, E. (2006). Development of male proactive and reactive physical aggression during adolescence. *Journal of Child Psychology and Psychiatry*, *47*, 783–790. doi: 10.1111/j.1469-7610.2005.01585.x

Barry, C. T., Golmaryami, F. N., Rivera-Hudson, N., & Frick, P. J. (2013). Evidence-based assessment of conduct disorder: Current considerations and preparation for DSM-5. *Professional Psychology: Research and Practice*, *44*, 56–63. doi: 10.1037/a0029202

Basseches, M., & Mascolo, M. F. (2010). *Psychotherapy as a developmental process*. New York, NY: Routledge/Taylor & Francis Group.

Baumeister, R. F., Smart, L., & Boden, J. M. (1996). Relation of threatened egotism to violence and aggression: The dark side of high self-esteem. *Psychological Review*, *103*, 5–33. doi: 10.1037/0033-295X.103.1.5

Bavelier, D., Green, C. S., Schrater, P., & Pouget, A. (2012). Brain plasticity through the life span: Learning to learn and action video games. *Annual Reviews of Neuroscience*, *35*, 391- 416.

Benikos, N., Johnstone, S. J., & Roodenrys, S. J. (2013a). Short-term training in the Go/Nogo task: Behavioural and neural changes depend on task demands. *International Journal of Psychophysiology*, *87*(3), 301–312. doi: 10.1016/j.ijpsycho.2012.12.001

Benikos, N., Johnstone, S. J., & Roodenrys, S. J. (2013b). Varying task difficulty in the Go/Nogo task: The effects of inhibitory control, arousal, and perceived effort on ERP components. *International Journal of Psychophysiology*, *87*, 262–272. doi: 10.1016/j.ijpsycho.2012.08.005

Bergman, L. R., & Vargha, A. (2013). Matching method to problem: A developmental science perspective. *European Journal of Developmental Psychology*, *10*(1), 9–28. doi: 10.1080/17405629.2012.732920

Bibace, R., & Kharlamov, N. A. (2013). The spiral: The concept of development after Werner and Kaplan. *Culture & Psychology*, *19*(4), 453–462. doi: 10.1177/1354067X13500324

Bierman, K. L. (2011). The promise and potential of studying the "invisible hand" of teacher influence on peer relations and student outcomes: A commentary. *Journal Of Applied Developmental Psychology*, *32*(5), 297–303. doi: 10.1016/j.appdev.2011.04.004

Blackburn, R., Logan, C., Donnelly, J. P., & Renwick, S. D. (2008). Identifying psychopathic subtypes: Combining an empirical personality classification of offenders with The Psychopathy Checklist-Revised. *Journal of Personality Disorders*, *22*(6), 604–622. doi: 10.1521/pedi.2008.22.6.604

Block, J. H., & Block, J. (1980). The role of ego-control and ego-resiliency in the organization of behavior. In W. A. Collins (Ed.), *Minnesota symposium on child psychology* (Vol. *13*, pp. 39–101). Hillsdale, NJ: Erlbaum. doi: 10.1521/pedi.2008.22.6.604

Boylan, K., Vaillancourt, T., Boyle, M., Szatmari, P. (2007). Comorbidity of internalizing disorders in children with oppositional defiant disorder. *European Child and Adolescent Psychiatry 16*(8): 484–494.

Bongers, I. L., Koot, H. M., van der Ende, J., & Verhulst, F. C. (2003). The normative development of child and adolescent problem behavior. *Journal of Abnormal Psychology*, *112*, 179–192. doi: 10.1037/0021-843X.112.2.179

Borsboom, D., Cramer, A. O. J., Schmittmann, V. D., Epskamp, S., & Waldorp, L. J. (2011). The small world of psychopathology. *PloS one*, *6*, e27407. doi: 10.1371/journal.pone.0027407

Bornstein, M. H. (2013). Mother-infant attunement: A multilevel approach via body, brain, and behavior. In M. Legerstee, D. W. Haley, & M. H. Bornstein (Eds.), *The infant mind: Origins of the social brain* (pp. 266–298). New York, NY: Guilford Press.

Bostow, D. E., & Bailey, J. B. (1969). Modification of severe disruptive and aggressive behavior using brief timeout and reinforcement procedures. *Journal of Applied Behavior Analysis*, *2*(1), 31–37. doi: 10.1901/jaba.1969.2-31

Boysen, G. A. (2007). An evaluation of the DSM concept of mental disorder. *Journal of Mind and Behavior*, *28*, 157–173.

Brendgen, M., Vitaro, F., Tremblay, R. E., & Lavoie, F. (2001). Reactive and proactive aggression: Predictions to physical violence in different contexts and moderating effects of parental monitoring and caregiving behavior. *Journal of Abnormal Child Psychology*, *29*, 293–304. doi: 10.1023/A:1010305828208

Bretherton, I., & Waters, E. (1985). *Growing points of attachment theory and research. Monographs of the Society for Research in Child Development*, *50*(1-2, Serial No. 209).

Breuer, J., & Freud, S. (1955). *Studies on hysteria* (A. Strachey & J. Strachey, Trans.). In J. Strachey (Ed.), *Standard edition of the complete psychological works of Sigmund Freud* (Vol. *2*). London, UK: Hogarth Press. (Original publication 1895.)

Brown, A. L., & Reeve, R. (1987). Bandwidths of competence: The role of supportive contexts in learning and development. In L. S. Liben (Ed.), *Development and learning: Conflict or congruence?* Hillsdale, NJ: Erlbaum.

Brugman, D., & Mancini, T. (2012). School moral atmosphere and normative orientation to explain aggressive and transgressive behaviours at secondary school. *Journal of Moral Education*, *41*(1), 1–22, doi: 10.1080/03057240.2011.652520

Bushman, B. J., & Anderson, C. A. (2001). Is it time to pull the plug on hostile versus instrumental aggression dichotomy? *Psychological Review*, *108*, 273–279. doi: 10.1037/0033-295X.108.1.273

Cairns, R. B. (1983). The emergence of developmental psychology. In P. H. Musen & W. Kessen, *Handbook of Child Psychology: Vol. 1: History, Theory, and Methods* (4th ed., pp. 41–102). New York: Wiley.

Calverley, R., Fischer, K. W., & Ayoub, C. (1994). Complex splitting of self-representations in sexually abused adolescent girls. *Development and Psychopathology*, *6*, 195–213.

Canadian Broadcasting Corporation. (1990). *To a safer place*. Video program. Ottowa, Ontario.

Carlson, V., Cicchetti, D., Barnett, D., & Braunwald, K. G. (1989). Finding order in disorganization: Lessons from research on maltreated infants' attachments to their caregivers. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 494–528). Cambridge, UK: Cambridge University Press.

Card, N. A., & Little, T. D. (2006). Proactive and reactive aggression in childhood and adolescence: A meta-analysis of differential relations with psychosocial adjustment. *International Journal of Behavioral Development*, *30*, 466–480. doi: 10.1177/0165025406071904

Case, R., & Edelstein, W. (Eds.) (1993). The new structuralism in cognitive development: Theory and research on individual pathways. In *Contributions to human development*, *23*. Basel, Switzerland: Karger.

Castellanos, X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: the search for endophenotypes. *Nature Reviews Neuroscience 3*, 617–628.

Cepeda, N., Blackwell, K., & Munakata, Y. (2013). Speed isn't everything: Complex processing speed measures mask individual differences and developmental changes in executive control. *Developmental Science*, *16*, 269–286. doi: 10.1111/desc.12024

Chen, C., McComas, J. J., Hartman, E., & Symons, F. J. (2011). A prospective sequential analysis of the relation between physical aggression and peer rejection acts in a high-risk preschool sample. *Early Education and Development*, *22*, 574–592. doi: 10.1080/10409289.2010.481706

Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development*, *55*(1), 1–7. doi: 10.2307/1129830

Cicchetti, D. (1989). How research on child maltreatment has informed the study of child development: Perspectives from developmental psychopathology. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 377–431). Cambridge, UK: Cambridge University Press. doi: 10.1017/CBO9780511665707.014

Cicchetti, D. (2013). Annual research review: Resilient functioning in maltreated children—Past, present, and future perspectives. *Journal of Child Psychology and Psychiatry*, *54*(4), 402–422.

Cicchetti, D. (2010). Developmental psychopathology. In M. E. Lamb, A. M. Freund, & R. M. Lerner (Eds.), *The handbook of life-span development, Vol 2: Social and emotional development* (pp. 511–589). Hoboken, NJ: John Wiley & Sons Inc.

Cicchetti, D., & Beeghly, M. (1987). Symbolic development in maltreated youngsters: An organizational perspective. In D. Cicchetti & M. Beeghly (Eds.), *Atypical symbolic development. New directions for child development* (Vol. *36*, pp. 5–29). San Francisco, CA: Jossey-Bass. doi: 10.1002/cd.23219873605

Cicchetti, D., & Toth, S. L. (2005). Child maltreatment. *Annual Review of Clinical Psychology*, *1*, 409–438.

Cicchetti, D., & Toth, S. L. (2009). The past achievements and future promises of developmental psychopathology: The coming of age of a discipline. *Journal of Child Psychology and Psychiatry*, *50*(1–2), 16–25. doi: 10.1111/j.1469-7610.2008.01979.x

Cherkes-Julkowski, M., & Mitlina, N. (1999). Self-organization of mother–child instructional dyads and later attention disorder. *Journal of Learning Disabilities*, *32*, 6–21. doi: 10.1146/annurev.clinpsy.1.102803.144029

Clark, K. E., & Ladd, G. W. (2000). Connectedness and autonomy support in parent–child relationships: Links to children's socioemotional orientation and peer relationships. *Developmental Psychology*, *36*, 485–498. doi: 10.1037/0012-1649.36.4.485

Cole, P. M., & Putnam, F. W. (1992). Effect of incest on self and social functioning: A developmental psychopathology perspective. *Journal of Consulting and Clinical Psychology*, *60*,174–184. doi: 10.1037/0022-006X.60.2.174

Connor, D. F., Chartier, K. G., Preen, E. C., & Kaplan, R. F. (2010). Impulsive aggression in attention-deficit-hyperactivity disorder: Symptom severity, comorbidity, and attention-deficit/hyperactivity disorder subtype. *Journal of Child and Adolescent Psychopharmacology*, *20*, 119–126. doi: 10.1089/cap.2009.0076

Conroy, M. A., Sutherland, K. S., Snyder, A. L., & Marsh, S. (2008). Classwide interventions effective instruction makes a difference. *Teaching Exceptional Children*, *40*(6), 24–30.

Cortese, S., (2012). The neurobiology and genetics of Attention-Deficit/Hyperactivity Disorder (ADHD): What every clinician should know. *European Journal of Paediatric Neurology*, *16*(5), 422–433. doi: http://dx.doi.org/10.1016/j.ejpn.2012.01.009

Cowan, N. (2003). Comparisons of developmental modeling frameworks and levels of analysis in cognition: connectionist and dynamic systems theories deserve attention, but don't yet explain attention. *Developmental Science*, *6*(4), 440–447. doi: 10.1111/1467-7687.00299

Cox, M. J., Mills-Koonce, R., Propper, C., & Gariépy, J. (2010). Systems theory and cascades in developmental psychopathology. *Development and Psychopathology*, *22*, 497–506. doi: 10.1017/S0954579410000234

Cramer, A. O. J., Waldorp, L. J., der Maas van, & Borsboom, D. (2010a). Comorbidity: A network perspective. *Behavioral and Brain Sciences*, *33*, 137–150.

Cramer, A. O. J., Waldorp, L. J., der Maas van, & Borsboom, D. (2010b). Complex realities require complex theories: Refining and extending the network approach to mental disorders. *Behavioral and Brain Sciences*, *33*, 178–193

Crittenden, P. M. (1988). Relationships at risk. In J. Belsky & T. Nezworski (Eds.), *Clinical implications of attachment theory* (pp. 136–174). Hillsdale, NJ: Erlbaum.

Danchin, É., Charmantier, A., Champagne, F. A., Mesoudi, A., Pujol, B., & Blanchet, S. (2011). Beyond DNA: integrating inclusive inheritance into an extended theory of evolution. *Nature Reviews Genetics*, *12*(7), 475–486.

Dandreaux, D. M., & Frick, P. J. (2009). Developmental pathways to conduct problems: A further test of the childhood and adolescent-onset distinction. *Journal of Abnormal Child Psychology*, *37*, 375–385.

De Zeeuw, P.; Weusten, J., van Dijk, S., van Belle, J., & Durston, S. (2012) Deficits in cognitive control, timing and reward sensitivity appear to be dissociable in ADHD. *PLoS ONE*, *7*(12): e51416. doi: 10.1371/journal.pone.0051416

Dempsey, A. G., & Storch, E. A. (2008). Relational victimization: The association between recalled adolescent social experiences and emotional adjustment in early adulthood. *Psychology in the Schools*, *45*, 310–322. doi: 10.1002/pits.20298

de Rivera, J. (2006). Conceptual encounter: The experience of anger. In C. T. Fischer (Ed.), *Qualitative research methods for psychologist: Introduction through empirical studies* (pp. 213–245). San Diego, CA: Elsevier Academic Press. doi: 10.1016/B978-012088470-4/50011-9

de Rivera, J. (1991). The structure and dynamics of emotion. In D. J. Ozer, J. R. Healy, & A. J. Stewart (Eds.) , *Perspectives in personality, Vol. 3: Part A: Self and emotion; Part B: Approaches to understanding lives* (pp. 191–212). London, UK: Jessica Kingsley Publishers.

Dirks, M. A., De Los Reyes, A., Briggs-Gowan, M., Cella, D., & Wakschlag, L. S. (2012). Annual research review: Embracing not erasing contextual variability in children's behavior—theory and utility in the selection and use of methods and informants in developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *53*(5), 558–574.

Dishion, T. J., Nelson, S. E., Winter, C. E., & Bullock, B. M. (2004). Adolescent friendship as a dynamic system: Entropy and deviance in the etiology and course of male antisocial behavior. *Journal of Abnormal Child Psychology*, *32*, 651–663. doi: 10.1023/B:JACP.0000047213.31812.21

Dishion, T. J., Eddy, J., Haas, E., Li, F., & Spracklen, K. (1997). Friendships and violent behavior during adolescence. *Social Development*, *6*, 207–223. doi: 10.1111/1467-9507.ep11631575

Dodge, K. A., Lochman, J. E., Harnish, J. D., Bates, J. E., & Pettit, G. S. (1997). Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. *Journal of Abnormal Psychology*, *106*, 37–51. doi: 10.1037/0021-843X.106.1.37

Dodge, K. A., & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology*, *39*, 349–371. doi: 10.1037/0012-1649.39.2.349

Edens, J. F., Campbell, J. S., & Weir, J. M. (2007). Youth psychopathy and criminal recidivism: A meta-analysis of the psychopathy checklist measures. *Law and Human Behavior*, *31*, 53–75. doi: 10.1007/s10979-006-9019-y

Eisenberg, N., Hofer, C., & Vaughan, J. (2007). Effortful Control and Its Socioemotional Consequences. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 287–306). New York, NY: Guilford Press.

Eley, T. C., Lichtenstein, P., & Moffitt, T. E. (2003). A longitudinal behavioral genetic analysis of the etiology of aggressive and nonaggressive antisocial behavior. *Development and Psychopathology*, *15*, 383–402. doi: 10.1017/S095457940300021X

Epkins, C. C., & Heckler, D. R. (2011). Integrating etiological models of social anxiety and depression in youth: Evidence for a cumulative interpersonal risk model. *Clinical Child and Family Psychology Review*, *14*, 329–376. doi: 10.1007/s10567-011-0101-8

Erdelyi, M. H. (1985). *Psychoanalysis: Freud's cognitive psychology*. New York: W.H. Freeman.

Eron, L., Huesmann, R., Spindler, A., Guerra, N., Henry, D., & Tolan, P. (2002). A cognitive-ecological approach to preventing aggression in urban settings: Initial outcomes for high-risk children. *Journal of Consulting and Clinical Psychology*, *70*(1), 179–194. doi: 10.1037/0022-006X.70.1.179

Estes, W. K., (1956). The problem of inference from curves based on group data. *Psychological Buletin*, *53*, 134–140.

Evans, D. E., & Rothbart, M. K. (2009). A two-factor model of temperament. *Personality and Individual Differences*, *47*, 565–570. doi: 10.1016/j.paid.2009.05.010

Evans, C. A., Nelson, L. J., & Porter, C. L. (2012). Making sense of their world: Sensory reactivity and novelty awareness as aspects of temperament and correlates of social behaviours in early childhood. *Infant and Child Development*, *21*(5), 503–520. doi: 10.1002/icd.1754

Evans, C. A., & Porter, C. L. (2009). The emergence of mother-infant co-regulation during the first year: Links to infants' developmental status and attachment. *Infant Behavior & Development*, *32*, 147–158. doi: 10.1016/j.infbeh.2008.12.005

Famularo, R., Kinscherff, R., Fenton, T., & Ayoub, C. (1994). Maternal and child posttraumatic stress disorder in cases of child maltreatment. *Child Abuse and Neglect*, *18*, 27–36.

Fanti, K. A. (2013). Individual, social, and behavioral factors associated with co-occurring conduct problems and callous-unemotional traits. *Journal of Abnormal Child Psychology*, *41*, 811–824. doi: 10.1007/s10802-013-9726-z

Fanti, K. A., & Kimonis, E. R. (2012). Bullying and victimization: The role of conduct problems and psychopathic traits. *Journal of Research on Adolescence*, *22*, 617–631. doi: 10.1111/j.1532-7795.2012.00809.x

Fanti, K. A., Frick, P. J., & Georgiou, S. (2009). Linking callous-unemotional traits to instrumental and non-instrumental forms of aggression. *Journal of Psychopathology and Behavioral Assessment*, *31*, 285–298.

Farmer, T. W., Farmer, E. M. Z., Estell, D. B., & Hutchins, B. C. (2007). The developmental dynamics of aggression and the prevention of school violence. *Journal of Emotional and Behavioral Disorders*, *15*(4), 197–208.

Farrell, A. D., Henry, D. B., Mays, S. A., & Schoeny, M. E. (2011). Parents as moderators of the impact of school norms and peer influences on aggression in middle school students. *Child Development*, *82*(1), 146–161. doi: 10.1111/j.1467-8624.2010.01546.x

Feffer, M. (1982). *The structure of Freudian thought*. NewYork, NY: International Universities Press.

Feiring, C., Cleland, C. M., & Simon, V. A. (2010). Abuse-specific self-schemas and self-functioning: A prospective study of sexually abused youth. *Journal of Clinical Child and Adolescent Psychology*, *39*, 35–50. doi: 10.1080/15374410903401112

Ferguson, T. J., Eyre, H. L., & Ashbaker, M. (2000). Unwanted identities: A key variable in shame–anger links and gender differences in shame. *Sex Roles*, *42*, 133–157.

Ferguson-Smith, A. C. (2011). Genomic imprinting: the emergence of an epigenetic paradigm. *Nature Reviews Genetics*, *12*(9), 663.

Ferrer, E., & Helm, J. L. (2013). Dynamical systems modeling of physiological coregulation in dyadic interactions. *International Journal of Psychophysiology*, *88*(3), 296–308. doi: 10.1016/j.ijpsycho.2012.10.013

Ferrer, E., & Steele, J. (2012). Dynamic systems analysis of affective processes in dyadic interactions using differential equations. In J. R. Harring & G. R. Hancock (Eds.), *Advances in longitudinal methods in the social and behavioral sciences* (pp. 111–134). Charlotte, NC: IAP Information Age Publishing.

Fischer, K. W. (1980). A theory of cognitive development: The control and construction of hierarchies of skills. *Psychological Review*, *87*, 477–531. doi: 10.1037/0033-295X.87.6.477

Fischer, K. W. (2008). Dynamic cycles of cognitive and brain development: Measuring growth in mind, brain, and education. In A. M. Battro, K. W. Fischer, & P. J. Léna (Eds.), *The educated brain: Essays in neuroeducation* (pp. 127–150). New York, NY: Cambridge University Press. doi: 10.1017/CBO9780511489907.010

Fischer, K. W., Bullock, D., Rotenberg, E. J., & Raya, P. (1993). The dynamics of competence: How context contributes directly to skill. In R. H. Wozniak & K. W. Fischer (Eds.), *Development in context: Acting and thinking in specific environments* (pp. 93–117). Hillsdale, NJ: Erlbaum.

Fischer, K. W., & Pipp, S. L. (1984). Development of the structures of unconscious thought. In K. Bowers & D. Meichenbaum (Ed.), *The unconscious reconsidered* (pp. 88–148). New York, NY: Wiley.

Fischer, K. W., & Rose, S. P. (1994). Dynamic development of coordination of components in brain and behavior: A framework for theory and research. In G. Dawson & K. W. Fischer (Eds.), *Human behavior and the developing brain* (pp. 3–66). New York, NY: Guilford Press.

Fischer, K. W., Shaver, P., & Carnochan, P. G. (1990). How emotions develop and how they organize development. *Cognition and Emotion*, *4*, 81–127. doi: 10.1080/02699939008407142

Fischer, K. W., & Watson, M. W. (1981). Explaining the Oedipus conflict. In K. W. Fischer (Ed.), *Cognitive development. New directions for child development* (Vol. *12*, pp. 79–92). San Francisco, CA: Jossey-Bass.

Fischer, K. W., Yan, Z., & Stewart, J. (2003). Adult cognitive development: Dynamics in the developmental web. In J. Valsiner & K. Connolly (Eds.), *Handbook of developmental psychology* (pp. 491–516). Thousand Oaks, CA: Sage.

Fisher, A. J., Newman, M. G., & Molenaar, P. M. (2011). A quantitative method for the analysis of nomothetic relationships between idiographic structures: Dynamic patterns create attractor states for sustained posttreatment change. *Journal of Consulting and Clinical Psychology*, *79*(4), 552–563. doi: 10.1037/a0024069

Flavell, J. (1982). On cognitive development. *Child Development*, *53*, 1–10.

Foà, C., Brugman, D., & Mancini, T. (2012) School moral atmosphere and normative orientation to explain aggressive and transgressive behaviours at secondary school. *Journal of Moral Education*, *41*:1, 1–22, doi: 10.1080/03057240.2011.652520

Fogel, A. (1993). *Developing through relationships: Origins of communication, self, and culture*. Chicago, IL: University of Chicago Press.

Fogel, A., Garvey, A., Hsu, H., & West-Stroming, D. (2006). *Change processes in relationships: A relational-historical research approach*. New York, NY: Cambridge University Press. doi: 10.1017/CBO9780511489686.

Fogel, A., & Thelen, E. (1987). Development of early expressive and communicative action: Reinterpreting the evidence from a dynamic systems perspective. *Developmental Psychology*, *23*, 747–761. doi: 10.1037/0012-1649.23.6.747

Foulkes, D. (1982). *Children's dreams: Longitudinal studies*. New York, NY: Wiley.

Frances, A. (2013). *Saving normal: An insider's revolt against out-of-control psychiatric diagnosis, DSM-5, Big Pharma, and the medicalization of ordinary life*. New York, NY: William Morrow & Co.

Freud, A. (1966). *The ego and the mechanisms of defense* (C. Baines, Trans.). New York, NY: International Universities Press. (Original publication 1936.)

Freud, S. (1961/1963). *Introductory lectures on psychoanalysis*. In J. Strachey (Ed.), *Standard edition of the psychological works of Sigmund Freud* (Vol. 15 & 16). London, UK: Hogarth Press. (Original publication 1917.)

Freud, S. (1961). *The ego and the id* (J. Riviere & J. Strachey, Trans.). In J. Strachey (Ed.), *Standard edition of the complete psychological works of Sigmund Freud* (Vol. 19). London, UK: Hogarth Press. (Original publication 1923.)

Frick, P. J., & Viding, E. (2009). Antisocial behavior from a developmental psychopathology perspective. *Development and Psychopathology*, *21*, 1111–1131. doi: 10.1017/S0954579409990071

Frick, P. J., & White, S. F. (2008). Research review: The importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. *Journal of Child Psychology and Psychiatry*, *49*, 359–375. doi: 10.1111/j.1469-7610.2007.01862.x

Frijda, N. H. (1986). *The emotions*. Cambridge, UK: Cambridge University Press.

Frijda, N. H. (2009). Emotion experience and its varieties. *Emotion Review*, *1*, 264–271. doi: 10.1177/1754073909103595

Gullotta, T. P., & Blau, G. M. (2008). (Eds.), *Handbook of childhood behavioral issues: Evidence-based approaches to prevention and treatment*. New York, NY: Routledge/Taylor & Francis Group

Gogtay, N., & Thompson, P. M. (2010). Mapping Gray Matter Development: Implications for typical development and vulnerability to psychopathology. *Brain and Cognition*, *72*(1), 6.

Goldin-Meadow, S., Alibali, M., & Church, R. B. (1993). Transitions in concept acquisition: Using the hand to read the mind. *Psychological Review*, *100*, 279–297. doi: 10.1037/0033-295X.100.2.279

Goodman, S. H., & Brand, S. R. (2009). Maternal depression and infant mental health. *Handbook of infant mental health*. 3rd Ed. New York, NY: Guilford.

Gorenstein, E. E., & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review*, *87*(3), 301–315. doi: 10.1037/0033-295X.87.3.301

Granic, I. (2005). Timing is everything: Developmental psychopathology from a dynamic systems perspective. *Developmental Review*, *25*(3–4), 386–407. doi: 10.1016/j.dr.2005.10.005

Granic, I., & Hollenstein, T. (2006). A survey of dynamic systems methods for developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, Vol 1: Theory and method* (2nd ed., pp. 889–930). Hoboken, NJ: John Wiley & Sons Inc.

Granic, I., & Patterson, G. R. (2006). Toward a comprehensive model of antisocial development: A dynamic systems approach. *Psychological Review*, *113*, 101–131. doi: 10.1037/0033-295X.113.1.101

Granott, N. (2002). How microdevelopment creates macrodevelopment: Reiterated sequences, backward transitions, and the zone of current development. In N. Granott & J. Parziale (Eds.), *Microdevelopment: Transition processes in development and learning* (pp. 213–242). Cambridge, UK: Cambridge University Press. doi: 10.1017/CBO9780511489709.009

Gottlieb, G., & Halpern, C. (2002). A relational view of causality in normal and abnormal development. *Development and Psychopathology*, *14*, 421–435. doi: 10.1017/S0954579402003024

Gottlieb, G. (1991). Experiential canalization of behavioral development: Results. *Developmental Psychology*, *27*(1), 35–39. doi: 10.1037/0012-1649.27.2.35

Haan, N. (1977). *Coping and defending*. New York, NY: Academic Press.

Haggis, T. (2008). 'Knowledge must be contextual': Some possible implications of complexity and dynamic systems theories for educational research. *Educational Philosophy and Theory*, *40*(1), 158–176.

Hastings, P. D., Fortier, I., Utendale, W. T., Simard, L. R., & Robaey, P. (2009). Adrenocortical functioning in boys with attention-deficit/hyperactivity disorder: Examining subtypes of ADHD and associated comorbid conditions. *Journal of Abnormal Child Psychology*, *37*, 565–578. doi: 10.1007/s10802-008-9292-y

Harter, S., & Buddin, B. (1987). Children's understanding of the simultaneity of two emotions: A five-stage developmental sequence. *Developmental Psychology*, *23*, 388–399. doi: 10.1037/0012-1649.23.3.388

Hawes, D. J., Dadds, M. R., Brennan, J., Rhodes, T., & Cauchi, A. (2013). Revisiting the treatment of conduct problems in children with callous-unemotional traits. *Australian and New Zealand Journal of Psychiatry*, *47*, 646–653.

Hayling, C. C., Cook, C., & Gresham, F. M. State, T., & Kern, L. (2008). An analysis of the status and stability of the behaviors of students with emotional and behavioral difficulties. *Journal of Behavioral Education*, *17*(1), 24–42.

Hechtman, L. (2009). Comorbidity and neuroimaging in attention-deficit hyperactivity disorder. *Canadian Journal of Psychiatry*, *54*, 649–650. doi: 10.1177/0004867413484092

Heider, K. (1991). *Landscapes of emotion: Mapping three cultures of emotion in Indonesia*. New York, NY: Cambridge University Press.

Hejdenberg, J., & Andrews, B. (2011). The relationship between shame and different types of anger: A theory-based investigation. *Personality and Individual Differences*, *50*, 1278–1282. doi: 10.1016/j.paid.2011.02.024

Hencke, R. W. (1996). *Self stories: Effects of children's emotional styles on their appropriation of self-schemata*. Unpublished doctoral dissertation, Harvard University, Cambridge, MA.

Herman, J. (1992). *Trauma and recovery*. New York, NY: Basic Books.

Herman, K. C., Ostrander, R., Walkup, J. T., Silva, S. G., & March, J. S. (2007). Empirically derived subtypes of adolescent depression: Latent profile analysis of co-occurring symptoms in the Treatment for Adolescents with Depression Study (TADS). *Journal of Consulting and Clinical Psychology*, *75*(5), 716–728. doi: 10.1037/0022-006X.75.5.716

Hilgard, E. R. (1977). *Divided consciousness*. New York, NY: Wiley.

Hoekzema, E., Carmona, S., Tremols, V., Gispert, J. D., Guitart, M., Fauquet, J., & Vilarroya, O. (2010). Enhanced neural activity in frontal and cerebellar circuits after cognitive training in children with attention-deficit/hyperactivity disorder. *Human Brain Mapping*, *31*(12), 1942–50. doi: 10.1002/hbm.20988

Hollenstein, T. (2013). *State space grids: Depicting dynamics across development*. New York, NY: Springer Science + Business Media. doi: 10.1007/978-1-4614-5007-8

Howe, M. L., & Lewis, M. D. (2005). The importance of dynamic systems approaches for understanding development. *Developmental Review*, *25*(1), 247–251.

Hubbard, J. A., McAuliffe, M. D., Morrow, M. T., & Romano, L. J. (2010). Reactive and proactive aggression in childhood and

adolescence: Precursors, outcomes, processes, experiences, and measurement. *Journal of Personality*, *78*, 95–118. doi: 10.1111/j.1467-6494.2009.00610.x

Hughes, J., & Chen (2011). Reciprocal effects and prediction of academic self-efficacy. *Journal of Applied Developmental Psychology*, *32*, 247–256.

Hulette, A. C., Kaehler, L. A., & Freyd, J. J. (2011). Intergenerational associations between trauma and dissociation. *Journal of Family Violence*, *26*, 217–225. doi: 10.1007/s10896-011-9357-5

Huxley, J. (1942). Evolution: The Modern Synthesis.

Immordino-Yang, M. H., & Damasio, A. R. (2007). We feel, therefore we learn: The relevance of affective and social neuroscience to education. *Mind, Brain and Education*, *1*, 3–10.

Isely, P. J., Isely, P., Freiburger, J., & McMackin, R. (2008). In their own voices: A qualitative study of men abused as children by Catholic clergy. *Journal of Child Sexual Abuse*, *17*(3–4), 201–215.

Ivanova, M. Y., & Israel, A. C. (2006). Family stability as a protective factor against psychopathology for inner-city children receiving psychological services. *Journal of Clinical Child and Adolescent Psychology*, *35*, 564–570.

Izard, C. E., Ackerman, B. P., Schoff, K. M., & Fine, S. E. (2000). Self-organization of discrete emotions, emotion patterns, and emotion–cognition relations. In M. D. Lewis & I. Granic (Eds.), *Emotion, development, and self-organization: Dynamic systems approaches to emotional development* (pp. 15–36). New York, NY: Cambridge University Press. doi: 10.1017/CBO9780511527883.003

Jablonka, E., & Lamb, M. J. (2006). The evolution of information in the major transitions. *Journal of Theoretical Biology*, *239*(2), 236–246.

Jacobs, N., & Harper, B. (2013). The effects of rejection sensitivity on reactive and proactive aggression. *Aggressive behavior*, *39*(1), 3–12.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Dodge, K. A., Rutter, M., Taylor, A., & Tully, L. A. (2005). Nature × nurture: Genetic vulnerabilities interact with physical maltreatment to promote conduct problems. *Development and Psychopathology*, *17*(1), 67–84. doi: 10.1017/S0954579405050042

Ji, L., Wei, X., Chen, L., & Zhang, W. (2012). Peer relationship adversities and children's aggression during late childhood: The mediating roles of self-conception and peer beliefs. *Acta Psychologica Sinica*, *44*, 1479–1489. doi: 10.3724/SP.J.1041.2012.01479

John, Y. J., Bullock, D., Zikopoulos, B., & Barbas, H. (2013). Anatomy and computational modeling of networks underlying cognitive-emotional interaction. *Frontiers in Human Neuroscience*, *7*, 1–26. doi: 101 .10.3389/fnhum.2013.00101

Kagan, J. (2007). A trio of concerns. *Perspectives on Psychological Science*, *2*, 361–376. doi: 10.1111/j.1745-6916.2007.00049.x

Kagan, J. (1997). Conceptualizing psychopathology: The importance of development profiles. *Development and Psychopathology*, *9*, 321–334. doi: 10.1017/S0954579497002071

Kagan, J., & Snidman, N. (1990). Temperamental contributions to human development: The biological characteristics of infants influence their initial behavior to unfamiliar contexts. *Research & Clinical Center for Child Development*, *12*, 59–70.

Kagan, J., Snidman, N., McManis, M., Woodward, S., & Hardway, C. (2002). One measure, one meaning: Multiple measures, clearer meaning. *Development and Psychopathology*, *14*(3), 463–475. doi: 10.1017/S0954579402003048

Kahn, R. E., Byrd, A. L., & Pardini, D. A. (2013). Callous-unemotional traits robustly predict future criminal offending in young men. *Law and Human Behavior*, *37*, 87–97. doi: 10.1037/b0000003

Kawa, S., & Giordano, J. (2012). A brief historicity of the Diagnostic and Statistical Manual of Mental Disorders: Issues and implications for the future of psychiatric canon and practice. *Philosophy, Ethics, and Humanities in Medicine*, *7*(2). 5341–5347.

Kempes, M., Matthys, W., de Vries, H., & van Engeland, H. (2005). Reactive and proactive aggression in children: A review of theory, findings and the relevance for child and adolescent psychiatry. *European Child & Adolescent Psychiatry*, *14*, 11–19. doi: 10.1007/s00787-005-0432-4

Kelso, J. (1995). *Dynamic patterns: The self-organization of brain and behavior*. Cambridge, MA: MIT Press.

Kemp, F. (1996). The ideology of aversive treatment as applied to clients and colleagues. *Child & Family Behavior Therapy*, *18*(1), 9–27. doi: 10.1300/J019v18n01_02

Kenardy, J., Smith, A., Spence, S. H., Lilley, P.-R., Newcombe, P., Dob, R., & Robinson, S. (2007). Dissociation in children's trauma narratives: An exploratory investigation. *Journal of Anxiety Disorders*, *21*, 456–466. doi: 10.1016/j.janxdis.2006.05.007

Kim, J., & Cicchetti, D. (2006). Longitudinal trajectories of self-system processes and depressive symptoms among maltreated and nonmaltreated children. *Child Development*, *77*, 624–639. doi: 10.1111/j.1467-8624.2006.00894.x

Kochanska, G., & Aksan, N. (2006). Children's conscience and self-regulation. *Journal of Personality*, *74*, 1587–1617.

Kochanska, G., Aksan, N., & Joy, M. E. (2007). Children's fearfulness as a moderator of parenting in early socialization: Two longitudinal studies. *Developmental Psychology*, *43*, 222–237. doi: 10.1111/j.1467-6494.2006.00421.x

Kochanska, G., & Aksan, N. (2004). Development of mutual responsiveness between parents and their young children. *Child Development*, *75*, 1657–1676. doi: 10.1111/j.1467-8624.2004.00808.x

Kroesbergen, E. H., & Van Luit, J. E. (2003). Mathematics interventions for children with special educational needs a meta-analysis. *Remedial and special education*, *24*(2), 97–114.

Kunnen, S. (2012a). *A dynamic systems approach to adolescent development*. New York, NY: Psychology Press.

Kunnen, S. (2012b). The art of building dynamic systems models. In S. Kunnen (Ed.), *A dynamic systems approach to adolescent development* (pp. 99–116). New York, NY: Psychology Press.

Lahat, A., Hong, M., & Fox, N. A. (2011). Behavioural inhibition: Is it a risk factor for anxiety?. *International Review of Psychiatry*, *23*, 248–257. doi: 10.3109/09540261.2011.590468

Lane, K. L., Barton-Arwood, S., Nelson, J. R., & Wehby, J. H. (2008). Academic performance of students with emotional and behavioral disorders served in a self-contained setting. *Journal of Behavioral Education*, *17*, 43–62.

Lane, K. L., Wehby, J. H. & Barton-Arwood, S. (2005). Students with and at-risk for emotional and behavioral disorders: Meeting their social and academic needs. *Preventing School Failure*, *49*, pp. 6–9.

Lewandowski, G. R., & Strohmetz, D. B. (2009). Actions can speak as loud as words: Measuring behavior in psychological science. *Social and Personality Psychology Compass*, *3*, 992–1002. doi: 10.1111/j.1751-9004.2009.00229.x

Lamborn, S. D., Fischer, K. W., & Pipp, S. (1994). Constructive criticism and social lies: A developmental sequence for understanding honesty and kindness in social interactions. *Developmental Psychology*, *30*, 495–508. doi: 10.1037/0012-1649.30.4.495

Lazarus, R. S. (1991). *Emotion and adaptation*. New York. NY: Oxford University Press.

Lerner, R. M., Hess, L. E., & Nitz, K. (1990). A developmental perspective on psychopathology. In M. Hersen & C. G. Last (Eds.), *Handbook of child and adult psychopathology: A longitudinal perspective* (pp. 9–32). Elmsford, NY: Pergamon Press.

Lewis, M. D. (1996). Self-organising cognitive appraisals. *Cognition and Emotion*, *10*, 1–25. doi: 10.1080/026999396380367

Li, J., Wang, L., & Fischer, K. W. (2004). The organization of Chinese shame concepts. *Cognition and Emotion*, *18*, 767–797.

Lindhout, I. E., Markus, M. H., Hoogendijk, T. G., & Boer, F. (2009). Temperament and parental child-rearing style: Unique contributions to clinical anxiety disorders in childhood. *European Child & Adolescent Psychiatry*, *18*, 439–446. doi: 10.1007/s00787-009-0753-9

Loeber, R., Stouthamer-Loeber, M., & Green, S. M. (1991). Age at onset of problem behaviour in boys, and later disruptive and delinquent behaviours. *Criminal Behaviour and Mental Health*, *1*, 229–246.

Lickliter, R., & Honeycutt, H. (2013). A developmental evolutionary framework for psychology. *Review of General Psychology*, *17*, 184–189. doi: 10.1037/a0032932

Luckner, & Pianta, R. (2011). Teacher–student interactions in fifth grade classrooms: Relations with children's peer behavior. *Journal of Applied Developmental Psychology*, *32*, 266–275. doi: 10.1016/j.appdev.2011.02.010

Maddux, J. E., Gosselin, J. T., & Winstead, B. A. (2005). Conceptions of psychopathology: A social constructionist perspective. In J. E. Maddux & B. A. Winstead (Eds.), *Psychopathology: Foundations for a contemporary understanding* (pp. 3–18). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Main, M., & Hesse, E. (1990). Parents' unresolved traumatic experiences are related to infant disorganized attachment status: Is frightened and/or frightening parental behavior the linking mechanism? In M. T. Greenberg, D. Cicchetti, & E. M. Cummings (Eds.), *Attachment in the preschool years* (pp. 161–182). Chicago, IL: University of Chicago Press.

Mascolo, M. F. (2005). Change processes in development: The concept of coactive scaffolding. *New Ideas in Psychology*, *23*, 185–196. doi: 10.1016/j.newideapsych.2006.05.002

Mascolo, M. F., & Fischer, K. W. (2010). The dynamic development of thinking, feeling, and acting over the lifespan. In R. M. Lerner & W. F. Overton (Eds.), *Handbook of life-span development. Vol. 1: Biology, cognition, and methods across the lifespan* (pp. 149–194). Hoboken NJ: Wiley. doi: 10.1002/9780470880166.hlsd001006

Mascolo, M. F., Fischer, K. W., & Li, J. (2003). Dynamic development of component systems of emotions: Pride, shame and guilt in China and the United States. In R. J. Davidson, K. R. Scherer, & H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 375–408). New York, NY: Oxford University Press.

Mascolo, M. F., Harkins, D., & Harakal, T. (2000). The dynamic construction of emotion: Varieties in anger. In M. D. Lewis & I. Granic (Eds.), *Emotion, development, and self-organization: Dynamic systems approaches to emotional development* (pp. 125–152). New York, NY: Cambridge University Press. doi: 10.1017/CBO9780511527883.007

Martel, M. M., Gremillion, M., Roberts, B., von Eye, A., & Nigg, J. T. (2010). The structure of childhood disruptive behaviors. *Psychological Assessment*, *22*(4), 816–826. doi: 10.1037/a0020975

Martel, M. M., Goth-Owens, T., Martinez-Torteya, C., & Nigg, J. T. (2010). A person-centered personality approach to heterogeneity in attention-deficit/hyperactivity disorder (ADHD). *Journal of Abnormal Psychology*, *119*, 186–196. doi: 10.1037/a0017511

Masten, A. S. (2007). Resilience in developing systems: Progress and promise as the fourth wave rises. *Development and Psychopathology*, *19*, 921–930.

McAssey, M., Helm, J., Hsieh, F., Sbarra, D. A., & Ferrer, E. (2013). Methodological advances for detecting physiological synchrony during dyadic interactions. *Methodology*, *9*(2), 41–53. doi: 10.1027/1614-2241/a000053

McAuliffe, M. D., Hubbard, J. A., Rubin, R. M., Morrow, M. T., & Dearing, K. F. (2006). Reactive and proactive aggression: Stability of constructs and relations to correlates. *The Journal of Genetic Psychology: Research and Theory on Human Development*, *167*, 365–382. doi: 10.1017/S0954579407000442

McMahon, R. J., & Frick, P. J. (2005). Evidence-based assessment of conduct problems in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, *34*, 477–505. doi: 10.1207/s15374424jccp3403_6

Merrell, K. W., & Walker, H. M. (2004). Deconstructing a definition: Social maladjustment versus emotional disturbance and moving the EBD field forward. *Psychology in the Schools*, *41*(8), 899–910. doi: 10.1002/pits.20046

Mischel, W., & Mischel, F. (1958). Psychological aspects of spirit possession: A reinforcement analysis. *American Anthropologist*, *60*, 249–260.

Molenaar, P. M. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement: Interdisciplinary Research and Perspectives*, *2*, 201–218. doi: 10.1207/s15366359mea0204_1

Molenaar, P. C., & Campbell, C. G. (2009). The new person-specific paradigm in psychology. *Current Directions in Psychological Science*, *18*(2), 112–117. doi: 10.1111/j.1467-8721.2009.01619.x

Molenaar, P. M., & Goode, T. (2013). New methods for sequential behavior analysis. In G. J. Madden, W. V. Dube, T. D. Hackenberg, G. P. Hanley, & K. A. Lattal (Eds.), *APA handbook of behavior analysis, Vol. 1: Methods and principles* (pp. 267–280). Washington, DC: American Psychological Association. doi: 10.1037/13937-012.

Molenaar, P. M., & Ram, N. (2010). Dynamic modeling and optimal control of intraindividual variation: A computational paradigm for nonergodic psychological processes. In S. Chow, E. Ferrer, & F. Hsieh (Eds.), *Statistical methods for modeling human dynamics: An interdisciplinary dialogue* (pp. 13–37). New York, NY: Routledge/Taylor & Francis Group.

Muñoz, L. C., Qualter, P., & Padgett, G. (2011). Empathy and bullying: Exploring the influence of callous-unemotional traits. *Child Psychiatry and Human Development*, *42*, 183–196. doi: 10.1007/s10578-010-0206-1

Muris, P., van Brakel, A. L., Arntz, A., & Schouten, E. (2011). Behavioral Inhibition as a Risk Factor for the Development of Childhood Anxiety Disorders: A Longitudinal Study. *Journal of Child and Family Studies*, *20*(2), 157–170. doi: 10.1007/s10826-010-9365-8

Myin-Germeys, I., Marcelis, M., Krabbendam, L., Delespaul, P., & van Os, J. (2005). Subtle fluctuations in psychotic phenomena as functional states of abnormal dopamine reactivity in individuals at risk. *Biological Psychiatry*. *58*, 105–110.

Nandi, A., Beard, J. R., Galea, S. (2009). Epidemiologic heterogeneity of common mood and anxiety disorders over the lifecourse in the general population: A systematic review. *BMC Psychiatry*, *9*, 31.

Navarro, J., Arrieta, C., & Ballén, C. (2007). An approach to the study of the dynamics of work motivation using the diary method. *Nonlinear Dynamics, Psychology, and Life Sciences*, *11*(4), 473–498.

Nigg, J. T. (2005). Neuropsychologic theory and findings in attention-deficit/hyperactivity disorder: The state of the field and salient challenges for the coming decade. *Biological Psychiatry*, *57*(11), 1424–1435. doi: 10.1016/j.biopsych.2004.11.011

Orobio de Castro, B., Brendgen, M., Van Boxtel, H., Vitaro, F., & Schaepers, L. (2007). 'Accept me, or else': Disputed overestimation of social competence predicts increases in proactive aggression. *Journal of Abnormal Child Psychology*, *35*, 165–178. doi: 10.1007/s10802-006-9063-6

Osgood, C. E., Suci, G. J., & Tannenbaum, P. (1957). *The measurement of meaning*. Urbana, IL: University of Illinois Press.

Pearce, M. J., Jones, S. M., Schwab-stone, M. E., & Ruchkin, V. (2003). The protective effects of religiousness and parent involvement on the development of conduct problems among youth exposed to violence. *Child development*, *74*(6), 1682–1696.

Peer, J., Strachan, E., & Spaulding, W. (2008). Heterogeneity in behavioral treatment response in severe mental illness. *Journal of Nervous and Mental Disease*, *196*, 198–206.

Pepler, D. J., Craig, W. M., Connolly, J. A., Yuile, A., McMaster, L., & Jiang, D. (2006). A developmental perspective on bullying. *Aggressive Behavior*, *32*, 376–384. doi: 10.1002/ab.20136

Piaget, J. (1951). *Play, dreams, and imitation in childhood* (C. Gattegno & F.M. Hodgson, Trans.). New York, NY: Harcourt Brace. (Original publication 1946.)

Piaget, J. (1957). Logique et équilibre dans les comportements du sujet. *Études d'Épistémologie Génétique*, *2*, 27–118.

Piaget, J., & Szeminska, A. (1952). *The child's conception of number* (C. Gattegno & F.M. Hodgson, Trans.). London, UK: Routledge & Kegan Paul. (Original publication 1941.)

Platt, S. A., & Sanislow, C. A. (1988). Norm-of-reaction: Definition and misinterpretation of animal research. *Journal of Comparative Psychology*, *102*, 254–261. doi: 10.1037/0735-7036.102.3.254

Poulin, F., & Boivin, M. (2000). Reactive and proactive aggression: Evidence of a two-factor model. *Psychological Assessment*, *12*, 115–122. doi: 10.1037/1040-3590.12.2.115

Prior, M., Smart, D., Sanson, A., & Oberklaid, F. (2000). Does shy-inhibited temperament in childhood lead to anxiety problems in adolescence? *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*, 461–468. doi: 10.1097/00004583-200004000-00015

Putnam, F. W. (1989). *Diagnosis and treatment of multiple personality disorder*. New York, NY: Guilford.

Putnam, F. W. (1994). Dissociation and disturbances of self. In D. Cicchetti & S. L. Toth (Eds.), *Disorders and dysfunctions of the self* (Vol. *5*, pp. 251–266). Rochester, NY: University of Rochester.

Putnam, F. W., Helmers, K., & Trickett, P. K. (1993). Development, reliability, and validity of a child dissociation scale. *Child Abuse & Neglect*, *17*, 731–741.

Raeff, C. (2011). Distinguishing between development and change: Reviving organismic-developmental theory. *Human Development*, *54*(1), 4–33. doi: 10.1159/000324334

Rahm, G., Renck, B., & Ringsberg, K. C. (2013). Psychological distress among women who were sexually abused as children. *International Journal of Social Welfare*, *22*(3), 269–278. doi: 10.1111/j.1468-2397.2012.00898.x

Ram, N., Brose, A., & Molenaar, P. M. (2013). Dynamic factor analysis: Modeling person-specific process. In T. D. Little (Ed.), *The Oxford handbook of quantitative methods: Statistical analysis* (Vol. 2, pp. 441–457). New York, NY: Oxford University Press.

Rapee, R. M., Schniering, C. A., & Hudson, J. L. (2009). Anxiety disorders during childhood and adolescence: Origins and treatment. *Annual Review of Clinical Psychology*, *5311-341*. doi: 10.1146/annurev.clinpsy.032408.153628

Raya, P. (1996). *Pretense in pair-play therapy: Examining the understanding of emotions in young at-risk children*. Unpublished doctoral dissertation, Harvard University, Cambridge, MA.

Reid, R., Gonzalez, J. E., Nordness, P. D., Trout, A., & Epstein, M. H. (2004). A meta-analysis of the academic status of students with emotional/behavioral disturbance. *The Journal of Special Education*, *38*, 130–144.

Reijntjes, A., Kamphuis, J. H., Prinzie, P., Boelen, P. A., van der Schoot, M., & Telch, M. J. (2011). Prospective linkages between peer victimization and externalizing problems in children: A meta-analysis. *Aggressive Behavior*, *37*, 215–222. doi: 10.1002/ab.20374

Reijntjes, A., Thomaes, S., Bushman, B. J., Boelen, P. A., de Castro, B., & Telch, M. J. (2010). The outcast-lash-out effect in youth: Alienation increases aggression following peer rejection. *Psychological Science*, *21*, 1394–1398. doi: 10.1177/0956797610381509

Romero-Canyas, R., Downey, G., Berenson, K., Ayduk, O., & Kang, N. (2010). Rejection sensitivity and the rejection-hostility link in romantic relationships. *Journal of Personality*, *7*, 119–148. doi: 10.1111/j.1467-6494.2009.00611.x

Rose, L. T., Rouhani, P., & Fischer, K. W. (2013). The Science of the Individual. *Mind, Brain, & Education*, *7*, 3, 152–158.

Roth-Hanania, R., Davidov, M., & Zahn-Waxler, C. (2011). Empathy development from 8 to 16 months: Early signs of concern for others. *Infant Behavior & Development*, *34*, 447–458. doi: 10.1016/j.infbeh.2011.04.007

Rubin, K. H., Burgess, K. B., Dwyer, K. M., & Hastings, P. D. (2003). Predicting preschoolers' externalizing behaviors from toddler temperament, conflict, and maternal negativity. *Developmental Psychology*, *39*, 164–176. doi: 10.1037/0012-1649.39.1.164

Rutter, M. (2005). Multiple meanings of a developmental perspective on psychopathology. *European Journal of Developmental Psychology*, *2*, 221–252.

Saarni, C., & Lewis, M. (Eds.). (1993). *Lying and deception in everyday life*. New York, NY: Guilford.

Sapienza, J. K., & Masten, A. S. (2011). Understanding and promoting resilience in children and youth. *Current Opinion in Psychiatry*, *24*, 267–273. doi: 10.1097/YCO.0b013e32834776a8

Schensul, J. (2009). Community, culture and sustainability in multilevel dynamic systems intervention science. *American Journal of Community Psychology*, *43*(3–4), 241–256. doi: 10.1007/s10464-009-9228-x

Schiepek, G. (2003). A dynamic systems approach to clinical case formulation. *European Journal of Psychological Assessment*, *19*(3), 175–184. doi: 10.1027/1015-5759.19.3.175

Schmittmann, V. D., Cramer, A. O. J., Waldorp, L. J., Epskamp, S., Kievit, R. A., & Borsboom, D. (2013). Deconstructing the construct: A network perspective on psychological phenomena. *New Ideas in Psychology*, *31*, 43–53. doi: 10.1016/j.newideapsych.2011.02.007

Shanahan, S., Jones, J., Thomas-Peter, B. (2011). Are you looking at me, or am I? Anger, aggression, shame and self-worth in violent individuals. *Journal of Rational Emotive Cognitive Behavioral Therapy*, *29*(1), 77–91. doi: 10.1007/s10942-0090105-1

Shaver, P. R., Schwartz, J., Kirson, D., & O'Connor, C. (1987). Emotion knowledge: Further exploration of a prototype approach. *Journal of Personality and Social Psychology*, *52*, 1061–1086. doi: 10.1037/0022-3514.52.6.1061

Shen, L., & Bigsby, E. (2010). Behavioral activation/inhibition systems and emotions: A test of valence vs. action tendency hypotheses. *Communication Monographs*, *77*, 1–26. . doi: 10.1080/03637750903514268

Siegler, R. S., & Jenkins, E. (1989). *How children discover new strategies*. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Siegler, R. S., & Stern, E. (1998). Conscious and unconscious strategy discoveries: A micorgenetic analysis. *Journal of Experimental Psychology: General*, *127*, 337–397.

Silk, J. S., Davis, S. S., McMakin, D. L., Dahl, R. E., & Forbes, E. E. (2012). Why do anxious children become depressed teenagers? The role of social evaluative threat and reward processing. *Psychological Medicine*, *42*, 2095–2107. doi: 10.1017/S0033291712000207

Smith, L. B. (2005). Cognition as a dynamic system: Principles from embodiment. *Developmental Review*, *25*, 278–298. doi: 10.1016/j.dr.2005.11.001

Sneed, J. R., Hamagami, F., McArdle, J. J., Cohen, P., & Chen, H. (2007). The dynamic interdependence of developmental domains across emerging adulthood. *Journal of Youth and Adolescence*, *36*, 351–362. doi: 10.1007/s10964-006-9081-2

Sommerfeld, P., & Hollenstein, L. (2011). Searching for appropriate ways to face the challenges of complexity and dynamics. *British Journal of Social Work*, *41*(4), 668–688. doi: 10.1093/bjsw/bcr057

Sonuga-Barke, E., Bitsakou, P., & Thompson, M. (2010). Beyond the dual pathway model: Evidence for the dissociation of timing, inhibitory, and delay-related impairments in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *49*, 345–355. doi: 10.1097/00004583-201004000-00009

Sonuga-Barke, E. S., Wiersema, J. R., van der Meere, J. J., & Roeyers, H. (2010). Context-dependent dynamic processes in attention deficit/hyperactivity disorder: Differentiating common and unique effects of state regulation deficits and delay aversion. *Neuropsychology Review*, *20*, 86–102. doi: 10.1007/s11065-009-9115-0

Sonuga-Barke, E. S., Sergeant, J. A., Nigg, J., & Willcutt, E. (2008). Executive dysfunction and delay aversion in attention deficit hyperactivity disorder: Nosologic and diagnostic implications. *Child and Adolescent Psychiatric Clinics of North America*, *17*(2), 367–384. doi: 10.1016/j.chc.2007.11.008

Spencer, J. P., Austin, A., & Schutte, A. R. (2012). Contributions of dynamic systems theory to cognitive development. *Cognitive Development*, *27*(4), 410–418. doi: http://dx.doi.org/10.1016/j.cogdev.2012.07.006

Spencer, T. J., Biederman, J., & Mick, E. (2007). Attention-deficit/hyperactivity disorder: Diagnosis, lifespan, comorbidities, and neurobiology. *Journal of Pediatric Psychology*, *32*, 631–642. doi: 10.1093/jpepsy/jsm005

Sportel, B., Nauta, M. H., de Hullu, E., & de Jong, P. J. (2013). Predicting internalizing symptoms over a two year period by BIS, FFFS and attentional control. *Personality and Individual Differences*, *54*, 236–240. doi: 10.1016/j.paid.2012.08.043

Sroufe, L. (2007). The place of development in developmental psychopathology. In A. S. Masten (Ed.), *Multilevel dynamics in developmental psychopathology: Pathways to the future* (pp. 285–299). New York, NY: Taylor & Francis Group/Lawrence Erlbaum Associates.

Steenbeek, H., Jansen, L., & van Geert, P. (2012). Scaffolding dynamics and the emergence of problematic learning trajectories. *Learning and Individual Differences*, *22*, 64–75. doi: 10.1016/j.lindif.2011.11.014

Steenbeek, H., & van Geert, P. (2013). The emergence of learning-teaching trajectories in education: a complex dynamic systems approach. *Nonlinear Dynamics, Psychology, and Life Sciences*, *17*(2), 233–267.

Steenbeek, H., & Van Geert, P. (2008). An empirical validation of a dynamic systems model of interaction: do children of different sociometric statuses differ in their dyadic play? *Developmental Science*, *11*, 253–281. doi: 10.1111/j.1467-7687.2007.00655.x

Steenbeek, H. W., & van Geert, P. L. C. (2007). A theory and dynamic model of dyadic interaction: Concerns, appraisals, and contagiousness in a developmental context. *Developmental Review*, *27*(1), 1–40. doi: 10.1016/j.dr.2006.06.002

Stein, Z., Dawson, T., & Fischer, K. W. (2010). Redesigning testing: Operationalizing the new science of learning. In M. S. Khine & I. M. Saleh (Eds.), *New science of learning: Cognition, computers, and collaboration in education* (pp. 207–224). New York, NY: Springer.

Stone, C. (1998). The metaphor of scaffolding: Its utility for the field of learning disabilities. *Journal of Learning Disabilities*, *31*, 344–364. doi: 10.1177/002221949803100404

Sullivan, H. S. (1953). *The interpersonal theory of psychiatry*. New York, NY: Norton.

Sutherland, K. S., Alder, N., & Gunter, P. L. (2003). The effect of increased rates of opportunities to respond on the classroom behavior of students with emotional/behavioral disorders. *Journal of Emotional and Behavioral Disorders*, *11*(4), 239–248.

Tangney, J. P., Wagner, P., Fletcher, C., & Gramzow, R. (1992). Shamed into anger? The relation of shame and guilt to anger and self-reported aggression. *Journal of Personality and Social Psychology*, *62*(4), 669–675. doi: 10.1037/0022-3514.62.4.669

Tavory, I., Jablonka, E., & Ginsburg, S. (2012). Culture and Epigenetics.

Terr, L. C. (1991). Childhood traumas: An outline and overview. *American Journal of Psychiatry*, *148*, 10–20.

Thelen, E., & Smith, L. B. (2006). Dynamic systems theories. In R. M. Lerner & W. Damon (Eds.), *Handbook of child psychology: Theoretical models of human development* (6th ed., Vol. 1, pp. 258–312). Hoboken, NJ: John Wiley & Sons Inc.

Thomas, K. M., Hopwood, C. J., Woody, E., Ethier, N., & Sadler, P. (2013). Momentary assessment of interpersonal process in psychotherapy. *Journal of Counseling Psychology*, *1-14*, 61(1). doi: 10.1037/a0034277

Thornton, L. C., Frick, P. J., Crapanzano, A., & Terranova, A. M. (2013). The incremental utility of callous-unemotional traits and conduct problems in predicting aggression and bullying in a community sample of boys and girls. *Psychological Assessment*, *25*, 366–378. doi: 10.1037/a0031153

Tomasi, D. , & Volkow, N. D. (2012). Abnormal functional connectivity in children with attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *71*, 443–450. doi: 10.1016/j.biopsych.2011.11.003

Trickett, E., & Schensul, J. (2009). Summary comments: Multi-level community based culturally situated interventions. *American Journal of Community Psychology*, *43*(3–4), 377–381. doi: 10.1007/s10464-009-9237-9

Tseng, V., & Seidman, E. (2007). A systems framework for understanding social settings. *American Journal of Community Psychology*, *39*(3–4), 217–228. doi: 10.1007/s10464-007-9101-8

Ursache, A., Blair, C., Stifter, C., & Voegtline, K. (2013). Emotional reactivity and regulation in infancy interact to predict executive functioning in early childhood. *Developmental Psychology*, *49*, 127–137. doi: 10.1037/a0027728

Vaillant, G. E. (1977). *Adaptation to life*. Boston, MA: Little, Brown.

Valentino, K., Cicchetti, D., Rogosch, F. A., & Toth, S. L. (2008). True and false recall and dissociation among maltreated children: The role of self-schema. *Development and Psychopathology*, *20*, 213–232. doi: 10.1017/S0954579408000102

van Derbur Atler, M. (1991). Address presented at the Kempe National Center for Prevention and Treatment of Child Abuse and Neglect, Denver, CO.

van der Aalsvoort, G., van Geert, P., & Steenbeek, H. (2008). Possibilities and drawbacks of microgenetic methodology related to research on learning and instruction. In: Kumpulainen, K. (Ed.). *Investigating Classroom interaction: Methodologies in action*. pp. 245–278. Sense publishers. Rotterdam.

van der Meere, J., & Stemerdink, N. (1999). The development of state regulation in normal children: An indirect comparison with children with ADHD. *Developmental Neuropsychology*, *16*, 213–225.

van Geert, P. (2009). Nonlinear complex dynamical systems in developmental psychology. In S. J. Guastello, M. Koopmans, & D. Pincus (Eds.), *Chaos and complexity in psychology: The theory of nonlinear dynamical systems* (pp. 242–281). New York, NY: Cambridge University Press.

van Geert, P. (2004). A dynamic systems approach to diagnostic measurement of SLI. In L. Verhoeven & H. van Balkom (Eds.), *Classification of developmental language disorders: Theoretical issues and clinical implications* (pp. 327–348). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Van Geert, P. (1994). *Dynamic systems of development: Change between complexity and chaos*. New York, NY: Harvester Wheatsheaf.

Van Geert, P. (1986). The concept of development. *Advances in Psychology*, *36*, 3–50.

van Geert, P. P., & Steenbeek, H. H. (2005). The dynamics of scaffolding. *New Ideas in Psychology*, *23*(3), 115–128. doi: 10.1016/j.newideapsych.2006.05.003

van Rijn, P. W., & Molenaar, P. M. (2005). Logistic models for single-subject time series. In L. van der Ark, M. A. Croon, & K. Sijtsma (Eds.), *New developments in categorial data analysis for the social and behavioral sciences* (pp. 125–145). Mahwah, NJ: Lawrence Erlbaum Associates Publishers. doi: 10.1080/08856250802596717

Vinogradov, S., Fisher, M., & de Villers-Sidani, E. (2012). Cognitive training for impaired neural systems in neuropsychiatric illness. *Neuropsychopharmacology*, *37*, 43–76. doi: 10.1038/npp.2011.251

Visser, M., Kunnen, S. E., & Van Geert, P. L. (2010). The impact of context on the development of aggressive behavior in special elementary school children. *Mind, Brain, and Education*, *4*(1), 34–43

Visser, M., Singer, E., van Geert, P. L., & Kunnen, S. E. (2009). What makes children behave aggressively? The inner logic of Dutch children in special education. *European Journal of Special Needs Education*, *24*(1), 1–20. doi: 10.1080/08856250802596717

Vitaro, F., & Brendgen, M. (2005). Proactive and reactive aggression: A Developmental perspective. In R. E. Tremblay, W. W. Hartup, & J. Archer (Eds.), *Developmental origins of aggression* (pp. 178–201). New York, NY: Guilford Press.

von Eye, A., & Bergman, L. R. (2003). Research strategies in developmental psychopathology: Dimensional identity and the person-oriented approach. *Development and Psychopathology*, *15*, 553–580.

Vondra, J. I., Barnett, D., & Cicchetti, D. (1989). Perceived competence and actual competence among maltreated and comparison school children. *Development and Psychopathology*, *1*, 237–255. doi: 10.1017/S0954579400000432

Wakschlag, L. S., Tolan, P. H., & Leventhal, B. L. (2010). Research review: 'Ain't misbehavin': Towards a developmentally-specified nosology for preschool disruptive behavior. *Journal of Child Psychology and Psychiatry*, *51*, 3–22. doi: 10.1111/j.1469-7610.2009.02184.x

Wallbott, H. G., & Scherer, K. R. (1993). Cultural differences in experiencing shame and guilt. In J. P. Tangney & K. W. Fischer (Ed.), *The self-conscious emotions: Pride, shame, and guilt* (pp. 465–487). New York, NY: Guilford.

Warren, S. L., Huston, L., Egeland, B., & Sroufe, L. (1997). Child and adolescent anxiety disorders and early attachment. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 637–644. doi: 10.1097/00004583-199705000-00014

Waschbusch, D. A., Porter, S., Carrey, N., Kazmi, S., Roach, K. A., & D'Amico, D. A. (2004). Investigation of the heterogeneity of disruptive behaviour in elementary-age children. *Canadian Journal of Behavioural Science*, *36*, 97–11. doi: 10.1037/h0087221

Watson, M. W., Fischer, K. W., Andreas, J., & Smith, K. W. (2004). Pathways to aggression in children and adolescents. *Harvard Educational Review*, *74*, 404–430.

Watson, M. W., & Getz, K. (1991). The relationship between oedipal behaviors and children's family role concepts. *Merrill-Palmer Quarterly*, *36*, 487–505.

Weaver, I. C., Champagne, F. A., Brown, S. E., Dymov, S., Sharma, S., Meaney, M. J., Szyf, M. (2005). Reversal of maternal programming of stress responses in adult offspring through methyl supplementation: altering epigenetic marking later in life. *Journal of Neuroscience*, *25*(47), 11045–11054.

Wehby, J. H., Lane, K. L., & Falk, K. B. (2003). Academic instruction for students with emotional and behavioral disorders. *Journal of Emotional and Behavioral Disorders*, *11*, 194–197.

Westen, D., & Cohen, R. (1992). The self in borderline personality disorder: A psychodynamic perspective. In Z. Segal & S. Blatt (Eds.), *Self-representation and emotional disorder: Cognitive and psychodynamic perspectives* (pp. 334–368). New York, NY: Guilford Press.

Westen, D., Malone, J. C., & DeFife, J. A. (2012). An empirically derived approach to theclassification and diagnosis of mood disorders. *World Psychiatry*, *11*, 172–180.

White, B. A., & Kistner, J. A. (2011). Biased self-perceptions, peer rejection, and aggression in children. *Journal of Abnormal Child Psychology*, *39*, 645–656. doi: 10.1007/s10802-011-9506-6

Wichstrøm, L., Berg-Nielsen, T., Angold, A., Egger, H., Solheim, E., & Sveen, T. (2012). Prevalence of psychiatric disorders in preschoolers. *Journal of Child Psychology and Psychiatry*, *53*, 695–705. doi: 10.1111/j.1469-7610.2011.02514.x

Wilson, L. C., & Scarpa, A. (2011). The link between sensation seeking and aggression: A meta-analytic review. *Aggressive Behavior*, *37*, 81–90. doi: 10.1002/ab.20369

Winkielman, P., Niedenthal, P. M., & Oberman, L. M. (2009). Embodied perspective on emotion-cognition interactions. In J. A. Pineda (Ed.), *Mirror neuron systems: The role of mirroring processes in social cognition* (pp. 235–257). Totowa, NJ: Humana Press.

Winsler, A. (1998). Parent–child interaction and private speech in boys with ADHD. *Applied Developmental Science*, *2*, 17–39. doi: 10.1207/s1532480xads0201_2

Witherington, D. C. (2011). Taking emergence seriously: The centrality of circular causality for dynamic systems approaches to development. *Human Development*, *54*, 66–92. doi: 10.1159/000326814

Witherington, D. C. (2007). The dynamic systems approach as metatheory for developmental psychology. *Human Development*, *50*, 127–153. doi: 10.1159/000100943

Witherington, D. C., & Crichton, J. A. (2007). *Emotion*, *7*, 628–637. doi: 10.1037/1528-3542.7.3.628

Witherington, D. C., & Margett, T. E. (2009). Systems and dynamic systems: The search for inclusive merger. *Human Development*, *52*, 251–256, doi: 10.1159/000215075

Wolf, C., & Linden, D. E. J. (2012). Biological pathways to adaptability – interactions between genome, epigenome, nervous system and environment for adaptive behavior. *Genes, Brain and Behavior*, *11*: 3–28. doi: 10.1111/j.1601-183X.2011.00752.x

Wood, D., Bruner, J. S., & Ross, G. (1976). The role of tutoring in problem solving. *Journal of Child Psychology and Psychiatry*, *17*(2), 89–100.

Wright, K., Gudjonsson, G. H., & Young, S. (2008). An investigation of the relationship between anger and offence-related shame and guilt. *Psychology, Crime & Law*, *14*, 415–423. doi: 10.1080/10683160701770369

Wundt, W. (1907). *Outlines of psychology* (Judd, C.H., Trans.). New York, NY: Stechert. (Original publication 1905).

Yoshikawa, H., & Hsueh, J. (2001). Child development and public policy: Toward a dynamic systems perspective. *Child Development*, *72*(6), 1887–1903. doi: 10.1111/1467-8624.00384

Zachar, P., & Kendler, K. S. (2010). Philosophical issues in the classification of psychopathology. In T. Millon, R. F. Krueger, & E. Simonsen (Eds.), *Contemporary directions in psychopathology: Scientific foundations of the DSM-V and ICD-11* (pp. 127–148). New York, NY: Guilford Press.

CHAPTER 16

A Survey of Dynamic Systems Methods for Developmental Psychopathology

ISABELA GRANIC, TOM HOLLENSTEIN, and ANNA LICHTWARCK-ASCHOFF

INTRODUCTION 717
THE DEVELOPMENTALIST'S DILEMMA 718
PRINCIPLES OF DYNAMIC SYSTEMS 719
State Space, Attractors, and Dynamic Stability 720
Interrelations Between Time Scales 720
Hierarchically Embedded Levels of Organization 721
Perturbations, Phase Transitions, and Nonlinear Change 721
RESEARCH DESIGN STRATEGIES INFORMED BY DS PRINCIPLES 723
SUITABLE DATA FOR DS ANALYSES 725
DS METHODS 727
Real-Time Measures 727
Event History Analysis 731
Developmental-Time Measures 738
Latent Class Analysis 740
STATE SPACE GRID ANALYSIS: A GRAPHICAL AND STATISTICAL MIDDLE ROAD 746
State Space Grids 746
State Space Grid Analysis: Within Grids 748
State Space Grid Analysis: Between-Grid Analysis 750
FUTURE DIRECTIONS: IMPLICATIONS FOR CLINICAL RESEARCH 750
CONCLUSION 752
REFERENCES 752

INTRODUCTION

Decades ago, Lewin (1931) criticized psychology for its overreliance on methodologies that were originally designed for studying closed, physical systems. These methods, he warned, were inappropriate for the study of complex, developmental processes. Over 70 years later, the same criticisms continue to be raised with increasing urgency. Specifically, leading theorists (Cicchetti & Cohen, 1995b; Ford & Lerner, 1992; Hinde, 1992; Kagan, 1992; Keating, 1990; Overton & Horowitz, 1991; Richters, 1997) suggest that there is a fundamental incompatibility between developmentalists' organismic, open systems models and the mechanistic research methods with which these models are tested. Developmental psychopathologists have been particularly concerned with their inherited mechanistic paradigm. Many of them have developed heuristically rich, complex models based on open systems concepts, but little headway has been made in finding alternative analytic tools appropriate for testing these models—a predicament Richters (1997) dubbed *the developmentalist's dilemma*.

In this chapter, we suggest that methods derived from dynamic systems (DS) theory may be useful for addressing this dilemma. There are three main objectives: (1) to outline key dynamic systems principles and highlight their commensurability with developmental psychopathologists' core conceptual concerns; (2) to provide a survey of research designs, methodological techniques, and measurement strategies currently being used and refined by developmental DS researchers; and (3) to elaborate on one specific DS method, state space grid analysis and provide several empirical examples using this method. The state space grid approach (Lewis, Lamey, & Douglas, 1999) was developed as a middle road between DS methods that are mathematically demanding on the one hand (and, thus, often inaccessible or inappropriate for developmental psychopathology) and purely descriptive on the other. We have included most DS methods currently available for developmentalists. The methodological techniques and specific examples we have selected were chosen because

An earlier, less detailed version of this chapter appeared in *Development and Psychopathology, 15* (2003). We gratefully acknowledge the financial support of Grant 1 R21 MH 67357 from the National Institute of Mental Health (to D. Pepler and I. Granic) and Grant 410-2003-1335 from the Social Sciences and Humanities Research Council of Canada (to the first author).

Color versions of Figures 16.7 and 16.14 are available at http://onlinelibrary.wiley.com/book/10.1002/9781119125556

they seemed most appropriate for addressing the types of research questions developmental psychopathologists tend to pursue. In the end, however, our main purpose is to provide a clear enough picture of various DS methods to inspire developmental psychopathologists to expand their analytic repertoire and, thus, test their systems-based models more directly.

THE DEVELOPMENTALIST'S DILEMMA

Before proceeding to discuss DS theory and its methodological bag of tricks, we will review briefly the strong rationale for developing new methods for the field of developmental psychopathology (also see Cicchetti & Cohen, 1995b). Developmental psychopathologists have adopted an organismic, holistic, transactional framework for conceptualizing individual differences in normal and atypical development (e.g., Cicchetti, 1993; Cicchetti & Cohen, 1995a, 1995b; Cummings, Davies, & Campbell, 2000; Garmezy & Rutter, 1983; Sameroff, 1983, 1995; Sroufe & Rutter, 1984). These scholars often frame their models in terms of organizational principles and systems language. The systems theories that inform these models include General Systems Theory (Sameroff, 1983, 1995; von Bertalanffy, 1968); Developmental Systems Theory (Ford & Lerner, 1992); the ecological framework (Bronfenbrenner, 1979); contextualism (Dixon & Lerner, 1988); the transactional perspective (Dumas, LaFreniere, & Serketich, 1995); the organizational approach (Cicchetti & Schneider-Rosen, 1986; Erickson, Egeland, & Pianta, 1989; Garmezy, 1974; Sroufe, 1979, 1986; Sroufe & Rutter, 1984); the holistic-interactionistic view (Bergman & Magnusson, 1997); and the epigenetic view (Gottlieb, 1991, 1992). As a class of models, these approaches focus on process-level accounts of human behavior and on the context dependence and heterogeneity of developmental phenomena. They are concerned with the equi- and multifinality of development, the hierarchically embedded nature of intrapersonal (e.g., neurochemical activity, cognitive and emotional processes), interpersonal (e.g., parent–child relationships; peer networks), and higher order social systems (e.g., communities, cultures). They are also fundamentally concerned with the mechanisms that underlie change and novelty (as well as stability) in normal and clinically significant trajectories.

As a result of inadequate measurement techniques, however, the complex developmental models informed by the language of systems thinking remain largely untested (Richters, 1997). For example, in the field of childhood aggression, a number of leading scholars have become concerned with highlighting the heterogeneity of aggressive youth and advocating the development of causal models that recognize the equifinality of aggression (e.g., Cicchetti & Richters, 1993; Hinshaw & Zupan, 1997; Moffitt, 1993). But it remains difficult to test these models because most of our current research methods and analytic techniques (e.g., regression analysis, t-tests, path analysis) rely on strategies that aggregate overtly similar subjects into one group or another (e.g., aggressive and nonaggressive children) to conduct group-level statistical analyses. Thus, although we may know that aggressive children show the same behavioral pattern for very different reasons (e.g., abuse, permissive parenting, marital conflict, birth of a new sibling), this variability cannot be systematically addressed because multivariate analytic strategies carry an a priori assumption of within-group homogeneity. This is not just a niggling statistical detail. Several leading methodologists have argued that in most cases, these assumptions are completely unfounded and have likely led to serious misinterpretations of data (e.g., Hinshaw, 1999; Lykken, 1991; Meehl, 1978; Richters, 1997).

How did this gap between methods and models come about? Numerous past critics (e.g., Hinde, 1992; Meehl, 1978; Lykken, 1991; Overton & Horowitz, 1991; Richters, 1997) have pointed to psychology's original sin for an explanation: in an effort to gain credibility and align itself with the hard sciences, psychology appropriated the methods and analytic techniques of mechanistic, nineteenth-century physical sciences. This paradigm is inappropriate for the study of self-organizing, active, reactive, interactive, and adaptive organisms (i.e., the stuff of psychology). The irony is that psychology's embrace of this mechanistic paradigm came at the same time as the physical and biological sciences were advancing a radically new one—one based on open systems concepts (Overton & Horowitz, 1991; Richters, 1997).

For some domains of psychology, adopting techniques from statistical mechanics may not be as paralyzing as it has become for developmental psychology (Thelen & Smith, 1994, 1998; Thelen & Ulrich, 1991; van Geert, 1998a, 1998c). At the heart of developmental questions, however, is how things *change*. By what process do novel structures (e.g., formal operational thought) or skills (e.g., emotion regulation, walking, language) emerge? The pioneers of developmental science (i.e., Piaget, Vygotsky, Werner) concerned themselves with the pursuit of abstract laws or properties that govern development: the structural explanation of how development unfolds (Cicchetti, 1990; van Geert, 1998a, 1998c). As van Geert (1998a) argued, however, change and the emergence of novelty may no longer be the focus of contemporary developmental psychology. This state of affairs can be traced to the

"adoption of a statistics that was designed for different purposes, namely distinguishing populations characterized by some special feature . . . and estimating the linear association between the variance of some independent variable on the one hand and a dependent variable on the other" (p. 146). The adoption of such statistics seems to have missed the point of the original questions laid out by the founding scholars of developmental science (Thelen & Smith, 1994; van Geert, 1998a).

Developmental psychopathology, having grown in part from this tradition, inherited the same schism. But developmental psychopathology also grew from several other disciplines that were less hampered by this statistical baggage. Embryology (e.g., Kuo, 1967; Spemann, 1938) was one of these disciplines (for a review, see Cicchetti, 1990). In the pursuit of understanding normal embryological functioning, "early embryologists derived the principles of a dynamically active organism and of a hierarchically integrated system that were later used in investigations of the processes of abnormal development within the neurosciences, embryology, and experimental psychopathology" (Cicchetti, 1990, p. 4). These insights formed the basis for the organizational approach in developmental psychopathology. Another avenue of influence comes from the discipline of ethology, which views the organism as a comprehensive whole and advocates a "multidomain, multidiscipline approach to psychopathology," (Cicchetti, 1990, p. 19), an approach that is at the heart of developmental psychopathology (see also Hinde, 1983).

Interestingly, developmental psychopathology also shares its roots with the founders of child psychoanalysis (e.g., A. Freud, Klein, Winnicott, Bowlby, Erikson) who, like Piaget and Vygotsky, formalized theories based on detailed observations of children in their natural environments (Cicchetti, 1990; Cicchetti & Cohen, 1995b). These original, individual-based, ethnographic methods, however, are rarely applied in contemporary research (but see Cicchetti & Aber, 1998, for an exception). Yet it seems clear to us that these organismic, systems-oriented roots lend themselves well to DS principles and the methods they suggest.

As it stands now, the field of developmental psychopathology seems to be at an impasse. On one hand, some scholars have suggested that systems approaches to studying development have provided an interesting metaphor, but offer little more (Cox & Paley, 1997; Reis, Collins, & Bersheid, 2000; Vetere & Gale, 1987). Thus, one option is to give up the grail, abandon this well-intentioned enterprise, and build simpler, more linear models that can be tested with established statistical rigor. Another option is offered by Richters (1997): "Resolving the developmentalist's dilemma will require more than a recognition of the inadequacies of the existing paradigm. It will require intensive efforts to develop indigenous research strategies, methods, and standards with fidelity to the complexity of developmental phenomena" (p. 226). In the closing of his essay, Richters offered some general instructions for how this new generation of studies should proceed: (1) There should be intense focus placed on understanding individuals and the causal structures that underpin specific individuals' development, with particular attention paid to well-characterized exemplars (i.e., nonextreme) cases; (2) no single method should be held as superior or inferior (e.g., case based, variable based, cross sectional, longitudinal, historical, ethnographic), but instead methodological pluralism should be encouraged and may vary in degree depending on the phenomena investigated; (3) ritualized hypothesis testing should be generally abandoned for more exploratory, creative approaches that emphasize the discovery process; and (4) a narrow focus on explained variance and prediction should take a secondary role to explanatory power. These directions provide the springboard from which to consider the potential contributions dynamic systems methods can make to addressing systems-inspired models.

PRINCIPLES OF DYNAMIC SYSTEMS

Developmental psychopathologists will be familiar with most of the concepts in DS theory because of their long-standing familiarity with systems concepts in general. Nevertheless, for the sake of clarity and precision, we believe it is important to delineate this framework from the systems approaches mentioned previously (Lewis & Granic, 2000). Formally, a *dynamical system* is a set of mathematical equations that specify how a system changes over time. The various patterns and processes that emerge from this set of equations rely on a technical language originally developed in the fields of mathematics and physics. The concepts derived from this mathematical framework comprise the principles of dynamic systems approaches. The terms that are most commonly associated with this framework include attractors, repellors, state space, perturbations, bifurcations, catastrophe, chaos, hysteresis, complexity, nonlinearity, far from equilibrium, and so on. Thus, what we refer to as *dynamic systems* theory or *dynamic systems* principles is a *meta-theoretical* framework (Witherington, 2007) that encompasses a set of abstract principles that have been applied in different disciplines (e.g., physics, chemistry, biology, psychology)

and to various phenomena (e.g., lasers, ant colonies, brain dynamics) at vastly different scales of analysis (from cells to economic trends and from milliseconds to millennia).

Consistent with developmental DS theorists (e.g., Fogel, 1993; Lewis, 2000; Thelen & Smith, 1994, 1998), we use the term *dynamic systems* to refer to the systems themselves (not the equations) that change over time. DS principles provide a framework for describing how novel forms emerge and stabilize through a system's own internal feedback processes (Prigogine & Stengers, 1984). This process is known as *self-organization* and refers to the spontaneously generated (i.e., emergent) order in complex, adaptive systems. In fields as various as physics (e.g., Haken, 1977), chemistry (e.g., Prigogine & Stengers, 1984), biology (e.g., Kauffman, 1993), and neuroscience (Freeman, 1995), DS principles have proven essential for providing process-level accounts of the structure and organization of system behavior, and changes in that structure over time (Lewis & Granic, 2000).

DS principles resonate with most systems concepts in general. Dynamic systems approaches to development emphasize the multiple reciprocal interactions among system elements that are hierarchically nested and mutually influential. The context or ecology in which the system is embedded is critical for understanding a dynamic system's behavior (Hollenstein, 2012). Also consistent with various systems perspectives, development is conceptualized as movement toward greater levels of complexity through the interplay between positive and negative feedback cycles (Lewis & Granic, 1999b).

As will become clear from our selection of methods, we are most strongly influenced by the pioneering work of Esther Thelen, Linda Smith, Alan Fogel, Marc Lewis, and Paul van Geert—developmental psychologists who brought DS principles to the attention of the field at large. Because our focus is on methods specifically, a thorough theoretical discussion of DS concepts and their relevance to developmental science is precluded; thus, the reader is strongly encouraged to supplement the current theoretical introduction with reviews by Thelen & Smith (1994, 1998), Fogel (1993), and Lewis (2000; 2011). In the following discussion, we highlight some key principles and then move on to their methodological implications.

State Space, Attractors, and Dynamic Stability

Dynamic, self-organizing systems share several key properties, some of which have already been mentioned. One key feature of open systems is that, although theoretically they have the potential to exhibit an enormous number of behavioral patterns, they tend to stabilize in a limited range of these possibilities. Stable patterns emerge through feedback among many lower order (more basic) system elements; these emergent patterns are referred to as *attractors* in DS terminology. In real time, attractors may be understood as absorbing states that "pull" the system from other potential states. Behavior moves in a trajectory across the state space toward these attractors. Over developmental time, attractors represent recurrent patterns that have stabilized and are increasingly predictable. As noted by Thelen and Smith (1994), all developmental acquisitions can be described as attractor patterns that emerge over weeks, months, or years.

As recurring stable forms, attractors are often represented topographically as valleys on a dynamic landscape (Figure 16.1). The deeper and wider the attractor, the more likely it is that behavior falls into it and remains there, and the more resistant it is to small changes in the environment. *Repellors*, or states that the system tends to avoid or be repelled from, are represented as hills on this landscape—states in which the system cannot rest. As the system develops, a unique *state space*, defined as a model of all possible states a system can attain, is configured by several attractors and repellors. Critically, living systems are characterized by *multistability* (Kelso, 1995); that is, their state space (i.e., behavioral repertoire) includes several coexisting attractors. Contextual constraints probabilistically guide behavior toward a particular attractor at any given moment in time.

As we will see later, the concepts of state space, attractors and multistability have informed several research designs and methodologies in recent years. The operationalization of these principles, either graphically, mathematically, or heuristically, have helped DS researchers uncover previously undetected behavioral variability, as well as the processes by which this variability stabilizes into unpredicted, but nevertheless stable, attractor patterns.

Interrelations Between Time Scales

DS researchers are always fundamentally concerned with the interplay between different time scales. From a DS

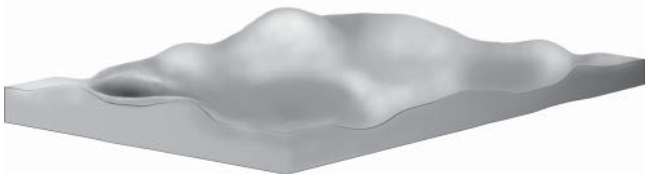


Figure 16.1 A state space with three attractors (the wells) and one repellor (the hill). *Source:* Hollenstein T. (2007). State space grids: Analyzing dynamics across development. *International Journal of Behavioral Development*, *31*, 384–396.

perspective, the same principles of change and stability can be applied at the moment-to-moment scale (real time) as they can to developmental time (weeks, months, years). The interplay between nested time scales is constant and reciprocal (Thelen & Smith, 1994, 1998). Self-organization at the real-time scale constrains self-organization at the developmental scale, which in turn constrains real-time behavior (van Gelder & Port, 1995). Thelen and Smith (1998) elaborated:

> . . . each behavioral act occurs *over time* . . . but every act changes the overall system and builds a history of acts over time . . . Habituation, memory, learning, adaptation, and development form one seamless web built on processes over time—activities in the real world. (p. 593)

Research designs based on DS principles almost always measure behavior on at least two time scales (Hollenstein, 2012; Hollenstein, Lichtwarck-Aschoff, & Potworowski, 2013). The manner and extent to which the two or more levels of analysis are related to each other is subsequently examined. Thus, DS-informed studies often involve collecting real-time, observational data over repeated sessions in a longitudinal design such that moment-to-moment behavioral patterns, and changes in those patterns, can be traced along a longitudinal trajectory (Lee & Karmiloff-Smith, 2002).

Hierarchically Embedded Levels of Organization

Dynamic systems are not only nested processes in time, they are also coupled hierarchically. Mechanisms in development may be defined at different levels of organization (e.g., physiological, emotional, behavioral, social) and these levels are thought to be embedded, linked by feedback processes, and mutually constraining. Importantly, DS theorists insist that no level of organization is any more "basic" or "primary" in terms of causality. Thus, for example, from a DS perspective, neural processes are accorded no higher causal privilege than psychological or social processes. Instead, "descriptions of change of many components are needed so that multilevel processes and their mutual interactions can be fully integrated . . . Moreover, explanations at every level must be consistent and ultimately reconcilable (Thelen & Smith, 1998, pp. 596–597). This view of causality contrasts with most approaches in psychology (but see Cicchetti & Dawson, 2002a).

Rather than simple linear relationships, DS researchers often understand relations among multiple levels in terms of *circular causality* (Hakens, 1977). Circular causality suggests that interactions among lower order elements provide the means by which higher order patterns emerge and these emergent attractors exert top-down influences to maintain the entrainment of lower level components. Developmental psychopathologists, particularly those in the neurosciences, have recently become interested in understanding how different levels of analysis can shed light on typical and atypical development (Calkins & Fox, 2002; Cicchetti & Dawson, 2002b; Nelson et al., 2002). The DS meta-theoretical framework, with concepts such as circular causality, may help researchers organize their findings and parsimoniously integrate the various theories about different levels into one coherent model. Indeed, it is likely that common dynamic principles of self-organization govern different levels of organization (e.g., neural, cardiac, emotional, behavioral, societal); if this is so, then synergistic DS methods that can be applied similarly at different levels of analysis will provide a powerful means by which integrative models can be developed and empirically supported. The extent to which real- and developmental-time scales are interrelated and hierarchical levels of organization are embedded is further clarified when considering perturbations and their relation to phase transitions.

Perturbations, Phase Transitions, and Nonlinear Change

Through the amplification properties of positive feedback, nonlinear changes in the organizational structure of a dynamic system can be observed. As shown in Figure 16.2, these abrupt changes are referred to as phase transitions and they occur at points of bifurcation, or junctures in the

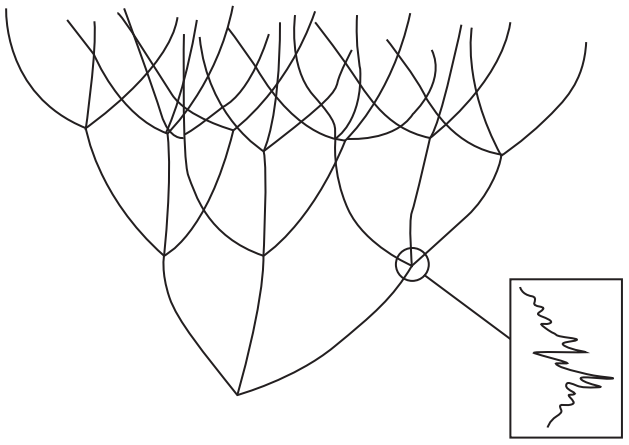


Figure 16.2 Alternative developmental trajectories. Phase transitions occur at regular junctures in development. Increased variability at phase transitions is shown in magnified segment. *Source:* From Lewis, M. D. & Douglas, L. (1998) A dynamic systems approach to cognition-emotion interactions in development (pp. 159–188), in *What Develops in Emotional Development?* M. F. Mascolo & S. Griffins (Eds.). New York: Plenum Press.

system's development. At these thresholds, small fluctuations have the potential to disproportionately affect the interactions of other elements leading to the emergence of new forms. Novelty does not have to originate from outside the system; it can emerge spontaneously through feedback within the system. During a phase transition, systems are extremely sensitive to perturbations. Between these points, however, self-organizing systems tend toward coherence and stability.

Phase transitions are characterized by interrelated changes in real and developmental time. In developmental time, periods of stability and relative predictability are followed by a period of disequilibrium in which established patterns are destabilized. After this period of flux, developmental systems restabilize and settle into new habits of interactions. Corresponding to this developmental profile, real-time behavior during a phase transition is more variable, flexible, and more sensitive to perturbations; behavior may change from one state to another frequently, and is less likely to settle in any one state for very long (Thelen & Ulrich, 1991). However, before and after the phase transition, real-time behavior is far less variable; only a small number of behavioral states are available to the system, and once the system settles into one of these stable patterns, it tends to remain there for an extended time period (e.g., Thelen & Smith, 1994; van Geert, 1998b; van der Maas & Molenaar, 1992).

DS researchers have used the concept of perturbations on a real-time scale as an empirical design innovation to test the relative stability of observed behavioral patterns. Perturbations have the potential to abruptly push the system from one stable pattern to another (Fogel, 1993; Thelen & Smith, 1994). But this is only a potential—whether and how a system becomes reorganized is determined by its underlying structure. A perturbation can be considered a focal aspect of environmental context, and sensitivity to perturbations (which typifies phase transitions) is a special case of the more general sensitivity of dynamic systems to their contexts—a sensitivity that changes with system development gradually or more suddenly. Thus, context-sensitivity for DS researchers is not just "a form of jargon for anything environmental, as if invoking the term suggests compliance with current scientific and conceptual canons" (Boyce et al., 1998, p. 145). DS researchers systematically observe changes in behavior, as it varies with contextual forces, to infer the underlying structure of the system (e.g., Fogel, 1993; Granic & Lamey, 2002; Lewis & Granic, 1999b, 2000; Thelen & Smith, 1994; Thelen & Ulrich, 1991).

On a developmental scale, principles of nonlinear change, phase transitions and perturbations have been most often used for the explicit purpose of studying the structural profile of developmental transitions (e.g., Fogel & Thelen, 1987; Granic, Hollenstein, Dishion, & Patterson, 2003; Lewis, 2000; Lewis & Granic, 2000; Thelen & Ulrich, 1991; van Geert, 1991, 1994). Neo-Piagetian scholars such as van der Maas and Molenaar (1992) have used a particular type of dynamic model, the cusp-catastrophe model, to represent the nonlinear nature of stage transitions. Borrowing from Gilmore (1981), they suggest a number of criteria or flags that can be used to operationalize a transition. Among the transition flags are: a sudden jump from one parameter value to another, evidence of hysteresis (i.e., when the same conditions elicit different behaviors, depending on the immediate prior history of the system), anomalous variance, and an increased sensitivity to perturbations. Transitions in motor, cognitive, linguistic, and socioemotional development have been successfully modeled by the application of variants of these flags (Case et al., 1996; Lewis et al., 1999, 2004; van der Maas & Molenaar, 1992; van Geert, 1991, 1994). Structural changes in parent–adolescent interactions at the early adolescent stage transition have also been shown to exhibit the properties of a phase transition (Granic, Dishion, & Hollenstein, 2003; Granic et al., 2003).

Many of the DS concepts described here are clearly resonant with other systems views. But as a point of distinction, we suggest that four principles central to the DS framework are either neglected or less emphasized in other approaches and hold great promise for new empirical directions in developmental psychopathology. First, DS theory is primarily concerned with the emergence of novelty through the process of self-organization, whereas, with some notable exceptions (Ford & Lerner, 1992), most of the emphasis in more general systems views is on mechanisms of stability (i.e., negative feedback processes, for examples see cybernetic models; Granic, 2000; Lewis & Granic, 1999a). Second, although systems views may acknowledge the nonlinear nature of change in developmental systems, this is the hallmark principle of DS approaches to development and it has led to the exploration of radical new strategies such as catastrophe theory (e.g., van der Maas & Molenaar, 1992), developmental growth curve modeling (van Geert, 1991, 1994), and the study of phase transitions (e.g., Thelen & Ulrich, 1991; Thelen & Smith, 1994). Third, variability represents critical information in DS research. Conventionally, variability in developmental data has been seen as the result of measurement error and, thus, a source of noise that should be minimized. DS theorists make a radical departure from this approach: variability is considered a rich source of information, indexing impending change and "the essential ground for exploration and selection" (van Geert & van Dijk, 2002, p. 342). Methods that

tap changes in variability are a mainstay of DS researchers (e.g., Thelen & Ulrich, 1991; van Geert & van Dijk, 2002). Finally, DS theorists are fundamentally concerned with the interrelations between time scales of development and put a great deal of emphasis on understanding the unfolding patterns of real-time behavior (Thelen & Smith, 1998). This final principle is critical in terms of its methodological implications, and it is most often ignored in other systems frameworks.

In the following sections, we describe a number of dynamic systems approaches to research designs and measurement strategies. To limit the scope of our review, we will not be discussing the exciting work emerging in the neurosciences (a field that has long embraced the principles of self-organization). This work is clearly relevant to developmental psychopathologists and the reader is referred to the neuroimaging methods detailed in the most recent volume of *Developmental Psychopathology,* 3rd edition. We also spend less time proportionally on the mathematical modeling techniques than the graphical, descriptive and statistical ones because we believe that the latter group of methodologies are generally more accessible and may ultimately prove more appropriate for the types of research questions put forward by developmental psychopathologists.

We first review some general DS-informed research design concepts and strategies put forth by Thelen and her colleagues (e.g., Thelen & Smith, 1994; Thelen & Ulrich, 1991). Next, we discuss the types of data most suitable for DS analyses. We follow by providing a list of graphical techniques and quantitative strategies appropriate for the analysis of real-time and developmental data; these descriptions are supplemented with actual or hypothetical examples relevant to developmental psychopathologists. We also highlight the limitations inherent in some of the techniques and argue that a newly developed DS methodology, state space grid analysis, may help address a number of these weaknesses. The last part of this paper provides a detailed description of the state space grid technique, which combines graphical methods with statistical analysis in a way that maintains fidelity to DS concepts. We provide several examples of programs of research and individual studies that have used variations of this approach.

RESEARCH DESIGN STRATEGIES INFORMED BY DS PRINCIPLES

Thelen and her colleagues have explicitly laid out a methodological strategy for developmental psychologists interested in dynamic analyses (Thelen & Smith, 1994, 1998; Thelen & Ulrich, 1991). Their strategy includes six steps:

1. *Identify the collective variable of interest*. A collective variable must be an observable phenomenon (not a construct or latent variable) that captures the coordination of the elements of a multidimensional system. Because Thelen and Ulrich (1991) were interested in motor development, they chose the phasing of alternating steps as the collective variable that condensed the many aspects of interlimb coordination. Changes in this collective variable can then be tracked over developmental time. This is the first challenging step—unlike physical systems, it is difficult to identify a collective variable in psychological systems. Extensive developmental observations and experiments are recommended as a first step toward this goal. An example relevant to developmental psychopathologists might be the observed intensity of a child's oppositional behavior—a collective variable that may capture the coordination of mood states, arousal level, appraisal processes, and so on (these processes themselves would need to be assessed in multiple contexts).
2. *Describe the attractors for that system*. This involves mapping the real-time trajectory of the collective variable in various contexts across different developmental periods and identifying its relative stability. Thus, the contexts in which a child's oppositional behavior is most stable can be identified (e.g., with a parent), as well as the contexts in which such behavior is less stable (i.e., more easily changed), is never observed, or is rarely observed. High stability indicates an attractor state.

   Attractor states may be tested by examining the variability of the collective variable given particular contexts (e.g., how often does the child become oppositional in response to a request to clean up at home versus at school). Additional features can help identify attractors as well. First, an attractor may be present if behavior takes a relatively short time to return back to the state once it has left it (latency to return to the attractor is short). Second, behavior tends to stay in an attractor for longer durations than other states. Third, it should take a much larger perturbation to move behavior out of a strong attractor state relative to weaker attractors or other regions of the state space. This last point is also a reminder that living systems exhibit multistability. The implication is that researchers should attempt to identify all or many of the attractors that are available to a particular system. Then the critical question becomes: what are the conditions under which behavior will gravitate toward one attractor versus another? Returning to

the oppositional child, most conventional approaches focus on describing and predicting the child's negative, destructive behavior. But a DS approach would map out not only the negative behavior attractors but also the interested/engaged states, the joyful and the neutral states, and, importantly, the pathways to and from these attractors. Although these latter states may be weaker attractors, the goal would be to track the contextual conditions under which the child moves from more positive to more negative attractors and vice versa to get a better picture of the underlying dynamics of this child's behavioral landscape.

3. *Map the individual developmental trajectories of the collective variable*. This step requires collecting observations at many time points in a longitudinal design (also see Fogel, 1993). The density of time samples depends on the developmental period in question (i.e., in infancy, weekly observations may be needed, whereas in late childhood data collected monthly might suffice; Fogel, 1993). Then, developmental profiles can be graphed on a case-by-case basis and the similarities and differences among profiles can be described. Stable (i.e., fixed or cycling) segments of the time series denote an attractor. At this stage, the multifinality and equifinality of developmental trajectories can be discovered. Some developmental profiles may start out looking similar and then, from very small differences that become amplified, trajectories may diverge. Other developmental profiles may show the opposite pattern of different initial conditions being "pulled" toward a particular attractor. The key at this stage is to create individual profiles rather than aggregate across subjects; otherwise, the variability inherent in developmental processes will be obscured. As described in more detail later, an alternative to averaging developmental profiles is to cluster individual trajectories into groups that share profile characteristics (Hollenstein, Granic, Stoolmiller, & Snyder, 2004). In developmental psychopathology, a similar point has been emphasized by researchers doing case-based, or person-oriented, analysis (e.g., Bergman & Magnusson, 1997).
4. *Identify phase transitions in development*. As described earlier, transitions in development are characterized by increased variability, a breakdown of stable patterns, and the emergence of new forms. The various catastrophe flags described earlier can help researchers identify points of transition. Transition periods are critical to mark because they allow researchers to access and manipulate mechanisms underlying change. This point is particularly relevant for developmental psychopathologists interested in clinical interventions. For instance, there may be normative stage transitions in children's development during which, as a result of normal maturational processes, the coordination among system elements begins to break down, previous attractors are destabilized, and there is an increased potential for new patterns to emerge (e.g., Granic, Hollenstein, Dishion, & Patterson, 2003; Lewis & Granic, 1999b; Lewis, Zimmerman, et al., 2004; Lewis et al., 1999). Clinical interventions may have their greatest impact if they are targeted at these sensitive periods.

   The idea of fluctuations at phase transitions is not new to developmental psychopathology, although it has rarely been pursued empirically. One fascinating example comes from a study conducted by Inhelder (1976), who found that some mentally retarded children's skill performance oscillated between various stage levels prior to reaching a new stage. Interpreting these findings, Cicchetti (1990) suggested two possibilities: the processes accounting for these children's development may simply be different from those of normal children, or, more intriguingly, "their oscillations are universal phenomena that become visible only in retarded children because of the slower nature of their development" (p. 16; see also Cicchetti & Sroufe, 1976).

   Bertenthal (1999) further emphasizes the importance of variability at phase transitions. He suggests that variability is not just an index of change, but actually helps *drive* change. According to Bertenthal, "variability offers flexibility, which drives development following Darwinian principles. Principles of variation and selection cause successful behaviors to be stored and repeated more frequently than the less successful" (van Geert & van Dijk, 2002, p. 343; Thelen & Smith, 1994). The theoretical implications for understanding intervention effects are compelling. Successful interventions may *induce* a phase transition during which behavioral, cognitive and affective variability increases, providing the fertile ground from which more positive, or less distressing patterns can be selected, repeated, and potentially stabilized. This possibility can be empirically verified by using simple descriptive statistics (e.g., looking for an increase in standard deviations and variance, a breakdown of correlations, and changes in entropy; additional measures of variance are described later) or more formal techniques (described in the section on state space grids).
5. *Identify control parameters*. In DS language, control parameters are the "agents of change." The purpose

of tracking the collective variable across different contexts and developmental transition points is to ultimately identify the mechanisms underlying processes of change. Control parameters are not simply independent variables (although they can be considered a special type). Usually, independent variables are static measures that are assumed to have a linear effect on outcomes. Control parameters are better understood as mediators with special properties; continuous and small changes in the values of these parameters can result in abrupt threshold effects on a collective variable. Moreover, these nonlinear changes occur at different values depending on whether the control parameter is increasing or decreasing. For example, through fine-grained longitudinal observations, Thelen and Ulrich (1991) were able to identify overall changes in muscle mass as the control parameter that was related to improvements in infants' treadmill stepping. In many areas of developmental psychopathology, however, this step is the most difficult, because psychological systems are incredibly complex and the problem of identifying and manipulating one or very few causal mechanisms is often insoluble. Moreover, a control parameter may not always be something that *can* be manipulated (e.g., temperament, parental depression). Nevertheless, DS researchers urge us to at least keep the concept in mind.

6. *Manipulate control parameters to experimentally generate phase transitions*. Despite its difficulty, this suggestion is a familiar one to many developmental psychopathologists. Simply put, once a causal factor has been inferred from careful descriptive analysis, it should be experimentally manipulated to examine whether it does indeed trigger the expected shift in behavior. In this respect, intervention studies are an exceptional avenue for testing the role of specific control parameters in developmental psychopathology (c.f. Dishion, Bullock, & Granic, 2002; Dishion & Patterson, 1999; Eddy & Chamberlain, 2000; Forgatch & DeGarmo, 1999). One of the best examples of following this proposed strategy comes from the work on the etiology and treatment of aggressive behavior. For instance, based on decades of microsocial observational studies with families, coercion has been identified as a mediating causal mechanism underlying the etiology of childhood aggression (e.g., Patterson, 1982; Patterson, Reid, & Dishion, 1992). To confirm this supposition experimentally, Forgatch and DeGarmo (1999) investigated the impact of a randomized control intervention that aimed to decrease the rate at which parents engaged in coercive interchanges. Results confirmed that, indeed, changes in coercion resulted in decreases in children's aggressive behavior. From a DS perspective, parental discipline strategies, for example, could have been the control parameter that was adjusted through the intervention (i.e., coercion decreased).

Despite the advantages of Thelen and colleagues' approach, many researchers are likely to experience some problems with implementing their general strategy. First, it requires collecting continuous time-series data (e.g., physiological data, behavioral observations coded in real time); this type of data is time-consuming and expensive to collect. More importantly, time-series data may not capture the type of information pertinent to many developmental psychopathologists. Second, and related, unlike motor or cognitive development in which some skill or task performance increases or decreases quantitatively over time, psychopathology may not involve such graded changes (Lewis & Granic, 1999b; Lewis et al., 2004). Instead, the development of psychopathology may better be characterized as emergent patterns of interconnected changes in different domains (e.g., biological, cognitive, emotional) that are nonlinearly related to one another and change qualitatively, as well as quantitatively (i.e., it is often categorical or ordinal). We will address this issue at greater length when we discuss state space grid analysis and the limitations it addresses in this regard.

SUITABLE DATA FOR DS ANALYSES

Developmental psychopathologists collect several types of data that are appropriate for DS analyses. Because a DS perspective is fundamentally about changes in time, the most important data characteristic is that there are multiple measurements over time. Thus, questionnaire data collected at one time point would be inappropriate for tapping dynamic processes (e.g., Cummings et al., 2000). Below, we list the four types of data that lend themselves most easily to DS analyses and name some examples of analytic techniques that can be conducted. These techniques are then explained in more detail in the following section. Table 16.1 lists the techniques, the appropriate data types for each, and provides some (non-exhaustive) examples of empirical papers that have applied the various DS methods.

1. *Observational data—continuous*. Includes data obtained in time units less than one second; these data are often composed of physiological measures. The density of

TABLE 16.1 Summary of DS Techniques and DS Concepts and Examples of Studies That Have Applied These Techniques

Techniques Real-time	DS concept	Examples
Case studies	Self-organization	Fogel (1990, 1993)
Time series	Attractors	Bakeman & Gottman (1997)
		Granic & Dishion (2003)
Phase plots	Phase space attractors	Sabelli, Carlson-Sabelli, Patel, Levy, & Diez-Martin (1995)
		Totterdell, Briner, Parkinson, & Reynolds (1996)
Event history analysis	Attractors	Snyder, Stoolmiller, Wilson, & Yamamoto (2003)
		Lougheed, Hollenstein, Lichtwarck-Aschoff, & Granic (2015)
Fourier analysis	Attractors	Newtson (1994,1998)
		Schroeck (1994)
Karnaugh maps	Phase transition	Dumas, Lemay, & Dauwalder (2001)
	State space	
Coupled equations	Feedback	Gottman, Guralnick, Wilson, Swanson, & Murray (1997)
	Attractors	Nowak & Vallacher (1998)
		Ryan, Gottman, Murray, Carrere, & Swanson (2000)
		Gottman, Murray, Swanson, Tyson, & Swanson (2002)
Nonlinear dynamics	Attractors	Guastello (2001)
	Phase space feedback	Heath (2000)
	Entropy	Newell & Molenaar (1998)
	Chaos & determinism	Dishion, Nelson, Bullock, & Winter (2004)
		Pincus (2001)
SSG	State space	Lewis, Lamey, & Douglas (1999)
	attractors	Hollenstein, Granic, Stoolmiller, & Snyder (2004)
	Phase transitions	Granic & Lamey (2002)
	Perturbation	Granic, O'Hara, Pepler, & Lewis (2007)
	variability	Hollenstein (2007, 2013)
		Hollenstein & Lewis (2006)
		Hollenstein, Allen, & Sheeber (in press)
Developmental		
Descriptive developmental	Phase space attractors	Thelen & Ulrich (1991)
profile	Self-organization	Thelen & Smith (1994)
	Phase transition	Van Geert & van Dijk (2002)
Latent class analysis	Attractors	van der Maas (1998)
	Self-organization	
	Phase transition	
Dynamic growth modeling	Phase space attractors	van Geert (1994, 1998)
	Self-organization	Ruhland & van Geert (1998)
	Phase transition	Thelen, Schoner, Scheier, & Smith (2001)
	feedback	Steenbeek & van Geert (2005, 2007, 2008)
Connectionist modeling	Attractors	McClelland (1979)
	Bifurcations	Rumelhart & McClelland (1986)
	Phase transition	Johnson & Morton (1991)
	Chaos	Smith (1993, 1995)
		Smith & Thelen (2003)
		Thelen et al. (2001)
Catastrophe modeling	Phase transition Hysteresis	Hartleman, van der Maas, & Molenaar (1998)
		van der Maas & Molenaar (1992)
SSG	Phase transition	Granic, Dishion, & Hollenstein (2003)
	State space	Granic, Hollenstein, Dishion, & Patterson (2003)
		Lewis, Zimmerman, Hollenstein, & Lamey (2004)
		Lewis, Lamey, & Douglas (1999)
		van der Giessen, Hollenstein, Hale, Koot, Meeus, & Branje (2015)

data points allows for the more sophisticated techniques adopted from the natural sciences, where this type of data is most common. These techniques are often based on time-series analysis and include the domain of elaborate mathematical models based on coupled equations and dynamical catastrophe models. Quantitative measures of chaos—Lyupanov exponents, entropy, correlational dimensions, and so on—can also be derived from such continuous data. Furthermore, all of the methods available to the other data types are available with this kind of data, either as time series or summaries of time series.

2. *Observational data—discrete*. Includes live and taped observational data that are converted to codes along

time units as small as one second. These codes can represent the sequence of behavior for one or more subjects and are typically inappropriate for time-series techniques unless they have a sufficiently large number of data points. By applying DS graphical techniques such as state space grids, Karnaugh maps, recurrence plots, and phase plots, the temporal patterns embedded in the temporal sequence can be uncovered. These data can be used to identify attractors, perturbations, phase transitions, and other DS patterns.

3. *Longitudinal data—short*. This type of data can include, for example, hourly/daily/weekly self-report measures (e.g., diary or beeper studies), repeated phone interviews, or repeated observational sessions (e.g., therapy sessions). The time points may be frequent enough to allow the use of some of the real-time techniques available to the first type of data or may be analyzed using techniques applicable to developmental-time data (type 4).
4. *Longitudinal data—long*. Includes any combination of the above data types collected at different time points that may span weeks, months, or years. The main distinction from data type 3 is the time span between first and last measurements. Data collected in three or more waves is often used to depict change, growth, or intervention effects and a variety of DS methods including state space grid analysis, dynamic growth modeling, developmental profile analysis, and catastrophe modeling can be applied. Developmental phase transitions are detectable through the application of these techniques.

DS METHODS

Real-Time Measures

As most teachers of research methods and statistics in general will insist, eyeballing your data is an important part of the analytic procedure. For DS researchers, graphical techniques provide the core of their analytic armament. Perhaps because dynamic systems theory is a descriptive framework and because it aims to describe phenomena in geometric terms (recall our discussion of behavioral trajectories on a state space), plotting data is the mainstay of DS researchers (Abraham, Abraham, & Shaw, 1992; Norton, 1995). A number of these real- and developmental-time graphical methods can be complemented with various quantitative tools. We have grouped the following methods under the rubric of real-time methods because that is how they have tended to be applied. But it is important to note that because the same principles of self-organization apply at different time scales, many of these techniques can be adapted easily to study phenomena in developmental time.

Case Studies

Perhaps the one common recommendation DS researchers offer is to start with fine-grained, real-time observations of the phenomenon of interest and follow this behavior across a significant developmental period. One of most basic first steps toward this end is the careful description of case studies. Fogel's (1990, 1993) research on mother–infant relationship processes is exemplary in this respect. His microgenetic research relies heavily on detailed descriptions of videotaped interactions, as they proceed in real time. He uses these case histories as a "means to seek patterns in sequences of action in a context, in both real and developmental time scales" (Fogel, 1990, p. 343). Metaphors based on DS principles serve as guides for identifying dynamically stable dyadic patterns (frames) and changes in those patterns across development. Although these narrative descriptions are rich in detail and provide ample fodder for generating hypotheses, they are intentionally not quantified at this stage. As such, this method does not address developmental psychopathologists' search for quantitative techniques to test their conceptual models. To quantify impressions from case studies and statistically test hypotheses, Fogel and his colleagues used a number of techniques (for a review, see Lavelli, Pantoja, Hsu, Messinger, & Fogel, 2005) including simple descriptive statistics, individual growth curve modeling (Fogel, 1995; Hsu & Fogel, 2001), conditional probabilities (Hsu & Fogel, 2003a), and event history analysis (Hsu & Fogel, 2003b) to measure the emergence, strength, and dissipation of parent–infant attractors. These methods are discussed in later sections of this chapter.

Time-Series Analysis

The group of methods that fall under time-series analysis offer some useful approaches to characterizing the real-time behavior of dynamic systems. The methods include visual representations as well as simple statistics that can be used to examine the qualities and predictive power of hypothesized attractor patterns. To illustrate the utility of this approach, we summarize the rationale, time-series procedures, and results from a study by Granic and Dishion (2003) using data from antisocial and normal friendship interactions.

Past research suggested that antisocial adolescents can be distinguished from their prosocial counterparts by the

extent to which they engage in reciprocal deviant talk (e.g., talk about stealing, lying; Dishion et al, 1995, 1996, 1997). Observational studies showed that antisocial peers had a higher mean duration of deviant talk (or "rule-break" talk) than prosocial peers. Central tendency measures, however, do not speak directly to the processes underlying these interactions. Moreover, they obscure potentially critical temporal patterns. To come closer to a process-level explanation, we began by conceptualizing deviant talk as an attractor for antisocial, but not prosocial peers (Granic & Dishion, 2003). Our interest was not in examining whether one group showed more deviant talk than another, but whether, over the course of an interaction, antisocial adolescents became stuck in an antisocial talk attractor. That is, did they become increasingly engrossed in topics organized around deviancy? One way to explore this hypothesis was to examine whether, as the interaction unfolded and antisocial dyads repeatedly returned to talking about deviant topics, they also spent increasingly more time in that deviant pattern. Time-series procedures were well suited for our purposes.

Deviant (or rule-break talk) and normative talk was coded continuously from videotaped interactions between best friends. Time-series plots were derived for each dyad, with the duration of each deviant talk bout on the y axis and each successive bout represented along the x axis (Figures 16.3 and 16.4). The slope of that time-series (i.e., the standardized beta) was then calculated using simple regression analysis. Slope is one parameter that can

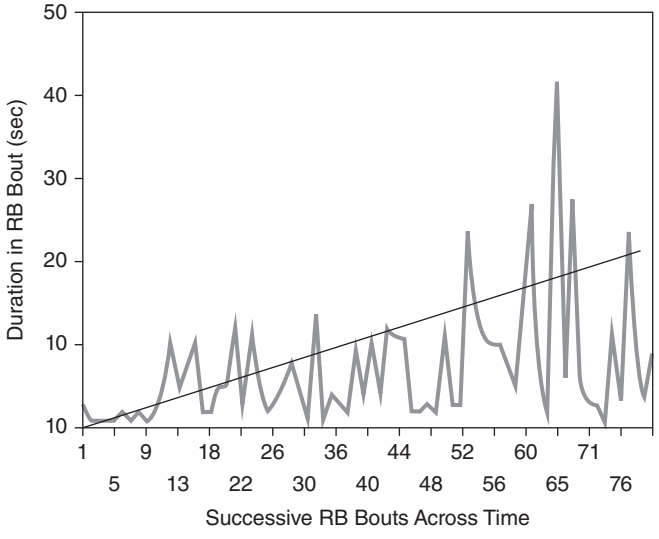


Figure 16.3 Example of a time-series for an antisocial youth with a positive rule-break bout slope. *Source:* From Granic, I., & Dishion, T. J. (2003). Deviant talk in adolescent friendships: A step toward measuring a pathogenic attractor process. *Social Development, 12*, 314–334.

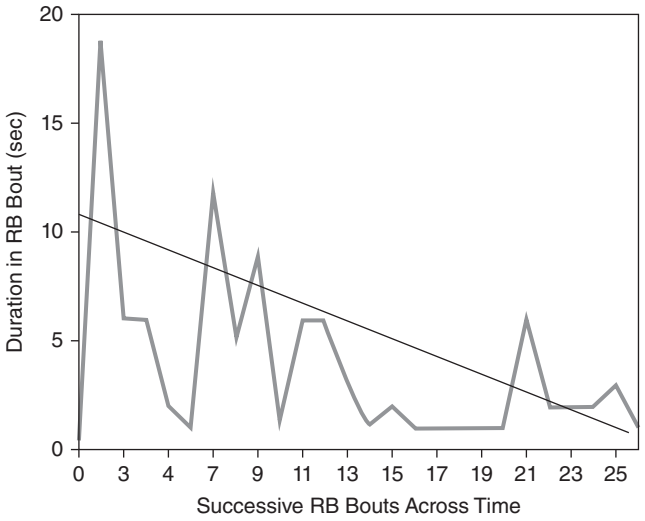


Figure 16.4 Example of a time-series for a normal adolescent with a negative rule-break bout slope. *Source:* From Granic, I., & Dishion, T. J. (2003). Deviant talk in adolescent friendships: A step toward measuring a pathogenic attractor process. *Social Development, 12*, 314–334.

be identified from a time series. Others include cyclicity (number of cycles in a series, length of cycles) and autocorrelation. We used the slope measure in a somewhat unique way to highlight a key DS principle. If deviant talk indeed functioned as an attractor for antisocial youth, then we expected to see a time-series that showed a positive slope, as exemplified in Figure 16.3. If it was not an attractor for a dyad, then we expected to see a time-series with either a flat or negative slope, as shown in Figure 16.4. Thus, each participant was assigned a deviant-talk slope value and these values were then used in regression analyses to predict antisocial outcomes 3 years later.

The results are summarized in Table 16.2. As hypothesized, the attractor index (the slope of deviant talk bouts) predicted serious authority conflict (e.g., number of arrests, school expulsion) and drug abuse three years later. These results were particularly strong because they remained statistically significant even after controlling for arguably the three most predictive risk factors in childhood: prior deviant behavior, family coercion, and deviant peer associations in childhood. We also showed that the mean duration of deviant talk bouts was not sufficient to predict these outcomes. Thus, these findings showed that reconceptualizing deviant talk as an attractor is not simply an exercise in relabeling; there are specific hypotheses that are engendered by applying this approach. From a DS perspective, although the frequency and amount of time spent talking about deviant topics are important, even more critical is the process by which dyadic behavior continues

TABLE 16.2 Regression Results Predicting Adolescent Authority Conflict and Substance Abuse in Midadolescence from the Strength of the Deviant Talk Attractor in Early Adolescence

	Antisocial behavior		Substance abuse	
Steps in regression	R^2 change	Total R^2	R^2 change	Total R^2
1. Child deviancy	.28[3]		.16[2]	
2. Child family coercion	.02	.30	.02	.18
3. Child deviant peer affiliation	.07[2]	.36	.06[1]	.24
4. Attractor strength (slope of deviant talk)	.05[2]	.41	.06[1]	.30

[1] $p < .05$.
[2] $p < .01$.
[3] $p < .001$.

to be drawn toward, and held, in this deviant pattern. Both antisocial and prosocial peers discussed deviant acts and breaking rules. It was the dynamic (time-based) characteristics of these conversations—as revealed by a simple time-series analysis–that differentiated antisocial and normal friendships.

Bakeman and Gottman (1997) used a similar analytic approach to analyze data from marital couples' interactions using this time-series technique, except they plotted the interevent interval between successive displays of negative affect (i.e., the time in between one negative affect and the next, across the interaction). They showed that, for distressed dyads, the time between each negative affect display became shorter and shorter over the course of the conversation (i.e., return time to the attractor became shorter over the interaction). Thus, negative affect was an absorbing state for distressed, but not happily married, couples.

Recurrence Quantification Analysis

Recurrence quantification analysis (RQA; Marwan, Carmen Romano, Thiel, & Kurths, 2007; Webber & Zbilut, 2005) is a nonlinear time series analysis technique that has found many applications across scientific disciplines (Webber, Marwan, Facchini, & Giuliani, 2009). We here discuss an adaptation of the RQA for categorical data because these are likely the kind of data that clinical researchers and developmental psychopathologists will use. In studies of behavior observation the data are usually time series that represent the occurrence of a particular type of behavior at a particular point in time. In that respect the data may be characterized as a temporal sequence of unordered behavioral responses, that is, as categorical time-serial data. RQA quantifies the temporal order in time series at each and all possible timescales or delays during the observation. RQA for categorical data is based on the dichotomous distance matrix, in which it is noted for all behaviors when each behavior that occurs at some instance in the time series also occurs at some other instance in that same time series, either earlier or later. Such a reoccurring value is called a recurrent point, which is the basic measure of RQA. A two-dimensional graphical representation of this matrix is called the recurrence plot. In a recurrence plot all the points are plotted that represent recurring values (Figure 16.5). When recurrent points occur in subsequent time steps in the time series, line or

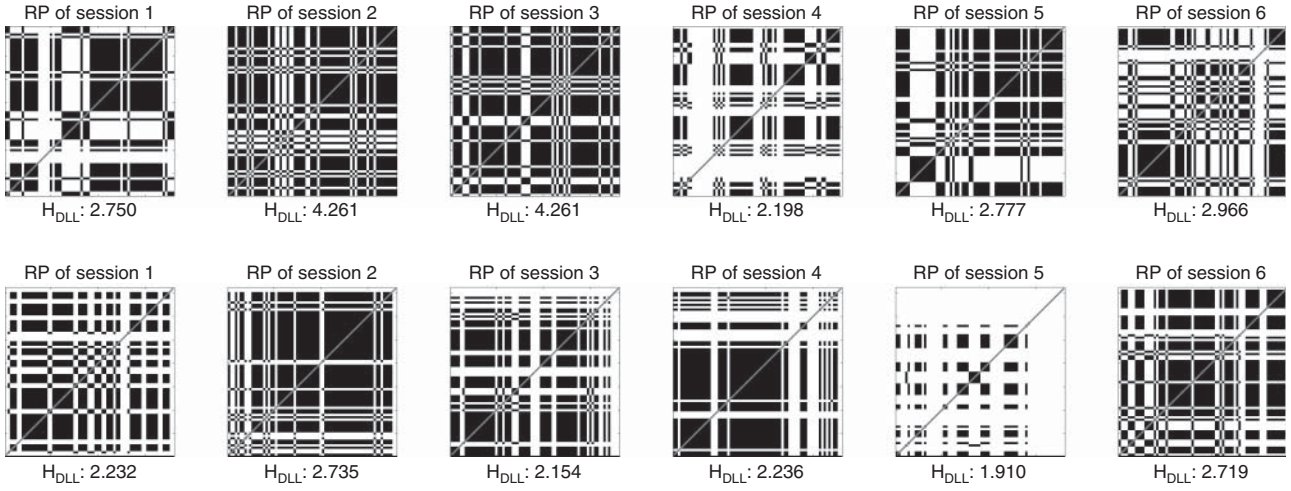


Figure 16.5 Recurrence plots of two exemplary dyads across observation sessions. Gray diagonal line represents the line of identity. Dyad in the upper row is showing the peak in entropy and dyad in the lower row does not show a peak in entropy. *Source:* With permission to reprint figure from Dr. David Pincus, Nonlinear Dynamics, Psychology, and Life Sciences (NDPLS) Permissions Editor. From Lichtwarck-Aschoff, A., Hasselman, F., Cox, R., Pepler, D., & Granic, I. (2012). A characteristic destabilization profile in parent–child interactions associated with treatment efficacy for aggressive children. *Nonlinear Dynamics, Psychology, and Life Sciences*, *16*, 353–379.

block structures emerge in the recurrence plot. These structures reflect that certain sequences of values are repeatedly occurring (i.e., recurring) over time. One could say the dyad returns to a previously displayed pattern of behaviors. The (average) length of such structures indicates how stable (or prominent) this particular pattern of behavior is in the interaction. Note that various structures that are located at different regions in the recurrence plot may represent different behavioral patterns, not only in duration but also in terms of their particular constituent behaviors (i.e., content). As can be seen in Figure 16.5, the recurrence plots are symmetrical around the line of identity (see grey diagonal line). This line represents the time series as it was observed. Several different measures can be derived from those recurrence plots (e.g., determinism, predictability, entropy, trapping time, and divergence; for an elaborate discussion see Webber & Zbilut, 2005).

In a recent study Lichtwarck-Aschoff and colleagues (2012) applied RQA to examine changes in entropy to investigate whether successful treatment outcomes were associated with having gone through a phase transition. Repeated mother–child interactions of children being treated for aggression were analyzed over the course of a 12-week treatment period (treatment was a combined Cognitive Behavioral Therapy and Parent Management Training). Interventions can be seen as causing perturbations to a (mother–child) system. To be effective in eliciting a qualitative shift to a more healthy attractor state, treatment should trigger increasing levels of variability and disorganization up until the point that the system can no longer resist and is forced to give up the old maladaptive attractor state (see also Granic & Patterson, 2006). The temporary change in the natural variability of the system, the increased within-system variability, is functional in the sense that it allows the system to explore new patterns and configurations (De Weerth & Van Geert, 2002; Thelen & Smith, 1994; Van Geert & Van Dijk, 2002). After the destabilization phase, the system settles down again, organizing into a new (dynamically) stable regime of the system (Van Orden, Kloos, & Wallort, 2009). Thus, it was expected to find a change pattern, signaling an impeding phase transition that was characterized by a temporary increase in random fluctuations or breaking down of stable recurring patterns of dyadic behaviors following the start of the intervention.

The RQA measure that quantifies this particular aspect of the dynamics is the Shannon entropy of the distribution of diagonal line structures in the recurrence plot (see also Stephen et al., 2009; Stephen, Dixon, & Isenhower, 2009). A subsequent latent class growth curve analysis

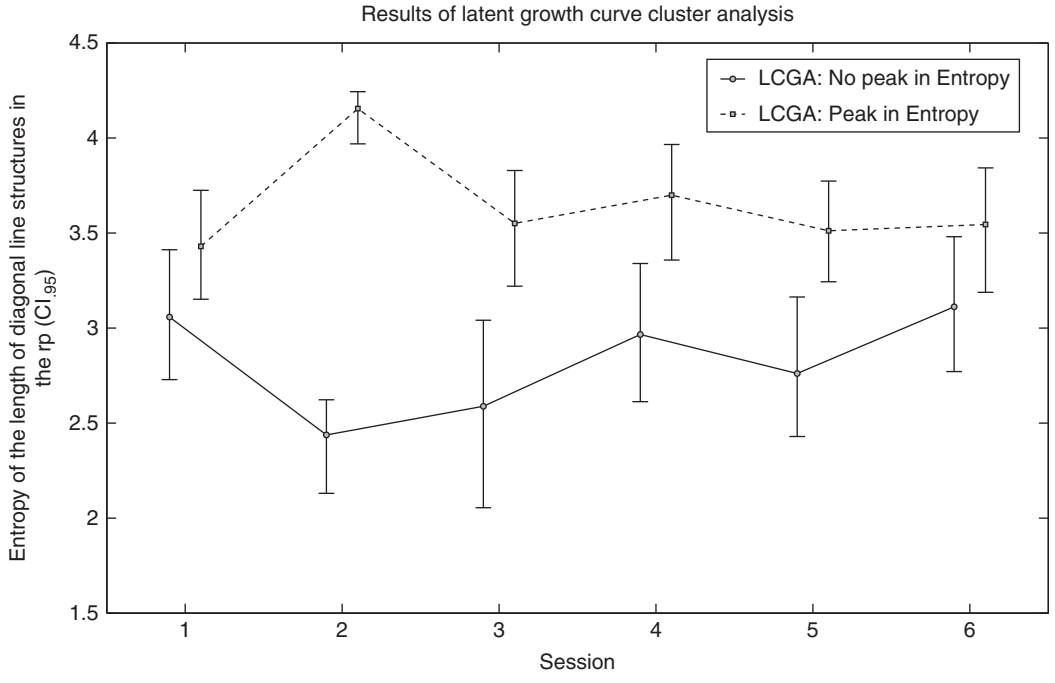


Figure 16.6 Result from the LCGA analysis of values for entropy of the diagonal line structures in the recurrence plots of each dyad across the six observation sessions (95% confidence intervals are based on 15,000 bootstrap replications). *Source:* With permission to reprint figure from Dr. David Pincus, Nonlinear Dynamics, Psychology, and Life Sciences (NDPLS) Permissions Editor. From Lichtwarck-Aschoff, A., Hasselman, F., Cox, R., Pepler, D., & Granic, I. (2012). A characteristic destabilization profile in parent–child interactions associated with treatment efficacy for aggressive children. *Nonlinear Dynamics, Psychology, and Life Sciences*, *16*, 353–379.

on the sequences of entropy values revealed two distinct classes of dyads, with one class showing a clear peak in entropy over the six measurement points (Figure 16.6). The latent class membership variables showed a significant systematic relationship with observed dyad improvement (as rated by clinicians). The class with the peak in entropy over the sessions consisted largely of treatment improvers (Figure 16.6). Further analysis revealed that improvers and nonimprovers could not be distinguished based on content-specific changes (e.g., more positivity or less negativity during the interaction). This study thus supports the DS view that treatment functions through creating the conditions that facilitate dynamic reorganization (Schiepek, 2009) rather than a linear input–output model in which a series of equally powerful weekly interventions force the system to a particular reaction or end state.

Event History Analysis

As can be seen with the previous examples, and others to follow, it is not always necessary to invent new methods to explore DS-inspired hypotheses. Another method that can help researchers explore the different characteristics of attractor processes is event history analysis. A variant of survival analysis and Cox regression models, hazard analysis calculates transition rates (also called hazard rate or latency to recurrence) of state changes in categorical time series (Allison, 1984; Blossfeld & Rohwer, 2002; Stoolmiller & Snyder, 2006). The rate of transition in and out of a behavioral state (e.g., tantrum, laugh) can be interpreted as an index of attractor strength. Several other measures including the frequency, duration, total time, and the time since the last occurrence of the behavior can be included in these models to determine the absorbing quality of a hypothesized attractor. Furthermore, time-invariant (e.g., gender, child's problem behavior, maternal depression) and time-varying (e.g., changes in context, affective behavior of the partner, time) covariates can be added to these models as predictors of the transition rate.

Figure 16.7 shows a schematic diagram of an interactive sequence. The bottom plot line represents the time line of events being modeled. In this case, there are only two states possible: in the proposed attractor or out of that state. This example illustrates a dichotomous response set, but it is not necessary for event streams to be binary for event history analyses; multiple states can be modeled as competing risks. The three episodes in this example correspond to the start and end times of the behavior of interest. The first episode lasts 14 seconds, the second episode lasts 18 seconds, and the third is 14 seconds long. With just this information it is possible to model the probability of transitions into the attractor as one instantaneous rate, and the duration since the last episode can be added into the model as a predictor.

Presumably, whatever behavior is being researched does not occur in a vacuum. There is the nature of the task at hand, the influence of other people and even factors unique to each individual that must be considered. Thus, as shown by the time-varying covariates (upper 2 lines) in Figure 16.7, more elaborate models can tease apart the relative contributions of various covariates to the recurrence of a particular behavior. In a series of studies Snyder,

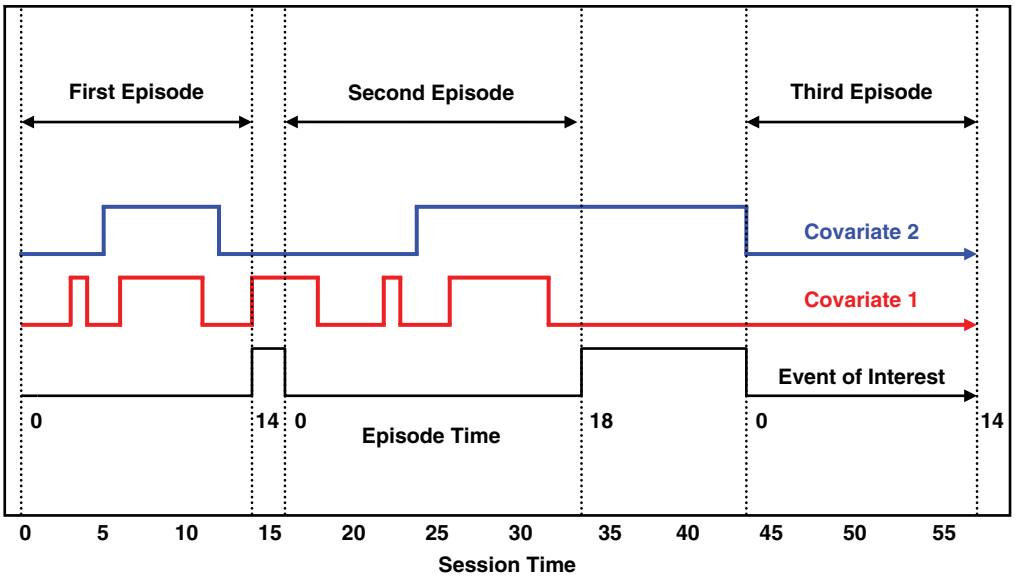


Figure 16.7 Schematic diagram of a behavioral interaction with some of the components that can be analyzed through event history analysis. See footnote 1. *Source:* Adapted from: Snyder, J., Stoolmiller, M., Wilson, M., & Yamamoto, M. (2003). Child anger regulation, parental responses to children's anger displays, and early child antisocial behavior. *Social Development, 12*, 335–360.

Stoolmiller, and colleagues have used hazard analyses to investigate a wide range of parameters of the state space for emotion response states in parent–child interactions (with clinical and control children), and the relationship of those parameters to time fixed and time-varying covariates. In the first study a repeated-events, competing-risks hazard analysis was used to estimate the return time for child anger displays using both time fixed and time-varying predictors (Snyder, Stoolmiller, Wilson, & Yamamoto, 2003). In state space terms, return time may be interpreted as an indication of the strength of the anger attractor state. Results showed that the longer it took a child to down regulate an anger display, the more quickly that anger display would reoccur. Further, the authors found that the speed of child's return time to an anger display (at time $t + 1$) was amplified by the cumulative frequency of parent negative responses (a cumulative time-varying covariate). Using a time-fixed covariate, poor parental discipline was also related to a faster return rate to anger. Individual differences in the hazard for return to anger displays were also associated with children's conduct problems (as rated by teachers and parents).

In a second study a complementary hazard analysis was applied to investigate the relationship of the child's hazard to move to an angry start state (given mom was aversive) to a neutral state to child overt and covert antisocial behavior (Dagne & Snyder, 2009). It was found that children high on overt antisocial behavior relative to the average child were slower to move to a neutral emotional state. Children high on covert antisocial behavior relative to the average child, in contrast, were faster to move to a neutral affective state. Additionally, maternal self-reported trait hostility (a time-fixed covariate) was associated with a reduced hazard for mothers to move from an aversive to a nonaversive state in the context of child anger displays, which indicates that hostile mothers are slower to cool down in negative encounters with their children.

In a third study Stoolmiller and Snyder (2006) examined the hazard of children's transition from a neutral state to positive, sad/fearful, or angry emotion states when their mothers initiated an aversive behavior. It appeared that on average, the hazard for children to move to a negative emotion state (either angry or sad/fearful) increased as mothers initiated an aversive behavior. Children high on antisocial behavior were significantly slower to move from neutral to sadness/fear, but there was no relationship between antisocial behavior and children's hazard to move from neutral to anger.

Finally, in a recent study Snyder, Bockman, and Stoolmiller (2012) investigated the hazard of down-regulating anger and sadness/fear relative to neutral in relation to children's antisocial behavior. The results showed that overt and covert antisocial behaviors were associated with an underutilization and rapid down regulation of sadness/fear relative to neutral. Further, children in overt antisocial behavior also showed an overutilization and slow down regulation of anger relative to neutral affect.

These studies demonstrate that hazard analysis is a sophisticated and flexible mathematical approach to model a system's behavior by capturing temporal and well as sequential shifts among various emotion states. These models can be run on individuals or pooled across an entire sample and it is possible to model the state space of emotion displays from a dyadic as well as an individual perspective (Stoolmiller & Snyder, 2006). It is not only the potential of identifying predictors of attractor strength that is the appeal for DS researchers but the fact that *time* itself can be used as a predictor. Probably the most difficult process of event history analysis is restructuring data into the appropriate format. Despite this difficulty, we expect that event history analysis will be used with increasing frequency as a method to model attractor dynamics.

Fourier analysis or *spectral decomposition* can be used for finding periodicities (i.e., cyclic patterns) in time-series data such as those used in phase plots. In general, this procedure treats a time-series as a conglomerate wave form, breaks it down into a collection of pure waves (each of uniform frequency), and identifies the most prominent waves (Schroeck, 1994). Newtson (1994, 1998) used this method to analyze the coupled dynamics of dyadic interactions. The relative amplitudes and temporal synchrony of these behavior waves were associated with the degree of mutuality or competition in interpersonal relationships. In other applications, different types of attractors including oscillating and periodic attractors can be identified. For behavioral scientists, however, this method has its limitations. Like all time-series procedures, it requires the researcher to collapse meaningful categorically coded observational data into one or very few continuous dimensions (e.g., Bakeman & Gottman, 1997). For example, most observational coding schemes used in developmental psychopathology (e.g., SPAFF—Gottman, McCoy, Coan, & Collier, 1996; FPC—Dishion, Gardner, Patterson, Reid, & Thibodeaux, 1983; MAX—Izard, 1979) code discrete behaviors like *contempt, argue, belligerence,* and *whining*. To conduct Fourier analysis, these codes would have to fall along a single dimension (i.e., intensity of negativity). This type of collapsing is often either conceptually unfeasible or it is unappealing because of its oversimplification.

Karnaugh Maps

Inspired by synergetics, a type of dynamics developed by Haken (1977), Dumas, Lemay, and Dauwalder (2001) adapted this technique from Boolean algebra to study parent–child interactions. Karnaugh maps depict all possible combinations of up to four binary variables in one table or grid. A simple two-variable Karnaugh map is basically a four-cell cross-tabulation of event frequencies with one dichotomous variable to each axis. Three- and four-variable maps are somewhat more complicated. Figure 16.8 shows a schematic four-variable Karnaugh map. Each variable (A, B, C, and D) can have a value of 0 or 1, and each is displayed on one side of the square table (bottom, left, top, and right). Each cell in the map can be represented as a unique combination of the binary values of the four variables. Thus, a Karnaugh map is a state space of all possible states of the system and represents the relative frequencies in each state. Dumas and colleagues extended the application of these maps by plotting the transitions between states to depict temporal patterns across the space.

In their study, Dumas et al. (2001) plotted every minute of six-hour home observations according to four parent and child dichotomous variables that included control, compliance, aversive and positive behavior. Each behavior was plotted according to the four-variable configuration and successive behaviors were connected by a trajectory. They were primarily interested in comparing the maps of clinically referred dyads with randomly selected controls. They analyzed the recurring transactions (i.e., attractors) that were found most frequently in each group and aggregated these findings graphically on one summary Karnaugh map. Their results showed differences in transactional patterns between the groups, mostly involving positive interactions and cycles of maternal control and child compliance.

In addition to the graphical depiction of interaction sequences, the authors computed a complexity index that was designed to quantify each map on a continuum from completely deterministic to completely random. They found that all maps, regardless of group assignment, were neither random nor deterministic but somewhere in between. While this approach was unique and interesting (particularly from a methodological standpoint), from a DS perspective there is no reason to think that social behavior, especially in dyads with a rich history, is ever random or ever completely determined. Nevertheless, the complexity measure has a great deal of potential. For example, it might be used to determine whether stable coercive parent–child interaction patterns become less determined (i.e., the old attractor patterns break down) during developmental transitions such as puberty.

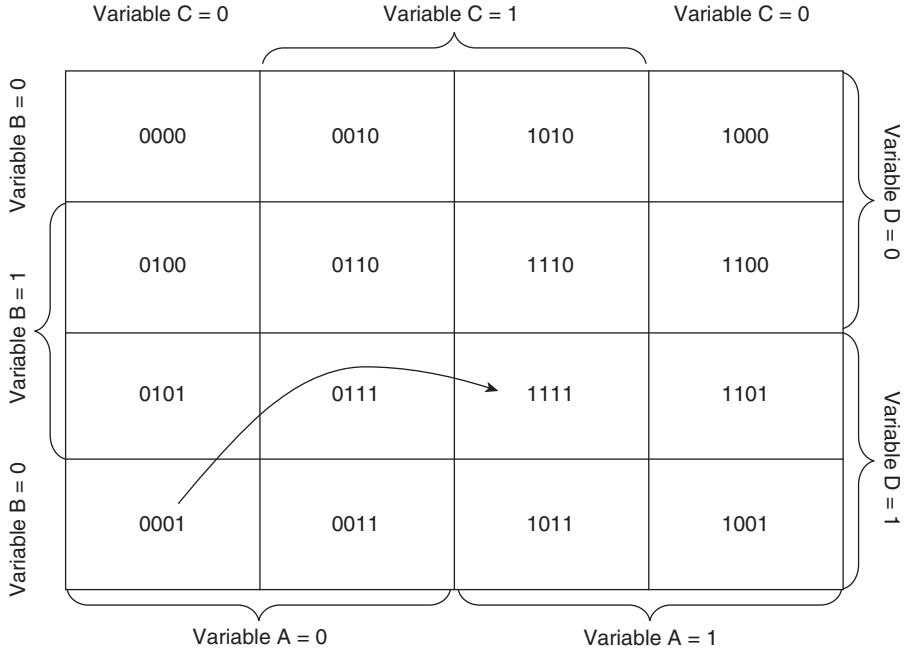


Figure 16.8 Schematic of a four-variable Karnaugh map. Each cell is a unique combination of four binary variables. The arrow shows a sample transition from one state, where only variable D is present (i.e., only D equals 1), to the next state in time, where all 4 variables are present (i.e., all equal 1).

A very similar approach has been applied in a study by Lichtwarck-Aschoff and colleagues (2009) to investigate intraindividual variability on a different time scale: diary entries of mother–daughter conflict episodes. Indices of the episode-to-episode variability in emotional states (i.e., composition of emotions) were used to assess whether girls in conflictual mother–daughter relationships would show signs of being stuck in a certain (emotional) conflict pattern. In line with research on real-time variability (Granic et al., 2007; Hollenstein & Lewis, 2006; Hollenstein et al., 2004) it was hypothesized that conflictual relationships would be characterized by a lack of variability across episodes. However, in contrast to real-time measures, which reflect changes from one emotion to another moment by moment, diary entries describe emotional states of entire conflict episodes that can be composed by several different emotions. Thus, changes in a multidimensional (emotional) space have to be assessed. (Note that this emotional space is therefore different from the state space grids developed by Lewis et al., 1999, described later in this chapter.)

To do so, measures based on symbol dynamics (Daw, Finney, & Tracy, 2003), which actually extends the Karnaugh maps in terms of the dimensions that can be included, were developed. The basic rationale behind this approach is the transformation of the system's trajectory or geographical movement in time into a sequence of specific symbols, corresponding to partitions within the state space (Dale & Spivey, 2005). First, the emotional state space for each girl was computed, which contained all her reported emotions across all her conflict episodes. Second, within each girl's state space all the emotional states that represented how a girl felt during a particular conflict were described. An emotional state could take the form of a single emotion or a combination of (many) different emotions. The mathematical representation of every state consisted of a particular string of ones (emotions being present) and zeros (emotions being absent). One way to assess the emotional variability was to count the number of different emotional states across all conflict episodes. Further, the Hamming distance (Hamming, 1950; Teşileanu & Meyer-Ortmanns, 2006) was used to compute additional variability measures. The Hamming distance calculates the distance (absolute difference) between two symbolic strings (here, two emotional states). The distance between the emotional states then indicates how much the emotional states differ from each other and in this sense represents the variation between the states (compare this with the intergrid distance used by Lewis et al., 1999). In this study a sequential and an overall Hamming distance was computed.

As expected the results revealed an inverse U-shaped relation between girls' emotional variability and the number of conflicts. Thus, the emotional variability decreased when relationships became highly conflictual. Moreover, girls who showed limited variability in emotional states across conflict episodes tended to attach the same emotional state to divergent conflict topics. For these girls, many different conflict contexts still induced the same emotional state. In other words, these girls felt the same, no matter what. Hence, the conflictual mother–daughter systems seemed to be stuck or frozen into a small number of emotional states and had lost their situational sensitivity (i.e., sensitivity and flexibility in response to different conflict topics).

Karnaugh maps and symbol dynamics hold considerable promise for several reasons. First, because these methods allow for the representation of behavior in multiple dimensions (including time), a great deal of the complexity in (interactional) behavior can be nicely captured. In case of the Karnaugh maps the temporal quality of dyadic behavior is also maintained and can be tracked easily through visual inspection. Moreover, the systemic properties of dyadic interactions are kept intact, in contrast to conventional methods that often require analyzing each dyad member separately. The approach described in the Lichtwarck-Aschoff et al. (2009) study offers the possibility to study changes and variability of multidimensional behavior which is especially relevant for studies on larger time scales where measures often reflect some aggregated unit of analysis (retrospective reports of conflict episodes rather than real-time presentations of emotions). However, some limitations need to be addressed before these techniques can be applied to a broad range of behavioral data. One problem is that data have to be collected or converted into dichotomous variables that occur concurrently. Developmental psychopathologists are often interested in data that cannot be meaningfully transformed into dichotomous values; continuous or categorical data cannot be adequately captured with these techniques. Also, these methods do not provide direct quantifiable tests of the strength of (dyadic) patterns, nor opportunities for comparing patterns. In this respect, the new developments in state space grid analysis may provide some promising avenues. Some of the quantitative measures that may be derived from state space grids may also be applicable to Karnaugh maps.

Coupled Equations

Most generally, this method refers to the use of paired equations derived from two synchronized time-series

that produce parameters that describe the underlying dynamics of a system. The use of coupled equations may involve mathematically demanding procedures and often require fine-grained time-series data or simulated data. One particularly successful application in the field has been the work of John Gottman and colleagues, who used coupled differential equations to model the dynamics of marital couples and to predict from those dynamics couples who will remain married or end up divorcing (Gottman, Coan, Carrere, & Swanson, 1998; Ryan, Gottman, Murray, Carrere, & Swanson, 2000). They also used this method to study how peer interactions influence the behavior of developmentally delayed versus normal children (Gottman, Guralnick, Wilson, Swanson, & Murray, 1997) and parent–infant interaction (Gottman, Murray, Swanson, Tyson, & Swanson, 2002).

Gottman and colleagues' modeling procedures, based on mathematical biology, are an attempt to mathematically formalize theoretical assertions made by general systems theorists such as von Bertalanffy (1968). Their model itself represents a theory about the mechanisms and dynamics of marriage. Their approach is to model maritial interactions using the mathematics of difference and differential equations. These equations express hypothesized mechanisms of change over time. Gottman and colleagues offered their model of marital interactions as a first attempt to create an explanatory theory of marriage; they recognize the value of cycling from theory development to modeling to experiments and back to theory development. As the authors comment, "The theory may be dead wrong, but it is precise" (Gottman et al., 2002, p. 67).

The modeling process is guided by posing two questions: (1) What are the steady states; and (2) which of these states are stable (i.e., the attractors) and which are unstable (i.e., repellors)? The first step is to set up the phase space such that each axis represents a variable corresponding to each participant. (This procedure is not the same as the one we described earlier in lag-1 phase plots.) The next step was to identify the steady states by graphing the null clines, curves in phase space for which variables stay constant. These curves help to determine the steady states by partitioning the phase space into regions where changes in each variable are increasing or decreasing.

Gottman et al.'s (2002)'s technique uses a time-series of coded observational data to create an equation for each person in the interaction. For this method, Gottman et al. uses variables that are codes or categories of codes that fall along a single continuous dimension from very negative to very positive with neutral in the middle. Equations are created to reflect the theoretical presumption that one person's behavior at time t is a function of his or her own behavior at time $t - 1$ as well as the other participant's behavior at time $t - 1$. Thus, there is an uninfluenced and an influenced factor in each equation that determines the behavior at each time t. The uninfluenced component is what each person brings to the interaction independent of the other. The uninfluenced set point can be thought of as a temperamental factor that is assumed to be the attractor a person, left on his or her own, will eventually approach. Mathematically, this is denoted by a parameter that represents the rate of change toward the uninfluenced set point, also called emotional inertia, plus a constant that is specific to the person. The influenced component is a mathematical function representing the other person's previous behavior. These functions can be used to plot curves that describe the degree of influence based on one assumption: positive behavior influences positively and negative behavior influences negatively. Thus, the equations for a husband, H, and wife, W, are

$$W_{t+1} = r_1 W_t + a + I(H_t)$$
$$H_{t+1} = r_2 H_t + b + I(W_t)$$

where r is the emotional inertia, a and b are constants specific to the individual, and I is the influence function. These equations are then manipulated to estimate the parameters and to determine the uninfluenced and influenced set points. Plotting both of the null clines (the curves for which the values of the variables remain constant) in phase space allows for the identification of stable steady states—the points where the two curves intersect.

Consider the example in Figure 16.9a, which shows a bilinear null clines for a husband and wife. There is one positive and one negative stable state, or attractor, for this couple (the circles at the intersection of the two null clines).

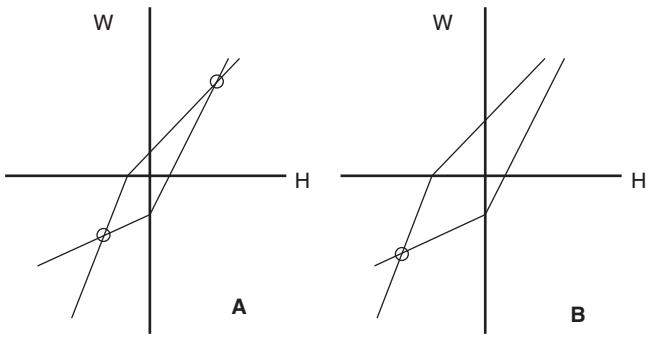


Figure 16.9 Null clines and the stability of steady states. Steady states are circled and represented by the point at which the null clines intersect. *Source:* Adapted from Gottman, J. M. (2002). *The mathematics of marriage: Dynamic nonlinear models*. London, UK: MIT Press.

Over the course of the interaction their behavior is drawn to one or the other of these attractors. One of the interesting uses of this modeling technique is its depiction of how a system exhibits sensitivity to initial conditions and how marriages can change over time. For example, situations at work may lead the husband to feel a lot of stress and he may begin to express this stress in his interactions with his wife. This would be reflected in a change in the parameter b in the model that corresponds to the husband's uninfluenced state, or what he brings to the interaction. A change in this constant shifts his influence function to the left (Figure 16.9b) to the point where the positive attractor no longer exists. Thus, this type of modeling can distinguish between superficial change (i.e., a move to different stable state) and deeper change (i.e., a fundamental change in the influence function that eliminates or adds a stable state to the marital couple's behavioral repertoire).

Gottman and colleagues (2002) have used this basic model and more elaborated versions to identify mechanisms related to specific types of marriages. Some couples only have one stable steady state, if it is positive the marriage is happy, if it is negative the marriage is unhappy. Typically, the situation is more complex. Happy couples also tend to have both positive and negative attractors. In fact, high influence and high inertia couples tend to have multiple steady states and thus will most likely have at least one negative attractor. It may be the relative strengths of these attractors that best distinguish distressed and nondistressed couples.

Gottman and colleagues' approach to mathematical modeling seems to hold exciting potential for developmental psychopathologists who are interested in understanding social processes of various kinds. For example, their model can be easily adjusted to simulate interactions that have different types of power imbalances: parent–child interactions, therapist–client sessions, sibling interactions, bully–victim relations, and so on. Developmental psychopathologists have constructed detailed theoretical models that hypothesize specific influence functions among social partners (e.g., coercion processes in families with aggressive children, authoritative parenting, and anxiety-disordered children). Gottman et al.'s approach challenges scholars in the field to translate these theories into mathematically specified mechanisms. This approach may be particularly rewarding if developmental principles can be formalized and included in these endeavors.

Nonlinear Dynamics

These methods are derived from mathematical procedures in physics and other sciences that aim to measure and model nonlinear phenomena (Abraham, Abraham, & Shaw, 1990; Heath, 2000; Norton, 1995). The simplest of these is nonlinear regression wherein the parameters are exponential functions or the predictive combinations are not simply additive. Other applications from this area are those related to chaos theory (Newell & Molenaar, 1998). One can use nonlinear dynamic techniques to find the embedding dimension, entropy, determinism, recurrence, or fractal dimension of any time series of sufficient length and sufficient precision. This class of techniques is typically applied to continuous time-series data (type 1), often physiological. For those developmental psychopathologists who are increasingly collecting this sort of data (e.g., heart rate, skin conductance, neural activity), this class of methods holds a great deal of promise, particularly if there are reasons to hypothesize nonlinearities. Moreover, there are many software packages available for free or at minimal cost that can calculate these measures for any appropriate time series. The challenge, therefore, lies not in the calculation but in the psychological interpretation of these measures.

There are a number of diverse applications of this class of techniques. One of the main reasons for using these methods is to determine the degree of orderliness, complexity, or predictability in the behavior of a given system. One example in developmental psychopathology comes from the work of Dishion, Nelson, Bullock, and Winter (2004). They applied a technique that calculated the degree of complexity of peer interactions, in the hopes of characterizing and differentiating prosocial versus antisocial peer interactions. The authors drew upon information theory and applied Shannon's entropy measure (Shannon & Weaver, 1949) as an index of the degree of disorganization, uncertainty, or information in a dyadic interaction. In information theory, uncertainty and information are related because "information about an unknown event is more useful as one knows less about the event" (Wickens, 1989, p. 231). For example, if 100% of a set of communications is of one type (e.g., deviant talk), there is total certainty and no information is conveyed from one communication to another. This is a case of low entropy. Conversely, if there are a variety of types of communications and all are equally probable, then there is total uncertainty and a maximal amount of information is gained by finding out which event occurred. This is an example of maximum entropy.

Shannon's entropy is typically used with contingency tables where the values in each cell represent the probability of a different two-event sequence. A simple way to think of entropy is the average number of dichotomous

decisions (yes–no) that need to be made to classify an event in that table (Wickens, 1989). Thus, in the previous example, the situation where only one event is observed would have a minimal entropy value of 1 because only one dichotomous decision is necessary to classify any event. Shannon's entropy is calculated as follows:

$$\text{Entropy} = H = \Sigma p_{ij}(\log_2(1/p_{ij}))$$

where p_{ij} is the conditional probability in the cell that is in row i and column j of a contingency table.

Dishion et al. (2004) used turn-taking observational data of boys interacting with their closest friend to calculate entropy. Their conversational turns were coded into five categories: negative engagement, directive, converse, positive engagement, and other. Thus, there was a 5×5 matrix of two-event sequences for each peer dyad. The frequencies in each cell of the matrix were used to calculate the conditional probabilities necessary for the entropy calculation. The findings from this study showed that all of the friendship dyads' interactions (regardless of whether or not they were antisocial) became more ordered (less entropic) over three time points, ages 14, 16, and 18 years old. This finding in itself is compelling. It supports the DS view that development is characterized by increasing orderliness—a crystallization of interaction patterns. As relationships of all kinds develop, they stabilize and, thus, become more predictable over time. When the authors compared early-onset antisocial boys with prosocial boys, they found that antisocial boys' interactions corresponded to higher entropy scores, suggesting a more disorganized pattern, and were characterized by more deviant talk than the typically developing boys. These results may suggest that antisocial boys are less skilled than prosocial boys at carrying on a fluid, appropriately contingent conversation.

Some other potentially useful applications of this approach are found in clinical therapy research (e.g., Badalamenti & Langs, 1992; Burlingame, Fuhriman, & Barnum, 1995; Butz, Chamberlain, & McCown, 1997; Lichtenberg & Heck, 1986; Pincus, 2001; Tschacher, Scheier, & Grawe, 1998). Lichtenberg and Heck (1986) examined the "Gloria" psychotherapy tapes and compared the results of Shannon's entropy measure, Markov chain analysis, and sequential analysis. Each of these approaches showed some merit and each had associated limitations. In terms of therapeutic process and outcome, two therapy studies have applied Shannon's entropy measure with somewhat contradictory results: one found that, over the course of intervention, verbal utterances became increasingly complex (Badalamenti & Langs, 1992) while the other found that client and therapist measures of therapy sessions became increasingly coherent (i.e., less complex, according to the entropy measure) and this tendency was related to treatment success (Tschacher et al., 1998).

Finally, a study conducted by Pincus (2001) set out to demonstrate that when treatment works, therapy sessions become both more complex and more coherent. This study may be particularly relevant to developmental psychopathologists because it was based on assumptions from family systems theory (Minuchin, 1974). Pincus was interested in empirically testing one of the basic assumptions of family systems therapy: that families maintain a state of equilibrium through negative feedback processes which dampen deviations. These deviations (e.g., depressive withdrawal, angry outbursts) manifest as symptoms for a particular family member, but also serve to maintain homeostasis in the family (e.g., Minuchin, 1974; Minuchin & Fishman, 1974). For example, a child may become oppositional to (consciously or unconsciously) distract the family from the real problem: intense marital discord. When the child acts out, the parents join forces and attend to the disruptive behavior, which precludes escalation of the marital conflict (dampening process); thus, the family maintains a state of equilibrium, however dysfunctional it may be. The goal of family therapy is twofold: "to disrupt or block these negative feedback processes, promoting family disequilibrium and reorganization" and to "capitalize on natural deviation-amplifying processes within family systems in order to bring about systemic change" (positive feedback; Pincus, 2001, p. 145). In short, the goal of therapy is to induce a phase transition in the family system.

As a first step toward validating these assumptions, Pincus conducted a pilot study in which he analyzed the conversations of one family undergoing therapy. He used an analytic technique called *orbital decomposition* (OD; Guastello, Hyde, & Odak, 1998), which combined Shannon's entropy and measures of Lyapunov dimensionality, as well as others, to identify hierarchical patterns, the extent of randomness in a time series, and the optimal level of analysis for any particular series (i.e., the number of units in a string that best captures the information for a time series). The results confirmed that, indeed, the family exhibited evidence of both coherence and complexity across treatment sessions. Because this was a pilot study, we are hesitant to make too much of these findings. But OD may hold particular promise for developmental psychopathologists because it was developed for analyzing *categorical* time series data, instead of the usual continuous series. As such, it addresses a major limitation that has

been repeatedly raised with the other time series techniques reviewed here.

The methods for analyzing nonlinear dynamics described in this section have been applied to single time series. These are appropriate for some biological measures with high temporal resolution (e.g., heart rate) but another form of continuous data, electrical activity in the brain, presents a more complex challenge. There are increasingly more developmental psychopathologists who are integrating brain activity measures in their research programs (e.g., Calkins & Fox, 2002; Cicchetti & Dawson, 2002a; Nelson et al., 2002). Although there is clearly not enough space to discuss the various brain imaging methods in the current review, a word on these techniques is warranted. Ongoing electrical activity in the brain, measured by electroencephalogram (EEG), can involve as many as 256 separate time series, one per electrode. Methods that analyze these kinds of complex data are designed to find relations and patterns among and across these various time series. For example, developmental neuroscientists interested in the self-organization of brain dynamics have found real-time synchronization across distal cortical areas to be correlated with attentional states or focused perception (Lewis, 2005). Transient, organized activity in different regions oscillating in phase (phase synchrony) and cortical coherence (a similar measure that incorporates both amplitude and phase) are detected through various temporal correlation methods and have been found to correspond with attentional (e.g., Engels, Fries, & Singer, 2001) and emotional (Paré & Collins, 2000) events.

We close this section with a cautionary note that applies to any dynamical mathematical technique—because these techniques are derived from other fields, oftentimes it may be easier to input numbers into the equations than to sensibly interpret the results of the computations. A number of DS researchers have been criticized for interpreting their mathematical functions too literally, either the data inserted into the equations do not conform to the mathematical assumptions or, perhaps more seriously, they are potentially arbitrary in terms of their psychological meaning. These methods hold a great deal of promise, but a great deal of theoretical and empirical work remains to be done before the psychological meaning of nonlinear dynamic techniques is clearly understood.

Developmental-Time Measures

Descriptive Developmental Profile Analysis

The empirical work by Thelen and colleagues (e.g., Thelen & Smith, 1994; Thelen & Ulrich, 1991) has been characterized as descriptive in that it is nonparametric, uses descriptive statistics, and often relies heavily on displaying individual developmental profiles graphically. These researchers most often collect continuous time-series data (e.g., number of alternating steps, degree of displacement of foot, proportion of stepping cycle) over repeated occasions across a significant developmental period. They create developmental profiles on a case-by-case basis and describe the similarities and differences among these profiles. A core concern in this type of analysis is to identify periods of transition during which variability dramatically increases and old behavioral habits dissolve, giving rise to new ones (i.e., from crawling to walking).

The most straightforward way to graph developmental growth profiles is to simply plot scores for the collective variable (e.g., alternate stepping) on the y axis across several developmental time points on the x axis, and to do so for each case individually. Van Geert and van Dijk (2002) offered a number of additional methods for visualizing and describing intraindividual time-series data over repeated observations. These methods were specifically created for measuring changes in variability across developmental time and they are well suited for one-dimensional continuous longitudinal data. In addition, with any individual developmental profile plot, several trajectories can be grouped into clusters that show similar profiles by using variations of a cluster analytic procedure (e.g., Hollenstein, Granic, Stoolmiller, & Snyder, 2004).

One simple but elegant plotting technique is referred to as the *moving min-max graph* (van Geert & van Dijk, 2002). This graph represents the developmental trend line but also the variability around this trend line in a bandwidth of observed scores. A moving window is used—that is, a time frame selected by the researcher (e.g., 2 weeks, 3 months) is moved over one position all the way across the time series such that each window partly overlaps with the previous window. There is no absolute window length that is required, but van Geert and van Dijk (2002) suggest that the window size should be about one-tenth the size of the entire data set and no less than five data points. The maximum and minimum score at each time window is plotted and then the bandwidth of these scores can be examined in terms of its stability and change. In addition, a moving average that corresponds to the moving min-max window can be computed and displayed along the middle of the bandwidth. Figure 16.10 shows a graph taken from van Geert and van Dijk (2002) in which they showed the developmental trend for one child's acquisition of spatial prepositions. They used a time window of 18 days across a 10-month period. The graph shows that there is a moderate sized bandwidth in the beginning of the trajectory and then, after a couple of oscillations, a

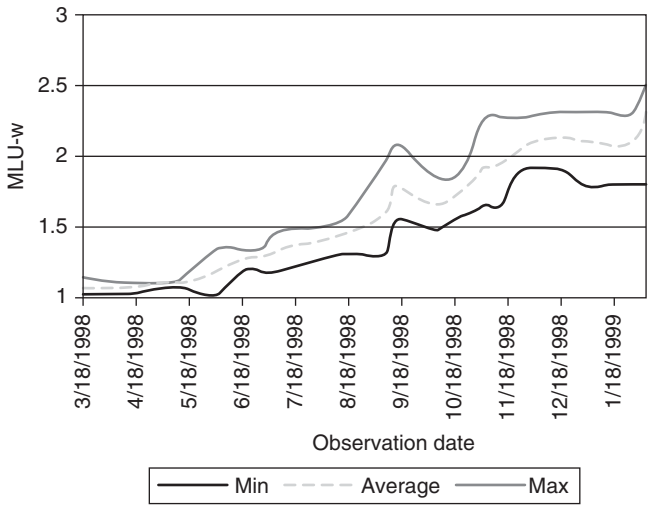


Figure 16.10 Moving min-max graph representing one child's acquisition of spatial prepositions (time window of 18 days, last window, 15 days) From van Geert, P., & van Dijk, M. 2002). Focus on variability: New tools to study intra-individual variability in developmental data. *Infant Behavior and Development, 25*, 340–370.

large increase in the bandwidth is maintained until the end of the observation period. The authors interpreted this plot as suggesting a developmental transition (one that showed the characteristics of a phase transition) in the use of spatial prepositions. For a more detailed examination of the values within the bandwidth, the authors also propose using an "altitude line graph," which simply depicts intermediate values as well (e.g., the second highest and second lowest values, percentiles) with a line connecting these values. The result is a plot that resembles the contour lines in a geographical relief map. A number of other variations of these variability charts can be found in van Geert and van Dijk (2002).

To explore developmental trends in individual or group data, developmentalists commonly plot idealized or processed estimations of a data series (e.g., regression models, smoothing techniques) that obscure information on variability (this is what they are designed to do: smooth out fluctuations). In general, developmentalists constrain themselves to a very small repertoire of smoothing techniques and, thus, lose potentially critical information about the characteristics of transitions and the idiosyncratic nature of development (van Geert & van Dijk, 2002). Van Geert and van Dijk (2002) argued for the application of more sophisticated smoothing models "which follow the actual rise and fall of the data as faithfully as one wishes" (p. 351). They provide alternatives, many of which are options available in commonly used statistical packages including SPSS. Some of these techniques include spline models and local polynomial regression models, or loess smoothers, which allow researchers to detect fluctuations and nonlinear trends in their data sets (Simonoff, 1996; van Geert & van Dijk, 2002).

To complement the impressions gained from the graphical developmental profiles, DS researchers may use simple statistics such as within-subject measures of variance (e.g., standard deviations) and correlations to track increases in variability across developmental transition points. The well-known standard deviation (SD) measure is sometimes problematic, however, because of its sensitivity to the mean (i.e., if distributions are censored at a low score, the SD tends to increase when the mean increases), making SDs difficult to compare across individual subjects or samples. To address this limitation, another less common measure of variability can be used: the coefficient of variation (CV; van Geert & van Dijk, 2002). The CV is simply the SD divided by the mean and, although it too has limitations, it can be used as an alternative to the SD.

Skewness is another easily calculated measure that provides a great deal of information about the pattern of variability in a given trajectory (van Geert & van Dijk, 2002). A skewed distribution is one potential indicator of bimodality, one of the catastrophe flags previously mentioned. In DS terms, bimodality can provide information about the extent to which a particular skill has been acquired or a pattern has been consolidated. For example, in the context of acquiring theory of mind (ToM), a 3.5-year-old child who is just beginning to understand that others may hold false beliefs about the child will likely show very few correct responses to ToM tests over several weeks of repeated testing; these will show up as positive outliers. In general, the child will still be responding "incorrectly," as if she has no false belief understanding; thus, the distribution will be positively skewed. At the age of 4, however, this child will likely show a negatively skewed distribution of responses—her ToM skills will have consolidated and now only very rarely will she respond as if she did not understand that others' can hold false beliefs.

A similar example might be used for thinking about children in treatment. For instance, at first, a child undergoing cognitive-behavioral therapy for problems with anxiety may respond to frightening or challenging situations with predominantly avoidant strategies and very few proactive, assertive behaviors (i.e., the distribution will be positively skewed, with most behaviors falling toward the avoidant end of the continuum and a few outliers falling toward the assertive end). But if treatment is successful, this child may end therapy showing a pattern of responses that is negatively skewed. The point at which the distribution shifts from being positively to negatively skewed may provide key information about the timing and profile of change

across treatment. From a DS perspective, it is likely that this child's distribution of avoidant-assertive scores from pre- to posttreatment will shift from positively skewed to bimodal to negatively skewed—that is, at no point will he exhibit responses that show a normal distribution. This pattern would suggest an abrupt shift (a phase transition) in response styles may be related to treatment success. The main point here is that instead of trying to normalize distributions at any stage, information about skewness and variability can provide insights about the nature of transitions and developmental change.

Standard statistical packages (e.g., SPSS, SAS) provide tests for assessing skewness. A Kolmogorow-Smirnov test provides a statistic for sample sizes (or number of observations) greater than 50 and the Shapiro-Wild test for sample sizes less than 50 (van Geert & van Dijk, 2002). In addition, van Geert and van Dijk (2002) described bootstrapping methods that provide researchers a random sample with which to compare statistically significant differences in skewness values across a given developmental trajectory.

Finally, individual growth curve modeling procedures can be applied to examine different profiles of change on both the individual and group level. This method is familiar to many developmentalists; it has been applied to examine different trajectories of antisocial behavior (e.g., Nagin & Tremblay, 1999), infant vocal development (Hsu & Fogel, 2001), infant-mother interactions (vanden Boom & Hoeksma, 1994), as well as many other domains. Several easily accessible computer programs are available to run these modeling procedures including multilevel analysis and hierarchical linear modeling (HLM; Willett, 1997).

Latent Class Analysis

Descriptive developmental profile analyses, as we have described them, are generally restricted to continuous one-dimensional data. Categorical and nominal data are difficult to use with these approaches (without somehow transforming them into continuous data). In this regard, combining different types of developmental profile analyses with methods that can capture content-specific changes in categorical or ordinal variables may be important. Another elegant option for investigating the bimodal properties of a distribution is offered by van der Maas (1998): latent class analysis (LCA). This technique is appropriate for analyzing categorical data. A number of developmental psychopathologists familiar with modeling procedures such as structural equation modeling (SEM) and HLM may also be aware of this method. LCA is conceptually similar to cluster analytic procedures in that it identifies subgroups or types. But it has additional power because it also provides a maximum likelihood estimation and is adaptable to different data structures with no a priori assumptions about the distributions.

As a cognitive developmentalist, van der Maas (1998) was particularly interested in this method for modeling the bimodal response distributions identified on various cognitive tasks at developmental transiton points or across various contexts (Quinlan, van der Maas, Jansen, Booij, & Rendell, 2007; van Rijn, Someren, & van der Maas, 2003). He argued that cognitive strategies can be best understood as attractors that are more or less available at different ages and in different contexts. In his approach, response patterns to multiple tasks are analyzed in terms of a cusp catastrophe model and multistable states (bimodality) are identified through LCA procedures. By presenting several examples of the application of rule and strategy detection in multiple domains, van der Maas (1998) showed that LCA may be one of the more promising psychometric models for identifying attractors and multistable states with categorical data.

Dynamic Growth Modeling

A group of scholars from the Netherlands can be credited for having pioneered the use of dynamic growth models in the study of cognitive developmental transitions (e.g., van der Maas, 1998; van der Maas & Molenaar, 1992; van Geert, 1994, 1995ab, 1997). The class of techniques advocated by these researchers is often grouped under the heading *nonlinear dynamics*, in reference to the modeling techniques and equations that are associated with this branch of mathematics. Although the techniques have often been applied to real-time data, these scholars explore various applications to developmental modeling instead.

Dynamic growth modeling was developed to simulate change over time (or growth) using logistic difference equations. Van Geert (1994) has used this procedure to model the processes underlying stage-like transitions in the growth of syntactic forms. The basic DS premise of van Geert's technique is that development of cognitive capacities is much like the self-organized proliferation of multiple species over the course of evolution. Van Geert models cognitive growers that constitute a complex system of intraindividual and environmental relations. Like the real-time coupled equations described earlier in reference to Gottman's work, the modeling procedure is realized with iterative equations in which behavioral states are updated by the amplifying and dampening forces inherent in the system's (i.e., the child's) experiential history combined with current external (contextual) resources and limitations. The

growth models depict the kinds of nonlinear developmental profiles predicted by stage theorists (e.g., Piaget and Vygotsky). They also emphasize the use of graphical procedures to plot empirical data from longitudinal studies of children's cognitive skill acquisition to match these empirical profiles to the data from simulations derived from the equations. The mathematical formulations used in these models are common in other disciplines; what makes these models unique is the application of both psychological and DS concepts to identify the mechanisms of growth that model observed developmental profiles. Another critical strength of this approach is that the process of developing simulations forces the researcher to specify the null hypothesis explicitly. The conventional a priori comparison between the hypothesized result and a lack of any effect is replaced by a specific null hypothesis of an alternative pattern of results.

More recently van Geert and his colleagues have broadened their cognitive developmental focus and have become interested in modeling children's peer play interactions (Steenbeek & van Geert, 2005, 2007, 2008). This new modeling endeavor may be more relevant to developmental psychopathologists because of its content (i.e., peer interactions), but more importantly, it may prove more generalizable to a large body of social interaction problems that developmental psychopathologists are interested in tackling.

Similar to previous endeavors, the procedure begins with a theoretical model. In this case, Steenbeek and van Geert (2005, 2007) developed a theoretical model that stipulated that differences in peer interactions with rejected versus popular children can be understood as a function of the following reciprocally interacting factors: each child's goals (autonomy and involvement), the drive that results from the extent to which these goals are met, the emotional appraisal at each given moment, and the resulting emotional expression and behavior. These theoretical factors are then formalized into a mathematical model that stipulates the mechanism differentiating rejected and popular children's dyadic interactions. Because this is a DS-based model, the mathematical formulation necessarily includes reciprocal, iterative processes and feedback cycles within and across occasions, as well as sensitivity to random or chance variations, and the potential for nonlinear results. Next, the simulation is run repeatedly and the results of the simulation are compared to theoretical expectations. Finally, the results of the simulation are compared to empirical data from real dyadic interactions.

The model includes a number of input parameters that can be set at the outset. These values can be start values for a particular variable or the strength of the connection between two variables. Rejected and popular children, for example, are assigned different preference values according to the original theoretical model. Figure 16.11 is a representation of the simulation model with all of the reciprocal and iterative processes depicted by arrows. Table 16.3 shows the different parameters and what values can be manipulated at the outset. The table also shows the parameter

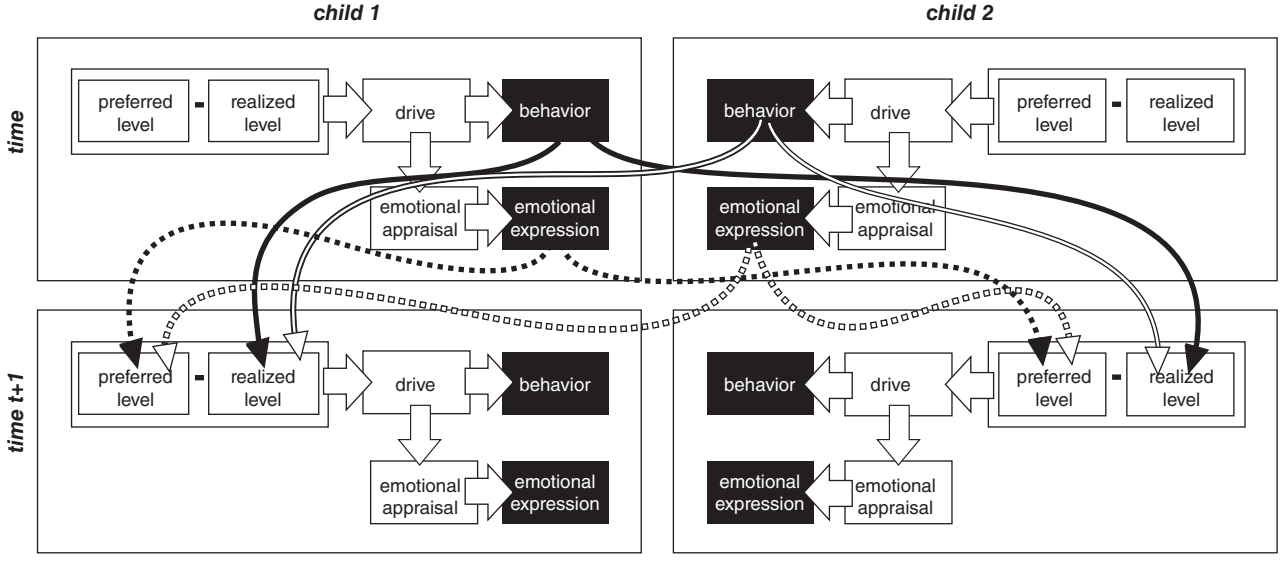


Figure 16.11 Steenbeek and van Geert's (2005) peer interaction simulation model. The first row of boxes represents the first moment in time, *t*, and the second row represents the second moment, $t + 1$. Thin arrows represent the iterative feedback components (the output of one iteration is the input for the next). *Source:* Steenbeek, H., and van Geert, P., 2005. A Dynamic Systems Model of Dyadic Interaction during Play of Two Children. *European Journal of Developmental Psychology*, *2*(2), 105–145.

TABLE 16.3 Parameter profiles for various types of dyads

Type of dyad	*Rejected dyad*		*Average dyad*		*Popular dyad*	
Status of child	*Rejected*		*Average*		*Popular*	
Status of play partner		*Average*		*Average*		*Average*
1 Concerns	I *much stronger* than A	I *little bit stronger* than A	I *stronger than* A	I *stronger than* A	I *little bit stronger* than A	I *much stronger* than A
2 Influence of behaviour on realization of concerns	I = average A = *high*	I = *high* A = average	I = average A = average	I = average A = average	I = *high* A = average	I = average A = *high*
3 Relation between emotional appraisal and emotional expression	Positive = moderate Negative = *difficult*	Positive = moderate Negative = moderate	Positive = moderate Negative = moderate	Positive = moderate Negative = moderate	Positive = moderate Negative = moderate	Positive = moderate Negative = *difficult*
4 Influence of emotional expression on preference of concerns	Positive = *big* Negative = *big*	Positive = average Negative = average	Positive = average Negative = average	Positive = average Negative = average	Positive = average Negative = average	Positive = *big* Negative = *big*
5 Nonintentional principles of behaviour	Continuity = average Symmetry = *high*	Continuity = average Symmetry = *low*	Continuity = average Symmetry = average	Continuity = average Symmetry = average	Continuity = average Symmetry = *low*	Continuity = average Symmetry = *high*

Notes: [a]The table can be read as follows: a child with a rejected status that plays with a play partner with an average status (rejected dyad; upper left of the table) has as setting for the first input parameter group concerns that his/her concern involvement is much stronger than his/her concern autonomy, which is expressed as "I *much stronger than* A". [b]I = concern involvement, A = concern autonomy. The average dyad has standard settings (average, moderate), Everything that differs from these standard settings is printed *in italics (high, low, difficult, big)*. [c]Information about the corresponding ranges of numerical values of these settings of input-parameter groups for children of different statuses in the context of playing with a play partner with a different status can be found in Appendix 2.

Source: Steenbeek, H., and van Geert, P. (2005). A dynamic systems model of dyadic interaction during play of two children. *European Journal of Developmental Psychology*, *2*(2), 105–145.

profiles for each type of dyad (the various combinations with rejected, popular, and average children). The model is run, for example, 1,000 times with the particular inputs specified in Table 16.3 for children with different sociometric status.

One of the unexpected results that emerged from running the simulations was that, contrary to what previous literature suggested, the data representing rejected children playing with normal-status children showed more positive emotional expressions than that for popular children with normal-status children (Steenbeek & van Geert, 2005, 2008). When these simulation results were compared to actual empirical data, the results matched (statistical tests described by the authors in detail were run to compare the simulated and empirical results). The authors concluded that, because rejected children seldom had a chance to play with other children in general, the new experience of playing with someone else led to the expression of more happy emotions than expected. The popular children, on the other hand, were not as pleased with the experience because they were not playing with their regular friends and had plenty of opportunities to play with other children.

The authors suggest that the modeling results are consistent with contextualist, functionalist, and dynamic systems perspectives. Indeed, their models remain true to fundamental DS assumptions. Broadly, the simulation technique demonstrated that emotions and behavior:

> . . . Are not a function of] personality characteristics that are relatively independent of the contexts and of the local and temporal realization of the concerns . . . [instead, the findings] emphasize the importance of iterative and interacting processes that take place in real time. It overthrows the classical distinction between independent and dependent variables in favor of a process of mutually interacting and simultaneous processes. (Steenbeek & van Geert, 2005)

Van Geert (1998) emphasized the critical role mathematical modeling plays in developmental research: "In order to find out the implications of theories, they have to be transformed into mathematical models that capture the major dynamic principles of such models and that can be used to explore the range of developmental trajectories under all possible or likely parameter conditions" (p. 155; also see Newtson, 1994; van Geert, 1994). But despite van Geert's concerted efforts to make his approach accessible, its impact on developmental psychology and developmental psychopathology may be limited because of its use of mathematical procedures that are daunting to most psychologists. More importantly, a great deal of the developmental phenomena that researchers are interested in examining are not conducive to this sort of modeling because they are not easily quantified as continuous variables. Also, regardless of the domain in which this method is applied, and as with all simulation techniques, the correspondence of the parameters in the model with genuine psychological mechanisms is often difficult to evaluate and runs the risk of seeming arbitrary. Nevertheless, this modeling technique and others may prove useful for developmental psychopathologists who have very precise hypotheses that can be faithfully translated into mathematical functions that specify mechanisms of change, influence, and interaction.

Connectionist Modeling

Connectionist and dynamic systems theories have separate histories, but share a great deal of commonalities (Spencer & Thelen, 2003). Although it is not quite accurate to characterize connectionism as a dynamic systems methodology, we would be missing an important contribution if we were not to include this modeling approach. Many (but not all) connectionist models are indeed dynamic systems and researchers working with these models often turn to concepts such as attractors, bifurcations, and chaos to understand their properties (Smith & Samuelson, 2003).

Connectionism is often characterized as a framework for simulating idealized brain-like, or neural network, processing. This is because connectionist modeling starts with the propagation of activation across a network of abstract units that can be hypothetically likened to networks of neurons that fire or do not, depending on the context. Knowledge, representation, or meaning is understood as being stored not in one or two units, but in connection weights between processing units. Learning and development is evidenced by changes in connections between these units. These changes result from statistical regularities in the input to the network and these regularities are governed by the Hebbian learning principle: units that fire together, wire together. Thus, "the connection weights between layers [of units]—the response of the network to a specific input—depend on the statistical regularities in the network's *history* of experiences" (Thelen & Smith, 1998, p. 580). Through their own real-time activity, without any additional intervention, connectionist networks change their own connections and, thus, teach themselves. One of the best-known examples of a connectionist model is Rumelhart and McClelland's (1986) demonstration of a network that acquired irregular and regular forms of past-tense, and overgeneralized (like real children were thought to do) the irregular forms. Their breakthrough was to show that a network could behave as if it was

governed by higher order rules with no such rules having been prespecified.

In most connectionist models, activation among units is a continuous temporal process. Differential equations are used to simulate this continuous activation process such that weighted connections link the rate of change in one variable to the inputs received from the other units (e.g., Munakata & McClelland, 2003). In many recent models, the activation process is assumed to be nonlinear. A simple example is provided by a nonlinear adjustment to McClelland's (1979) cascade model:

$$\frac{dneta_i}{dt} = k(\Sigma a_j w_{ij} - a_i)$$
$$a_i = f(net_i)$$

where a represents the activation of receiving unit i, a_j is the activation of sending unit j, w_{ij} represents the connection weight from unit j to unit i, and k is a time constant that sets the rate of change. $neta_i$ refers to the net input to unit i, and f is a nonlinear function (Munakata & McClelland, 2003).

In terms of development, connectionist simulations based on variations of the nonlinear differential equation have been developed to understand category and word learning (Plunkett, 1993; Smith, 1993, 1995), children's knowledge of dimension words and their selective attention to each dimension (Gasser & Smith, 1996; Smith, 1993), the development of face recognition (Johnson & Morton, 1991), and the A-not-B error (Munakata, 1998), among others. In terms of the A-not-B error, Thelen and colleagues (Thelen, Schoener, Scheier, & Smith, 2001; Smith & Thelen, 2003) extended past connectionist models by applying the dynamic systems principles of multicausality and nested time scales and developed a dynamic field model. They incorporated the idea that instabilities in network activities (i.e., variability) index qualitative changes in systemic behavior. Spencer and Schoner (2003) summarize this dynamic connectionist model: "Instabilities disrupt the one-to-one input–output mapping and make dynamic fields nonstandard neural networks" (p. 406).

Connectionist modeling, like other dynamical modeling techniques listed, requires either a great deal of technical expertise or collaboration among researchers with varying talents (e.g., computer programming, mathematics, developmental theory). We are aware of very few efforts in developmental psychopathology to apply this type of simulation procedure, but there may be a number of phenomena in the field that could benefit from such an approach. For example, different types of friendship formations on the playground may be modeled through connectionist procedures. Children seem to self-organize into coalitions or cliques very quickly at the beginning of a school year. These emergent groupings may be modeled as networks of units that become strengthened over time. Different types of coalitions (e.g., bullies and victims versus supportive friendship relationships) may exhibit unique properties (e.g., bullies/victims may be less stable, more perturbable, and they may be less likely to include new members than other types of friendships). These unique properties may suggest adjustments to existing theoretical models of friendship formation and even have the potential to suggest change mechanisms that may be targeted through intervention.

Readers interested in applying connectionist modeling are directed to *Rethinking Innateness: A Connectionist Perspective on Development* (Elman et al., 1996) and the companion book *Exercises in Rethinking Innateness: A Handbook for Connectionist Simulations* (Plunkett & Elman, 1997). These volumes provide a thorough primer in connectionist modeling and give novices an opportunity for hands-on practice. The authors make important distinctions between mechanisms of change and development and the content of those changes. Of special concern is the attempt to understand emergence and nested interactions at multiple time scales. The exercise book is a practical step-by-step manual that includes computer software and several example simulations that model, for example, stage-wise development and how children learn the past tense.

Catastrophe Modeling

This is a mathematical procedure wherein several control parameters can be used to account for discontinuous change in one of seven topological forms (catastrophe models; Thom, 1975; Guastello, 1995). As such, it has some appeal for developmental psychopathologists interested in incorporating insights from developmental stage theories or modeling nonlinear changes in behavior due to treatment or trauma. These models show how each of the possible combinations of control parameters result in different values of a dependent variable. The potential values of the dependent variable are represented on a plane, much like a state space. Nonlinear shifts can be depicted as a fold or curl in the plane showing where behavior changes suddenly rather than continuously.

The simplest and most commonly used catastrophe model is a cusp catastrophe, as depicted in Figure 16.12. The top surface of this three-dimensional figure is the

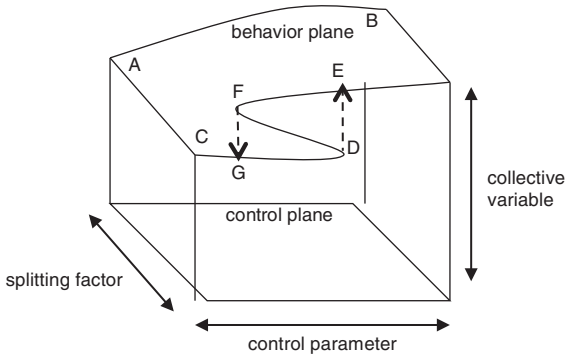


Figure 16.12 Basic cusp catastrophe model.

behavior plane, which is made up of all the possible values of the response variable (i.e., collective variable). The location of a point in the behavior plane is predicted by the values of the control parameter and the splitting factor (the axes on the bottom plane called the control plane), which are equivalent to independent variables in these models. The top left position on the behavior plane (point A), for instance, is a combination of a low value of the control parameter and a low value of the splitting factor. Keeping the splitting factor constant for the moment, follow the movement from point A to point B. Here we see that there is a linear response in behavior as the control parameter increases continuously. However, when the values of the splitting factor are high, there are some values of the control parameter that result in two possible locations on the behavior plane (points D and E or F and G). This cusp fold in the behavior plane is the region that describes the nonlinear response dynamics of the system. As the control parameter increases from the lowest value at point C, the behavior increases through points G and D. However, at point D the collective variable value suddenly jumps to point E on the plane. When the control parameter decreases from its maximum value, behavior passes through point E to point F in a linear response. Now at point F, behavior suddenly jumps down to point G, a much lower value for the collective variable. This difference in response depending on whether the control parameter is increasing or decreasing is one of the unique features represented by cusp models and exemplifies the process of hysteresis.

For example, consider the relationship between frustration and anger. The first step in catastrophe modeling is to identify that a catastrophe exists. The catastrophe flags mentioned earlier are used for this purpose. Presence of hysteresis, divergence from linear response, sudden jumps, bimodality, anomalous variance, and critical slowing down can all serve as flags. For the current example, suppose that through experimentation a researcher discovers a bimodality in the distribution of anger responses to frustration. In cases where frustration is moderate, anger is either high or low but never in between. This would be an indication that a cusp catastrophe could be an appropriate model. Moreover, suppose this bimodality only occurs in high reward contexts, and that frustration and anger have a positive linear relationship in low reward contexts. Thus, for this example, anger is the collective variable of interest, frustration is the control parameter, and amount of reward in the context is the splitting factor (Figure 16.13). In a low-reward task (e.g., no motivational incentive other than following instructions), as frustration increases, the anger response would increase gradually (path A to B in Figure 16.13). In a high-reward task (e.g., in a gambling context), as frustration increases, the anger response would break from linearity at point D. Anger would suddenly jump to a higher level at point E. If frustration continues to increase, there would again be a linear increase in anger. However, frustration would have to subside well below the levels at which the previous jump occurred (point F) before anger decreased nonlinearly to its former level.

The next step of the modeling procedure is to fit the model to empirical data, much like linear regressions are fit to data. Catastrophe models have been used to model diverse developmental transitions such as conservation of liquid (van der Maas & Molenaar, 1992); acquisition of syntax (Ruhland, & van Geert, 1998); infant reaching with grasping (Wimmers, Savelsbergh, van der Kamp, & Hartelman, 1998); dating (Tesser & Achee, 1994); blame responses of victims of assault (Lanza, 1999); adolescent smoking (Byrne, Mazanov, & Gregson, 2001); alcohol use (Clair, 1998); and anxiety in preuniversity students (Haslett, Smyrnios, & Osbourne, 1998). These and other

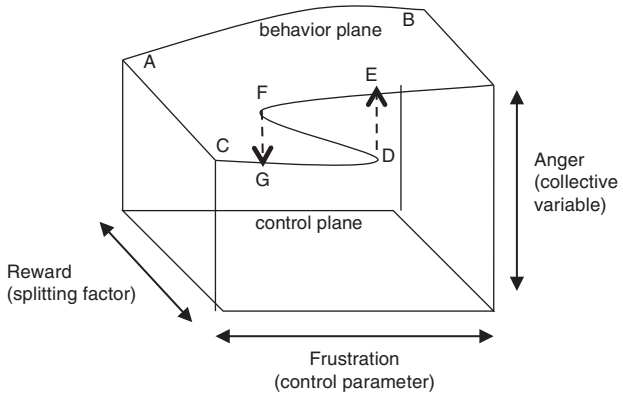


Figure 16.13 Cusp catastrophe model representing the relation between anger and frustration in different contexts.

examples indicate that, despite the difficulty in specifying or measuring control parameters, as discussed earlier, important insights about the profiles and mechanisms of change can be gained. Catastrophe theory and models will likely become more applicable as more researchers address the nonlinearities in their data and work to measure hypothesized control parameters.

STATE SPACE GRID ANALYSIS: A GRAPHICAL AND STATISTICAL MIDDLE ROAD

The various DS techniques introduced thus far have considerable potential for addressing some of the analytic challenges faced by developmental psychopathologists. However, we have also pointed out some obstacles for implementing these techniques. In general, most techniques require continuous data, whereas ordinal and categorical variables are more common in developmental psychopathology, especially in observational studies. In addition, many of the techniques are either solely descriptive, preventing researchers from testing the strength and reliability of their findings, or require complex mathematical procedures that may be inaccessible or irrelevant to most developmental psychopathologists. Recently, hybrid strategies have been developed, providing a middle ground by combining graphical techniques that capture the descriptive richness of DS concepts with simple statistical procedures that stay true to systems assumptions (Hollenstein, 2007, 2012, 2013; Lamey et al., 2004; Lewis et al., 1999). The state space grid method is a graphical and statistical strategy that links the analysis of real- and developmental time patterns and allows for the identification of individual and group differences. Thus, the flexibility of this methodology has proven valuable for developmental psychopathologists. We begin by describing this technique and the various measures that can be derived for statistical analysis.

State Space Grids

Recall that DS theorists use the concept of a *state space* to represent the range of behavioral habits, or attractors, for a given system. In real time, behavior is conceptualized as moving along a trajectory on this hypothetical landscape, being pulled toward certain attractors and freed from others (Figure 16.1). Based on these abstract formalizations, Lewis, Lamey, and Douglas (1999) developed a graphical approach that utilizes categorical time series structured as two ordinal variables that define the state space for any particular system. Though these state variables can represent two measures of one individual's behavior (e.g., Lewis et al., 1999, Lewis et al., 2004) so far the most frequent use of state space grids has been the analysis of dyadic interactions (Branje, 2008; Cerezo, Trenado, & Pons-Salvador, 2012; Connell, Hughes-Scalise, Klostermann, & Azem, 2011; Dishion et al., 2004; Gardner & Wampler, 2008; Granic, Dishion, & Hollenstein, 2003; Granic & Lamey, 2002; Granic et al., 2003, 2007; Granic et al., 2012; Hollenstein, 2007; Hollenstein & Lewis, 2006; Hollenstein et al., 2004; Lunkenheimer, Albrecht, & Kemp, 2012; Lunkenheimer et al., 2001, 2012; Mainhard, Pennings, Wubbels, & Brekelmans, 2012; Moore et al., 2012; Ribeiro, Bento, Salgado, Stiles, & Gonçalves, 2011; Sravish, Tronick, Hollenstein, & Beeghly, 2013; van Dijk, Hunnius, & van Geert, 2012; van der Giessen, Branje, Frijns, & Meeus, 2013; van der Giessen et al., 2015). With this application, a dyad's trajectory (i.e., the sequence of behavioral states) is plotted as it proceeds in real time on a grid representing all possible behavioral combinations. Categories of one dyad member's (e.g., parent) behavior forms the columns on the x axis and the other member's (e.g., child) behavior forms the rows on the y axis. Each cell of the grid is the joint x–y state (e.g., parent–child) and plot points in these cells represent the occurrences of these joint states, and their durations if applicable. Moreover, a trajectory is drawn through the successive dyadic points in the temporal sequence they were observed. Any time the behavior changes a line is drawn connecting the new point and the previous point. For example, a hypothetical trajectory representing 15 seconds of coded behavior is presented in Figure 16.14. With the duration of each joint state represented by the size of the plot point and the direction of each transition indicated by an arrow, this sequence begins with the parent and child in conflict (shared negativity/hostility, bottom left cells) and ends in repair (shared positivity, top right cell).

The SSG methodology can tap both the content of interactions (e.g., mutual hostility, permissive parenting) and the structure of these interactions. When we refer to structure, we mean the patterning of behavior, regardless of content, such as the relative flexibility versus rigidity of interaction patterns (Hollenstein, Lichtwarck-Aschoff, & Potworowski, in press). With SSGs, we are able to examine whether behavior clusters in very few or many states (i.e., cells), or regions (i.e., a subset of cells) of the state space. We can also track how long the trajectory remains in some cells but not others, and how quickly it returns to particular cells. If a dyadic trajectory remains in a small number of cells, and makes very few transitions between cells, this system may be thought of as rigid, inflexible, or stuck. In contrast, a trajectory that moves around to many cells in the state space grid and makes frequent changes between

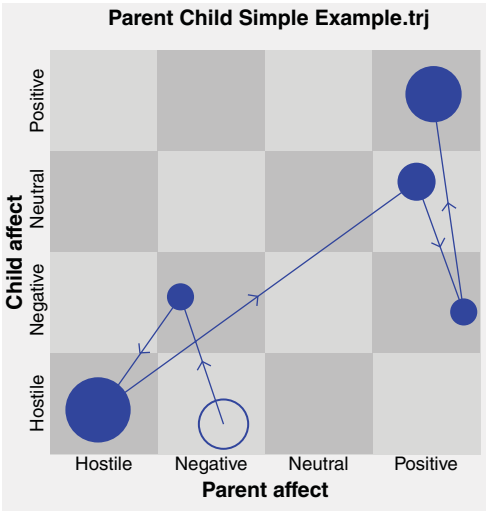


Figure 16.14 Example of a state space grid with a hypothetical trajectory representing 10 seconds of coded behavior, one arrow head per second. Plotting begins in the lower left part of the cell and moves in a diagonal as each second is plotted, ending in the upper right. See footnote 1.

these cells may indicate a highly flexible, or variable, system. We can identify attractors as those cells to which behavior is drawn repeatedly, in which it rests over extended periods, and/or to which it returns quickly. Moreover, as discussed in the following sections, a range of variables that capture the relative stability of particular attractors may be derived from SSGs and these values can be tested statistically for changes in real and developmental time.

A major advantage of SSGs is that they provide an intuitively appealing way to view complex, interactional behavior; thus they are first and foremost a useful tool for exploratory analysis. An examination of the heterogeneity of family interactions with aggressive children may help illustrate this point (Granic & Lamey, 2002). SSGs were used to explore differences in the parent–child interactions of "pure" externalizing children (EXT) and children comorbid (MIXED) for externalizing and internalizing problems. This study is useful not only for demonstrating how the grids work, but also to demonstrate design innovations based on DS principles that are useful with or without SSGs, in this case, a systematic perturbation.

Parents and clinically referred children discussed a problem for four minutes and then tried to "wrap up and end on a good note" in response to a signal (the perturbation) within the next two minutes. The perturbation was intended to increase the emotional pressure on the dyad, triggering a reorganization of their behavioral system. We hypothesized that, as a function of differences in the underlying structure of their relationships, EXT and MIXED dyads would be differentially sensitive to the perturbation and would reorganize to different parts of the state space. Prior to the perturbation, however, we expected dyads' interactions to look relatively similar. Separate grids were constructed for the pre- and postperturbation interaction sessions. For this study, the lines (trajectories) are less important to notice than the points that show clustering in particular cells. Figure 16.15 shows an example of an

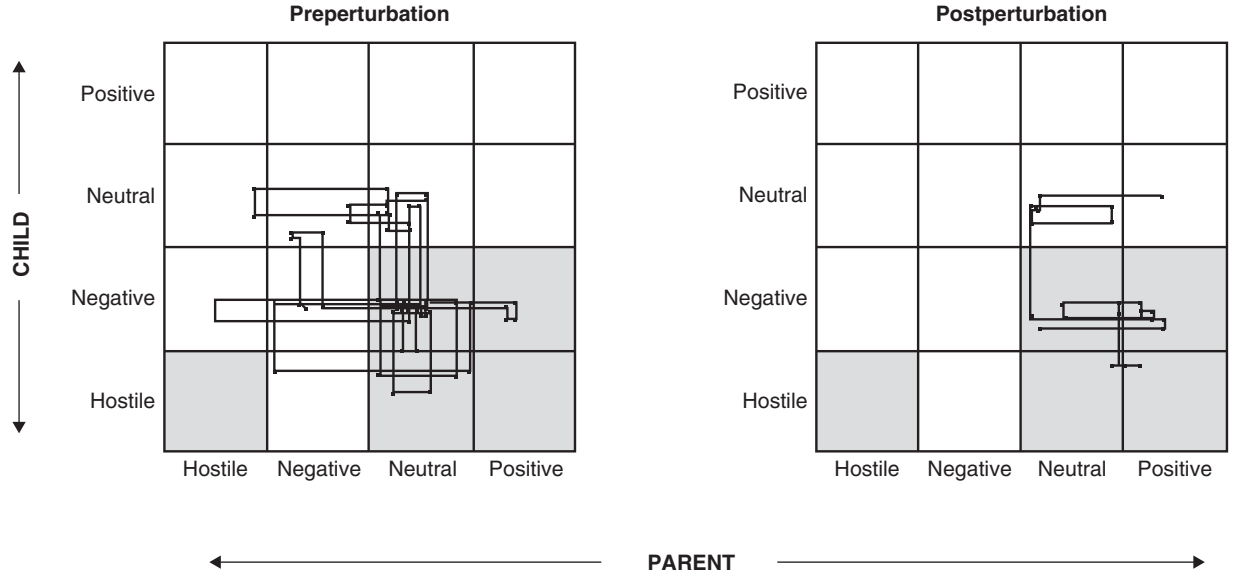


Figure 16.15 Pre- and postperturbation state space grids for an EXT dyad. *Source:* From Granic, I., & Lamey, A. V. (2002). Combining dynamic-systems and multivariate analyses to compare the mother–child interactions of externalizing subtypes. *Journal of Abnormal Child Psychology*, *30*, 265–283.

interaction between a pure externalizing child and his parent, pre- and postperturbation. As exemplified in these grids, EXT dyads tended to go to the permissive region (child hostile–parent neutral/positive) of the state space grid, as well as other regions (i.e., mutual neutrality and negativity), before the perturbation. After the perturbation, EXT dyads tended to remain and stabilize in the permissive region. Figure 16.16 represents the interaction of a MIXED dyad. Similar to EXT dyads, the MIXED dyads occupied the permissive region, as well as other areas, before the perturbation. But in contrast with the EXT group, MIXED dyads tended to move toward the mutual hostility, or mutual negativity, region of the state space grid after the perturbation. Granic and Lamey (2002) concluded that the perturbation was a critical design innovation that provided the means by which clinical subtypes could be differentiated.

Another contribution of this study was a more general one—the use of SSGs, with their rich case-by-case temporal narratives, provided a technique to further parse interaction processes that have been previously assumed to represent one coherent pattern. In this case, the coercive process (Patterson, 1982; Patterson, Reid, & Dishion, 1992; Snyder et al., 1994) was shown to constitute two separate microsocial patterns—two separate attractors on a state space (Granic & Patterson, 2006). Moreover, the conditions under which dyads would be drawn toward one region or the other were found to differ for subtypes. The use of SSGs to uncover heterogeneous processes may be relevant to a variety of phenomena in developmental psychopathology including variability in the real-time unfolding of attachment patterns (Coleman & Watson, 2000), in bullying interactions on the playground (Pepler, Craig, & O'Connell, 1999) and in parent–adolescent interactions during puberty (Granic, Dishion, et al., 2003; Granic, Hollenstein, et al., 2003).

State Space Grid Analysis: Within Grids

In general, there are two ways to use SSGs for analysis: deriving measures from one specific state space arrangement (within-grid analysis) and analyzing similarities and differences between grids, such as with longitudinal designs (between-grid analysis). Within a given SSG, patterns can be quantified and used as variables for statistical analyses. Measures that capture the temporal and spatial patterning of behavior have been developed for time-based (e.g., second-by-second coding, daily diary entries) as well as event-based (e.g., conversational turns) data (Hollenstein, 2013). These variables can all be obtained via the SSG software program, GridWare (Lamey et al., 2004), available for free on the Internet (www.statespacegrids.org) and may be used to map intra- or interpersonal behavioral trajectories. Here, we highlight two within-grid analysis approaches relevant to the development of psychopathology (for a comprehensive review, see Hollenstein, 2013): attractor (or content) analysis and whole-grid (or structural) analysis. In general, long durations or frequent recurrences of

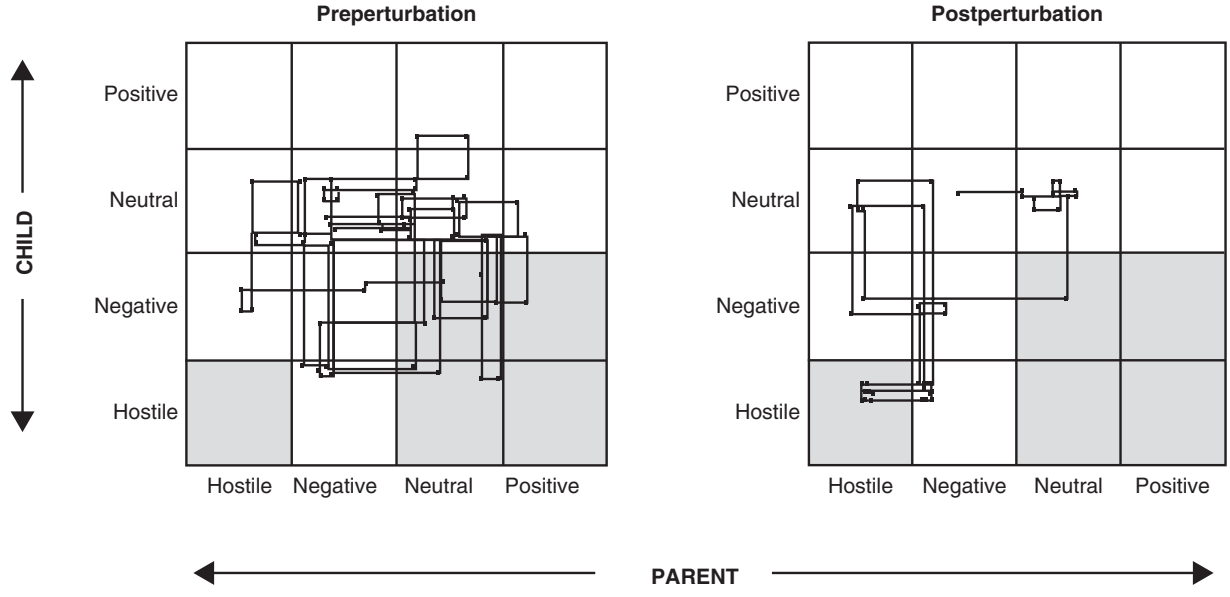


Figure 16.16 Pre- and postperturbation state space grids for a MIXED dyad. *Source:* From Granic, I., & Lamey, A. V. (2002). Combining dynamic-systems and multivariate analyses to compare the mother–child interactions of externalizing subtypes. *Journal of Abnormal Child Psychology*, *30*, 265–283.

behavior in a particular cell or region suggest an attractor on the state space, and these hypothetical attractors can be compared and tested within individuals across development as well as between individuals.

Attractor analysis can be theoretically driven (expected attractor states are compared across subjects; e.g., Granic & Lamey, 2002) or empirically derived for each case (e.g., Lewis et al., 1999). Lewis et al. (1999) is still the best example of empirically derived attractors for analysis. First, attractor cells or regions were identified via a winnowing technique that used the individual cell durations. Through iterations removing one cell at a time, the number of possible attractor states was winnowed down to only those cells in which durations exceeded chance. Each trajectory was found to have at least one cell—and sometimes two cells—that qualified as attractors. Once identified, analytical options for theoretically and empirically derived attractors are the same. For within-grid analyses, this primarily consists of determining the *strength* or relative pull of the attractor on the system's trajectory. On top of relatively straightforward attractor indices such as cumulative, proportional, mean, or maximum durations in cells or groups of cells (i.e., regions) of interest, attractor strength can be measured via return time. Return time is the latency to return to a cell or region following an event in that cell or region. This can be measured in units of time, number of events, or number of unique cells visited en route. For example, Figure 16.17 shows three grids with attractor regions shaded in gray. Grid B shows a duration in cells 2/2 (cell labels follow the x/–y convention) and 2/3 and a very low duration in cell 1/3. In grid C, cell 3/1 shows a high mean duration (each time behavior goes to that cell, it tends to stay there for some time) whereas cell 1/1 in that same grid shows a low perseverance value. Finally, grid B shows a very low return time for cell 2/2 (every time behavior leaves that cell, it returns in approximately one turn), but a high return time for grid A, cell 1/0. Thus, multiple attractor strength indices can be used for subsequent statistical analyses.

Whole-grid analysis can be used to derive measures of the overall variability or structure of trajectories on the SSG. There are two classes of overall variability measures: dispersion-based and transition-based. Measures of dispersion capture the overall spread of the trajectory based on the number of unique cells visited. With duration-based data, this dispersion can also be calculated controlling for the relative durations in each cell—cells with longer duration count more in the index than cells with low durations. Thus, high levels of dispersion indicate a more even distribution of behavior across the entire state space. Transitions are somewhat independent of dispersion. A trajectory can have a high number of transitions (i.e., the number of lines in the trajectory on an SSG plot) among many cells or only a few. Thus, a simple count of the number of transitions (or transitions-per-minute for studies with trajectories of unequal length) can indicate the dynamic flexibility of the system, as complementary information to dispersion. As discussed earlier, another transition-based measure is entropy, which is based on the lag 1 event sequences—higher entropy indicates less predictability. Both dispersion and transition measures of dyadic variability have been shown to correlate with children's mood and behavior problems. Low variability, interpreted as low flexibility or high rigidity, in parent–child interactions has been associated with externalizing and internalizing problems in early childhood (Hollenstein et al., 2004), lack of response to treatment for externalizing problems (Granic et al., 2007), and maternal depression (Lunkenheimer et al., 2012). Returning to Figure 16.17, grids A and C show high dispersion, compared with grid B, and grid A shows a low stability 1 value, compared with grid B.

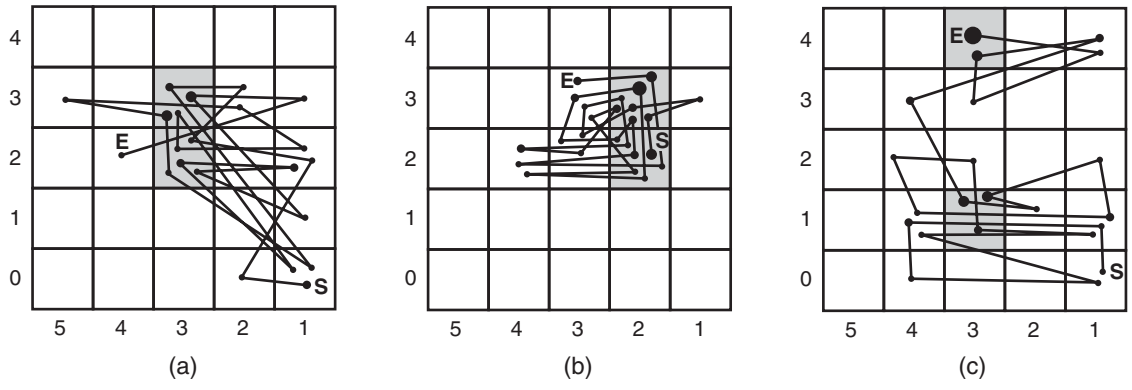


Figure 16.17 Examples of three state space grids from Lewis, Lamey, and Douglas (1999). *Source:* From Lewis, M. D., Lamey, A. V., & Douglas, L. (1999). A new dynamic systems method for the analysis of early socioemotional development. *Developmental Science*, *2*, 457–475.

State Space Grid Analysis: Between-Grid Analysis

After computing the within-grid parameters that are most relevant for a particular research question, longitudinal statistical techniques (most of which are quite familiar to developmental psychopathologists) may be applied. We recommend using these statistical procedures in such a way that maintains the integrity of the individual (or dyadic) case (e.g., growth clusters). However, multivariate analyses including one-way analyses of variance (ANOVAs), regressions, and SEM can just as easily be run on the grid variables. For example, we have shown over the course of five longitudinal observations that parent–boy variability (dispersion and transitions) peaked when the son was 13–14, consistent with the hypothesis that adolescence is a developmental phase transition of temporarily heightened variability in interpersonal interactions. The use of within-grid measures of repeated observations in this way is statistically relatively straightforward, thus we will not elaborate further here However, two between-grid techniques enable the kind of person-centered analyses of interest to system-minded researchers.

Lewis et al. (2004) developed two techniques to depict month-to-month stability and change in toddler's socioemotional habits. Observed once a month for 11 months (ages 14–24 months), each subject had 11 SSGs of the behavioral trajectories. The study was designed to test the hypothesis that the 18–20-month transition was a developmental phase transition—a temporary period of instability and fluctuation as the children developed from one stable behavioral pattern to another. Thus, the authors derived two indices from SSGs to see if the lowest month-to-month stability (i.e., greater change) occurred at 18–20 months. The first technique they used was to calculate the *inter-grid distance* score (IDS). Grid-to-grid Euclidian distance scores yield a global metric of the difference in behavioral landscapes from month to month, based on the sum of squared differences across all cell pairs. For each grid cell pair, the difference in duration values over two consecutive months is calculated, then squared, and then these values are summed for all cells. Next, the square root of this sum is taken as the distance score between the two grids. The second technique was to put all SSG cell variables for all participants at every month into one cluster analysis. Each SSG was then identified as a member of a homogenous cluster. Then, returning back to a case-by-case computation, a cluster change score was calculated: a value of zero was assigned if the cluster membership of one month was the same as the subsequent month and a value of 1 was assigned if the cluster membership changed from one month to the next. Thus, from 11 SSGs, 10 cluster change scores were calculated for each case. Both the IDS and cluster change score were highest during the 18–20-month period as hypothesized. Thus, SSG-to-SSG change can reveal patterns of development that go beyond using within-grid measures in standard statistical analysis.

Although the SSG method is clearly still in its early stages of development, we are encouraged by its potential. One of the important advantages to this technique is its inherent flexibility. At the very least, it is a visual, exploratory tool to develop and refine hypotheses. At best, this methodology provides a source from which novel, temporal-based, process-level predictors may be tapped and used to strengthen current models of normal and atypical development. Researchers are not limited to using continuous time-series, as is the case with many other DS methods. Categorical and ordinal data are also appropriate for this type of analysis. Also, the grids are malleable in that they can represent systemic behavior on the individual as well as dyadic level. In addition to the aforementioned examples, changes in peer, romantic couples, and sibling interactions, for example, can easily be tracked using SSGs. In fact, apart from the difficulties in visually representing the data, the variables derived from the grids can be extended past the two dimensions on which we have focused. For instance, SSGs have been used to analyze triadic (Lavictoire et al., 2012) and group (Martin et al., 2005) interactions.

Another benefit of this approach is the extent to which it remains user-friendly and does not require expertise in mathematical modeling. In addition, we have developed a SSG software program that will output grids, compute all the grid parameters mentioned previously, and export these measures for statistical analyses on these parameters: GridWare (Lamey et al., 2004), available for free on the Internet (www.statespacegrids.org).

FUTURE DIRECTIONS: IMPLICATIONS FOR CLINICAL RESEARCH

There are no limits to the diverse topics and varied problems that can potentially be addressed with the application of DS methods; clearly listing all of them here would be impossible. In this final section, we have chosen to limit our discussion to the implications for clinical research, an area in which many developmental psychopathologists share an interest. Because DS methods are specifically designed to capture change processes, and because the study of psychopathology often breaks down into the study of individual patterning, one of the most exciting potential

applications of DS methods may be in treatment research. Although randomized controlled trials have helped to identify the most effective interventions for a variety of childhood mental health problems, there remains variability in outcomes (e.g., Eyberg, Nelson, & Boggs, 2008; In-Albon & Schneider, 2007) and almost no understanding about the underlying processes and mechanisms of change (Hinshaw 2002; Kazdin, 2000, 2001, 2002, 2009). This problem highlights our lack of understanding of the change process itself. Information about the mechanisms responsible for the success of interventions is critical for guiding clinicians in making informed decisions about how to tailor interventions to different contexts and for unique individuals and families. Also, identifying mechanisms of change is a crucial step toward more effective program dissemination in community settings (Kazdin, 2000).

DS principles and methods should be able to provide a microsocial, process-level account of how family and peer relationships change over the course of treatment and why some may fail to do so (Gardner & Wampler, 2008; Granic et al., 2007; Lichtwarck-Aschoff et al., 2012). In the following, we present three hypotheses that can be generalized to any evidence-based intervention.

First, phase transitions in normative development may be critical to mark because they allow clinicians and researchers to more efficiently, and perhaps more successfully, access and manipulate mechanisms of change. There may be normative stage transitions in children's development (e.g., early childhood 3–5 years, early adolescence 11–14 years), as well as idiosyncratic transition periods (e.g., divorce or birth of a sibling) during which, the coordination among system elements begins to break down, previous attractors are destabilized, and new patterns have the potential to emerge (e.g., Lewis et al., 1999). During phase transitions, the system is much more open to environmental shifts, and seemingly small changes have the potential to radically alter the trajectory of relationships and individuals. As a result, prevention and intervention efforts that target youth mental health problems may have their maximal effect during these periods. Thus, based on DS ideas we would expect that clinical interventions and prevention efforts will be most effective if they are targeted at these sensitive periods. The same intervention targeted before or after a phase transition is hypothesized to be less successful. To test this hypothesis rigorously, it would be important to design treatment studies that examine the differential impact of the same evidence based intervention before, during, and after a recognized transition period.

Second, psychotherapy researchers suggest that, for improvements to be made, treatment must trigger a reorganization of affective, cognitive, and behavioral systems (e.g., Caspar, Rothenfluh, & Segal, 1992; Greenberg, Rice, & Elliott, 1996; Mahoney, 1991). To induce a major reorganization, "old patterns must be shaken loose or destabilized to allow for new configurations to emerge or to be discovered" (Hayes & Strauss, 1998, p. 940). Thus, "destabilization is viewed as a necessary and natural process that allows for growth and change" (p. 940). Although this destabilization period has been theoretically proposed, very few empirical studies have investigated this profile of change in therapeutic contexts partly because, until recently, we lacked the appropriate methodological tools for doing so (Cicchetti & Cohen, 1995). The study by Lichtwarck-Aschoff et al. (2012) discussed earlier explicitly investigated the destabilization profile in aggression treatment and found that children who improved through treatment showed characteristics of a reorganization in the parent–child dyad. In line with that study we expect that treatment gains—across a wide range of psychopathologies and interventions—will be evident only after a phase transition, operationalized as a significant increase in the variability of behavioral patterns. Without evidence of a destabilization period, treatment is expected to be less successful.

It is important to keep in mind that system destabilization, or a lack thereof, is part of a process description, revealing potential forms of treatment progressions. They are never the cause of success or failure of the treatment itself. Therefore, it is important that future efforts are targeted on unraveling the underlying causes of the reorganization that are triggered by the treatment perturbation. One way to do this would involve a model building and simulation approach (discussed in this chapter; e.g., Gottman, Murray, Swanson, Tyson, & Swanson, 2002; Van Geert, 1994; Van Geert & Lichtwarck-Aschoff, 2005), emphasizing the client's internal dynamics, together with the interaction with external influences such as treatment. A second way of understanding the causal mechanisms in terms of change processes is the investigation of (changes in) control parameters (Van Orden et al., 2009). A prime candidate for a control parameter within the context of treatment is motivation to change (Guastello, 1995, 2002; Hayes & Strauss, 1998; Kowalik, Schiepek, Kumpf, Roberts, & Elbert, 1997; Mahoney, 1991; Schiepek, 2009). Regarding organizational change, Guastello (1995, 2002) described pressure for change and resistance to change as two orthogonal control parameters. When resistance is low, even minor pressures to change (i.e., intervention efforts) may lead to gradual and small changes in behavior. When resistance is high, old patterns of behavior will not

change if pressure to change is low. However, when both resistance and pressure to change are high, radical, nonlinear change is likely to occur. This conception is in line with the struggle-and-work-through hypothesis (Chamberlain et al., 1984; Stoolmiller et al., 1993) where it has been demonstrated that families that show low resistance from baseline to termination are unlikely to show significant change and benefit from treatment (Stoolmiller at al., 1993). Hence, the time-serial investigation of changes in control parameters (e.g., motivation to change) in relation to structural changes in real-time behavior is needed to enhance our understanding of causal mechanisms producing successful treatment outcomes.

Finally, an implicit, if not explicit, assumption made by most clinicians and clinical researchers is that, when treatment works, family interactions change from being emotionally negative or angry to positive or supportive. But according to emotion theorists (e.g., Izard 1977; Magai & McFadden 1995; Tomkins 1963), the expression of anger or any other negative emotion is not pathogenic; all emotions are adaptive and important to express in appropriate contexts. It is the effective—flexible—regulation of these emotions that is critical for healthy development (e.g., Bonanno, Papa, Lalande, Westphal, & Coifman, 2004). Being emotionally flexible is the hallmark of healthy functioning at any age (Kashdan & Rottenberg, 2010). Being locked into any one emotional state is often the definition of certain psychopathologies: perseveration of fearful vigilance is the mark of anxiety, unyielding sadness is diagnostic of depression, and long-lasting elation is mania. Developmentalists agree that, in general, regulation skills are learned and practiced in the context of parent–child interactions. We go on to propose that flexibility in these interactions (i.e., the ability to shift from one emotional state to another, according to contextual demands) may be more important for healthy development than the complete avoidance of negative affect (see also Gottman & Notarius, 2000, for a similar suggestion for marital relationships). Indeed the study by Granic and colleagues (2007) discussed earlier showed that children that improved through treatment could be distinguished from the ones that did not based on the flexibility of their parent–child interactions. The improvers had changed their rigid interactions into emotionally flexible and reparative ones not simply into positive interactions. But flexibility can also be assessed on different levels. As an illustration, we are currently investigating whether flexibility is associated with positive treatment outcomes in an anxiety treatment for children on the basis of changes in the coordination of body movements in parent–child interactions (see for a similar approach Ramseyer & Tschacher, 2011). Here we expect that improver dyads show a more balanced body synchronization in contrast to a parent dominated coordination. In sum, parent–child and peer interactions are predicted to become more flexible as a function of successful treatment. Rigidity in parent–child and peer interactions on the other hand gives rise to a variety of cascading constraints that contribute to the emergence and maintenance of problem behavior.

These three hypotheses provide an illustration of how DS ideas and methodologies can promote the study of change processes in clinical research. Providing a finer degree of resolution than pre-post designs and thereby revealing predictors, moderators, mediators, and mechanisms of change (Hayes et al., 2007). The evidence-based treatment movement has set out to identify what works for whom by implementing randomized clinical trials. The ideas and methodologies described in this chapter offer the possibility of enriching this work to better understand the when, how, and why of change.

CONCLUSION

From the beginning of its establishment as a discipline, one of the core priorities in developmental psychopathology has been methodological diversity (e.g., Cicchetti & Cohen, 1995b; Cummings et al., 2000; Richters, 1997). One reason for encouraging this analytic pluralism is the recognition of the disparity between systems-based models of developmental psychopathology and the inadequate methodological tools that are available to test them (Richters, 1997). We have argued that DS approaches to development offer research methods that show greater fidelity to the complex, heterogeneous, temporal nature of developmental phenomena. Clearly no set of analytic methods can address the mismatch of methods and models entirely; thus, we are not arguing for the complete abandonment of well-established techniques. Instead, our purpose in providing a survey of DS methods is to encourage developmental psychopathologists to begin examining empirically questions that may have previously seemed out of analytic reach.

REFERENCES

Abraham, F. D., Abraham, R. H., & Shaw, C. D. (1990). *A visual introduction to dynamical systems theory for psychology*. Santa Cruz, CA: Aerial Press.

Allison, P. D. (1984). *Event history analysis: Regression for longitudinal event data*. Beverly Hills, CA: Sage.

Badalamenti, A. F., & Langs, R. J. (1992). The thermodynamics of psychotherapeutic communication. *Behavioral Science*, *37*, 152–180. doi: 10.1002/bs.3830370302

Bakeman, R., & Gottman, J. M. (1997). *Observing interaction: An introduction to sequential analysis* (2nd ed.). Cambridge, UK: Cambridge University Press.

Bergman, L. R., & Magnusson, D. (1997). A person oriented approach in research on developmental psychopathology. *Development and Psychopathology*, *9*, 291–319. doi: 10.1017/S095457949700206X

Bertenthal, B. (1999). Variation and selection in the development of perception and action. In G. Savelsbergh, H. van der Maas, & P. van Geert (Eds.), *Non-linear developmental processes* (Vol. 175, pp. 105–121). Amsterdam, The Netherlands: Royal Netherlands Academy of Arts and Sciences.

Blossfeld, H., & Rohwer, G. (2002). *Techniques of event history modeling: New approaches to causal analysis* (2nd ed.). Mahwah, NJ: Erlbaum.

Bonanno, G. A, Papa, A., Lalande, K., Westphal, M., & Coifman, K. (2004). The importance of being flexible: The ability to both enhance and suppress emotional expression predicts long-term adjustment. *Psychological Science*, *15*, 482–487. doi: 10.1111/j.0956-7976.2004.00705.x

Boyce, W. T., Frank, E., Jensen, P. S., Kessler, R. C., Nelson, C. A., & Steinberg, L. (1998). Social context in developmental psychopathology: Recommendations for future research from the MacArthur Network on Psychopathology and Development. *Development & Psychopathology*, *10*, 143–164. doi: 10.1017/S0954579498001552

Branje, S. J. (2008). Conflict management in mother–daughter interactions in early adolescence. *Behaviour*, *145*, 1627–1651. doi: 10.1163/156853908786131315

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design*. Cambridge, MA: Harvard University Press.

Burlingame, G. M., Fuhriman, A., & Barnum, K. R. (1995). Group therapy as a nonlinear dynamical system: Analysis of therapeutic communication for chaotic patterns. In F. D. Abraham & A. R. Gilgen (Eds.), *Chaos theory in psychology* (pp. 87–105). Westport, CT: Greenwood Press.

Butz, M. R., Chamberlain, L. L., & McCown, W. G. (1997). *Strange attractors: Chaos, complexity, and the art of family therapy*. New York, NY: John Wiley & Sons Inc.

Byrne, D. G., Mazanov, J., Gregson, R. A. M. (2001). A cusp catastrophe analysis of changes to adolescent smoking behavior in response to smoking prevention programs. *Nonlinear Dynamics, Psychology, & Life Sciences*, *5*, 115–137. doi: 10.1023/A:1026467401041

Calkins, S. D., & Fox, N. A. (2002). Self-regulatory processes in early personality development: A multilevel approach to the study of childhood social withdrawal and aggression. *Development and Psychopathology*, *14*, 477–498. doi: 10.1017/S095457940200305X

Capaldi, D. M., & Patterson, G. R. (1987). An approach to the problem of recruitment and retention rates for longitudinal research. *Behavioral Assessment*, *9*, 169–177.

Case, R., Okamoto, Y., Griffin, S., McKeough, A., Bleiker, C., Henderson, B., & Stephenson, K. M. (1996). The role of central conceptual structures in the development of children's thought. *Monographs of the Society for Research in Child Development*, *246*, 1-295. doi: 10.2307/1166077

Caspar, F., Rothenfluh, T., & Segal, Z. (1992). The appeal of connectionism for clinical psychology. *Clinical Psychology Review*, *12*, 719–762. doi: 10.1016/0272-7358(92)90022-Z

Cerezo, M. A., Trenado, R. M., & Pons-Salvador, G. (2012). Mother–infant interaction and quality of child's attachment: A nonlinear dynamical systems approach. *Nonlinear Dynamics in Psychology and the Life Sciences*, *16*, 243–267.

Chamberlain, P., Patterson, G. R., Rid, J. B., Kavanagh, K., & Forgatch, M. S. (1984) Observation of client resistance. *Behavior Therapy*, *15*, 144–155. doi: 10.1016/S0005-7894(84)80016-7

Cicchetti, D. (1990). A historical perspective on the discipline of developmental psychopathology. In J. E. Rolf, A. S. Masten, D. Cicchetti, & K. H. Nuechterlein (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 2–28). New York, NY: Cambridge University Press.

Cicchetti, D. (1993). Developmental psychopathology: Reactions, reflections, projections. *Developmental Review. Special Issue: Setting a path for the coming decade: Some goals and challenges*, *13*, 471–502. doi: 10.1006/drev.1993.1021

Cicchetti, D., & Cohen, D. J. (1995a). *Developmental psychopathology, Vol. 1: Theory and methods. Wiley series on personality processes*. New York, NY: Wiley.

Cicchetti, D., & Cohen, D. J. (1995b). Perspectives on developmental psychopathology. In D. Cicchetti & Cohen, D. J. (Eds.), *Developmental psychopathology, Vol. 1: Theory and methods. Wiley series on personality processes* (pp. 3–20). New York, NY: John Wiley & Sons.

Cicchetti, D., & Dawson, G. (2002a). *Special issue: Multiple levels of analysis, development and psychopathology, Vol. 14*. New York, NY: Cambridge University Press.

Cicchetti, D., & Dawson, G. (2002b). Editorial: Multiple levels of analysis. *Development and Psychopathology*, *14*, 417–420. doi: 10.1017/S0954579402003012

Cicchetti, D., & Richters, J. E. (1993). Developmental considerations in the investigation of conduct disorder. *Development and Psychopathology*, *5*, 331–334. doi: 10.1017/S0954579400004429

Cicchetti, D., & Schneider-Rosen, K. (1986). Theoretical and empirical considerations in the investigation of the relationship between affect and cognition in atypical populations of infants. In C. E. Izard, J. Kagan, & R. B. Zajonc (Eds.), *Emotions, cognition, and behavior*. (pp. 366–406). New York, NY: Cambridge University Press.

Cicchetti, D., & Sroufe, L. A. (1976). The relationship between affective and cognitive development in Down's syndrome infants. *Child Development*, *47*, 920–929. doi: 10.2307/1128427

Clair, S. (1998). A cusp catastrophe model for adolescent alcohol use: An empirical test. *Nonlinear Dynamics, Psychology, & Life Sciences*, *2*, 217–241. doi: 10.1023/A:1022376002167

Connell, A. M., Hughes-Scalise, A., Klostermann, S., & Azem, T. (2011). Maternal depression and the heart of parenting: Respiratory sinus arrhythmia and affective dynamics during parent–adolescent interactions. *Journal of Family Psychology*, *25*, 653–662. doi: 10.1037/a0025225

Coleman, P., & Watson, A. (2000). Infant attachment as a dynamic system. *Human Development*, *43*, 295–313. doi: 10.1159/000022691

Cox, M. J., & Paley, B. (1997). Families as systems. *Annual Review of Psychology*, *48*, 243–267. doi: 10.1146/annurev.psych.48.1.243

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2000). *Developmental psychopathology and family process: Theory, research, and clinical implications*. New York, NY: Guilford Press.

Dagne, G., & Snyder J. (2011). Relationship of maternal negative moods to child emotion regulation during family interaction. *Development and Psychopathology*, *23*, 211–223. doi: 10.1017/S095457941000074X

Dale, R., & Spivey, M. J. (2005). From apples and oranges to symbolic dynamics: A framework for conciliating notions of cognitive representation. *Journal of Experimental & Theoretical Artificial Intelligence*, *17*, 317–342. doi: 10.1080/09528130500283766

Daw, C. S., Finney, C. E. A., & Tracy, E. R. (2003). A review of symbolic analysis of experimental data. *Review of Scientific Instruments*, *74*, 915–930. doi: 10.1063/1.1531823

De Weerth, C., & Van Geert, P. (2002). Changing patterns of infant behavior and mother-infant interaction: Intra- and interindividual variability. *Infant Behavior & Development*, *24*, 340–374. doi: 10.1016/S0163-6383(02)00083-8

Dishion, T. J., Bullock, B. M., & Granic, I. (2002). Pragmatism in modeling peer influence: Dynamics, outcomes, and change processes. How prevention intervention studies in the field of developmental psychopathology can inform developmental theories and models. [Special Issue]. *Development and Psychopathology*, *14*, 969–981. doi: 10.1017/S0954579402004169

Dishion, T. J., Capaldi, D. M., Spracklen, K. M., & Li, F. (1995). Peer ecology of male adolescent drug use. *Development and Psychopathology*, *7*, 803–824. doi: 10.1017/S0954579400006854

Dishion, T. J., Eddy, J. M., Haas, E., Li, F., & Spracklen, K. (1997). Friendships and violent behavior during adolescence. *Social Development*, *6*, 207–223. doi: 10.1111/1467-9507.00034

Dishion, T. J., French, D. C., & Patterson, G. R. (1995). The development and ecology of antisocial behavior. In D. Cicchetti & D. Cohen (Eds.), *Manual of developmental psychopathology* (pp. 421–471). New York, NY: Wiley.

Dishion, T. J., Gardner, K., Patterson, G. R., Reid, J. B., & Thibodeaux, S. (1983). *The family process code: A multidimensional system for observing family interactions*. Unpublished technical manual. Eugene, OR: Oregon Social Learning Center.

Dishion, T. D., Nelson, S. E., Bullock, B. M., & Winter, C. E. (2004). Adolescent friendship as a dynamic system: Entropy and deviance in the etiology and course of male antisocial behavior. *Journal of Abnormal Child Psychology*, *32*, 651–663. doi: 10.1023/B:JACP.0000047213.31812.21

Dishion, T. J., & Patterson, G. R. (1999). Model building in developmental psychopathology: A pragmatic approach to understanding and intervention. *Journal of Clinical Child Psychology*, *28*, 502–512. doi: 10.1207/S15374424JCCP2804_10

Dishion, T. J., Spracklen, K. M., Andrews, D. W., & Patterson, G. R. (1996). Deviancy training in male adolescent friendships. *Behavior Therapy*, *27*, 373–390. doi: 10.1016/S0005-7894(96)80023-2

Dixon, R. A., & Lerner, R. M. (1988). A history of systems in developmental psychology. In M. H. Bornstein & M. E. Lamb (Eds.), *Developmental psychology: An advanced textbook* (2nd ed., pp. 3–50). Hillsdale, NJ: Erlbaum.

Dumas, J. E., LaFrenier, P. J., & Serketich, W. J. (1995). "Balance of power": A transactional analysis of control in mother-child dyads involving socially competent, aggressive, and anxious children. *Journal of Abnormal Psychology*, *104*, 104–113. doi: 10.1037/0021-843X.104.1.104

Dumas, J. E., Lemay, P., & Dauwalder, J. P. (2001). Dynamic analyses of mother-child interactions in functional and dysfunctional dyads: A synergetic approach. *Journal of Abnormal Child Psychology*, *29*, 317–329. doi: 10.1023/A:1010309929116

Eddy, J. M., & Chamberlain, P. (2000). Family management and deviant peer association as mediators of the impact of treatment condition on youth antisocial behavior. *Journal of Consulting & Clinical Psychology*, *68*, 857–863. doi: 10.1037/0022-006X.68.5.857

Elman, J. L., Bates, E. A., Johnson, M. H., Karmiloff-Smith, A., Parisi, D., & Plunkett, K. (1996). *Rethinking innateness: A connectionist perspective on development*. Cambridge, MA: MIT Press.

Erickson, M. F., Egeland, B., & Pianta, R. (1989). The effects of maltreatment on the development of young children. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 647–684). New York, NY: Cambridge University Press.

Eyberg, S. M., Nelson, M. M., & Boggs, S. R. (2008). Evidence-based psychosocial treatments for children and adolescents with disruptive behavior. *Journal of Clinical Child and Adolescent Psychology*, *37*, 215–237. doi: 10.1080/15374410701820117

Fogel, A. (1990). The process of developmental change in infant communicative action: Using dynamic systems theory to study individual ontogenies. In J. Colombo & J. Fagen (Eds.), *Individual differences in infancy: Reliability, stability, prediction* (pp. 341–358). Hillsdale, NJ: Erlbaum.

Fogel, A. (1993). *Developing through relationships: Origins of communication, self and culture*. Chicago, IL: University of Chicago.

Fogel, A. (1995). Development and relationships: A dynamic model of communication. *Advances in the Study of Behavior*, *24*, 259–290. doi: 10.1016/S0065-3454(08)60396-4

Fogel, A., & Thelen, E. (1987). The development of early expressive and communicative action: Re-interpreting the evidence from a dynamic systems perspective. *Developmental Psychology*, *23*, 747–761. doi: 10.1037/0012-1649.23.6.747

Ford, D. H., & Lerner, R. M. (1992). *Developmental systems theory: An integrative approach*. Newbury Park, CA: Sage.

Forgatch, M. S., & DeGarmo, D. S. (1999). Parenting through change: An effective prevention program for single mothers. *Journal of Consulting & Clinical Psychology*, *67*, 711–724. doi: 10.1037/0022-006X.67.5.711

Freeman, W. (1995). *Societies of brains: A study in the neuroscience of love and hate*. Hillsdale, NJ: Erlbaum.

Gardner, B. C., & Wampler, K. S. (2008). Uncovering dynamical properties in the emotional system of married couples. *Contemporary Family Therapy*, *30*, 111–126. doi: 10.1007/s10591-008-9056-4

Garmezy, N. (1974). The study of competence in children at risk for severe psychopathology. In E. J. Anthony & C. Koupernik (Eds.), *The child in his family: Children at psychiatric risk*. Oxford, UK: John Wiley & Sons.

Garmezy, N., & Rutter, M. (Eds.). (1983). *Stress, coping, and development in children*. Baltimore, MD: Johns Hopkins University Press.

Gasser, M., & Smith, L. B. (1996). Learning nouns and adjective meanings: A connectionist account. *Language and Cognitive Processes*, *13*, 269–306. doi: 10.1080/016909698386537

Gilmore, R. (1981). *Catastrophe theory for scientists and engineers*. New York, NY: Wiley.

Gottlieb, G. (1991). Epigenetic systems view of human development. *Developmental Psychology*, *27*, 33–34. doi: 10.1037/0012-1649.27.1.33

Gottlieb, G. (1992). *Individual development and evolution: The genesis of novel behavior*. London, UK: Oxford University Press.

Gottman, J. M., Coan, J., Carrere, S., & Swanson, C. (1998). Predicting marital happiness and stability from newlywed interactions. *Journal of Marriage and the Family*, *60*, 5–22. doi: 10.2307/353438

Gottman, J. M., Guralnick, M. J., Wilson, B., Swanson, C. C., & Murray, J. D. (1997). What should be the focus of emotion regulation in children? A nonlinear dynamic mathematical model of children's peer interaction in groups. *Development and Psychopathology*, *9*, 421–452. doi: 10.1017/S0954579497002113

Gottman, J. M., McCoy, K., Coan, J., & Collier, H. (1996). *The specific affect coding system (SPAFF) for observing emotional communication in marital and family interaction*. Mahwah, NJ: Erlbaum.

Gottman, J. M., Murray, J. D., Swanson, C. C., Tyson, R., & Swanson, K. R. (2002). *The mathematics of marriage: Dynamic nonlinear models*. London, UK: MIT Press.

Gottman, J. M., & Notarius, C. I. (2000). Decade review: Observing marital interaction. *Journal of Marriage and the Family*, *62*, 927–947. doi: 10.1111/j.1741-3737.2000.00927.x

Granic, I. (2000). The self-organization of parent–child relations: Beyond bi-directional models. In M. D. Lewis & I. Granic (Eds.), *Emotion, development and self-organization: Dynamic systems approaches to emotional development* (pp. 267–297). New York, NY: Cambridge University Press.

Granic, I. (2003, April). *Dynamic systems methods tap previously undetected socialization processes associated with antisocial outcomes*. Paper presented at the Biennial Meeting of the Society for Research in Child Development, Tampa, FL.

Granic, I., & Dishion, T. (2003). Deviant talk in adolescent friendships: A step toward measuring a pathogenic attractor process. *Social Development*, *12*, 314–334. doi: 10.1111/1467-9507.00236

Granic, I., Dishion, T. J., & Hollenstein, T. (2003). The family ecology of adolescence: A dynamic systems perspective on normative development. In G. Adams & M. Berzonsky (Eds.), *Blackwell Handbook of Adolescence* (pp. 60–91). New York: Blackwell.

Granic, I., Hollenstein, T., Dishion, T. J., & Patterson, G. R. (2003). Longitudinal analysis of flexibility and reorganization in early adolescence: A dynamic systems study of family interactions. *Developmental Psychology*, *39*, 606–617. doi: 10.1037/0012-1649.39.3.606

Granic, I., Meusel, L. A., Lamm, C., Woltering, S., & Lewis, M. D. (2012). Emotion regulation in children with behavior problems: Linking behavioral and brain processes. *Development and psychopathology*, *24*, 1019–1029. doi: 10.1017/S095457941200051X

Granic, I., & Patterson, G. R. (2001, July). *Innovations in the study of coercive microsocial processes in middle childhood: New methods and models based on dynamic systems principles*. Paper presented at the meeting of the International Society for Research in Child and Adolescent Psychopathology, Vancouver, British Columbia.

Granic, I., & Patterson, G. (2006). Towards a comprehensive model of antisocial development: A dynamic systems approach. *Psychological Review*, *113*, 101–131. doi: 10.1037/0033-295X.113.1.101

Granic, I., & Lamey, A. V. (2002). Combining dynamic-systems and multivariate analyses to compare the mother-child interactions of externalizing subtypes. *Journal of Abnormal Child Psychology*, *30*, 265–283. doi: 10.1023/A:1015106913866

Granic, I., O'Hara, A., Pepler, D., & Lewis, M. (2007). A dynamic systems analysis of parent–child changes associated with successful real-world interventions for aggressive children. *Journal of Abnormal Child Psychology*, *35*, 845–857. doi: 10.1007/s10802-007-9133-4

Greenberg, L. S., Rice, L. N., & Elliott, R. K. (1996). *Facilitating emotional change: The moment-by-moment process*. New York, NY: Guilford Press.

Guastello, S. J. (1995). *Chaos, catastrophe, and human affairs: Applications of nonlinear dynamics to work, organizations, and social evolution*. Mahwah, NJ: Lawrence Erlbaum Associates.

Guastello, S. J. (2002). *Managing emergent phenomena: Nonlinear dynamics in work organizations*. Mahwah, NJ: Lawrence Erlbaum Associates.

Guastello, S. J., Hyde, T., & Odak, M. (1998). Symbolic dynamic patterns of verbal exchange in a creative problem solving group. *Nonlinear Dynamics, Psychology, and Life Sciences*, *2*, 35–58. doi: 10.1023/A:1022324210882

Haken, H. (1977). *Synergetics An introduction: Nonequilibrium phase transitions and self-organization in physics, chemistry and biology*. Berlin, Germany: Springer-Verlag.

Hamming, R. W. (1950). Error detecting and error correcting codes. *Bell Systems Technical Journal*, *25*, 147–160. doi: 10.1002/j.1538-7305.1950.tb00463.x

Hartelman, P., van der Maas, H., & Molenaar, P. (1998). Detecting and modeling developmental transitions. *British Journal of Developmental Psychology*. *Special Issue: Developmental Transitions, 16*, 97–122. doi: 10.1111/j.2044-835X.1998.tb00751.x

Haslett, T., Smyrnios, K. X, Osbourne, C. (1998). A cusp catastrophe analysis of anxiety levels in pre-university students. *Journal of Psychology*, *132*, 5–24. doi: 10.1080/00223989809599260

Hayes, A. M., & Strauss, J. L. (1998). Dynamic systems theory as a paradigm for the study of change in psychotherapy: An application to cognitive therapy for depression. *Journal of Consulting and Clinical Psychology*, *66*, 939–947. doi: 10.1037/0022-006X.66.6.939

Hayes, A. M., Laurenceau, J-P., Feldman, G., Strauss, J. L & Cardaciotto, L-A. (2007). Change is not always linear: The study of nonlinear and discontinuous patterns of change in psychotherapy. *Clinical Psychology Review*, *27*, 715–723. doi: 10.1016/j.cpr.2007.01.008

Heath, R. A. (2000). *Nonlinear dynamics: Techniques and applications in psychology*. Mahwah, NJ: Lawrence Erlbaum Associates.

Hinde, R. A. (1983). Ethology and child development. In M. Haith & J. Campos (Eds.), *Carmichael's manual of child psychology, Vol. 2* (pp. 27–94). New York, NY: Wiley.

Hinde, R. A. (1992). Some complexities in aggressive behavior. In A. Fraczek & H. Zumkley (Eds.), *Socialization and aggression* (pp. 3–10). New York, NY: Springer-Verlag.

Hinshaw, S. P., & Zupan, B. A. (1997). Assessment of antisocial behavior in children and adolescents. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 36–50). New York, NY: Wiley.

Hinshaw, S. P. (1999, June). *Risk-factor research in developmental psychopathology*. Symposium conducted at the biennial meeting of the International Society for Research in Child and Adolescent Psychopathology, Barcelona.

Hollenstein, T. (2007). State space grids: Analyzing dynamics across development. *International Journal of Behavioral Development*, *31*, 384–396. doi: 10.1177/0165025407077765

Hollenstein, T. (2012). The dynamic systems perspective: What is the system? In L. C. Mayes & M. Lewis (Eds.), *The Cambridge handbook of environment in human development*. (pp. 138–151). New York, NY: Cambridge University Press.

Hollenstein, T. (2013). *State space grids: Depicting dynamics across development*. New York, NY: Springer.

Hollenstein, T., Allen, N. B., & Sheeber, L. (in press). *Affective patterns in triadic family interactions: Associations with adolescent depression*. Development and Psychopathology.

Hollenstein, T., Granic, I., Stoolmiller, M., & Snyder, J. (2004). Rigidity in parent–child interactions and the development of externalizing and internalizing behavior in early childhood. *Journal of Abnormal Child Psychology*, *32*, 595–607. doi: 10.1023/B:JACP.0000047209.37650.41

Hollenstein, T., & Lewis, M. D. (2006). A state space analysis of emotion and flexibility in parent– child interactions. *Emotion*, *6*, 656–662. doi: 10.1037/1528-3542.6.4.656

Hollenstein, T., Lichtwarck-Aschoff, A., & Potworowski, G. (2013). A Model of socioemotional flexibility at three time scales. *Emotion Review*, *5*, 397–405. doi: 10.1177/1754073913484181

Hsu, H., & Fogel, A. (2001). Infant vocal development in a dynamic mother-infant communication system. *Infancy*, *2*, 87–109. doi: 10.1207/S15327078IN0201_6

Hsu, H., & Fogel, A. (2003a). Social regulatory effects of infant non-distress vocalization on maternal behaviors. *Developmental Psychology*, *39*, 976–991. doi: 10.1037/0012-1649.39.6.976

Hsu, H., & Fogel, A. (2003b). Stability and transitions of mother-infant face-to-face communication during the first six months: A

micro-historical approach. *Developmental Psychology*, *39*, 1061–1082. doi: 10.1037/0012-1649.39.6.1061

Inhelder, B. (1976). Some pathologic phenomena analyzed in the perspective of developmental psychology. In B. Inhelder & H. Chipman (Eds.), *Piaget and his school* (pp. 221–227). New York, NY: Springer.

In-Albon, T., & Schneider, S. (2007). Psychotherapy of childhood anxiety disorders: a Meta-analysis. *Psychotherapy and Psychosomatics*, *76*, 15–24. doi: 10.1159/000096361

Izard, C. E. (1977). *Human emotions*. New York, NY: Plenum.

Izard, C. E. (1979). *The maximally discriminative facial movement coding system (MAX)*. Newark, NJ: Instructional resources center, University of Delaware.

Johnson, M. H., & Morton, J. (1991). *Biology and cognitive development: The case of face recognition*. Oxford, UK: Blackwell.

Kagan, J. (1992). Yesterday's premises, tomorrow's promises. *Developmental Psychology*, *28*, 990–997. doi: 10.1037/0012-1649.28.6.990

Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review*, *7*, 865–878. doi: 10.1016/j.cpr.2010.03.001

Kauffman, S. A. (1993). *The origins of order: Self-organization and selection in evolution*. New York, NY: Oxford University Press.

Kazdin, A. E. (2000). *Psychotherapy for children and adolescents: Directions for research and practice*. New York, NY: Oxford University Press.

Kazdin, A. E. (2001). Progression of therapy research and clinical application of treatment require better understanding of the change process. *Clinical Psychology*, *8*, 143–151. doi: 10.1093/clipsy.8.2.143

Kazdin, A. E. (2002). Psychosocial treatments for conduct disorder in children and adolescents. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (2nd ed., pp. 57–85). London, UK: Oxford University Press.

Kazdin, A. E. (2009). Understanding how and why psychotherapy leads to change. *Psychotherapy Research*, *19*, 418–428. doi: 10.1080/10503300802448899

Keating, D. P. (1990). Adolescent thinking. In G. R. Elliott & S. S. Feldman (Eds.), *At the threshold: the developing adolescent* (pp. 54–89). Cambridge, MA: Harvard University Press.

Kelso, J. A. S. (1995). *Dynamic patterns: The self-organization of brain and behavior*. Cambridge, MA: Bradford/MIT Press.

Kowalik, Z. J., Schiepek, G., Kumpf, K., Roberts, L. E., & Elbert, T. (1997). Psycho-therapy as a chaotic process 2: The application of nonlinear analysis methods on quasi time series of the client-therapist interaction: A nonstationary approach. *Psychotherapy Research*, *7*, 197–218. doi: 10.1080/10503309712331331973

Kuo, Z. Y. (1967). *The dynamics of behavior development*. New York, NY: Random House.

Lamey, A., Hollenstein, T., Lewis, M. D., & Granic, I. (2004). GridWare (Version 1.1). [Computer software]. http://statespacegrids.org

Lanza, M. L. (1999). Catastrophe theory: Application of nonlinear dynamics to assault victim responses. *Journal of the American Psychiatric Nurses Association*, *5*, 117–121. doi: 10.1177/107839039900500403

Lavelli, M., Pantoja, A. P. F., Hsu, H., Messinger, D., & Fogel, A. (2005). Using microgenetic designs to study change processes. In D. G. Teti (Ed.), *Handbook of research methods in developmental psychology*. London, UK: Blackwell.

Lee, K., Karmiloff-Smith, A. (2002). Macro- and microdevelopmental research: Assumptions, research strategies, constraints, and utilities. In N. Granott & J. Parziale (Eds.), *Microdevelopment: Transition processes in development and learning. Cambridge studies in cognitive perceptual development* (pp. 243–292). New York, NY: Cambridge University Press.

Lewin, K. (1931). The conflict between Aristotelian and Galilean models of thought in contemporary psychology. *Journal of General Psychology*, *5*, 147–177. doi: 10.1080/00221309.1931.9918387

Lewis, M. D. (2000). The promise of dynamic systems approaches for an integrated account of human development. *Child Development*, *71*, 36–43. doi: 10.1111/1467-8624.00116

Lewis, M. D. (2011). Dynamic systems approaches: Hot enough? Cool enough? *Child Development Perspectives*, *5*, 279–285. doi: 10.1111/j.1750-8606.2011.00190.x

Lewis, M. D., & Douglas, L. (1998). A dynamic systems approach to cognition-emotion interactions in development (pp. 159–188), in *What Develops in Emotional Development?* M. F. Mascolo & S. Griffins (Eds.) New York: Plenum Press.

Lewis, M. D., & Granic, I. (1999a). Self-organization of cognition-emotion interactions. In T. Dalgleish & M. Power (Eds.), *The handbook of cognition and emotion* (pp. 683–701). Chichester, UK: Wiley.

Lewis, M. D., & Granic, I. (1999b). Who put the self in self-organization? A clarification of terms and concepts for developmental psychopathology. *Development and Psychopathology*, *11*, 365–374. doi: 10.1017/S0954579499002102

Lewis, M. D., & Granic, I. (Eds.) (2000). *Emotion, development and self-organization: Dynamic systems approaches to emotional development*. New York, NY: Cambridge University Press.

Lewis, M. D., Lamey, A. V., & Douglas, L. (1999). A new dynamic systems method for the analysis of early socioemotional development. *Developmental Science*, *2*, 457–475. doi: 10.1111/1467-7687.00090

Lewis, M. D., Zimmerman, S., Hollenstein, T., & Lamey, A. V. (2004). Reorganization of coping behavior at 1 1/2 years: Dynamic systems and normative change. *Developmental Science*, *7*(1), 56–73. doi: 10.1111/j.1467-7687.2004.00323.x

Lichtenberg, J. W., & Heck, E. J. (1986). Analysis of sequence and pattern in process research. *Journal of Counseling Psychology*, *33*, 170–180. doi: 10.1037/0022-0167.33.2.170

Lichtwarck-Aschoff, A., Hasselman, F., Cox, R., Peppler, D., & Granic, I. (2012). A characteristic destabilization profile in parent–child interactions associated with treatment efficacy for aggressive children. *Nonlinear Dynamics, Psychology, and Life Sciences*, *16*, 353–379.

Lichtwarck-Aschoff, A., Kunnen, S., &, van Geert, P. (2009). Here we go again: A dynamic systems perspective on emotional rigidity across parent–adolescent conflicts. *Developmental Psychology*, *45*, 1364–1375. doi: 10.1037/a0016713

Lougheed, J., Hollenstein, T., Lichtwarck-Aschoff, A., & Granic, I. (2015). Maternal regulation of child affect in externalizing and typically-developing children. *Journal of Family Psychology*, *29*, 10–19.

Lunkenheimer, E. S., Albrecht, E. C., & Kemp, C. J. (2013). Dyadic flexibility in early parent–child interactions: Relations with maternal depressive symptoms and child negativity and behaviour problems. *Infant and Child Development*, *22*, 250–269. doi: 10.1002/icd.1783

Lunkenheimer, E. S., Hollenstein, T., Wang, J., & Shields, A. M. (2012). Flexibility and attractors in context: Family emotion socialization patterns and children's emotion regulation in late childhood. *Nonlinear Dynamics in Psychology and the Life Sciences*, *16*, 269–291.

Lunkenheimer, E. S., Olson, S. L., Hollenstein, T., Sameroff, A., & Winter, C. (2011). Dyadic flexibility and positive affect in parent–child coregulation and the development of children's behavior problems. *Development and Psychopathology*, *23*, 577–591. doi: 10.1017/S095457941100006X

Lykken, D. T. (1991). What's wrong with psychology anyway? In D. Cicchetti & W. M. Grove (Eds.), *Thinking clearly about psychology* (Vol. 1, pp. 3–39). Minneapolis, MN: University of Minnesota Press.

Mahoney, M. J. (1991). *Human change processes: The scientific foundations of psychotherapy*. New York, NY: Basic Books, Inc.

Magai, C., & McFadden, S. H. (1995). *The role of emotions in social and personality development: History, theory, and research*. New York, NY: Plenum Press.

Mainhard, M. T., Pennings, H. J., Wubbels, T., & Brekelmans, M. (2012). Mapping control and affiliation in teacher–student interaction with state space grids. *Teaching and Teacher Education*, *28*, 1027–1037. doi: 10.1016/j.tate.2012.04.008

Marwan, N., Carmen Romano, M., Thiel, M., & Kurths, J. (2007). Recurrence plots for the analysis of complex systems. *Physics Reports*, *438*, 237–329. doi: 10.1016/j.physrep.2006.11.001

McClelland, J., L. (1979). On the time relations of mental processes: An examination of systems of processes in cascade. *Psychological Review*, *86*, 287–330. doi: 10.1037/0033-295X.86.4.287

Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, *46*, 1–42. doi: 10.1037/0022-006X.46.4.806

Minuchin, S. (1974). *Families & family therapy*. Cambridge, MA: Harvard University Press.

Minuchin, S., & Fishman, C. H. (1974). *Family therapy techniques*. Cambridge MA: Harvard University Press.

Moffitt, T. E. (1993). "Adolescence-limited" and "life-course persistent" antisocial behaviour: A developmental taxonomy. *Psychological Review*, *100*, 674–701. doi: 10.1037/0033-295X.100.4.674

Moore, G. A., Powers, C. J., Bass, A. J., Cohn, J. F., Propper, C. B., Allen, N. B., & Lewinsohn, P. M. (2013). Dyadic interaction: Greater than the sum of its parts? *Infancy*, *18*, 490–515. doi: 10.1111/j.1532-7078.2012.00136.x

Munakata, Y. (1998). Infant perseveration and implications for object permanence theories: A PDP model of the AB task. *Developmental Science*, *1*, 161–184. doi: 10.1111/1467-7687.00021

Munakata, Y., & McClelland, J. L. (2003). Connectionist models of development. Connectionism and dynamic systems theory: Are these really different approaches to development? [Special issue]. *Developmental Science*, *6*, 413–429. doi: 10.1111/1467-7687.00296

Nelson, C. A., Bloom, F. E., Cameron, J. L., Amaral, D., Dahl, R. E., & Pine, D. (2002). An integrative, multidisciplinary approach to the study of brain-behavior relations in the context of typical and atypical development. *Development and Psychopathology*, *14*, 499–520. doi: 10.1017/S0954579402003061

Newell, K. M., & Molenaar, P. (1998). *Applications of nonlinear dynamics to developmental process modeling*. Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Newtson, D. (1994). The perception and coupling of behavior waves. In R. R. Vallacher & A. Nowak (Eds.), *Dynamical systems in social psychology* (pp. 139–166). San Diego, CA: Academic Press.

Newtson, D. (1998). Dynamical systems and the structure of behavior. In K. M. Newell & P. C. Molenaar (Eds.), *Applications of nonlinear dynamics to developmental process modeling* (pp. 129–160). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Norton, A. (1995). Dynamics: An introduction. In R. F. Port & T. van Gelder (Eds.), *Mind as motion: Explorations in the dynamics of cognition* (pp. 45–68). Cambridge, MA: MIT Press.

Nowak, A., & Vallacher, R. R. (1998). *Dynamical social psychology*. New York, NY: Guilford Press.

Overton, W. F., & Horowitz, H. A. (1991). Developmental psychopathology: Integrations and differentiations. In D. Cicchetti & S. L. Toth (Eds.), *Rochester symposium on developmental psychopathology, Vol. 3: Models and integrations*, (pp. 1–42). Rochester, NY: University of Rochester Press.

Quinlan, P. T., Van Der Maas, H. L. J., Jansen, B. R. J., Booij, O., & Rendell, M. (2007). Re-thinking stages of cognitive development: An appraisal of connectionist models of the balance scale task. *Cognition*, *103*, 413–459. doi: 10.1016/j.cognition.2006.02.004

Patterson, G. R. (1982). *Coercive family processes*. Eugene, OR: Castalia.

Patterson, G. R., Reid, J. B., & Dishion, T. (1992). *Antisocial boys*. Eugene, OR: Castilia.

Pepler, D., Craig, W., & O'Connell, P. (1999). Understanding bullying from a dynamic systems perspective. In A. Slater & D. Muir (Eds.), *The Blackwell reader in developmental psychology* (pp. 440–451). Malden, MA: Blackwell Publishers.

Pincus, D. (2001). A framework and methodology for the study of nonlinear, self-organizing family dynamics. *Nonlinear Dynamics, Psychology, and Life Sciences*, *5*, 139–173. doi: 10.1023/A:1026419517879

Plunkett, K. (1993). Lexical segmentation and vocabulary growth in early language acquisition. *Journal of Child Language*, *20*, 43–60. doi: 10.1017/S0305000900009119

Plunkett, K., & Elman, J. L. (1997). *Exercises in rethinking innateness: A handbook for connectionist simulations*. Cambridge, MA: MIT Press.

Port, R. F., & van Gelder, T. (1995). *Mind as motion: Explorations in the dynamics of cognition*. Cambridge, MA: MIT Press.

Prigogine, I., & Stengers, I. (1984). *Order out of chaos*. New York, NY: Bantam.

Ramseyer, F., & Tschacher, W. (2011). Nonverbal synchrony in psychotherapy: Coordinated body-movement reflects relationship quality and outcome. *Journal of Consulting and Clinical Psychology*, *79*, 284–295. doi: 10.1037/a0023419

Reis, H. T., Collins, W. A., & Bersheid, E. (2000). The relationship context of human behavior and development. *Psychological Bulletin. Special Issue: Psychology in the 21st Century*, *126*, 844–872. doi: 10.1037/0033-2909.126.6.844

Ribeiro, A. P., Bento, T., Salgado, J., Stiles, W. B., & Gonçalves, M. M. (2011). A dynamic look at narrative change in psychotherapy: A case study tracking innovative moments and protonarratives using state space grids. *Psychotherapy Research*, *21*, 54–69. doi: 10.1080/10503307.2010.504241

Richters, J. E. (1997). The Hubble hypothesis and the developmentalist's dilemma. *Development and Psychopathology*, *9*, 193–229. doi: 10.1017/S0954579497002022

Ruhland, R., & van Geert, P. (1998). Jumping into syntax: Transitions in the development of closed class words. *British Journal of Developmental Psychology*, *16*, 65–95. doi: 10.1111/j.2044-835X.1998.tb00750.x

Rumelhart, D. E., & McClelland, J. L. (Eds.). (1986). *Parallel distributed processing: Explorations in the microstructure of cognition: Vol. 1. Foundations*. Cambridge, MA: Bradford Books/MIT Press.

Ryan, K. D., Gottman, J. M., Murray, J. D., Carrere, S., & Swanson, C. (2000). Theoretical and mathematical modeling of marriage. In M. D. Lewis & I. Granic (Eds.), *Emotion, development and self-organization: Dynamic systems approaches to emotional development* (pp. 349–372) New York, NY: Cambridge University Press.

Sabelli, H. C., Carlson-Sabelli, L., Patel, M., Levy, A., & Diez-Martin, J. (1995). Anger, fear, depression, and crime: Physiological and psychological studies using the process method. In R. Robertson & A. Combs (Eds.), *Chaos theory in psychology and the life sciences* (pp. 65–88). Mahwah, NJ: Lawrence Erlbaum Associates.

Sameroff, A. J. (1983). Developmental systems: Contexts and evolution. In W. Kessen (Ed.), *History, theory, and methods* (4th ed., Vol. 1, pp. 237–294). New York, NY: Wiley.

Sameroff, A. J. (1995). General systems theories and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental*

psychopathology, Vol. 1: Theory and methods. Wiley series on personality processes (pp. 659–695). New York, NY: Wiley & Sons.

Schiepek, G. (2009). Complexity and nonlinear dynamics in psychotherapy. *European Review*, *17*, 331–356. doi: 10.1017/S1062798709000763

Schroeck, F. E. (1994). New mathematical techniques for pattern recognition. In R. R. Vallacher & A. Nowak (Eds.), *Dynamical systems in social psychology* (pp. 139–166). San Diego, CA: Academic Press.

Shannon, C. E., & Weaver, W. (1949). *The mathematical theory of communication*. Urbana, IL: University of Illinois Press.

Simonoff, J. S. (1996). *Smoothing methods in statistics*. New York, NY: Springer-Verlag.

Smith, L. B. (1993). The concept of same. *Advances in Child Development and Behavior*, *24*, 216–253.

Smith, L. B. (1995). Self-organizing processes in learning to learn words: Development is not induction. In C. A. Nelson (Ed.), *The Minnesota symposia on child psychology: Basic and applied perspectives on learning, cognition, and development* (Vol. 28, pp. 1–32). Mahwah, NJ: Erlbaum.

Smith, L. B., & Samuelson, L. K. (2003). Different is good: Connectionism and dynamic systems theory are complementary emergentist approaches to development. Connectionism and dynamic systems theory: Are these really different approaches to development? [Special issue]. *Developmental Science*, *6*, 434–439. doi: 10.1111/1467-7687.00298

Smith, L. B., & Thelen, E. (2003). Development as a dynamic system. *Trends in Cognitive Science*, *7*, 343–348. doi: 10.1016/S1364-6613(03)00156-6

Snyder, J., Edwards, P., McGraw, K., Kilgore, K., & Holton, A. (1994). Escalation and reinforcement in mother-child conflict: Social processes associated with the development of physical aggression. *Development and Psychopathology*, *6*, 305–321. doi: 10.1017/S0954579400004600

Snyder, J., Stoolmiller, M., Wilson, M., & Yamamoto, M. (2003). Child anger regulation, parental responses to children's anger displays, and early child antisocial behavior. *Social Development. 12*, 335–360. doi: 10.1111/1467-9507.00237

Spemann, H. (1938). *Embryonic development and induction*. New Haven, CT: Yale University Press.

Spencer, J. P., & Schoner. (2003). Bridging the representational gap in the dynamic systems approach to development. Connectionism and dynamic systems theory: Are these really different approaches to development? [Special issue]. *Developmental Science 6*, 392–412. doi: 10.1111/1467-7687.00295

Spencer, J. P., & Thelen, E. (2003). Connectionism and dynamic systems theory: Are these really different approaches to development? [Special issue]. *Developmental Science*, *6*, 375–377. doi: 10.1111/1467-7687.00293

Sravish, A. V., Tronick, E., Hollenstein, T., & Beeghly, M. (2013). Dyadic Flexibility during the Face-to-Face Still-Face Paradigm: A dynamic systems analysis of its temporal organization. *Infant Behavior and Development*, *36*(3), 432–437. doi: 10.1016/j.infbeh.2013.03.013

Sroufe, L. A. (1979). The coherence of individual development: Early care, attachment, and subsequent developmental issues. *American Psychologist*, *34*, 834–841. doi: 10.1037/0003-066X.34.10.834

Sroufe, L. A. (1986). Appraisal: Bowlby's contribution to psychoanalytic theory and developmental psychology—attachment, separation, loss. *Journal of Child Psychology & Psychiatry & Allied Disciplines. Special Issue: 30th anniversary of the Association for Child Psychology and Psychiatry and Allied Disciplines*, *27*, 841–849

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, *55*, 17–29. doi: 10.2307/1129832

Steenbeek, H., & van Geert, P. (2005). A dynamic systems model of dyadic child-peer play interactions. *European Journal of Developmental Psychology*, *2*, 105–145. doi: 10.1080/17405620544000020

Steenbeek, H., & van Geert, P. (2007). A dynamic systems approach to dyadic interaction in children: Emotional expression, action, dyadic play, and sociometric status. *Developmental Review*, *27*, 1–40. doi: 10.1016/j.dr.2006.06.002

Steenbeek, H., & van Geert, P. (2008). The empirical validation of a dynamic systems model of interaction; Do children of different sociometric statuses differ in their dyadic play interaction. *Developmental Science*, *11*, 253–281. doi: 10.1111/j.1467-7687.2007.00655.x

Stephen, D. G., Boncoddo, R. A., Magnuson, J. S., & Dixon, J. A. (2009). The dynamics of insight: Mathematical discovery as a phase transition. *Memory & Cognition*, *37*, 1132–1149. doi: 10.3758/MC.37.8.1132

Stephen, D. G., Dixon, J. A., & Isenhower, R. W. (2009). Dynamics of representational change: Entropy, action and cognition. *Journal of Experimental Psychology: Human Perception and Performance*, *35*, 1811–1832. doi: 10.1037/a0014510

Stoolmiller, M., & Snyder, J. (2006). Modeling heterogeneity in social interaction processes using multi-level survival analysis. *Psychological Methods*, *26*, 164–177. doi: 10.1037/1082-989X.11.2.164

Teşileanu, T., & Meyer-Ortmanns, H. (2006). Competition of languages and their hamming distance. *International Journal of Modern Physics C*, *17*(2), 259–278. doi: 10.1142/S0129183106008765

Tesser, A., & Achee, J. (1994). Aggression, love, conformity, and other social psychological catastrophes. In R. R. Vallacher & A. Nowak (Eds.), *Dynamical systems in social psychology* (pp. 95–109). San Diego, CA: Academic Press.

Thelen, E., & Smith, L. B. (1994). *A dynamic systems approach to the development of cognition and action*. Cambridge, MA: Bradford/MIT Press.

Thelen, E., & Smith, L. B. (1998). Dynamic systems theories. In W. Damon (Ed.), *Handbook of Child Psychology: Theoretical models of human development* (5th ed., Vol. 1, pp. 563–634). New York, NY: Wiley.

Thelen, E., & Ulrich, B. D. (1991). Hidden skills: A dynamic systems analysis of treadmill stepping during the first year. *Monographs of the Society for Research in Child Development*, *56*(1), 1–98. doi: 10.2307/1166099

Thelen, E., Schoner, G., Scheier, C., & Smith, L. B. (2001). The dynamics of embodiment: A field theory of infant perseverative reaching. *Behavioral and Brain Sciences*, *24*, 1–34. doi: 10.1017/S0140525X01003910

Thom, R. (1975). *Structural stability and morphogenesis*. New York, NY: Benjamin-Addison-Wesley.

Tomkins, S. S. (1963). *Affect, imagery, consciousness: Vol. II. The negative affects*. New York, NY: Springer.

Tschacher, W., Scheier, C., & Grawe, K. (1998). Order and pattern formation in psychotherapy. *Nonlinear Dynamics, Psychology, and Life Sciences*, *2*, 195–216. doi: 10.1023/A:1022324018097

van der Maas, H. (1995). Beyond the metaphor? *Cognitive Development*, *10*, 621–642. doi: 10.1016/0885-2014(95)90029-2

van der Maas, H. (1998). The dynamical and statistical properties of cognitive strategies: Relations between strategies, attractors, and latent classes. In K. M. Newell & P. C. M. Molenaar (Eds.), *Applications of nonlinear dynamics to developmental process modeling* (pp. 161–176). Mahwah, NJ: Erlbaum.

van der Maas, H., & Molenaar, P. (1992). Stagewise cognitive development: An application of catastrophe theory. *Psychological Review*, *99*, 395–417. doi: 10.1037/0033-295X.99.3.395

van der Giessen, D., Branje, S. J., Frijns, T., & Meeus, W. H. (2013). Dyadic variability in mother-adolescent interactions: Developmental trajectories and associations with psychosocial functioning. *Journal of Youth and Adolescence*, *42*, 96–108. doi: 10.1007/s10964-012-9790-7

van der Giessen, D., Hollenstein, T., Hale, W. W., Koot, H. M., Meeus, W., & Branje, S. (2015). Emotional variability in mother-adolescent conflict interactions and internalizing problems of mothers and adolescents: Dyadic and individual processes. *Journal of Abnormal Child Psychology*, *43*, 339–353. doi 10.1007/s10802-014-9910-9

van Dijk, M., Hunnius, S., & van Geert, P. (2012). The dynamics of feeding during the introduction to solid food. *Infant Behavior and Development*, *35*, 226–239. doi: 10.1016/j.infbeh.2012.01.001

van Geert, P. (1991). A dynamic systems model of cognitive and language growth. *Psychological Review*, *98*, 3–53. doi: 10.1037/0033-295X.98.1.3

van Geert, P. (1994). *Dynamic systems of development: Change between complexity and chaos*. New York, NY: Prentice Hall/Harvester Wheatsheaf.

van Geert, P. (1995). Dimensions of change: A semantic and mathematical analysis of learning and development. *Human Development*, *38*, 322–331. doi: 10.1159/000278338

van Geert, P. (1997a). Nonlinear dynamics and the explanation of mental and behavioral development. *Journal of Mind & Behavior*, *18*, 269–290.

van Geert, P. (1997b). Que sera sera: Determinism and nonlinear dynamic model building in development. In A. Fogel, M. C. D. P. Lyra, & J. Valsiner (Eds.), *Dynamics and indeterminism in developmental and social processes* (pp. 13–38). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

van Geert, P. (1998a). A dynamic systems model of basic developmental mechanisms: Piaget, Vygotsky, and beyond. *Psychological Review*, *105*, 634–677. doi: 10.1037/0033-295X.105.4.634-677

van Geert, P. (1998b). Dynamic modeling of cognitive and language development: From growth processes to sudden jumps and multimodality. In K. M. Newell & P. C. Molenaar (Eds.), *Applications of nonlinear dynamics to developmental process modeling* (pp. 129–160). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

van Geert, P. (1998c). We almost had a great future behind us: The contribution of non-linear dynamics to developmental-science-in-the-making. *Developmental Science*, *1*, 143–159. doi: 10.1111/1467-7687.00020

van Geert, P., & Lichtwarck-Aschoff, A. (2005). A dynamic systems approach to family assessment. *European Journal of Psychological Assessment*, *21*, 240–248. doi: 10.1027/1015-5759.21.4.240

van Geert, P., & van Dijk, M. (2002). Focus on variability: New tools to study intra-individual variability in developmental data. *Infant Behavior and Development*, *25*, 340–370. doi: 10.1016/S0163-6383(02)00140-6

van Orden, G. C., Kloos, H., & Wallort, S. (2009). Living in the pink: Intentionality, wellbeing and complexity. In C. Hooker (Ed.), *Handbook of the philosophy of science* (pp. 639–682). Amsterdam, The Netherlands: Elesvier.

van Rijn, H., Van Someren, M., & Van der Maas, H. (2003). Modeling developmental transitions on the balance scale task. *Cognitive Science*, *27*, 227–257. doi: 10.1016/S0364-0213(02)00116-7

Vetere, A., & Gale, A. (1987). The family: A failure in psychological theory and research. In A. Vetere, A. Gale, S. Lewis, C. Jolly, & S. Reynolds (Eds.), *Ecological studies of family life* (pp. 1–17). Chichester, UK: Wiley.

von Bertalanffy, L. (1968). *General systems theory*. New York, NY: Braziller.

Webber Jr., C. L., & Zbilut, J. P. (2005). Recurrence quantification analysis of nonlinear dynamical systems. In M. A. Riley & G. C. Van Orden (Eds.), *Tutorials in contemporary nonlinear methods for the behavioral sciences* (pp. 26–94). Washington, DC: National Science Foundation.

Webber Jr., C. L., Marwan, N., Facchini, A., & Giuliani, A. (2009). Simpler methods do it better: Success of recurrence quantification analysis as a general purpose data analysis tool. *Physics Letters A*, *373*, 3753–3756. doi: 10.1016/j.physleta.2009.08.052

Wickens, T. D. (1989). *Multiway contingency table analysis for the social sciences*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Wimmers, R. H., Savelsbergh, G. J. P., van der Kamp, J., & Hartelman, P. (1998). A developmental transition in apprehension modeled as a cusp catastrophe. *Developmental Psychobiology*, *32*, 23–35. doi: 10.1002/(SICI)1098-2302(199801)32:1<23::AID-DEV3>3.0.CO;2-V

Witherington, D. C. (2007). The dynamic systems approach as metatheory for developmental psychology. *Human Development*, *50*, 127–153. doi: 10.1159/000100943

CHAPTER 17

Missing Data

TODD D. LITTLE, KYLE M. LANG, WEI WU, and MIJKE RHEMTULLA

STATISTICAL ISSUES: WHAT HAPPENS WHEN DATA GO MISSING? 761
Bias 761
Type I Error 761
Type II Error/Power 761
MECHANISMS OF MISSINGNESS 763
Missing Completely at Random 764
Missing at Random 766
Missing Not at Random 766
MODERN MISSING DATA METHODS 767
FULL INFORMATION MAXIMUM LIKELIHOOD 767
Multiple Imputation 768
PRACTICAL CONSIDERATIONS 774
Assumptions 775
Auxiliary Variables 776
Fraction of Missing Information 777
Assessing Model Fit with Missing Data 777
Mediation Analysis 780
When to Use FIML Versus MI 782
REVIEW OF MISSING DATA PRACTICES IN PSYCHOLOGICAL RESEARCH 782
WHY CHANGE? 784
PLANNED MISSING DATA DESIGNS 784
Multiform Planned Missing Protocols 785
Two-Method Planned Missing Design 787
Wave-Missing Designs 789
CONCLUSIONS 790
REFERENCES 791

Missing data are ubiquitous in most human science data, and many prominent social scientists and statisticians have written on the subject of addressing nonresponse in social science studies (e.g., Enders, 2013; Graham, Cumsille, & Shevock, 2012; Graham, Hofer, Donaldson, MacKinnon, & Schafer, 1997; Graham, Hofer, & Piccinin, 1994; Hofer & Hoffman, 2007; Little & Schenker, 1995; Nakagawa & Freckleton, 2010; Sinharay, Stern, & Russell, 2001). The treatments for missing data vary considerably across the disciplines covered under the human sciences umbrella. Unfortunately, the traditional procedures that have been in common use through the last century are prone to bias except in very circumscribed conditions. Fortunately, recent advances in the capabilities of computer hardware and software have made a new breed of contemporary approaches both accessible and easy to implement. These contemporary treatments are versatile and powerful in the ways that unplanned missing data can be handled. And, as we will describe in detail, the power of the modern missing data approaches can be leveraged to allow remarkable uses in the form of planned missing data designs. The goal of this chapter is to supplement, in an accessible manner, the burgeoning literature on modern missing data treatments, with a particular focus on planned missing data designs. Later in this chapter, we will describe in some detail the two contemporary approaches: full information maximum likelihood (FIML) estimation and multiple imputation (MI). For book-length treatments of these contemporary approaches to addressing missing data, see Allison (2002), Enders (2010), Graham (2012), Little and Rubin (2002), and van Buuren (2012).

Our discussions around the topic of missing data will focus on examples derived from clinical research in the area of developmental psychopathology (see also Little, Jorgensen, Lang, & Moore, 2014). Our goal is to fuel the movement toward a data-collection paradigm shift

Partial support for this project was provided by grant NSF 1053160 to the first and third authors, a Banting postdoctoral fellowship from the Social Sciences and Humanities Research Council of Canada to the fourth author, and the Center for Research Methods and Data Analysis at the University of Kansas (when it was under the direction of Todd D. Little). Any opinions, findings, and conclusions or recommendations expressed in this material are those of the author(s) and do not necessarily reflect the views of the funding agencies. We would like to thank Richard Kinai for assisting with creating Table 17.2.

that embraces planned missing data designs and improves study protocols to better address the inevitable unplanned missing data that will arise. In this regard, missing data are not a bane in the research process, but rather appropriate application of modern methods provide solace for the unplanned missing values and dramatic opportunities in the context of planned missing data designs.

STATISTICAL ISSUES: WHAT HAPPENS WHEN DATA GO MISSING?

Regardless of the reasons for missing data, the problem cannot simply be ignored (Allison, Goodson, & Neilands, 2008; Raghunathan, 2004), and the presence of nonresponse necessarily complicates the statistical analysis (Brown, 1994; Cheung, 2007; Finkbeiner, 1979; Gleason & Staelin, 1975; Haitovsky, 1968; Harel, Hofer, Hoffman, Pedersen, & Johansson, 2007; Kim & Bentler, 2002; Lee, 1986; Little, Howard, McConnell, & Stump, 2011; Little, Schnabel, & Baumert, 2000; Longford, 2005; McArdle, 1994; McArdle & Hamagami, 1992; McArdle & Hamagami, 2001; Rovine, 1994; Song & Lee, 2002; Wothke, 2000). More specifically, three statistical issues emerge when data have gone astray: bias, type I error, and type II error (i.e., power).

Bias

The first key issue is bias. Is there anything systematic about why the data are missing? If so, then the remaining data for the selected sample are no longer representative of the population from which the sample was drawn. Any parameter estimates derived from the remaining data will no longer generalize to the population and any conclusions will be, to some degree, invalid (Little & Rubin, 2002; Schafer, 1997). As we will illustrate in more detail below, the classical, or *ad hoc*, treatments for missing data (e.g., listwise or pairwise deletion) do nothing to correct for potential bias, whereas the modern, or *principled*, treatments attempt to correct for any biasing influences (Little & Rubin, 2002; Rubin, 1987).

Modern missing data treatments address the bias induced by a systematic influence associated with the missing data by adjusting the parameter estimates to reflect, as closely as possible, what they would have been had there been no missing data (Rubin, 1996). The extent to which this is possible depends on the causes of missing data, and whether those causes are represented in the data (i.e., the missing data mechanism; more on this later). In other words, when data go missing we lose information both in the statistical sense of lower power and in the substantive sense that we know less than what we should know.

Type I Error

The second issue to contend with in the face of missing data is type I error. Many traditional methods for dealing with missing data (e.g., any single-imputation technique) result in standard errors that are smaller than they should be, given that the data are not complete. The problem here is similar to what would happen if one collected data from 20 people but computed a standard error using $N = 200$. The standard error is based on an assumption that the data contain more statistical precision than they really do, so the estimated standard error is too small. Too-small standard errors translate to a higher-than-nominal probability of finding a significant result when there is no effect in the population.

Type II Error/Power

The third issue when data go missing is power loss (i.e., higher type II error rates). As implied in the previous paragraph, missing data means less statistical precision, so it will always result in less powerful analyses than a complete data set (unless the missing values were completely redundant with the nonmissing values; Davey & Savla, 2010; Graham, Olchowski, & Gilreath, 2007; Savalei & Rhemtulla, 2012). Listwise deletion leads to the greatest loss in power of all missing data methods (Enders, 2010; Little & Rubin, 2002; Schafer & Graham, 2002). Single imputation methods, such as mean substitution or regression imputation, will lead to greater power; however, these methods rarely lead to unbiased estimates and are associated with increased type I error rates (Enders, 2010; Rubin, 1987, 1996; Schafer & Graham, 2002). Modern treatments to missing data are *efficient*, which means they result in the most powerful analysis possible, while also eliminating much of the bias due to selective missingness (Graham et al., 2007).

Table 17.1 presents a list of missing data treatments along with descriptions of how each treatment deals with (or fails to deal with) bias and type I and II error. As is plain from this table, the conditions under which the traditional ad hoc methods will be valid are quite limited and often unrealistic in contemporary studies across the human sciences. To fully understand the reasons for the poor performance of classical methods, we need to define the three mechanisms for missing data.

TABLE 17.1 Methods of Treating Missing Data

Method Deletion	Bias/power/error	Description
Listwise deletion/complete case analysis	Bias under MAR & MNAR Loss of power under MCAR, MAR, & MNAR	All cases with any missing values are deleted. Analyses are done on the remaining cases. Under MCAR, this technique produces unbiased parameter estimates. Standard errors (and thus confidence intervals and *p*-values) are larger, reflecting the loss of data. As the proportion of missing data increases, power can decrease substantially. Under MAR/MNAR, the sample after deletion is not representative of the complete data set, resulting in biased parameter estimates (e.g., means, correlations, regression coefficients). Bias increases as the complete case sample grows more different from the complete sample, and as the proportion of missing data increases.
Pairwise deletion/available case analysis	Bias under MAR & MNAR Loss of power under MCAR, MAR, & MNAR	Each statistic is computed using cases that have complete data available for that statistic. A case with missing data on V3 is not used to find the correlation between V2 and V3, but is used to find the correlation between V1 and V2. Power loss is less than with listwise deletion but still a problem. Estimates are biased under MAR and MNAR. Every statistic is based on a different sample size, which may result in an impossible correlation matrix, which can cause serious estimation problems. As sample size is a key element in calculating standard errors, lack of a constant N results in some software packages using the average sample size per variable for these computations. This has the potential of underestimating/overestimating the standard error for some variables. Lack of a single value for a sample size affects the accuracy of the standard error and the testing of model fit.
Single imputation		Single-imputation techniques address prediction error but not error of estimation, leading to deflated standard error estimates. This leads to inflated type I error rates. When variance is underestimated, correlations suffer and significance testing tends to result in more type II errors.
Mean substitution	Bias under MCAR, MAR, & MNAR Error inflation (type I error) under MCAR, MAR, & MNAR	Each missing value is replaced with that variable's mean. The imputed values have zero variance because they all take on the same value. As such, estimates of the variable's variance and relations with other variables (e.g., covariances, correlations, regression coefficients) are underestimated. This problem exists regardless of the missingness mechanism and gets worse as the proportion of missing data increases.
Regression imputation	Bias under MCAR, MAR, & MNAR Error inflation (type I error) under MCAR, MAR, & MNAR	Each missing value is replaced with its predicted value based on a regression equation using all other variables as predictors. Information from the complete variables is used to fill in the incomplete variables. The imputed values are perfectly predicted by the other variables in the data set, with no error added. Variances will be underestimated (because error variance is lacking), and variable relations (correlations, covariances, regressions) will be overestimated. This problem exists regardless of the missingness mechanism, and gets worse as the proportion of missing data increases.
Stochastic regression imputation	Bias under MNAR Error inflation (type I error) under MCAR, MAR, & MNAR	As with regression imputation, each missing value is replaced with its predicted value, but it has a normally distributed residual error term added to the imputed predicted score. The added residual variance adds variability to the imputed variable and adds noise to the estimated relations between variables. Thus, under MCAR and MAR, parameter estimates are unbiased. Under MNAR, the required information to achieve accurate estimates is missing, so bias persists under MNAR. As with every single-imputation technique, the imputed data are treated like complete data, so standard errors will resemble what they would have looked like with complete data. In other words, the information lost due to missing data is not accounted for, so standard errors will be too small. Inflated type I error rates (under every mechanism) result.
Expectation-maximization (EM) imputation	Bias under MNAR Error inflation (type I error) under MCAR, MAR, & MNAR	The EM algorithm produces maximum likelihood (ML) estimates of the covariance matrix $\boldsymbol{\Sigma}$ and mean vector $\boldsymbol{\mu}$ from incomplete data. The algorithm iterates through two steps. In the expectation (E) step, the missing values are filled in with their expected values after conditioning on the observed data (similar to stochastic regression imputation). The subsequent maximization (M) step applies complete data ML estimation to the imputed data from the E-step to derive updated estimates of the covariances and the means. These updated estimates are used by the next E-step to build the regression equations used to fill in the missing data. The algorithm repeatedly iterates between the E- and M-steps until the difference in the maximized likelihood values from adjacent M-steps falls below a specified criterion. The imputed values from the last E-step may be saved as a single imputed data set. EM is an important building block of more complex missing data methods (e.g., Enders, 2003; 2004; Enders & Peugh, 2004; Lauritzen, 1995; Savalei & Bentler, 2009). The estimated $\boldsymbol{\Sigma}$ and $\boldsymbol{\mu}$ are the same as those produced by FIML. However, a single EM imputation has the same problem as any other single imputation, which is that uncertainty cannot be accounted for in a single-imputed data set. Type I error rates will be inflated.

(*continued*)

TABLE 17.1 *(Continued)*

Method Deletion	Bias/power/error	Description
Hot deck imputation	Bias under MNAR Error inflation (type I error) under MCAR, MAR, & MNAR	Both hot and cold deck imputation techniques are common in survey research. Hot deck imputation works by finding participants who match the case with missing data on other variables. For example, missing scores on job performance are replaced by randomly sampling job performance scores from cases with identical age and gender. Depending on how the imputation is actually performed, this method might or might not preserve the correlations among variables. This method is nonparametric and less sensitive to model misspecification. It is not good for small sample sizes.
Cold deck imputation	Bias under MNAR Error inflation (type I error) under MCAR, MAR, & MNAR	In cold deck imputation, information from external sources is used to determine the matching variables. An external source could be another available data set from which to impute missing values. This technique results in small standard errors (as missing information is not taken into account) and adjusted R^2, denoting decreased variability in the data used for these analyses.
Averaging available items (person-mean imputation / prorated scale score)	Bias under MCAR, MAR, & MNAR Error inflation under MCAR, MAR, & MNAR	A technique used on multiple item scale data with item-level missing data. A scale score is computed by averaging the available items for each case. This technique is equivalent to replacing the missing values for each case with the mean of that case's values. This technique only works for MCAR assuming *parallel items*: all items have same mean, same true-score variance, and the same error variance—this is rarely the case. When this assumption is violated, estimates will be biased even when data are MCAR. More specifically, coefficient alpha estimates will be biased, as will measures of association and variance.
Last observation carried forward	Bias under MCAR, MAR, & MNAR Error inflation (type I error) under MCAR, MAR, & MNAR	Missing values in longitudinal data are replaced with the latest previous observation. A key assumption here is that the most recent observation is the best guess for subsequent missing values. The implication is that scores remain constant after the last observed measurement, an assumption that is not reasonable in longitudinal data. Parameter estimates will be biased in unpredictable directions, depending on the particular features of the data. The magnitude of bias is not predictable. Treatment group differences can be attenuated. Type I error rates are inflated through failure to account for the effect of missing information.
Modern methods		
Multiple imputation (MI)	Bias under MNAR only Power is maximized and error rates are correct for MCAR and MAR	In MI, multiple values for each missing data-point are imputed. The information lost due to missing data in estimating model parameters is quantified using the variability in parameter estimates between imputed data sets. As such, standard error estimates are unbiased, resulting in accurate type I error rates. Imputation uses all the information in the data set, resulting in unbiased parameter estimates under MCAR and MAR. MI commonly assumes multivariate normal data, but imputation algorithms for other distributions are possible. With the correct assumptions on data distributions and missing data mechanism and a large number of imputations, MI is asymptotically unbiased and efficient.
Full information maximum likelihood (FIML)	Bias under MNAR only Power is maximized and error rates are correct for MCAR and MAR	FIML estimates model parameters and standard errors directly using all the available data. FIML aims at identifying the population parameters that are most likely to have generated the observed data (i.e., maximize the likelihood of observing sample data). FIML is a model-based missing data solution, which means that its accuracy depends on the accuracy of the model being estimated. Typically multivariate normality is assumed, though it is possible (but computationally intensive) to use FIML with binary and other distributions. When the assumptions are met, FIML leads to asymptotically unbiased and efficient estimates.

MECHANISMS OF MISSINGNESS

Missing data can occur because a specific datum for given person is not present (e.g., a participant skipped an item due to an error or not wanting to answer) or because data on all variables at a specific measurement occasion for a specific person is not present (e.g., a participant dropped out of a longitudinal study after the second wave of data collection). The first type of missing data is often referred to as item missing. The second type of missing data is often called wave missing, or if the wave missingness occurs because of participants permanently dropping out of a study, attrition. Terms like attrition, wave missing, and item missing are general descriptors of the types of missing data that occur because a desired response was not obtained (Enders, 2010; Rubin, 1987; Shafer & Graham, 2002). This type of missing data should be distinguished from structural missingness. Structural missingness refers

to elements of a data matrix that are not possible to collect (e.g., items that ask about pregnancy for participants who are not or cannot become pregnant, empty cells following a negative response to a "gatekeeper" item, or data for participants after they are deceased). The various descriptors of the types of nonresponse that can occur should not be confused with the mechanisms of missing data. A mechanism of missing data is the causal process associated with whether a data point is missing or not but not necessarily what the value of the missing information would have been.[1]

Missingness mechanisms distinguish the causes of missingness. The pattern of missing data can be described using a missingness matrix, R, which has the same dimensions as the data matrix but contains 0 whenever a datum is observed and 1 whenever it is missing. The key question in establishing the missingness mechanism is: what predicts R? There are three potential sources of prediction, which are the three missingness mechanisms (Rubin, 1976). These causes together account for all missing data. In describing these mechanisms, we will refer to Table 17.2, which provides their definitions in terms of the formal probability distributions of the missing data patterns as originally described by Rubin (1976). Figure 17.1 provides a graphic schematic of the mechanisms of missing data.

By way of a running example, we will describe a hypothetical study of the relationship between parental involvement (a scale administered as part of a larger parent protocol) and child school adjustment (a scale administered as part of a larger teacher protocol). As a purely hypothetical point of discussion, we will pick an arbitrary true correlation of .5 between these two constructs. This correlation of .5 exists in the population and in the hypothetical sample that is drawn from the population; that is, sampling error is held constant so we can focus on the influence of the missing data mechanisms.

Missing Completely at Random

The first mechanism of missing data is termed missing completely at random (MCAR). When the data are MCAR it means that the missing data patterns have zero associations with any variables included in the data set or with any unmeasured variables that are associated with the missing values themselves.[2] This lack of association does not mean that a datum is not missing for a reason—under MCAR, the reasons are essentially random. In this regard, the label for this mechanism clearly communicates, even to naïve readers, the reason for the missingness. In this regard, MCAR can be a truly random process or a process that happens to have a zero association with all variables in an investigation. Unfortunately, MCAR data are not as common as we would hope. As indicated in Table 17.1,

[1]Strictly speaking, the missingness mechanism can't provide any information about what the missing value may have been. It is only a way to describe the process that gave rise to the nonresponse. From the perspective of Rubin (1976), every cell in a data set is associated with two values: an actual data value Y_{ij} (which may or may not be observed); and a missingness indicator R_{ij} (which takes a value of 1 when the corresponding Y_{ij} is missing and 0 if it is observed). The missingness mechanisms, as they are traditionally conceptualized, are concerned only with describing the process by which R_{ij} takes a value of 1 or 0 (i.e., whether or not Y_{ij} is missing or observed), but they do not address what the correct value for any missing Y_{ij} might be. This is an important distinction. Knowing what the missing values would have been implies knowledge of the sampling distribution of the missing values. Knowing the missingness mechanism, on the other hand, is a weaker form of information that implies only knowledge of the more general process by which a value is or is not observed (but not anything about what that value may look like). Rubin (1987, p. 17) overtly distinguishes between these two types of information when he says: "In general, it is impossible to estimate a population quantity such as the mean without making assumptions about either the distribution of values for nonrespondents or the process that creates nonresponse."

[2]Every incident of nonresponse, as with any natural occurrence, has some cause. If MCAR missing had no relationship to any variable at all, then it would not exist. In other words, if there is no reason for a value to go missing, it will be observed. This claim can lead to confusion if we describe MNAR as the probability of missingness being predictable by some set of unmeasured variables. This misunderstanding can be largely remedied if we recognize that this definition of MNAR is what Enders (2010) called *indirect MNAR*. The issue can be illuminated by expanding the description of indirect MNAR slightly. Indirect MNAR is only problematic because the unobserved variables that predict R are also related to the Y_{mis}. This mutual association induces a spurious correlation between Y_{mis} and R. This is one reason that the original Rubin (1976) definitions for the missingness mechanisms are given only in terms of the statistical patterns in the data set. In practice, whether the MNAR is direct or indirect, the observed pattern of relationships in the data will be the same. Namely, there will be an apparent relationship between Y_{mis} and R, even after controlling for the relationship between Y_{obs} and R. As discussed above, the probability of MCAR missing is necessarily predictable by some set of unmeasured variables (otherwise the data wouldn't be missing). However, unlike the indirect MNAR case, those unmeasured variables are *not* associated with the missing values. Therefore, in the MCAR case, there is no spurious correlation induced between Y_{mis} and R, and there is no apparent connection between Y_{mis} and R in the data set.

TABLE 17.2 Missing Data Mechanisms

Mechanism	Mathematical definition[1]	Verbal definition
Missing completely at random (MCAR)	$P(R\|Y_{Obs}, Y_{Mis}) = P(R)$	Missingness on Y is unrelated to any of the variables of interest. The missing data are a pure random sample of the complete data.
Missing at random (MAR)	$P(R\|Y_{Obs}, Y_{Mis}) = P(R\|Y_{Obs})$	Missingness on Y is related to the observed data and not Y itself. The missing data are a random sample of the complete data, after conditioning on the observed data.
Missing not at random (MNAR)	$P(R\|Y_{Obs}, Y_{Mis}) = P(R\|Y_{Obs}, Y_{Mis})$	The missingness on Y is dependent on the missing values themselves. The distribution of the missing values is unrecoverable because the predictors of nonresponse are missing.

[1] R represents the missing data pattern (i.e., 1 if the value is missing, 0 if the value is observed). Y_{Mis} is the notation for the values of the missing data; Y_{Obs} represents the values of the observed scores.

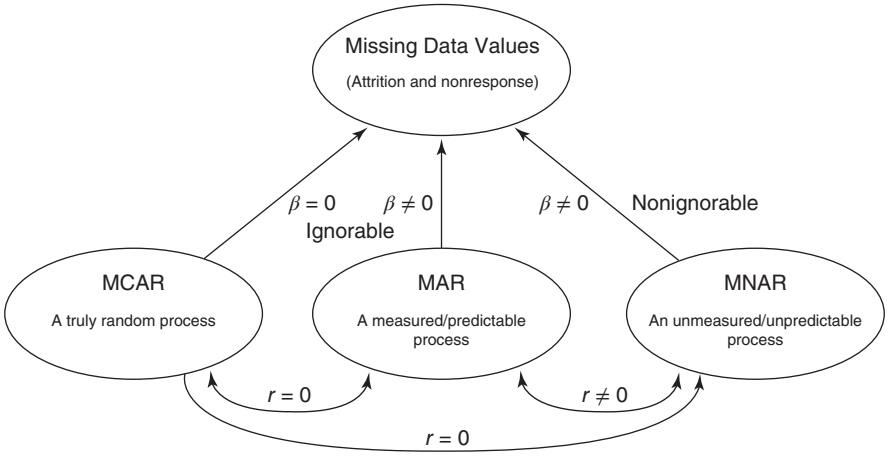


Figure 17.1 The mechanisms of missing data and their potential associations with the missing data and the missing values. (*Note:* The MCAR variables are necessarily uncorrelated with MAR variables and MNAR variables. MAR and MNAR variables can have varying overlap and the higher the overlap the less the influence of the MNAR process can be. The regression betas reflect the implied strength of the multiple linear prediction of the missing values from the set of possible variables that are classified as either in MCAR, MAR, or MNAR set.) *Source:* Little (2013). Used with permission of Todd D. Little.

many classic methods for treating missing data assume that all missing data are due to the MCAR mechanism (Little & Rubin, 2002). When the data are MCAR it means that every missing data treatment, except for single imputation techniques, will produce unbiased parameter estimates. When the pairwise deletion method is used with MCAR missing data, for example, estimates will be perfectly accurate. Here, the problem is that the missing data have undermined the power of the statistical model and type II errors are now likely to arise (i.e., failing to detect a true effect as being significant; Schafer & Graham, 2002).

Figure 17.1 shows the lack of association between the MCAR mechanism and the missing data as a predictive relationship that is necessarily zero and the correlation of the MCAR mechanism with the other two mechanisms is also necessarily zero. Table 17.2 shows that the conditional probability of the missingness, R, is not associated with the values of the missing variables (Y_{Mis}) or with the values of the observed variables (Y_{Obs}), and thus conditional independence holds and the missingness can be ignored.

Using the hypothetical example above, some children are missing the teacher-rated adjustment score because one teacher had recently moved, one teacher had the flu, one teacher refused because the child had just arrived in his classroom, one teacher lost the protocol, one teacher was distracted by a health issue, and so on. In each of these cases, a unique reason for each missing data point is to blame—the missing data are due to the whims of truly chance events, none of which are related to any of the variables under examination. Similarly, if a random subset of the completed adjustment protocols were accidentally shredded, this would also constitute an MCAR mechanism. In these scenarios, a principled missing data tools will, on average, reproduce the .5 correlation that exists in the population. In other words, the truly chance events have not introduced bias in the estimated correlation. The

only problem in this scenario is the loss in power that the lost data have caused. In this scenario the cause of missingness is "ignorable" because it does not need to be explicitly accounted for in the imputation or analysis models (as opposed to the missing not at random mechanism; Rubin, 1976). In other words, when using principled missing data tools, the MCAR mechanism will not have an impact on the estimated parameters of an analysis model (Figure 17.1).

Missing at Random

The second mechanism of missing data is labeled, confusingly, missing at random (MAR). When data are MAR it means that the causes of the missing data are in the observed data. Here, the values of the missing data can be regarded as a random effect that can be predicted by other variables in the data set. The MAR mechanism, therefore, is also an ignorable mechanism in so far as values of the missing data can be accurately recovered using a modern missing data model. In this regard, therefore, both MCAR and MAR are considered ignorable because the consequences for conclusions are negligible; that is, when employing modern, principled missing data tools, the parameter estimates of any statistical model fit to the data will be unbiased when either or both of these mechanisms are involved. As seen in Table 17.2, under the MAR mechanism, the only predictors of the missing data pattern (R) are contained in the observed data (Y_{obs}). So the missing values (Y_{mis}) can be considered a purely random sample of the complete data (Y), after accounting for the values of the observed data (Y_{obs}).

Using the hypothetical example above, some children are missing the parental involvement scores because a large percentage of primary care givers failed to fill out portions of the parental protocol. The reason these primary care providers did not complete the protocol is because they are consumed with the responsibilities and demands of care providing. The questionnaire protocol contains variables that record important characteristics of these care providers that were collected during the intake assessment by a trained interviewer. The variables include the person's role as a primary care provider or not, the number of hours per day spent providing care, employment status, the number of hours spent working per week, and the like. These variables are strong predictors of the reason for the missing responses. Using these variables in a modern treatment procedure would recover the lost information and would provide unbiased parameter estimates from a given statistical model.

The MAR assumption is often tested by predicting the missing data pattern from variables that are on the data set (e.g., by using the Little, 1988, test for MCAR or logistic regression). If the results of these tests are nonsignificant, then one of two reasons is possible: either the data are MCAR, or they are MNAR. Unfortunately, the ability to differentiate between these two mechanisms is challenging. In many ways, finding evidence of predictive links would be a preferred outcome of a missing data screening because it would indicate evidence of an ignorable MAR process.

Missing Not at Random

The third mechanism of missing data is the nefarious one. Here, the data are missing for a systematic reason but this systematic reason for the missingness is not accessible to the researcher. In this situation, data are MNAR. That is, there is not a predictor of the missingness that a researcher can use. In our example, parents who have low involvement in their children's education may be the least likely to turn in the survey for all the same reasons that they have low involvement to begin with (e.g., being overly busy). This selective responding results in a situation where the lowest values on parental involvement are missing, and the variable that predicts missingness is parental involvement itself.

In situations like these, the variable needed to predict the missingness is, itself, missing. Another way to describe this mechanism is that the data are missing for a reason, but the variable representing the reason is not on the observed data set (i.e., because it is part of Y_{Mis} in Table 17.2). Under the MNAR mechanism, the equation for predicting the missingness (R) does not simplify—unlike MCAR and MAR, the missing values themselves (Y_{Mis}) predict unique information in R. This means that the information that is available in the data set is not sufficient to recover the relations among variables. The MNAR circumstance is a non-ignorable situation because it will have consequences on the validity of any conclusions. The information needed to correct the data for the reasons is simply not available because it is missing. Because the observed data cannot predict the missing values, if these missing values were imputed, the imputations would be constructed as though the missingness mechanism were MCAR. However, the results would be biased because the reason for the missingness is unrecoverable.

Models that are designed to recover missing information when the data are MNAR are beyond the scope of our discussion because (1) the methods for preventing bias are complex models that require making strong untestable assumptions (see Enders, 2010, 2011; Muthen, Kaplan, &

Hollis, 1987); and (2) with planning, an MNAR mechanism can be converted to a MAR mechanism (Collins, Schafer, & Kam, 2001; Schafer & Graham, 2002; Yoo, 2009; Yuan, 2009a). For example, a classic example of an MNAR mechanism applies to income reported in net or gross annual salary. Very rich participants are more likely to leave this information missing than are the less wealthy participants. In this case the cause of the missingness is itself missing. There is no variable on the data set that would predict it and, therefore, it would not be recoverable. On the other hand, the research protocol could include variables that are known correlates of income. For example, job title, years of education, square-footage of the primary home, make and model of the primary automobile, and political affiliation. Variables such as these will, in a multivariate combination, provide information that now correlates with the pattern of missingness on income. Variables such as these are proxies of the missing information. A well-planned protocol would strive to have such proxies of important information that might be missing as an MNAR process. In the developmental psychopathology literature, sensitive variables such as physical, emotional, and sexual abuse might be missing because the person has been abused in some way. If the protocol contains variables that are expected to predict abuse the process would fall under the MAR mechanism because these variables predict missingness.

MODERN MISSING DATA METHODS

Table 17.1 reviews a range of classic techniques for dealing with missing data, from the simplest (e.g., listwise deletion, mean imputation) to more complex (e.g., stochastic regression imputation, hot deck imputation). As described there, none of these methods produce ideal results. At best, some of these methods can produce unbiased parameter estimates when the missingness is MCAR (e.g., listwise or pairwise deletion) or when it is MAR (e.g., stochastic regression imputation). At worst, some of the methods produce biased results even under MCAR (e.g., mean imputation, regression imputation). In addition, all of these methods either lack power (i.e., any deletion method) or result in inflated type I error rates (i.e., any single imputation method). Though it can be justifiable to use one of these methods when there is only a very small amount of missingness, using any of these techniques to deal with a substantial amount of missing data risks biased estimates, low power, or inflated type I error. In contrast to all of these methods, multiple imputation and full information maximum likelihood produce unbiased and fully efficient (i.e., powerful) parameter estimates when their assumptions are met. In the next sections, we describe these two methods in detail.

FULL INFORMATION MAXIMUM LIKELIHOOD

Full information maximum likelihood (FIML) is a state-of-the-art missing data tool that represents the culmination of an extensive history of research into maximum likelihood solutions to the missing data problem (e.g., Beale & Little, 1975; M. B. Brown, 1974; C. H. Brown, 1983; Carter & Meyers, 1973; Chen & Fienberg, 1976; Dempster, Laird, & Rubin, 1977; Enders, 2001b; Enders & Bandalos, 2001; Gold & Bentler, 2000; Hartley, 1958; Hartley & Hocking, 1971; Healy & Westmacott, 1956; Jamshidian & Bentler, 1999; Kenward, Lesaffre, & Molenberghs, 1994; Kenward & Molenberghs, 1998; Little & Rubin, 1989; Orchard & Woodbury, 1972; Rubin, 1991). The framework for FIML was formalized by Anderson (1957) as a way to derive ML estimates directly from incomplete data. Through clever manipulation of the likelihood function, FIML can hide any missingness from the estimator and allow one to fit a statistical model directly in the presence of incomplete data, without any need to estimate values for the missing data (Arbuckle, 1996). This approach has a distinct advantage over EM and imputation-based missing data tools. Because there is no estimation of values for the missing data, there is also no artificial certainty introduced into the treated data (as opposed to EM and single imputation techniques) nor is there any need to quantify the uncertainty in the accuracy of the imputed values (as must be done with multiple imputation).

To get a conceptual idea of how FIML operates, consider your computer's monitor. The image on this monitor is made up of many rows of pixels, just as a data set is made up of many rows of responses. If a pixel dies in your monitor, you are still able to understand the image on the screen because you can use the information from surrounding pixels to infer the whole image, passing over the damaged one. This principle will hold true even if a relatively large proportion of the pixels in your monitor were to fail, so long as there are enough pixels remaining for you to connect the dots and extrapolate the implied complete image from the partial image on your screen. Similarly, the FIML log likelihood function (equation 17.2) achieves an analogous effect when used to fit a statistical model to incomplete data. By using only what is known from the observed data, FIML can estimate around the missing data and infer what the whole model should look

like without needing to know what the missing responses would truly be. In this way, just as you can look at a damaged computer monitor and still understand how the complete image would appear, FIML can be applied to an incomplete data set to produce estimates that correctly describe the entire sample.

While the underlying mathematics can get complicated, the overarching principles guiding FIML estimation are relatively simple. First we must consider how a maximum likelihood estimator transforms the information contained in the observed data into a set of parameter estimates. Since FIML is based on normal-theory ML estimation, the multivariate normal loglikelihood function is a natural starting point for this discussion. Equation 17.1 gives the multivariate normal loglikelihood function:

$$\ell(\boldsymbol{\mu}, \boldsymbol{\Sigma}|\mathbf{Y}) = \sum_{i=1}^{N}\left[-\frac{k}{2}\ln(2\pi) - \frac{1}{2}\ln|\Sigma| - \frac{1}{2}(\mathbf{Y}_i - \boldsymbol{\mu})^T\Sigma^{-1}(\mathbf{Y}_i - \boldsymbol{\mu})\right], \tag{17.1}$$

where $\mathbf{Y}$ is a $N \times k$ matrix of incomplete data, $\boldsymbol{\mu}$ is the k-variate vector of unknown, population-level means of the variables in $\mathbf{Y}$, and $\boldsymbol{\Sigma}$ is the $k \times k$, unknown, population-level covariance matrix of the variables in $\mathbf{Y}$. The derivation of this equation falls beyond the scope of this chapter. However, for the sake of the current discussion, the reader need only recognize that equation 17.1 represents a reformulation of the multivariate normal distribution that provides a more computationally efficient way to compute ML estimates of $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$.

The beauty of FIML estimation is that it accomplishes its objective with only a small modification to equation 17.1. In the presence of missing data, equation 17.1 becomes the following:

$$\ell_{FIML}(\boldsymbol{\mu}, \boldsymbol{\Sigma}|\mathbf{Y}) = \sum_{i=1}^{N}\left[-\frac{k}{2}\ln(2\pi) - \frac{1}{2}\ln|\Sigma_i| - \frac{1}{2}(\mathbf{Y}_i - \boldsymbol{\mu}_i)^T\Sigma_i^{-1}(\mathbf{Y}_i - \boldsymbol{\mu}_i)\right]. \tag{17.2}$$

The only difference between equations 17.1 and 17.2 is that equation 17.2 includes subscripted versions of $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$. This subtle distinction allows each row in $\mathbf{Y}$ to have unique versions of $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$, which allows the dimensions of $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$ to differ between observations. When the dimensions of $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$ can differ across observations, it is possible for the final aggregated estimates of those parameters to be based on only the observed data, thereby hiding the nonresponse from the estimator and removing the need to delete any data or fill in any values for the missing data. This small change to the likelihood function is the mathematical procedure that allows FIML to estimate around the missing data and infer a complete set of parameter estimates from incomplete input. As long as there is sufficient overlap in the questions with observed responses (i.e., the *coverage* is adequate), FIML estimation can borrow enough information across observations to effectively remove any need to consider what the true values of the missing data would have been.

In practice, FIML estimation is quite convenient because it works behind the scenes to produce accurate parameter estimates without requiring the user to give much thought to the missing data (Enders, 2006, 2010; Muthen & Muthen, 2012). Consider the case of many participants filling out the same survey (i.e., a standard cross-sectional design). Even if some of those subjects do not complete the survey, we would still like to fit a model that will allow us to draw accurate conclusions about the entire sample, but we may not wish to go through the process of filling in values for the missing data. FIML estimation can help us accomplish this by using the observed responses to supplement the loss of information due to the missing responses (Enders, Dietz, Montague, & Dixon, 2006). In the end, FIML will produce unbiased and efficient estimates of the model parameters that we're actually interested in, without ever requiring us to consider the true values of the missing data.

While FIML is often the preferred method of addressing nonresponse in psychological studies, there are certain situations in which maximum likelihood methods are not applicable. For example, if a researcher wishes to calculate scale scores or parcels from incomplete data, they will find that there is no way to apply FIML estimation to those analyses without an additional loss of information (Enders, 2010). In such circumstances, it is beneficial to begin the substantive analysis with a complete data set. Thus, the analyst will be better served by a technique that fills in the missing values through imputation (Rubin, 1996).

Multiple Imputation

Single-imputation techniques all use some prediction mechanism (some better than others) to fill in the missing value with a plausible value. Once the missing data are thus filled in, the analysis of interest is run on the completed data set. A fundamental problem with this approach is that there is no way to quantify the additional uncertainty that results from the missingness (Rubin, 1987; Schafer, 1997). For example, a data set with 200 cases and 5% missing data

contains much more information than the same data set with 75% missing data. With a single-imputation approach, both situations will result in statistics that have similarly sized standard errors and confidence intervals. Intuitively, we can see that this should not be the case—statistics based on data with 75% missingness should have more uncertainty than those based on data with 5% missingness. This is where single imputation fails.

Multiple imputation overcomes this problem through introducing a second source of variation by repeating the imputation process multiple times. In MI, a modified single imputation technique (usually some form of regression imputation) is repeated to simulate multiple plausible values for each missing data point. Because there are small differences in the values chosen to replace the missingness from one iteration of the MI algorithm to the next, the final answer is a plausible *distribution* for each missing value, rather than an arbitrarily chosen single value that replaces each missing datum (Rubin, 1987; Schafer, 1997).

With more than one imputation (traditionally three to five, but these days more often 20 or even 100), the amount of information loss can be measured by looking at how different the imputed data sets are (Harel, 2007). If very little information is lost (e.g., if only a few values are missing, or if the missing values are highly redundant with complete values), then the imputed data sets will be highly similar to each other; that is, there will be low "between-imputation" variability. If a lot of information is lost (e.g., if a high proportion of data are missing, or if the missing values contain a lot of unique information), then the imputed data sets will differ a lot, and there will be high between-imputation variability. When doing an analysis on imputed data sets, the standard errors of the produced statistics are adjusted using the between-imputation variability—the greater the between-imputation variability, the larger the standard error becomes, to reflect the missing information. In this way, multiple imputation produces both unbiased estimates and correct standard errors.

If we think of the missing data as random variables with a distribution of possible values—of which the imputed values are just one possibility from an infinitely large pool—it is natural to compare the rationale for the multiplicity of MI to the rationale for collecting large samples, in general (Rubin, 1987). For most substantive research, gathering larger samples is advisable because basing your inferences upon a larger pool of observations gives you more certainty in the veracity of your conclusions. The exact same reasoning can be applied to the repeated imputations of MI.

For the sake of argument, consider the following extreme example. Say you wish to examine the prevalence of claustrophobia among elementary school students. You could go to a local elementary school and ask a single student to rate his or her fear of confined spaces on a scale from 1 to 10. If that student gives a response of 9, and you don't question any other children, then you are limited to the information carried by that student's data when constructing your inference. In this situation, your only option would be to conclude that elementary school students universally suffer from severe claustrophobia. This approach is plainly ridiculous, and it is clear that sampling more students would likely alter your finding.

While most researchers are naturally skeptical of statistical inferences based on very small samples, they may not automatically apply this same reasoning to missing data analysis. However, just as relying on only one child's data to model the severity of childhood claustrophobia can lead to highly erroneous findings, so too can blindly trusting the single set of imputed values produced by a single-imputation technique (Schafer, 1997). In the preceding example, it would be advisable to sample many children and use that large sample to derive a probability distribution that describes the levels of claustrophobia among elementary school students. The same is true of imputation. It is bad practice to impute a single value and trust it without question. A much more trustworthy answer can be achieved by simulating many different values for the missing data (i.e., create multiple imputations) so that we can derive a distribution to describe the plausible values that the missing data may take in the population. This distribution of plausible values helps us understand the degree of uncertainty associated with any of the parameter estimates of a given model.

At a conceptual level, the MI approach is relatively straightforward. An MI analysis is a piecemeal endeavor that is broken into three phases: the imputation phase, the analysis phase, and the pooling phase (Enders, 2010). Suppose you wish to analyze an incomplete data set $Y = \{Y_{obs}, Y_{mis}\}$, where Y_{obs} is the observed portion of the data and Y_{mis} is the missing portion. In the imputation phase, you must fill in Y_{mis} with M different sets of plausible values (i.e., you must create M imputations). This process will result in M imputed data sets $Y^{(1)} = \{Y_{obs}, Y_{imp}^{(1)}\}$, $Y^{(2)} = \{Y_{obs}, Y_{imp}^{(2)}\}$, …, $Y^{(M)} = \{Y_{obs}, Y_{imp}^{(M)}\}$ in which the original missing values of Y are replaced by the M sets of imputations. In the analysis phase, you will use these imputed data sets to fit M replicates of your statistical model. This step will result in M distinct sets of model parameter estimates $\widehat{Q}^{(1)}$, $\widehat{Q}^{(2)}$, …, $\widehat{Q}^{(M)}$

(these could be fitted regression coefficients, for example). Finally, in the pooling phase, you will combine these M sets of estimates into a single pooled set of estimates from which you will draw your conclusions. This final pooling is most commonly accomplished by applying Rubin's Rules (Rubin, 1987).

The original framework for MI was proposed by Rubin (1978) and greatly expanded by Rubin (1987), but the flexibility of the underlying concept has left the door open for the development of many different algorithms with which the imputations themselves can actually be created. Table 17.3 shows a list of currently popular software packages that can implement MI. Although there are seven distinct software packages listed in Table 17.3, the estimation methods they use to produce the imputations can be broken into three basic classes, namely: Bayesian data augmentation (or Bayesian MCMC), multiple imputation with chained equations (or sequential regression imputation), and bootstrapped EM imputation. These three approaches can be further categorized into joint modeling and fully conditional specification techniques. The joint modeling techniques (data augmentation and bootstrapped EM imputation) treat all missing data simultaneously by applying a single parametric model that describes the joint distribution of all the missing values. Fully conditional specification techniques (multiple imputation with chained equations), on the other hand, treat each individual variable sequentially and model the conditional distribution of each individual variable's missing data separately (van Buuren & Groothuis-Oudshoorn, 2011).

We will begin by discussing joint modeling techniques because they were the first to become widely available (Schafer, 1997; Schafer & Olsen, 1998), and fully conditional specification techniques can be discussed as special cases of the joint modeling approach.

Data Augmentation

Data augmentation was one of the first algorithms to become popular for use in creating multiple imputations. Data augmentation is a special application of Gibbs sampling that was proposed by Tanner and Wong (1987) as a method of estimating a Bayesian posterior distribution of model parameters when the underlying data are incomplete. These parameters can then be used to construct the multivariate regression equations needed to impute the missing data. The process is iterative, and accomplished in two steps. The first step, or imputation (I) step, is basically a multivariate stochastic regression imputation. During the I-step, the most up-to-date values for the mean vector $\boldsymbol{\mu}$ and the covariance matrix $\boldsymbol{\Sigma}$ (user specified starting values are used in the first I-step) are used to construct the coefficients of a series of multivariate regression equations (one for each distinct pattern of missingness). A randomly drawn, multivariate normally distributed vector of residual terms is then added to each of the predicted values from these equations and the resulting quantities are used to replace the missing values. Thus, after each I-step, all missing data are filled in, and the data set contains no missingness. From the Bayesian perspective, this is equivalent to creating the imputations by randomly sampling from a *posterior predictive distribution* of the missing data (Enders, 2010).

The second step, or posterior (P) step, uses the imputed data set from the preceding I-step to calculate updated values for $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$. These new values for $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$ are then used to construct the Bayesian posterior distributions for the mean vector $P(\boldsymbol{\mu}|\mathbf{Y}, \boldsymbol{\Sigma})$ and covariance matrix $P(\boldsymbol{\Sigma}|\mathbf{Y}, \boldsymbol{\mu})$. Finally, MCMC simulation techniques are used to randomly draw simulated values for the mean vector $\boldsymbol{\mu}^*$ and the covariance matrix $\boldsymbol{\Sigma}^*$ from these posterior distributions. These simulated values are then passed on to the next I-step, where they are used to construct updated regression coefficients for use in the next round of stochastic regression imputation.

This process is repeated a large number of times (e.g., 5000 I and P step pairs) and every n-th imputed data set produced by the I-step is saved. It is important to ensure that the number of imputed data sets to discard ($n - 1$) is large enough that the imputed values in subsequently saved data sets are statistically independent of each other (Baraldi & Enders, 2010). When M data sets have been saved, the algorithm terminates and these saved data sets make up the final collection of multiply imputed data sets.

Bootstrapped EM

Another popular joint modeling technique is the bootstrapped EM algorithm that was developed by Honaker and King (2010) and implemented in the R (R Core Team, 2012) package Amelia II (Honaker, King, & Blackwell, 2011) as a way to address the computational inefficiency of traditional data augmentation approaches. The bootstrapped EM algorithm is very simple at a conceptual level. First, standard bootstrapping procedures are applied and the incomplete data are resampled with replacement to create M incomplete bootstrapped samples of the same dimension as the original data set. Next, the EM algorithm is applied to each of these M samples to create M ML estimates of the mean vector $\boldsymbol{\mu}$ and covariance matrix $\boldsymbol{\Sigma}$

TABLE 17.3 Software Packages That Implement Multiple Imputation

Software (citation)	Estimation technique	Pros	Cons
Amelia II (Honaker, King, & Blackwell, 2011)	Bootstrapped EM Algorithm	Robust convergence Models time when imputing longitudinal data Freeware/Open Source	Only supports MVN imputation models Applies the same imputation model to all variables
mice (van Buuren & Groothuis-Oudshoorn, 2011)	Chained Equations	Accommodates mixed variable types Applies the correct imputation model to categorical and count variables Maintains factor labels for imputed values Offers numerous elemental methods Allows unique imputation models for each variable Freeware/Open Source Imputes multilevel data	Imputed values can be sensitive to the order that the variables are visited
mi (Su, Gelman, Hill, & Yajima, 2011)	Chained equations	Accommodates mixed variable types Applies the correct imputation model to categorical and count variables Allows unique imputation models for each variable Freeware/Open Source Includes many imputation diagnostics	Imputed values can be sensitive to the order that the variables are visited
Mplus (Muthen & Muthen, 2012)	Bayesian MCMC	Accommodates many different imputation models Imputes multilevel data Allows user-specified imputation models Categorical variables are imputed using probit models	Unrestricted models can exhibit poor convergence for large imputation models Applies the same imputation model to all variables Does not implement the full flexibility of the MICE approach
	Chained equations	Accommodates mixed variable types Allows unique imputation models for each variable	Imputed values can be sensitive to the order that the variables are visited Does not implement the full flexibility of the MICE approach Limited elemental methods
SAS 9.3: Proc MI (SAS Institute, 2011)	Data augmentation	Uses one of the best studied MCMC algorithms available	Only supports MVN imputation models Applies the same imputation model to all variables
	Chained equations	Accommodates mixed variable types Applies the correct imputation model to categorical variables Allows unique imputation models for each variable	Does not implement the full flexibility of the MICE approach Imputed values can be sensitive to the order that the variables are visited
SPSS: Missing Values (IBM Corp, 2012)	Chained equations	Accommodates mixed variable types Applies the correct imputation model to categorical variables Allows unique imputation models for each variable	Imputed values can be sensitive to the order that the variables are visited Does not implement the full flexibility of the MICE approach
Stata: mi (StataCorp, 2013)	Chained equations	Accommodates mixed variable types Applies the correct imputation model to categorical variables Allows unique imputation models for each variable	Imputed values can be sensitive to the order that the variables are visited
	Data augmentation	Uses one of the best studied MCMC algorithms available	Only supports MVN imputation models Applies the same imputation model to all variables

(Table 17.1). Finally, the M estimates of $\boldsymbol{\mu}$ and $\boldsymbol{\Sigma}$ are used to carry out M multivariate stochastic regression imputations which are applied to the original incomplete data, just as in the I-step of traditional data augmentation. This process results in M replicates of the original data, each of which contain a different set of imputed values, in other words, M multiply imputed data sets.

Multiple Imputation with Chained Equations

Multiple imputation with chained equations (MICE) has been gaining in popularity over recent years. It is unique among the algorithms discussed here because it is a fully conditional specification technique rather than a joint modeling technique. Therefore, MICE does not seek to treat all variables simultaneously; rather, the MICE

approach sequentially addresses each variable individually (van Buuren, 2012). While this is an important distinction, with considerable implications for the usage of the MICE approach, the estimation process underlying MICE is essentially identical to data augmentation. The only meaningful difference is that joint modeling techniques treat the imputation as a large multidimensional problem, in which the I-step performs a multivariate stochastic regression imputation and the P-step draws a k-variate $\boldsymbol{\mu}^*$ and $k \times k$ $\boldsymbol{\Sigma}^*$ from the joint posterior distributions $P(\boldsymbol{\mu}|\mathbf{Y}, \boldsymbol{\Sigma})$ and $P(\boldsymbol{\Sigma}|\mathbf{Y}, \boldsymbol{\mu})$. The MICE I- and P-steps, on the other hand, reduce to repeatedly solving k univariate analogues of the data augmentation I- and P-steps, where k is the number of incomplete variables to be imputed. In other words, the MICE I-step imputes the missing portion of only a single variable at a time, and the MICE P-step simulates only the univariate mean μ^* and variance σ^{2*} of that focal item.

This approach affords a great deal of flexibility to the analyst because each variable's imputations can be created from its own distinct regression equation (van Buuren & Groothuis-Oudshoorn, 2011). This approach also allows specification of appropriate posterior distributions for the parameters simulated in the P-step even in cases where the joint distribution of all of these parameters may not have a well-defined form (van Buuren, 2012). For example, suppose you have collected data from middle school students and you wish to analyze three variables: age, gender, and a count of aggressive outbursts in class. If there is missingness on these variables, you will be limited in your ability to treat that missingness with data augmentation or bootstrapped EM, since both techniques are based on the assumption that the incomplete data are jointly multivariate normally distributed. However, MICE allows an imputation model where age is imputed with a normal-theory regression model, gender with a logistic regression model, and the count of aggressive outbursts with a Poisson regression model. Thus, the MICE approach will produce correctly distributed imputed values for each special variable type while only requiring the user to run a single instance of the multiple imputation procedure.

Rubin's Rules

Rubin's rules are a simple set of formulae given by Rubin (1987) that allow pooling of the M distinct sets of parameter estimates derived from the analysis phase of an MI analysis. Recall that the product of the MI analysis phase is a set of M parameter estimates $\widehat{Q}^{(1)}, \widehat{Q}^{(2)}, \ldots, \widehat{Q}^{(M)}$. The *multiple imputation point estimate* is simply the arithmetic mean of these M individual parameter estimates:

$$\overline{Q} = \frac{1}{M}\sum_{m=1}^{M} \widehat{Q}^{(m)}, \tag{17.3}$$

where $\overline{Q}$ is the pooled point estimate, $\widehat{Q}^{(m)}$ is the parameter estimate from the m-th imputation, and M is the number of imputations.

Pooling the variance components is a little more involved than pooling the point estimates and requires three distinct steps. The pooled within-imputation variance is simply the arithmetic mean of the M parameters' sampling variances:

$$\overline{U} = \frac{1}{M}\sum_{m=1}^{M} SE^2_{\widehat{Q}(m)}, \tag{17.4}$$

where $\overline{U}$ is the pooled within-imputation variance, $SE^2_{\widehat{Q}(m)}$ is the squared standard error for the parameter estimate from the m-th imputation, and M is the number of imputations. The next step is calculating the between-imputation variance. The between-imputation variance simply captures the dispersion in the imputed values across all M imputations. It is calculated via the following formula:

$$B = \frac{1}{M-1}\sum_{m=1}^{M} \left(\widehat{Q}^{(m)} - \overline{Q}\right)^2, \tag{17.5}$$

where B is the between-imputation variance, $\widehat{Q}^{(m)}$ is the parameter estimate from the m-th imputation, $\overline{Q}$ is the multiple imputation point estimate, and M is the number of imputations. Finally, these two sources of variance must be combined into the *total sampling variance*. The total sampling variance is simply a weighted sum of the within and between-imputation variances:

$$T = \overline{U} + (1 + M^{-1})B, \tag{17.6}$$

where T is the total sampling variance, $\overline{U}$ is the pooled within-imputation variance, B is the between-imputation variance, and M is the number of imputations. With a large number of imputations, the influence of the weighting term in equation 17.66 becomes negligible and the total sampling variance reduces to a simple sum of the within and between-imputation variance.

Once the multiple imputation point estimate and total sampling variance have been calculated, they can be

combined into familiar test statistics. The MI Wald statistic is produced by combining the pooled components in the natural way:

$$t(v)_{MI} = \frac{\overline{Q} - Q'}{\sqrt{T}}, \tag{17.7}$$

where $\overline{Q}$ is the multiple imputation point estimate, Q' is the null hypothesized value of the parameter estimate, and T is the total sampling variance. This test statistic can be compared with a student's t distribution with degrees of freedom given by the following:

$$v = (M-1)\left[1 + \frac{\overline{U}}{\left(1 + M^{-1}\right)B}\right]^2, \tag{17.8}$$

where v is the degrees of freedom, $\overline{U}$ is the pooled within-imputation variance, B is the between-imputation variance, and M is the number of imputations. These components can also be combined into the following confidence interval for the multiple imputation point estimate:

$$(1-\alpha)\%CI_{MI} = \overline{Q} \pm t_{v,1-\alpha/2}\sqrt{T}, \tag{17.9}$$

where α is the probability of a type I error (i.e., significance level), $\overline{Q}$ is the multiple imputation point estimate, $t_{v,1-\alpha/2}$ is the cutoff value on the student's t distribution corresponding to the desired significance level and the degrees of freedom calculated via equation 17.8, and T is the total sampling variance.

Convergence

To ensure the quality of the imputed data sets when MCMC is used to impute data, convergence of MCMC needs to be carefully monitored. An MCMC procedure is deemed converged if the posterior distribution of the parameters in the imputation model reaches a stationary point. The convergence of MCMC ensures that the imputed data are drawn from the right posterior distribution. The iterations prior to convergence, termed burn-in iterations, need to be disregarded. Depending on the specific program one chooses, the number of burn-in iterations of MCMC is either decided by the program automatically or needs to be specified by researchers themselves. In Mplus, convergence of MCMC is monitored using a criterion termed potential scale reduction (PSR; Gelman & Rubin, 1992). The number of burn-in iterations is then specified based on this criterion. Detailed explanation of the PSR is beyond the scope of the chapter. What applied researchers need to know is that there will be one PSR for each parameter in the imputation model at each iteration of MCMC. According to Asparouhov and Muthén (2012), MCMC is deemed as converged if the worst PSR becomes less than 1.05 or 1.1 (Asparouhov & Muthén, 2012). In addition, graphical tools such as trace plots and autocorrelation plots can be very useful in assessing MCMC convergence. Trace plots display the change of simulation draws of each of the imputation parameters over iterations. When MCMC converges, the simulation draws should show random fluctuations over iterations. Autocorrelation plots are plots of autocorrelations between the simulated draws for each of the imputation parameters against lag. The autocorrelations usually decrease to zero after a certain number of lags. The faster the autocorrelations decrease to zero, the faster an MCMC procedure converges.

Due to their autocorrelations, the imputed values from successive iterations are also correlated. Because the imputed data sets need to be independent to produce correct standard error estimates, it is necessary to allow a number of iterations to lapse every time before saving an imputed data set. This strategy is termed *thinning* and the number of iterations between imputed data sets are termed *between-imputation iterations*. Usually 100 between-imputation iterations would be sufficient for most of cases. However, if one has a difficult data set with a large fraction of missing information, a larger value should be specified for the thinning interval. The autocorrelation plots can inform researchers of the appropriate number of between-imputation iterations.

There are also many things researchers can do to facilitate the convergence of MCMC (Asparouhov & Muthén, 2012). First, researchers need to make sure that there are no variables in the imputation model that are highly correlated with one another. Second, try not to include variables that do not predict the missing values in the imputation model (e.g., ID). Although, including them has no harm on the parameter estimates (Collins, Schafer, & Kam, 2001), they might cause convergence problems. Third, if there are missing data indicators (binary variables created to indicate whether a score is missing or not), they should not be included in the imputation model as their correlations with the variables having missing data are not identified. Fourth, the imputation model has to be identified. For single-level data, the number of observations on each of the variables

should be no less than the number of variables included in the imputation model. For example, if the variable with most of the missing data has 50 observations, then including more than 50 variables in the imputation model would create convergence problem. Finally, If MI does not converge and you don't know which variable causes the problem, try to include a subset of variables and add variables sequentially into the model.

Number of Imputations

The number of imputations that are needed for MI to give accurate and efficient standard errors depends on the amount of missing information (Bodner, 2008; Graham et al., 2007). Although MI standard errors are less efficient than those of FIML as long as the number of imputations is finite, practically speaking the incremental gain in efficiency from more imputations drops off very quickly (von Hippel, 2005). Until recently, five imputations was considered a sufficient number of imputations to yield standard errors virtually as efficient as FIML (Rubin, 1987). More recently, 20 imputations has emerged as the new minimum and many analysts recommend producing 100 imputed data sets to ensure comparability to FIML estimates (Bodner, 2008; Enders, 2010; Graham et al., 2007; Little, 2013).

A key consideration in deciding the number of imputations is the amount of missing information, which can be quantified in the *fraction of missing information* (FMI) statistic (Bodner, 2008; Graham et al., 2007; Savalei & Rhemtulla, 2011). Recall that when more information is missing, there is greater variability between imputations. As such, each individual imputed data set is less reliable. A lower FMI means that the missing data are highly predictable (or there are very few missing data) and thus fewer imputations are needed to produce a reliable result, whereas a high FMI indicates that more imputations are needed due to the uncertainty of the missing values (Fraley, 1999; Rubin, 1987). White, Royston, and Wood (2011) suggested a rule of thumb that the number of imputations should be at least the fraction of missing information multiplied by 1000. That is, if 50% of the information is missing (FMI = .5), then the minimum number of imputations should be 50. Unfortunately, estimates of the FMI itself can fluctuate dramatically when few imputations are used. To get a reliable estimate of the fraction of missing information from MI, several hundred imputations are needed. As such, with advances in computing making it relatively quick and easy to do many imputations and combine the results across them using automated software, we recommend using 100 imputations whenever possible.

Above, we mentioned a type of missing data that we called structural missing such as items on a questionnaire that would not be asked of certain individuals because the items do not pertain to the person (e.g., asking boys about their menstruations). Multiple imputation procedures are unable to distinguish between structural missing and the nonresponse forms of missingness that we desire to recover. In these situations, we recommend creating a flag variable to indicate true missing from structural missing. The imputed values for the structural missing can then be reset to missing by using the flag variable.

PRACTICAL CONSIDERATIONS

Keep in mind that the primary goal of a modern missing data procedure is to reproduce the summary parameter estimates (Rubin, 1996). In the case of FIML estimation, the goal is to provide unbiased values of the parameter estimates from the statistical model being fit to the data (Arbuckle, 1996; Enders, 2010). As mentioned previously, all observations are used to inform the model's parameter estimates. With MI, the goal is to produce sufficient statistics that are unbiased (Rubin, 1987; 1996) and accurately reflect the statistical precision in the data. The sufficient statistics are the means, variances, and covariances among the variables (note that the range is not a sufficient statistic). The statistical model is fit at the level of the sufficient statistics and it produces unbiased parameter estimates that are equivalent to those of FIML (all other things being equal; Graham, et al., 2007). With MI, the person-level information is not part of the imputation objective (Rubin, 1996; Schafer & Graham, 2002). The imputation model does use plausible values for the cells that are missing but these values are not treated as scores for the person because many different plausible values can be used to optimize recovery of the unbiased sufficient statistics. The value assigned to a cell is more of a temporary place holder that is used to infer the population sufficient statistics. When a statistical model is fit to the aggregate data, the parameter estimates of this model will be unbiased. Imputed values for a given person, therefore, should be treated with caution. The distribution of the imputed values for a given person can be quite variable and any one imputation would not be a trustworthy estimate for that person (Schafer, 1997). It would be possible to produce an estimated value from the imputed scores that would be the mean of the imputed values with a confidence interval around this value to be used for assessment purposes, for example.

A second important objective of missing data analysis is accurate quantification of the information inherent in the

incomplete data (Harel, 2007). The missing information principle of Orchard and Woodbury (1972) states that the information of an incomplete data set can be partitioned into two quantities: the observed information and the missing information. Modern missing data tools allow us to differentiate between these two types of information and accurately estimate their values (Savalei & Rehemtulla, 2012). While both MI and FIML quantify the data's information, they address the problem in two different ways. MI quantifies the missing information as a function of the between-imputation variance (Rubin, 1987; Schafer, 1997). As suggested above, this way of quantifying is another reason that it is advantageous to construct a large number of imputations—doing so will produce a more accurate estimate of the missing information. FIML, on the other hand, implicitly derives a corrected estimate of the observed information by ascribing more certainty to those parameter estimates that are derived from a larger number of observed responses (Enders, 2010).

Assumptions

Both FIML and MI are model based in the sense that the accuracy of their results rely on the assumptions underlying their respective models. For FIML, the missing data model is the analysis model (e.g., a structural equation model or regression model). For MI, the missing data model is the imputation model, which is specified separately. In both cases, to the extent that the model is false, the results of the procedure will suffer (Little & Rubin, 2002; Schafer & Olsen, 1998).

One common assumption is that the associations between variables, including the associations that account for missingness, are linear (Honaker & King, 2010). If the missingness is actually due to an interaction between two variables, for example, and that interaction term is omitted from the missing data model, the missingness will not be accounted for. In MI, new variables such as powered polynomials and product terms can be inserted into the imputation model to represent potential nonlinear effects of missingness. For example, socioeconomic status (SES) is a common linear correlate of dropout in longitudinal studies. If the rate of dropout increased in a nonlinear fashion at lower levels of SES, the squared powered term of SES (SES^2) can be included in the imputation model to recover the nonlinearity of the MAR process (Graham, 2009; von Hippel, 2009). In FIML, these nonlinear variables may be used as auxiliary variables (see next section).

Another common assumption is that of independence, which is violated when data are nested. Nested data are common in longitudinal data and samples collected from schools, where data points are nested within individuals, within classrooms, or within schools. In these cases, if the missing data model does not take into account the nested structure of the data, the complex relations between variables within and across clusters can be obscured, resulting in incorrect estimates of between- and within-cluster variability and incorrect standard errors (Beunckens, Molenberghs, Thij, & Verbeke, 2007). When dealing with nested data and using FIML, if the nested structure is accounted for in the model (e.g., by using a multilevel model or multilevel structural equation model; Muthén & Asparouhov, 2009) maximum likelihood estimation of the model will successfully recover the relations among the variables at the levels included in the model.

When using MI, it is important to remember to incorporate the nesting structure into the imputation model as well as the analysis model. If the imputation model does not account for nesting, the imputed values will underestimate the amount of variance at the individual level, leading to too-small standard errors, even if a correct multilevel model is used as the analysis model (Schafer, 1997). When more than one level of analysis is of interest, for example, if a researcher is interested in both the effects of student-level predictors such as student motivation as well as teacher-level predictors such as teacher experience, the former effects will be overestimated and the latter effects will be underestimated if the nesting is not properly modeled during multiple imputation (Reiter, Raghunathan, & Kinney, 2006). Several methods have recently been implemented in software to account for nesting in multiple imputation (e.g., mice—van Buuren & Groothuis-Oudshoorn, 2011; Pan—Schafer &Yucel, 2002; SHRIMP—Yucel & Raghunathan, 2006).

Another assumption of many modern missing data treatments is that the variables are multivariate normally distributed (Allison, 2006; Demirtas, Freels, & Yucel, 2008; Enders, 2001a; Gold, Bentler, & Kim, 2003; Honaker & King, 2010; Savalei, 2010a). Only a few studies have been done on the degree to which MI and FIML are robust to violations of this assumption and whether some modern procedures are better than others when the normality assumption is violated (e.g., Savalei & Bentler, 2005; Shin, Davidson, & Long, 2010). Although some work suggests that both normal-theory MI and FIML are robust to violations of normality (Demirtas et al., 2008; Enders, 2001a; Graham & Schafer, 1999; Yuan, 2009b), further research in this area is needed. We still recommend a modern imputation approach even when the assumptions are violated because the consequences of using another method

are graver than the lack of precision that nonnormal data might introduce (Graham, 2009; Schafer & Graham, 2002).

Auxiliary Variables

Auxiliary variables are those that are associated with the missing data pattern or the values of the missing responses, but are not substantively interesting to the researchers (Collins et al., 2001; Enders & Gottschall, 2011). That is, they are variables that capture a MAR missing data process. Recall that FIML and MI only result in unbiased estimates when the causes of missingness are included in the model (FIML) or the imputation model (MI).

When FIML estimation is used, auxiliary variables must be included in the analysis model. Graham (2003) described a method for including auxiliary variables in any structural equation model without changing the interpretation of the substantively interesting model parameters, called the "saturated correlates" approach. In a nutshell, this approach introduces auxiliary variables into the model by allowing them to correlate with other exogenous variables (e.g., predictors) in the model, without allowing them to predict unique variance in any dependent variables. In this way, the paths of interest are not affected, but the auxiliary variables are nonetheless part of the model. Once they are part of the model, the influence of auxiliary variables on missingness is taken into account. If auxiliary variables are not used when FIML estimation is employed, the MAR process is not likely to be represented (unless the MAR-associated variables just happen to be part of the desired analysis model; Enders, 2008, 2010; Graham, 2003).

Auxiliary variables are easier to deal with in MI. Here, auxiliary variables are just included in the imputation model, which typically contains the complete data set (Collins, et al., 2001; Schafer, 1997). In MI, there is no need to explicitly distinguish auxiliary variables from analysis variables—auxiliary variables can be any variables in the imputation model, whereas analysis variables are whichever variables are chosen, post-imputation, for use in the analysis. Once imputation is complete, there is no further need for auxiliary variables.

Two general strategies for including auxiliary variables are recommended. The first is the *inclusive strategy* (Collins et al., 2001). Here, all variables on a data set that are not part of the analysis model are included in the analysis as auxiliary variables. This strategy ensures that if a MAR association is contained among the set of auxiliary variables, its impact will be represented (Collins et al., 2001; Kreuter & Olson, 2011; Yoo, 2009). The inclusive strategy will generally become intractable when too many variables are included, particularly because the auxiliary variables themselves will often contain missing values (Enders, 2008; 2010). The large number of variables coupled with the variety of missing data patterns that occurs can lead to an insurmountable estimation problem—the software simply will not converge on a solution (Honaker & King, 2010).

The *restrictive strategy* is an alternative approach that entails selecting variables that have an observable relationship with the missing data patterns, that is, those variables with a significant unique predictive association to be included as auxiliary variables (Enders, 2010). Van Buuren (2012) suggested including all variables that appear in the analysis model, the variables that are related to the variables having missing data, and the variables that are highly correlated with the target variables. In addition, including an incomplete auxiliary variable is beneficial, even if that variable contains MNAR missingness. However, variables that have too many missing data might introduce estimation problems, and should not be included in an imputation model. In general, the restrictive strategy tends to be less effective than the inclusive strategy (Collins et al., 2001). However, it is sometimes necessary to reduce convergence problems.

Both the inclusive strategy and the restrictive strategy often ignore potential nonlinearity in the MAR association of the variables on the data set (Collins et al., 2001). To allow an effective use of the inclusive strategy and to encourage broader representation of the potential MAR process, Howard, Rhemtulla, and Little (2015) introduced the use of principal components scores as auxiliary variables. The principal components strategy relies on the characteristic of component scores as distilled sources of much of the variance contained in a set of variables.

The basic strategy involves (1) doing a single imputation of the missing values using a stochastic regression imputation or EM imputation, (2) computing nonlinear variables including powered and interaction terms, and (3) reducing the resulting very large data set down to a handful of meaningful component scores. The first step uses precise predicted values as temporary substitutes for the values that are missing. The uncertainty issues for statistical inference are not a concern in this step because the predicted values are simply estimates for the calculation of the component scores. In the second step, when nonlinear terms are calculated, the strategy is to be very broad. For example, each variable could be squared and all possible two-way interaction terms among the set of variables could conceivably be calculated.

Ongoing simulation work and comparative analysis on existing data sets indicates that using around 10 component scores is sufficient to capture the missing data relevant

information that is contained on many practical data sets (Garnier-Villarreal et al., 2013). These 10 component scores are included as the auxiliary variables in an FIML or MI analysis model.

Fraction of Missing Information

As suggested earlier, fraction of missing information (FMI) is probably the most useful value for quantifying the effect that missing data has on a statistical analysis. Allison (2002) gave a conceptual definition of FMI as "how much information is lost about each coefficient because of missing data" (p. 48). FMI can also be interpreted as the proportion of the total sampling variance of a focal parameter Q_j that is attributable to the missing data (Rubin, 1987), in this sense it is much like an R^2 statistic of the missing data (Enders, 2010). Alternatively, the FMI shows how much more variable a given parameter estimate is with missing data than it would have been had all of the data been fully observed, thereby conveying information about the statistical efficiency lost to nonresponse (Harel, 2007; Savalei & Rhemtulla, 2012). FMI has a fundamental place in any missing data analysis because it is intimately tied to many aspects of the missing data problem. The FMI directly impacts power loss due to missingness (Savalei & Rhemtulla, 2012), rates of convergence for missing data algorithms (Fraley, 1999; Harel, 2007; Schafer, 1997), and the number of imputations required for MI (Bodner, 2008; Graham et al., 2007).

Although it seems natural to quantify the scope of a missing data problem by the raw percentage of cells in the data set that are missing (i.e., percent missing [PM]), FMI is actually the more appropriate metric from which to draw guidance (Harel, 2007). FMI can be viewed as showing the impact of the raw PM after accounting for the strength of the item interrelations in the observed data (Enders, 2010; Rubin, 1987). Consequently, FMI will often be lower than the simple PM because high variable correlations may mean that there is less missing information than missing data. On the other hand, particularly when the missing data mechanism is MAR, FMI can be much higher than the raw percentage of missing values (Rhemtulla, Savalei, & Little, 2014; Savalei & Rhemtulla, 2011).

The formula for FMI is usually given in terms of the variance components of an MI analysis. Schafer and Graham (2002) gave the following formula for a sample estimate of the FMI for a focal model parameter Q_j:

$$FMI_{j,\mathrm{MI}} = \frac{(1 + M^{-1})B_j}{\overline{U}_j + (1 + M^{-1})B_j} \approx \frac{B_j}{\overline{U}_j + B_j}, \qquad (17.10)$$

where B_j is the between-imputation variance of the focal parameter, $\overline{U}_j$ is the average within-imputation variance for the focal parameter, and M is the number of imputations. With a large number of imputations the formula for FMI reduces to the approximation shown rightmost in equation 17.10 (Savalei & Rhemtulla, 2012). This approximation makes clear the conceptual parallel of FMI and the R^2 statistic. Replacing B_j with between-subject variance and $\overline{U}_j$ with within-subject variance gives the familiar R^2 statistic from ANOVA.

FMI is often discussed in the literature as a natural consequence of MI that is not easily derived from ML-based missing data tools (e.g., McKnight, McKnight, Sidani, & Figueredo, 2007; Schafer, 2001), but Savalei and Rhemtulla (2012) appealed to the missing information principle of Orchard and Woodbury (1972) to give a simple method for estimating FMI in the course of a FIML-based analysis. The computational formula given by Savalei and Rhemtulla (2012) is the following:

$$FMI_{j,FIML} = 1 - \frac{SE^2_{j,Comp}}{SE^2_{j,FIML}}. \qquad (17.11)$$

where $SE^2_{j,Comp}$ is the squared standard error of the focal parameter $\widehat{Q}_{j,Comp}$ derived from complete-data ML estimation and $SE^2_{j,FIML}$ is the squared standard error of the focal parameter $\widehat{Q}_{j,FIML}$ derived from a FIML analysis.

The natural stumbling block of this approach is the apparent inability to estimate $\widehat{Q}_{j,Comp}$ from incomplete data, but an asymptotic approximation is readily available. The method described by Savalei and Rhemtulla (2012) involves breaking the analysis into two phases. Phase 1 consists of estimating $\widehat{Q}_{j,FIML}$ by applying FIML to the incomplete data. Phase 2 consists of using ordinary ML estimation to fit an identical model to the model implied mean vector and covariance matrix produced by Phase 1. Because FIML produces model implied moments that are asymptotically equal to their complete-data analogues, the estimates derived from the Phase 2 model are asymptotically equal to the complete data ML estimates $\widehat{Q}_{j,Comp}$. Furthermore, because $SE^2_{j,Comp}$ and $SE^2_{j,FIML}$ are natural byproducts of estimating $\widehat{Q}_{j,Comp}$ and $\widehat{Q}_{j,FIML}$, these two models produce all the necessary components of equation 17.11, and FMI is easily calculated.

Assessing Model Fit with Missing Data

In many popular modeling paradigms assessing how well an estimated model recreates the relationships observed in

the sample data (i.e., the *model fit*) is an integral component of the research process (Davey, Savla, & Luo, 2005). Latent variable modeling techniques, in particular, have been developed hand-in-hand with ever more sophisticated model fit indices. Consequently, the consumers of research that employs latent variable models have come to expect a complement of fit indices to be reported alongside parameter estimates and test statistics. The ability to accurately assess model fit, therefore, is of paramount importance to any researcher seeking to employ a latent variable model. Unfortunately, missing data complicate this issue.

As with any other statistic we may wish to estimate, fit indices were developed under the assumption of completely observed data, and special consideration must be given to their calculation when they are derived from models fit to incomplete data (Davey et al., 2005; Savalei, 2010b). The assessment of model fit is problematic whether employing FIML or MI, but the issues inherent in the two approaches are unique, so we will address them separately below.

If using FIML estimation, one of the biggest issues arises from the unique form of the likelihood function that FIML maximizes to derive its parameter estimates. Traditional, complete-data, ML estimation is customarily accomplished by minimizing the discrepancy between the observed data moments and the model implied moments via the discrepancy function given by Joreskog (1967):

$$F_{ML} = \ln|\Sigma| + \text{tr}\left(S\Sigma^{-1}\right) - \ln|S| - p \tag{17.12}$$

where Σ is the model implied covariance matrix, S is the covariance matrix of the observed data, p is the total number of manifest variables, $|\cdot|$ represents the determinant of a matrix, and tr(.) is the trace of a matrix. Once F_{ML} is calculated, it can be used to compute the likelihood ratio (chi-squared) statistic in the following way:

$$\chi^2_{ML} = (N-1)F_{ML}, \tag{17.13}$$

where F_{ML} is the minimized discrepancy function from equation 17.12, and N is the total sample size. However, as described previously, FIML maximizes the joint case-wise loglikelihoods given by equation 17.2. Unfortunately, the FIML χ^2 cannot be calculated by simply replacing F_{ML} in equation 17.13 with ℓ_{FIML} from equation 17.2 (even though they are analogous quantities; Enders, 2001c).

Fortunately, the FIML χ^2 can be computed without a great deal of additional effort on the part of the researcher. Rather than naively replacing terms in equation 17.13, the FIML χ^2 is calculated by taking the difference between the maximized loglikelihood functions associated with the researcher's hypothesized model and a fully saturated model:

$$\chi^2_{FIML} = -2\left[\ell_{FIML}\left(\boldsymbol{\mu}_1, \boldsymbol{\Sigma}_1|\mathbf{Y}\right) - \ell_{FIML}\left(\boldsymbol{\mu}_0, \boldsymbol{\Sigma}_0|\mathbf{Y}\right)\right] \tag{17.14}$$

where $\ell_{FIML}\left(\boldsymbol{\mu}_1, \boldsymbol{\Sigma}_1|\mathbf{Y}\right)$ represents the loglikelihood function from equation 17.2 fit to the mean vector $\boldsymbol{\mu}_1$ and covariance matrix $\boldsymbol{\Sigma}_1$ implied by the researcher's hypothesized model, and $\ell_{FIML}\left(\boldsymbol{\mu}_0, \boldsymbol{\Sigma}_0|\mathbf{Y}\right)$ represents this same loglikelihood function fit to a fully saturated mean vector $\boldsymbol{\mu}_0$ and covariance matrix $\boldsymbol{\Sigma}_0$. The fit of the hypothesized model can then be assessed by comparing χ^2_{FIML} with a central χ^2 distribution with degrees of freedom equal to the difference in the number of free parameters in the hypothesized and fully saturated models. Model fit can also be judged by computing derived fit indices (e.g., CFI, TLI, RMSEA) by substituting χ^2_{FIML} for χ^2_{ML} in their standard formulae (Wu, West, & Taylor, 2009).

The formulation of equation 17.14 may not map intuitively back to the two step procedure illustrated by equations 17.12 and 17.13, but the underlying rationale becomes more clear if we consider $\ell_{FIML}\left(\boldsymbol{\mu}_1, \boldsymbol{\Sigma}_1|\mathbf{Y}\right)$ to carry roughly the same information as the model-implied components of equation 17.12 and we think of $\ell_{FIML}\left(\boldsymbol{\mu}_0, \boldsymbol{\Sigma}_0|\mathbf{Y}\right)$ as carrying the same information as the observed data components of equation 17.12.

An additional concern arises when using incremental fit indices (i.e., CFI, TLI) to assess the fit of FIML-based models that employ the saturated correlates approach to include auxiliary variables (Enders, 2010; Graham, 2003). The saturated correlates approach necessarily adds extraneous variables to the model, but the software may not recognize the unique status of the new variables. Therefore, relying on the default independence model when calculating incremental fit indices will lead to incorrectly calculated degrees of freedom and fit indices that suggest better fit than they should. Interested readers can find a more detailed discussion of this issue and a description of the procedure for correct specification of the null model in Enders (2010, pp. 137–140). Although this issue still holds, in general, several software packages have implemented the ability to compute the correct null model when employing the saturated correlates approach. If the auxiliary variables are correctly entered into the model, the Mplus software (Muthén & Muthén, 2012) will calculate the correct null model (Asparouhov & Muthén, 2008). By using the R package semTools (Pornprasertmanit, Miller, Schoemann, & Rosseel, 2013) to introduce the auxiliary variables, the incremental fit indices from models fit with the R package

lavaan (Rosseel, 2012) are also calculated from the correct null model.

The biggest issue facing researchers who wish to assess model fit when employing MI is not calculating the χ^2 statistic, but correctly pooling the inevitable replicates of the χ^2. As with any implementation of MI, the analysis phase of an MI-based latent variable modeling enterprise will result in M distinct replicates of the χ^2 statistic. Unfortunately, Rubin's rules were not created to accommodate latent variable modeling, and they cannot be used to pool the χ^2. Therefore, as one would expect, the fit indices that are derived from the χ^2 statistic (e.g., RMSEA, CFI, TLI) cannot be pooled using Rubin's Rules either.

The issue of pooling MI-based fit indices has been explored in the statistical and methodological literature at length, but a widely applicable and easily implemented solution has yet to be proposed. Unfortunately, simply averaging the M χ^2 statistics is an untenable solution that will lead to a pooled statistic that is much too large and will reject the hypothesized model too often (i.e., the pooled statistic will have a highly inflated type I Error rate; Asparouhov & Muthén, 2010a; Lang, 2013).

One successful, albeit somewhat tedious, approach that has been implemented in several popular software packages was developed by Meng and Rubin (1992). Their approach is a multiple step procedure that centers on calculating a χ^2 statistic of the form given by Equation 17.14 (i.e., the difference in the minimized likelihoods of a full and restricted model). The first step involves aggregating the M χ^2 statistics and parameter estimates produced by the analysis phase of an ordinary implementation of MI:

$$\overline{\chi^2} = \frac{1}{M}\sum_{m=1}^{M}\chi^2_m, \tag{17.15}$$

$$\overline{Q_0} = \frac{1}{M}\sum_{m=1}^{M}\widehat{Q}_{0m}, \tag{17.16}$$

$$\overline{Q_1} = \frac{1}{M}\sum_{m=1}^{M}\widehat{Q}_{1m,} \tag{17.17}$$

where χ^2_m, $\widehat{Q}_{om}$, and $\widehat{Q}_{1m}$ are the estimated χ^2 statistic, null model (fully saturated) parameter estimates, and hypothesized model parameter estimates, respectively, derived from the m-th imputed data set, and M is the number of imputations.

The next step is to calculate an updated χ^2 statistic for each imputed data set by fixing the null and hypothesized parameter values at $\overline{Q_0}$ and $\overline{Q_1}$, respectively, and evaluating a special case of equation 17.14 within each of the imputed data sets:

$$\widetilde{\chi^2_m} = -2[\ell_{ML}(\overline{Q}_1|Y^{(m)}) - \ell_{ML}(\overline{Q}_0|Y^{(m)})], \tag{17.18}$$

where $\ell_{ML}(\overline{Q}_0|Y^{(m)})$ represents the ordinary, complete-data, loglikelihood function fit to the m-th imputed data set and the aggregated point estimates given by equation 17.16, and $\ell_{ML}(\overline{Q}_1|Y^{(m)})$ represents this same loglikelihood function fit to the same imputed data set and the aggregated point estimates given by equation 17.17.

Next, the m estimates of $\widetilde{\chi^2_m}$ are pooled by simply taking their average across the imputed data sets:

$$\overline{\widetilde{\chi^2}} = \frac{1}{M}\sum_{m=1}^{M}\widetilde{\chi^2_m}, \tag{17.19}$$

where $\widetilde{\chi^2_m}$ are the updated χ^2 statistics from equation 17.18, and M is the number of imputations. Finally, the adjusted χ^2 statistic can be calculated as follows:

$$\chi^2_{imp} = \frac{\overline{\widetilde{\chi^2}}}{1+r_3}, \tag{17.20}$$

where $\overline{\widetilde{\chi^2}}$ is the pooled χ^2 statistic from equation 17.19, and r_3 is a correction factor given by

$$r_3 = \frac{M+1}{(M-1)(p_1-p_0)}(\overline{\chi^2} - \overline{\widetilde{\chi^2}}), \tag{17.21}$$

$\overline{\chi^2}$ is the aggregate χ^2 statistic from equation 17.15, $\overline{\widetilde{\chi^2}}$ is the pooled χ^2 statistic from equation 17.19, p_1 and p_0 are the number of freely estimated parameters from the hypothesized and null models, respectively, and M is the number of imputations.

The χ^2_{imp} statistic has been shown to perform well when compared to the χ^2_{FIML} statistic given by equation 17.14 when the amount of missing data is low or the number of imputations is large (Asparouhov & Muthén, 2010a). Though the process of calculating χ^2_{imp} manually can be onerous, this approach is well implemented in the Mplus software package (Muthén & Muthén, 2012) as the default method of pooling χ^2 statistics when fitting ML-based, single-level structural equation models. It is also available in the R package semTools (Pornprasertmanit et al., 2013) as an option for pooling χ^2 statistics from structural equation models estimated with the R package lavaan (Rosseel, 2012).

The question of how to best assess model fit with missing data has been a very active area of missing data research over the course of the last 20 years, and many interesting applications have been developed whose discussion falls outside the scope of this chapter (e.g., the application of parametric bootstrapping to the assessment of model fit by Enders, 2002; Savalei & Yuan, 2009). One such family of techniques still warrants a brief introduction, though. These are the so-called two-stage estimators that are based on the EM algorithm. The two-stage estimation approach was developed by Yuan and Bentler (2000) as a way to carry out ML-based missing data analysis under difficult situations, such as under nonnormal data distributions. Before Graham's (2003) saturated correlates approach was developed and implemented, a two-stage approach was the only way to get auxiliary variables into an FIML analysis. It has remained a fruitful starting point for several recent advances in assessing model fit with missing data. For example, Cai and Lee (2009) adapt the two-stage estimation approach to produce a correctly distributed χ^2 statistic by incorporating the Meng and Rubin (1991) supplemental EM algorithm, and Savalei and Bentler (2009) derived their own correction to produce accurate standard errors and χ^2 statistics, which they show to perform comparably to FIML estimates. Savalei and Falk (2014) showed that this method can perform better than FIML when data are nonnormally distributed.

One downside of approaches like Meng and Rubin's (1992) technique and the corrected versions of the Yuan and Bentler (2000) two-stage estimator is that they tend to be difficult to implement without good software integration. To mitigate this difficulty certain authors have explored the possibility of pooling the imputed data structures prior to the analysis phase of MI. Yet, all such studies have found that the χ^2 statistics derived from these pooled data structures required a *post hoc* correction before they followed a χ^2 distribution (Lang, 2013; Lee & Cai, 2012). Lee and Cai (2012) derived such a correction by adapting Proposition 4 from Browne (1984) to produce a test statistic T_{MB} that asymptotically follows a central χ^2 distribution. They also provided a SAS Macro that can be used to implement their approach software.

Lang and Little (2014) proposed an approach that was designed to be easily implemented with any statistical analysis software without relying on specialized code. Their approach (which we dub the supermatrix technique) is essentially an expansion of the initial aggregation phase of the Lee and Cai (2012) two-stage approach. Using a Monte Carlo simulation study they provided empirical evidence to suggest that accurate hypothesis tests can be accomplished by appealing to nested-model χ^2 difference tests without the need for post hoc correction to the component χ^2 statistics (even though the component χ^2 statistics are biased measures of model fit when considered individually).

To illustrate how the supermatrix technique is implemented, consider a hypothetical missing data analysis in which you begin with an incomplete $N \times p$ data set. Application of the supermatrix technique can then be broken into four basic steps:

1. Create a set of M multiple imputations using any MI algorithm.
2. Stack the M imputed data sets into an aggregate $MN \times p$ data frame.
3. Calculate a single $p \times p$ covariance matrix from the stacked data from step 2.
4. Use the covariance matrix from step 3 and the original sample size N as the sufficient statistics and sample size, respectively, for subsequent model fitting.

Figure 17.2 gives a graphical summary of the supermatrix approach for a hypothetical example in which the original incomplete data consists of four observations of three variables. The results of a Monte Carlo simulation study conducted by Lang (2013) suggested that fit indices derived from structural equation models fit using the supermatrix as sufficient statistics will have inflated type I Error rates (i.e., they will overreject well-fitting models) and should not be used to assess overall model fit. However, simulation results from Lang and Little (2014) showed very good performance for nested-model $\Delta\chi^2$ statistics computed from parent and child models that are both fit to a supermatrix. When $\Delta\chi^2$ statistics were calculated from parent and child models fit to supermatrices, Lang and Little (2014) found that these $\Delta\chi^2$ statistics were nearly identical to analogous $\Delta\chi^2$ statistics calculated from parent and child models fit to fully observed data.

Mediation Analysis

Mediation is often of central interest to researchers in social and behavioral science research. Mediation is said to occur when the effect of an independent variable (X) on a dependent variable (Y) is transmitted by a third variable (M) (Baron & Kenny, 1986; Sobel, 1982). M is termed a mediator as it mediates the effect of X on Y. A mediating effect (also termed an indirect effect) can be quantified by and tested through the product of the two

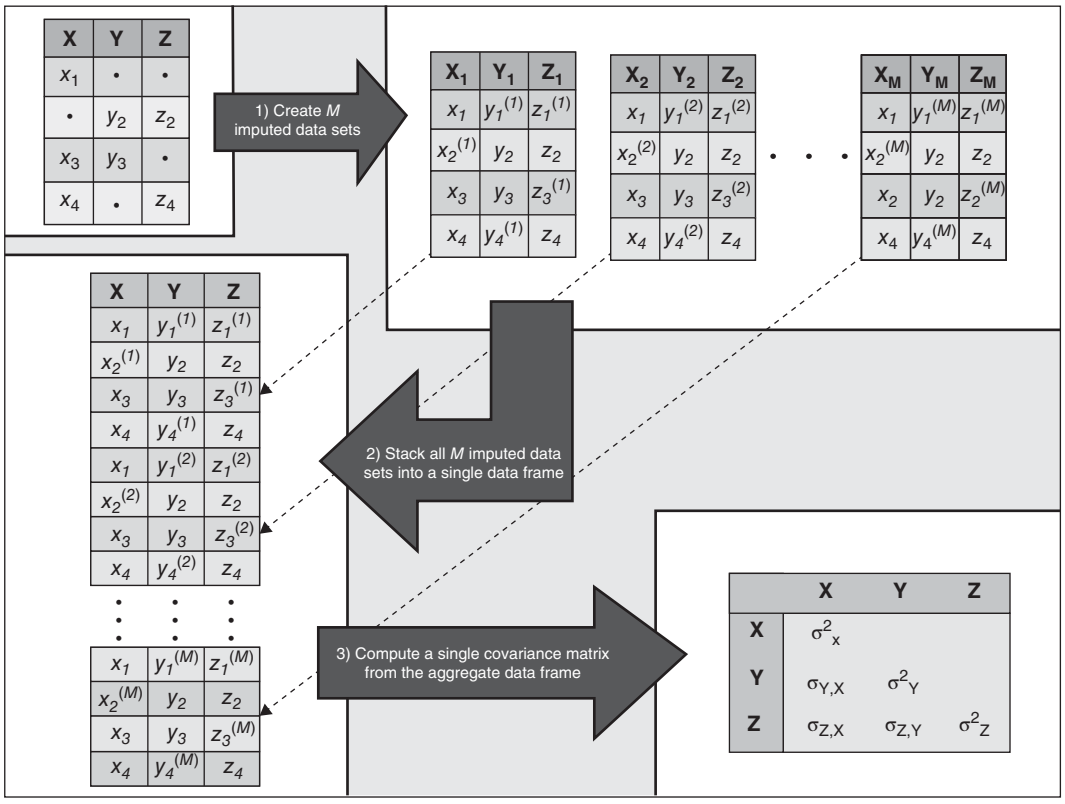


Figure 17.2 A graphical representation of the supermatrix technique as applied to a data set with four observations and three variables. *Source:* From Lang and Little (2013). Used with permission.

direct effects (denoted by *ab*): (1) the effect of *X* on *M* (*a* effect); and (2) the effect of M on Y controlling for *X* (*b* effect). A well-known strategy to test *ab* is to empirically establish the sampling distribution of *ab* through nonparametric bootstrapping and then use the confidence interval computed from the sampling distribution to test the null hypothesis that $ab = 0$ (MacKinnon, Lockwood, & Williams, 2004). A nonparametric bootstrapping procedure starts with drawing a large number of samples with replacement (e.g., 1,000) with the same sample size of the original data, assuming that the original sample is the population (Efron, 1979; Efron & Tibshirani, 1994). A mediation model is then fit to each of the bootstrap samples, resulting in a large number of point estimates of *ab,* which form the empirical sampling distribution of *ab*.

When there are missing data, nonparametric bootstrapping can be combined with FIML or MI to test mediation effect (Wu & Jia, 2013; Zhang & Wang, 2013). To combine nonparametric bootstrapping with FIML, one needs to bootstrap first and then run the target analysis using FIML for each bootstrapped sample (denoted by BOOT[FIML]). Many software packages such as Mplus and EQS have automated this procedure. To combine bootstrapping with MI, two procedures have been proposed. One is to bootstrap first and then run MI for each bootstrap sample. This procedure is computationally intensive because it requires running MI for each bootstrap sample. In other words, if there are 1,000 bootstrapped samples, the MI needs to be run 1,000 times. This large number of runs would impose a tremendous amount of computational burden. To mitigate this problem, Wu and Jia (2013) suggested running MI first to obtain a number of imputed data sets and then running the bootstrap procedure for each imputed data set (denoted by MI[BOOT]). Because MI(BOOT) requires only 1 run of MI, it is more computationally efficient than BOOT(MI). Wu and Jia (2013) showed that MI(BOOT) performed comparably to BOOT(FIML) when data are normally distributed and missing data are ignorable. However, one should be cautious about using either procedure to test mediation as MNAR, nonnormality, or both can distort the point and interval estimates of a nonzero *ab* for both procedures.

In addition to nonparametric bootstrapping, Bayesian estimation methods are well suited to mediation analysis and can be readily extended to treat missing data (Biesanz, Falk, & Savalei, 2010; Enders, Fairchild, & MacKinnon,

2013; Yuan & MacKinnon, 2009). The Bayesian method generates a posterior distribution of *ab* (i.e., a distribution of *ab* conditional on the observed data) based on which the statistical inference of *ab* can be made. However, past research on Bayesian approach is limited to normal data. More studies need to be conducted to examine the performance of the Bayesian approach when the normality assumption is violated and to compare it with the other strategies.

When to Use FIML Versus MI

FIML and MI provide comparable point and standard error estimates given that (1) the multivariate normality assumption is satisfied, (2) the missing data are ignorable (i.e., MCAR and MAR), (3) missing data model is the same, and (4) the number of imputations in MI approaches infinity (Collins, Schafer, & Kam, 2001; Graham et al., 2007; Little & Rubin, 2002; Schafer, 1997; Schafer & Graham, 2002). However, they have their own pros and cons in practice. Some of these have been mentioned in the previous sections. Here we provide a more detailed comparison of the two techniques.

There are many reasons researchers might prefer FIML. First, FIML is more efficient than MI. The standard error estimates from MI will generally be larger than those from FIML although the difference is trivial when the number of imputations is large. Second, FIML is also easier to implement because it is a one-step approach and available in most of the commercial software packages (e.g., Mplus, LISREL, EQS, AMOS, SAS PROC CALIS, and lavaan in R). In contrast, MI involves multiple steps that have not been fully automated. In addition, implementation of MI requires many decisions. As described before, researchers might need to decide on an appropriate number of imputations, the correct imputation model (with the relationships among the variables correctly specified), and an appropriate number of burn-in and between-imputation iterations. Finally, it is also easier to obtain model fit indices with FIML as described previously.

However, there are benefits of using MI. Certain imputation algorithms and imputation models might provide a better treatment of categorical variables and nonparametric relationships among the variables (Asparouhov & Muthén, 2010a; White, Royston, & Wood, 2011). In addition, MI creates complete data sets. As a result, statistical methods that are only implemented for use with complete data can be applied. Under certain circumstances, MI has to be adopted (Enders, 2010; Gottschall, West, & Enders, 2012). One circumstance occurs when there are item-level missing data. To illustrate, suppose *X, Y*, and *M* in a mediation model are scale scores that are computed by averaging multiple item scores. If some of the items have missing data, the best way to handle the missing data would be imputing them at the item level and then calculating the scale scores from the imputed item scores (Gottschall, West, & Enders, 2012). By doing this item-level imputation, the observed items' scores are used to recover the missing information. FIML is inferior in this case as it relies on the analysis model in which the item scores are not directly included, resulting in underuse of the available information. Another circumstance in which MI may work better is when the model contains categorical dependent variables. Though FIML can be used with categorical data, when there are more than two categories or the model is complicated, this becomes extremely computationally intensive. Instead, the most common estimation technique is to use a diagonally weighted least squares estimator on the matrix of polychoric correlations (this is called "WLSMV" in Mplus, where it is the default estimator for categorical variables). Because least squares estimators cannot handle missing data efficiently, MI allows the researcher to deal with the missingness first (e.g., by using MICE to impute categorical variables) and use the least-squares estimator to analyze the model on the imputed data sets (Asparouhov & Muthén, 2010a, 2010b).

REVIEW OF MISSING DATA PRACTICES IN PSYCHOLOGICAL RESEARCH

In his introduction to the special section of *Psychological Methods* on missing data analysis, West (2001) noted that a great many methodological advances had been made in the treatment of missing data analysis in the preceding 25 years. In an interview shortly thereafter, West predicts that increasing implementation of principled missing data tools will be hallmark of psychological research in the early twenty-first century (Azar, 2002). Many of the advances that West (2001) was referencing had, by the turn of the twenty-first century, yet to make their way into practice in the applied social science literature.

The *American Psychological Association* was clearly cognizant of the need for improved treatment of missing data. This concern is evident when reviewing the recommendations of the Task Force on Statistical Inference published in 1999. One of the key recommendations of this task force was that authors publishing in psychology journals should accurately report on the missingness in their studies so that its impact on the conclusions can be judged transparently (Wilkinson & Task Force on Statistical Inference, 1999). The committee also went on to advise

that researchers adopt optimal missing data treatments and eschew antiquated ad hoc approaches. They condemned the use of deletion methods, in particular, by stating, "The two popular methods for dealing with missing data that are found in basic statistics packages—listwise and pairwise deletion of missing values—are among the worst methods available for practical applications" (p. 598).

So how prophetic was West's claim? Have there been considerable improvements in missing data practices since 2000? Unfortunately, even in the shadow of an ever growing monolith of evidence that ad hoc missing data tools are universally inferior to modern principled techniques, psychological researchers have been relatively slow to adopt more sophisticated missing data tools. Reviews of the missing data practices employed in psychological studies published in 1999, 2003, and 2012 have all found that ad hoc techniques, and deletion based techniques in particular, remain the most prevalent treatment for missing data (Bodner, 2006; Little et al., 2014; Peugh & Enders, 2004). However, all hope is not lost, because there are signs that the field is on a trajectory of improvement.

Recognizing that the aforementioned recommendations of the Task Force on Statistical Inference represented a turning point for missing data analysis in the social sciences, Bodner (2006) and Peugh and Enders (2004) reviewed the missing data practices reported in samples of journal articles published in 1999. These studies' findings were convergent and bleak. Perhaps most troubling was the very high frequency with which missing data were not reported at all (i.e., the presence of missingness often had to be inferred from discrepancies in the degrees of freedom reported for test statistics). Additionally, for those cases in which the missing data treatment could be elucidated, a principled technique was never used, and deletion-based approaches were, by far, the most frequently applied treatments.

To judge the improvement in missing data analysis in the years following the recommendations of the Task Force on Statistical Inference, Peugh & Enders (2004) also reviewed the missing data practices reported in a sample of journal articles published in 2003. They found a discernible improvement in missing data practices, especially in terms of reporting practices. This second review found much more accurate reporting of missing data (i.e., a higher proportion of authors explicitly acknowledged the presence of missingness in their studies). They also note a higher frequency of authors explicitly discussing the assumptions underlying the missing data analysis (e.g., actively testing the MCAR assumption). Unfortunately, ad hoc approaches were still the norm, and deletion techniques remained the most common missing data treatment. However, they did record several instances of principled techniques being employed, whereas there had not been any use of principled approaches in their review of journal articles from 1999.[3]

Finally, Little et al. (2014) conducted a comprehensive review of the missing data practices reported in empirical studies from the 2012 volume of the *Journal of Pediatric Psychology* and found further evidence for a trajectory of improvement in missing data practices. The improvement in reporting practices vis-à-vis those found by Bodner (2006) and Peugh and Enders (2004) was considerable. The majority of the articles examined transparently acknowledged the presence of missingness in their studies and explicitly described their treatment thereof. There was also a considerable increase in the usage of principled missing data tools (especially FIML) relative to the rates reported by Bodner (2006) and Peugh and Enders (2004). A sizable minority of the articles examined employed FIML (or some variant of FIML) to treat the missingness in their studies. Unfortunately, ad hoc approaches were still the most frequently employed techniques, and several papers mixed ad hoc approaches with principled techniques (e.g., listwise deletion of attrition and imputation of the remaining arbitrary missingness).

An interesting finding of the Little et al. (2014) review was that every study that employed FIML either fit a structural equation model using Mplus (Muthén & Muthén, 2012) or it fit a multilevel model. Because FIML estimation is the default option when analyzing incomplete data in Mplus, and multilevel modeling relies on FIML (or some specialized variant of FIML) to accommodate the inherently unbalanced data for which it is designed, the

[3]Principled techniques are defined as any missing data technique that treats the nonresponse by considering the underlying distribution of the missing data and either fills in these missing values via predictive models constructed to produce predicted values that conform to that underlying distribution (in the case of MI) or partition the missingness out of the likelihood function by appealing to the Rubin (1976) partitioning of the complete data distribution, which is only possible when explicitly considering the distribution of the missing values (as in EM and FIML). These principled techniques are usually considered in contrast to ad hoc approaches that are so called because they are simple one-off convenience solutions to the missing data that basically ignore any missing data that ever existed. In the strictest sense principled and ad hoc are not mutually exclusive. You can have a principled ad hoc approach if it is built up from initial consideration of the distribution of the missing values, but it is specialized to a specific problem and not generalizable to any other analysis.

greater usage of principled techniques observed by Little et al. (2014) may not represent a meaningful improvement in practice. Although, this is a pessimistic outlook, the fact that all of the reported FIML analyses were conducted by naively employing a restrictive strategy (i.e., they did not include auxiliary variables in the FIML model) lends credibility to the suggestion.

Judging by the patterns of missing data practices observed in the reviews presented by Bodner (2006), Little, et al. (2014), and Peugh and Enders (2004) it is apparent that missing data are becoming a more active consideration for researchers as time goes on. Similar findings are reported by Wood, White, and Thompson (2004), who employed similar methodology to analyzed the missing data practices reported in a sample of studies published in the 2001 volume of several top-tier medical journals. However, the findings of Little et al. (2014) suggest that many applied researchers in psychology are still inadequately informed when it comes to optimal missing data treatments, and many decisions in a missing data analysis are guided by software defaults. While optimal missing data tools are becoming better integrated into popular software every year (Enders, 2010), the findings discussed above suggest that there is still a need for education on how to best implement these tools as they become available.

WHY CHANGE?

Too often, entreaties to employ modern approaches to address missing data or to incorporate planned missingness as a design feature are met with an unfounded fear or unwarranted hubris. As detailed in Table 17.1, the traditional methods of handling missing data are rife with error and bias. Maintaining a staunch commitment to the practices of old is an untenable position to take (Wilkinson & Task Force on Statistical Inference, 1999). The only time that *some* of these practices are warranted is when the missing data are MCAR and power is not an issue (Enders, 2010; Little & Rubin, 2002), or when the amount of missing data is very small (e.g., less than 5%; Graham, 2009). Otherwise, invalid conclusions will result. Fundamentally, failing to address the bias created by missing data represents a miscarriage of social justice. Because results are biased when traditional approaches are employed, policy recommendations based on the analyses of faulty data lead to failures in providing accurate guidance for stakeholders and decision makers. Efforts to simplify analyses by shortcutting the appropriate ways of addressing missing data are unconscionable. Baseless fears or unjustified hubris are not legitimate excuses to avoid appropriately treating the missing data.

Rather than ignoring the issue in the abject desire that the bad consequences of missing data won't be too bad, we should aggressively address the mechanisms of missing data to minimize their undermining influences (Wilkinson & Task Force on Statistical Inference, 1999). In this vein, thoroughly examining and reporting on potential predictors of missingness would benefit future research because investigators could identify potential predictors of missingness to be included into a data-collection protocol. Such information could be added to the supplemental materials for a published paper and posted on a reliable web page. Such virtual repositories would provide a wealth of information for future study planning.

PLANNED MISSING DATA DESIGNS

The preceding discussion of missing data has centered on the unplanned missingness that inevitably occurs in human science research. We now turn our attention to planned missing data designs and the recent research that is still emerging in this area (e.g., Graham, Hofer, & MacKinnon, 1996; Graham, Taylor, & Cumsille, 2001; Graham, Taylor, Olchowski, Cumsille, 2006; Jia et al., 2014; Jorgensen et al., 2014; Rhemtulla, Jia, Wu, & Little, 2014, Rhemtulla, Savalei, et al., 2014). Planned missing data designs have been around for some time but maybe not obviously recognized as such. The Solomon four-square design, for example, is a planned missing data design (see Table 17.4, Groups 1–4; see also Iacobucci, 1995). This design is often used with classical missing data handling procedures. As such, researchers have not taken full advantage of it. If classical analyses are used, it is a very underpowered design. If modern missing data handling procedures are used, on the other hand, it becomes a very powerful pre–post

TABLE 17.4 The Solomon Four-Group Design as a Planned Missing Design with the Addition of Two Additional Groups for a Complete Planned Missing Design

Group	Pretest[1]	Treatment/intervention	Posttest[1]
1	O_1	X	O_2
2	O_3		O_4
3	Planned missing	X	O_5
4	Planned missing		O_6
5	O_7	X	Planned missing
6	O_8		Planned missing

[1]O reflects the observed variables, including all demographics and outcome scores and X represents a treatment or intervention that has been implemented. The subscripts differentiate the different groups at the different testing occasions.

experimental design. The participants who do not receive the pretest scores are randomly assigned to be in Group 3 and Group 4 (Table 17.4). These missing pretest scores are imputed if a modern missing data approach is used, thereby increasing the power of the overall design. A fully planned missing data design might also add two additional groups (Groups 5 & 6 in Table 17.4) to allow for more power at less cost.

Planned missing data designs leverage the capabilities of the modern missing data handling procedures to minimize the loss in power that traditional approaches suffer. Planned missing data designs also are used because they can substantially reduce costs and, in many cases, increase validity by reducing participant burden/fatigue and minimize other threats to validity, such as test reactivity and unplanned missing data (Harel, Stratton, & Aseltine, 2011; Popham, 1993). Planned missing data designs are most useful in the context of large sample research involving latent variable SEM techniques that extend easily to longitudinal designs (Little, 2013). In the following, therefore, we will assume that multiple indicators of constructs are generally used, reasonably large sample sizes are available (e.g., > 140), and that latent variable SEM or one of its variants will be used (see Little, 2013, for a discussion of the various options). This latter assumption is not essential for using all of the planned missing designs we will discuss but it is for some of them.

Harel et al. (2011) investigated whether planned missingness can be used to mitigate test reactivity in a pre-post control group experimental design. In a suicide prevention assessment trial, students received either all or a subset of items from the Signs of Suicide 17-item questionnaire at pretest and the complete questionnaire 3 months postintervention. Three notable findings arose: first, students who were given more items to complete were less likely to complete all of them: 27% of those who were given all 17 items failed to answer all of them, compared to 14% who were given just 6 items and 18% who were given 8 items. Second, students who were given the full questionnaire at baseline were three times more likely to skip the posttest assessment entirely (21% compared with 7% for those who received a truncated questionnaire at baseline). Third, of those students who attempted the posttest questionnaire, those who received the full questionnaire at baseline were again less likely to complete all items at posttest (25% of these students skipped items compared with 15% of students who received a truncated questionnaire at baseline), despite the fact that all students received the full 17-item questionnaire at posttest. These results suggest that planned missing designs can significantly reduce rates of unplanned missing data—both item nonresponse and attrition—by substantially reducing participant burden.

In addition to reducing the rates of unplanned missingness, Harel et al. (2011) showed that the planned missing strategy alleviated the effects of test reactivity. Test reactivity occurs when persons assigned to the control group get better simply by virtue of being tested with protocols that clearly indicate the purpose of a study. With the planned missing protocol used by Harel et al., those participants who received only some of the signs of suicide items did not react to the protocol, whereas those who received the whole questionnaire showed changes at post-test that obscured the effect of the intervention.

The Harel et al. (2011) study is a unique application of a planned missing data design that highlights the utility of such designs. In the following, we will discuss more standard applications of planned missing data designs and extend them to the case of longitudinal data collections: (1) multiform designs, which focus on shortening the questionnaire protocol; (2) the two-method design, which focuses on leveraging high-quality measures to increase power; and (3) wave-missing designs, which focus on reducing the costs of longitudinal data collection.

Multiform Planned Missing Protocols

The simplest multiform example is the three-form planned missing data design, an instance of which is presented in Table 17.5. Multiform designs have been described in various literatures under various names and with various analysis models (e.g., Bunting & Adamson, 2000; Bunting, Adamson, & Mulhall, 2002; Dings, Childs, & Kingston, 2002; Gonzalez & Eltinge, 2008; Mislevy, Beaton, Kaplan, & Sheehan, 1992; Raghunathan & Grizzle, 1995; Shoemaker, 1973; Wacholder, Carroll, Pee, & Gail, 1994). As seen in the example of a three-forms design in Table 17.5, items are divided into four different item sets and then assembled to create three questionnaire protocol forms. Participants are randomly assigned to fill out one of the three forms. The items that are not included in a form are thereby missing completely at random (i.e., MCAR) because the investigator controls, through random assignment, which variables are missing for each person.

TABLE 17.5 Example of a Three-Form Planned Missing Design

Form	Common Set X	Variable Set A	Variable Set B	Variable Set C
1	Complete	Complete	Complete	Missing
2	Complete	Complete	Missing	Complete
3	Complete	Missing	Complete	Complete

Multiform questionnaire protocols are a way to create short forms that fit within a specified time frame, to reduce overall costs, and to minimize respondent burden and fatigue. Each participant receives a questionnaire protocol that can be substantially shorter than the full set of protocol questions. The benefit of the multiform design is that it leverages the capabilities of the modern missing data treatments to maintain the information (both reliability and validity) that is inherent in the full set of questions. Moreover, the planned missing protocol has the potential to increase validity by reducing burden, fatigue, unplanned missing, and test reactivity (e.g., Harel et al., 2011) as well as practice and retest effects with longitudinal data (Beglinger et al., 2005; Jorgensen et al., 2013; Salthouse & Tucker-Drob, 2008).

These many benefits notwithstanding, planned missing protocols can be less efficient than complete case protocols. Recall that efficiency, which is related to power, reflects the size of the standard error for a given parameter estimate. The power loss associated with the planned missing protocol can be offset by an increasing the sample size to produce the same efficiency as the design would have had with complete data. The extent to which parameter efficiency suffers depends on the pattern of missing data, the parameter type, and the strength of correlations between variables (Garnier-Villaréal et al., in press; Rhemtulla, Jia, et al., 2014; Rhemtulla, Savalei, et al., 2014).

One salient recommendation that has come out of our recent research on optimal planned missing data designs is that efficiency is maximized when groups of highly correlated items (e.g., items from the same scale) are split across item sets, such that every participant answers a proportion of the items on every scale. Intuitively, this makes sense—once a participant has responded to four items on a six-item conscientiousness scale, four items on a six-item neuroticism scale, and four items on a six-item extraversion scale, the incremental benefit of responding to another two items on each of those scales is small. In contrast, if a participant responds to all six items on the conscientiousness scale and the neuroticism scale, and zero items on the extraversion scale, even though in both cases the participant has answered 12/18 questions, more information is missing when a whole scale is absent. In the first case, we have a pretty good estimate of that participant's personality along all three dimensions; in the second case, we have a slightly better estimate of personality along two dimensions but no estimate at all on the third. As such, planned missing designs where scales are split across sets result in much more powerful analyses.

Similarly, having indicators of constructs in the X block minimizes the reduction in efficiency for many parameters. As with most complex modeling endeavors, we recommend careful planning and preliminary simulation runs to identify any potential weakness of a planned missing data design. Even if poorly constructed, however, a planned missing data design brings more benefits than costs when compared with the business as usual approach that permeates current literature.

As with many statistical techniques, the details of how to create a multiform design can be more cumbersome than a first glance may suggest. A multiform design is no different. A number of important issues need to be considered when using a multiform design in general. We will discuss these issues in the context of the three-form design. By way of example, we will walk through the creation of three forms of the Inventory of the Forms and Functions of Aggression (iFFA) survey (Little, Jones, Henrich, & Hawley, 2003). The iFFA measures six constructs of aggression each with six items: Pure Overt, Instrumental-Overt, Reactive-Overt, Pure Relational, Instrumental-Relational, and Reactive-Relational. The iFFA is a self-report battery that is age appropriate from elementary age to late adolescence. For this hypothetical instrument, we will also collect demographic information and an important screening item for suicide ideation—"During the past 3 months, did you ever seriously consider attempting suicide (yes or no)?"—as well as self-reports reports on truancy, detentions, and visits to the principal's office.

As shown in Table 17.5, the three-form design consists of an X block of items, which is administered to all participants. In creating a three-form protocol from these variables, all singly indicated constructs (i.e., constructs that are measured by just one item, including truancy frequency, detention frequency, and number of principal visits), demographic variables (age, gender, race/ethnicity), and any critical screening variables (suicide ideation) are assigned to the X block. For the latent constructs with multiple indicators, the items for each construct would be assigned based on the item characteristics. Indicators of constructs can be classified as (1) parallel, (2) tau-equivalent, or (3) congeneric. Parallel items are psychometrically identical in their measurement of a construct (as can be found in abilities testing, for example). In the behavioral and social sciences, constructs are more likely to be either tau-equivalent or congeneric. With tau-equivalent items, each items is about equally good at representing the construct (i.e., each item loads on the construct to about the same degree). With congeneric items, some items are stronger indicators of the construct (i.e., have high

loadings) then other items of the construct (i.e., items with lower loadings). The iFFA is a battery with tau-equivalent indicators. A loneliness scale with items such as "I feel lonely" and "I feel like no one likes me" would likely have congeneric qualities with the "I feel lonely" item being a marker variable of the construct loneliness and the "I feel like no one like me" would be a lower loading (yet important) indicator of the construct.

With this slightly tangential discussion in mind, items representing the same construct would be spread among the X, A, B, and C blocks of times. If the items are tau-equivalent the process can be essentially random. With six items for a construct, two of the four blocks of items would have two items assigned to them. The key here would be to balance the number of items in the A, B, and C blocks to ensure about equal numbers so that each of the three forms is of equal length. If one of the forms is longer than the other two, unplanned missing data might emerge at a greater frequency for the longer form. A set of dummy codes representing the assigned form could be used in the imputation model to account for this feature if it were to occur. If items are congeneric and one item can be identified as the strongest representative of the construct, it would be assigned to the X block. The remaining items would be distributed as evenly as possible among the A, B, and C blocks.

As mentioned previously, when building a multiform protocol, the efficiency of the parameters estimated from the resulting data set is increased if the between block correlations are high. Spreading items of the same construct across the item blocks helps ensure that the between block correlations are high. Putting all the items of a given construct into the same item block will increase the within block correlations but will have a tendency to reduce the between block correlations, which can lower efficiency. Sometimes, questions are blocked or created as a testlet. Here, the set of items in a block or testlet cannot be spread among the different item sets. In this case, the block of items or testlet must be assigned to one of the items sets. If only one block or testlet is involved it would be best placed in the X set. If two or more of them exist, then they could be assigned to different blocks of items.

Extending the three-form design longitudinally is also a matter for consideration. As mentioned, the three-form planned missing data design allows researchers to measure more items from more participants on more occasions using the same budget as a complete data design. It also reduces burden, fatigue, and response reactivity. After randomly assigning participants to complete one of three forms on the first occasion, for subsequent measurements, one might assign participants to complete the same form, a different form that they haven't seen yet, or use random assignment again. Retest effects are expected when making repeated measurements, and assigning different forms over time would decrease the number of variables that participants see on consecutive occasions, thus decreasing the degree to which retest effects manifest in variables not included in the X block. Using a Monte Carlo simulation approach, Jorgensen et al. (2014) compared the different assignment strategies in the context of autoregressive latent-variable panel models, a commonly used type of longitudinal model. Data were simulated based on a fixed, moderately large sample size using a range of population parameter values, and certain patterns (corresponding to each of the three methods of assigning forms) of complete data were replaced with missingness, replacing either 11% or 22% of complete data with planned missingness. The results indicated negligible differences between assignment methods only in the absence of retest effects, which is not a likely scenario in practice. When they introduced practice effects in the form of small (Cohen's $d = 0.1$) mean increases for items seen on consecutive occasions, strong invariance models (with intercepts constrained to equality across occasions) showed an increase in the latent mean, although the population mean was zero. This bias manifested less when forms were randomly assigned on each occasion, but not nearly so much as when different forms were assigned systematically, such that no one would see the same form twice until seeing all other forms first. Reduction of bias due to retest effects makes it preferable to systematically assign different forms over time.

Two-Method Planned Missing Design

In many studies, researchers are put in the difficult situation of choosing between two methods for measuring a critical construct. Often, one method, such as blood cortisol levels for measuring stress, is a highly valid but expensive tool whereas a second method, such as a self-report battery of perceived stress, is a less valid but easily (inexpensively) administered tool. In situations like this, the expensive measure is considered the gold standard for assessing a construct, but the costs of using it are prohibitive. If the expensive measure is chosen, the costs are often reduced by reducing the sample size, which typically results in an underpowered or highly simplified study. In contrast, if the inexpensive measure is chosen, the increases in sample size and design complexity are offset by the fact that the inexpensive measure contains systematic bias (e.g., due to the self-report nature of the measure).

This scenario does not have to be an either–or choice. Instead, the two-method planning missing data design provides an ingenious way to leverage the capabilities of both modern missing data treatments and latent variable structural equation modeling. When two methods for measuring the same underlying construct exist (or can be developed), the two-method design allows for larger sample sizes while maintaining the high validity of using only an expensive gold standard tool. Table 17.6 provides a list of candidate constructs for which two methods exist. From physiological indices, to classroom observations, to clinical diagnoses, many constructs have expensive gold standard ways of measuring them. Many of these constructs also have easily administered alternatives. In any of these situations, the two-method design can be implemented.

One key to the two-method planned missing data design is the use of latent variable structural equation modeling. Figure 17.3 shows a couple of examples of two-method construct representations. As shown in Figure 17.3, the two-method planned missing data design relies on the bifactor model and the common factor model to represent the construct of interest as well as a bias factor. It also relies on the ability to represent both the gold standard measure and the biased measure with multiple indicators. Using multiple indicators is essential to disentangle the reliable from the unreliable variance and then to further disentangle the biased information from the unbiased information about the focal construct of interest.

A second key to the two-method planned missing data design is that all participants are assigned to receive the biased measure. For the unbiased, gold standard measure, only a subset of participants are randomly assigned to be assessed. The ratio of expensive to cheap observations depends on the degree of correlation between the two methods of measuring the construct and on the degree of reliability of the two measures. Graham et al. (2006) provided a straightforward way to calculate the ratio that hits the sweet spot in terms of power and cost. Generally speaking, the amount of planned missing data for this design can be quite high if the biased measure is not too biased and both measures are measured reasonably reliably.

TABLE 17.6 Possible Candidate Constructs for Use with the Two-Method Planned Missing Data Design

Construct	Expensive/Gold Standard	Inexpensive/Biased Measure
Stress	Cortisol assays	Self-report perceived stress
Intelligence	Wechsler WAIS assessments	Multiple-choice test of ability
Smoking cessation	Blood draws	Self-report
Student/teacher behavior	Classroom observations	Self-, Peer-, or Teacher-report
Nutrition	Face-to-face-interview	Self-report survey
Adiposity	Densitometry, hydrostatic weighing, or dual energy x-ray absorptiometry	Body mass index

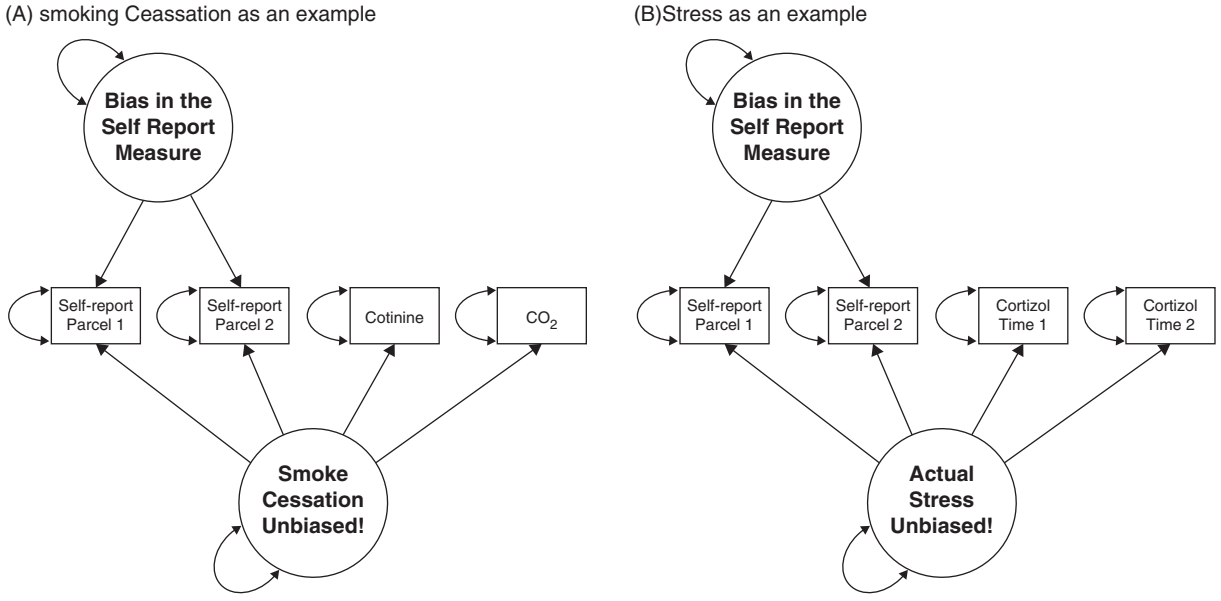


Figure 17.3 Two variations of a two-method planned missing model. (*Note*: Only a subset of individuals are randomly selected to receive the expensive measure.)

Obtaining an optimal number of indicators for each construct is not difficult if one considers the use of parceling techniques to provide the correct level of aggregation needed to create multiple indicators for each construct. Although the use of parcels is somewhat a matter of debate (and the nature of the debate is beyond the scope of this chapter), the ways parcels can be used for purposes of the two-method planned missing data design are straightforward (see, e.g., Little, Cunningham, Shahar, & Widaman, 2002; Little, Rhemtulla, Gibson, & Schoemann, 2013).

To extend the two-method planned missing data design to longitudinal data collection procedures, we need to discuss some nuances for how it should be implemented. In a simulation study of the power and efficiency of the two-method planned missing data design, Garnier-Villarréal et al. (2013) and Rhemtulla and Little (2013) provide a number of useful recommendations for extending this model to the longitudinal case.

Wave-Missing Designs

A number of wave-missing designs are available to developmental researchers. These designs and their variants have different nuances and considerations that must be considered to implement them well. Some of the wave-missing designs involve random assignment and thus make the missing information MCAR. Recall that MCAR missing (missing completely at random) is the hallmark of planned missing data designs because there is no introduction of bias with MCAR missingness, only a decrease in power. As such, these designs provide tremendous potential. Some wave-missing designs, however, are not based on random assignment to missing data pattern. One such design is the accelerated longitudinal design (Table 17.7). In this design, the missing information is not randomly assigned but, instead, is a feature of the cohort from which participants are selected. Such missing data, however, are due to a predictable MAR process and can therefore be recovered to provide unbiased parameter estimates. In the following, we discuss three wave missing designs: the developmental lag missing design introduced by McArdle and Woodcock (1997); the accelerated longitudinal design originally introduced by Bell (1953); and the growth curve wave missing design introduced by Graham and colleagues (2001).

McArdle and Woodcock (1997), for example, introduced a planned missing data design that allowed them to examine memory loss and retest effects over 8 days. Their design involved only two measurement occasions for each participant but the interval of time between the two measurements ranged across participants. Putting the responses into bins of baseline and six successive waves, they used growth curve modeling procedures to represent a slope construct of change over time and a separate retest construct. In one model, the retest effect was estimated to be a constant value regardless of the length of time between measurement occasions. In a second model, the retest effect was specified to be an exponentially declined effect. This exponentially declining retest construct fit the data better than the constant effect model, suggesting that retest effects are more pronounced with shorter intervals of measurement and less pronounced as the time span between occasions gets longer.

Bell (1953) introduced the accelerated longitudinal design as a way to estimate age trends from cross-sequential data. Figure 17.4 shows the three sequential data designs

TABLE 17.7 Converting a Cohort-Sequential Data Set into an Accelerated Longitudinal Design

	Age in years; months for each cohort at each assessment																			
Age/cohort	0 Months			4 Months			8 Months			12 Months			16 Months			20 Months				
Age 11 years	11;0			11;4			11;8			12;0			12;4			12;8				
Age 12 years	12;0			12;4			12;8			13;0			13;4			13;8				
Age 13 years	13;0			13;4			13;8			14;0			14;4			14;8				
Age 14 years	14;0			14;4			14;8			15;0			15;4			15;8				
Age 15 years	15;0			15;4			15;8			16;0			16;4			16;8				
Age 16 years	16;0			16;4			16;8			17;0			17;4			17;8				
	Full span of the ages covered																			
	11;0	11;4	11;8	12;0	12;4	12;8	13;0	13;4	13;8	14;0	14;4	14;8	15;0	15;4	15;8	16;0	16;4	16;8	17;0	17;4	17;8
Age 11 years	W1	W2	W3	W4	W5	W6															
Age 12 years				W1	W2	W3	W4	W5	W6												
Age 13 years							W1	W2	W3	W4	W5	W6									
Age 14 years										W1	W2	W3	W4	W5	W6						
Age 15 years													W1	W2	W3	W4	W5	W6			
Age 16 years																W1	W2	W3	W4	W5	W6

Source: Little (2013). Used with permission.

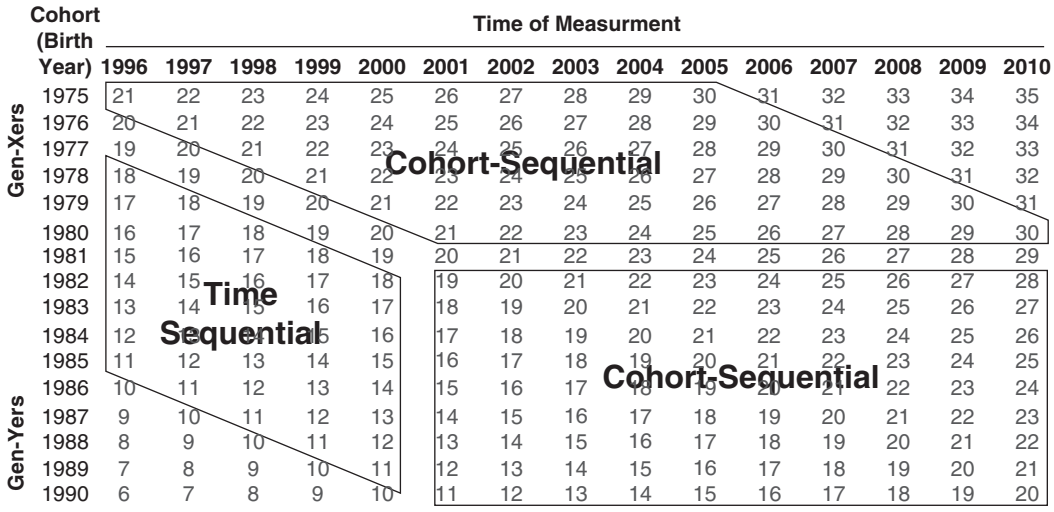


Figure 17.4 The three traditional sequential designs used to disentangle age, cohort, and time of measurement effects. *Source:* Little (2013). Used with permission.

that are used to disentangle two of the three confounded effects in longitudinal data. Specifically, age, cohort, and time of measurement cannot be separated in a single cohort longitudinal study. By broadening the measurement frame, however, two of the three effects can be adequately disentangled (see Little, 2013; Wohwill, 1973). With the cross-sequential design (which is often mislabeled the cohort-sequential design), it is possible to realign the data set to represent the ages of the participants regardless of the cohort or wave at which the information was assessed. Figure 17.4 shows how the cross-sequential data are transformed into an accelerated longitudinal design.

The final design in this section is the growth curve planned missing design. Rhemtulla, Jia, et al. (2014) examined the performance of three types of planned missing designs in multivariate latent growth models, including a three-form design (where participants are assigned to be missing a subset of items at each occasion), a wave-missing design (where participants are assigned to be missing a subset of measurement occasions), and a design that combined both types of missingness. They found that the three-form design resulted in high convergence rates, little bias, and high efficiency relative to complete data for almost all parameter types. In contrast, the wave-missing and combined designs resulted in biased parameter estimates at sample sizes smaller than 500, and dramatically lower power for many of the structural parameters, including the variance and covariances among latent slopes. Based on these results, they recommended that wave missing designs are only well suited to latent growth curve models when the sample is very large (e.g., $N > 1000$) or effect sizes are large. In contrast, three-form missingness is an easy and effective way to achieve the advantages of a planned missing design without sacrificing accuracy or precision.

CONCLUSIONS

As should be clear, missing data should no longer be considered a bane to researchers. Modern techniques, when applied in an appropriately principled manner, will yield unbiased and suitably powered parameter estimates for any statistical model chosen. Accurate inferences are possible even under conditions of selective nonresponse (i.e., when the missing data mechanism is MAR). To optimize the ability to recover from the losses associated with unplanned missing data, we admonish researchers to identify and measure all known and likely predictors of nonresponse. To facilitate this practice, researchers should carefully scrutinize extant data sets to identify variables that are predictive of the unplanned missing data. The details of such an analysis can easily be included as an online appendix for interested researchers to access. This online appendix can also contain summary reports of the fraction of missing information in the means and variances of the variables on the data set (perhaps in the form of a histogram of the FMI values of the means and the FMI values for the variances). As well as provide a detailed log of the steps and options that were selected in treating the missing data. With the ease and capacity of web repositories, we can easily be much more transparent in sharing information about a given data set.

Another admonishment that we offer is to leverage the advantages of using planned missing data designs in

conjunction with modern treatments and latent variable analyses. The clear gains in cost savings and the potential gains in validity are both sufficient reasons to employ these designs. We acknowledge that the details of how to properly implement planned missing data designs can be more cumbersome than the traditional collect-everything-from-everyone approach. The rapidly expanding literature in this area provides the needed details for proper guidance in constructing data collection protocols with planned missing elements (as new works emerge, they will be added to our online bibliography at immap.educ.ttu.edu). We see the benefits of these designs in terms of the costs, veracity, and verisimilitude of research enterprise as far outweighing the time and thought needed to optimally include planned missing data designs in all future studies. In fact, planned missing elements can also be implemented in active longitudinal studies going forward.

REFERENCES

Allison, P. D. (2002). *Missing data*. Thousand Oaks, CA: Sage Publications.

Allison, P. D. (2006). Multiple imputation of categorical variables under the multivariate normal model. Retrieved from http://www.statisticalhorizons.com

Allison, E. R., Goodson, P., & Neilands, T. B. (2008). Out of sight, not out of mind: Strategies for handling missing data. *American Journal of Health Behavior*, *32*(1), 83–92.

Anderson, T. W. (1957). Maximum likelihood estimates for a multivariate normal distribution when some observations are missing. *Journal of the American Statistical Association*, *52*(278), 200–203. doi:10.1080/01621459.1957.10501379

Arbuckle, J. L. (1996). Full information estimation in the presence of incomplete data. In G. A. Marcoulides & R. E. Schumacker (Eds.), *Advanced structural equation modeling* (pp. 243–277). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Asparouhov, T., & Muthén, B. O. (2008). *Auxiliary variables predicting missing data*. Technical report. Retrieved from http://www.statmodel.com/do

Asparouhov, T., & Muthén, B. (2010a). *Multiple imputation with Mplus*. Technical report. Retrieved from http://www. statmodel.com

Asparouhov, T., & Muthén, B. (2010b). Weighted least squares estimation with missing data. Technical report. Retrieved from http://www.statmodel.com/download/GstrucMissingRevision.pdf

Azar, B. (2002). Finding a solution for missing data. *Monitor on Psychology*, *33*, 70.

Baraldi, A. N., & Enders, C. K. (2010). An introduction to modern missing data analyses. *Journal of School Psychology*, *48*, 5–37. doi: 10.1016/j.jsp.2009.10.001

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182. doi: 10.1037/0022-3514.51.6.1173

Beale, E. M. L., & Little, R. J. A. (1975). Missing values in multivariate analysis. *Journal of the Royal Statistical Society, Series B*, *37*, 129–145.

Beglinger, L. J., Gaydos, B., Tangphao-Daniels, O., Duff, K., Kareken, D. A., Crawford, J., … Siemers, E. R. (2005). Practice effects and the use of alternate forms in serial neuropsychological testing. *Archives of Clinical Neuropsychology*, *20*, 517–529. doi: 10.1016/j.acn.2004.12.003

Bell, R. Q. (1953). Convergence: An accelerated longitudinal approach. *Child Development*, *24*(2), 145–152. doi: 10.2307/1126345

Beunckens, C., Molenberghs, G., Thij, H., & Verbeke, G. (2007). Incomplete hierarchical data. *Statistical Methods in Medical Research*, *16*, 457–492. doi: 10.1177/0962280206075310

Biesanz, J. C., Falk, C., & Savalei, V. (2010). Assessing mediational models: Testing and interval estimation for indirect effects. *Multivariate Behavioral Research*, *45*, 661–701. doi: 10.1080/00273171.2010.498292

Bodner, T. E. (2006). Missing data: Prevalence and reporting practices. *Psychological Reports*, *99*, 675–680. doi: 10.2466/PR0.99.3.675-680

Bodner, T. E. (2008). What improves with increasing missing data imputations? *Structural Equation Modeling*, *15*, 651–675.

Brown, C. H. (1983). Asymptotic comparison of missing data procedures for estimating factor loadings. *Psychometrika*, *48*, 269–291. doi: 10.1007/BF02294022

Brown, M. B. (1974). Identification of the sources of significance in two-way tables. *Applied Statistics*, *23*, 405–413. doi: 10.2307/2347132

Brown, R. L. (1994). Efficacy of the indirect approach for estimating structural equation models with missing data: A comparison of five methods. *Structural Equation Modeling*, *1*, 287–316. doi: 10.1080/10705519409539983

Browne, M. W. (1984). Asymptotically distribution-free methods for the analysis of covariance structures. *British Journal of Mathematical and Statistical Psychology*, *37*, 62–83. doi: j.2044-8317.1984.tb00789.x

Bunting, B., & Adamson, G. (2000). Assessing reliability and validity in the context of planned incomplete data structures for multitrait-multimethod models. *Developments in Survey Methodology*, *15*, 37–53.

Bunting, B. P., Adamson, G., & Mulhall, P. K. (2002). A Monte Carlo examination of an MTMM model with planned incomplete data structures. *Structural Equation Modeling*, *9*, 369–389. doi: 10.1207/S15328007SEM0903_4

Cai, L., & Lee, T. (2009) Covariance structure model fit testing under missing data: An Application of the supplemented EM algorithm. *Multivariate Behavioral Research*, *44*, 281–304. doi: 10.1080/00273170902794255

Carter, W. H., Jr., & Meyers, R. H. (1973). Maximum likelihood estimation from linear combinations of discrete probability functions. *Journal of the American Statistical Association*, *68*, 203–206. doi: 10.1080/01621459.1973.10481366

Chen, T., & Fienberg, S. (1976). The analysis of contingency tables with incompletely classified data. *Biometrics*, *32*, 133–144. doi: 10.2307/2529344

Cheung, M. (2007). Comparison of methods of handling missing time-invariant covariates in latent growth models under the assumption of missing completely at random. *Organizational Research Methods*, *10*, 609. doi: 10.1177/1094428106295499

Collins, L. M., Schafer, J. L., & Kam, C. M. (2001). A comparison of inclusive and restrictive strategies in modern missing data procedures. *Psychological Methods*, *6*, 330–351. doi: 10.1037/1082-989X.6.4.330

Davey, A., & Savla, J. (2010). *Statistical power analysis with missing data. A structural equation modeling approach*. New York, NY: Routledge.

Davey, A., Savla, J., & Luo, Z. (2005). Issues in evaluating model fit with missing data. *Structural Equation Modeling*, *12*, 578–597. doi: 10.1207/s15328007sem1204_4

Demirtas, H., Freels, S. A., & Yucel, R. M. (2008). Plausibility of multivariate normality assumption when multiply imputing non-Gaussian continuous outcomes: A simulation assessment. *Journal*

of Statistical Computation and Simulation, *78*, 69–84. doi: 10.1080/10629360600903866

Dempster, A. P., Laird, N. M., & Rubin, D. B. (1977). Maximum likelihood from incomplete data via the EM algorithm. *Journal of the Royal Statistical Society, Series B*, *39*, 1–38.

Dings, J., Childs, R., & Kingston, N. (2002). *The effects of matrix sampling on student score comparability in constructed-response and multiple-choice assessments*. Washington, DC: Council of Chief State School Officers.

Efron, B. (1979). Bootstrap methods: Another look at the jackknife. *Annals of Statistics*, *7*, 1–26. doi: 10.1214/aos/1176344552

Efron, B., & Tibshirani, R. J. (1994). *An introduction to the bootstrap*. New York, NY: Chapman & Hall/CRC.

Enders, C. K. (2001a). The impact of nonnormality on full information maximum likelihood estimation for structural equations models with missing data. *Psychological Methods*, *6*, 352–370. doi: 10.1037/1082-989X.6.4.352

Enders, C. K. (2001b). The performance of the full information maximum likelihood estimator in multiple regression models with missing data. *Educational and Psychological Measurement*, *61*, 713–740. doi: 10.1177/0013164401615001

Enders, C. K. (2001c). A primer on maximum likelihood algorithms available for use with missing data. *Structural Equation Modeling*, *8*(1), 128–141. doi: 10.1207/S15328007SEM0801_7

Enders, C. K. (2002). Applying the Bollen-Stine bootstrap for goodness-of-fit measures to structural equation models with missing data. *Multivariate Behavioral Research*, *37*(3), 359–377. doi: 10.1207/S15327906MBR3703_3

Enders, C. K. (2003). Using the expectation maximization algorithm to estimate coefficient alpha for scales with item-level missing data. *Psychological Methods*, *8(3)*, 322–337. doi: 10.1037/1082-989X.8.3.322

Enders, C. K. (2004). The impact of missing data on sample reliability estimates: Implications for reliability reporting practices. *Educational and Psychological Measurement*, *64*, 419–436. doi: 10.1177/0013164403261050

Enders, C. K. (2006). A primer on the use of modern missing-data methods in psychosomatic medicine research. *Psychosomatic Medicine*, *68*, 427–436. doi: 10.1097/01.psy.0000221275.75056.d8

Enders, C. K. (2008). A note on the use of missing auxiliary variables in full information maximum likelihood-based structural equation models. *Structural Equation Modeling*, *15*(3), 434–448. doi: 10.1080/10705510802154307

Enders, C. K. (2010). *Applied missing data analysis*. New York, NY: Guilford.

Enders, C. K. (2011). Missing not at random models for latent growth curve analysis. *Psychological Methods*, *16*, 1–16. doi: 10.1037/a0022640

Enders, C. K. (2013). Dealing with missing data in developmental research. *Child Development Perspectives*, *7*, 27–31. doi: 10.1111/cdep.12008

Enders, C. K., & Bandalos, D. L. (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling*, *8*, 430–457. doi: 10.1207/S15328007SEM0803_5

Enders, C. K., Fairchild, A. J., & MacKinnon, D. P. (2013). Using Bayesian parameter simulation to evaluate mediation effects with missing data. *Multivariate Behavioral Research*, *48*, 340–369. doi: 10.1080/00273171.2013.784862

Enders, C. K., Dietz, S., Montague, M., & Dixon, J. (2006). Modern alternatives for dealing with missing data in special education research. In T. E. Scruggs & M. A. Mastropieri (Eds.), *Advances in learning and behavioral disabilities* (Vol. 19, pp. 101–130). New York, NY: Elsevier.

Enders, C. K., & Gottschall, A. C. (2011). Multiple imputation strategies for multiple group structural equation models. *Structural Equation Modeling*, *18*, 35–54. doi: 10.1080/10705511.2011.532695

Enders, C. K., & Peugh, J. L. (2004). Using an EM covariance matrix to estimate structural equation models with missing data: Choosing an adjusted sample size to improve the accuracy of inferences. *Structural Equation Modeling*, *11*, 1–19. doi: 10.1207/S15328007SEM1101_1

Finkbeiner, C. (1979). Estimation for the multiple factor model when data are missing. *Psychometrika*, *44*, 409–420. doi: 10.1007/BF02296204

Fraley, C. (1999). On computing the largest fraction of missing information for the EM algorithm and the worst linear function for data augmentation. *Computation Statistics & Data Analysis*, *31*, 13–26. doi: 10.1016/S0167-9473(99)00003-1

Garnier Villarreal, M., Dowsett, C., & Little, T. D. (2013). *Using component scores as auxiliary variables: How many components are needed?* Manuscript in preparation.

Garnier Villarreal, M., Rhemtulla, M., & Little, T. D. (2014). Two-method planned missing designs for longitudinal research. *International Journal of Behavioral Development, 38*, 411–422. doi: 10.1177/0165025414542711

Gelman, A., and Rubin, D. B. (1992), Inference from iterative simulation using multiple sequences. *Statistical Science*, *7*, 457–472. doi: 10.1214/ss/1177011136

Gleason, T. C., & Staelin, R. (1975). A proposal for handling missing data. *Psychometrika*, *40*, 229–252. doi: 10.1007/BF02291569

Gold, M. S., & Bentler, P. M. (2000). Treatments of missing data: A Monte Carlo comparison of RBHDI, iterative stochastic regression imputation, and expectation-maximization. *Structural Equation Modeling*, *7*, 319–355. doi: 10.1207/S15328007SEM0703_1

Gold, M. S., Bentler, P. M., & Kim, K. H. (2003). A comparison of maximum-likelihood and asymptotically distribution-free methods of treating incomplete nonnormal data. *Structural Equation Modeling*, *10*, 47–79. doi: 10.1207/S15328007SEM1001_3

Gonzalez, J. M., & Eltinge, J. L. (2008). Adaptive Matrix Sampling for the consumer expenditure quarterly interview survey. *Proceedings of the Section on Survey Research Methods, American Statistical Association*, 2081–2088.

Gottschall, A. C., West, S. G., & Enders, C. K. (2012). A comparison of item-level and scale-level multiple imputation for questionnaire batteries. *Multivariate Behavioral Research*, *47(1)*, 1–25. doi: 10.1080/00273171.2012.640589

Graham, J. W. (2003). Adding missing-data-relevant variables to FIML-based structural equation models. *Structural Equation Modeling*, *10*, 80–100. doi: 10.1207/S15328007SEM1001_4

Graham, J. W. (2009). Missing data analysis: Making it work in the real world. *Annual Review of Psychology*, *60*, 549–576. doi: 10.1146/annurev.psych.58.110405.085530

Graham, J. W. (2012). *Missing data: Analysis and design*. New York, NY: Springer. doi:10.1007/978-1-46144018-5_1

Graham, J. W., Cumsille, P. E., & Shevock, A. E. (2012). Methods for handling missing data. In J. A. Schinka & W. F. Velicer (Eds.), *Handbook of Psychology, Research methods in psychology* (2nd ed.,Vol. 2, pp. 109–141). New York, NY: John Wiley & Sons. doi: 10.1002/9781118133880.hop202004

Graham, J. W., Hofer, S. M., Donaldson, S. I., MacKinnon, D. P., & Schafer, J. L. (1997). Analysis with missing data in prevention research. In K. J. Bryant, M. Windle, & S. G. West (Eds.), *The science of prevention: Methodological advances from alcohol and substance abuse research* (pp. 325–366). Washington, DC: American Psychological Association. doi: 10.1037/10222-010

Graham, J. W., Hofer, S. M., & MacKinnon, D. P. (1996). Maximizing the usefulness of data obtained with planned missing value patterns: An application of maximum likelihood procedures. *Multivariate Behavioral Research*, *31*, 197–218. doi: 10.1207/s15327906mbr3102_3

Graham, J. W., Hofer, S. M., & Piccinin, A. M. (1994). Analysis with missing data in drug prevention research. In L. M. Collins & L. Seitz (Eds.), *Advances in data analysis for prevention intervention research* (pp. 13–63). Washington, DC: National Institute on Drug Abuse.

Graham, J. W., Olchowski, A. E., & Gilreath, T. D. (2007). How many imputations are really needed? Some practical clarifications of multiple imputation theory. *Prevention Science*, *8*, 206–213. doi: 10.1007/s11121-007-0070-9

Graham, J. W., & Schafer, J. L. (1999). On the performance of multiple imputation for multivariate normal data with small sample size. In R. H. Hoyle (Ed.), *Statistical strategies for small sample research* (pp. 1–29). Thousand Oaks, CA: Sage.

Graham, J. W., Taylor, B. J., & Cumsille, P. E. (2001). Planned missing data designs in the analysis of change. In L. M. Collins & A. G. Sayer (Eds.), *New methods for the analysis of change* (pp. 335–353). Washington, DC: American Psychological Association. doi: 10.1037/10409-011

Graham, J. W., Taylor, B. J., Olchowski, A. E., & Cumsille, P. E. (2006). Planned missing data designs in psychological research. *Psychological Methods*, *11*, 323–343. doi: 10.1037/1082-989X.11.4.323

Haitovsky, Y. (1968). Missing data in regression analysis. *Journal of the Royal Statistical Society, Series B*, *30*, 67–82.

Harel, O. (2007). Inferences on missing information under multiple imputation and two-stage multiple imputation. *Statistical Methodology*, *4*, 75–89. doi: 10.1016/j.stamet.2006.03.002

Harel, O., Hofer, S., Hoffman, L., Pedersen, N., & Johansson, B. (2007). Population inference with mortality and attrition in longitudinal studies on aging: A two-stage multiple imputation method. *Experimental Aging Research*, *33*, 187–203. doi: 10.1080/03610730701239004

Harel, O., Stratton, J., & Aseltine, R. H. Jr. (2011). *Designed missingness to better estimate efficacy of behavioral studies*. Technical Report 11-15. Storrs, CT: Department of Statistics, University of Connecticut.

Hartley, H. O. (1958). Maximum likelihood estimation from incomplete data. *Biometrics*, *14*, 174–194. doi: 10.2307/2527783

Hartley, H. O., & Hocking, R. R. (1971). The analysis of incomplete data. *Biometrics*, *27*, 783–823. doi: 10.2307/2528820

Healy, M., & Westmacott, M. (1956). Missing values in experiments analysed on automatic computers. *Applied Statistics*, *5*, 203–206. doi: 10.2307/2985421

Hofer, S. M., & Hoffman, L. (2007). Statistical analysis with incomplete data: A developmental perspective. In: T. D. Little, J. A. Bovaird, & N. A. Card (Eds.), *Modeling ecological and contextual effects in longitudinal studies of human development* (pp. 13–32). Mahwah, NJ: LEA.

Honaker, J., & King, G. (2010). What to do about missing values in time-series cross-section data. *American Journal of Political Science*, *54*(2), 561–581. doi: 10.1111/j.1540-5907.2010.00447.x

Honaker, J., King, G., & Blackwell, M. (2011). Amelia II: A program for missing data. *Journal of Statistical Software*, *45*, 1–47. Retrieved from http://www.jstatsoft.org/v45/i07

Howard, W., Rhemtulla, M., & Little, T. D. (2015). Using principal components as auxiliary variables in missing data estimation. *Multivariate Behavioral Research*, *50*, 285–299. doi: 10.1080/00273171.2014.999267

Iacobucci, D. (1995). The analysis of variance for unbalanced data. *Marketing Theory and Applications*, *6*, 337–343.

IBM Corp. (2012). *IBM SPSS Statistics for Windows, Version 21.0*. Armonk, NY: IBM Corp.

Jamshidian, M., & Bentler, P. M. (1999). ML estimation of mean and covariance structures with missing data using complete data routines. *Journal of Educational and Behavioral Statistics*, *24*, 21–41. doi: 10.3102/10769986024001021

Jia, F., Moore, E. W. G., Kinai, R., Crowe, K. S., Schoemann, A. M., & Little, T. D. (2014). Planned missing data design on small sample size: How small is too small? *International Journal of Behavioral Development*, *38*, 435–452. doi: 10.1177/0165025414531095

Jöreskog, K. G. (1967). Some contributions to maximum likelihood factor analysis. *Psychometrika*, *32*(4), 443–482. doi: 10.1007/BF02289658

Jorgensen, T. D., Schoemann, A. S., McPherson, B., Rhemtulla, M., Wu, W., & Little, T. D. (2013). Optimal assignment methods in three-form planned missing data designs for longitudinal panel studies. *International Journal of Behavioral Development*, *38*, 397–410. doi: 10.1177/0165025414531094

Kenward, M. G., Lesaffre, E., & Molenberghs, G. (1994). An application of maximum-likelihood and generalized estimating equations to the analysis of ordinal data from a longitudinal-study with cases missing at random. *Biometrics*, *50*(4), 945–953. doi: 10.2307/2533434

Kenward, M. G., & Molenberghs, G. (1998). Likelihood based frequentist inference when data are missing at random. *Statistical Science*, *13*, 236–247. doi: 10.1214/ss/1028905886

Kim, K. H., & Bentler, P. M. (2002). Tests of homogeneity of means and covariance matrices for multivariate incomplete data. *Psychometrika*, *67*, 609–624. doi: 10.1007/BF02295134

Kreuter, F., & Olson, K. (2011). Multiple auxiliary variables in nonresponse adjustment. *Sociological Methods & Research*, *40*(2), 311–332. doi: 10.1177/0049124111400042

Lang, K. M. (2013). *Streamlining missing data analysis by aggregating multiple imputations at the data level: A Monte Carlo simulation to assess the tenability of the supermatrix approach*. Unpublished master's thesis, University of Kansas, Lawrence, KS.

Lang, K. M., & Little, T. D. (2014). The supermatrix technique: A simple framework for hypothesis testing with missing data. *International Journal of Behavioral Development*, *38*, 461–470. doi: 10.1177/0165025413514326

Lauritzen, S. (1995). The EM algorithm for graphical association models with missing data. *Computational Statistics & Data Analysis*, *19*, 191–201. doi: 10.1016/0167-9473(93)E0056-A

Lee, S.-Y. (1986). Estimation for structural equation models with missing data. *Psychometrika*, *51*, 93–99. doi: 10.1007/BF02294002

Lee, T., & Cai, L. (2012). Alternative multiple imputation inference for mean and covariance structure modeling. *Journal of Educational and Behavioral Statistics*, *37*(6), 675–702. doi: 10.3102/1076998612458320

Little, R. J. A. (1988). A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Association*, *83*, 1198–1202. doi: 10.1080/01621459.1988.10478722

Little, R. J. A., & Rubin, D. B. (1989). The analysis of social science data with missing values. *Sociological Methods and Research*, *18*, 292–326. doi: 10.1177/0049124189018002004

Little, R. J. A., & Rubin, D. B. (2002). *Statistical analysis with missing data*. Hoboken, NJ: John Wiley & Sons.

Little, R. J. A., & Schenker, N. (1995). Missing data. In G. Arminger, C. C. Clogg, & M. E. Sobel (Eds.), *Handbook of statistical modeling for the social and behavioral sciences* (pp. 39–75). New York, NY: Plenum. doi: 10.1007/978-1-4899-1292-3_2

Little, T. D. (2013). *Longitudinal structural equation modeling*. New York, NY: Guilford.

Little, T. D., Cunningham, W. A., Shahar, G., & Widaman, K. F. (2002). To parcel or not to parcel: Exploring the question, weighing the merits. *Structural Equation Modeling*, *9*(2), 151–173. doi: 10.1207/S15328007SEM0902_1

Little, T. D., Howard W. J., McConnell, E. K., & Stump, K. N. (2011). Missing data in large data projects: Two methods of missing data imputation when working with large data projects. Department of Psychology, University of Kansas, *KUant Guides*. Retrieved from www.quant.ku.edu/resources/guides.html

Little, T. D., Jones, S. M., Henrich, C. C., Hawley, P. H. (2003). Disentangling the "whys" from the "whats" of aggressive behavior. *International Journal of Behavioral Development*, *27*(2), 122–133. doi: 10.1080/01650250244000128

Little, T. D., Jorgensen, T. D., Lang, K. M., & Moore, E. W. G. (2014). On the joys of missing data. *Journal of Pediatric Psychology*, *39*, 151–161. doi: 10.1093/jpepsy/jst048

Little, T. D., Rhemtulla, M., Gibson, K., & Schoemann, A. M. (2013). Why the items versus parcels controversy needn't be one. *Psychological Methods*, *18*, 285–300. doi: 10.1037/a0033266

Little, T. D., Schnabel, K. U., & Baumert, J. E. (Eds.). (2000). *Modeling longitudinal and multilevel data: Practical issues, applied approaches, and specific examples*. Mahwah, NJ: Lawrence Erlbaum Associates.

Longford, N. T. (2005). *Missing data and small-area estimation: Modern analytical equipment for the survey statistician*. New York, NY: Springer.

McArdle, J. (1994). Structural factor analysis experiments with incomplete data. *Multivariate Behavioral Research*, *29*, 409–454. doi: 10.1207/s15327906mbr2904_5

McArdle, J., & Hamagami, F. (1992). Modeling incomplete longitudinal and cross-sectional data using latent growth structural models. *Experimental Aging Research*, *18*, 145–166. doi: 10.1080/03610739208253917

McArdle, J. J., & Hamagami, F. (2001). Linear dynamic analyses of incomplete longitudinal data. In L. Collins & A. Sayer (Eds.), *Methods for the analysis of change* (pp. 139–175). Washington, DC: American Psychological Association.

McArdle, J. J., & Woodcock, R. W. (1997). Expanding test–retest designs to include developmental time-lag components. *Psychological Methods*, *2*, 403–435. doi: 10.1037/1082-989X.2.4.403

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research*, *39*(1), 99–128. doi: 10.1207/s15327906mbr3901_4

McKnight, P. E., McKnight, K. M., Sidani, S., & Figueredo, A. J. (2007). *Missing data: A gentle introduction*. New York, NY: Guilford.

Meng, X.-L., & Rubin, D. B. (1991). Using EM to obtain asymptotic variance-covariance matrices: The SEM algorithm. *Journal of the American Statistical Association*, *86*, 899–909. doi: 10.1080/01621459.1991.10475130

Meng, X.-L., & Rubin, D. B. (1992). Performing likelihood ratio tests with multiply-imputed data sets. *Biometrika*, *79*(1), 103–111. doi: 10.1093/biomet/79.1.103

Mislevy, R. J., Beaton, A. E., Kaplan, B., & Sheehan, K. M. (1992). Estimating population characteristics from sparse matrix samples of item responses. *Journal of Educational Measurement*, *29*, 133–161. doi: 10.1111/j.1745-3984.1992.tb00371.x

Muthén, B., & Asparouhov, T. (2009). Multilevel regression mixture analysis. *Journal of the Royal Statistical Society, Series A*, *172*, 639–657. doi: 10.1111/j.1467-985X.2009.00589.x

Muthén, B., & Asparouhov, T. (2012). Bayesian structural equation modeling: A more flexible representation of substantive theory. *Psychological Methods*, *17*(3), 313–335. doi: 10.1037/a0026802

Muthén, B., Kaplan, D., & Hollis, M. (1987). On structural equation modeling with data that are not missing completely at random. *Psychometrika*, *51*, 431–462. doi: 10.1007/BF02294365

Muthén, L. K., & Muthén, B. O. (2012). *Mplus user's guide* (7th ed.). Los Angeles, CA: Muthén & Muthén.

Nakagawa, S., & Freckleton, R. P. (2010). Model averaging, missing data and multiple imputation: A case study for behavioral ecology. *Behavioral Ecology and Sociobiology*, *65*, 103–116. doi: 10.1007/s00265-010-1044-7 .

Orchard, T., & Woodbury, M. A. (1972). *A missing information principle: Theory and applications*. Paper presented at the sixth Berkeley symposium on mathematical statistics and probability, University of California, Berkeley, CA.

Peugh, J. L., & Enders, C. K. (2004). Missing data in educational research: A review of reporting practices and suggestions for improvement. *Review of Educational Research*, *74*, 525–556. doi: 10.3102/00346543074004525

Popham, W. J. (1993). Circumventing the high costs of authentic assessment. *Phi Delta Kappan*, *7*, 470–473.

Pornprasertmanit, S., Miller, P., Schoemann, A. M., & Rosseel, Y. (2013). *semTools: Useful tools for structural equation modeling*. R package version 0.4-0. Retrieved from http://cran.r-project.org/web/packages/semTools/index.html

R Core Team. (2012). *R: A language and environment for statistical computing (version 2.15.2)* [Computer software]. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://www.R-project.org/

Raghunathan, T. E. (2004). What do we do with missing data? Some options for analysis of incomplete data. *Annual Review of Public Health*, *25*, 88–117. doi: 10.1146/annurev.publhealth.25.102802.124410

Raghunathan, T. E., & Grizzle, J. E. (1995). A split questionnaire survey design. *Journal of the American Statistical Association*, *90*, 54–63. doi: 10.1080/01621459.1995.10476488

Reiter, J. P., Raghunathan, T. E., & Kinney, S. (2006). The importance of the sampling design in multiple imputation for missing data. *Survey Methodology*, *32*(2), 143–150.

Rhemtulla, M., Jia, F., Wu, W., & Little, T. D. (2014). Planned missing designs to optimize the efficiency of latent growth parameter estimates. *International Journal of Behavioral Development*, *38*, 423–434. doi: 10.1177/0165025413514324

Rhemtulla, M. & Little, T. D. (2013). Planned missing data designs for longitudinal organizational research. In M. Raukko & E. Paavilainen-Mäntymäki (Eds.), *Handbook of longitudinal research methods in organisation and business studies* (pp. 56–77). Northampton, MA: Edward Elgar Publishing. doi: 10.4337/9780857936790.00012

Rhemtulla, M., Savalei, V., & Little, T. D. (2014). On the asymptotic relative efficiency of planned missingness designs. *Psychometrika, Advance online publication.* doi: 10.1007/s11336-014-9422-0

Rosseel, Y. (2012). lavaan: An R package for structural equation modeling. *Journal of Statistical Software*, *48*, 1–36. Retrieved from http://lavaan.ugent.be/

Rovine, M. J. (1994). Latent variables models and missing data analysis. In A. von Eye & C. C. Clogg (Eds.), *Latent variables analysis: Applications for developmental research* (pp. 181–225). Thousand Oaks, CA: Sage.

Rubin, D. B. (1976). Inference and missing data. *Biometrika*, *63*, 581–592. doi: 10.1093/biomet/63.3.581

Rubin, D. B. (1978). Multiple imputations in sample surveys—A phenomenological bayesian approach to nonresponse. *Proceedings of the Survey Research Methods Section of the American Statistical Association*, 30–34.

Rubin, D. B. (1987). *Multiple imputation for nonresponse in surveys*. New York, NY: Wiley. doi: 10.1002/9780470316696

Rubin, D. B. (1991). EM and beyond. *Psychometrika*, *56*, 241–254. doi: 10.1007/BF02294461

Rubin, D. B. (1996). Multiple imputation after 18+ years. *Journal of the American Statistical Association*, *91*, 473–489. doi: 10.1080/01621459.1996.10476908

Salthouse, T., & Tucker-Drob, E. (2008). Implications of short-term retest effects for the interpretation of longitudinal change. *Neuropsychology*, *22*, 800–811. doi: 10.1037/a0013091

SAS Institute (2011). *SAS/STAT software, version 9.3*. Cary, NC: SAS Institute Inc.

Savalei, V. (2010a). Small sample statistics for incomplete nonnormal data: extensions of complete data formulae and a Monte Carlo comparison. *Structural Equation Modeling*, *17*, 241–264. doi: 10.1080/10705511003659375

Savalei, V. (2010b). Expected versus observed information in SEM with incomplete normal and nonnormal data. *Psychological Methods*. doi: 10.1037/a0020143

Savalei, V., & Bentler, P. M. (2005). A statistically justified pairwise ML method for incomplete nonnormal data: A comparison with direct ML and pairwise ADF. *Structural Equation Modeling*, *12*, 183–214. doi: 10.1207/s15328007sem1202_1

Savalei, V., & Bentler, P. M. (2009). A two-stage approach to missing data: Theory and application to auxiliary variables. *Structural Equation Modeling*, *16*, 477–497. doi: 10.1080/10705510903008238

Savalei, V., & Falk, C. F. (2014). Robust two-stage approach outperforms robust FIML with incomplete non-normal data. *Structural Equation Modeling*, *21*, 280–302. doi: 10.1080/10705511.2014.882692

Savalei, V., & Rhemtulla, M. (2011). *Some explorations of the local and global measures of missing information*. Paper presented at the the 76th annual and 17th international meeting of the Psychometric Society, Hong Kong.

Savalei, V., & Rhemtulla, M. (2012). Teacher's corner: On obtaining estimates of the fraction of missing information from FIML. *Structural Equation Modeling*, *19*, 477–494. doi: 10.1080/10705511.2012.687669

Savalei, V., & Yuan, K. (2009). On the model-based bootstrap with missing data: Obtaining a p-value for a test of exact fit. *Multivariate Behavioral Research*, *44*, 741–763. doi: 10.1080/00273170903333590

Schafer, J. L. (1997). *Analysis of incomplete multivariate data*. New York, NY: Chapman Hall.

Schafer, J. L. (2001). Multiple imputation with PAN. In L. M. Collins & A. G. Sayer (Eds.), *New methods for the analysis of change* (pp. 357–377). Washington, DC: American Psychological Association. doi: 10.1037/10409-012

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, *7*, 147–177. doi: 10.1037/1082-989X.7.2.147

Schafer, J. L., & Olsen, M. K. (1998). Multiple imputation for multivariate missing-data problems: A data analyst's perspective. *Multivariate Behavioral Research. Special Issue: Innovative Methods for Prevention Research, 33*(4), 545–571. doi: 10.1207/s15327906mbr3304_5

Schafer, J. L., & Yucel, R. M. (2002). Computational strategies for multivariate linear mixed models with missing values. *Journal of Computational and Graphical Statistics*, *11*, 421–442.

Shin, T., Davidson, M. L., & Long, J. D. (2010). Effects of missing data methods in structural equation modeling with nonnormal longitudinal data. *Structural Equation Modeling*, *16*, 70–98. doi: 10.1080/10705510802569918

Shoemaker, D. M. (1973). *Principles and procedures of multiple matrix sampling*. Cambridge, MA: Ballinger Publishing.

Sinharay, S., Stern, H. S., & Russell, D. (2001). The use of multiple imputation for the analysis of missing data. *Psychological Methods*, *6*, 317–329. doi: 10.1037/1082-989X.6.4.317

Sobel, M. E. (1982). Asymptotic intervals for indirect effects in structural equations models. In S. Leinhart (Ed.), *Sociological methodology 1982* (pp. 290–312). San Francisco, CA: Jossey-Bass.

Song, X. Y., & Lee, S. Y. (2002). Analysis of structural equation model with ignorable missing continuous and polytomous data. *Psychometrika*, *67*, 261–288. doi: 10.1007/BF02294846

StataCorp. (2013). *Stata Statistical Software: Release 13*. College Station, TX: StataCorp LP.

Su, Y., Gelman, A., Hill, J., & Yajima, M. (2011). Multiple imputation with diagnostics (mi) in R: Opening windows into the black box. *Journal of Statistical Software*, *45*(2), 1–31.

Tanner, M. A., & Wong, W. H. (1987). The calculation of posterior distributions by data augmentation. *Journal of the American Statistical Association*, *82*, 528–540. doi: 10.1080/01621459.1987.10478458

Van Buuren, S. (2012). *Flexible imputation of missing data*. Boca Raton, FL: CRC Press. doi: 10.1201/b11826

Van Buuren, S., & Groothuis-Oudshoorn, K. (2011). MICE: Multivariate imputation by chained equations in R. *Journal of Statistical Software*, *45*(3), 1–67.

Von Hippel, P. T. (2005). How many imputations are needed? A comment on Hershberger and Fisher (2003). *Structural Equation Modeling*, *12*, 334–335. doi: 10.1207/s15328007sem1202_8

Von Hippel, P. T. (2009). How to impute interactions, squares, and other transformed variables. *Sociological Methodology*, *39*, 265–291. doi: 10.1111/j.1467-9531.2009.01215.x

Wacholder, S., Carroll, R. J., Pee, D., & Gail, M. H. (1994). The partial questionnaire design for case-control studies. *Statistics in Medicine*, *13*, 623–634. doi: 10.1002/sim.4780130523

West, S. G. (2001). New approaches to missing data in psychological research: Introduction to the special section. *Psychological Methods*, *6*(4), 315–316. doi: 10.1037/1082-989X.6.4.315

White, I. R., Royston, P., & Wood, A. M. (2011). Multiple imputation using chained equations: Issues and guidance for practice. *Statistics in Medicine*, *30*(4), 377–399. doi: 10.1002/sim.4067

Wilkinson, L., & Task Force on Statistical Inference (1999). Statistical methods in psychology journals: Guidelines and explanations. *American Psychologist*, *54*(8). 594–604. doi: 10.1037/0003-066X.54.8.594

Wohwill, J. F. (1973). *The study of behavioral development*. New York, NY: Academic Press.

Wood, A. M., White, I. R., & Thompson, S. G. (2004). Are missing outcome data adequately handled? A review of published randomized

controlled trials in major medical journals. *Clinical Trials Review*, *1*, 368–376. doi: 10.1191/1740774504cn032oa

Wothke, W. (2000). Longitudinal and multi-group modeling with missing data. In T. D. Little, K. U. Schnabel, & J. Baumert (Eds.), *Modeling longitudinal and multiple group data: Practical issues, applied approaches and specific examples* (pp. 219–240). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Wu, W., West, S. G., & Taylor, A. B. (2009). Evaluating model fit for growth curve models: Integration of fit indices from SEM and MLM frameworks. *Psychological Methods*, *14*(3), 183–201. doi: 10.1037/a0015858

Wu, W., & Jia, F. (2013). A new procedure to test mediation with missing data through nonparametric bootstrapping. *Multivariate Behavioral Research*, *48*, 663–691. doi: 10.1080/00273171.2013.816235

Yoo, J. E. (2009). The effect of auxiliary variables and multiple imputation on parameter estimation in confirmatory factor analysis. *Educational and Psychological Measurement*, *69*(6), 929–947. doi: 10.1177/0013164409332225

Yuan, K. (2009a). Identifying variables responsible for data not missing at random. *Psychometrika*, *74*, 233–256. doi: 10.1007/s11336-008-9088-6

Yuan, K. (2009b). Normal distribution based pseudo ML for missing data: With applications to mean and covariance structure analysis. *Journal of Multivariate Analysis*, *100*, 1900–1918. doi: 10.1016/j.jmva.2009.05.001

Yuan, K.-H., & Bentler, P. M. (2000). Three likelihood-based methods for mean and covariance structure analysis with nonnormal missing data. *Sociological Methodology*, *30*, 165–200. doi: 10.1111/0081-1750.00078

Yuan, Y., & MacKinnon, D. P. (2009). Bayesian mediation analysis. *Psychological Methods*, *14*(4), 301–322. doi: 10.1037/a0016972

Yucel, R. M., & Raghunathan, T. (2006). *Sequential hierarchical regression imputation (SHRIMP)*. Paper presented at the annual meeting of the American Statistical Association, Seattle, WA.

Zhang, Z., & Wang, L. (2013). Methods for mediation analysis with missing data. *Psychometrika*, *78(*1*),* 154–184. doi: 10.1007/s11336-012-9301-5

CHAPTER 18

Person-Oriented Approaches

G. ANNE BOGAT, ALEXANDER VON EYE, and LARS R. BERGMAN

CHAPTER OVERVIEW 797
DESCRIPTION OF THE VARIABLE- AND PERSON-ORIENTED APPROACHES 799
Variable-Oriented Approach 800
Person-Oriented Approach 804
THREE EARLY PROTAGONISTS OF THE PERSON-ORIENTED APPROACH 812
William Stern 813
Kurt Lewin 814
Jack Block 815
STATISTICAL APPROACHES 818
Methods of Person-Oriented Data Analysis 818
Structural Equation Modeling 819
Log-Linear Modeling 822
Cluster Analysis 826
CONCLUSION AND FUTURE DIRECTIONS 837
REFERENCES 839

CHAPTER OVERVIEW

The variable-oriented approach has been the dominant paradigm in psychological research for most of its history. Person-oriented approaches have been advocated strongly at several junctures in this history; for example, at the turn of the twentieth century by James, Dewey, and Stern, in the 1930s by Lewin and Allport, in the 1960s by Block, and in the 1980s to the present by Magnusson, Bergman, and von Eye. Yet, even though there has been a noticeable increase in the use of person-oriented approaches, the majority of published research is still variable-oriented.

By variable-oriented approaches we mean theoretical and statistical approaches that discuss or demonstrate relationships among variables. For example, when we find that childhood depression is positively related to maternal depression, we are discussing the relationship between two variables. This may say little about a specific child. Assuming a moderate positive correlation, there will be many children whose childhood depression and maternal depression scores are far from the regression line, for instance, being fairly high in childhood depression and fairly low in maternal depression, contrary to the positive linear relationship indicated by the correlation coefficient. In developmental research, variable-oriented research focuses on constancy and change among the variables. Variable-oriented research also assumes that there are universal laws involving human behavior, thus, information is not lost when specific variable relationships that are part of a larger process are examined and when data are aggregated across individuals. In contrast, person-oriented approaches focus on patterns or profiles that describe individuals or groups of individuals, the constancy and change of these patterns or profiles, and whether or how these patterns or profiles relate to other profiles or patterns within as well as events outside the individual.

Kuhn (1962/2012) argued that it is inevitable that paradigms[2] become entrenched. Scientific training focuses on inculcating the values, theory, and methods of a specific paradigm; in essence, students are schooled in anticipation of joining a specific scientific community whose members were also indoctrinated in the paradigm. Thus, the basic building blocks upon which the student and then the scientist conduct research are unlikely to be questioned. In fact, Kuhn argued "that commitment and the apparent consensus it produces are prerequisites for normal science, i.e.,

[1]Color versions of Figures 18.1, 18.4–18.7 are available at http://onlinelibrary.wiley.com/book/10.1002/9781119125556

[2]In the 1969 postscript to his book, Kuhn noted that paradigm had two meanings: (1) "the entire constellation of beliefs, values, techniques, and so on shared by the members of a given community," a meaning Kuhn believed was better exemplified by the term *disciplinary matrix*; and (2) exemplars, examples to which scientists refer to understand the common knowledge base of a discipline. In this chapter, the first meaning of paradigm is used.

for the genesis and continuation of a particular research tradition" (p. 11).

This commitment and consensus to the dominant paradigm are two of the major reasons that paradigm shifts occur slowly. For example, there is no doubt few, if any, undergraduate or graduate students understand that the science they are being taught uses a variable-oriented paradigm. Without specifically defining the paradigm, the presentation of material in textbooks assumes the validity and worth of research that elaborates the relationships among variables. Statistics courses, beginning at the undergraduate level, further instill the variable-oriented approach, focusing on methods that aggregate individuals (e.g., analysis of variance [ANOVA], regression) and often make questionable assumptions about linearity, data distributions, and random error.

The dominant paradigm finds a way to integrate anomalous findings until the moment when it cannot. Kuhn (1962/2012) noted that over time a crisis is reached that makes a new paradigm more acceptable. At present, psychology is dominated by the variable orientation. There does not yet appear to be a crisis in psychological research that necessitates the abandonment of the variable-oriented approach or that propels the person-oriented approach to greater ascendance. However, at the same time, there is a slow but steady accretion of person-oriented research in various fields of psychology, such as developmental and developmental psychopathology (e.g., Martinez-Torteya, Bogat, von Eye, & Levendosky, 2009), clinical (e.g., Lundh, Saboonchi, & Wangby, 2008), personality (e.g., Laible, Carlo, Panfile, Eye, & Parker, 2010), and industrial organizational (e.g., Foti, Thompson, & Allgood, 2011), and in other disciplines such as education (e.g., Boscardin, Muthén, Francis, & Baker, 2008), criminal justice (e.g., Sampson & Laub, 2005), kinesiology (e.g., Sjoquist et al., 2010), medicine (e.g., Axen et al., 2011), public health (e.g., Lennon, McAllister, Kuang, & Herman, 2005), and sociology (e.g., Kim & Fram, 2009). There is also a burgeoning focus on complementary theory and methods to the person-oriented approach, including idiographic science (e.g., Molenaar, 2004, 2007, 2010).

In Kuhnian terms, the person-oriented approach is an alternative paradigm, because "it regularly raises questions that cannot be resolved by the criteria of normal science" (Kuhn, 1962/2012, p. 109). If a paradigm shift from variable- to person-oriented approaches occurs, or if the person-oriented approach becomes a promising alternative to the variable-oriented approach, it will be because the person-oriented approach affects a change in what are considered legitimate problems to solve as well as what are acceptable solutions to address those problems.

The type versus trait debate in personality is a good example of how difficult it is to affect a paradigm shift. Research on personality has centered for years on a specific question: "Should the field conceptualize personality in terms of the individual attributes that vary across people or the overarching configuration of attributes that defines each person?" (Donnellan & Robins, 2010, p. 1072). The trait approach, consistent with a variable-oriented approach, searches for universals based on the presence of attributes that vary across people. The five-factor model (FFM; McCrae & Alik, 2002) is the most widely used trait approach. The type approach, consistent with the person-oriented approach, focuses on understanding the personality of the individual. There is strong research evidence for three personality types: ego resilients, overcontrollers, and undercontrollers (Robins, John, Caspi, Moffitt, & Stouthamer-Loeber, 1996).

Various rationales have been offered against types and for traits. One is that the three types have not been found in all samples (e.g., Costa, Herbst, McCrae, Samuels, & Ozer, 2002). Even Donnellan and Robins (2010), who found merit in types and defend the person-oriented approach, suggested that the inability to replicate the three types in every sample may be the result of, among other factors, sampling error. Building on this reasoning, Costa et al. (2002) argued that the failure to replicate the three personality types in every sample indicates the superiority of the trait (FFM) approach. Both assertions disregard research that the five factors also have not been replicated in every sample (e.g., Vassend & Skrondal, 2011). These statements reflect how deeply ingrained the variable-oriented approach is in scientific thinking, or, stated another way, to most psychologists, the variable-oriented approach *is* current scientific thinking. When scientists search for universals and cannot consistently find them, they conclude that there is a flaw in their theory or in the specific methods they used (e.g., sampling error). The paradigm—that is, the overarching perspective that guides their specific theories and research methods and underpins these inconsistent findings—is rarely considered.

Using the person-oriented approach as a paradigm, finding or not finding the three personality types in every sample does not mean that these types are wrong, inconclusive, or not useful. It simply means that certain profiles are good descriptors of only certain individuals. Individuals or groups of individuals who do not fit into one of the three types are not exemplars of theory or methods failure, but, rather, may represent individuals or groups that occur at low frequency within certain samples. That is, there is likely to be heterogeneity in samples.

The tension between a variable- and person-oriented approach is also apparent in psychopathology research

and practice. To illustrate, we focus on the issue of heterogeneity vs. homogeneity in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM; American Psychiatric Association, 2013)*. The categorical classification of the *DSM*, based on a medical diagnosis approach, is structured so that mental disorders are defined by the presence or absence of specific symptoms. Advantages to a categorical diagnostic classification system include providing a common language among mental health professionals that improves communication as well as scaffolding that allows researchers and practitioners to determine the reliability and validity of their clinical decisions (e.g., Maughan, 2005).

The *DSM* categories are based on empirical research that finds general patterns of specific behaviors/symptoms across individuals who meet a specific diagnosis. Thus, the categories consist of criteria that, in general, are associated with a particular disorder. The authors of the *DSM* acknowledge that there will be heterogeneity within diagnostic categories. However, the diagnostic categories start with the premise that heterogeneity of symptom features is not the most salient feature in understanding a specific type of disorder.

Ignoring heterogeneity or subgroups within diagnostic categories may have significant implications for both practice and research. For example, in randomized clinical trials of empirically supported treatments, diagnoses are made before clients/participants are enrolled. If the presence of subgroups is not assessed, the results of the clinical trial may be less clearly understood. We know that treatments do not have salutary effects on all participants and that there is a range of drop out in clinical trials. Certainly there are many factors affecting these two outcomes, but individual differences are no doubt one of them. In the 1960s, psychotherapy researchers (e.g., Kiesler, 1966; Strupp & Bergin, 1969) called for methods to elaborate specific therapist behaviors that would help specific clients and produce specific types of behavior change—a charge consistent with a person-oriented approach. Kiesler challenged the notion that all therapists, clients, and treatments were alike, and he labeled the desire of researchers to treat them as such *uniformity myths*.

One possible solution to the problems with heterogeneity identified by Kiesler and others would have been to conceptualize psychotherapy and psychotherapy research from a person-oriented approach. Such a perspective would have, in part, focused on understanding patterns of clients, therapists, treatments, and behaviors that led to successful outcomes. But the variable-oriented approach was and is the dominant paradigm; thus, it framed the concern with heterogeneity identified by Kiesler and others. The effect of the criticism was that many psychotherapy researchers attempted to reduce heterogeneity by creating more homogenous treatments (e.g., manualized treatments) and more homogeneous patient groups exposed to treatment (e.g., *DSM* diagnosable conditions, such as depression). The attempt to create homogeneous patient groups may have been illusory, given the inherent heterogeneity in the diagnostic categories[3]. And, importantly, the creation of homogeneous groups and the subsequent use of variable-oriented methods to analyze data did not answer the basic criticism of the field, which suggested that understanding the effects of specific treatments and therapists on specific patients should be the focus of researchers' efforts. It is easy to see how a person-oriented approach to psychotherapy outcome research would have led the field in different directions, focusing on patterns and profiles of therapist, client, and treatment factors that are related to change.

The purpose of this chapter is to elucidate the person-oriented approach. To do this, we organize the chapter into four sections. In the first section, we describe the tenets and criticisms of the variable- and person-oriented approaches. In the second section, we provide a historical context for the modern formulation of the person-oriented approach by focusing on the work of three early protagonists who advocated this approach at different times during the twentieth century (Stern, Lewin, and Block). In the third section, we discuss three methods of data analysis (structural equation modeling, log-linear modeling, and cluster analysis) to determine whether and how they can be used in the context of person-oriented research. Finally, the last section concludes with a summary and future directions for person-oriented research.

DESCRIPTION OF THE VARIABLE- AND PERSON-ORIENTED APPROACHES

Kuhn (1962/2012) used the term *paradigm* and later *disciplinary matrix* to refer to theory and methods of a normal science. In discussing the person orientation, Sterba and Bauer (2010) use the term *approach*, meaning an overall rubric that encompasses theoretical principles and the methods used to elucidate those principles. Regardless of the nomenclature, the point is that any scientific

[3]There is a movement in psychopathology research away from taxonomies and toward dimensional conceptualizations of mental health disorders. However, the dimensional approaches are variable oriented.

perspective cannot divorce theory and methods, as they are intimately tied to and inform one another. Theory and data analytic techniques from one paradigm can reach conclusions only consonant with that paradigm (Cairns, 1983; Magnusson, 1985, 1995). We will elucidate this essential quality of paradigms as we describe both the variable- and person-oriented approaches.

Variable-Oriented Approach

The dominance of the variable-oriented approach in scientific research makes it difficult to explain or define it—the variable-oriented approach is likely to be considered science itself, and not just a paradigm guiding scientific inquiry (Bergman & Andersson, 2010). We discuss two broad assumptions that characterize the variable-oriented approach.

First, that objects are best understood by focusing on their component parts in isolation is a foundation of the variable-oriented approach (Kelso, 2000). The variable-oriented approach is based on the premise that examining one *aspect* of the whole is thought to reflect the inherent properties of the whole, and that characteristics of the entire entity "have properties irreducible to the individual-level yet nevertheless affecting individual-level phenomena" (Subramanian, Jones, Kaddour, & Krieger, 2009, p. 350). A variable is considered important to the extent that individuals are the same or different on the specific dimension it measures. The focus is on individual variables, not patterns or profiles of variables. Bergman and Andersson (2010) also noted that when statistical analyses of the variables are undertaken, simplifying assumptions often are made about the structure of the variables.

Second, the variable-oriented approach focuses on finding universal laws that describe and predict human behavior. As theory and hypotheses are driven by this assumption, researchers expect to find homogeneity across the samples or populations studied. To the extent that these universals are not found within a specific sample or population, the theory or methods are considered flawed (see earlier discussion of the type vs. trait controversy). When heterogeneity is considered, it is often based on subgroups defined by the researcher that derive from a priori assumptions that those subgroups differ on relevant factors (e.g., males vs. females; schizophrenics vs. nonschizophrenics); these assumptions may or may not be valid.

The focus on universals often means that assumptions underlying widely used statistical analyses are ignored. For example, aggregation of data, which we discuss at length later in this section, is a widely used technique. Because, in the search for universal laws, the variable-oriented approach focuses on the importance of interindividual differences rather than intraindividual differences, aggregation is considered appropriate. "Theories are often formalized in terms of causal relationships between constructs which are regarded as dimensions on which a single individual may vary across time and on which different individuals vary" (Bergman & Andersson, 2010, p. 155).

Related to aggregation is the problem of outliers. Variable-oriented researchers are usually willing to eliminate or replace them. Outliers are, by definition, statistically rare. There are different types of outliers, including distance outliers (those far from the mean), leverage outliers (those that have undue influence vis-à-vis other scores on specific parameters), and inliers (scores close to other scores but which still have a proportionally extreme effect on specific parameters). Statistical methods have been proposed for dealing with outliers, including considering the outlier scores missing and imputing values to replace the deleted scores. The assumption is that outliers represent error rather than true values. This assumption of outliers as error fails to consider that (1) outliers may be members of a different population than the majority of individuals in the sample, and (2) the study of this different population (regardless of how small it is) vis-à-vis the other population may be important (von Eye et al., 2015).

In addition, there is a form of aggregation that occurs in calculating the value of a specific variable. Most variables are scored using a cumulative technique where the more answers an individual gives the more of that variable (e.g., depression) the individual is assumed to have. This is in contrast to other techniques, such as differential scale measurement, where an individual's pattern on the various questions that constitute the variable might be considered; for example, ogive rules, where extreme responses are given more weight than responses that approximate the median (Loevinger & Wessler, 1970). Although not typically used in person-oriented research, approaches such as item response theory (IRT) can be adapted (see von Eye et al., 2015).

In addition, commonly employed statistical analyses often assume that relationships among variables are linear and that "errors are random, follow a normal distribution, and are not related to the true scores" (Bergman & Andersson, 2010, p. 156). And, finally, factors that might affect an individual's score on the key variables are considered nuisance variables that confound understanding of the key variables; thus, they are typically controlled in the analyses. The problems with these assumptions are

discussed in more detail below in the section on limitations of the variable-oriented approach.

Like any scientific paradigm, the variable-oriented approach has legitimate advantages. For example, Kuhn (1962/2012) argued that the areas of inquiry toward which a paradigm directs scientists "leads to a detail of information and to a precision of the observation-theory match that could be achieved in no other way" (p. 64). That is, in the social sciences, variable-oriented theories and methods identify, distinguish, and classify the component parts of the object under investigation (e.g., the child, the family, the school classroom). The variable-oriented approach provides the researcher with objectivity as a result of "clearly defined measurements, the additional information value that comes out of good scales, the usefulness of inferential statistics based on variables, the strength of model testing, and the possibilities for causal inferences that may arise from the study of relationships with proper control for confounders . . . " (Bergman & Andersson, 2010, p. 156). The variable-oriented approach, through its focus on understanding the relationships among precisely defined variables, also can aid identification of mechanisms that occur in the overall process under investigation (Magnusson & Törestad, 1993). Some argue that the variable approach provides the foundation on which science rests—without it we could not move beyond basic descriptive information or idiographic approaches (e.g., Mandara, 2003).

As with any paradigm, there are situations or circumstances about which the variable-oriented approach provides completely accurate data and conclusions. Even Jack Block, a major proponent of the person-oriented approach, whom we shall discuss in detail later, suggested that sometimes the variable-oriented approach was appropriate. Regarding data analysis, the person-oriented approach may not be needed if (1) the variable-oriented approach provides sufficient explanation of the theory or hypothesis tested (e.g., in those cases in which the theory tested and data analysis conducted are focused only on making statements about the aggregate, and not the individual), and (2) the process studied over time is ergodic; that is, the process is stationary and inter- and intraindividual change are identical (Molenaar, 2004).[4] In a Kuhnian sense, it is only through the refinement of the variable-oriented approach that psychologists can ascertain anomalies; that is, we see the novel only when we know what to expect and when the expected does not occur.

[4]However, Molenaar argued that ergodicity can almost never exist in development.

Limitations of the Variable-Oriented Approach

Two overall disadvantages with the variable-oriented approach have been articulated. The first concerns assumptions of variable-oriented data analytic techniques. A major problem in this regard is the logical error of using variable-oriented data to make conclusions about individuals or subgroups. Leo Tolstoy's famous opening line of *Anna Karenina* captures this problem: "Happy families are all alike; every unhappy family is unhappy in its own way." The first phrase is variable-oriented—if you've seen one happy family you've seen them all; whereas the second phrase is person-oriented—unhappy families find myriad ways to make themselves miserable. Perhaps Tolstoy knew very few happy families, and his assessment was that they all had similar traits; for example, perhaps all of these families had a high level of involvement and the children and parents were typically happy. Researchers using the variable-oriented approach might draw the same conclusion as Tolstoy by assessing numerous families, measuring family involvement and happiness, and then examining the covariance between the two variables. Such research tells us something about the aggregate—how the variables involvement and happiness are generally related, but it tells us little about a specific family. Although it would be easy for researchers if all happy families were alike, there is no indication that this is the case. Involvement is likely only one of several variables that are associated with family happiness.

An *ecological fallacy* exists when "aggregated data are analyzed and the results are assumed to apply to relationships at the individual level" (Everitt, 1998, p. 110). Robinson (1950) first demonstrated the ecological fallacy by examining the data from the 1930 U.S. census. Individual correlations indicated a negative correlation ($r = -0.11$) between illiteracy and nativity (i.e., whether one was foreign-born or not), suggesting that the foreign-born were less literate than native born Americans. However, when the same correlation was calculated at the state level, the correlation was $r = .53$, suggesting just the opposite—that foreign-born individuals were more literate than the American born. Another sociologist, in a discussion of proper replication as it related to Durkheim's suicide data and his methods, coined the term *ecological fallacy*, defined as "the invalid transfer of aggregate results to the individual" (Selvin, 1958, p. 613).

In the field of epidemiology, there has been a lively debate disputing Robinson's (1950) admonition that the aggregate may not apply to the individual case. Subramanian et al. (2009) summarized some of the critiques, including searching for instances where there might be

correspondence between aggregate- and individual-level associations, reducing bias in estimates of aggregate models so they more closely approximate the individual, demonstrating that the individual-level relationships among variables can be as error-filled as the aggregate level relationships among those variables, and elucidating "solutions that approximate individual-level relationship based on the information that bounds them at the aggregate level" (p. 343). Finally, the critique extends to the term ecological fallacy itself, which some researchers feel diminishes the importance of the ecological and inappropriately privileges individualism; Susser (1994) indicated the better label might be *aggregative fallacy*.

From a Kuhnian perspective, these attempts to discredit the general assertion—that the aggregate does not always apply to the individual—speak to how strongly the variable-oriented approach is held in the field of epidemiology (and elsewhere) and exemplifies Kuhn's (1962/2012) principle that until a paradigm shift occurs, scientists will continue to fit anomalous data into the dominant paradigm. Regardless of the critiques suggesting that applying the aggregate to the individual may not be problematic, much research since Robinson (who demonstrated the fallacy using bivariate correlations) has indicated that the problem occurs frequently and the same fallacy can exist for any parameter that is estimated at the individual and aggregate level (e.g., Schmitz, 2000; von Eye & Bergman, 2003; von Eye & Bogat, 2006).

von Eye and colleagues (2003, 2015) provide a striking example of the ecological fallacy in the development of alcoholism (Perrine, 1995). The data came from a sample of male adults, collected over the course of 3 years, who provided daily information regarding how much alcohol they had consumed the day prior to the interview. In one article (von Eye & Bergman, 2003), the first four participants in the data set were examined (respondents 3000, 3004, 3005, and 3007). Autocorrelograms, a visual representation of the relationship of each participant's scores with each other as a function of time, were created for each individual as well as an aggregate for the four participants. As the authors noted, the aggregate autocorrelogram indicated that the group had a specific pattern of drinking behavior that consisted of high and positive correlations during weekdays. Unfortunately, this pattern did not represent any of the individual participants' drinking patterns. For example, one participant demonstrated a fairly erratic pattern that was not predictable day-to-day or over time. Another participant's pattern was also erratic, yet it was possible to predict his day-to-day consumption even if his consumption could not be predicted over a longer time period. In a more recent chapter, von Eye and colleagues (von Eye, Bergman, & Hsieh, 2015) demonstrated that these results were not a coincidence of subject selection. They chose the next four participants in the data set, conducted similar analyses, and found similar results; that is, the aggregate autocorrelation plot does not represent any of the individual participants' plots.

Schmitz (2000) developed four theorems, based on methods characteristics, that highlight problems of aggregating data and then applying it to the individual. The first theorem is that individual trajectories cannot be determined from aggregated trajectories. Schmitz's second theorem is that cross sectional and interindividual relationships cannot be used to describe processes within the individual. The third focuses on asynchronous relationships. In longitudinal research, cross-lagged panel analyses may not capture the relationships among the variables that are true of any given individual. Finally, the fourth theorem suggests that the sequence of aggregating and prediction should start with the individual, not the group.

The variable-oriented approach, which generalizes from the aggregate to the individual, assumes that the data are perfect (e.g., aggregate- and individual-level data have the same parameter estimates). However, we do not live in a perfect world, and, because of this, the researcher is faced with two general problems when he or she generalizes from the aggregate to the individual. First, data in each individual case do not always speak to the same effect. Individual- and aggregate-level information can only be equal if the parameter estimates are unbiased; that is, correctly specified. Second, aggregating is often confounded with unmeasured variables. *Lurking variables* are those omitted from the design or data analysis that might lead the researcher to unknowingly make false or misleading conclusions. Lurking variables might also underlie spurious relations among other variables as well as mask heterogeneity in a population or sample (von Eye et al., 2015). There is, as yet, no perfect answer to problems with aggregation, although sampling, design, and data analysis solutions have been proposed (Subramanian et al., 2009; von Eye & Bogat, 2006).

In addition to problems with aggregation, four other methodological problems often occur with the variable-oriented approach (Bergman & Andersson, 2010). First, rarely is it the case that models generated using all subjects in a sample or population describe specific individuals or subgroups. Researchers seldom decompose these models to examine possible subgroup differences. Variable-oriented solutions to the problems of subgroup differences (e.g., adding exogenous variables such as gender to a structural

equation model) are usually not true resolutions. For example, the inclusion of an exogenous variable, such as socioeconomic status, in a structural equation model does not address whether the relationships among the variables are similar or different among subgroups that differ on socioeconomic status.

The second issue is that model fit is often considered a statement about the fit of the model to the data, but this is not always the case. Bergman and Andersson (2010) noted that the accuracy of model fit depends on the veracity of the underlying assumptions. Most models create a data summary matrix based on pairwise linear relationships, which cannot account for the presence of higher order interactions or nonlinear relationships. Models are based on the goodness of the data of which they are composed. This means that, fairly often, the model fit only tells us about "the fit to a flawed summary of the data" (p. 156). Meehl's paradox (1950) demonstrated there are occasions in which no significant main effects or lower order interactions are present, and the highest order effect explains all the variability (Krauth & Lienert, 1973; von Eye, 2002). Thus, omitting higher order interactions in a specific model will sometimes completely misrepresent the data and the underlying relationships among the variables.

Third, Toomela (2009) argued that post–World War II psychological science is built on the assumption that cause–effect relationships are linear; therefore, standard data analytic techniques, which assume linear relationships among variables, are considered sufficient to answer most research questions. Unfortunately, it is unlikely that most human behavior is linear. For example, two variables may have a relationship with each other, but only under certain conditions. Interventions might have a negative effect on a participant early on, but later show a positive effect; for other participants, the positive effect may occur early and then erode and become negative over time. There are numerous examples of how development is typically not linear. For example, Sampson and Laub (2005) discussed turning points; that is, rare events in the life of an individual that lead to a cessation in the trajectory of crime. And Fogel, Garvey, Hsu, & West-Stroming (2006) noted a bridging frame that explains how mother-infant communication followed an inverted U-shaped curve as it moved from a dyadic mode (mother–infant) to a triadic guided mode (mother shows infant toy) to a triadic not-guided mode (mother shows infant toy and infant plays with it) in the first months of life.

It is also a basic assumption in most statistical analyses that errors are random and normally distributed. In addition, random effects are assumed to be independent of model covariates. These assumptions are unlikely to hold and can have serious consequences for understanding data. For example, statistical models make implicit assumptions about the variables we *do not* measure or include—that they are equal across individuals or situations (Ram & Nesselroade, 2007). For example, psychological research on depression assumes that environmental factors are not an important influence on individuals' depressive symptoms. Environmental factors may not be measured or various aspects of the environment (e.g., socioeconomic status) may be controlled, thus ignoring epidemiological research indicating that, in the United States, neighborhood characteristics are associated with depression, independent of individual characteristics (e.g., see Kim, 2008, for a review).

Before closing this section, we discuss two metatheoretical disadvantages of the variable-oriented approach: (1) whether developmentalists are, in fact, concerned with understanding individual behavior, and (2) if they are, what is the best way to accomplish this goal. First, in some fields of research, it may not be necessary or important to understand the individual. One of the points made by Subramanian et al. (2009) is that epidemiologists and sociologists are not always interested in the individual. For example, an individual's income may not be the same (it might be higher or lower) as the average income in the neighborhood or census tract where he or she lives. However, the effect of the overall neighborhood aggregation of SES may play an important role in individual behavior, or it, by itself, may be the focus of interest. However, studies in developmental psychopathology are interested in individual behavior.

The second metatheoretical issue concerns systems theory. If systems theory is, at present, the most accurate way to describe human behavior, then it is not possible to understand an individual by studying aspects of the individual in isolation; we can only understand an individual by studying multiple aspects of the person and his or her environment and how these aspects relate to each other. Magnusson and Törestad (1993) noted that to understand an individual's dynamic psychological functioning, at a minimum, we must understand his or her thoughts and actions as an intentional, active being; his or her biological systems, including genetics; and his or her interface with the social environments as well as how all of these factors affect each other in the present and over time. This relates to Lerner and Busch-Rossnagel's (1981) conception of the individual as someone who generates his or her development. Thus far, the variable-oriented approach has not been especially effective at elucidating humans as dynamic systems.

Person-Oriented Approach

The modern-day person-oriented approach derives from a zeitgeist shift from research on development and developmental psychopathology using an individualistic perspective to research that considers the importance of a holistic, dynamic system in studying the individual (e.g., Bergman & Magnusson, 1997; Cicchetti & Scheider-Rosen, 1986; Magnusson, 1988). Bergman and Magnusson (1997) argued that the current emphasis on holistic-dynamic systems theory derives from three sources: (1) the scientific movement to integrate our understanding of biological processes with behavioral, cognitive, and environmental factors; (2) recent advances in methods focused on nonlinear dynamic systems; and (3) a renewed interest in longitudinal research.

Dynamic systems[5] stand in contrast to static, linear models (e.g., Bergman & Andersson, 2010). "The problem of reconciling phenomena that at the macroscopic level appear ordered, irreversible and determined with phenomena that at the microscopic level appear variable, reversible and stochastic lies at the heart of the DSP [dynamic systems perspective] and self-organization . . . " (Witherington, 2007, p. 134). Dynamic systems theory and models focus attention on understanding change and when and how something new results from that change. Whatever process or variable is studied, at a particular moment, that process or variable resulted from an assemblage of dynamic components that formed, through nonlinear processes, into patterns. Dynamic systems are characterized by moments of equilibrium and moments of disequilibrium; at any given moment, the system can stay stable or become unstable (Kelso, 1995).

Bergman and Magnusson (Bergman & Magnusson, 1991, 1997; Magnusson, 1988) described five tenets of the person-oriented approach. These have been further expanded and elaborated by von Eye and colleagues (e.g., von Eye & Bogat, 2006; von Eye & Bergman, 2003). We start with the original five (Bergman & Magnusson, 1997, all quotes p. 293).

1. "The process is partly specific to the individual." Sterba and Bauer (2010) labeled this the *individual specificity* principle. Although it is tempting to look for universals that describe human behavior, it is unlikely that many universals, at least as related to complex behavior, exist. Even as related to brain function, where we are tempted to state unequivocally that a specific function is controlled by a specific part of the brain, we know that in some individuals, other parts of the brain can take over the same function.

There will not be a universal that fully describes every individual. Even researchers taking a variable-oriented approach recognize this problem. For example, Morey and Hopwood (2009) employed item response theory to differentiate an individual's development along an alcohol dependence continuum to aid understanding of the effectiveness of a treatment protocol. Such efforts attempt to increase predictive power by creating greater homogeneity, when there is known or suspected heterogeneity in the sample or population. But the focus on prediction can interfere with progress in understanding certain kinds of human phenomena such as development, personality, or psychopathology (Magnusson & Törestad, 1993). Prediction can be a goal, a "conceptual and methodological tool" (Magnusson & Törestad, 1993, p. 442), or both. If prediction is a goal, then it takes front and center in our scientific theory and methods. However, if we consider prediction a tool in our research arsenal, sometimes aligned with prediction as a goal, then it can help to elucidate aspects of the phenomena we wish to understand. Magnusson and Törestad (1993) expressed concern that the current focus on prediction, solely as a goal, and its attendant research methods, has led psychology to understand mechanisms, but not processes.

Importantly, individual specificity implies that there are real individual differences that are not the result of errors in measurement or sampling. These individual differences may appear in different samples or within subgroups within one sample. von Eye et al. (2015) noted that outliers (or inliers), typically excluded from variable-oriented approaches, may, in some cases, typify this individuality. A variable-oriented approach, which often uses linear models to determine the relationship among variables across a sample or population, cannot describe how those variables relate in the life of a specific individual or subgroups of individuals (e.g., Magnusson & Törestad, 1993).

2. "The process is complex and is conceptualized as involving many factors that interact at various levels which may be mutually related in a complicated manner." This tenet focuses on the complexity of human development and functioning; Sterba and Bauer (2010) labeled this principle *complex interactions*. Magnusson & Törestad (1993)

[5]There are at least two competing metatheoretical approaches to dynamic systems: contextualism and organicism–contextualism (see Witherington, 2007, for a discussion). It is beyond the scope of this chapter to discuss these. In the discussion of dynamic systems in this chapter, we are referring to the organicism–contextualist metatheory, which best complements the person-oriented approach.

labeled this multidetermination; that is, scientific understanding of the human system will be determined by multiple factors or conditions. It is unlikely that one or two variables and their interactions could describe an individual sufficiently.

The complexity of human psychological functioning has been compared to general systems theory with its emphasis on dynamic, nonlinear, complex processes (e.g., Magnusson & Törestad, 1993). The separation of specific aspects of a human process or the assumption that a linear relationship is present among several aspects cannot capture human behavior at any given time or its course and development over time. Numerous writers have noted the importance of examining the whole person as a system with the underlying assumption that the whole is more than the sum of its parts.

Human processes involve transactions among psychological, biological, and environmental factors. Recent theoretical and empirical work has begun to integrate these. For example, adaptive calibration models suggest that children react to specific environments based on biological responsivity (Del Giudice, Ellis, & Shirtcliff, 2011; Del Giudice, Hinnant, Ellis, & El-Sheikh, 2012; Ellis, Del Giudice, & Shirtcliff, 2013). Vedeler and Garvey (2009) proposed an integration of epigenesis and dynamic systems; in other words, human functioning at all levels (gene to culture) is understood as a dynamically evolving system.

3. "There is a meaningful coherence and structure (a) in individual growth and (b) in differences between individuals' process characteristics. The lawfulness of the processes, within functionally organized structures, is reflected in the development and functioning of all subsystems as well as in the functioning of the organized totality." Sterba and Bauer (2010) labeled this principle *interindividual differences in intraindividual change*. The dynamic, holistic perspective suggests that the organism consists of coherent processes (e.g., biological, psychological). This integration occurs with lower order processes, which then coordinate with higher order processes. However, there may well be individual differences in how various systems are organized and integrated (Magnusson & Törestad, 1993). From a developmental perspective, the organization and integration of these systems may differ, as some may come online earlier or later in particular individuals. These differences in development can have important implications. For example, prior to puberty, genes account for essentially 0% of the variance in disordered eating, but genetic effects become important (>50%) in mid-to-late adolescence (e.g., Klump, Burt, McGue & Iacono, 2007); the genetic effects are quite specific—they only occur midpuberty (Klump, Keel, Sisk, & Burt, 2010) and only for girls (Klump et al., 2012).

4. "Processes occur in a lawful way within structures that are organized and function as patterns of operating factors, where each factor derives its meaning from its relations to the others." This tenet focuses on *interdependence* (Magnusson & Törestad, 1993), and it may be one of the most important tenets of the person-oriented approach. Sterba and Bauer (2010) labeled this principle *pattern summary*; von Eye et al. (2015) suggest a revised label that focuses on the link between theory and methods—*principle of patterns as units of analysis*. This tenet also relates to Sterba and Bauer's (2010) additional sixth principle—*holism* (generated from other writings on the person-oriented approach; Bergman & Magnusson, 1997; von Eye & Bergman, 2003; von Eye & Bogat, 2006) wherein our understanding of any given factor is based on the relationship it has to other factors.

One criticism of the person-oriented approach is that there is nothing inherently distinctive about it. In other words, if person-oriented methods have, at their core, variables, then how is this approach different? The difference lies in the last phrase of the aforementioned principle. Variables are not of interest in and of themselves; they have meaning only insofar as they are one indivisible part of a variable pattern (Bergman & Magnusson, 1997). Because any one variable cannot describe an individual or process—in fact, specific variables can have meaning for one person but not for others—the person-oriented researcher focuses on patterns, profiles, or configurations of these variables as they relate to each other and other profiles. If the profiles or patterns are meaningful, they should provide more information than a variable-oriented approach (see, e.g., Martinez-Torteya et al., 2009, on resilience).

5. "Although there is, theoretically, an infinite variety of differences with regard to process characteristics and observed states at a detailed level, at a more global level there will often be a small number of more frequently observed patterns ('common types'). The assumption applies both intraindividually (viewed over time for the same person) and interindividually (for different individuals at the same time or over time)." This tenet clarifies the concept of *individual* and *individuality*. The person approach holds that no individual is identical to another. However, as von Eye et al. (2010) noted, some differences can be ignored, as they are not important, are not interpretable, or are not significant in all contexts or specific contexts (e.g., a GRE score of 670 vs. 671). Because

measurement error is inherent in any variable or process we measure, small differences are usually meaningless. These inherent properties of measurement mean that there will not be a limitless number of meaningful subgroups in any sample or population. Sterba and Bauer (2010) labeled this principle *pattern parsimony*. Patterns involve some level of coordination and synergy; thus, it will not be necessary to study all the components of an action (or emotion, thought, etc.). For example, Kelso (1995) proposed that dynamic systems are characterized by dissipation and disequilibrium (the analogy is to thermal dynamics). Because of this, he argued that only some factors contribute to a specific behavior. He gives the example that when a baby speaks "ba" there are 36 separate muscles involved; we do not usually need to understand the coordination of this musculature to study phonemes.

In translating this tenet to research methods, consideration of the search for the precise number of subgroups in the data is important. For example, if cluster analysis is to be the statistical method used, Bergman and collaborators (Bergman et al., 2003) recommended having a sample size large enough to generate about 6–10 clusters. von Eye and Bergman (2003) suggest that known effect sizes should be taken into account in making groups, and a statistical base model, based on theoretical considerations, should also provide guidance.

Three assumptions, relevant to this tenet and the search for subgroups, have been advanced (Bogat et al., 2005; von Eye & Bogat, 2006). We explain each of these, and give examples where appropriate. The first is that "a sample is analyzed under the assumption that it was drawn from more than one population" (von Eye & Bogat, 2006, p. 394). There are a few ways the researcher can address this assumption. One is to determine a priori groups (e.g., younger vs. older children, males vs. females). This approach may have merit if the a priori groups are homogeneous relative to the variables under consideration. Theory often guides the generation of hypotheses that predict a priori group differences; however, often the research on which those hypotheses is based is variable-oriented, and the prior researchers may not have searched for heterogeneity within the groups. If a priori groups are used in data analysis, the researcher should establish that the groups are homogeneous by, for example, determining that there are not outliers in the groups, validating groups on variables in the space of variables not used to make the groupings, and ensuring that dimensional identity (see Tenet 6) can be demonstrated. In other words, *typical* subgroups (e.g., age, gender) may not be homogeneous groups; these variables may only be one component of a pattern that defines a more consistent subgroup.

Another way to form groups is to decompose data using techniques such as cluster analysis, latent class analysis, or other methods of finite mixture decomposition. One advantage of these approaches is that they can generate groups of different sizes, including groups that may include only a few individuals; from a person-oriented perspective, this flexibility is important (von Eye, Bogat, & Rhodes, 2006). An example of this type of group formation was undertaken by Bogat et al. (2012). The relationship between stress and HPA axis dysregulation is multiply determined, affected by characteristics of the stress itself as well as the psychological consequences of the stress (see Miller et al., 2007, for a review). Research to date has typically examined one or two aspects of the stress or the psychological response to it and, predictably, findings are mixed as to whether these factors are associated with dysregulation at all or hyper- or hypo-corticolism.

Bogat et al. (2012) tested whether individuals have heterogeneous responses to stress and whether profiles of women who differed on specific variables might better elucidate the relationship between stress and HPA axis dysregulation. Data from a cross sectional study of mothers (n = 182) experiencing (or not experiencing) intimate partner violence were analyzed. Initial attempts to fit the psychological, cortisol, and intimate partner violence data using structural equation modeling were unsuccessful. Thus, latent profile analysis was conducted using scores on pregnancy intimate partner violence, postpartum intimate partner violence, depression, anxiety, PTSD symptoms, the cortisol awakening response slope (awakening to 30 minutes postawakening), and the morning to evening cortisol slope (30 minutes postawakening to 5 p.m.). Models with one to six latent profiles were estimated using Mplus6. Fit indices (AIC and BIC) suggested that the model with five latent profiles, each of a different size, was the best fitting model (Figure 18.1).

Latent Profile 1 (n = 51) included women with significant levels of depression and anxiety, moderate levels of IPV during pregnancy, and lack of the expected cortisol awakening response. Women in Latent Profile 2 (n = 85) were characterized by relatively healthy adaptation, with low levels of IPV during pregnancy and postpartum, the lowest levels of depressive, anxiety, and PTSD symptoms, and the expected pattern of diurnal cortisol secretion. Latent Profile 3 (n = 11) consisted of women with high levels of IPV during pregnancy and postpartum with moderate levels of depression, anxiety, and PTSD, as well as dampened cortisol levels in the morning with a flattened diurnal. Latent Profile 4 (n = 3) consisted of women with the most severe violence scores at both pregnancy and

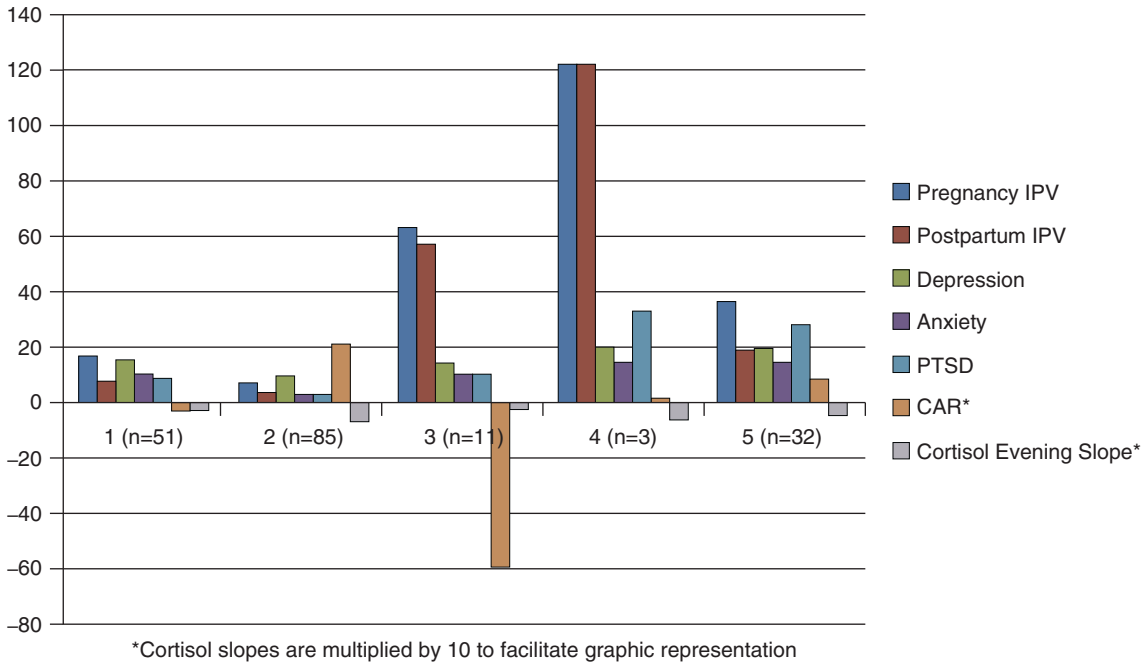


Figure 18.1 Five biopsychological profiles of women experiencing intimate partner violence. See footnote 1.

postpartum, high levels of psychopathology, no cortisol awakening response; and a normal evening slope. Latent Profile 5 (n = 32) included women with moderate levels of IPV at pregnancy and postpartum, the highest levels of psychopathology but who showed a cortisol diurnal pattern similar to that of the healthy group.

A third way to form groups is to ascertain patterns of function or development that characterize certain individuals (e.g., changers vs. nonchangers). An example of this type of grouping was research that identified trajectories of early reading difficulties among children studied from kindergarten to Grade 2 (Boscardin, Muthén, Francis, & Baker, 2008). Growth modeling with mixture components (e.g., Muthén, 2000, 2001; Muthén & Muthén, 2000), using 12 data points, was used to create the groups. This method was chosen, in part, because it allowed for the possibility of heterogeneous subgroups within the model. The number of classes for phonological awareness in kindergarten and word recognition development in Grades 1 and 2 were modeled separately. When these models were combined, the authors allowed for the possibility that the children might stay in the same class or change class membership over time. The final model was composed of 10 classes of students with different trajectories related to reading prerequisite skills and later deficits in reading development.

Finally, groups can be formed by asking "whether groups of cases exist that deviate systematically from predictions that can be made based on parameters that had been estimated under the assumption that only one population exists" (von Eye & Bogat, 2006, p. 394). An example of forming groups in this manner was research conducted by Bogat, Levendosky, DeJonghe, Davidson, and von Eye (2004). The participants (n = 163) were part of a larger, prospective longitudinal study examining risk and resilience among women and children experiencing intimate partner violence. This particular research examined the temporal effects of intimate partner violence on women's mental health using women's data from three time points: the last trimester of pregnancy (Time 1), the child's first birthday (Time 2), and the child's second birthday (Time 3). The authors asked whether there were groups of women that could be formed based on temporal experiences of intimate partner violence that would differ from the sample as a whole.

The women were grouped into eight patterns of intimate partner violence based on their reports at each of the three time periods (e.g., 000 = no violence at any time; 110 = violence at time 1 and time 2). An 8 (patterns) × 3 (observation points) mixed effect repeated measures ANOVA was performed for each of the dependent measures (self-esteem, anxiety, depression, and posttraumatic stress symptoms), decomposing them into linear and quadratic orthogonal polynomials; this allowed for the examination of different mental health trajectories. Here,

we focus on whether there were group differences in the time × pattern interactions. There were for self-esteem and depression but not for anxiety and posttraumatic stress disorder symptoms. For example, regarding self-esteem, across all participants, self-esteem increased during Time 1 and Time 2 and then tapered off at Time 3. There were, however, group differences regarding the quadratic polynomials. The no violence group (i.e., 000) showed a steady, linear increase; the chronic violence group (i.e., 111) showed an increase followed by a decline; and the group with only Time 2 violence (i.e., 010) showed high levels of self-esteem, which then tapered off. For depression, across all participants, symptoms declined over time. However, there was a significant group × quadratic effect for three groups (chronic violence, 111; Time 2 violence only, 010; Time 3 violence only, 001), indicating an increase in depressive symptoms at Time 3. Thus, examining mental health trajectories over the course of more than two years indicated that the chronicity and timing of intimate partner violence mattered; that is, in this sample, there were subgroup differences based on intimate partner violence experiences.

The second assumption relevant to this tenet is that "attempts are made to establish external validity of the groupings." If methods such as those suggested above are used (e.g., cluster analysis, latent class or profile analysis), a relatively small number of groups are generated with group-specific profiles. External validity can be established by comparing these groups on variables not used to constitute the group profiles. Different techniques for group comparisons are available. For example, in latent profile analysis, covariates can be included within the model to determine uncertainty within profiles (Roeder, Lynch, & Nagin, 1999). Another approach, to validate latent classes, involves rerunning the final latent class model by adding additional variables, on which groups are expected to differ. One can then conduct difference tests to compare models in which those additional variables are allowed to vary across the classes with models that assume the additional variables do not vary (e.g., see Ansara & Hindin, 2010).

Finally, the third assumption is that "the groups are interpreted based on theory." It is not difficult to find groupings of individuals in a data set, but whether these groups are distinct in important ways only can be ascertained relative to theory. Bergman, Magnusson, and El-Khouri (2003) indicated the importance of either a priori meaning of the groups or the ability to interpret the groups based on extant theory.

A recent paper, examining profiles of behavioral and adrenocortical functioning among 7-month-olds, illustrates these three assumptions concerning groups and the person orientation (Towe-Goodman et al., 2012). The authors employed latent profile analysis to generate profiles of infants, covariates were used to test the validity of the profiles, and theory was used to interpret the results. We briefly describe the rationale for the hypotheses, the findings regarding the profiles, and the interpretation of one aspect of the findings.

Understanding relationships among the biological and behavioral indicators of coping with stress is important as problems in these domains in early childhood are associated with vulnerability or resilience to stress across the life span (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007; Gunnar & Quevedo, 2007). However, little is known about the coordination of these systems in infancy, and research has failed to find consistent relationships between these systems (e.g., Keenan, Grace, & Gunthorpe, 2003; Ramsay & Lewis, 2003). The lack of reliable findings suggested to Towe-Goodman et al. that there might be patterns or configurations of adrenocorticol and behavioral stress responses among infants exposed to interparental conflict. This was the case; a four-class solution provided the best fit to the data. Compared with the reference group ("low reactors"), children who were in the "high cortisol reactivity, moderate negative behavior" group were more likely to have higher negative affect and to live in households where conflict occurred as well as less likely to demonstrate successful self-regulatory behaviors under stress. The findings were interpreted, in part, based on the emotional security hypothesis (Davies & Forman, 2002). Children might maintain emotional security in households with high levels of parent conflict by minimizing overt displays of distress, even when they are experiencing high physiological arousal. This behavioral suppression may result from learning that it is dangerous to draw attention to themselves, as this might lead the parents to aggress toward them.

These are the basic tenets of the person orientation. Other important additions to these tenets have been suggested; we discuss two of them.

6. *Dimensional identity*. There have been developing statements about the meaning of dimensional identity. The expression originated with Schmidt (1977), who postulated that scales used for comparison be commensurable. Von Eye and Bergman (2003) suggested that scales used for inter- or intraindividual comparisons should also have the same dimensional structure and be invariant over time. If dimensional identity is not given, scales cannot be used for comparison. The result that dimensional identity does not exist is of interest in itself. It points to the possibility

that multiple populations exist. In addition, this result indicates where these populations possibly differ from each other.

7. *The ecological context of human behavior*. Three additions to the original person-oriented tenets, relating to the ecological context, were developed by Bogat (2009), using the basic structure and language of the original tenets put forward by Bergman and Magnusson (1997). *1. The structure and dynamics of individual behavior are, at least in part, specific to the environment in which the individual lives and works. 2. There is lawfulness and structure to (a) intrasystemic constancy and change and (b) intersystemic differences in constancy and change. These processes can be described by patterns of the involved factors. 3. Validity is specific to individuals and environments*.

In discussing the individual as a holistic, dynamic being, Bergman and Magnusson (1997) emphasized that an individual's behavior can be influenced by factors from the neuronal to the cultural. The setting in which an individual works, lives, or loves is as much a factor in his or her development as cognitive, biological, and psychological processes. The effects of context can be dramatic. For example, marriage dampens criminal behavior, an effect that seems to be contextual rather than based on an individual personality characteristic (Horney, Osgood, & Marshall, 1995; Sampson, Laub, & Wimer, 2006).

Bronfenbrenner and Morris (1983) defined context as "any event or condition outside the organism that is presumed to influence, or be influenced by, the person's development" (p. 359). Within this broad definition, Bronfenbrenner (1977) elaborated four nested levels of the environment: (1) the *microsystem* (social settings in which the individual is embedded, such as the family); (2) the *mesosystem* (the connections between the various microsystems, such as the family's relationship with the church); (3) the *exosystem* (larger structures that subsume the micro and mesosystems, such as neighborhoods); and (4) the overarching *macrosystem* (i.e., culture and its various components, such as political systems). Later formulations included a fifth level—the *chronosystem*—which described how interactions within or among the four other levels, were influenced by time (Bronfenbrenner, 1986a, 1986b).

This five-level, broad contextual rubric helps focus the researcher's attention on the wide array of contexts that can affect an individual; however, *which* specific contexts should be measured or included in a specific research study are less obvious. Bronfenbrenner's contextual levels focus on the social environment; however, others have argued that the physical environment might also play an important role in child development (e.g., Widaman, 2007).

Unfortunately, of all the components of the person-oriented approach, the contextual component is probably the least developed and understood. In the limitations section, we explain why this might be the case.

Limitations of the Person-Oriented Approach

Here we discuss three limitations to the person-oriented approach: (1) generating testable hypotheses; (2) conducting pattern analyses; and (3) measuring and analyzing the contexts in which human behavior occurs.

The first limitation is that the person-oriented approach does not lead easily to testable hypotheses. Bergman and colleagues (Bergman & Anderson, 2010; Bergman & Trost, 2006) argued that, in part, hypothesis generation is difficult because the person-oriented approach "is quite general and its translation into specific theory for a scientific problem can be difficult" (p. 158). On this point, we take a slightly different perspective. If the person-oriented and variable-oriented approaches are considered general paradigms for scientific inquiry, then, in and of themselves, they do not lead to specific testable hypotheses. Rather, they provide the structure under which theories and hypotheses are generated.

However, even so, hypotheses from a person-oriented approach may be difficult to generate because there has not been enough attention to the match between theory and methods. Sterba and Bauer (2010) rightly object to the casual use of the term person-oriented any time individuals are grouped. They emphasize the importance of understanding what person-oriented methods can be used to test what principles of the person orientation. For example, they propose a matrix in which the person-oriented principles are crossed with four person-oriented methods (i.e., less restrictive variable-oriented approaches, classification, hybrid classification, and single subject). They conclude that the correspondence between principle and method may sometimes be a match (e.g., pattern summary × single subject), sometimes a mismatch (e.g., pattern summary cannot be tested with less restrictive variable-oriented methods), or sometimes a limited or conditional match (e.g., individual specificity is only conditionally testable with hybrid classification models). Of course, as statistical techniques evolve, these correspondences may change [e.g., in the same special issue in which Sterba & Bauer's article appeared, Molenaar (2010) argued that all person-oriented principles can be tested with dynamic factor analysis].

Generating testable hypotheses using a person-oriented approach may also be difficult because most extant research

is variable-oriented. As Bergman and Andersson (2010) note, it is often difficult to use variable-oriented findings to develop person-oriented theory and methods. However, sometimes when an accrual of variable-oriented research on a specific topic generates contradictory findings in different samples or when different variables are included in the analyses, a reconceptualization of the research using person-oriented theory and methods may be beneficial and advance the science (e.g., the infant and adult cortisol research, cited earlier).

The second limitation involves how to conduct data analysis of patterns (Bergman & Andersson, 2010). Person-oriented theory focuses on the importance of understanding patterns; however, the statistical techniques for analyzing patterns are underdeveloped. For example, Bergman and Andersson (2010) noted that many current data analytic techniques to determine patterns depend on dissimilarity matrices (e.g., cluster analysis), yet there is not widespread agreement on what the appropriate measure of dissimilarity should be. Configural frequency analysis is a particularly promising technique because it creates all possible patterns within the variable space; the researcher can then identify which patterns occur more or less frequently than expected under a specific hypothesis.

In addition to understanding patterns, in and of themselves, there is the additional problem of modeling dynamic processes in those patterns that occur over time. Kelso (1995) noted that the principles of dynamic pattern formulation confront us with two problems. The first is *the problem of complexity of substance*—how a pattern is constructed from a large number of components. The second is the *problem of pattern complexity*—how patterns accommodate complexity in whatever function they ultimately serve (e.g., by changing, by selection, by bifurcation). In the field of developmental psychopathology (as in most fields), we are far from understanding how patterns form and how they operate. However, we can assume that the relationship of the components of patterns as well as the relationship of patterns with other patterns is most often nonlinear. Thus, new data analytic techniques will be necessary to capture these processes.

Dynamic systems modeling approaches, focused on recursive differential and difference equations, show some promise, and there has been, of late, much interest in this strategy (e.g., Kunnen, 2012; Valsiner, Molenaar, Lyra, & Chaudhary, 2009). Other approaches, for example, state-space nonstationary time series modeling (e.g., Molenaar, Sinclair, Rovine, Ram, & Corneal, 2009), which puts no constraints on the parameter estimates, has been used in idiographic approaches.

Analyzing patterns involves decisions about which variables are essential to constitute the patterns, since we cannot measure everything in every research study. In discussing patterning of contextual influence, Widaman (2007) suggested three contextual patterns to consider. Big Bang events, which are likely to be one-time, unrepeatable, stressful life events (e.g., death of a parent), are known to have a strong influence on development, but only in the context of other factors (e.g., age of child, socioeconomic status). Repetitious, smaller events, as the name implies, occur frequently, over the course of time. Big Bang events correspond to major life stressors; repetitious smaller events correspond to daily hassles. Finally, there are intermittent but salient events (e.g., inconsistent parenting behavior).

The third limitation concerns context. What is context and how should it be measured? What contexts are most relevant to assess in the field of developmental psychopathology? These are not simple questions, and it is beyond the purview of this chapter to answer them fully. However, here we address several problems with the conceptualization and measurement of context.

One problem faced by the researcher is how to define context in terms of how it differs from as well as might be dependent on individual-level data. For example, Oakes (2009), cited the work of Coleman (1990), who put forward a simple model of how social change occurs: society is composed of individuals and individuals constitute society. That is, "Institutions and other social phenomena play a key role in analyses, but these phenomena must be grounded to the activity of individuals.... Group-level phenomena are never simple aggregations but rather complex dynamic and multilevel phenomena" (Oakes, 2009, p. 364).

There are conceptual as well as statistical problems associated with determining and measuring context. Sometimes context is "defined purely on the aggregate" as in whether or not there is a Head Start program in a specific community; sometimes context is a summary statistic of an aggregate of individuals (e.g., prevalence or incidence data, neighborhood socioeconomic status; Greenland, 2001).

Greenland (2001) offered an example from the field of epidemiology regarding how aggregate measures can introduce problems of cross-level confounding when measuring the effect of context. Table 18.1 (from Greenland, 2001) presents data for two areas, *A* and *B* (which differ on a contextual variable, such as a law or social program), and their influence on the rate of a health outcome (e.g., smoking). The rates in Panel 1 indicate no contextual effect; in essence

TABLE 18.1 An Example Demonstrating the Complete Confounding of Contextual and Individual Effects in Ecologic Data

	Area A			Area B		
	$X = 1$	$X = 0$	Total	$X = 1$	$X = 0$	Total
1. Ecologic (marginal) data						
Y = 1	?	?	560	?	?	560
N	60	40	100	40	60	100
Rate	?	?	5.6	?	?	5.6
2. Possibility 1 ($RRx = 2$, $RR_A = 7/8$)						
Y = 1	420	140	560	320	240	560
N	60	40	100	40	60	100
Rate	7.0	3.5	5.6	8.0	4.0	5.6
3. Possibility 2 ($RR_X = 1/2$, $RR_A = 8/7$)						
Y=1	240	320	560	140	420	560
N	60	40	100	40	60	100
Rate	4.0	8.0	5.6	3.5	7.0	5.6

Note: The ecologic data cannot identify the effect of group (A versus B) on the rate of the outcome $Y = 1$ when only a marginal summary of the individual-level covariate X is available. N = denominator (in thousands of person-years); RR_A and RR_X are the rate ratios for the true effects of A versus B and of $X = 1$ versus $X = 0$, respectively.
Source: Greenland, S. (2001). Ecologic versus individual-level sources of bias in ecologic estimates of contextual health effects. *International Journal of Epidemiology, 30*, 1343–1350. Reprinted with permission from Oxford University Press.

$RR_A = 1$ (the true effects of A vs. B). Because the presence of a risk factor (X) is unknown, it cannot affect RR_A. Panels 2 and 3 show circumstances under which the same result as Panel 1 can be obtained, even though risk and context are different. For example, in Panel 2, area A has a positive contextual difference ($RR_A = 7/8$; that is <1), but that is obscured by a higher prevalence of a harmful risk factor ($RR_X = 2$; that is >1). Panel 3 demonstrates how this confounding can take place when the situation is reversed; that is, when the contextual difference is negative ($RR_A = 8/7$) and there is a higher prevalence of a beneficial factor ($RR_X = 1/2$).

Greenland argued that in some cases, the differences in risk factors between areas A and B might be negligible. However, he noted that in other cases, the differences might be substantial, for example, in the case of alcohol consumption. At moderate levels alcohol consumption may be protective, whereas at high levels it may be detrimental. Thus, without knowing more about the distribution of moderate and heavy drinkers within an area, the direction or the strength of confounding of RR_X on RR_A would be difficult to determine.

Another problem is that the meaning of context may vary over time. We noted earlier that marriage itself is associated with decreased criminal behavior. However, Gottfredson (2005) suggested that marriage is an age-dependent variable, and the meaning of marriage will differ accordingly. "At very young ages, marriage may index low self-control . . . , during the early to late thirties higher levels [of self-control] . . . And repeated marriage may signify low rather than high self-control" (p. 51). This presents a very specific problem of dimensional identity, which may have strong effects on the data, and, thus, the conclusions that are drawn from them.

Although developmental psychology and developmental psychopathology researchers study contextual factors (e.g., parenting, SES, prenatal programming), the focus is usually on a specific variable; rarely is the focus on the associations among multiple factors and levels in which individuals exist. For example, of the articles published in psychology from 2000–2005, at least one-third that had a significant focus on race, did not have a significant focus on socioeconomic status (Carlson, 2006, as cited in Cauce, 2008). Culture, race, and ethnicity are important contextual factors, yet, at present, we understand little about how they transact with individuals.

The field of developmental psychopathology often focuses on identifying early risk factors that predict later problem behaviors. Sampson and Laub (2005) argued that sometimes the search for childhood risk factors may be misguided, especially when the focus is on "early childhood attributes that are presumed to be stable over the life course" (p. 40). Their research, examining criminal behavior among males from age 7 to 70 (Laub & Sampson, 2003), found that risk factors generally did not predict future behavior. When the sample was divided into

high- and low-risk groups (based on early detrimental family and individual factors), both groups had identical trajectories of criminal behavior—criminal behavior peaked at age 15 and then desisted over time to negligible levels. Decomposing the sample another way, a five-group solution was found; each group had a slightly different trajectory of peak and drop off of criminal activity. However, similar to the two-group solution, criminal activity declined for all groups over time. In addition, the authors found no evidence that two childhood risk factors (parental deviance and mental health) differentiated the five groups in meaningful ways.

One solution to the focus on risk perspective is for person-oriented research to examine positive development. For example, Richard Lerner's (e.g., Lerner, Taylor, & von Eye, 2002; Lerner, von Eye, Lerner, & Lewin-Bizan, 2009) positive youth development model focuses on strengths, including those of the adolescent (school engagement, self-regulation, and hope about the future) and the environment (social networks, individuals, institutions, and resources). Healthy development (as exemplified by competence, confidence, character, caring, and connection) occurs as a result of positive synergy between youth and environmental assets.

This section has elucidated the principles of both the variable- and person-oriented approaches as well as outlined limitations of each. In the following section, we discuss the genesis of many of the tenets of the person-oriented approach by focusing on the theories and research of three seminal psychologists.

THREE EARLY PROTAGONISTS OF THE PERSON-ORIENTED APPROACH

In this section, we discuss the work of William Stern, Kurt Lewin, and Jack Block, three key personality theorists and researchers, whose ideas presaged the modern-day person-oriented approach. In examining their work, it is evident that the field of personality has debated the merits of some variant of the person-oriented vs. the variable-oriented approach, since its inception (e.g., holistic vs. individualistic; idiographic vs. nomothetic; traits vs. types). We recognize that there are other important figures (e.g., Allport, Binet, Brunswik, Cairns); however, the scope of this chapter does not allow us to cover the entire history of the field.

The three protagonists worked during the ascendance of the variable-oriented approach. Various historical explanations have been offered as to why the variable-oriented approach came to dominate psychological science. One idea is that a consensus was reached in psychology regarding which statistical methods were scientific. For example, Stam (2010) focused on the Galtonian view in psychology, where "numbers . . . *represent* some empirical reality" (p. 147, italics in the original). He argued that this focus moved psychologists away from people and toward characteristics of tests, as aggregation occurred hand-in-hand with this representation. Lamiell (2009) discussed attempts to integrate experimental and correlational methods, which resulted in neo-Galtonian notions that "the statistical concepts and methods common to both forms of inquiry—involving the analysis of means, variances, and covariances (i.e., correlations)—made the merger of these two research programs both possible and eminently sensible" (p. 36). Another idea, advanced by Toomela (2009), suggests that causality, understanding, and explanation, after World War II, focused almost exclusively on linear models. In doing so, psychology adopted simple, mechanistic models and rejected more complicated models for understanding human behavior (see also Stam, 2010). Magnusson (2000) focused on the move to a stimulus-response paradigm in the 1950s and 1960s, which focused on molar behaviors rather than a holistic, integrated formulation of human behavior.

As we mentioned earlier, Kuhn (1962/2012) argued that paradigms stay dominant, in part, because researchers are able to integrate anomalous findings into these paradigms. This certainly occurred with the person- and variable-oriented approaches. Lamiell (2003) provided an example in his discussion of Allport and McClelland's perspectives on the findings of Hartshorne, May, and Shuttleworth (the Character Education Inquiry; Hartshorne & May, 1928, 1929; Hartshorne, May, & Shuttleworth, 1930). In this classic research, numerous components of specific child traits (e.g., helpfulness, dishonesty) were measured across settings; however, the correlations among these components were negligible, leading Hartshorne and May (1928) to conclude that these were not traits, but simply habits. Allport (1937), attempting to integrate nomothetic and idiographic approaches, suggested that the findings proved "only that children are not consistent *in the same way*, not that they are inconsistent with *themselves*" (p. 250). In other words, there might be unmeasured traits influencing the results (an early example of the lurking variable argument), and/or that individual children might have unique motives for the same behavior (e.g., one child might steal because of a *bravado* trait, another because of a *social inferiority* trait). Alternatively, as Lamiell (2003) pointed out, McClelland (1951), defending the nomothetic tradition, argued that the research had methodological

flaws; the children were too young to demonstrate traits (which were learned from past experiences), a significant methodological problem.[6]

The three historical figures we discuss here were immersed in the debates about the prominence the individual should have in psychological science. They all came down on the side of the individual.

William Stern

William Stern (1871–1938) is well known in the field of psychology for specific contributions (e.g., developing the intelligence quotient), yet he is almost unknown as relates to his philosophical writings on critical personalism. In fact, Lamiell (e.g., 2009) suggested that Allport might have been one of the few psychologists who valued Stern's theoretical contributions.[7] His lack of renown is unfortunate, given that Stern's ideas foreshadow important principles of the person-oriented approach.

Stern began his career by developing the field of differential psychology; that is, the study of individual differences. to study individual differences, differential psychologists developed tests and compared individuals and/or groups on these tests. For example, as is well known, in the late nineteenth and early twentieth century, Binet and Cattell were developing intelligence tests and administering them to different individuals and populations. Stern's book on differential psychology first appeared in 1900, with a revision in 1911. He was interested in specifying differences between individuals and groups, ascertaining what caused these differences, and how the variations were demonstrated; however, although the book dealt with both inter- and intraindividual differences, Stern's emphasis was interindividual differences (Lamiell, 2010a). Stern became concerned with the testing movement (especially America's obsession with intelligence testing and its reliance on norms determined through aggregation), and ultimately repudiated his own contributions to the field (Lamiell, 2010a). In the process, Stern embraced his interests in metaphysics and philosophy, articulating his theory of critical personalism—"a genuine differential psychology" (Stam, 2010, p. 149).

For Stern (1917/2009), the individual was a unitas multiplex; that is, multiple components that are organized in a coherent, and purely teleological, manner. The idea of the individual as unitas multiplex augurs Bergman and Magnusson's (1997) second and third tenets concerning the complexity of process and the meaningful coherence in development and functioning. Stern was concerned that psychology had focused primarily on elucidating the multiple components of human experience at the expense of understanding the "unified individual." Stern noted that simply aggregating all the components of the individual did not, in and of itself, explain higher-order processes.

The unity of the individual was to be understood on four levels: "phenomena (mental experiences), acts (doings), dispositions (strivings, capabilities), and subjects (I)" (Stern, 1917/2009, p. 115, n.b. all quotes below from this source). Although heuristically, one could look at each in isolation, none could be understood fully without an appreciation of how their interrelationships created a totality. By *phenomena*, Stern meant individual self-perceptions, such as ideas or feelings. *Acts* involved the interconnections and unifications of self-perceptions that occurred within a short time frame or within the moment. However, such a temporal framework, if taken in isolation, provides a misleading picture of the individual. Stern extended the time frame by considering *dispositions*, enduring qualities or attributes of the individual that predispose him/her to various perceptions. The relationship between these three components is as follows: Acts mediate the relationship between dispositions and perceptions. For example, Stern noted that intelligence "is not the power to set into course thoughts or thought sequences of a particular kind, but instead the disposition to adjust oneself through acts of thought to new situations and demands" (p. 122).

The fourth component was the I—the unification of the dispositions. Stern defined the I as existence itself. "The I experiences phenomena, executes acts, [and] owns dispositions" (p. 124). The I included both the psychological and physical aspects of the person.

From these four components, Stern defined personality as "an entity that we encounter as a living whole which, that in and of itself strives for and is capable of realizing the enduring purposes of that whole" (p. 131). This definition puts front and center Stern's focus on the teleology of the personality system. The personality was both self- and other-oriented (*autotelie* and *heterotelie*, respectively), and worked to integrate these two purposes, even though they were sometimes in opposition. Stern referred to this as *introception*. In addition, personality was not fixed by the four components, they simply potentiated the individual's transactions with the environment. Here Stern's writings

[6]The reader may be interested in a more recent exemplification of fitting anomalous data into the dominant paradigm of personality research (Borkenau & Ostendorf, 1998).

[7]Over the last 30 years, James Lamiell has, nearly single-handedly, brought Stern's ideas to an American audience.

relate to the ecological context tenet of the person-oriented approach. The transaction of the person and the environment, he termed *convergence*. In Stern's view, an individual's goal focus was insufficient; it had to be augmented by the environment. The environment would influence the individual, but the individual's specific dispositions would influence his/her experience with the environment.

It is apparent that Stern's theories relate closely to the tenets of the person-oriented approach. In addition, his delineation of research methods (separating, for instance, research on the individual and research on groups of individuals), also echoes person-oriented approaches. As Lamiell (2010b) noted, Stern believed in large-scale research and the use of modern statistical methods; at the same time, he understood that there was a distinction between knowledge about an individual and knowledge collected across a group regarding specific attributes that individuals might have. This concern presaged the dilemma of the ecological fallacy, discussed earlier, and specifically relates to Jack Block's writings decades later that the search for universal traits is problematic because they say little about the individual.

Kurt Lewin

Lewin (1890–1947) and Stern knew of each other's work (Stam, 2010). Although their theories diverged in important ways, they shared a concern regarding psychology's misguided focus on aggregation and the search for universals and lawfulness.

Lewin (1931) believed that psychology was tethered, in problematic ways, to Aristotelian logic. He was concerned that psychology focused on regularity, specifically in the sense of frequency. Thus, psychologists were primarily concerned with lawfulness (regularities across individuals) and unconcerned with the individual case. In fact, "the individual event seems to him [the psychologist] fortuitous, unimportant, scientifically indifferent" even though it may have "critically determined the destiny of the person involved" (p. 151). Lewin argued that the focus on lawfulness prevented psychology from investigating large swaths of human experience (e.g., will and affect), because these types of experiences were likely to occur less frequently than other experiences. As is obvious, Lewin, like Stern, was concerned that psychologists discounted the importance of the individual, the first tenet of the person-oriented approach.

According to Lewin, in their quest to discover universal laws of human behavior, psychologists established groupings of convenience, leading to incomplete, mechanistic understandings. For example, he wrote:

> Whatever is common to children of a given age is set up as the fundamental character of that age. The fact that three-year-old children are quite often negative is considered evidence that negativism is inherent in the nature of three-year-olds, and the concept of a negativistic age or stage is then regarded as an explanation (though perhaps not a complete one) for the appearance of negativism in a given particular case. (p. 153)

Again, these sentiments reflect tenets of the person-oriented approach, including avoiding the ecological fallacy, the importance of creating meaningful groups, and a focus on understanding process rather than simply mechanisms.

Lewin also felt that psychology's concentration on classes and frequency of behavior led to the use of statistics that obscured individual differences. Although psychology was preoccupied with developing fundamental principles, in practice, Lewin believed, research described averages across individuals. "It is forgotten that there is just no such thing as an 'average situation' any more than an average child" (p. 172). Lewin's critique of the use of averages in statistical methods focused on the lack of precision that was inherent in this approach. He wrote: "The demands of psychology upon the *stringency* of its propositions go no farther than to require a validity 'in general,' or 'on the average,' or 'as a rule.' . . . *psychology does not regard exceptions as counter-arguments so long as their frequency is not too great*" (p. 156, italics in the original). Here again, Lewin's ideas foreshadow the importance of avoiding the ecological fallacy and the need to focus on individual behavior.

Lewin (1931) anticipated that psychology would move from an Aristotelian to a Galilean concept formation; he based this idea on Ernst Cassirer's analysis of t he fields of mathematics and physics. In part, a Galilean formulation would lead psychologists to develop theories that provided complete, not partial, explanations for the phenomena under study. Central to these theories was the notion that the causes of human behavior were multiply determined, and did not rest exclusively within the individual. He noted that numerous factors might affect an individual, including his or her personality, the groups within which an individual was embedded, culture, and economic forces. Lewin (1939) was greatly influenced by the Gestalt psychologists; however, he felt that the whole and its individual parts were of equal importance. "The whole is not 'more' than the sum of its parts, but it has different properties. The statement should be: 'The whole is different from the sum of its parts'" (p. 885).

Lewin's theory of multicausality ultimately evolved into his "field theory," which proposed that human behavior resulted from the interactions among many different

systems; that is, a dynamic field. Lewin's (1936) famous formula, expressed in mathematical terms, stated that behavior is a function of the person and the environment: $B = F\ (P,\ E)$. Lewin argued that the focus for psychological research was on the life space, which consisted of the needs of the individual as well as the environment in which he or she existed [$B = F\ (P,\ E) = F(L\ Sp)$] (Lewin, 1939). The focus on multicausality and the inclusion of the environment, as well as the person, in theory and method, are preludes to the second and third tenets of the person-oriented approach—that processes involve many interacting factors, and that within these interactions there is coherence and lawfulness. Lewin's work also reflects the fourth tenet with its focus on the importance of understanding each factor only in relation to other factors.

Lewin (1931) was also an experimentalist, who believed in creating ideal or paradigmatic situations rather than simply recreating conditions that occurred frequently.

> The general validity, for example, of the law of movement on an inclined plane is not established by taking the average of as many cases as possible of real stones actually rolling down hills, and then considering this average as the most probable case. It is based rather upon the 'frictionless' rolling of an "ideal" sphere down an 'absolutely straight' and hard plane, that is, upon a process that even the laboratory can only approximate, and which is most extremely improbable in daily life. (p. 161)

For example, Lewin, Lippitt, and White (1939) studied aggressive behavior by organizing clubs of 10-year old boys that were guided by three types of leaders (i.e., laissez-faire, authoritarian, and democratic). Process data were collected, including a written verbatim account of all conversations, visual recordings, and moment-by-moment structural, quantitative, and interpretative accounts of group process. The authors found that most of the acts of aggression occurred during emotion-laden moments (e.g., criticism of the group from an outsider). These acts of aggression seemed to be spontaneous acts resulting from a build-up of tensions based on leadership style and the restriction in "space of free movement" that resulted from that style. Other factors considered important were the "rigidity of the group structure," which provides a restraining force but also increases tension within the individual, and whether the child came from a culture where aggression was an accepted way of acting.

This is necessarily a very brief review of Lewin's theory. We do not discuss the aspects of his theory that are, at present, most well known, including action research, group process, and leadership styles. What is most interesting to us is that the criticisms of psychology that Lewin raised, which are similar to those raised by advocates of the person-oriented approach, are largely forgotten. We now turn our attention to Jack Block.

Jack Block

Block (1924–2010) is often rightly cited as one of the fathers of the modern person-oriented approach. He is perhaps most widely known today as a contrarian (a label he gave himself) who offered strong critiques of the Five-Factor Model (FFM) of personality (e.g., Block, 1995, 2001, 2010), as it came to dominate the theory and research of personality. Although researchers have acknowledged the validity of some of his critiques of the FFM (e.g., McAdams & Walden, 2010), the overarching concern in which the specific critiques are subsumed—that variables, rather than people, had become the focus of personality research—is often overlooked or downplayed.

Block began his career at a time when the debate about how personality was to be defined (whether there were universal truths about personality as well as which statistical techniques provided the best way to understand personality) was quite active. In 1946, Cattell defined the *R-technique* as an approach to studying personality that focused on correlating variables; hence, a variable-oriented approach to analyzing data. Alternatively, the *Q-technique* correlated persons. Some researchers (e.g., Eysenck, 1954) felt that the two approaches were the same; that is, they always led to similar results. Along with others (e.g., Cronbach, 1953; Stephenson, 1953), Block (1955) disputed this assertion, arguing that depending on the heterogeneity or homogeneity of the sample, the results would be different. In other words, in a heterogeneous sample, composed of more than one subgroup, it was possible that the covariance structures for specific variables would differ by subgroup. In homogeneous samples, the covariance structures would be the same.

Writings throughout his career reflected Block's concern that personality psychologists focused too much attention on variables and not enough on individuals. His strongest defense and advocacy of the person orientation can be found in *Lives Through Time*, Block's (1971) seminal investigation of personality development. Many of the principles of the person orientation that he elaborated can be seen in the following quote from this book, when he wrote that psychologists should aspire to understand personality in the way that laypeople do, which is "[a] configuration and systematic connection of personality variables as these dynamically operate within a particular

person" (Block, 1971, p. 13),[8] a point made by some current personality researchers, as well (e.g., Donnellan & Robins, 2010).

There are a number of important components to this statement. First, laypeople naturally discuss people, not variables; whereas the opposite tends to be true of many psychologists. Block acknowledged that this problem was not unique to the study of personality. For example, he cited an article wherein the authors ask "where is the perceiver in perceptual theory?" The second important aspect of this statement is that personality consists of a configuration of characteristics. One characteristic cannot describe someone's personality adequately. A person who is quiet in large social situations may be perceived as shy; however, when that same person is seen talking freely and intimately among his close friends, the observer may need to adjust his or her perception.

Finally, the third component of Block's statement speaks to the importance of configurations of personality variables operating in a dynamic fashion. He argued that personality theory put too much emphasis on examining stability (i.e., uniformity of development across people and time) at the expense of overlooking instances of change and what drives such change. For example, when personality psychologists find differences in individuals across time, they are likely to ascribe these to measurement error in the assessment instrument (in particular when the instrument was constructed based on classical test theory), random error, or the choice of an unimportant (i.e., nonuniversal) variable. Block disputed this bias by demonstrating in his longitudinal research that there were groups of individuals whom he labeled changers and nonchangers; importantly, these subgroups were better able to explain personality development compared to a priori groupings based on such variables as gender.

Block recognized that there was strong pressure among personality researchers to adhere to the uniformity myth.[9] Many researchers had argued that if uniformity across people did not exist, the result would be a lack of lawfulness; that is, the discovery of too many different types of personality would undermine the search for universal principles or types. Block argued against this perspective by decoupling lawfulness and universality. He wrote, when the two are conflated it means that "the seeking of one may preclude finding the second . . . [in fact, in not doing so] a greater lawfulness may be discerned as what is general and undifferentiated is partitioned into smaller but more homogeneous classes for study" (1971, p. 11). In fact, Block believed smaller groups were likely of great interest to the researcher, and, furthermore felt that random samples might not include certain types of infrequently occurring individuals. He felt so strongly about this that his recommendation for future longitudinal researchers was to *not* recruit a random sample, but rather to focus theory and research questions on a relevant, non-random, sample.

As mentioned earlier, Block felt that theory and research were synergistic. In other words, it was inevitable that researchers holding variable-oriented theories would find support for these theories, as they employed variable-oriented assessments and data analytic techniques that could not find support for person-oriented theories. This was a point he made continually throughout his life. For example, in his critique of the FFM, he eschewed the notion that five personality factors derived for adults could be readily transposed onto childhood personality (Block, 2010). In other words, he implied that researchers who believed in stability rather than change and who focused on variables, rather than people, might conclude (falsely) that adult and child personality traits were interchangeable. Thus, early in his career, to examine the person in personality, Block turned to a new assessment tool just gaining popularity.

Q-Technique

Although often attributed to Block, the *Q-technique* (also called *Q-set* or *Q-sort*) was not invented by him. Stephenson (1935) introduced this technique in an article aptly titled "Correlating Persons Instead of Tests." Stephenson was concerned with a specific problem within psychology—that large numbers of participants were needed to collect the data that would allow psychologists to correlate tests or estimates and then conduct factor analyses, especially as related to Spearman's *g*, *c*, and *w* factors.

He went on to argue that laboratory experiments become almost "burdensome" because of the need to collect data from so many individuals. He proposed the *Q-technique* as an antidote to this problem: "Can we make factor studies on a *few* individuals, and bring the

[8]Block was not arguing that laypeople should provide scientific definitions of personality for the researcher. In fact, one of his ongoing critiques of the FFM was that the list of adjectives used to define the traits had been derived from undergraduate students asked to list traits that best described themselves and their acquaintances.

[9]The term uniformity myth, as discussed earlier, was also used by psychotherapy outcome researchers to suggest the folly in assuming that clients, therapists, and behavior were similar and that individual differences did not matter (e.g., Kiesler, 1966).

methods of correlations and factor analysis into the laboratory?" (p. 18, italics in original; see also Nesselroade & Molenaar, 2010).

His solution was to invert the typical process. Instead of obtaining a few tests on a large number of people, he proposed that psychologists obtain many tests on a few people. Stephenson's demonstration of his new technique was a study on taste (i.e., aesthetics), what he considered an important, but understudied, facet of an individual's personality. He took 60 color samples (no blacks, pure grays, light creams or whites were included) and gave them to 12 women and eight men with the instruction to grade each quickly and spontaneously from best to least liked. His small experiment found two factors; that is, two groups of individuals whose preferences correlated positively with each other. Factor 1 was composed of 12 individuals who preferred the subtle colors (which Stephenson labeled as the most tasteful); Factor 2 was composed of eight individuals who preferred the pure bright colors (which Stephenson labeled as vivid but crude).

Interestingly, although Stephenson's procedure had the participants sort the cards (self-report), Block's initial Q-set was developed so that professionals could make appraisals of others (other-report). Also, although Stephenson developed this technique as a special type of factor analysis, the method became used as an assessment tool unrelated to factor analysis (e.g., Rogers, 1961).

Block's Q-Set

Block's (1961) original purpose in developing his *California Q-set* was to define more precisely the characteristics of personality widely used by professionals (e.g., authoritarian personality) as an aid to future research, including better communication and precision among scientists. Thus, as stated already, the Q-set was given to professionals to evaluate patients or research participants. Although it had earlier permutations, the Q-set Block presented in his 1961 monograph consisted of 100 statements, each of which described a specific personality trait (e.g., "is an interesting, arresting person; seeks reassurance from others; emphasizes being with others; gregarious"). Although the statements do not represent conceptualizations from any specific theoretical orientation, Block felt they reflected current informed opinion among professionals in personality psychology, clinical psychology, and psychiatry.

Block conceived of the standard statements as a language and the sorting algorithm of the traits as a grammar. Each observer is given the 100 statements on separate cards; the task is to sort the statements into nine categories where one represents the category least representative of the person and nine represents the category that is most representative. The number of statements that the observer places in each category differ and range from 5 to 18.

Block saw the Q-set as offering both *ipsative* and *normative* data. That is, the information provided a distinct profile of characteristics specifically about the individual being assessed; however, across all the participant data, Block was able to find representative types. The normative data were particularly interesting over time; for example, certain of the 100 characteristics in the Q-set were rarely if ever used to rate individuals at specific stages of development.

As noted earlier, Block was essentially concerned with issues of change and stability over time, as he felt that such knowledge would lead us to understand the developmental paths that lead to the same outcome (equifinality) or similar developmental paths that lead to different outcomes (multifinality). To study trends, Block developed profiles of personality variables based on their correspondence, salience level, and salience heterogeneity over time. This resulted in seven categories. For example, the category "sameness" described personality variables where the correspondence across time was high, and changes in the salience level and salience heterogeneity were minimal. The category "decreasing salience values, order maintained" described personality variables that correlated well over time but whose salience, as a group, declined over time. Using these categories of personality variables, Block examined continuity and change in specific groups (e.g., men, adolescents). The findings using this approach were deemed uninteresting by Block, " . . . the plethora of slight to moderate across-time relationships supports only the by-now banal recognition that human beings do indeed remain somewhat true to themselves over considerable lengths of time" (p. 88). Block understood that there was an inherent flaw in summarizing information about variables across diverse people; he recognized that such an approach would mask subgroups.

For example, in taking a different approach (i.e., factor analyzing the individual profiles), Block found that there were some subjects who did not fit the majority types he found. He labeled these individuals *residuals* and noted that they had one of two characteristics. The first were individuals who did not fit any of the types; the second were individuals who fit well within multiple types. It should be noted that in 1971, Block was not entirely convinced that factor analysis was the best data analytic strategy to find types or residuals. In fact, he wished to find natural groupings, but did not have the computational power to use techniques that could find them. In later years, some of his most pointed criticisms of the FFM were based on

researcher's reliance on factor analysis (e.g., Block, 1995, 2001, 2010), including the assumption that mathematically forced orthogonality accurately represented psychological understanding (i.e., the five factors did not operate independently in an individual).

It is obvious that Block's theory of personality and research touched on each of the person-oriented tenets described earlier. One of the important components of the modern person-oriented approach is that context matters. Unfortunately, the Q-set was deficient in measuring context; a problem of which Block was aware. The Q-set questions tried to take into account individual behavior in diverse settings, but the questions did not specifically focus on this dimension of human behavior. When an individual's behavior varied, Block often attributed this to context. In his longitudinal research, Block did not have the assessment tools or methods to explore the context of an individual's behavior, so he simply averaged the various discrepancies.

In summary, Block was considered a contrarian, and he focused many of his critical essays in his later years, and, even the year that he died, on arguing against the FFM, which had come to dominate personality research. He offered many specific criticisms; however, these were all embedded in his most fundamental belief, the person-orientation. He promoted a focus on the person, rather than variables, with specific emphasis on examining profiles; he encouraged psychologists to develop intraindividual theories rather than theories of interindividual differences; and he promoted the notion of the individual as a dynamic system. Block (1995) wrote:

> Once the parameters that define the personality system of a generic individual have been conceptually posited and empirically identified, these parameters become the essential, nonarbitrary, overarching variables or concepts for a personality of interindividual differences. The differences between individuals would then be understandable as due to the different specific values these parameters take in different individual personality systems. It would be a sweet intellectual accomplishment if the theoretical constructs required for dynamically understanding within-individual functioning could also be used for understanding the differences between individuals. Personality psychologists might aspire to this goal. In that effort, our science—in its theoretical reach and empirical grasp—may better realize its deep aspirations: to provide a bio-social theory of intraindividual development, the moment-to-moment psychodynamic functioning within the individual, the life themes coherently reflected in the adaptive changes of individuals over time and context, *and the* personality differences among individuals. (p. 210)

STATISTICAL APPROACHES

Methods of Person-Oriented Data Analysis

In this section, we discuss and exemplify application of methods of person-oriented data analysis. With these aims in mind, we adopt the role of the data analyst. This poor soul is often handed a data set along with two requests. First, test the hypotheses that had been specified when the grant proposal was written. Second, let the data speak a little as well, and perform exploratory analyses, just to make sure anything interesting was not overlooked. In the current context, there may be a third request, which is that analyses be performed from a person-oriented perspective. For the third request to be realistic, data have to be collected so that person-oriented analyses can be performed. von Eye and Bogat (2007; von Eye, 2010) specified a set of conditions for person-oriented data collection and analysis. Two conditions are most important. First, the sample should be large enough for person-oriented analysis to become possible. When data are collected with the goal of performing person-oriented data analysis, the number of cases must correspond to the anticipated number of groups or populations the data are assumed to be collected from. Each of the subpopulations must be large enough for the analysis to be possible in each of the subpopulations, and for comparative analyses to be possible as well. This desiderate applies both when subpopulations are known prior to data collection and when the researchers just assume that more than one population might exist. In the first case, power analysis can be used to determine the required size of samples from each population. In the second case, the number of populations may be unknown, rendering the a priori determination of sample sizes complicated. The second condition for person-oriented data analysis is that dimensional identity prevails (see the earlier section on the tenets of person-oriented research). Only if variables, test instruments, or dimensions have the same meaning and characteristics in each of the subpopulations, comparative statements with these variables, instruments, or dimensions are possible.

Person-oriented research implies that a specific set of methods of data analysis is employed; however, it also implies that theory, data collection, and data analysis complement each other. This is reflected in a statement by Sterba and Bauer (2010), according to which

> person-oriented approach = person-oriented theory + person-oriented methods

The remainder of this section is structured as follows. We discuss three popular methods of analysis and ask whether and how they can be used in the context of person-oriented research. The methods are structural equation modeling, log-linear modeling, and cluster analysis. Configural frequency analysis (Lienert, & Krauth, 1975; von Eye, 2002; von Eye, & Gutierrez-Pena, 2004), among the chief methods used in person-oriented research, is presented in a separate chapter in this volume (von Eye & Mun, 2015). For each method, we highlight the elements of person-oriented research that are represented, and we give an empirical data example. We posit that each of these methods requires unique assumptions and allows researchers to analyze data in respects of importance for person-oriented research.

Structural Equation Modeling

Structural equation modeling (SEM; Bollen, 1989; Jöreskog & Sörbom, 2001; Kline, 2011; McArdle, 2012; Muthén & Muthén, 1998–2012) allows one to combine methods of factor analysis with methods of regression analysis. In SEM, factor analysis is enriched by providing the option of classifying variables into dependent and independent, and regression analysis is enriched by allowing latent variables to be regressed onto each other. It is also possible to perform exploratory or confirmatory factor analysis without distinguishing between dependent and independent variables, and to estimate regression or path models at the level of manifest variables. In Jöreskog and Sörbom's (2001) LISREL notation, a structural model can be described as

$$\eta = -\mathrm{B}\eta + \Gamma\xi + \zeta$$

where

- η is a [m, 1] random vector of latent variables (factors) on the dependent variable side (the y-side of the model);
- $-\mathrm{B}$ is an [m, m] matrix of regression coefficients that describe the structural relations among the η factors; the diagonal elements of β are zero; β can be symmetric or asymmetric;
- ξ is a [n, 1] random vector of latent variables (factors) on the independent variable side (the x-side of the model);
- Γ is an [m, n] matrix of regression coefficients that describe the structural relations among factors on the x- and the y-sides of the model (factors from the y-side are regressed onto factors on the x-side of the model); and
- ζ is a [m, 1] vector of residuals; this vector represents imperfections of the model in the sense that not all of the variance of the η factors can be explained by the model; these are random disturbances, also called equation errors.

The measurement models for the manifest variables on the x- and the y-sides of the model describe the factorial structure of the dependent and the independent variables. The models are

$$y = \Lambda y\eta + \varepsilon,$$

and

$$x = \Lambda x\xi + \delta,$$

where

- x is a [p, 1] vector of observed (manifest) variables on the x-side of the model, with p indicating the number of manifest x-variables;
- y is a [q, 1] vector of observed variables on the y-side of the model, with q indicating the number of y-variables;
- $\Lambda\, x$ is a [q, n] matrix of the loadings of the x-variables on their latent variables, ξ;
- $\Lambda\, y$ is a [p, m] matrix of the loadings of the y-variables on their latent variables, η; and
- ξ and δ are the residuals of the manifest y- and x-variables, respectively; ε and δ are convolutions of measurement errors and model imperfections.

Later, in this section, we present an example of a structural model in which we only use manifest variables. One characteristic of the structural model that is important for the present discussion of methods of data analysis from a person-oriented perspective is that the input matrix for the estimation of the model is a variance-covariance matrix, that is, a matrix with the variances of the observed variables in its diagonal and all covariances in the off-diagonal cells. Here, it is not important that this matrix can be substituted, under certain conditions, by a matrix of, for example, correlations or tetrachoric coefficients. Important is that, by definition, information about individuals disappears when variances and covariances are calculated. The individual is no longer visible. There are, however, two interesting exceptions. The first involves estimating models at the level of individuals (see, e.g., Nesselroade & Molenaar, 1998; Molenaar & Newell, 2010). The second involves identifying groups by using, for example, mixture distribution models (Muthén & Muthén, 1998–2012) or defining groups, and then estimating multigroup models. In this chapter, we focus on *multigroup models*.

Either of these approaches to modeling development is of great importance for person-oriented research. They

allow characteristics of individuals or groups of individuals to be modeled. This way, justice is done to unique characteristics of individuals or groups of individuals instead of averaging differences out and estimating models that describe the average individual, which, according to Walls and Schafer (2006), may not exist (see also the earlier section on Kurt Lewin).

Multigroup models allow researchers to specify hypotheses concerning the parameters in which they do versus do not allow the comparison groups to differ. Specifically, let G groups be given, then, the model for group g involves the eight parameter matrices $\Lambda_y^g, \Lambda_x^g, B^g, \Phi^g, \Psi^g, \Theta_\varepsilon^g, \Theta_\delta^g$, where the superscript indexes the groups. Now, to give an example of the hypotheses that can be specified, suppose that, in a two-group comparison, the structural parameters in the B and the Γ matrices are supposed to be equal. This can be expressed as $B^1 = B^2$ and $\Gamma^1 = \Gamma^2$. Under these hypotheses, the parameters in all other matrices can be different. In LISREL, the following options for parameter constraints and estimation are available. Matrices in the comparison groups can be constrained

- to have the same patterns of fixed and freed elements,
- to have the same patterns and starting values in the iteration process, and
- to have invariant elements, that is, estimates are constrained to be numerically exactly the same for the comparison groups.

It is interesting to note that the number of variables does not have to be equal in the comparison groups. In addition, residual variances and covariances can be estimated for individual groups only. Hierarchies of constraints have been proposed, but here, we will not use any of the hierarchical procedures.

Data Example

The following example uses data that were collected for a study on the development of aggression in adolescence. Finkelstein, von Eye, and Preece (1994) administered a questionnaire to 75 11-year-old girls and 39 same-age boys who indicated the degree to which they perceived in themselves aggressive impulses (AI), were verbally aggressive against adults (VAAA), and physically aggressive against peers (PAAP). The questionnaire was administered again when the adolescents were 13 and 15 years of age. We hypothesize that, at age 13, aggressive impulses are explanatory of physical aggression against peers and that this process is mediated by verbal aggression against adults. To test this hypothesis, we first estimate a path model without the direct path from AI85 to PAAP85, that is, the direct effects model

$$PAAP85 = \alpha_1 + \beta_{21} VAAA85 + \zeta_1$$

and

$$VAAA85 = \alpha_2 + \gamma_{11} AI85 + \zeta_2$$

If needed, we also estimate the model that does include the direct path

$$PAAP85 = \alpha_3 + \gamma_{21} AI85 + \zeta_3$$

The first model is, in a first run, estimated for the entire group of respondents. The model – data fit for this model is excellent. We obtain $\chi^2 = 0.68$ ($df = 1; p = 0.41$), RMSEA = 0.00, CFI = 1.00, and GFI = 1.00 and decide to retain the model. Because of this excellent fit, the model with the direct path does not need to be estimated.

In a first person-oriented step of analyzing the relations among aggressive impulses, verbal aggression against adults, and physical aggression against peers, we ask whether any of the questionnaire variables can be predicted from gender. In other words, we ask whether responses are gender-specific. To answer this question, we include gender in the model. For the following model, we place gender on the x-side of the model, and AI85, VAAA85, and PAAP85 on the y-side. We predict PAAP85 from VAAA85, which, in turn is predicted from AI85. Each of these variables is predicted from gender. The model – data fit for this model is excellent as well. We obtain $\chi^2 = 0.56$ ($df = 2; p = 0.75$), RMSEA = 0.00, CFI = 1.00, and GFI = 1.00 and decide to retain the model. Figure 18.2 displays this model (standardized scores given).

In the model in Figure 18.2, the path hypotheses are confirmed. AI85 predicts VAAA85, which predicts PAAP85. In contrast, only the PAAP responses are gender-specific ($t = 4.89; p < 0.01$). The paths from Gender to AI85 and to VAAA85 remain nonsignificant ($t = 0.07$, n.s.; $t = 1.64$, n.s.). From this result, we conclude that the two gender groups may differ in their responses to the aggression questionnaire, and we specify a two-group model. We adopt a descending strategy where the strictest model is estimated first. In this model, we set all parameters equal for the two groups. Specifically, we set $\theta_\delta^1 = \theta_\delta^2, B^1 = B^2, \Gamma^1 = \Gamma^2$, and $\Theta_\varepsilon^1 = \Theta_\varepsilon^2$. Not unexpectedly, this model fails to describe the data well. Based on the overall goodness-of-fit $\chi^2 = 22.58$ ($df = 7; p = 0.002$), RMSEA = 0.20, CFI = 0.63, and GFI = 0.89, none of

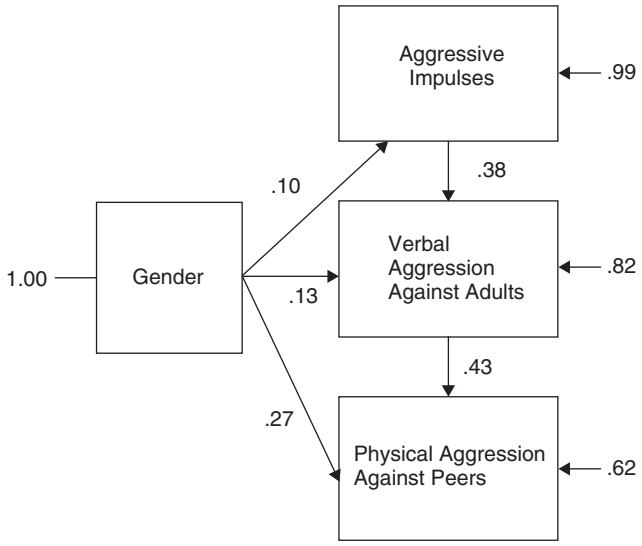


Figure 18.2 Path model of aggressive impulses, verbal aggression against adults and physical aggression against peers. All predicted from gender. *Source*: Data from 1985.

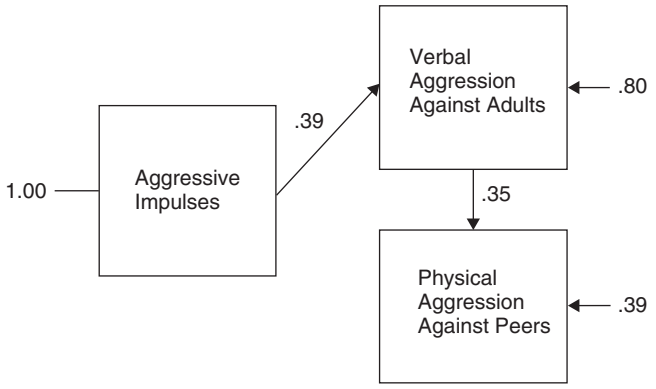


Figure 18.3 Two-group model of aggressive impulses, verbal aggression against adults, and physical aggression against peers; standardized solution for female respondents given. *Source*: Data from 1985.

which is in support of the model, we reject it. We conclude already at this point that male and female respondents differ in their answers to Finkelstein et al.'s (1994) aggression questionnaire.

We now relax the restrictions placed in the first model. Instead of navigating through one of the hierarchies of imposing or relaxing restrictions, we consider that Gender differences seem to exist in the responses concerning physical aggression against peers (PAAP85; Figure 18.2). In the present model, the parameters related to PAAP85 are located in matrix B (parameter β_{21}) and in matrix Ψ (parameter ψ_{33}). We relax the parameter constraints, and now specify that the parameters in the matrices B and Ψ exhibit the same pattern for the two comparison groups, but they are allowed to differ from each other in magnitude. The resulting model describes the data very well. Specifically, we obtain the overall goodness-of-fit $\chi^2 = 1.22$ ($df = 4; p = 0.87$), RMSEA = 0.0, CFI = 1.00, and GFI = 1.00. Each of these statistics is in support of the model, and we decide to retain it. This model is given in Figure 18.3 (standardized solution given for female respondents).

Several elements of this model reflect group differences. First, we note that the model describes the male responses slightly better than the female responses. The female submodel contributes 79.02% to the overall χ^2, and the male submodel contributes only 20.98%. These differences are tiny, but they are interpreted as pointing in this direction. More important are possible differences between the parameters that were relaxed for this solution. In Figure 18.3, we see that, for the female respondents, the parameter for the path from AI85 to VAAA85 is estimated to be 0.39. For male respondents, this parameter was fixed to be equal. In contrast, the parameter from VAAA85 to PAAP85 is 0.35 in the female group and 0.64 in the male group. The difference between these two estimates is significant when only the β parameters are compared and the ψ parameters are assumed to be invariant across the comparison groups ($\Delta\chi^2 = 18.85; \Delta df = 2; p < 0.01$). We conclude that female and male adolescent respondents differ in the path from VAAA85 to PAAP85 in the sense that this path, although significant in both comparison groups, is stronger in males than in females.

Discussion

Not surprisingly, structural modeling of manifest variable path models allows one to identify the parameters in which comparison groups differ. In this respect, structural modeling is well capable to test hypotheses that are compatible with a person-oriented research perspective. SEM offers many options, including, for example, hierarchical modeling, modeling data of individuals, growth curve modeling in multigroup designs, or means modeling. Still, it is hard to label SEM a standard method for person-oriented research. We, therefore, have to identify the elements that make SEM suitable for such research. We discuss three options.

First, as is well known, the input matrix for standard SEM is a covariance matrix. Therefore, the individual does not play a role or, even worse, the individual is not visible. However, most SEM programs are able to make individuals visible, in some form. For example, to the best of our knowledge, every SEM program is capable of creating and saving factor scores. These scores will, in person-oriented

research, not necessarily be used to estimate factor score regression models (for options, see Hoshino & Bentler, 2011). However, the individual's relative position in a factor space can be ascertained. This way, outliers can be made out and factor scores from different factors can be related to each other, with the options of identifying outliers, or specifying individual profiles. In other words, every SEM program allows one to identify those individuals for which a factor solution is less than perfect in the sense that the individual becomes an outlier. From a person-oriented perspective, these are the individuals for which a model may not apply. (Note: When factor scores are used, the covariance matrix that is used as input information must contain all relevant information. That is, one must assume that higher order interactions do not exist or explain only negligible portions of the targeted variability, that is, the variability that allows one to distinguish between individuals.)

The second option, and this was illustrated in the data example on aggression development, is that SEM is a very powerful methodology that can be used to distinguish among subpopulations that are known a priori. Differences in most any parameter can be ascertained.

The third option involves SEM mixture modeling (Arminger & Stein, 1997; Dolan & van der Maas, 1997). Two approaches to SEM mixture modeling have been proposed (Vermunt & Magidson, 2004). The first involves specifying an SEM model and then modeling within-class covariance matrices under the assumption that there are multivariate normal mixtures. This approach is also known as latent class clustering or model-based clustering (Banfield & Raftery, 1993; Mun, von Eye, Bates, & Vaschillo, 2008). In the context of SEM mixture modeling, the class-specific mean and covariances are restricted to the specified SEM structure. This structure reflects, for instance, factors or growth curves. The second approach involves incorporating latent classes into a structural model, thus capturing unobserved heterogeneity. Here, the unobserved variable is class membership. SEM mixture models can be estimated by some SEM software packages (e.g., Mplus; Muthén & Muthén, 1998/2012) and is of importance in particular when researchers assume that data were collected from more than one population, but number and size of populations are unknown.

These three options show that SEM is suitable for person-oriented research—when certain assumptions apply (Sterba & Bauer, 2010; von Eye, 2010). Most important, in the present context, is the assumption of *dimensional identity* that was previously discussed, in the context of the tenets of person-oriented research (von Eye & Bergman, 2003). Dimensional identity implies that the variables that are used to describe individuals are the same in all parameters in the comparison groups. That is, they have the same meaning, the same factor and covariance structures, and they are commensurable, both cross sectionally and longitudinally. If this assumption holds, individuals and groups of individuals can be compared using these variables. If, however, this assumption must be rejected and there is, for example, *differential item functioning*, that is, item characteristics fail to be invariant across comparison groups, scores on the scales that include such items are no longer comparable. Not all is lost in such a case. The information that there is differential item functioning or that factorial structures vary across comparison groups may be of interest itself. However, group means cannot be compared, and neither can scale scores. Before testing multigroup structural models or estimating SEM mixture models, researchers, therefore, are well advised to make sure that dimensional identity prevails.

There exists, however, an interesting exception. There are variables that have natural meaning. Examples of such variables are physiological measures, voxel counts for brain activity, response time, number of items recalled, and many other measures that are not based on instruments constructed using psychometric tools. When this kind of variable is employed, differences in means or covariance structures are most important for the person-oriented researcher because they are no longer indicative of flaws in instruments but of possibly meaningful differences between groups or individuals.

Log-Linear Modeling

Types of Log-Linear Models

Log-linear models are special cases of generalized linear models in which categorical variables are analyzed (Agresti, 2012; von Eye & Mun, 2013). The generalized linear model (GLM) relates a vector, η, to one or more explanatory variables by way of a linear model, $\eta = X\beta$, where X contains the predictors or the effects posited by a model, and β is a parameter vector. In a linear regression model, β contains the regression weights. Now, let η be related to the expected value of the linear model by $\eta = g(\mu)$, where μ is the expected value, and g(.) is a monotone function. This function is called the *link function*. For cases of the general linear model (e.g., analysis of variance or regression), the link function is the identity function. That is, $\eta = \mu$. In log-linear modeling, one obtains

$$\log \mu = X\beta$$

and for the general linear model, one obtains

$$\mu = X\beta$$

These equations illustrate that the general linear model and the log-linear model have the same form (see Agresti, 2012; McCullagh & Nelder, 1989; von Eye & Mun, 2013). Differences are evident in the link function and in the nature of the expected values. In regression and in analysis of variance, expected values are estimated for an observed variable, the outcome or dependent variable, typically in the units of this variable. In log-linear modeling, expected values typically are estimated for counts. By implication, main effects in regression or analysis of variance involve two variables, the independent and the dependent variables, and in log-linear modeling, main effects concern the distribution of the categories of just one variable. This applies accordingly to interactions.

Three ways of specifying log-linear models have been discussed. The first involves *hierarchical log-linear models*, the second involves *nonhierarchical models*, and the third involves *nonstandard models* (Mair & von Eye, 2007; von Eye & Mun, 2013). Log-linear models are hierarchical if

- all lower order relatives of terms included in a model are also part of the model;
- the model contains only terms that can be expressed within the hierarchy of effects; and
- no constraints are specified for parameters (except zero-sum constraints; see von Eye & Mun, 2013).

Log-linear models are nonhierarchical if

- they contain only a selection of the possible lower order relatives of the terms in the model; and
- if the second and the third condition for hierarchical models are also fulfilled.

Log-linear models are nonstandard if

- they contain terms that cannot be specified in the hierarchy of models; or
- if they include covariates, parameter constraints, or special effects that cannot be specified in the hierarchy of models.

In the following sections, we first present examples of log-linear models, and we review parameter interpretation. This is followed by a data example and a discussion of log-linear modeling in the context of person-oriented research. For the examples, consider the four categorical variables, A, B, C, and D. A first model that can be considered for these variables (and is not the null model of no effects) is that of variable independence. This model includes all main effects but proposes that there are no interactions. The model is

$$\log m = \lambda + \lambda^A + \lambda^B + \lambda^C + \lambda^D$$

where m is the vector of model frequencies, and the superscripts indicate the variables involved in the main effects. A second model could propose that A is associated with B and C is associated with D. This model is

$$\log m = \lambda + \lambda^A + \lambda^B + \lambda^C + \lambda^D + \lambda^{AB} + \lambda^{CD}$$

where the double superscripts indicate the associations (interactions) included in the model.

When, for instance, the main effect of any variable is removed from this model, for example, when, after removal of the main effect of A

$$\log m = \lambda + \lambda^B + \lambda^C + \lambda^D + \lambda^{AB} + \lambda^{CD}$$

researchers consider a nonhierarchical model. This model is nonhierarchical because the main effect of variable A that was removed is a lower order relative of the interaction between A and B.

An example of a nonstandard model is

$$\log m = \lambda + \lambda^A + \lambda^B + \lambda^C + \lambda^D + \lambda^{AB} + \lambda^{CD} + \lambda^{Covariate}$$

where the covariate is a term that cannot be expressed in terms of elements of a hierarchical model. Examples of covariates are continuous variables whose cell-specific averages are used as special effects.

Parameter Interpretation

The interpretation of log-linear model parameters is based on three arguments (von Eye & Mun, 2013). The first is statistical significance. To test the null hypothesis that an individual parameter is zero, researchers routinely employ t-tests. Second, the magnitude of parameters is of interest. Parameters can be transformed into odds ratios that have a natural interpretation. Third, and most important, is the meaning of a parameter. The meaning is expressed in terms of contrasts in the columns of the design matrix, X. For clear-cut parameter interpretation, that is, interpretation

that is independent of other parameters in the model, the λ parameters can be interpreted, based on X, as

$$\lambda = (X'X)^{-1}X' \log m$$

where X is the design matrix that contains, in its column vectors, the effects included in a model.

It is most important that the interpretation corresponds to the contrasts specified in the design matrix. To give an illustration of this type of parameter interpretation, we use the example given by von Eye and Mun (2013, p. 135). Consider a 2×2 table that results from crossing the variables A and B. For this matrix, we specify the saturated model, that is, the model with all possible parameters. The model is, in explicit form,

$$\begin{bmatrix} \log m_{11} \\ \log m_{12} \\ \log m_{21} \\ \log m_{22} \end{bmatrix} = \begin{bmatrix} 1 & 1 & 1 & 1 \\ 1 & 1 & -1 & -1 \\ 1 & -1 & 1 & -1 \\ 1 & -1 & -1 & 1 \end{bmatrix} \begin{bmatrix} \lambda \\ \lambda^A \\ \lambda^B \\ \lambda^{AB} \end{bmatrix} + \begin{bmatrix} \epsilon_{21} \\ \epsilon_{12} \\ \epsilon_{21} \\ \epsilon_{22} \end{bmatrix}$$

The first column vector of this model contains the logarithms of the observed cell frequencies. The first column vector in the design matrix represents the constant. The second, representing the main effect of Variable A, contrasts the first category of A with the second category. The third vector, representing the main effect of Variable B, contrasts the two categories of Variable B. The last column vector represents the A × B interaction. It results from element-wise multiplication of the main effect vectors of the interacting variables. After the design matrix follow the parameter and the residual vectors. One parameter is estimated for each effect, that is, each column in the design matrix.

The inner products (and the correlations) of any two of the vectors of the design matrix in the previous equation are zero. Therefore, parameter interpretation is easy. Inserting into

$\lambda = (X'X)^{-1}X' \log m$ yields, for the four parameters of this model,

$$\lambda = 1/4(\log m_{11} + \log m_{12} + \log m_{21} + \log m_{22}),$$
$$\lambda^A = 1/4(\log m_{11} + \log m_{12} - \log m_{21} - \log m_{22}),$$
$$\lambda^B = 1/4(\log m_{11} - \log m_{21} + \log m_{21} - \log m_{22}),$$
$$\lambda_{AB} = 1/4(\log m_{11} - \log m_{12} - \log m_{21} + \log m_{22}).$$

Each of these equations corresponds to the effects specified in the design matrix. This can be seen by the equal weights of the logarithms of the cell frequencies and the signs of each term. These are the same as in the design matrix. For more examples, and for examples of design matrices that do not possess this characteristic, see von Eye and Mun (2013).

Multiple approaches have been developed for log-linear analysis of longitudinal data. Models that come with a straightforward interpretation include those for which repeatedly observed variables are crossed (see, for example, von Eye & Mun, 2013). In the following data example, we present such a model.

Data Example

For the following example, we again use the data from the Finkelstein et al. (1994) study on the development of aggression. We ask whether the development of aggressive impulses over a span of 4 years (AI83 and AI87) is related to the development of physical aggression against peers (PAAP83 and PAAP85). For the following analyses, we dichotomize these variables at the grand median of aggressive impulses and the grand median of physical aggression against peers. This approach allows us to identify individuals who, overall, stay constant or display change relative to the average.

We estimate two models. The first is the model of variable independence,

$$\log m = \lambda + \lambda^{\text{AI83}} + \lambda^{\text{AI87}} + \lambda^{\text{PAAP83}} + \lambda^{\text{PAAP87}}$$

This model is estimated to have a reference for more complex models and to find out whether effects exist at all in the AI83 × AI85 × PAAP83 × PAAP87 cross-classification. For the model of variable independence, we obtain an overall goodness-of-fit maximum likelihood χ^2 of 56.50, which, for $df = 11$ suggests that this model fails to describe the data well ($p < 0.01$). We need a more complex model. Skipping the model development steps in which we successively added terms to the reference model, we describe the final model. This model includes the associations between the repeated observations of AI and PAAP as well as the cross sectional associations between AI83 and PAAP83 and AI87 and PAAP87. In other words, this is the model

$$\log m = \lambda + \lambda^{\text{AI83}} + \lambda^{\text{AI87}} + \lambda^{\text{PAAP83}} + \lambda^{\text{PAAP87}} + \lambda^{\text{AI83,AI87}} + \Lambda^{\text{PAAP83,PAAP87}} + \lambda^{\text{AI83,PAAP83}} + \lambda^{\text{AI87,PAAP87}}$$

The new terms are presented in the second row of the equation. The overall goodness-of-fit maximum likelihood

TABLE 18.2 Observed and Expected Frequencies for Log-Linear Model of the Development of Aggressive Impulses and Physical Aggression Against Peers in Adolescents

AI83 AI87 PAAP83 PAAP87	Observed frequencies	Expected frequencies
1111	24	24.49
1112	2	2.50
1121	12	12.82
1122	8	6.19
1211	4	4.79
1212	4	2.22
1221	4	2.51
1222	3	5.48
2111	8	7.22
2112	1	0.74
2121	10	9.47
2122	3	4.57
2211	6	5.50
2212	1	2.54
2221	6	7.21
2222	18	15.76

χ^2 of 6.64 suggests that, for $df = 11$, this model does describe the data well ($p = 0.47$). Table 18.2 displays the observed and the expected cell frequencies for this model. Table 18.2 shows that the observed and the expected cell frequencies are close to each other throughout. The largest standardized deviate is 1.20 (for cell 1212). This value is nonsignificant ($p = 0.12$), thus supporting the model. Table 18.3 displays the significance tests for the model parameters.

The significance tests in Table 18.3 suggest that, of the parameters of interest (these are the interactions), only one is nonsignificant (AI83 × PAAP83). We conclude that the auto-associations AI83 × AI85 and PAAP 83 × PAAP87 and the cross sectional association AI87 × PAAP87 are sufficient to explain the data in Table 18.2. There is no need to include the cross-lagged associations AI83 × PAAP87 and PAAP83 × AI87. Before finally retaining this model, we ask whether the parameters of interest come with a clear-cut interpretation. This is the case. The column vectors of the design matrix (not shown here) correlate to zero, and each of the parameters can be interpreted as specified in the design matrix.

We present one example, the parameter for the interaction between AI83 and AI87. This parameter has the interpretation

$$\begin{aligned}\lambda^{AB} = 1/16 \log m_{1111} + \log m_{1112} + \log m_{1121} + \log m_{1122} \\ - \log m_{1211} - \log m_{1212} - \log m_{1221} - \log m_{1222} \\ - \log m_{2111} - \log m_{2112} - \log m_{2121} - \log m_{2122} \\ + \log m_{2211} + \log m_{2212} + \log m_{2221} + \log m_{2222}\end{aligned}$$

We, therefore, retain the model. The substantive interpretation of this model is that self-perceived aggressive impulses and physical aggression against peers are stable over a span of four adolescent years and that, at age 15 (but not at age 11), aggressive impulses are significantly related to physical aggression against peers.

Discussion

The example given in the last section illustrates standard log-linear modeling. Many other and more complex models have been proposed (see, e.g., Agresti, 2012; von Eye & Mun, 2013). Examples of such models include logistic regression models (which can always equivalently be expressed in terms of log-linear models; see von Eye, Mair, & Bogat, 2005), latent class models, mixture distribution models, or path models. Even some IRT models can be expressed in terms of log-linear models (Meiser, 1996). Clearly, one of the greatest benefits of using log-linear models lies in their generality. We now ask whether log-linear models can be used for person-oriented research. This is possible in a number of designs.

First, log-linear models allow researchers to naturally include classification or stratification variables. This is possible when sampling is multinomial, that is when every

TABLE 18.3 Parameters of the Model of the Development of Aggressive Impulses and Physical Aggression Against Peers in Adolescents

Term tested	The model without the term				Removal of term from model		
	LOG(MLE)	Chi-square	df	*p*-value	Chi-square	df	*p*-value
AI83	−31.318	6.795	8	0.559	0.153	1	0.696
AI87	−31.635	7.429	8	0.491	0.787	1	0.375
PAAP83	−34.707	13.574	8	0.094	6.932	1	0.008
PAAP87	−36.575	17.311	8	0.027	10.668	1	0.001
AI83*AI87	−37.025	18.210	8	0.020	11.568	1	0.001
PAAP83*PAAP87	−37.662	19.484	8	0.012	12.842	1	0.000
AI83*PAAP83	−33.954	12.068	8	0.148	5.425	1	0.020
AI87*PAAP87	−37.940	20.040	8	0.010	13.398	1	0.000

new case can, in principle, be assigned to any cell of a cross-classification, but also when sampling is product-multinomial, that is, when the number of cases in a stratum or group of respondents is determined a priori. Parameter estimation is the same for both cases, but there are constraints concerning the models that can be estimated. In product-multinomial sampling designs, the estimated model must always reproduce the marginal probabilities of the variables for which these probabilities are fixed. In either case, log-linear models allow one to compare groups of individuals. The comparison can result in interactions that indicate the variable relations that are group-specific.

Alternatively, one can estimate Mantel-Haenszel-type statistics that allow one to answer the question whether interactions differ across groups (for an overview, see Fidalgo, 2005). The important characteristic of the log-linear methods that allow one to compare groups of individuals, under certain conditions and over time, is that this comparison already represents an approach to person-oriented research. These comparisons presuppose that researchers hypothesize that the comparison groups differ from each other, for example in a conditional pattern of interactions among variables. The only condition that must be met for this comparison is that of dimensional identity. Variables must have the same meaning for all comparison groups.

Interestingly, if interaction patterns are defined to be constituting elements of dimensional identity, there should be no differential interaction patterns. If, however, a researcher asks whether such patterns vary across comparison groups under the condition that, otherwise, dimensional identity prevails, results of such a comparison would indicate where dimensional identity is violated (if groups differ). This result in itself amounts to a statement of interest to person-oriented researchers. If, in contrast, interaction patterns are not part of the definition of dimensional identity, a study on differential interaction patterns can be of interest to person-oriented, idiographic, and differential developmentalists alike.

In brief, when dimensional identity can be made plausible, the comparison of a priori existing groups is of interest from a person-oriented research perspective. In this respect, log-linear models are comparable to analysis of variance (von Eye & Bogat, 2007) and structural modeling. In many instances, however, researchers just assume that multiple populations exist. Number and size of these populations are unknown. In these cases, groups need to be identified and group differences need to be established. Groups can be identified by using such methods as cluster analysis, latent class analysis, or mixture distribution decomposition.

It is important to realize that group differences can manifest in any parameter-(s). When metric variables are on the outcome side, mean differences can be examined (ANOVA). When variables are categorical, marginal distributions can be compared. In either case, groups can differ in variances, covariances, factor structures, parameters of structural models, trajectories, validity and reliability of variables, or any combination of these. Person-oriented researchers are interested in all of these.

It is also important to realize that groups may not come in prespecified sizes. Groups can be as large as standard populations and as small as just one individual. Person-oriented as well as idiographic developmental research recommend estimating parameters first at the level of the individual. Aggregation is done at the level of parameter estimates, if it is done at all. The development of methods of analysis of individual data and small group-data needs to make progress for this to be possible. Fortunately, methods for the analysis of single-subject data, in particular longitudinal data, are being developed at a rapid pace and integrated into the canon of methods for the analysis of longitudinal developmental data (e.g., Dolan, Molenaar, & Boomsma, 1989; Molenaar, 1997; Molenaar, Huizenga, & Nesselroade, 2003; Molenaar & Newell, 2010; Rovine, Molenaar, & Corneal, 1999). This development includes metric as well as categorical outcome variables (van Rijn, 2008; van Rijn & Molenaar, 2005).

Cluster Analysis

Taxometric methods, in particular cluster analysis, are the most frequently used methods for the identification of groupings that are unknown before analysis. Using these methods, one can extract groupings from a data set that contain members that

- are maximally similar to each other, while being
- maximally dissimilar to members of other groupings

The number of taxometric methods is very large. The decision about which method to select is important, because results can differ dramatically depending on the method selected. von Eye, Mun, and Indurkhya (2004) present an example of an artificial data set that can lead to classifications that are completely unrelated to each other when different taxometric methods are used. Specifically, the cross-classification of the clusters created for this data set comes with a $\chi^2 = 0.0$.

The most fundamental issue with taxometric methods concerns the measurement of the degree of similarity or

dissimilarity between individuals. The choice of which variables to include in the profile to be analyzed and the type of similarity or dissimilarity measure to be used must be carefully considered and theoretically motivated. The scaling of the variables must be commensurable and, in most cases, continuous variables must be standardized before analysis (see Bergman, Magnusson, & El-Khouri, 2003, for exceptions).

In the following section of this chapter, we first present a specification of the taxometry problem. We then review, discuss, and extend eight decisions to be made when selecting a taxometric procedure for person-oriented researchers by von Eye et al. (2004). This is followed by a data example.

Taxometry: Specification of the Problem

To illustrate the problem, we use a data example. We use Finkelstein et al.'s data on the development of aggression again. In this study, the variable self-perceived Aggressive Impulses (AI) was observed three times, in 1983, 1985, and 1987, in a sample of 114 adolescents (67 females). The trajectories AI are depicted in Figure 18.4, by Gender.

Figure 18.4 shows that all possible patterns of ups and downs were observed, and that not a single adolescent gave the same responses at all three occasions. Now, proceeding as von Eye et al. (2004) with the artificial data set, we illustrate that two cluster solutions of the same data can result in quite different classifications. We apply two methods of clustering to the aggressive impulse data, thus creating clusters of trajectories of aggressive impulses during adolescence, and then examine the cross-classification of these two cluster solutions. We create a first cluster solution by using the popular Ward method, based on Euclidean distances between the trajectories. We create the second solution with Ward's method again, but, this time, based on the correlations between the trajectories. For the distance-based classification, we select the four cluster solution, and for the correlation-based classification, we select the three cluster solution. The cross-classification appears in Table 18.4.

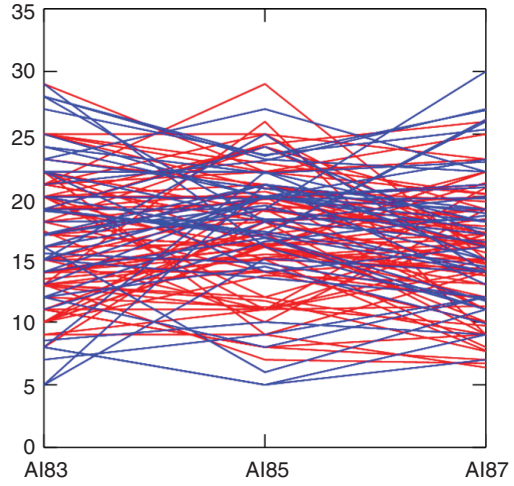


Figure 18.4 Developmental trajectories of self-perceived aggressive impulses in 114 adolescents. See footnote 1.

TABLE 18.4 Cross-Classification of Two Cluster Solutions

	1	2	3	Total
1	21	9	0	30
2	20	9	0	29
3	24	15	1	40
4	3	12	0	15
Total	**68**	**45**	**1**	**114**

Table 18.4 shows that the clusters of the distance-based solution differ in size (row totals), but each contains at least 14 of the 114 cases of the sample, that is, more than 13% of the total. In contrast, the three clusters of the correlation-based solution differ dramatically in size. The smallest cluster contains a singleton, that is, just one individual. The largest contains 68 respondents, that is, almost 60% of the total. The cross-classification of the two cluster solutions created for the aggression data comes with a $\chi^2 = 14.25$. For $df = 6$, this value suggests that knowledge of one of the two cluster solutions also contains information about the second solution ($p = 0.03$). One might be tempted to interpret this result as indicating that the two solutions are *similar*. Fact is, however, that not one of the clusters from the distance-based solution remains intact when the two solutions are crossed. Each of the two larger clusters of the correlation-based solutions feeds from the four clusters of the distance-based solution. The singleton of the correlation-based solution is a peaceful member of the third cluster of the distance-based solution. We now proceed to the specification of the taxometry problem.

In this section, we give a general definition of the clustering or classification problem (Bock, 1974, p. 22; Hartigan, 1975). Consider N objects (i.e., cases), $O_1, \ldots, O_N$. For these objects, a data matrix, x_{ki}, a similarity matrix,[10] d_{jk}, or a relation, $\prec$, can be calculated, the elements x_{ki}, d_{jk}, or $\prec$ of which represent the similarity structure of the set of objects, $S = \{O_1, \ldots, O_N\}$. Searched for is a *classification*,

[10] In later sections of this section we also discuss distance matrices. For the present purposes, we specify that the term *similarity matrix* subsumes distance matrices and other matrices that can be created to describe relations between objects.

$A = (A_1, A_2, \dots)$ of S. The subsets of this classification, that is, $A_1, A_2, \dots$ are classes (groups or clusters) that reflect the similarity structure of the objects, and imply data reduction. These two requirements can be fulfilled if both the objects within a group are maximally similar to each other and cross-class dissimilarity is maximized. The first characteristic is called *homogeneity*; the second is called *separation*. In the following sections, we review and extend the decisions that von Eye et al. (2004) discussed for the selection of a method of clustering.

Decisions for Clustering

von Eye et al. (2004) considered the following eight cluster characteristics: clusters are (1) disjoint versus overlapping; (2) hierarchical versus nonhierarchical; (3) agglomerative versus divisive; (4) exhaustive versus selective; (5) stochastic versus deterministic; (6) based on correlation versus distance measure; (7) convex versus nonconvex; and (8) represent manifest versus latent variables. The authors also discussed methods of classification that are based on such a priori set criteria as cutoffs or ranges of admissible scores and natural classifications that result from cross-classifying categorical variables. Here, we focus on taxometric clustering and on natural classifications. Cross-classifications of categorical variables are discussed in the earlier section on log-linear modeling.

Before discussing the eight criteria, it should be emphasized that there is no such thing as a true cluster solution. Instead, researchers use classification algorithms to structure data. The resulting clusters or groups reflect characteristics of the methods that were applied as well as data characteristics. Different methods are sensitive to different data characteristics. Therefore, different cluster solutions describe different data characteristics to the extent that different clustering methods are capable of depicting them.

Criterion 1: Disjunct Versus Overlapping Clusters

Disjunct classifications have the characteristic that each object O_j belongs to only one class, A_i, or cluster. In contrast, overlapping classifications allow each object O_j to belong to more than one class. Rarely, clusters naturally will be completely disjunct. Groups of cases will exist that are not easily assigned to a group. How can one deal with situations of this kind? We discuss two solutions. First, one can create nonoverlapping clusters by changing cluster parameters. This can be achieved by, for instance, reducing the diameters of clusters as long as necessary for them to no longer overlap. The number of cases located outside clusters will increase as the diameters shrink. This way, the overlap can be reduced to zero. The cases not located inside the (imaginary) cluster hull can be (1) treated as a separate group, (2) considered members of no cluster, (3) assigned to clusters at random, (4) assigned to the cluster to which they most likely belong, or (4) allowed to form separate small groupings, even singletons.

Second, one can consider whether overlapping cluster solutions may be natural or appropriate. There may be cases for which it is reasonable to assume that they belong to more than one group. For instance, an athlete may be both a soccer and a basketball player, or a student may be both a stellar performer and a political activist.

Separation of clusters indicates the degree to which clusters are disjunct. Separation can be measured only if groupings are created that are allowed to overlap. If groupings are not naturally disjunct—groupings based on manifest categorical variables are naturally disjunct—the idea that clusters are nonoverlapping clusters is perceived, by some, as artificial. Therefore, application of methods that create nonoverlapping clusters by default may require justification.

Based on this, it is surprising that a look at the literature suggests that, in empirical applications of clustering methods, researchers overwhelmingly use methods that result in nonoverlapping clusters. Similarly, most statistical software packages do not even include methods that allow researchers to create disjunct clusters. Two programs that provide this option include ADCLUS by Arabie and Caroll (1980) and Pyramid by Aude, Diaz Lazcoz, Codani, and Risler (1999; Everitt, Landau, & Leese, 2001).

Criterion 2: Hierarchical Versus Nonhierarchical Clusters

Classification algorithms can create solutions that are either hierarchical or nonhierarchical. A strength of a hierarchical cluster analysis is the simple relationship between solutions with different numbers of clusters. (For instance, a six-clusters solution is identical to a seven-clusters solution except that two clusters of the second solution are fused to constitute one cluster in the first solution.) This information is helpful for understanding the classification structure and in choosing the number of clusters. It also makes it feasible to present findings based on both a fine-grained classification with many clusters and a simpler classification with fewer clusters (since the relationship between the two classifications is straightforward and easily explained).

Hierarchical clustering algorithms create series of cluster solutions. The series are either *agglomerative* or *divisive*. Agglomerative series result from grouping objects together

into larger and larger groups. One of the chief characteristics of agglomerative hierarchical procedures is that smaller numbers of clusters result from merging clusters. Cases who were members of the same cluster will stay members of the same cluster. Divisive procedures are hierarchical if they create greater numbers of clusters by splitting larger clusters (the choice between agglomerative and divisive algorithms is discussed later under Criterion 3). A clustering method is hierarchical if cases that are members of the same cluster when clusters contain fewer cases are members of the same cluster when the cluster contains more cases. The results of hierarchical clustering are routinely displayed in the form of dendrograms (cluster trees). Figure 18.5 displays the dendrogram that results when Ward's method, based on Euclidean distances, is used to cluster the aggressive impulses trajectories shown in Figure 18.4.

Clustering algorithms are nonhierarchical when they create just one solution instead of a series of solutions. Examples of such procedures include methods that identify space density maxima and assign objects to the cluster that is defined by the nearest density center. Examples of such algorithms include the methods discussed by von Eye and Wirsing (1978, 1980) and the well-known k-means method. The number of clusters in nonhierarchical clustering is either user-determined as in k-means, or is determined by the number of space density maxima that were found based on a priori specified criteria. Hierarchical methods always create $N - 1$ solutions. A decision as to which solution to interpret is a key part of hierarchical cluster analysis.

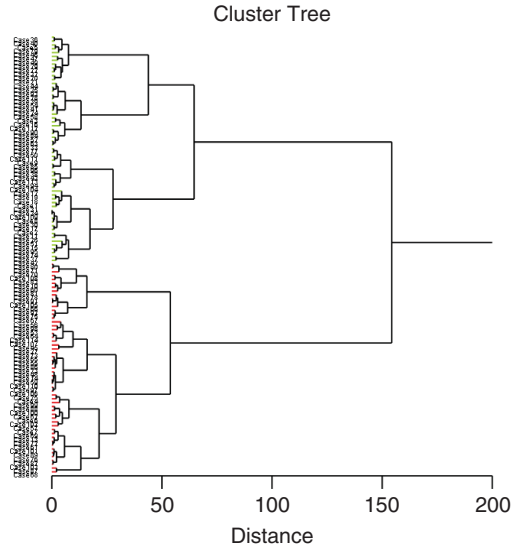


Figure 18.5 Dendrogram of Ward's clustering of aggressive impulses in adolescents. See footnote 1.

Several methods have been proposed to select from the hierarchy of cluster solutions. One involves examining a distance scale as the one used for the x-axis in Figure 18.5. Based on distance scales, the solution is selected that fulfills two conditions:

1. The number of clusters is near the desired number (see Bergman's, 2000, postulate of targeting a number of 4–10 clusters)
2. A large increase in the distance scale value has occurred

When these two conditions are fulfilled, one selects the solution right before the large increase on the distance scale. This large distance suggests that two heterogeneous clusters (clusters that are far apart from each other) had been fused into one. In the example in Figure 18.5, many researchers will select either the four- or the two-cluster solution. Because of weaknesses of selecting solutions based on distances, many other cluster evaluation methods have been discussed. Among these are methods that use various forms of the Rand Index (1971). This index measures the similarity between two cluster solutions, for example a random solution and the solution preferred by the researchers. Recent investigations have shown that the Hubert-Arabie-adjusted Rand index performs well in cluster solution evaluation (Steinley, 2004; Volkovich, Avros, & Golani, 2011). Interestingly, it has been shown that the Hubert-Arabie-adjusted Rand index is a twin of the well-known Cohen's kappa (Cohen, 1960; Warrens, 2008).

The choice between hierarchical or nonhierarchical methods can be based on two criteria. The first is whether the hierarchy of cluster solutions can be interpreted. When this is the case, hierarchical methods are selected. When, however, the series of cluster solutions is not interpreted or not interpretable, and when researchers have expectations concerning the number of clusters, nonhierarchical solutions may be selected. Only a very small number of solutions—maybe only one—needs to be calculated, and interpretation is typically straightforward. The second criterion is thus the extent to which researchers use prior knowledge when performing a cluster analysis.

Criterion 3: Agglomerative Versus Divisive Clustering

In this section, we discuss the choice between *agglomerative* and *divisive* clustering. Agglomerative clustering starts under the assumption that each object, O_j, constitutes a separate cluster. With the goals of reducing redundancy and creating a parsimonious solution, agglomerative methods join the clusters (at the beginning of the procedure, these are all individuals) that are the most similar to each

other, or, in more general terms, clusters are joined when some loss, measured, for instance, in units of decrease in information, increase on a distance scale, or difference in R^2, is minimal. This is repeated until either a stopping criterion has been reached or until the final two groupings are joined.

Divisive clustering considers, before the first step is taken, all case members of one large cluster. Beginning with the second step, divisive clustering splits the existing cluster(s). This is done such that a benefit, measured in units similar to the ones used for agglomeration, is maximized. This step is repeated until either a stopping criterion, for instance, some R^2 threshold, has been reached or until each individual constitutes its own cluster. Evidently, what is the starting point for the agglomerative procedure is the end point for the divisive procedure.

The results of both agglomeration and division clustering can be depicted in the form of *dendrograms* (tree structures). Consider, again, Figure 18.5. If the dendrogram in this figure is the result of agglomeration, we read Figure 18.3 from left to right. At the left of the dendrogram, each individual is represented. In the first steps, the most similar objects are joined to form one cluster. In each of these steps a new cluster is created. Relatively soon, clusters are joined. In the last step, the clusters that resulted from the first $n - 2$ agglomeration steps are joined, and every case belongs to the one and only cluster. Researchers interpret the solution that best meets optimality criteria as the ones discussed previously, plausibility criteria, and criteria derived from theory.

To illustrate the divisive procedure, we use Figure 18.5 again. This time, however, we read it from right to left. Before the first step (at Step 0), the procedure considers all objects members of one big cluster. Beginning with Step 1, this big cluster is subdivided into ever smaller clusters, until each object constitutes its own cluster. As for all hierarchical procedures, criteria of optimality, theory, and plausibility can be used to select a solution.

When deciding which of the two procedures to select, agglomerative or the divisive clustering, researchers keep in mind that results from both methods are often the same. Therefore, method selection is largely guided by preferences and program availability. Most software packages offer exclusively agglomerative procedures. Examples of agglomerative procedures that are available in many software packages include Single Linkage (aka Nearest Neighbor), Complete Linkage (aka Furthest Neighbor), Average Linkage, Centroid Clustering, Median Clustering, Weighted-Average Linkage, Beta-Flexible, Density Linkage, and Ward's method.

Criterion 4: Exhaustive Versus Nonexhaustive Clustering

Classifications are *exhaustive* if every case, O_j, belongs to a cluster. Nonexhaustive classifications involve only some of the N objects. The cases assigned to clusters are sometimes called the *most important* objects. Cases not assigned to a cluster are often either ignored or discarded and thrown into a *poubelle*, that is, a garbage can. From a person-oriented perspective, the distinction between exhaustive and nonexhaustive classifications is not always conclusive. If a researcher assumes that populations can be as small as just one individual, nonclassified cases constitute classes also. In addition, Bergman (1988) suggested that it may be wise not to assign all cases to a grouping under all conditions. He called these unclassified cases a residue and argued that it can be important to remove them before cluster analysis for both theoretical reasons (true unique cases) and practical reasons (an outlier could be caused by errors of measurement that can distort the findings of a cluster analysis). Further, he did not regard them as a *poubelle* but as important cases to be analyzed separately and claimed that the proportion of residue cases in the sample can provide important information about the classificability of the data set. Still, in most applications, singletons (one-case clusters) are considered cases that are not classified. Even clusters with only two cases are often ignored in follow-up analyses or assigned to the poubelle.

One way to illustrate nonexhaustive classifications involves configural frequency analysis (CFA; Lienert & Krauth, 1975; von Eye, 2002; more detail on CFA can be found in the chapter on CFA in this volume). CFA examines cross-classifications of categorical variables under the question whether the observed number of cases in a cell differs from the expected number. If the null hypothesis of no difference is rejected for a cell, CFA states that this cell constitutes a type if more cases than expected were found, and an antitype, if fewer cases than expected were found. It is a routine result of CFA that only a selection of cells constitutes types and antitypes. The frequencies in other cells do not deviate from expectancy. The designation as 'belongs to a CFA type' or 'belongs to a CFA antitype' is, therefore, nonexhaustive.

Many methods of classification can create exhaustive as well as nonexhaustive solutions. In hierarchical classifications, a nonexhaustive solution would include one or more singletons or clusters that contain only two or three objects. In nonhierarchical classification, one can create solutions with relatively large numbers of clusters. Some of these may then contain very small numbers of cases. It is important to note that classification methods differ in their tendencies

to yield small clusters. Methods that build clusters by letting objects gravitate toward a center of gravity (centroid) tend to allow larger clusters to swallow smaller clusters. Examples of such methods are the centroid method, complete linkage, average linkage, and Ward's method.

Pros and cons for exhaustive versus nonexhaustive classification are evident. On the pro side of exhaustive classification is that every object is assigned to a group. By implication, the sample under study is not reduced in size. At the expense of this strategy is, however, that, for some cases (e.g., outliers), it may be unnatural to belong to a group. Also on the con side of exhaustive clustering is the possibility that single-case clusters result. In follow-up analyses, researchers often experience problems with single-case clusters because individual cases can be hard to statistically compare with other clusters. For instance, multivariate analysis of variance (MANOVA) can be problematic for subsequent comparisons when one or more cells contain only one case.

Criterion 5: Stochastic Versus Deterministic Clustering

Stochastic models consider data points realizations of random variables. This seems appropriate under the following assumptions: (1) there is natural variation within each grouping; (2) the manifest variables that are used for classification are measured with error; and (3) the cases used for classification are a random sample. When these assumptions are met, stochastic models allow researchers to test hypotheses concerning possible group structures. In contrast, *deterministic models* consider data points fixed sets of objects. Variability is not considered random.

Pros and cons of these two approaches are evident. Among the pros of stochastic models is that statistical decisions about hypotheses that are compatible with the existence of groupings (substructures) become possible. One downside is that a null hypothesis for the existence of a particular grouping structure is not always easily specified. Among the pros of deterministic models is that parametric assumptions do not need to be made. Another downside is that existing error structures are ignored.

The question as to which of the two approaches, stochastic and deterministic clustering, is preferred by users, comes with a clear answer. Most applications use deterministic models. There certainly are multiple reasons for this preference. One reason may be that deterministic models seem easy to interpret. Another reason is that programs for stochastic clustering are part of only a few of the general purpose statistical software packages; S-plus, R, and SAS are among these.

An example of stochastic classification can be seen in a method introduced by von Eye and Gardiner (2004; von Eye & Bogat, 2005). The authors propose splitting the multivariate space of the variables to be used for classification into sectors. For each sector, the probability is estimated from the data under a hypothesis of *data generation*. Examples of data generation processes are those that generate multinormally distributed data, or uniformly distributed data. Based on the estimated probability, an expected frequency can be calculated, which then is compared with the frequency that was observed for this sector. Algorithmically, the following six steps are performed.

1. Splitting each of the d variables into c_j segments, with $c_j > 1$ and $j = 1, \ldots, d$, where j indexes the variables;
2. Crossing the segmented variables; this results in a cross-classification with $\Pi_j\, c_j$ sectors;
3. Estimating the probability of each sector. Typically, these probability do not exist a priori. Therefore, they are estimated from the data. Each of the $\Pi_j\, c_j$ sectors has the boundaries c_i^1 and c_{i+1}^1 on the first variable, c_j^2 and c_{j+1}^2 on the second variable, …, and c_l^d and c_{l+1}^d on the d-th variable, where the subscripts indicate the segments and the superscripts indicate the variables. The probability of being located in the sector with these boundaries is

$$p\left(z_i^1 - z_{i+1}^1, z_j^2 - z_{j+1}^2, \ldots, z_l^d - z_{l+1}^d\right) = \int_{z_i^1}^{z_{i+1}^2} \int_{z_j^2}^{z_{j+1}^2} \cdots \int_{z_l^d}^{z_{l+1}^d} \Psi\left(z^1, z^2, \ldots, z^d\right) \mathrm{dz}^1\, \mathrm{dz}^2 \ldots \mathrm{dz}^{\mathrm{d}}$$

   A numerical solution for this expression was proposed by Genz (1992).
4. Estimating expected sector frequencies as $e_{i,j,\ldots,l} = Np_{i,j,\ldots,l}$, where $p_{i,j,\ldots,l}$ is short for the expression given in the last equation.
5. Performing sector-specific tests. The null hypothesis for each of the tests is $E[o_{i,j,\ldots,l}] = e_{i,j,\ldots,l}$, where $o_{i,j,\ldots,l}$ is the observed frequency for the sector with subscripts $i, j, \ldots, l$. The tests can be performed using most of the residual tests from log-linear modeling. These include, for example, the Pearson X^2–component test, $X_{i,j,\ldots,l} = (o_{i,j,\ldots,l} - e_{i,j,\ldots,l})^2/e_{i,j,\ldots,l}$, where $o_{i,j,\ldots,l}$ is the observed frequency for the sector with subscripts $i, j, \ldots, l$, and $e_{i,j,\ldots,l}$ is the corresponding expected frequency. α-protection is advisable.

Based on the sector-specific test, an omnibus test can be devised by summing up the X^2–components. The resulting summary statistic can be used to test whether, overall, the cross-classification of segments follows a multinormal distribution. The test is approximately distributed as χ^2 with $df = (\Pi^d{}_{j=1} c_j) - 2d - d_{cov} - 1$, where c_j is the number of segments of the jth variable. The term d_{cov} indicates the number of covariances taken into account, typically $d_{cov} = (d)$. That is, typically all covariances are taken into account.

6. Changing the number of segments and restarting at Step 1. This sequence of steps is repeated until the clusters (sectors) are detected that contain significantly more (or fewer) cases than expected. A data example follows after all of the eight criteria have been reviewed.

Criterion 6: The Selection of Base Measures

Many clustering methods start from an $N \times N$ similarity matrix, e.g., a covariance matrix, a correlation matrix, a distance matrix, or a matrix that contains coefficients that count [in percent] the number of incidences in which two objects' characteristics match. The coefficients in such similarity matrices are termed *base measures*. The results of simulation studies suggest that "... resulting clusters depend more on the underlying similarity criterion than on the physical process of cluster formation" (Wishart, 1970, p.1).

The most popular base measures used in applications are Pearson's correlation, r, and the Euclidean distance. Correlation measures classify objects as identical if their profiles are parallel. r is not sensitive to differences in standard deviation, mean, or both. Distance measures quantify spatial distance, regardless of whether profiles are parallel or not. Correlation and distance scores coincide only if the distance, $d = 0$. For an illustration of these relations, see von Eye, Mun, and Indurkhya (2004).

When it comes to selecting from the many available base measures, it is important to keep in mind that, as we said earlier in this section, there is no true cluster solution. Clustering algorithms, in particular base measures, are sensitive to specific data characteristics, but not to others. Therefore, before selecting a base measure, researchers can do worse than answering the question concerning the data characteristics they wish to base their cluster solution on. For example, when spatial proximity is considered, the focus is on level or magnitude of behavior over time, or, if there exists a distinction that separates top from bottom clusters of developmental trajectories, a distance measure makes sense. It should be noted that variables must be commensurable when Euclidean distances are selected. If variables are not commensurable, variables with larger scores and variances can dominate the cluster solution. When, however, the similarity of profiles is of importance, that is, the degree to which profiles are parallel, trend characteristics are more interesting than levels, when researchers are interested in shape, fluctuation, or curvature similarity, and that regardless of possible differences in location and spread, a correlation measure may be more appropriate.

In some instances, a selection of a particular base measure may be hard to justify. In these instances, two strategies can be considered. First, one can create classifications using a selection of base measures. The inspection of the resulting classifications may reveal that some are more meaningful than others. A second strategy can involve using base measures that possess the characteristics of distance as well as correlational base measures. An example of such a coefficient is Cattell's (1949) coefficient, r_P. This coefficient is sensitive to profile shape, mean differences, and differences in standard deviations. To the best of our knowledge, however, this coefficient is not included in any of the general-purpose statistical software packages.

Criterion 7: Convex Versus Nonconvex Clusters

The shape of clusters can be of importance, for at least two reasons. First, when clusters take a shape that can be depicted, for example, in the form of an ellipsoid or a rectangle, verbal description of clusters will be straightforward as well. Second, when new objects are classified based on an existing cluster structure, one can ask whether these objects are located within the (imaginary) hull that represents a cluster. Many classification methods do create clusters that are *convex* in shape. Subsets (clusters) A_i are convex if any two points (cases), O_i and O_j, for $i \neq j$, can be connected by a straight line that is entirely located within A_i. Examples of convex clusters include ellipses, squares, circles, rectangles, and other types of quadrics (von Eye & Wirsing, 1978, 1980).

A number of classification methods, however, create *nonconvex* clusters. Examples include algorithms that create clusters that can take the shape of bananas. These clusters are not convex because connecting straight lines between data points can be located outside the cluster hull. Among the classification methods that yield nonconvex clusters is the *single linkage* algorithm. This algorithm agglomerates clusters based on the shortest distance of any cluster member with another element that did not belong to the cluster before. Thus, it tends to create chains rather than convex agglomerates. Hartigan (1975) noted

that "single linkage clusters are famously strung out in long sausage shapes, in which objects far apart are linked together by a chain of close objects" (p. 200) (for graphical illustrations see Hartigan, 1975, pp. 201, 202).

Usually, researchers opt for convex clusters because they can be described by using measures of central tendency or parameters that represent their hull. In particular instances, however, chain structures may be interpretable. This can be the case, for example, when events such as the next best step are grouped or social network structures.

Criterion 8: Manifest Variable Versus Latent Variable Grouping

The clustering algorithms discussed thus far in this chapter operate at the level of *manifest*, that is, observed variables. Cases are classified based on their relative distances or similarities. The resulting clusters are described also at the level of manifest variables. In contrast, there are methods that create *latent*, that is, unobserved variables, based on relations among manifest variables. As in structural equation modeling or factor analysis, these latent variables are specified to explain the covariation among the observed variables. Most popular among these methods are *latent class analysis* (LCA; Lazarsfeld & Henry, 1968; Rost & Langeheine, 1997), and latent class mixture models (LCMM; Muthén, 2001; Nagin, 1999). In this section, we briefly review these methods of latent variable grouping.

LCMM is a variant of latent growth curve modeling that is used to depict heterogeneous latent classes of trajectories. In a first approach, Muthén (2001) used baseline data to cluster cases. Then, a latent variable structure is specified to model classes' trajectories. The method is Bayesian and classes' trajectories are conditional on class membership. For evaluation, this method uses posterior predictors of class membership. In a second approach, the semiparametric, group-based approach introduced by Nagin (1999) enables researchers to model data with reference to the zero-inflated Poisson, censored normal, and binary logit distributions. A unified approach that combines these methods with IRT and multilevel modeling was recently proposed by Hsieh and collaborators (Hsieh, von Eye, & Maier, 2010; Hiseh, von Eye, Maier, & Chen, 2013).

Unobserved latent classes are defined to explain individual differences from the aggregated developmental trajectory in a population. Fixed-effect growth, that is, a fixed trajectory, is hypothesized for each class. That is, all members of one latent class are hypothesized to exhibit the same within-class developmental trajectory. The trajectories are class-specific, that is, trajectories vary across classes.

LCA of categorical variables is based on the concept of *local independence*. For each latent class, the joint probability of the observed frequencies is expressed as the product of the marginal probabilities. In other words, within each latent class, the observed variables are independent. Note, however, that local independence is a common characteristic only of the original latent class models. Mixed Markov models, in particular those for longitudinal data, do not create latent classes based on the restriction of local independence (Langeheine & van de Pol, 1993).

When it comes to describing individuals, LCA models share the characteristic that all members of a latent class have the same response probabilities for the categories of the variables included in an analysis. In other words, individuals within the same latent class are treated as identical both in their developmental trajectories and their response probabilities. In addition, members of a latent class are different than individuals in other latent classes who are also considered identical to each other. This concept is more restrictive than the concepts used in cluster analysis where members of a cluster can differ from each other but are, on average, more similar to each other than to members of other clusters.

Many researchers consider LCA the categorical variable analogue to factor analysis (Molenaar & von Eye, 1994). In the context of classification, the analogy to cluster analysis may be more interesting: LCA can also be considered a method of cluster analysis for categorical variables.

Data Example

For the following example, we reanalyze data used by von Eye and Bogat (2005). The data were collected for the first wave of a longitudinal study examining male violence toward their female partners and their children. When the women were pregnant, they were given a structured interview (the Working Model of the Child Interview; Zeanah, Benoit, Hirshberg, Barton, & Regan, 1994) to assess their perceptions and subjective experiences of their unborn children. The interviews were audio taped, transcribed, and coded on 5-point Likert scales. The three variables analyzed here—fear, joy, and anxiety—represent affective features of maternal representations (for more detail see Huth-Bocks, Levendosky, Theran, & Bogat, 2004). Each scale was coded so that low scores represent low levels of the mood represented by the scale.

We now analyze these variables using three taxometric methods, complete linkage, Ward's method, and the space-segmenting method proposed by von Eye and Gardiner (2004; von Eye & Bogat, 2005). The selection of these three methods can be explained as follows. The first

two methods are both hierarchical. We use the Euclidean distance as base measure to make results comparable and because the space-segmenting method is sensitive to agglomerations in data space as well. Of the two hierarchical algorithms, Ward's method is known for small groupings to be swallowed by larger groupings. Therefore, we expect larger clusters to result from this method than from complete linkage. The space-segmenting method can be based on the assumption of multinormality. It is, thus, a stochastic method. The other two are deterministic. All three of the methods used here are exhaustive although, theoretically, singletons could result from either hierarchical method and the space segmenting method could result in empty sectors and sectors with very small numbers of cases.

Based on the distance scales in the dendrograms (not shown here), we select for both hierarchical methods the three-cluster solution. The density plots of the three clustering variables for the three clusters from Ward's method are given in Figure 18.6. The corresponding plots for the three clusters from complete linkage are given in Figure 18.7.

Cluster 1 from Ward's method contains 48 cases. It is the smallest of the three clusters. Women in this group report average levels of fear, above average levels of joy, and slightly above average levels of anxiety. Cluster 2 is the largest cluster. It contains 103 women. These women report below average levels of fear, average levels of joy, and below average levels of anxiety. The 50 women in the third cluster report slightly above average levels of fear, clearly below average levels of joy, and elevated levels of anxiety.

Cluster 1 from complete linkage is the same as Cluster 1 from Ward's method. Cluster 2 contains 67 women who report below average levels of fear, a rather wide range with an average close to the scale midpoint of joy, and below average levels of anxiety. Cluster 3 contains 86 women who report slightly below average levels of fear, below average levels of joy, and elevated levels of anxiety.

We do not see any problems interpreting any of these profiles. More important, however, for the present discussion is that the two hierarchical solutions overlap only in part. This is shown in the cross-tabulation of the two cluster solutions in Table 18.5.

Table 18.5 shows that the first clusters from both methods contain the same respondents. The big second cluster from Ward's method is split such that 67 of the 103 women are in the second cluster from complete linkage, and the remaining 36 women are now in the third cluster from complete linkage. The third cluster from Ward's method goes entirely into the third cluster from complete linkage.

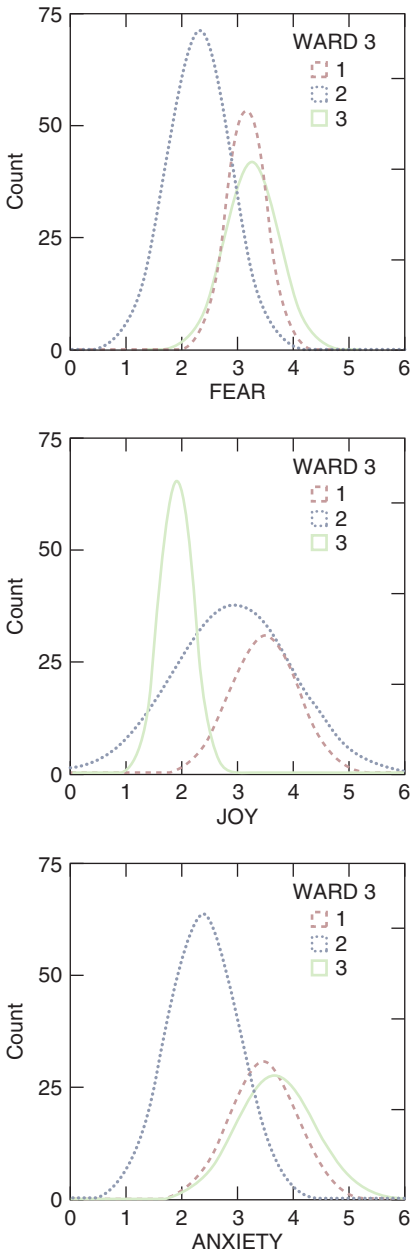


Figure 18.6 Density plots of fear, joy, and anxiety in three clusters (Ward's method). See footnote 1.

From this pattern of results, we conclude that the women in Cluster 1 are different enough from the other respondents in their location in the Fear × Joy × Anxiety space that the different characteristics of the two clustering algorithms do not affect the composition of this group. Cluster 2 from Ward's method is the largest cluster. It is possible that it resulted from swallowing small numbers of groups that, in complete linkage, stay together until they join Cluster 3. In support of this argument are (1) the agglomeration sequences that can be seen in the dendrograms (not shown

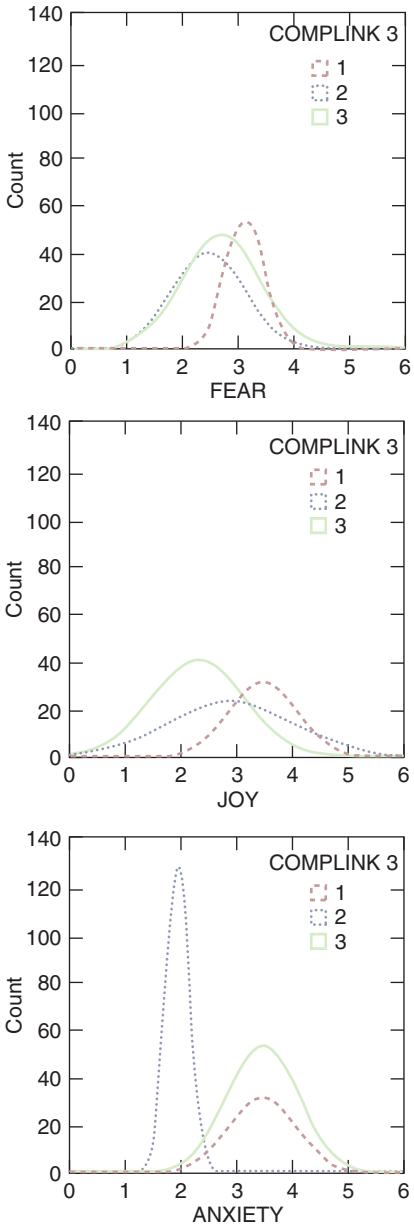


Figure 18.7 Density plots of fear, joy, and anxiety in three clusters (complete linkages). See footnote 1.

here) and (2) the fact that, in all three variables, Cluster 2 in Ward's solution has the largest variances (Figure 18.6).

The third classification of the fear–joy–anxiety data was created with the space segmenting methodology. To keep the number of sectors in the data space manageable, we dichotomized the variables at their scale-specific mean. To compare the cross-classification of the three dichotomized variables with the solutions from the hierarchical methods, we crossed the 2 × 2 × 2 table of JoyD, FearD, and AnxietyD (where the D indicates that the variables

TABLE 18.5 Cross-Tabulation of the Three-Cluster Solutions from Ward's Method (Rows) and Complete Linkage (Columns)

	1	2	3	Total
1	48	0	0	48
2	0	67	36	103
3	0	0	50	50
Total	**48**	**67**	**86**	**201**

are dichotomized) with the classifications from Ward's method and complete linkage. Table 18.6 displays the cross-classification with the solution from Ward's method.

Table 18.6 shows an interesting pattern. The first of Ward's clusters appears unchanged in the cross-classification of the three dichotomized variables. These are the individuals with the variable profile 2 2 2. The third of Ward's clusters remains intact also. These are the respondents with variable profile 2 2 1. In contrast, the second cluster from Ward's method is distributed over six of the eight possible variable profiles. This supports, from a new perspective, the interpretation of this cluster as possibly heterogeneous.

When the parallel cross-classification is created with the solution from complete linkage (table not shown here), a slightly different picture emerges. The first cluster—it is identical with the one from Ward's method—remains intact again. The second cluster is spread over the first four of the eight possible variable profiles, and the third cluster is spread over three of the eight variable profiles. This difference is not surprising, considering that the two hierarchical solutions differed in the second and the third clusters.

We estimated the cell frequencies of the 2 × 2 × 2 three-way cross-classification based on the assumption of symmetric distributions, we examined each of the eight cells by using the exact binomial test, and we protected the significance threshold α by invoking Bonferroni's procedure. Table 18.7 displays the results.

Table 18.7 shows, in its left column, the eight patterns from low levels of fear, joy, and anxiety (Pattern 1 1 1) through high levels in all three variables (Pattern 2 2 2). In its second column, the table shows the observed frequencies, m, for each pattern. This is followed by the expected frequencies, $\hat{m}$, and the binomial tail probabilities, p. The final column contains asterisks for those three patterns that were observed at different rates than expected under the assumption of independent symmetric distributions. The asterisks thus indicate clusters that deviate from the expected density in the sector that is described by the pattern in the first column. The frequencies in the remaining five patterns do not differ from expectancy.

TABLE 18.6 Cross-Classification of JoyD, FearD, and AnxietyD with the Three-Cluster Solution Created with Ward's Method

WARD3	ANXIETYD	FEARD	JOYD	Frequency	Cumulative frequency	Percent	Cumulative percent
1	1	1	1	0	0	0.000	0.000
1	1	1	2	0	0	0.000	0.000
1	1	2	1	0	0	0.000	0.000
1	1	2	2	0	0	0.000	0.000
1	2	1	1	0	0	0.000	0.000
1	2	1	2	0	0	0.000	0.000
1	2	2	1	0	0	0.000	0.000
1	2	2	2	48	48	23.881	23.881
2	1	1	1	13	61	6.468	30.348
2	1	1	2	20	81	9.950	40.299
2	1	2	1	12	93	5.970	46.269
2	1	2	2	22	115	10.945	57.214
2	2	1	1	13	128	6.468	63.682
2	2	1	2	23	151	11.443	75.124
2	2	2	1	0	151	0.000	75.124
2	2	2	2	0	151	0.000	75.124
3	1	1	1	0	151	0.000	75.124
3	1	1	2	0	151	0.000	75.124
3	1	2	1	0	151	0.000	75.124
3	1	2	2	0	151	0.000	75.124
3	2	1	1	0	151	0.000	75.124
3	2	1	2	0	151	0.000	75.124
3	2	2	1	50	201	24.876	100.000
3	2	2	2	0	201	0.000	100.000

TABLE 18.7 Space-Segment Clusters of the Cross-Classification of the Dichotomized Variables Fear, Joy, and Anxiety

Pattern	m	$\hat{m}$	p
111	13.	25.942	.00241467 *
112	13.	16.435	.22963101
121	20.	32.740	.00701107
122	23.	17.065	.08851520
211	12.	16.267	.16502180
212	50.	31.565	.00051574 *
221	22.	17.254	.14311317
222	48.	27.492	.00007017 *

The first cluster with a discrepant density is constituted by Pattern 1 1 1. Almost 26 respondents were expected to report below average levels of fear, joy, and anxiety, but only 13 did display this pattern. The second cluster with a discrepant density is constituted by Pattern 2 1 2. These are women who report above average levels of fear and anxiety and below average levels of joy. Fifty women exhibit this pattern, but only about 31 had been expected. The third cluster with a discrepant number of cases is constituted by Pattern 2 2 2. These are women who indicate above-average levels in all three emotions. About 27 had been expected to show this pattern, but 48 did.

Comparison of Cluster Solutions

The two hierarchical cluster solutions, created with Ward's method and complete linkage, can only differ because of differences in the agglomeration algorithms. As is well known, in Ward's method smaller clusters tend to gravitate toward larger clusters, thus overriding, in part, distance characteristics of cases. This can be illustrated by the large variances of the big, second cluster of the emotion data in Figure 18.6. This cluster exhibits large variances in all three variables that were used for clustering. Ward's method is, therefore, the method of choice when researchers assume that one large group of individuals exists as well as smaller groups that are remote in the data space from the large group. Ward's method also tends to create convex and, by default, nonoverlapping clusters. So does complete linkage. However, with complete linkage, there is less of a tendency for larger clusters to absorb smaller ones.

The space-segmenting method creates natural groupings by segmenting the data space in blocks. The solution of this method can be optimized by varying the number of segments (more segments result in smaller numbers of cases per segment) and by varying assumptions concerning the data-generating mechanism. For example, researchers can test the hypothesis that agglomerations near the centroid of the data space are excessive assuming the data are multinormally or, alternatively, uniformly distributed. Different numbers of segments and different distributional assumptions can result in different segments that contain more cases or segments that contain fewer cases than expected under these assumptions.

Discussion: Cluster Analysis and Person-Oriented Research

With respect to the tenets of person-oriented research, cluster analytic methods, mixture distribution decomposition, latent class analysis or, in general, taxometric methods are most interesting. These methods allow one to create groups of individuals that are similar because of their profiles and not by definition. By implication, clusters are more homogeneous than populations. Developmental trajectories are, therefore, cluster-specific and do not describe everybody. They only describe those who belong to a particular cluster. If singletons are considered clusters, cluster analysis will allow researchers to also identify those cases that cannot meaningfully be assigned to a cluster. Reasons for these individuals to be different include unique profiles and, of course, measurement error.

Another most important characteristic of cluster solution is that it is far more likely that dimensional identity holds for a cluster than for an entire population. The meaning and the test statistical characteristics of measurement instruments can be expected to be the same for everybody within a cluster (*can be expected* means should be ascertained). This is less important when variables are nominal level. It is important, however, when the variables used in a study are test scores. As was discussed by von Eye and Bergman (2003), test statistical characteristics of measurement scales can differ across populations (this is also discussed as *differential validity* or *differential item functioning*; von Eye, Bergman, & Hsieh, 2015). Whenever items or tests differ in their characteristics across groups, scores from these instruments cannot easily be compared when they describe individuals from different groups, even if they were given the same score on a scale. Individuals from the same group, however, can be compared.

Because of these interesting characteristics, many studies in which researchers used taxometric methods have been published under the auspices of person-oriented research. There are downsides, however. The first of these is that, although taxometric methods are designed to capture unmeasured heterogeneity, it can be very hard to make sure the groups that appear as clusters do exist. To establish a cluster solution, researchers typically undertake two steps. In the first, the best solution is identified. This is done, when latent class analysis is performed, by comparing information criteria from solutions that differ in the number of latent classes. The most likely grouping can thus be identified. When hierarchical clustering algorithms are applied, researchers select from solutions that differ in their distance measure, R^2, or the Rand index (Steinley, 2004). The performance of these indexes is, however, still subject to discussion and research. This is why the second step is equally important. In this step, cluster members are compared on measures that have not been used for clustering. This way, external validity of clusters can be established.

There is another reason why these steps are very important. Many taxometric methods deliver solutions under virtually all conditions. Not all of these solutions, however, describe groups that naturally or meaningfully exist. Matters are complicated by the fact previously illustrated that multiple solutions can be created, and many of these may be interpretable and meaningful. The selection from multiple solutions that have these characteristics is no easy task. Still, taxometric methods are extremely useful options when researchers suspect that unmeasured heterogeneity exists that may be interesting and should be captured because it has the potential of fogging results that are created when this heterogeneity is ignored.

CONCLUSION AND FUTURE DIRECTIONS

There is a striking continuity that connects the ideas of Stern, Lewin, and Block with those of present-day advocates of the person-oriented approach (e.g., Magnusson, Bergman, von Eye). We briefly summarize those continuities as well as suggest where, going forward, person-oriented approaches might lead research and application in developmental psychopathology.

The basic principles for psychological inquiry from a person-oriented approach have existed for more than 100 years. Understanding the individual is widely held to be central to the enterprise of psychological science. Concerns regarding a focus on lawfulness at the expense of understanding important aspects of individual human experience have been raised consistently from Stern to the present. Importantly, all theorists focus on the complexity of the process of development, indicating that there will be multiple determinants of the phenomena or process under investigation. The analogy of the individual as a dynamic system, whose component parts are integrated in a meaningful and coherent way also underpins much of the writing. Within this framework, concerns with change as well as stability are considered important aspects of human behavior. Researchers also emphasize the false dichotomy of the psychological and physiological aspects of an individual and the importance of understanding the individual and how he or she transacts with the environment.

Although research methods have evolved dramatically in the past 100 years, basic concerns with the inadequacies

of extant statistical techniques as a means to elucidate individual behavior as well as attempts to develop alternative methods has been ongoing. Concerns with aggregation of data to explain individuals is a theme that continually emerges in person-oriented research. For example, as we noted previously, Block was interested in subgroups (i.e., heterogeneity within the aggregate) and concerned about the use of factor analysis in personality research, including his own. Lewin's sentiments about the uniqueness of the individual child, quoted earlier, speak to the *ecological fallacy* decades before it received its moniker. Modern researchers, as we have noted, critique methods that aggregate individuals as well as those that make questionable and simplifying assumptions about variable relationships, data distributions, and random error. These researchers have also developed new, and revised existing, methods that circumvent some of these problems. Finally, following from the dynamic systems view, with integrated components together reflecting the system, person-oriented researchers have long realized the need to make, as far as possible, the whole person the object of study. In the early days of psychology, this goal was strived for by applying subjective methods, for example, when psychiatrists used observations and interviews to divide a sample of patients into different types. During the last decades, objective statistical methods of pattern analysis and classification have been increasingly applied; methods that are based on the pattern of variable values as the analytic unit. Hence, the old objections against a typological approach as being subjective and unscientific are no longer valid.

What measures are used to assess individuals is another thread of continuity between the past and present. Early assessments of intelligence were highly individualized (e.g., Binet). Stern's consternation with the overuse of intelligence tests and the intelligence quotient led him to develop methods that reflected person–world convergence; that is, test instruments should not compare the individual to others (as in classical test theory) but rather to himself or herself vis-à-vis the environment in which he or she interacts (Lamiell, 2010a). Loevinger's focus on differential scaling predates von Eye et al.'s (2015) reformulation of IRT as a person-oriented tool. Block's Q-set, described earlier, attempted to better reflect the complexity of the individual. von Eye (e.g., von Eye & Bergman, 2003; von Eye et al., 2015) has consistently raised concerns regarding dimensional identity of assessment instruments—items, measures, and constructs may have different meaning for different individuals at different times in their development. Going forward, issues of scaling and measurement are in need of more attention; researchers still, too often, rely on the principles of classical test theory that do not align with the person-oriented approach.

How should the field of developmental psychopathology integrate the person-oriented approach as a legitimate paradigm and not just the poor stepchild of the variable-oriented approach? One idea is that developmental psychopathology researchers explicitly identify whether they employ a variable- or person-oriented approach. At present, as far as we are aware, only research that employs a person-oriented approach identifies it as such. von Eye et al. (2015) remind us that the search for universals, which is best done via variable-oriented approaches, can be important in the early stages of a new field of inquiry. However, research conducted from a variable-oriented approach cannot normally make statements about the individual. Thus, variable-oriented research should refrain from making such statements in the hypotheses and in the interpretation of results.

The language that researchers employ in this regard is important. Hypotheses and results must be formulated such that the person- versus variable-orientation of researchers becomes visible and, more important, defensible. For example, it is not valid to state that a person with long experience as a victim will be depressed, if the basis for such a conclusion is simply a correlation between duration of exposure and depression (that would amount to committing the sin of ecological fallacy). Person-oriented statements require person-oriented research and vice versa (but the first of these is more important).

Discussion sections often include statements that the findings cannot be generalized to a population other than the one studied, but limitations inherent in the variable-oriented approach are not presented (e.g., cross sectional data do not elucidate process, universals can only be deemed such if validated by person-oriented research, assessment instruments may not have dimensional identity, aggregation of data limits the interpretation of results). Framing the argument and hypotheses as either variable- or person-oriented and acknowledging the limitations of each approach in the discussion section would be more than a mere exercise; it would provide scaffolding for clearer thinking in theory and methods. The need for transparency about the underlying paradigm driving research is analogous in some ways to Stern's (1911) delineation of four specific types of research in differential psychology (Lamiell, 2010a). More recently, in discussing the tension between person- and variable-oriented approaches, Cervone (2005) noted the need to acknowledge explicitly that these two approaches lead to two different types of information.

Focusing specifically on person-oriented research, what does the future hold as it applies to the field of developmental psychopathology? The NIMH Research Doman Criteria (RDoC, 2011), in an attempt to generate innovative and integrative research on brain and behavior, provides a starting point. The research domain criteria matrix suggested has units of analysis (from genes to paradigms) as the columns and domains/constructs (e.g., negative valence systems, cognitive systems) as the rows. The initiative emphasizes linking behavior with brain functioning and genetics. Unfortunately, developmental and environmental aspects of psychopathology are not in the matrix, although NIMH indicates that these are critical elements for researchers to consider. Research from a person-oriented approach would include environment and development in the matrix itself. Because the individual is a dynamic system, the person-oriented approach cannot explain the transaction of brain and behavior factors without taking environment and development into consideration. In addition, the patterning of these different factors within individuals or groups of individuals would be necessary for the research to be person oriented.

Already we see the first signs of person-oriented research moving in this direction. The cortisol/behavior (Towe-Goodman et al., 2011) and cortisol/mental health/environment research (Bogat et al., 2012) cited earlier, research on ADHD that bases classifications on differential patterns of brain functioning (e.g., Fair, Bathula, Nikolas, & Nigg, 2012), and formulations of temperament calling for person-oriented methods that incorporate aspects of child behavior as well as central and peripheral nervous system factors (Nigg, 2006) all begin to integrate facets of the RDoC matrix.

The NIMH RDoC is, of course, part of a wider movement within psychology and medicine toward translational research; that is, taking findings from basic research and applying them to clinical research with the goal of enhancing health or preventing problems from developing in the first place. Person-oriented research has a strong role to play in translational research. In medicine, cutting-edge cancer research eschews a one-size-fits-all treatment approach. Person-oriented approaches, whereby physicians and researchers analyze genetic and molecular abnormalities in an individual's cancer, allow for more precise treatments to cure the cancer and cause the fewest side effects possible. Person-oriented approaches offer the same promise for psychological interventions. For example, batterer intervention programs (BIPs), often mandated by courts in cases where men perpetrate intimate partner violence, are generally ineffective. A meta-analysis by Feder and Wilson (2005) found that the programs had no effect on victim's reports of revictimization and only minimal effects on police reports of violence. An NIJ meeting convened to address this problem provided guidelines for practitioners and researchers going forward (National Institute of Justice, 2011); however, only one research recommendation was somewhat person oriented ("Study experiences in BIPs of men by ethnic group"). This is unfortunate. In the case of BIPs or other interventions, translational research, using person-oriented approaches to understand individual behavior (genetic, psychological, and environmental causes), holds the promise of individualized treatments to help the broadest number of individuals.

In conclusion, a core goal of developmental psychopathology is to understand individual development. The direct and natural path to follow is then to study *individual* development of persons in real life, taking into consideration that we function as integrated organisms in interaction with our environment. This standpoint naturally leads to a person-oriented approach. Those who claim they can understand individual development by applying variable-oriented methods must carry the burden of proof that such an indirect approach is defensible.

REFERENCES

Agresti, A. (2012). *Categorical data analysis* (3rd ed.). New York, NY: Wiley.

Allport, G. W. (1937). *Personality—a psychological interpretation*. New York, NY: Holt.

Ansara, D. L., & Hindin, M. J. (2010). Exploring gender differences in the patterns of intimate partner violence in Canada: a latent class approach. *Journal of Epidemiology & Community Health*, *64*, 849–854. doi: 10.1136/jech.2009.095208

Arabie, P., & Caroll, J. D. (1980). MAPCLUS: A mathematical programming approach to fitting the ADCLUS model. *Psychometrika*, *45*, 211–235.doi: 10.1007/BF02294077

Arminger, G., & Stein, P. (1997). Finite mixture of covariance structure models with regressors: loglikehood function, distance estimation, fit indices, and a complex example. *Sociological Methods and Research*, *26*, 148–182. doi: 10.1177/0049124197026002002

Aude, J. C., Diaz Lazcoz, Y., Codani, J. J., & Risler, J. L. (1999). Applications of the pyramidal clustering method to biological objects. *Computers and Chemistry*, *23*, 303–325. doi: 10.1016/S0097-8485(99)00006-6

Axen, I., Bodin, L., Bergström, G., Halasz, L., Lange, F., Lövgren, P. W., . . . Jensen, I. (2011). Clustering patients on the basis of their individual course of low back pain over a six month period. *BMC Musculoskeletal Disorders*, *12*, 99–108. doi: 10.1186/1471-2474-12-99

Banfield, J. D., & Raftery, A. E. (1993). Model-based Gaussian and non-Gaussian clustering. *Biometrics*, *9*, 803–821. doi: 10.2307/2532201

Bergman, L. R. (1988). You can't classify all of the people all of the time. *Multivariate Behavioral Research*, *23*, 425–441. doi: 10.1207/s15327906mbr2304_1

Bergman, L. R. (2000). The application of a person-oriented approach: Types and clusters. In L. R. Bergman, R. B. Cairns, L. G. Nilsson, & L. Nystedt (Eds.), *Developmental science and the holistic approach* (pp. 137–154). Mahwah, NJ: Lawrence Erlbaum.

Bergman, L. R., & Andersson, H. (2010). The person and the variable in developmental psychology. *Journal of Psychology*, *218*, 155–165. doi: 10.1027/0044-3409/a000025

Bergman, L. R., & Magnusson, D. (1991). Stability and change in patterns of extrinsic adjustment problems. In D. Magnusson, L. R. Bergman, & B. Törestad (Eds.), *Problems and methods in longitudinal research: Stability and change* (pp. 323–346). New York, NY: Cambridge University Press. doi: 10.1017/CBO9780511663260.017

Bergman, L. R., & Magnusson, D. (1997). A person-oriented approach in research on developmental psychopathology. *Development and Psychopathology*, *9*, 291–319. doi: 10.1017/S095457949700206X

Bergman, L. R., Magnusson, D., & El-Khouri, B. M. (2003). *Studying individual development in an interinidividual context: A person-oriented approach*. Mahwah, NJ: Erlbaum.

Bergman, L. R., & Trost, K. (2006). The person-oriented versus the variable-oriented approach: Are they complementary, opposites, or exploring different worlds? *Merrill-Palmer Quarterly*, *52*, 601–632. doi: 10.1353/mpq.2006.0023

Block, J. (1955). The difference between Q and R. *Psychological Review*, *62*, 356–358. doi: 10.1037/h0042760

Block, J. (1961). *The Q-sort method in personality assessment and psychiatric research*. Springfield, IL: Charles C. Thomas.

Block, J. (1971). *Lives through time*. Berkeley, CA: Bancroft Books. doi: 10.1037/13141-000

Block, J. (1995). A contrarian view of the five-factor approach to personality description. *Psychological Bulletin*, *117*, 187–215. doi: 10.1037/0033-2909.117.2.187

Block, J. (2001). Millennial contrarianism: The five-factor approach to personality description 5 years later. *Journal of Research in Personality*, *35*, 98–107. doi: 10.1006/jrpe.2000.2293

Block, J. (2010). The five-factor framing of personality and beyond: Some ruminations. *Psychological Inquiry*, *21*, 2–25. doi: 10.1080/10478401003596626

Bock, H. H. (1974). *Automatische Klassifikation* [Automatic Classification]. Gottingen, Germany: Vandenhoeck & Ruprecht.

Bogat, G. A. (2009). Is the person orientation necessary in community psychology? *American Journal of Community Psychology*, *43*, 22–34. doi: 10.1007/s10464-008-9215-7

Bogat, G. A., Leahy, K. L., von Eye, A., Maxwell, C., Levendosky, A. A., & Davidson, W. S. (2005). The influence of community violence on the functioning of battered women. *American Journal of Community Psychology*, *36*(1–2), 123–132. doi: 10.1007/s10464-005-6237-2

Bogat, G. A., Levendosky, A. A., DeJonghe, E., Davidson, W. S., & von Eye, A. (2004). Pathways of suffering: The temporal effects of domestic violence on women's mental health. *Maltrattamento e abuso all'infanzia*, *6*(2), 97–112.

Bogat, G. A., Levendosky, A. A., Lonstein, J. S., Martinez-Torteya, C., Muzik, M., & Granger, D. A. (2012, October). *Effects of pre and postpartum intimate partner violence (IPV) on maternal and HPA Axis functioning*. Paper presented at International Society for Developmental Psychobiology, New Orleans, LA.

Bollen, K. A. (1989). *Structural equations with latent variables*. New York, NY: Wiley.

Borkenau, P., & Ostendorf, F. (1998). The Big Five as states: How useful is the five-factor model to describe intra-individual variations over time? *Journal of Research in Personality*, *32*, 202–221. doi: 10.1006/jrpe.1997.2206

Boscardin, C. K., Muthén, B., Francis, D. J., & Baker, E. L. (2008). Early identification of reading difficulties using heterogeneous developmental trajectories. *Journal of Educational Psychology*, *100*, 192–208. doi: 10.1037/0022-0663.100.1.192

Bronfenbrenner, U. (1977). Toward an experimental ecology of human development. *American Psychologist*, *32*, 513–531. doi: 10.1037/0003-066X.32.7.513

Bronfenbrenner, U. (1986a). Ecology of the family as a context for human development: Research perspectives. *Developmental Psychology*, *22*, 723–742. doi: 10.1037/0012-1649.22.6.723

Bronfenbrenner, U. (1986b). Recent advances in research on the ecology of human development. In R. K. Silbereisen, K. Eyferth, & G. Rudinger (Eds.), *Development as action in context: Problem behavior and normal youth development* (pp. 287–309). Heidelberg, Germany: Springer-Verlag. doi: 10.1007/978-3-662-02475-1_15

Bronfenbrenner, U., & Morris, P. A. (1983). The evolution of environmental models in developmental research. In P. H. Mussen & W. Kessen (Eds.), *Handbook of child psychology: History, theory, and methods* (Vol. 1, pp. 357–414). New York, NY: Wiley.

Cairns, R. B. (1983). The emergence of developmental psychology. In P. H. Mussen (Series Ed.) & W. Kessen (Vol. Ed.), *Handbook of child psychology: History, theories, and methods*, (4th ed., Vol. 1, pp. 41–101). New York, NY: Wiley.

Cattell, R. B. (1946). *Description and measurement of personality*. Oxford, UK: World Book Company.

Cattell, R. B. (1949). *rp* and other coefficients of pattern similarity. *Psychometrika*, *14*, 279–298. doi: 10.1007/BF02289193

Cattell, R. B., & Tsujioka, B. (1964). The importance of factor-trueness and validity versus homogeneity and orthogonality in test scales. *Educational and Psychological Measurement*, *24*, 3–30. doi: 10.1177/001316446402400101

Cauce, A. M. (2008). Parenting, culture, and context: Reflections on excavating culture. *Applied Developmental Science*, *12*, 227–229. doi: 10.1080/10888690802388177

Cervone, D. (2005). Personality architecture: within-person structures and processes. *Annual Review of Psychology*, *56*, 423–452. doi: 10.1146/annurev.psych.56.091103.070133

Cicchetti, D., & Scheider-Rosen, K. (1986). An organizational approach to childhood depression. In M. Rutter, C. Izard, & P. Read (Eds.), *Depression in young people: Clinical and developmental perspectives* (pp. 71–134). New York, NY: Guilford.

Cohen, J. (1960). A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*, *20*, 37–46. doi: 10.1177/001316446002000104

Costa, P. T., Herbst, J. H., McCrae, R. R., Samuels, J., & Ozer, D. J. (2002). The replicability and utility of three personality types. *European Journal of Personality*, *16*, S73–S87. doi: 10.1002/per.448

Cronbach, L. J. (1953). Correlations between persons as a research tool. In O. H. Mowrer (Ed.), *Psychotherapy: Theory and research*, (pp. 376–388). New York, NY: Ronald. doi: 10.1037/10572-014

Davies P. T., & Forman, E. M. (2002). Children's patterns of preserving emotional security in the interparental subsystem. *Child Development*, *73*, 1880–1903. doi: 10.1111/1467-8624.t01-1-00512

Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., & Cummings, E. M. (2007). The role of child adrenocortical functioning in pathways between interparental conflict and child maladjustment. *Developmental Psychology*, *43*, 918–930. doi: 10.1037/0012-1649.43.4.918

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience & Biobehavioral Reviews*, *35*, 1562–1592. doi: 10.1016/j.neubiorev.2010.11.007

Del Giudice, M., Hinnant, J. B., Ellis, B. J., & El-Sheikh, M. (2012). Adaptive patterns of stress responsivity: A preliminary investigation. *Developmental Psychology*, *48*, 775–790. doi: 10.1037/a0026519

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders*, *5th edition*. Arlington, VA: American Psychiatric Association Publishing.

Dolan, C. V., Molenaar, P. C. M., & Boomsma, D. I. (1989). LISREL analysis of twin data with structured means. *Behavior Genetics*, *19*(1), 51–62. doi: 10.1007/BF01065883

Dolan, C. V., & van der Maas, H. L. J. (1997). Fitting multivariate normal finite mixtures subject to structural equation modeling. *Psychometrika*, *63*, 227–253. doi: 10.1007/BF02294853

Donnellan, M. B., & Robins, R. W. (2010). Resilient, undercontrolling, and overcontrolling personality types: Issues and controversies. *Social and Personality Psychology Compass*, *4*, 1070–1083. doi: 10.1111/j.1751-9004.2010.00313.x

Ellis, B. J., Del Giudice, M., & Shirtcliff, E. A. (2013). Beyond allostatic load: The stress response system as a mechanism of conditional adaptation. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (2nd ed., pp. 251–284). New York, NY: Wiley.

Everitt, B. S. (1998). *The Cambridge dictionary of statistics*. Cambridge, UK: Cambridge University Press.

Everitt, B. S., Landau, S., & Leese, M. (2001). *Cluster analysis* (4th ed.). London, UK: Arnold.

Eysenck, H. J. (1954). The science of personality: nomothetic! *Psychological Review*, *61*, 339–342. doi: 10.1037/h0058333

Fair, D. A., Bathula, D., Nikolas, M. A., & Nigg, J. T. (2012) Distinct neuropsychological subgroups in typically developing youth inform heterogeneity in children with ADHD. *Proceedings of the National Academy of Sciences*, *109*, 6769–6774. doi: 10.1073/pnas.1115365109

Feder, L., Wilson, D. B. (2005). A meta-analytic review of court-mandated batterer interventions programs: can courts affect abusers' behavior? *Journal of Experimental Criminology*, *1*, 239–62. doi: 10.1007/s11292-005-1179-0

Fidalgo, A. M. (2005). Mantel-Haenszel methods. In B. S. Everitt & D. C. Howell (Eds.), *Encyclopedia of statistics in behavioral science* (Vol. 3, pp. 1120–1126). Chichester, UK: Wiley. doi: 10.1002/0470013192.bsa364

Finkelstein, J. W., von Eye, A., & Preece, M. A. (1994). The relationship between aggressive behavior and puberty in normal adolescents: A longitudinal study. *Journal of Adolescent Health*, *15*, 319–326. doi: 10.1016/1054-139X(94)90605-X

Fogel, A., Garvey, A., Hsu, H., & West-Stroming, D. (2006). *Change processes in relationships: Relational-historical research on a dynamic system of communication*. Cambridge, UK: Cambridge University Press. doi: 10.1017/CBO9780511489686

Foti, R. J., Thompson, N. J., & Allgood, S. F. (2011). The pattern-oriented approach: A framework for the experience of work. *Industrial and Organizational Psychology–Perspectives on Science and Practice*, *4*(1), 122–125. doi: 10.1111/j.1754-9434.2010.01309.x

Genz, A. (1992). Numerical computation of multivariate normal probabilities. *Journal of Computational and Graphical Statistics*, *1*, 141–149. doi: 10.1080/10618600.1992.10477010

Gottfredson, M. R. (2005). Offender classifications and treatment effects in developmental criminology: A propensity/event consideration. *The ANNALS of the American Academy of Political and Social Science*, *602*, 46–56. doi: 10.1177/0002716205279937

Greenland, S. (2001). Ecologic versus individual-level sources of bias in ecologic estimates of contextual health effects. *International Journal of Epidemiology*, *30*, 1343–1350. doi: 10.1093/ije/30.6.1343

Gunnar, M. R., & Quevedo, K. M. (2007). Early care experiences and HPA axis regulation in children: A mechanism for later trauma vulnerability. *Progress in Brain Research*, *167*, 137–149. doi: 10.1016/S0079-6123(07)67010-1

Hartigan, J. A. (1975). *Clustering algorithms*. New York, NY: John Wiley.

Hartshorne, H., & May, M. A. (1928). *Studies in the nature of character. Vol. 1: Studies in deceit*. New York, NY: Macmillan.

Hartshorne, H., & May, M. A. (1929). *Studies in the nature of character. Vol 2: Studies in service and self control*. New York, NY: Macmillan. doi: 10.1037/11334-000

Hartshorne, H., May, M. A., & Shuttleworth, F. K. (1930). *Studies in the nature of character. Vol. 3*: *Studies in the organization of character*. New York, NY: Macmillan.

Horney, J. D., Osgood, D. W., & Marshall, I. H. (1995). Criminal careers in the short-term: Intra-individual variability in crime and its relation to local life circumstances. *American Sociological Review*, *60*, 655–673. doi: 10.2307/2096316

Hoshino, T., & Bentler, P. M. (2011). *Bias in factor score regression and a simple solution*. Department of Statistics, University of California–Los Angeles. Retrieved from http://www.escholarship.org/uc/item/8w9514jw

Hsieh, C. A., von Eye, A., & Maier, K. S. (2010). Using a multivariate multilevel Item Response Theory model to study parallel processes of change: The dynamic association between adolescents' social isolation and engagement with delinquent peers in the National Youth Survey. *Multivariate Behavioral Research*, *45*, 508–552. doi: 10.1080/00273171.2010.483387

Hsieh, C. A., von Eye, A., Maier, K. S., & Chen, S. H. (2013). A unified latent growth curve model. *Structural Equation Modeling: A Multidisciplinary Journal*, *20*, 592–615. doi: 10.1080/10705511.2013.824778

Huth-Bocks, A. C., Levendosky, A. A., Theran, S. A., & Bogat, G. A. (2004). The impact of domestic violence on mothers' prenatal representations of their infants. *Infant Mental Health Journal*, *25*, 79–98. doi: 10.1002/imhj.10094

Jöreskog, K. G., & Sörbom, D. (2001). *LISREL 8: User's reference guide*. Lincolnwood, IL: Scientific Software International.

Keenan, K., Grace, D., & Gunthorpe, D. (2003). Examining stress reactivity in neonates: Relations between cortisol and behavior. *Child Development*, *74*, 1930–1942. doi: 10.1046/j.1467-8624.2003.00647.x

Kelso, J. A. S. (1995). *Dynamic patterns: The self organization of brain and behavior*. Boston, MA: MIT Press.

Kelso, J. A. S. (2000). Principles of dynamic pattern formation and change for a science of human behavior. In L. R. Bergman, R. B. Cairns, L. G. Nilsson, & L. Nystedt (Eds.), *Developmental science and the holistic approach* (pp. 63–83). Mahwah, NJ: Erlbaum.

Kiesler, D. J. (1966). Some myths of psychotherapy research and the search for a paradigm. *Psychological Bulletin*, *65*, 110–136. doi: 10.1037/h0022911

Kim, D. (2008). Blues from the neighborhood? Neighborhood characteristics and depression. *Epidemiologic Reviews*, *30*, 101–117. doi: 10.1093/epirev/mxn009

Kim, J., & Fram, M. S. (2009). Profiles of choice: Parents' patterns of priority in child care decision-making. *Early Childhood Research Quarterly*, *24*(1), 77–91. doi: 10.1016/j.ecresq.2008.10.001

Kline, R. B. (2011). *Principles and practice of structural equation modeling* (3rd ed.). New York, NY: Guilford Press.

Klump, K. L., Culbert, K. M., Slane, J. D., Burt, S. A., Sisk, C. L., & Nigg, J. T. (2012). The effects of puberty on genetic risk for disordered

eating: Evidence for a sex difference. *Psychological Medicine*, *42*(3), 627–638. doi: 10.1017/S0033291711001541

Klump, K. L., Keel, P. K., Sisk, C. L., & Burt, S. A. (2010). Preliminary evidence that estradiol moderates genetic effects on disordered eating attitudes and behaviors during puberty. *Psychological Medicine*, *40*(10), 1745–1754. doi: 10.1017/S0033291709992236

Klump, K. L., Perkins, P., Burt, S. A., McGue, M., & Iacono, W. G. (2007). Puberty moderates genetic influences on disordered eating. *Psychological Medicine*, *37*, 627–634. doi: 10.1017/S0033291707000189

Krauth, J., & Lienert, G. A. (1973). *Die Konfigurationsfrekvenz-analyse (KFA)* [Configural frequency analysis (CFA)]. Freiburg, Germany: Karl Alber.

Kuhn, T. S. (1962/2012). *The structure of scientific revolutions: Fiftieth anniversary edition*. Chicago, IL: University of Chicago Press.

Kunnen, S. E. (2012). *A dynamic systems approach to adolescent development*. New York, NY: Psychology Press.

Laible, D., Carlo, G., Panfile, T., Eye, J., & Parker, J. (2010). Negative emotionality and emotion regulation: A person-centered approach to predicting socioemotional adjustment in young adolescents. *Journal of Research in Personality*, *44*(5), 621–629. doi: 10.1016/j.jrp.2010.08.003

Lamiell, J. T. (2003). *Beyond individual and group differences: Human individuality, scientific psychology, and William Stern's critical personalism*. Thousand Oaks, CA: Sage.

Lamiell, J. T. (2009). Reviving person-centered inquiry in psychology: Why it's erstwhile dormancy? In J. Valsiner, P. C. M. Molenaar, M. C. D. P. Lyra, & N. Chaudhary (Eds.), *Dynamic process methodology in the social and developmental sciences* (pp. 31–43). New York, NY: Springer. doi: 10.1007/978-0-387-95922-1_2

Lamiell, J. T. (2010a). Reflections on some neglected ideas about psychological measurement from the personalistic perspective of William Stern (1871–1938). In A. Toomela & J. Valsiner (Eds.), *Methodological thinking in psychology: 60 years gone astray?* (pp. 180–207). Charlotte, NC: Information Age Publishing, Inc.

Lamiell, J. T. (2010b). Why was there no place for personalistic thinking in 20th century psychology? *New Ideas in Psychology*, *28*, 135–142. doi: 10.1016/j.newideapsych.2009.02.002

Langeheine, R., & van de Pol, F. (1993). A unifying framework for Markov modeling in discrete space and discrete time. *Sociological Methods & Research*, *18*, 416–441. doi: 10.1177/0049124190018004002

Laub, J. H., & Sampson, R. J. (2003). *Shared beginnings, divergent lives: Delinquent boys to age 70*. Cambridge, MA: Harvard University Press.

Lazarsfeld, P. F., & Henry, N. W. (1968). *Latent structure analysis*. Boston, MA: Houghton Mifflin.

Lennon, M. C., McAllister, W., Kuang, L., & Herman, D. B. (2005). Capturing intervention effects over time: Reanalysis of a critical time intervention for homeless mentally ill men. *American Journal of Public Health*, *95*(10), 1760–1766. doi: 10.2105/AJPH.2005.064402

Lerner, R. M., & Busch-Rossnagel, N. (1981). Individuals as producers of their development: Conceptual and empirical bases. In R. M. Lerner & N. A. Busch-Rossnagel (Eds.), *Individuals as producers of their development: A life-span perspective* (pp. 1–36). New York, NY: Academic Press. doi: 10.1016/B978-0-12-444550-5.50008-0

Lerner, R. M., Taylor, C. S., & von Eye, A. (Eds.). (2002). *Pathways to positive youth development among diverse youth*. San Francisco, CA: Jossey-Bass.

Lerner, R. M., von Eye, A., Lerner, J. V., & Lewin-Bizan, S. (2009). Exploring the foundations and functions of adolescent thriving within the 3-H study of positive youth development: A view of the issues. *Journal of Applied Developmental Psychology*, *30*, 567–584. doi: 10.1016/j.appdev.2009.07.002

Lewin, K. (1931). The conflict between Aristotelian and Galileian modes of thought in contemporary psychology. *Journal of General Psychology*, *5*, 141–177. doi: 10.1080/00221309.1931.9918387

Lewin, K. (1936). *Principles of topological psychology*. New York, NY: McGraw-Hill. doi: 10.1037/10019-000

Lewin, K. (1939). Field theory and experiment in social psychology: Concepts and methods. *American Journal of Sociology*, *44*, 868–897. doi: 10.1086/218177

Lewin, K., Lippitt, R., & White, R. K. (1939). Patterns of aggressive behavior in experimentally created "social climates." *Journal of Social Psychology*, *10*, 271–299. doi: 10.1080/00224545.1939.9713366

Lienert, G. A., & Krauth, J. (1975). Configural frequency analysis as a statistical tool for defining types. *Educational and Psychological Measurement*, *35*, 231–238. doi: 10.1177/001316447503500201

Loevinger, J., & Wessler, R. (1970). *Measuring ego development: Vol. 1. Construction and use of a sentence completion test*. San Francisco, CA: Jossey-Bass.

Lundh, L. G., Saboonchi, F., & Wangby, M. (2008). The role of personal standards in clinically significant perfectionism. A person-oriented approach to the study of patterns of perfectionism. *Cognitive Therapy and Research*, *32*(3), 333–350. doi: 10.1007/s10608-006-9109-7

Magnusson, D. (1985). Implications of an interactional paradigm for research on human development. *International Journal of Behavioral Development*, *8*, 115–137. doi: 10.1177/016502548500800201

Magnusson, D. (1988). *Individual development from an interactional perspective: A longitudinal study*. Hillsdale, NJ: Erlbaum.

Magnusson, D. (1995). Individual development: A holistic, integrated model. In P. Moen, G. H. Elder, & K. Luscher (Eds.), *Examining lives in context* (pp. 19–60). Washington, DC: American Psychological Association. doi: 10.1037/10176-001

Magnusson, D. (2000). The individual as the organizing principle in psychological inquiry: A holistic approach. In L. R. Bergman, R. B. Cairns, L.-G. Nilsson, & L. Nystedt (Eds.), *Developmental science and the holistic approach* (pp. 33–47). Mahwah, NJ: Erlbaum.

Magnusson, D., & Törestad, B. (1993). A holistic view of personality: A model revisited. *Annual Review of Psychology*, *44*, 427–452. doi: 10.1146/annurev.ps.44.020193.002235

Mair, P., & von Eye, A. (2007). Application scenarios for nonstandard log-linear models. *Psychological Methods*, *12*, 139–156. doi: 10.1037/1082-989X.12.2.139

Mandara, J. (2003). The typological approach in child and family psychology: A review of theory, methods, and research. *Clinical Child and Family Psychology Review*, *6*, 129–146. doi: 10.1023/A:1023734627624

Martinez-Torteya, C., Bogat, G. A., von Eye, A., & Levendosky, A. A. (2009). Resilience among children exposed to domestic violence: The role of protective and vulnerability factors. *Child Development*, *80*, 562–577. doi: 10.1111/j.1467-8624.2009.01279.x

Maughan, B. (2005). Developmental trajectory modeling: A view from developmental psychopathology. *The ANNALS of the American Academy of Political and Social Science*, *602*, 118–130. doi: 10.1177/0002716205281067

McAdams, D. P., & Walden, K. (2010). Jack Block, the Big Five, and personality from the standpoints of actor, agent, and author. *Psychological Inquiry*, *21*, 50–56. doi: 10.1080/10478401003648740

McArdle, J. J. (2012). Foundational issues in contemporary modeling. In B. Laursen, T. D. Little, & N. A. Card (Eds.), *Handbook of developmental research methods* (pp. 385–410). New York, NY: Guilford.

McClelland, D. C. (1951). *Personality*. New York, NY: Holt. doi: 10.1037/10790-000

McCrae, R. R., & Alik, J. (Eds.). (2002). *The five-factor model of personality across cultures*. New York, NY: Kluwer. doi: 10.1007/978-1-4615-0763-5

McCullagh, P., & Nelder, J. A. (1989). *Generalized linear models* (2nd ed.). London, UK: Chapman and Hall. doi: 10.1007/978-1-4899-3242-6

Meehl, P. E. (1950). Configural scoring. *Journal of Consulting Psychology*, *14*, 165–171. doi: 10.1037/h0058049

Meiser, T. (1996). Loglinear Rasch models for the analysis of stability and change. *Psychometrika*, *61*, 629–645. doi: 10.1007/BF02294040

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*, 25–45. doi: 10.1037/0033-2909.133.1.25

Molenaar, P. C. M. (1997). Time series analysis and its relationship with longitudinal analysis. *International Journal of Sports Medicine*, *18*, 232–237. doi: 10.1055/s-2007-972720

Molenaar, P. C. M. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement: Interdisciplinary Research and Perspectives*, *2*, 201–218. doi: 10.1207/s15366359mea0204_1

Molenaar, P. C. M. (2007). On the implications of the classical ergodic theorems: Analysis of developmental processes has to focus on intra-individual variation. *Developmental Psychobiology*, *50*, 60–69. doi: 10.1002/dev.20262

Molenaar, P. C. M. (2010). Testing all six person-oriented principles in dynamic factor analysis. *Development and Psychopathology*, *22*, 255–259. doi: 10.1017/S0954579410000027

Molenaar, P. C. M., Huizenga, H. M., & Nesselroade, J. R. (2003). The relationship between the structure of interindividual and intraindividual variability: A theoretical and empirical vindication of developmental systems theory. In U. M. Staudinger & U. Lindenberger (Eds.), *Understanding human development: Dialogues with lifespan psychology* (pp. 339–360). Dordrecht: Kluwer Academic Publishers. doi: 10.1007/978-1-4615-0357-6_15

Molenaar, P. C. M., & Nesselroade, J. R. (1998). A comparison of pseudo-maximum likelihood and asymptotically distribution-free dynamic factor analysis parameter estimation in fitting covariance-structure models to block-Toeplitz matrices representing single-subject multivariate time-series. *Multivariate Behavioral Research*, *33*, 313–342. doi: 10.1207/s15327906mbr3303_1

Molenaar, P. C. M., & Newell, K. M. (Eds.) (2010). *Individual pathways of change: Statistical models for analyzing learning and development*. Washington, DC: American Psychological Association. doi: 10.1037/12140-000

Molenaar, P. C. M., & Rovine, M. (2015). Intraindividual analysis. In D. Cicchetti (Ed.), *Developmental psychopathology* (3rd ed.). New York, NY: Wiley.

Molenaar, P. C. M., Sinclair, K. O., Rovine, M. J., Ram, N., & Corneal, S. E. (2009). Analyzing developmental processes on an individual level using nonstationary time series modeling. *Developmental Psychology*, *45*, 260–271. doi: 10.1037/a0014170

Molenaar, P. C. M., & von Eye, A. (1994). On the arbitrary nature of latent variables. In A. von Eye & C. C. Clogg (Eds.), *Latent variables analysis — Applications for developmental research* (pp. 226–242). Newbury Park, CA: Sage.

Morey, L. C., & Hopwood, C. J. (2009). An IRT-based measure of alcohol trait severity and the role of traitedness in trait validity: A reanalysis of Project MATCH data. *Drug and Alcohol Dependence*, *105*, 177–184. doi: 10.1016/j.drugalcdep.2009.06.025

Mun, E. Y., von Eye, A., Bates, M. E., & Vaschillo, E. (2008). Finding groups using model-based cluster analysis: Heterogenous emotional self-regulatory processes and heavy alcohol use risk. *Developmental Psychology*, *44*, 481–495. doi: 10.1037/0012-1649.44.2.481

Muthén, B. (2000). Latent variable mixture modeling. In G. A. Marcoulides & R. E. Schumacker (Eds.), *Advanced structural equation modeling: New developments and techniques* (pp. 1–33). Mahwah, NJ: Erlbaum.

Muthén, B. (2001). Second-generation structural equation modeling with a combination of categorical and continuous latent variables: New opportunities for latent class-latent growth modeling. In L. M. Collins, & A. G. Sayer (Eds.), *New methods for the analysis of change* (pp. 291—322). Washington, DC: American Psychological Association. doi: 10.1037/10409-010

Muthén, B. O., & Muthén, L. M. (2000). Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcoholism: Clinical and Experimental Research*, *24*, 882–891. doi: 10.1111/j.1530-0277.2000.tb02070.x

Muthén, L. M., & Muthén, B. O. (1998–2012). *Mplus: User's guide* (7th ed.). Los Angeles, CA: Muthén & Muthén.

Nagin, D. S. (1999). Analyzing developmental trajectories: A semiparametric, group-based approach. *Psychological Methods*, *4*, 139–157. doi: 10.1037/1082-989X.4.2.139

National Institute of Justice. (2011). *Batterer intervention programs often do not change offender behavior*. Retrieved from http://www.nij.gov/topics/crime/intimate-partner-violence/interventions/Pages/batterer-intervention.aspx#noteReferrer2

National Institute of Mental Health. (2011). NIMH Research Domain Criteria (RDoC). http://www.nimh.nih.gov/research-funding/rdoc/nimh-research-domain-criteria-rdoc.shtml

Nigg, J. T. (2006). Temperament and development psychopathology. *Journal of Child Psychology and Psychiatry*, *47*, 395–422. doi: 10.1111/j.1469-7610.2006.01612.x

Nesselroade, J. R., & Molenaar, P. C. M. (2010). When persons should be few and occasions should be many—Modeling a different kind of longitudinal data. *International Society for the Study of Behavioral Development Bulletin. Serial No. 57*, 2–4.

Oakes, J. M. (2009). Commentary: individual, ecological, and multilevel fallacies. *International Journal of Epidemiology*, *38*, 361–368. doi: 10.1093/ije/dyn356

Perrine, M. W. B. (1995). *Daily self-reported drinking: Longitudinal patterns*. NIAAA grant AA09684.Vermont Alcohol Research Center, Essex, VT.

Ram, N., & Nesselroade, J. R. (2007). Modeling intraindividual and intracontextual change: Rendering developmental contextualism operational. In T. D. Little, J. A. Bovaird, & N. A. Card (Eds.), *Modeling contextual effects in longitudinal studies* (pp. 325–342). Mahwah, NJ: Erlbaum.

Ramsay, D., & Lewis, M. (2003). Reactivity and regulation in cortisol and behavioral responses to stress. *Child Development*, *74*, 456–464. doi: 10.1111/1467-8624.7402009

Rand, W. M. (1971). Objective criteria for the evaluation of clustering methods. *Journal of the American Statistical Association*, *66*, 846–850. doi: 10.1080/01621459.1971.10482356

Robins, R. W., John, O. P., Caspi, A., Moffitt, T. E., & Stouthamer-Loeber, M. (1996). Resilient, overcontrolled, and undercontrolled boys. Three replicable personality types. *Journal of Personality and Social Psychology*, *70*, 157–171. doi: 10.1037/0022-3514.70.1.157

Robinson, W. S. (1950). Ecological correlations and the behavior of individuals. *American Sociological Review*, *15*, 351–357. doi: 10.2307/2087176

Roeder, K., Lynch, K., & Nagin, D. (1999). Modeling uncertainty in latent class membership: A case study in criminology. *Journal*

of the American Statistical Association, *94*, 766–776. doi: 10.1080/01621459.1999.10474179

Rogers, C. R. (1961). *On becoming a person: A therapist's view of psychotherapy*. Boston, MA: Houghton Mifflin.

Rost, J., & Langeheine, R. (Eds.) (1997). *Applications of latent trait and latent class models in the social sciences*. Münster, Germany: Waxmann.

Rovine, M. J., Molenaar, P. C. M., & Corneal, S. E. (1999). Analysis of emotional response patterns for adolescent stepsons using P-technique factor analysis. In R. K. Silbereisen & A. von Eye (Eds.), *Growing up in times of social change* (pp. 261–285). Berlin, Germany: De Gruyter. doi: 10.1515/9783110810110.261

Sampson, R. J., & Laub, J. H. (2005). A life-course view of the development of crime. *ANNALS of the American Academy of Political and Social Science*, *602*, 12–45. doi: 10.1177/0002716205280075

Sampson, R. J., Laub, J. H., & Wimer, C. (2006). Does marriage reduce crime? A counterfactual approach to within-individual causal effects. *Criminology*, *44*, 465–508. doi: 10.1111/j.1745-9125.2006.00055.x

Schmidt, H. D. (1977). Methodologische Probleme der entwicklungspsychologischen forschung. *Probleme und Ergebnisse der Psychologie*, *62*, 5–27.

Schmitz, B. (2000). Auf der Suche nach dem verlorenen Individuum: Vier Theoreme zur Aggregation von Prozessen [Searching for the lost individual: Four theorems on the aggregation of processes.] *Psychologische Rundschau*, *51*, 83–92. doi: 10.1026//0033-3042.51.2.83

Selvin, H. C. (1958). Durkheim's suicide and problems of empirical research. *American Journal of Sociology*, *63*, 607–619. doi: 10.1086/222356

Sjoquist, E. S., Almqvist, L., Asenlof, P., Lampa, J., Opava, C. H., & Para Study, G. (2010). Physical-activity coaching and health status in rheumatoid arthritis: A person-oriented approach. *Disability and Rehabilitation*, *32*(10), 816–825. doi: 10.3109/09638280903314069

Stam, H. J. (2010). The tradition of personalism and its relationship to contemporary indeterminate functionalism. *New Ideas in Psychology*, *28*, 143–150. doi: 10.1016/j.newideapsych.2009.02.004

Steinley, D. (2004). Properties of the Hubert-Arabie adjusted Rand index. *Psychological Methods*, *9*, 386–396. doi: 10.1037/1082-989X.9.3.386

Stephenson, W. (1935). Correlating persons instead of tests. *Character and Personality*, *6*, 17–24. doi: 10.1111/j.1467-6494.1935.tb02022.x

Stephenson, W. (1953). *The study of behavior: Q technique and its methodology*. Chicago, IL: University of Chicago Press.

Sterba, S. K., & Bauer, D. J. (2010). Matching method with theory in person-oriented developmental psychopathology research. *Development and Psychopathology*, *22*, 239–254. doi: 10.1017/S0954579410000015

Stern, W. (1900). *Über Psychologie der individuellen Differenzen* (Ideen zu einer 'differentiellen Psychologie'). Leipzig, Germany: J.A. Barth.

Stern, W. (1911). *Die differentielle Psychologie in ihren methodischen Grundlagen*. Leipzig, Germany: J.A. Barth.

Stern, W. (1917/2010). Psychology and personalism (Trans. J. T. Lamiell). *New Ideas in Psychology*, *28*, 110–134. doi: 10.1016/j.newideapsych.2009.02.005

Strupp, H. H., & Bergin, A. E. (1969). Some empirical and conceptual bases for coordinated research in psychotherapy. *International Journal of Psychiatry*, *7*, 18–90.

Subramanian, S. V., Jones, K., Kaddour, A., & Krieger, N. (2009). Revisiting Robinson: The perils of individualistic and ecologic fallacy. *International Journal of Epidemiology*, *38*, 342–360. doi: 10.1093/ije/dyn359

Susser, M. (1994). The logic in the ecological. *American Journal of Public Health*, *84*, 825–835. doi: 10.2105/AJPH.84.5.825

Toomela, A. (2009). How methodology became a toolbox—and how it escapes from that box. In J. Valsiner, P. C. M. Molenaar, M. C. D. P. Lyra, & N. Chaudhary (Eds.), *Dynamic process methodology in the social and developmental sciences* (pp. 45–66). New York, NY: Springer. doi: 10.1007/978-0-387-95922-1_3

Towe-Goodman, N. R., Stifter, C. A., Mills-Koonce, W. R., Granger, D. A., & The Family Life Project Key Investigators. (2012). Interparental aggression and infant patterns of adrenocortical and behavioral stress responses. *Developmental Psychobiology*, *54*, 685–699. doi: 10.1002/dev.20618

Valsiner, J., Molenaar, P. C. M., Lyra, M. C. D. P., & Chaudhary, N. (Eds.), *Dynamic process methodology in the social and developmental sciences* (pp. 31–43). New York, NY: Springer.

van Rijn, P. W. (2008). *Categorical time series in psychological measurement*. Dissertation at the Faculty of Social and Behavioural Sciences of the University of Amsterdam. Retrieved from http://dare.uva.nl/document/104220

van Rijn, P. W., & Molenaar, P. C. M. (2005). Logistic models for single-subject time series. In L. A. van der Ark, M. A. Croon, & K. Sijtsma (Eds.), *New developments in categorical data analysis for the social and behavioral sciences* (pp. 125–146). London, UK: Erlbaum.

Vassend, O., & Skrondal, A. (2011). The NEO personality inventory revised (NEO-PI-R): Exploring the measurement structure and variants of the five-factor model. *Personality and Individual Differences*, *50*, 1300–1304. doi: 10.1016/j.paid.2011.03.002

Vedeler, D., & Garvey, A. P. (2009). Dynamic methodology in infancy research. In J. Valsiner, P. C. M. Molenaar, M. C. D. P. Lyra, & N. Chaudhary (Eds.), *Dynamic process methodology in the social and developmental sciences* (pp. 431–453). New York, NY: Springer. doi: 10.1007/978-0-387-95922-1_19

Vermunt, J. K., & Magidson, J. (2004). *Structural equation models: Mixture models*. Retrieved from http://statisticalinnovations.com/technicalsupport/2005c.pdf

Volkovich, Z., Avros, R., & Golani, M. (2011). A comparative approach to cluster validation. *Journal of Pattern Recognition Research*, *2*, 230–243. doi: 10.13176/11.208

von Eye, A. (2002). *Configural frequency analysis—Methods, models, and applications*. Mahwah, NJ: Erlbaum.

von Eye, A. (2010). Developing the person-oriented approach—Theory and methods of analysis. *Development and Psychopathology*, *22*, 277–285. doi: 10.1017/S0954579410000052

von Eye, A., & Bergman, L. R. (2003). Research strategies in developmental psychopathology: Dimensional identity and the person-oriented approach. *Development and Psychopathology*, *15*, 553–580. doi: 10.1017/S0954579403000294

von Eye, A., Bergman, L. R., & Hsieh, C. A. (2015). Person-oriented approaches. In W. Overton & P. C. M. Molenaar (Eds.), *Handbook of child development: Theory and methods* (Vol. 1, pp. 789–841). New York, NY: Wiley.

von Eye, A., & Bogat, G. A. (2005). Identifying sectors of deviations from multinormality. In K. Anaya Izquierdo, A. Contreras Cristan, A. Dominguez Molina, & E. Estrada Barragan (Eds.), *Memorias del XIX Foro Nacional de Estadistica* (pp. 201–207). Aguascalientes, Mexico: Instituto Nacional de Estadistica, Geografia e Informatica.

von Eye, A., & Bogat, G. A. (2006). Person-oriented and variable-oriented research: Concepts, results, and development. *Merrill-Palmer Quarterly*, *52*, 390–420. doi: 10.1353/mpq.2006.0032

von Eye, A., & Bogat, G. A. (2007). Methods of data analysis in person-oriented research. The sample case of ANOVA. In A. Ittel, L. Stecher, H. Merkens, & J. Zinnecker (Eds.), *Jahrbuch Jugendforschung 2006* (pp. 161–182). Wiesbaden: Verlag fur Sozialwissenschaften.

von Eye, A., Bogat, G. A., & Rhodes, J. (2006). Variable-oriented and person-oriented perspectives of analysis: The example of alcohol consumption in adolescence. *Journal of Adolescence*, *29*(6), 981–1004. doi: 10.1016/j.adolescence.2006.06.007

von Eye, A., & Gardiner, J. C. (2004). Locating deviations from multivariate normality. *Understanding Statistics*, *3*, 313–331.

von Eye, A., & Gutierrez-Pena, E. (2004). Configural frequency analysis: The search for extreme cells. *Journal of Applied Statistics*, *31*, 981–997. doi: 10.1080/0266476042000270545

von Eye, A., Mair, P., & Bogat, G. A. (2005). Prediction models for configural frequency analysis. *Psychology Science*, *47*, 342–355.

von Eye, A., & Mun, E. Y. (2013). *Log-linear modeling—Concepts, interpretation and applications*. New York, NY: Wiley.

von Eye, A., Mun, E. Y., & Indurkhya, A. (2004). Classifying developmental trajectories—A decision making perspective. *Psychology Science*, *46*, 65–98.

von Eye, A., & Wirsing, M. (1978). An attempt for a mathematical foundation and evaluation of MACS, a method for multidimensional automatical cluster detection. *Biometrical Journal*, *20*, 655–666.

von Eye, A., & Wirsing, M. (1980). Cluster search by enveloping space density maxima. In M. M. Barritt & D. Wishart (Eds.), *Compstat 1980, Proceedings in Computational Statistics* (pp. 447–453). Vienna, Austria: Physica.

Walls, T. A., & Schafer, J. L. (Eds.). (2006). *Models for intensive longitudinal data*. Oxford, UK: Oxford University Press. doi: 10.1093/acprof:oso/9780195173444.001.0001

Warrens, M. J. (2008). On the equivalence of Cohen's Kappa and the Hubert-Arabie Adjusted Rand Index. *Journal of Classification*, *183*, 177–183. doi: 10.1007/s00357-008-9023-7

Widaman, K. F. (2007). Intrauterine environment affects infant and child outcomes: Environment as direct effect. In T. D. Little, J. A. Bovaird, & N. A. Card (Eds.), *Modeling contextual effects in longitudinal studies* (pp. 387–436). Mahwah, NJ: Erlbaum.

Wishart, D. (1970). *The treatment of various similarity criteria in relation to Clustan 1A*. University of St. Andrews, St. Andrews, Scotland.

Witherington, D. C. (2007). The dynamic systems approach as metatheory for developmental psychology. *Human Development*, *50*, 127–153. doi: 10.1159/000100943

Zeanah, C. H., Benoit, D., Hirshberg, L., Barton, M. L., & Regan, C. (1994). Mothers' representations of their infants are concordant with infant attachment classifications. *Developmental Issues in Psychiatry and Psychology*, *1*, 9–18.

CHAPTER 19

Person-Specific Approaches to the Modeling of Intraindividual Variation in Developmental Psychopathology

MICHAEL J. ROVINE and PETER C. M. MOLENAAR

INTRODUCTION 846
Definition of Intraindividual Variation 848
Contrasting Intraindividual Variation with Interindividual Variation 848
MODELING INTERINDIVIDUAL VARIATION THROUGH GROUP-BASED INDIVIDUAL DIFFERENCE MODELS 849
The Multilevel Growth Curve Model 850
The Linear Latent Growth Curve Model 851
The Multilevel Autoregressive Model 852
Lagged Regression Models 852
The Vector Autoregression Model 853
THE DATA BOX AND FACTOR ANALYSIS 854
Factor Models for Longitudinal Data 855
Person-Oriented Approaches 857
***P-Technique* Factor Models** 857
The Dynamic Factor Model 857
DATA EXAMPLES 858
Data Example 1: The Borkenau and Ostendorf Data Set 858
Data Example 2: PANAS Data 858
DISCUSSION 861
FUTURE DIRECTIONS 862
REFERENCES 862

INTRODUCTION

Developmental psychopathology constitutes the confluence of two important research traditions that both are undergoing major methodological changes—developmental science and psychopathology. Developmental science is increasingly based on the metamodel of developmental systems theory (Ford & Lerner, 1992). Developmental systems theory conceives of development as the result of multiple coacting influences that (1) are context sensitive and contingent (e.g., Gottlieb, 2001), (2) affect constructive processes in which nonlinear epigenetic influences play central roles (e.g., Lickliter & Honeycutt, 2009), and (3) evolve at multiple time scales and at multiple levels (e.g., Smith & Thelen, 2003). Accordingly, the emphasis in developmental systems theory is on the inherently stochastic and person-specific nature of developmental processes. Applications of developmental systems theory therefore require methodological approaches in which the focus is on person-specific methods (Molenaar, Lerner, & Newell, 2014; Molenaar & Newell, 2010).

The medical sciences also are undergoing major methodological changes under the banner of personalized medicine (Hamburg & Collins, 2010). While the initial approaches to personalized medicine were heavily based on genotypic characterization of patients, this now has been generalized to include intensive longitudinal assessments of phenotypic features such as physiology, behavior, etc. (Schwarz & Collins, 2007). The availability of such intensive longitudinal assessments opens up the possibility to optimize therapeutic treatment by means of new methods derived from computational engineering. In these so-called dynamic treatment methods the sequence of therapeutic manipulations is increasingly optimized in real time by means of machine learning techniques conditional on the newly available longitudinal assessments (e.g., Chakraborty & Moodie, 2013).

The trend toward personalized and person-specific methodology also is corroborated by a recent development in the foundations of psychometrics. Using general theorems of mathematical ergodic theory (Birkhoff, 1931), Molenaar (2004) proved that pooling across individuals,

[1]Color versions of Figure 19.3 are available at http://onlinelibrary.wiley.com/book/10.1002/9781119125556

which is the hallmark of standard statistical methods in the analysis of psychological data, only is warranted if strong so-called ergodicty criteria are met. The criteria that define *ergodicity* imply that (1) all individuals making up a population (i.e., domain of generalization) should obey exactly the same dynamic model and (2) the dynamic model should be stationary. The latter stationarity criterion implies that the process under consideration should lack any developmental features (e.g., constant mean, constant variance, constant sequential dependencies). In case one or both of these criteria are violated, the statistical analysis should be based on person-specific approaches involving intensive measurements of individual subjects. Developmental process almost by definition violates the stationarity criterion and therefore should be investigated using person-specific methods.

In an important publication Sterba & Bauer (2010) scrutinized the match between person-oriented methods and person-oriented theory. They showed that person-specific methods like dynamic factor analysis (i.e., factor analysis of intensive longitudinal assessments obtained in single-subject or replicated time-series designs) provide the best match with person-oriented principles (see also Molenaar, 2010a). Accordingly, person-oriented principles should preferably be applied using person-specific methods.

Because person-specific methodology is relatively new (in contrast to other fields of research where time-series analysis often is the standard approach to data analysis, such as for instance physics, engineering, and econometrics), we highlight in what follows differences between standard developmental methods based on analysis of interindividual variation with person-specific methods based on intraindividual variation. In particular, we will argue that the individual differences created within standard statistical developmental methods such as latent growth curve modeling violate in important respects the tenets of person-specific methodology. This implies that these standard statistical methods based on interindividual variation cannot be used to accommodate violations of ergodicity and also may have limited applicability in personalized and person-oriented approaches (Molenaar & Campbell, 2009; Voelkle, et al., 2014).

Here, we suggest applying these approaches to the field of developmental psychopathology, In summarizing the field as part of the recognition of the fiftieth anniversary of the *Journal of Child Psychology and Psychiatry*, Cicchetti and Toth (2009) indicated the importance of an approach to research in developmental psychopathology that considered all methods that allowed the description of process that typically unfolds over time. They emphasized the importance of multiple levels of analysis to achieve this goal. An important level of analysis that has received less attention in the developmental psychopathology literature is the person-specific approach based on single subject models. Given their notion that there are "multiple pathways to similar manifest outcomes and that there are different outcomes [resulting from] the same pathway," (page 4) typical prediction models based on group characteristics may be lacking when the requirement would be a prediction that can have two different outcomes given identical conditions. Given that treatment may be suggested by the prediction, the person-specific single subject approach which would concentrate more on individual characteristics may be the better bet.

Add to this that there has been an increasing tendency in the field toward the notion of person-centered care (Adams & Grieder, 2005). The idea behind this is simple. Treatments developed based on sample characteristics and applied to the individual may not appropriately address that individual's needs. A treatment regimen tailored on the individual's unique requirements may produce better results. This is akin to developing a person-specific model. Molenaar (1987; Molenaar, 2010b) has shown how this modeling approach can be successful in the therapeutic situation.

There has also been a greater emphasis on shared decision making in the therapeutic setting (Adams & Drake, 2006; Deegan et al., 2008; Ptasznik, 2011). These tendencies suggest that the person-specific, single-subject modeling approach may be an important new tool for moving the field of developmental psychopathology forward.

As Cicchetti and Toth (2009) described the type of developmental analysis we suggest, it "presupposes change and novelty, highlights the critical role of timing in the organization of behavior, underscores multiple determinants, and cautions against expecting invariant relations between causes and outcomes" (p. 1). We extend this idea of expected invariance to include individual models that can be used to achieve optimal therapeutic outcomes.

In this chapter we would like to first demonstrate the way certain common analytic approaches such as longitudinal regression modeling, linear mixed modeling (e.g., growth curve and repeated measures one-way analysis of variance [ANOVA]), multilevel autoregressive modeling, factor modeling, and cluster analysis create individual difference measures based on a common underlying model. In this sense, they are modeling interindividual variation. After showing that these approaches depend on sufficient (summary) statistics and creating individual parameter predictions based on an underlying assumed probability model, we will then compare these methods

to more person-specific approaches. We will focus on analogous single subject time-series approaches (e.g., vector autoregressive approaches for cross-lagged regression, P-technique and dynamic factor models for cross sectional or longitudinal factor models based on relatively few occasions) and show how these person-specific methods create parameters based on models that can vary from individual to individual. We will demonstrate a true intraindividual approach using personality data collected by Borkenau and Ostendorf (1996) and emotional mood data from Corneal and Nesselroade (1994; see also Molenaar et al., 2009; Rovine, Molenaar, & Corneal, 1998). We first define the difference between the concepts of intra- and interindividual differences.

Definition of Intraindividual Variation

In its most basic form intraindividual variation is variation in time of repeated measurements obtained with a single subject. We use the term *subject* for the single entity (case) that is measured repeatedly, but we understand that this entity also can be a single group of subjects like a particular mother–infant dyad, a single family, or a school. The important point to note is that here, subject is fixed; that is, it is not considered to be a randomly drawn from some population of subjects. Even though the designation for each subject as a system or dynamic system would be technically more appropriate, as psychologists we select subject.

Intraindividual variation is understood to be random variation associated with a *stochastic process* which is thought to describe the evolution of some system over time. Here, the system is a single subject and the process is measured by a set of repeated measurements indexed by time. The general idea of a stochastic process was introduced by Kolmogorov (1931) and can be illustrated as follows: suppose the cognitive performance of a single subject on the same test is measured at T consecutive occasions. According to Kolmogorov's definition of a stochastic process, the actual score observed on each occasion t, say $t = 1$, constitutes a random draw from a probability distribution of possible scores that could have been obtained at $t = 1$ under the same circumstances. This allows the consideration of the mean of each time series. Moreover, each pair of scores, say at $t = 1$ and $t = 2$, is considered to be a random draw from a bivariate probability distribution. This allows the consideration of the correlation or covariance between each pair of occasions. Together the means, variances, correlations, and higher order moments of this each series can be modeled using methods typically called time-series analysis.

Time can be continuous or discrete. In this chapter we consider only discrete time processes. To simplify the presentation we suggest that the time interval between consecutive occasions is constant, although this assumption can be dropped when discussing data obtained in, for example, ecological momentary assessment (EMA) designs. By emphasizing stochastic processes we are ruling out deterministic process; for example, those that follow a predetermined shape or trajectory caused such as growth processes. Such processes can be described by deterministic differential or difference equations and would typically be modeled using a different set of methods (Banks, 1983).

Contrasting Intraindividual Variation with Interindividual Variation

The converse of intraindividual variation is interindividual variation. These two contrasting types of variation can be distinguished in the following way. In analysis of intraindividual variation, we model each subject separately treating the subject as fixed while the estimation proceeds by pooling across the replicated measurements obtained with this subject. Because its measurements are time-dependent, the hallmark of analysis of intraindividual variation is representing by pooling across repeated occasions. The finite set of observations for each subject can be conceived of as a sample from all possible times. As a consequence, the results of a standard analysis of intraindividual variation can be generalized to all times previous to the observation interval (retrodiction), to times within the observation interval for which no observations are available (interpolation), and to future times (prediction). Hence, the domain of generalization of a pure analysis of intraindividual variation is the time domain, while no statements regarding the generalization across subjects can initially be made.

In contrast, in analysis of interindividual variation the sample of subjects is conceived of as randomly drawn from a population of subjects. Estimation proceeds by pooling data across the sample of subjects. The hallmark of analysis of interindividual variation is pooling across subjects. As a consequence, the results obtained in an analysis of interindividual variation are generalized to the population of subjects. This is the typical analytical situation in longitudinal designs. Again, analysis of interindividual variation of these repeated measures proceeds by pooling across subjects. For instance, as we will show, the standard latent growth curve model is fitted by pooling across subjects. The result of this is that any longitudinal analysis is not analysis of intraindividual variation but instead is analysis of interindividual variation.

The difference in the definitions of inter- and intraindividual variation suggests how the results of a particular analysis can be generalized (generalization across populations of subjects versus generalization across time). We note

that one of the dangers of forgetting this distinction is that inappropriate generalizations are often made based on the results of analyses based on interindividual variation. We will emphasize these below.

MODELING INTERINDIVIDUAL VARIATION THROUGH GROUP-BASED INDIVIDUAL DIFFERENCE MODELS

Many popular analytical methods create values for what are described as individual difference parameters that are based on group models. For example, the linear multilevel (or latent) growth curve model (Bryk & Raudenbush, 1992; Goldstein, 1995; Grimm, Davoudzadeh, & Ram, 2014; McArdle & Epstein, 1987) can be used to generate individual intercepts and slopes in addition to the group intercept and slope that are typically the focus of the model. While the individual measures can be used to describe differences among participants in a study, they are created based on the group parameter estimates (e.g., regression weights, common covariance structure of the residuals). The individual variable values are then predicted using these group parameter values based on certain assumptions regarding the distribution of these variables (e.g., the individual values will be normally distributed with a zero mean). This approach represents the assumption that the same model holds for all study participants. With a relatively few number of occasions of measurement, this assumption cannot be tested.[2] With more intensive time-series data, however, this assumption can be tested, and individual models can be determined. It may be the case that different individuals require different models. There may be unknown subgroups whose data may be described by a common model, but this is not known a priori. With more intensive time-series data, a separate model can be determined for each study participant. Individuals with common models can then be clustered to form subgroups (Gonzales & Ferrer, 2014; Nesselroade & Molenaar, 1999).

One common model where this can become evident is the longitudinal lagged regression model based on a panel design. Here, we will compare how the group-based model relates to the person-specific vector autoregressive model.

A second family of analytic procedures that we will consider encompasses methods based on the linear mixed model, which include multilevel growth curve modeling, repeated measures ANOVA, and multilevel autoregressive models. As an example, for the linear multilevel growth curve model, individual slopes and intercepts are generated as part of the solution. These are often interpreted as individual difference measures. They are based on post hoc predictions of the model and are common to all participants in the sense that they represent variations in the parameters of a single underlying model under the assumption of a particular probability distribution for the residuals (Robinson, 1991). The common underlying model makes these individual difference measures much different than those that would result from a separate time-series model for each individual.

Another model we consider is the longitudinal factor model. With a few occasions of measurement investigators can test whether a factor structure is invariant across occasions and the degree of invariance that can be assumed. But this factor model is the result of a *subjects* × *variables* data set where the variables for the different occasions are the variables of the model. As an alternative we will consider a factor analysis based on collecting multiple *variables* on multiple *occasions* on a single *subject*. Here, *occasions* essentially takes the place of *subjects*. A correlation (or covariance) matrix can be constructed which can be used to estimate individual factor models. The factor loadings and factor correlations of these different individual models can be compared both to each other and to results of the group-based *subjects* × *variables* as we show for the data presented by Borkenau and Ostendorf (1998) and Corneal and Nesselroade (1991; Molenaar et al., 2009; Rovine, Molenaar, & Corneal, 1998).

Other modeling approaches attempt to account for heterogeneity of intraindividual variation in creating individual difference parameters. Latent class trajectory analysis (Nagin, 1999) and growth mixture modeling (Muthén & Sheden, 1999) create subgroups by implicitly clustering individuals with similar predicted parameter values. Although these methods allow heterogeneity in terms of differences in the predicted parameter values, there will still be strong distribution assumptions within subgroups (Sterba & Bauer, 2010). For example, a particular subgroup within a growth mixture model will have a specified mean and covariance structure that assumes homogeneity within that subgroup. These types of models still require a common group model of which the individual parameters represent instances of that model though with wider variation across subgroups. By requiring the common model, these approaches still describe interindividual variability.

Multilevel autoregressive (AR) models use an autoregressive model as the Level 1 (time-related level) model. The Level 2 or individual-level model allows covariates that are invariant across occasions to account for variability in the Level 1 parameters. However, this results in a set

[2] Unless mixture modeling is used, but then the same assumptions reapply in each of the finite number of subpopulations.

of fixed effect parameters that are added to the overall or group level equation again resulting in a common model for all individuals. As in the multilevel growth curve model, individual AR parameters are predicted post hoc under the strict assumptions that the same AR process (e.g., AR(1), AR(2)) holds for all individuals, and the individual parameter values deviate from the group parameter values by residual values that conform to a multivariate normal distribution. If the order of the process is not identical for individuals, or if the order of the process is the same, but the true distribution of the parameter does not follow a strict normal distribution, this approach represents a misspecification of the model.

The person-oriented literature (Bergman, 2012; Bergman & Andersson, 2010) often refers to a cluster analytic approach to time-series data. Given the strong similarity between many clustering methods and standard factor analysis, such person-oriented approaches are often really describing interindividual differences.

The Multilevel Growth Curve Model

The linear growth curve model (Bryk & Raudenbush, 1992; Goldstein, 1995; McArdle & Epstein, 1987) is member of the linear mixed model family (Laird & Ware, 1982).

A simple linear growth curve model with a time-invariant covariate x can be written as

$$y_{it} = \beta_0 + \beta_1 TIME + \beta_2 x_i + \beta_3 (x * TIME) + \upsilon_{i0} + \upsilon_{i1} TIME + r_{it} \quad (19.1)$$

where $\beta_0, \beta_1, \beta_2$, and β_3 are fixed effects regression coefficients, and $\upsilon_{i0}, \upsilon_{i1}$, and r_{it} are the random effects which are the deviation of the individual intercept around the group intercept, the deviation of the individual slope around the group slope, and the deviations of the individual observed variable values around their own regression line. As in all linear mixed models we estimate the expected values of the fixed effects and the covariance matrix of the random effects. These estimates are based on data pooled across individuals. The covariance matrix of the random effects is given by

$$\sigma^2 V = \sigma^2 (ZDZ^T + I) \quad (19.2)$$

where σ^2 is the pooled variance of the individual residuals around the individual's regression (assumed equal across occasions for the typical multilevel linear growth curve model), Z is the set of design column vectors for the random intercept and slope parameters, and D is covariance matrix of the random intercepts and slopes. It is a convention to refer to the total covariance matrix of the random effects as $\sigma^2 V$, which defines V by factoring the residual variance out of the total covariance.

This covariance matrix along with the fixed effects can be used to predict values of the individual random effects under the assumptions of this single common model under a multivariate normal distribution (Henderson, 1975). The equation for best linear unbiased predictions (BLUPs) was given by Robinson (1991) as

$$\hat{\gamma}_i = DZ^T \hat{V}^{-1} (y_i - X\hat{\beta}) \quad (19.3)$$

where $\hat{\gamma}_i$ is the vector of predicted random effects (intercept and slope) for subject i, X is the design matrix for the fixed effect parameters (group intercept and slope), and β is the set of estimated fixed effect parameters.

These random effects are often described as individual difference measures, and in the sense that they vary across individuals, they are. This interpretation is the result, in part, of the multilevel formulation of the linear mixed model as presented by Goldstein (1995), and included in the formatting of software such as HLM (Bryk & Raudenbush, 1992). For example, the multilevel model version of the linear growth curve shown above can be rewritten as

Level 1:

$$y_{it} = \pi_{i0} + \pi_{i1} TIME + r_{it}$$

Level 2:

$$\pi_{i0} = \beta_{00} + \beta_{10} x_i + \upsilon_{i0}$$
$$\pi_{i1} = \beta_{01} + \beta_{11} x_i + \upsilon_{i1} \quad (19.4)$$

where in Level 1, π_{i0} is the individual's intercept, and π_{i1} is the individual's linear slope. These random effects comprise the individual equation. In Level 2 we included a covariate, x, that varies from individual to individual but is otherwise time invariant. Here the individual intercept that has been passed on from Level 1 deviates around the group intercept, β_{00}, by an amount, υ_{i0}, and the individual slope deviates from the group slope, β_{01}, by an amount, υ_{i1}. In both Level 2 equations, we included a covariate, which is purported to account for variability in the individual slopes and intercepts. Here, the random effects are r_{it}, υ_{i0}, and υ_{i1}. The fixed effects are the group intercept, β_{00}, the group slope, β_{01}, and the regression coefficient related to the covariate, β_{11}. For the linear mixed model, we estimate the expected values of the fixed effects, and the covariance matrix of the random effects.

Given the format of this multilevel model, it appears that a separate equation is estimated for each individual. This appearance is part of the reason why the model is mistaken for a true model of intraindividual differences. However, substituting Level 2 into Level 1, we get

$$\begin{aligned} y_{it} &= (\beta_{00} + \beta_{10}x_i + \upsilon_{i0}) + (\beta_{01} + \beta_{11}x_i + \upsilon_{i1})TIME + r_{it} \\ y_{it} &= \beta_{00} + \beta_{01}TIME + \beta_{10}x_i + \beta_{11}(x * TIME) \\ &\quad + (\upsilon_{i0} + \upsilon_{i1}TIME) + r_{it} \end{aligned} \tag{19.5}$$

which is identical to the linear mixed model equation presented above in which the individual difference equation has disappeared and is replaced by a single equation with fixed and random effects. It is this equation which is estimated in programs like SAS PROC MIXED, SAS MIXED, and R LME.

The random effects here and as estimated by programs such as HLM are the same regression residuals predicted under the same set of assumptions under a specific group-based regression model. The empirical Bayes estimates of HLM are identical to the solution of the Henderson linear model equations implemented by programs such as SAS PROC MIXED (Littell et al., 2006).

This model assumes that the same trajectory shape describes each individual, and individuals only differ in, for example, the steepness of the slope, or the height of the intercept. When a common model does not hold, the growth curve approach may misrepresent how individuals differ resulting in an inference that gives a less than optimal description about the course of change (Liu et al., 2012). Given the short longitudinal data that are most typically available in growth curve analysis, the possibility of testing to see whether the model adequately describes the sample is often not available. The person-specific analog to the growth curve model would require fitting a separate polynomial equation to each individual. The proper order (i.e., the parameters that can describe the shape of the curve) would be empirically determined for each individual. With most studies, these data are not available. When sufficient data to estimate the individual curve would be available, the estimate of a relatively low order polynomial would only make sense for certain simple true growth processes. Since the growth curve model is often used for modeling some arbitrary change process that does not follow a known growth function, the fitting of a growth curve to time-series data may not even make much sense. For a relatively few number of repeated occasions of measurement, the growth curve approach often represents a necessary practical compromise.

The Linear Latent Growth Curve Model

Latent curve models (Bollen & Curran, 2006; Meredith & Tisak, 1990) represent a very common approach to fitting curves through the means of repeatedly measured data. Hearkening back to the work of Tucker (1958) and Rao (1958), these approaches are primarily based on factor analytic methods. A very popular set of models based on a parameterization is presented by McArdle and Epstein (1987) and McArdle and Aber (1990). The latent growth curve model with either fixed or estimated basis vector coefficients (actually fixed or estimated factor loadings), is strongly related to the multilevel growth curve model.

For example, the linear growth curve model uses a highly constrained factor model to estimate the same set of fixed effects parameters as the linear mixed model. Here the model is again used to define straight lines with different intercepts and different slopes. To vary things slightly we have included two groups in the model and no covariate. While we concentrate on the straight-line model, curves other than straight lines can be modeled either by using polynomial terms (powers, centered powers, or orthogonal polynomials) or by estimating factor loadings (McArdle & Hamagami, 1991).

This model can be estimated as a structural equation measurement model. Using LISREL notation, the general matrix equation for the measurement model is

$$\vec{y} = \Lambda\vec{\eta} + \vec{\varepsilon} \tag{19.6}$$

where $\vec{y}$ is the vector of observed variables, Λ is a partitioned matrix of fixed factor loadings corresponding to the fixed and random effects, $\vec{\eta}$ is a partitioned vector representing the fixed and random regression parameters, and $\vec{\varepsilon}$ is a vector of regression residuals.

For two groups with four repeated measures, the matrix form of the linear growth curve model is

$$\begin{bmatrix} y_{i1} \\ y_{i2} \\ y_{i3} \\ y_{i4} \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 & 0 & 1 & 0 \\ 1 & 1 & 0 & 0 & 1 & 1 \\ 1 & 2 & 0 & 0 & 1 & 2 \\ 1 & 3 & 0 & 0 & 1 & 3 \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \\ \beta_3 \\ \beta_4 \\ \gamma_{i1} \\ \gamma_{i2} \end{bmatrix} + \begin{bmatrix} \varepsilon_{i1} \\ \varepsilon_{i2} \\ \varepsilon_{i3} \\ \varepsilon_{i4} \end{bmatrix} \text{ Group 1}$$

$$\begin{bmatrix} y_{i1} \\ y_{i2} \\ y_{i3} \\ y_{i4} \end{bmatrix} = \begin{bmatrix} 1 & 0 & 1 & 0 & 1 & 0 \\ 1 & 1 & 1 & 1 & 1 & 1 \\ 1 & 2 & 1 & 2 & 1 & 2 \\ 1 & 3 & 1 & 3 & 1 & 3 \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \\ \beta_3 \\ \beta_4 \\ \gamma_{i1} \\ \gamma_{i2} \end{bmatrix} + \begin{bmatrix} \varepsilon_{i1} \\ \varepsilon_{i2} \\ \varepsilon_{i3} \\ \varepsilon_{i4} \end{bmatrix} \text{ Group 2} \tag{19.7}$$

where β_1 and β_2 are the respective intercept and slope for the first group, and β_3 and β_4 are the respective differences in intercept and slope between the two groups. Because columns 3 and 4 are 0s for Group 1, β_3 and β_4 are not added to the Group 1 equation. β_3 and β_4 are added to the model for Group 2. The βs are fixed for the sample. γ_1 and γ_2 are the random effects (in this case random slopes and intercepts). These correspond to columns 5 and 6. In theory the random effects for each individual provide the difference between the group intercept and the individual intercept (γ_1) and the group slope and the individual slope (γ_2). In this model, factor scores take the place of the random effects in the linear mixed model. In estimating the model, the covariance matrix of the random effects, along with the covariance matrix of the ε_t are estimated. Factor scores are then predicted post hoc to yield the individual random effects under the same assumptions as in the linear mixed model. This highly constrained factor model is identical to a two-group linear mixed model/multilevel growth model. As such, it is a model of interindividual differences.

The Multilevel Autoregressive Model

Given that it is now more common to collect more intensive longitudinal data, some researchers have begun to turn to time-series methods in an attempt to model these data. Some of these approaches look at estimating a separate model for each individual, but others have attempted to use a multilevel-type approach. One method that is gaining more popularity is the multilevel autoregressive (AR) model in which the Level 1 model is an autoregressive model of a particular order. These models are thought to be particularly appropriate for data that Nesselroade (1991) described as belonging to a stable process, one without a discernible trend. Having a Level 1 autoregressive model has resulted in what some authors refer to as dynamic multilevel modeling (Suls, Green, & Hillis, 1998). Using a first-order autoregressive model, they referred to the AR parameter as a measure of inertia in that in the presence of a perturbation (a movement away from equilibrium), the autoregressive parameter tended to return the series toward the equilibrium point at the mean of the series (Hamaker, 2012; Kuppens, Allen, & Sheeber, 2010).

Consider an AR(1) process that is considered appropriate to describe all members of a sample, the multilevel AR model can be written as

Level 1:

$$y_{i,t+1} = \pi_{i0} + \pi_{i1} y_{i,t} + r_{i,t+1}$$

Level 2:

$$\begin{aligned} \pi_{i0} &= \beta_{00} + \beta_{10} x_i + \upsilon_{i0} \\ \pi_{i1} &= \beta_{01} + \beta_{11} x_i + \upsilon_{i1} \end{aligned} \tag{19.8}$$

where an intercept, π_{i0}, and the AR coefficient, π_{i1}, are both conditioned on some covariate, x. As required by the stationarity condition, $-1 > \pi_{i1} > 1$.

As before, we could substitute Level 2 into Level 1 to come up with a single linear mixed model equation to estimate the fixed effects of the model along with the covariance matrix of the random effects (Rovine & Walls, 2006). But instead we just wish to point out that the same assumptions of the multilevel model hold here; namely, that the AR(1) process holds equally well for all individuals, and that the individual AR(1) coefficients, the random effects, can be predicted post hoc under a common model assuming that the coefficients conform to a multivariate normal distribution. Different processes for different individuals cannot be accommodated by this model. It is therefore a model concerned with interindividual variation.

One innovation in this approach has been the addition of a random innovation variance (Wang, Hamaker, & Bergeman, 2012), which determines each individual's innovation variance in addition to their AR parameter value. Again, these individual error variances are predicted post hoc based on a common model and a specific assumed error distribution. That makes this model markedly distinct from a true model of intraindividual differences.

Lagged Regression Models

As an example of a lagged regression we consider the cross-lagged regression model with two variables, x and y, each measured on four occasions with the assumption that x at each occasion predicts y at the next occasion, and in turn, y at each occasion predicts x at the next occasion (Kenny, 2005). The model for such data is shown in Figure 19.1. Given any path model, the set of regression equations that result is given by the set of tracing rules (Wright, 1934). Simply stated, each variable in the model is a dependent variable in its own regression equation. Predictor variables for that equation are connected via one-headed arrows. Two-headed arrows represent correlations[3] or covariances (depending on the scaling) between

[3]From this point when we use the term *correlation* it will be implicit that the association coefficient could be a correlation, covariance, or sum of the cross-products depending on the requirements of scaling for the model.

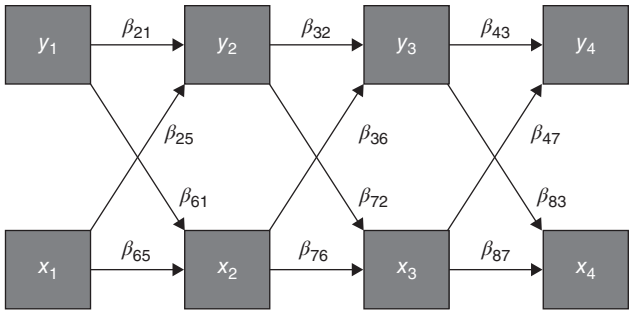


Figure 19.1 A cross-lagged regression model (regression coefficients only).

variables. Errors are linked to a particular dependent variable through one-headed arrows. To simplify the figures, we do not include errors in the models as shown.[4] We also do not include any concurrent regression relationships.

The set of regression equations resulting from the model in Figure 19.1 is

$$\begin{aligned} y_1 &= \zeta_1 \\ y_2 &= \beta_{21}y_1 + \beta_{25}x_1 + \zeta_2 \\ y_3 &= \beta_{32}y_2 + \beta_{36}x_2 + \zeta_3 \\ y_4 &= \beta_{43}y_3 + \beta_{47}x_3 + \zeta_4 \\ x_1 &= \zeta_5 \\ x_2 &= \beta_{61}y_1 + \beta_{65}x_1 + \zeta_6 \\ x_3 &= \beta_{72}y_2 + \beta_{76}x_2 + \zeta_7 \\ x_4 &= \beta_{83}y_3 + \beta_{87}x_3 + \zeta_8 \end{aligned} \tag{19.9}$$

where β represents a regression coefficient, and ζ represents an equation residual. As y_1 and x_1 are not predicted by other variables, they are considered *exogenous*, and all other variables (which are predicted) are considered *endogenous*. Exogenous variables are marked by the fact that their correlation equals the correlation between their equation residuals.

To estimate the regression coefficients and residual variances of the model, we use a covariance structure modeling approach that expresses the associations between variables (correlations, covariances, or sums of squares and cross-products) as functions of the parameters to be estimated. Here we use the LISREL model (Jöreskog & Sorböm, 1996) for these relationships. To conform to the LISREL model, we rename the variables as follows: $\eta_1 = y_1$; $\eta_2 = y_2$; $\eta_3 = y_3$; $\eta_4 = y_4$; $\eta_5 = x_1$; $\eta_6 = x_2$; $\eta_7 = x_3$; $\eta_8 = x_4$. Placing the ηs in equation (19.9) yields

$$\begin{aligned} \eta_1 &= \zeta_1 \\ \eta_2 &= \beta_{21}\eta_1 + \beta_{25}\eta_5 + \zeta_2 \\ \eta_3 &= \beta_{32}\eta_2 + \beta_{36}\eta_6 + \zeta_3 \\ \eta_4 &= \beta_{43}\eta_3 + \beta_{47}\eta_7 + \zeta_4 \\ \eta_5 &= \zeta_5 \\ \eta_6 &= \beta_{61}\eta_1 + \beta_{65}\eta_5 + \zeta_6 \\ \eta_7 &= \beta_{72}\eta_2 + \beta_{76}\eta_6 + \zeta_7 \\ \eta_8 &= \beta_{83}\eta_3 + \beta_{87}\eta_7 + \zeta_8 \end{aligned} \tag{19.10}$$

This can be summarized by the matrix equation

$$\vec{\eta} = B\vec{\eta} + \vec{\zeta} \tag{19.11}$$

where $\vec{\eta}$ is an 8×1 vector of variables, $\vec{\zeta}$ is an 8×1 vector of equation residuals, and β is an 8×8 matrix of regression weights indexed according to the previous regression weights. This is a regression equation with all variables acting as possible dependent variables in their own regression equation. Which variables predict each of the dependent variables is determined by the path model. Equation 19.11 can be used to develop an equation for the expected associations among all of the variables. The difference between the observed variable covariance matrix, S, and the expected $\hat{\Sigma}$ based on the model can be used to determine the fit of the model.

In terms of estimating this model, the required input for generating the regression estimates is the covariance matrix. Individual data points are not required for the estimation of the parameters. As in ordinary regression, the equation residuals, ζ, can be predicted post hoc using the parameter estimates. These residuals are model based in that the group model parameters must be known before the residuals can be calculated.

The Vector Autoregression Model

We now present the person-specific analog of the cross-lagged regression model. Here we consider two time series, x and y, measure on a single subject. We hypothesize that x_t predicts y_{t+1} and y_t predicts x_{t+1}. This model appears in Figure 19.2.

This is an example of a first-order vector autoregression model (Lutkepohl, 2006). Here, the order of the process represents which of the previous occasions are used to predict the next occasion. With just the previous occasion as a predictor, it is a first-order process. If we used the previous

[4] We also indicate the observed variables as squares. In many SEM programs, regression relationships among latent variables (often shown as circles) are modeled. For observed variable regression, latent variables are set as equivalent to observed variables.

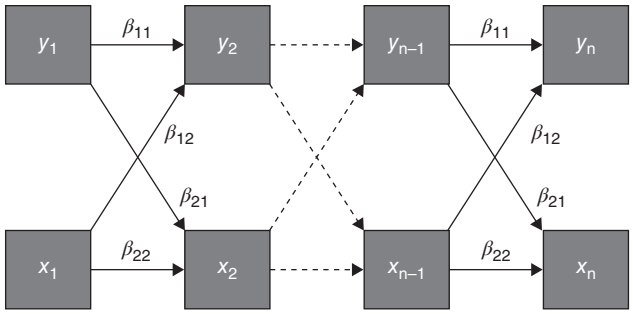


Figure 19.2 A first-order cross-legged vector autoregression model (regression coefficients only).

two occasions to predict, it would be a second-order process. The first-order model presented is equivalent to the set of equations

$$y_{t+1} = \beta_{11} y_t + \beta_{12} x_t + \varepsilon_{1t+1}$$
$$x_{t+1} = \beta_{21} y_t + \beta_{22} x_t + \varepsilon_{2t+1} \quad (19.12)$$

The matrix representation of these equations is

$$\begin{bmatrix} y_{t+1} \\ x_{t+1} \end{bmatrix} = \begin{bmatrix} \beta_{11} & \beta_{12} \\ \beta_{21} & \beta_{22} \end{bmatrix} \begin{bmatrix} y_t \\ x_t \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t+1} \\ \varepsilon_{2t+1} \end{bmatrix} \quad (19.13)$$

While this looks much like the cross-lagged regression model described above, here we estimate a separate model for each individual. As a result the $\begin{bmatrix} \beta_{11} & \beta_{12} \\ \beta_{21} & \beta_{22} \end{bmatrix}$ matrix is person specific and not dependent on other individuals' results.

By changing the regression weight matrix, we can create the analog for many different lagged regression models. For example, $\begin{bmatrix} \beta_{11} & 0 \\ 0 & \beta_{22} \end{bmatrix}$ represents the analog to the stability only model (i.e., x_t predicts x_{t+1}, y_t predicts y_{t+1}). The matrix, $\begin{bmatrix} \beta_{11} & \beta_{12} \\ 0 & \beta_{22} \end{bmatrix}$, represents prediction from x_t to y_{t+1}. This latter situation represents the test of Granger causality (Granger, 1969). Here, x is a Granger cause of y. Granger causality has been used in neural network analysis (Seth, 2008), connectivity mapping of fMRI brain scan data (Roebroeck et al., 2005), and econometric modeling (Hatemi, 2012).

Here we assume that the same order of autoregression and the same order of lagged prediction (i.e., x predicting y) would hold for each individual. However, in a true individual approach we would estimate distinct models, which would include determining the order of autoregression for each time series and the order of the lagged prediction. With the proper models, we could determine which individuals have the same models—for example, whether the order of their models is the same, whether the regression coefficients are the same, or whether the variance of the errors is the same. Group models could be constructed for those individuals who have identical models. Nesselroade and Molenaar (1999) suggested methods for pooling the results of individual time series. This approach of creating samples based on the equivalence of individual models guarantees that *ergodicity*, as defined already, is satisfied and that a subgroup with an identical pooled model adequately describes each individual in the subgroup. Using such a strategy, it is most likely that different subgroups based on different (sometimes qualitatively different) models can emerge. When there are different models, the underlying process generating the series would be expected to be different for the subgroups, and this becomes an important and interesting research result. These models can, of course easily be extended to any number of variables.

THE DATA BOX AND FACTOR ANALYSIS

The conceptual framework that allows one to consider the difference between intra- and interindividual approaches in multivariate studies is often linked to Cattell's data box. In his *Handbook for Multivariate Experimental Psychology*, Cattell (1952, 1966a) presented a classification scheme for determining the appropriate analysis for data collected in a multivariate study. The data box as shown in Figure 19.3 has three dimensions: *variables*, *individuals*, and *occasions*. Each two-dimensional slice or *facet* of the box represents a way of constructing an association matrix with each slice having a dimension of interest and a dimension across which the data will be pooled. Cattell gave each two-dimension facet a letter name. The most commonly used are the *R* and *P* facets. When interested in pooling across *individuals* to create a *variable* × *variable* correlation matrix, we are using the *R* facet resulting in *R-technique*. This results in the most common form of factor analysis of interindividual variation. Both common cross sectional and longitudinal factor analysis are examples of *R-technique*. In longitudinal factor analysis the variables collected on multiple occasions are treated along the variables dimension. The data are still pooled across individuals to form a correlation matrix among the variables. Since multilevel growth curve models, repeated measures ANOVA, and latent growth curve models are pooled across individuals, each can be thought of as belonging to the *R-technique* class of analyses.

For person-specific models, the *P* facet takes data for a single subject and pools across occasions to create

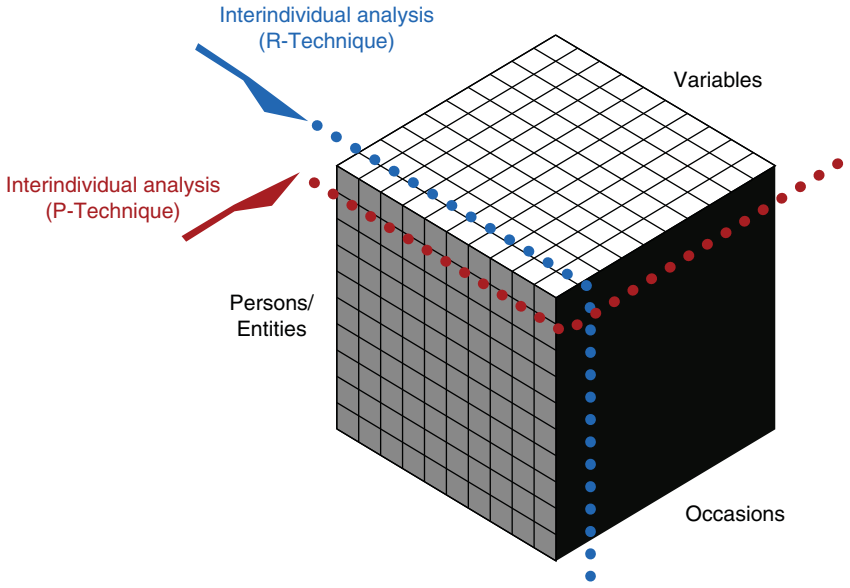


Figure 19.3 The Data Box. See footnote 1.

a *variable* × *variable* correlation matrix of intraindividual variation. Factor analysis of this type of data set is referred to as *P-technique*. The difference between *R*- and *P-technique* is thus the dimension across which one pools based on the way data are collected, where *R-technique* pools across persons and *P-technique* pools across occasions. Facets for both *R*- and *P-technique* appear in Figure 19.3.

Factor Models for Longitudinal Data

Factor models for longitudinal data represent an extension of the common factor model (Gorsuch, 1983; Harman, 1976). The common factor model can be expressed as

$$\vec{y} = \Lambda\vec{\eta} + \vec{\varepsilon} \tag{19.14}$$

where $\vec{y}$ represents the vector of observed variables, Λ represents the matrix of regression weights linking the latent variables to the observed variable, $\vec{\eta}$ represents the vector of latent variable factor scores, and $\vec{\varepsilon}$ represents the vector of measurement errors. Given any factor model the expected covariance matrix of the observed variable based on the model is

$$\hat{\Sigma} = \Lambda\Phi\Lambda^T + \Theta_\varepsilon \tag{19.15}$$

where $\hat{\Sigma}$ is the expected covariance matrix of the observed variables based on the model, Λ is the matrix of factor loadings (i.e., regression weights), Φ is the covariance matrix of the latent variables, and Θ_ε is the covariance matrix of the measurement errors or uniquenesses. In the exploratory common factor model with set number of factors, each observed variable loads on each factor. This means that with, for an example with three factors, each factor predicts each of the observed variables. With means added to the model

$$\mu_y = \tau + \Lambda\kappa$$

where μ_y is the vector of means, τ is the vector of intercepts, and κ is the vector of factor score means[5].

To consider a longitudinal version of factor analysis for the situation in which there are relatively few occasions of measurement, we move to the confirmatory version of the factor model based on *R-technique*. The exploratory longitudinal factor model (for instance, a longitudinal factor model in which at each occasion the factors are uncorrelated, there only are autoregressions between the factor scores, and in which the factor loadings at each occasion only have minimal identifiability constraints) still becomes a confirmatory factor model when fitted to the longitudinal covariance matrix. The confirmatory factor model allows the researcher to specify certain hypotheses based on the parameters of the model. For example, the researcher could expect certain variables to load only on certain factors.

[5]Additional constraints are required; otherwise, the means model would be overparameterized.

Consider the situation in which the researcher has collected the same three variables on three separate occasions assuming that the three variables are indicators of the same latent construct. A sample pattern based on a confirmatory model would have each factor represent the same latent variable at a particular occasion resulting in loadings of 0 for those variables on the other occasions. This would result in the following in the following Λ matrix:

$$\begin{array}{c} \\ y_{1T1} \\ y_{2T1} \\ y_{3T1} \\ y_{1T2} \\ y_{2T2} \\ y_{3T2} \\ y_{1T3} \\ y_{2T3} \\ y_{3T3} \end{array} \begin{array}{c} \begin{array}{ccc} \eta_{T1} & \eta_{T2} & \eta_{T3} \end{array} \\ \begin{bmatrix} \lambda_{11} & 0 & 0 \\ \lambda_{21} & 0 & 0 \\ \lambda_{31} & 0 & 0 \\ 0 & \lambda_{42} & 0 \\ 0 & \lambda_{52} & 0 \\ 0 & \lambda_{62} & 0 \\ 0 & 0 & \lambda_{73} \\ 0 & 0 & \lambda_{83} \\ 0 & 0 & \lambda_{93} \end{bmatrix} \end{array} \tag{19.16}$$

and each row represents a regression equation where the observed along margin represents the dependent variable that is predicted by the latent variables along the top with regression coefficients indicated by the factor loadings.

The covariance matrix among the factors is

$$\Phi = \begin{bmatrix} \phi_{11} & & \\ \phi_{21} & \phi_{22} & \\ \phi_{31} & \phi_{32} & \phi_{33} \end{bmatrix} \tag{19.17}$$

In exploratory factor models, the variance of the measurement errors, the uniquenesses, are estimated. In CFA under certain circumstances measurement errors can be correlated. In this model, we expect that the same variable measured on different occasions may result in correlated measurement errors. This would result in the following covariance matrix of measurement errors:

$$\Theta_\varepsilon = \begin{bmatrix} \theta_{11} & & & & & & & & \\ 0 & \theta_{22} & & & & & & & \\ 0 & 0 & \theta_{33} & & & & & & \\ \theta_{41} & 0 & 0 & \theta_{44} & & & & & \\ 0 & \theta_{52} & 0 & 0 & \theta_{55} & & & & \\ 0 & 0 & \theta_{63} & 0 & 0 & \theta_{66} & & & \\ \theta_{71} & 0 & 0 & \theta_{74} & 0 & 0 & \theta_{77} & & \\ 0 & \theta_{82} & 0 & 0 & \theta_{85} & 0 & 0 & \theta_{88} & \\ 0 & 0 & \theta_{93} & 0 & 0 & \theta_{96} & 0 & 0 & \theta_{99} \end{bmatrix} \tag{19.18}$$

In this model each factor is indicated by three observed variables. In addition to the regression relationships between the latent and observed variables (the factor loadings), characteristics of the latent variables could be of interest. For example, a research question may involve whether the means of the latent variables change across time, or whether there is a distinct pattern of correlation among the latent variables across occasions of measurement.

The set of variables could be, for example, indicators of problematic behavior (e.g., subscales of the Child Behavior Checklist, Achenbach & Edelbrock (1981)), measures of temperament, attachment, or any number of other constructs that are repeatedly measured. A natural extension would have more than one latent variable at each occasion, each one repeatedly measured.

To be able to discuss group differences or characteristics of change in a repeatedly measured latent variable, a certain level of measurement invariance must exist (Meredith, 1993; Millsap & Meredith, 2004; Reise et al., 1993; Vandenberg & Lance, 2000; Widaman & Reise, 1997). The underlying notion of factorial invariance is if that assumption is violated, the same latent construct may not be measured across all groups or across all occasions. This result would strongly limit the ability to interpret any change in the construct. Suggestions for how to deal with differing degrees of factorial invariance have not always been consistent.

Improved model fitting capabilities allow one to more easily test whether invariance has been violated without necessarily suggesting what to do in response to a particular violation (Bontempo et al., 2012; Millsap & Meredith, 2004). Millsap & Kwok (2004) gave a good set of recommendations for how to assess the effect of a particular violation. Millsap (2011) emphasized the multigroup case.

For the previously shown factor pattern, the parameters to be estimated are Λ, the factor loadings; Φ, the factor covariances; and Θ_ε, the covariance matrix of the measurement errors. The model above is based on a correlation or covariance matrix. We could include information about the means as input, which would allow us to also estimate parameters related to the mean structure[6].

[6]Means in longitudinal factor models should be modeled according to a method described by Jöreskog (1978) and Sörbom (1974) to ensure that the means model is appropriate for interval-scaled variables. This requires that at the first occasion the means are freely estimated in τ_y (a saturated means model at the first occasion). Next τ_y is constrained to be invariant across later occasions and only at these later occasions the changes in means are modeled by the factor means in α. If one models all means in terms of α (including at the first occasion and leaving out τ_y) then it is required that the variables have ratio scale.

A typical sequence for testing factorial invariance may include the following levels:

1. Configural invariance—the same patterns of nonzero loadings hold across groups or occasions.
2. Metric or pattern invariance—the values of the loadings are fully invariant across groups or occasions.
3. Strong factorial invariance—the pattern matrices and the latent intercepts are fully invariant across groups or occasions.
4. Strict factorial invariance—the pattern matrices, intercepts, and unique variances are fully invariant across groups or occasions.

The first two invariance criteria were introduced by Thurstone (1947) and the third and fourth by Meredith (1993). To test models of the latent variable means, strong factorial invariance is required.

Like the cross sectional common factor model, the longitudinal factor model requires as input the correlation (or covariance) matrix of the input variables. Individual values of the variables are not needed to estimate the model. The factor loadings are group-based, not person-specific. Factor scores are generated as post hoc predictions using a similar procedure to that of the latent growth curve's random effects. In fact, the multilevel growth curve model can estimated as a structural equations model (Rovine & Molenaar, 2000). In this form, the random effects are the factor scores of the model estimated using the fixed effects and the covariance matrix of the random effects under certain specified distributional assumptions. Since estimated models are based on input matrices pooled across individuals, they are models of interindividual differences. Despite the importance and utility of this model, it is not person specific.

Person-Oriented Approaches

Certain methods described as person-oriented approaches (Bergman & Magnusson, 1997) are typically described as a counterpoint to variable-oriented analysis (Bergman & Trost, 2006). These often involve cluster analyzing repeatedly measured data to identify subgroups of individuals who have similar sets of variable values across occasions of measurement. As can be shown, under certain circumstances, a cluster analysis can be transformed into a factor analysis. As such, this analytic approach may pool across individuals. Under these circumstances, cluster analysis generalizes across individuals, not occasions. This approach is thus an interindividual differences approach rather than a person-specific approach.

P-Technique Factor Models

As previously stated, the *ergodicity* hypothesis implies that a group-based result can apply to an individual only when that model holds for each individual. Without true time-series data that assumption cannot be tested. As Molenaar (2004) argued, the result of this is that cross sectionally or longitudinally derived factor models based on a covariance matrix pooled across individuals (R-technique) can be quite different from those derived separately for each individual. The individual approach, based on P-technique factor analysis, is a true intraindividual method.

P-technique factor analysis has been used to study stepchildren's emotional experiences (Corneal & Nesselroade, 1994), changes in mood (Bath, Daly, & Nesselroade, 1976), and changes in attachment security (Mitteness & Nesselroade, 1987) using essentially the standard (R-technique) factor analytic approach with occasions taking the place of individuals.

For the basic P-technique model, the data for each subject are pooled across occasions resulting in a variable × variable association matrix. For the analogous three variable factor model shown above as a longitudinal factor model, the set of equations represented by this model is represented by the matrix equation

$$\begin{bmatrix} y_1 \\ y_2 \\ y_3 \end{bmatrix}_t = \begin{bmatrix} \lambda_1 \\ \lambda_2 \\ \lambda_3 \end{bmatrix} [\eta]_t + \begin{bmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \varepsilon_3 \end{bmatrix}_t \tag{19.19}$$

where a single factor replaces the occasion factors of the longitudinal factor analysis and a single set of loadings represents the requirement of invariance across the time series. This model would be estimated separately for each subject.

The Dynamic Factor Model

The P-technique makes certain assumptions regarding the nature of relationships across occasions of measurement. Most importantly, the model assumes that there are no lagged relationships among the occasion variables. The association matrix that is analyzed pairs variables at each occasion to construct the correlation. While this model has been shown to be more widely applicable than one might expect (Molenaar & Nesselroade, 2009), models that address the question of lagged relationships and determine the order of lags represent an important approach. Molenaar (1985) presented an extension of the P-technique model, the dynamic factor model that properly addresses the question of whether or not the relationship among

variables is lagged, and if so, what is the order. The dynamic factor model is

$$y_t = \sum_{u=0}^{s} \Lambda_u \eta_{t-u} + \varepsilon_t \qquad (19.20)$$

where $\Lambda_u$contains the factor loading for lag, u. With this model the researcher can determine the degree to which latent variables and residuals are correlated over time. When $s = 0$, the model reduces to the state space model in which the evolution of factors is explained by a vector autoregressive process (VAR).[7] These models and their variants have appeared under somewhat different names (Nesselroade et al., 2002). Wood and Brown (1994) showed a relatively straightforward method for implementing this model as a structural equations model.

DATA EXAMPLES

Data Example 1: The Borkenau and Ostendorf Data Set

Here, we demonstrate an instance of that situation of a violation of ergodicity assumption for personality factors based on the big five using a data set provided by Borkenau and Ostendorf (1998). This data set included 22 individuals followed on a daily basis over 90 days with 30 personality items being repeatedly measured. The participants were psychology students from the University of Halle, Germany; course credit was given for participation. The sample consisted of 19 women and three men who were mostly 19-year-olds or in their early 20s, except for three females who were in their 30s or 40s. In the current example, an exploratory factor analysis is carried out at both the cross sectional group level (R-technique) and for several individuals at the person-specific time-series level (P-technique; Nesselroade & Jones, 1991). Here, we will demonstrate that the number of cross sectional factors gives a poor indication of the factor structure for each of the three individuals considered.

In the exploratory approach to factor analysis, we initially want to determine the number of factors or latent variables that underlie the set of observed variables considered. For the Borkenau and Ostendorf data set we first determined the number of factors cross sectionally (R-technique). The scree plot for these data appears in Figure 19.4. The scree plot (Cattell, 1966b) is a commonly used visual guide for determining the number of factors in a solution. In the plot the points represent the results of an eigenvalue decomposition of the input correlation matrix. The eigenvalues are ordered from largest to smallest and can be thought of as incrementally explaining the set of correlations among the variables. Each eigenvalue corresponds to one possible factor, and some subset of these factors will be able to adequately reproduce the input correlation matrix. The question is to find this optimal number of factors. The scree plot does so by indicating the point at which the remaining factors are most likely trivial. This point is usually depicted by an *elbow* in the scree. For the cross sectional data, the elbow seems to appear at the fourth eigenvalue. For Subject A, the elbow appears at either eigenvalue 2 or eigenvalue 4; for Subject B at the third eigenvalue; and for Subject C at the second, third, or fourth eigenvalue. Because this perusal approach can be somewhat subjective, several numerical criteria have been developed. Here we employ the acceleration factor (AF; Raiche et al., 2006). The AF has been established as a good selection procedure for both R- and P-technique. The approach looks at the derivative (the slope) of the scree plot and determines where the slope changes most rapidly (accelerates). This typically represents the elbow of the curve. Using this approach, we determined that the elbow did indeed occur at the fourth eigenvalue for the cross sectional data indicating a three-factor solution. For three individuals, Subject A had a one-factor solution (elbow at the second eigenvalue), and Subjects B and C both had a two-factor solution. The solution from the cross sectional data described none of the three subjects adequately.

This example demonstrates how the number of factors determined in a group-level R-technique factor analysis may be inconsistent with subsequent individual factor models obtained via individually applied P-technique analyses.

Data Example 2: PANAS Data

Using data taken from a study of stepchildren, Corneal and Nesselroade (1994; see also Corneal & Nesselroade, 1991; Molenaar et al., 2009; Rovine, Molenaar, & Corneal, 1998; Molenaar et al., 2009) showed a similar result based on time-series assessments of emotions.

The data were measures designed to analyze the emotional experiences of stepsons as they interacted with their stepfathers over a 2-month period. After a specified type of interaction with their stepfather (including mealtimes, leisure activities, conversations, arguments, and disciplines), the stepson would complete the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) to assess the emotional content of the interaction.

[7]This VAR is implicit in the general model (20) because it is simplest to treat the evolution of factor scores as a white-noise process. Molenaar and Nesselroade (2001) showed that other choices are possible, including VARs. Only these choices have to be fixed.

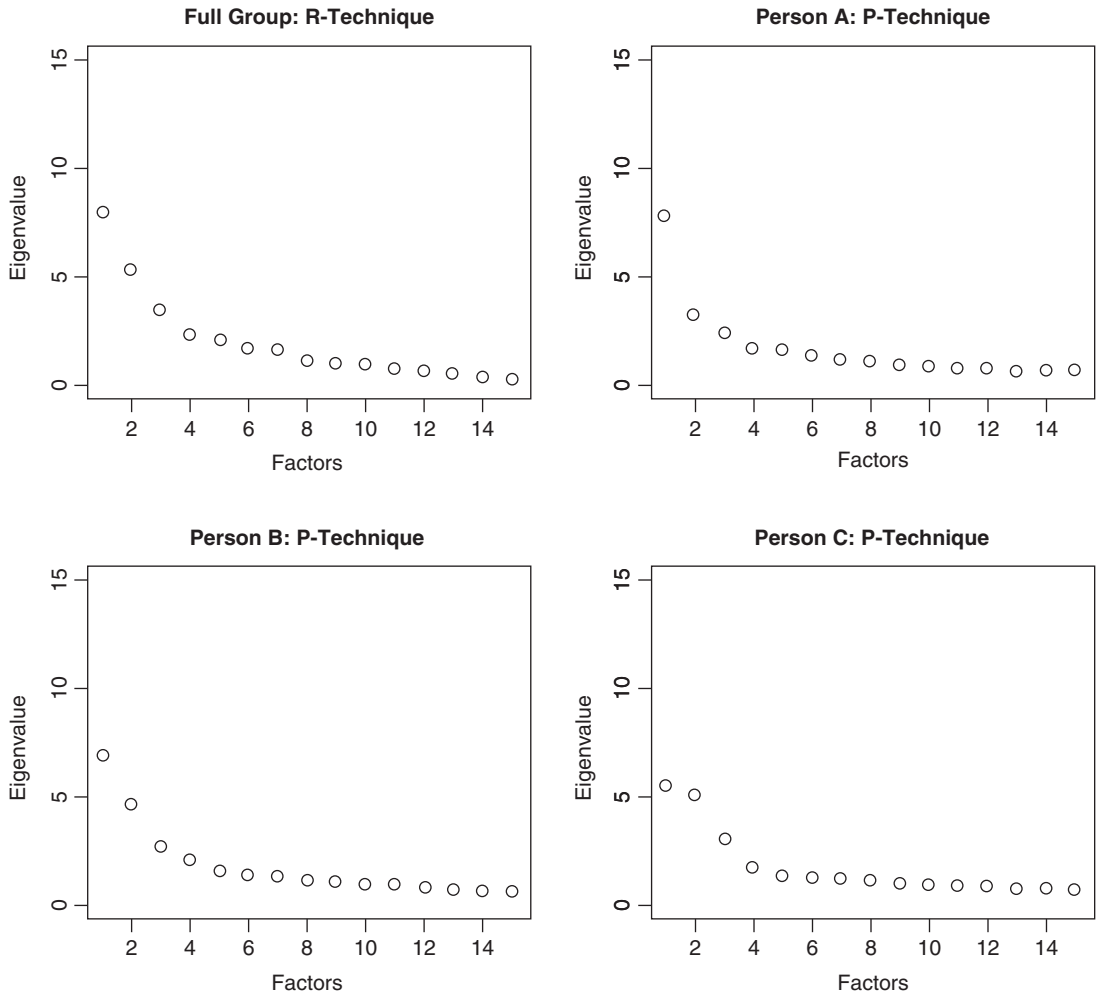


Figure 19.4 A comparison of scree plots of the Borkenau data.

To the 20 items of the PANAS, eight involvement items were added. Since the 20 items were developed for high internal consistency, the 28 items were expected to have a two-factor structure. The items representing this factor structure along the with-fit indices for Stepson 1 are shown in Table 19.1. While the two-factor solution typically shows up in the analysis of cross sectional data, the two-factor confirmatory solution showed a very poor fit. In fact the two-factor solution was a poor model for all stepsons included in this study. For each subject we first tested the two-factor model. After the model was rejected, we moved to an exploratory factor analysis to determine the proper number of factors. Using the salient loadings for each stepson, we then fit a confirmatory factor model. We include the results for three of the stepsons in Table 19.2.

TABLE 19.1 Positive and Negative Items for the Two-Factor Solution

Negative	Positive	Involved
Distressed	Satisfied	Interested
Upset	Content	Excited
Humiliated	Strong	Enthusiastic
Hostile	Proud	Alert
Ashamed	Liking	Inspired
Irritated	Accepted	
Active	Determined	
Scared	Closed	
Afraid	Loved	
Nervous		
Jittery		
Discouraged		
Not wanted		
Goodness-of-fit statistics		
Chi-square with 349 degrees of freedom = 1066.93 ($P = 0.0$)		
Nonnormed fit index (NNFI) 0.44		

TABLE 19.2 Confirmatory Factor Analysis for Stepsons 1, 5, and 6

Stepson 1: LISREL ML estimates

	Factors				
Variables	Interest	Anger	Anxiety	Affection	Closeness
Interested	0.65				
Excited	0.67				
Enthusiastic	0.72	−0.38			
Inspired	0.42	−0.36			
Alert	0.66		0.34		
Attentive	0.74		0.33		
Satisfied	0.62	−0.59			
Proud	0.66	−0.39			
Distressed		0.85			
Upset		0.87		−0.23	
Strong	0.51	0.30	0.23		
Hostile		0.91		0.29	
Irritable		0.88		−0.32	
Discouraged		0.74	0.33	0.24	
Accepted	0.46	−0.63			
Not wanted		0.97			
Determined	0.51	0.34		0.25	
Content	0.32	−0.46			
Scared			0.71		
Nervous			0.92		
Jittery		0.45	0.73	0.27	
Afraid			0.98		−0.13
Humiliated		0.35		−0.49	
Loved				0.43	
Active	0.43			0.37	
Liking					0.62
Ashamed					−0.41
Close					0.73

Factor intercorrelations

	Interest	Anger	Anxiety	Affection	Closeness
Interest	1.00				
Anger	0.36	1.00			
Anxiety	−0.06	−0.08	1.00		
Affection	0.13	−0.03	0.11	1.00	
Closeness	0.37	−0.36	0.14	0.23	1.00

Stepson 5: LISREL ML estimates

	Factors			
Variables	Anger	Interest	Strength	Shame
Distressed	0.71			
Liking	−0.68	0.25		
Upset	0.79	−0.38		
Scared	0.65	0.33		
Hostile	0.87		−0.19	
Irritable	0.75			
Not wanted	0.68			
Loved	−0.75			
Nervous	0.91	0.46		
Close	−0.72			
Jittery	0.55			
Afraid	0.33		−0.30	
Content	−0.49	0.26		
Interested		0.65		
Excited		0.70		
Enthusiastic		0.79		
Inspired		0.60		

TABLE 19.2 (*continued*)

Stepson 5: LISREL ML estimates

	Factors			
Variables	Anger	Interest	Strength	Shame
Alert		0.59		
Attentive		0.53		
Proud		0.60		−0.19
Strong			0.47	
Discouraged	0.45		−0.26	0.35
Determined			0.81	
Satisfied	−0.22		0.47	
Active			0.57	
Humiliated	0.56			
Accepted		0.55		−0.98
Ashamed				0.45

Factor Intercorrelations

	Anger	Interest	Strength	Shame
Anger	1.00			
Interest	−0.64	1.00		
Strength	−0.44	0.70	1.00	
Shame	0.27	0.25	−0.01	1.00

Stepson 6: LISREL ML Estimates

	Factors		
Variables	Anger	Interest	Strength
Distressed	0.96		−0.21
Upset	0.92		
Humiliated	0.57		
Scared	0.79		
Hostile	0.79		
Irritable	0.92		−0.21
Discouraged	0.82		
Ashamed	0.82		
Not wanted	0.74		
Nervous	0.78		
Satisfied	−0.58	0.61	
Jittery	0.81	0.14	
Afraid	0.85		
Content	−0.55	0.57	
Interested	0.17	0.77	
Liking	−0.16	0.72	
Excited		0.81	
Enthusiastic		0.81	
Accepted	−0.14	0.65	
Inspired	−0.22	0.56	
Loved		0.69	
Alert	0.20	0.77	
Attentive		0.58	0.21
Close		0.68	
Strong			0.56
Determined			0.75
Proud	−0.60		0.65
Active			0.81

Factor Intercorrelations

	Anger	Interest	Strength
Anger	1.00		
Interest	−0.10	1.00	
Strength	0.42	0.65	1.00

The results indicate the very intuitive result that different children respond to a situation by experiencing different combinations of emotions. For Stepson 1, a five-factor solution seemed the best fit. Looking at the items that load, the factors can be described as Interest, Anger, Anxiety, Affection, and Closeness. A number of variables loaded on more than one factor. Certain variables can be expected to help define more than one factor. For example, *enthusiasm* is an indicator of both Interest and Anger with a positive loading on Interest and a negative loading on Anger. The combination of variables that load on a particular define that factor for a specific stepson. While Anger may exist as a factor for each of the stepsons, its definition in terms of the variables that comprise the factor can vary. When Stepson 1 felt *anger* during interactions with his stepfather, he felt less *accepted*. For Stepsons 5 and 6, *anger* was accompanied by a feeling of *anxiety*. Stepson 5 also felt less loved when he felt *anger*.

Stepson 5 differed from Stepson 1 in some important ways. For Stepson 4, a four-factor solution seemed most appropriate. Two factors that we labeled Strength and Shame appeared for Stepson 5 that had not occurred in Stepson 1. Stepson 6 had a three-factor solution as the best fitting model. Here, Anger and Anxiety items appeared on a single factor. Interest and Affection items also tended to appear on the same factor. A Strength factor similar to Stepson 5 also appeared.

The results shown here indicate the importance of at least considering the person-specific approach in looking at factor structures. By pooling across subjects, the investigator is assuming that differences in individuals should be treated as essentially error. If the differences are systematic and represent important descriptive information, that information is lost when pooling to estimate models. The result of using the assumed cross sectional two-factor solution to describe each subject in this study would be that a wealth of information describing how these stepsons differ in their interactions with the stepfathers would be lost.

DISCUSSION

Clearly pooling across heterogeneous cases whether they be replications, subjects, or systems can be ill advised. Applying analysis of interindividual variation to heterogeneous data that involves pooling across subjects that obey subject-specific models can lead to results that poorly describe individuals and poorly describe the group. We argue, instead, that such data should be subjected to the analysis of intraindividual variation that is specifically designed to detect the kind of heterogeneity that analysis of interindividual variables essentially ignores.

However, carrying out separate single-subject analysis of the intraindividual variation of a sample of subjects may yield a bewildering collection of different subject-specific dynamic models. For this situation there is obviously an urgent need for methods to bring order to these separate models. This represents the need to arrive at nomothetic models for intraindividual variation.

One promising approach to accomplish this is the so-called Idiographic Filter proposed by Nesselroade et al. (2007). The basic idea underlying the Idiographic Filter is the recognition that the way latent factors manifest themselves in observed variables can be subject-specific. For instance, in our second data example, for Stepson 5, anger is accompanied by being nervous and scared, which for Stepson 1, that is not the case. The Idiographic Filter thus allows for subject-specific factor loadings while locating homogeneity at the latent level.

Molenaar (et al., 2012) has applied a generalized version of the Idiographic Filter (iFACE) to the analysis of multivariate time series obtained with a pair of genetically related subjects allowing for subject-specific heritabilities and environmental effects. A first application of the iFACE to multilead EEG obtained in an oddball task shows clear evidence for considerable subject-specificity of heritable and environmental effects.

While the Idiographic Filter can accommodate heterogeneity due to subject-specific factor loadings, Molenaar recently developed an alternative approach called the Group Iterative Multiple Model Estimation (GIMME; Gates & Molenaar, 2012) which can accommodate arbitrary subject-specificity of both factor loadings and any other parameters in state space models using a variant of the structural equations model referred to as the unified SEM (Gates *et al*., 2010).

GIMME has been used in a number of studies including the analysis of a large and multifaceted simulated data set considered in Smith et al. (2011), who tested 38 methods to estimate connectivity networks based on multivariate fMRI time series. The methods included Bayesian net approaches and vector autoregressive modeling. None of these methods did well in terms of recovering the presence of directed links among component series. This surprising result, obtained with realistic simulation of BOLD activities of brain regions of interest under a wide variety of conditions (varying signal-to-noise, varying dimensionality of the observed time series, varying number and strength of links, etc.), created quite a stir in the brain imaging community. When GIMME was applied

to these data, all directed links and the numerical values of their weights were recovered with close to or exactly 100% fidelity (see Gates & Molenaar, 2012, for a complete report of these results). GIMME has been most often applied to brain imaging data, but more recently it has been extended to the description of behavioral processes (Belz, et al., 2013). GIMME can be freely accessed at www.nitrc.org/projects/gimme/. We note that GIMME is unique in providing a valid way to determine common dynamic models for heterogeneous replicated time series, thus yielding generalizable (nomothetic) results about idiographic processes.

We feel that the importance of the subject-specific approach to the modeling of intraindividual variation in the statistical analysis of developmental processes in particular is clear. All nonergodic processes such as developmental processes should initially be analyzed at the level of intraindividual variation, i.e., single-subject time-series analysis. From this starting point several courses of action can be followed in attempts to obtain nomothetic knowledge about intraindividual variation. One could apply the methodologies used in cognitive neuroscience and/or neuropsychology to generalize to nomothetic knowledge from single case analyses. Or one could use the Idiographic Filter or GIMME approaches for the same purpose. We hope and expect that in addition to these well-established approaches other new alternative methodologies will be found to obtain nomothetic knowledge about intraindividual variation, because this is an essential endeavor to move our science forward.

FUTURE DIRECTIONS

Cicchetti and Toth (2009) emphasized the importance of "translat[ing] basic research knowledge into real world contexts." We believe that an important way to allow this clinical translation to occur is to ensure that appropriate models are used to describe a successful therapeutic intervention. The approaches to targeting interventions based on single subject, person specific modeling seem very appropriate to this task. In particular, the notion of optimal control, of moving the individual toward a specific goal or set of goals using a model that can assess whether the person in on target and suggest a change of direction when not on target seems to be an important tool to add to the field. This approach has been successful in many other areas and disciplines. To move the field of developmental psychopathology forward we believe that this approach should be added to the toolbox of researchers. Using these methods requires the collection of more intensive data, but given the increasing popularity of data collection methods such as those based on Ecological Momentary Assessment (EMA), the single subject, person-specific modeling approach is becoming more feasible. It may take some ingenuity on the part of the researchers to design approaches that make use of these methods, but that alone represents an important challenge for clinicians and other investigators. In some areas of research, sufficient data already exist to make use of single subject methods. For example, work in the area of experience dependent brain development makes use of brain-scan data. The development of models requires individual models of developing connectivities. Methods such as the GIMME method described above are designed specifically for such a problem. As mentioned above, all nonergodic processes such as developmental processes should be analyzed as a single-subject time-series analysis. Methods such as GIMME and the idiographic filter can and should be applied to a wide range of problems in the behavioral sciences. We believe that researchers in the area of developmental psychopathology should look for opportunities to apply these methods, which will hopefully help to better describe the pathways to and out of abnormal development while describing conditions of resilience and improving the outcomes for those experiencing these problems.

REFERENCES

Achenbach, T. M. & Edelbrock, C. S. (1981). Behavioral problems and competencies reported by parents of normal and disturbed children aged four through sixteen *Monographs of the Society for Research in Child Development*, *46*(1),1–82.

Adams, J., & Drake, R. (2006). Shared decision-making and evidence-based practice. *Community Mental Health Journal*, *42*(1), 87–105.

Adams, N., & Grieder, D. (2005). *Treatment planning for person-centered care: The road to mental health and addiction recovery*. New York, NY: Academic Press.

Banks, R. B. (1994). *Growth and diffusion phenomena: Mathematical frameworks and applications*. Berlin, Germany: Springer-Verlag.

Bath, K. E., Daly, D. L., & Nesselroade, J. R. (1976). Replicability of factors derived from individual P-technique analyses. *Multivariate Behavioral Research, 1*(2), 147–156.

Belz, A. M., Beekman, C., Molenaar, P. C. M., & Buss, K. A. (2013). Mapping temporal dynamics in social interactions with unified structural equation modeling: A description and demonstration revealing time-dependent sex differences in play behavior. *Applied Developmental Psychology*, *17*, 152–168. doi: 10.1080/10888691.2013.805953

Bergman, L. R. (2012). Advancing developmental science: Some challenges and obstacles. *International Journal of Developmental Science, 6,* 41–43.

Bergman, L. R., & Andersson, H. (2010). The person and the variable in developmental psychology. *Journal of Psychology, 218*(3), 155–165. doi: 10.1027/0044-3409/a000025

Bergman, L. R., & Magnusson, D. (1997). A person-oriented approach in research on developmental psychopathology. *Development and Psychopathology*, *9*, 291–319.

Bergman, L. R., & Trost, K. (2006). The person-oriented versus the variable-oriented approach: Are they complementary, opposites, or exploring different worlds? *Merrill-Palmer Quarterly: Journal of Developmental Psychology*, *52*, 601–632.

Birkhoff, G. (1931). Proof of the ergodic theorem. *Proceedings of the National Academy of Science USA*, *17*, 656–660.

Bollen, K. A., & Curran, P. J. (2006). *Latent curve models: A structural equation approach*. New York, NY: Wiley.

Bontempo, D., Grouzet, F., & Hofer, S. (2012). Measurement issues in the analysis of within-person change. In J. Newson, R. Jones, & S. Hofer (Eds.), *Longitudinal data analysis: A practical guide for researchers in aging* (pp. 97–142). New York, NY: Routledge.

Borkenau, P., & Ostendorf, F. (1998). The big five as states: How useful is the five-factor model to describe intraindividual variations over time? *Journal of Research in Personality, 32,* 202–221. doi: 10.1006/jrpe.1997.2206

Bryk, A., & Raudenbush, S. (1992). *Hierarchical linear models: Applications and data analysis methods*. Newbury Park, CA: Sage Press.

Cattell, R. B. (1952). The three basic factor-analytic designs—Their interrelations and derivatives. *Psychological Bulletin*, *49*, 499–520. doi: 10.1037/h0054245

Cattell, R. B. (1966a). The data box: Its ordering of total resources in terms of possible relational systems. In R. B. Cattell (Ed.), *Handbook of multivariate experimental psychology* (pp. 67–128). Chicago, IL: Rand-McNally.

Cattell. R. B. (1966b). The scree test for the number of factors. *Multivariate Behavioral Research*, *1*, 245–276. doi: 10.1207/s15327906mbr0102_10

Chakraborty, E., & Moodie, E. E. M. (2013). *Statistical methods for dynamic treatment regimes: Reinforcement learning, causal inference, and personalized medicine*. New York, NY: Springer.

Cicchetti, D., & Toth, S. L. (2009). The past achievements and future promises of developmental psychopathology: The coming of age of a discipline. *Journal of Child Psychological Psychiatry*, *50*(1–2), 16–25. doi: 10.1111/j.1469-7610.2008.01979.x.

Corneal, S. E., & Nesselroade, J. R. (1991). Multivariate, replicated, single-subject, repeated measures design: Studying change in the adolescent. In R. Lerner, A. Petersen, & J. Brooks-Gunn (Eds.), *Encyclopedia of adolescence* (pp. 681–687). New York, NY: Garland.

Corneal, S. E., & Nesselroade, J. R. (1994). A stepchild's emotional experience across two households: An investigation of response patterns by P-technique factor analysis. *Multivariate Experimental Clinical Research, 10*(3), 167–180.

Deegan, P. E., Rapp, C., Holter, M., & Riefer, M. (2008). A program to support shared decision-making in an outpatient psychiatric medication clinic. *Psychiatric Services*, *59*, 603–605.

Ford, D. H., & Lerner, R. M. (1992). *Developmental systems theory: An integrative approach*. Newbury Park, CA: Sage.

Gates, K. M., & Molenaar, P. C. M. (2012). Group search algorithm recovers effective connectivity maps for individuals in homogeneous and heterogeneous samples. *NeuroImage*, *63*, 310–319. doi: 10.1016/j.neuroimage.2012.06.026

Gates, K. M., Molenaar, P. C. M., Hillary, F. G., Ram, N., & Rovine, M. J. (2010). Automatic search for fMRI connectivity mapping: An alternative to Granger causality testing using formal equivalences among SEM path modeling, VAR and unified SEM. *NeuroImage*, *50*, 1118–1125. doi: 10.1016/j.neuroimage.2009.12.117

Goldstein, H. (1995). *Multilevel statistical models* (2nd ed.). London, UK: Edward Arnold.

Gonzales, J. E., & Ferrer, E. (2014). Individual pooling for group-based modeling under the assumption of ergodicity. *Multivariate Behavioral Research, 49*(3), 245–260. doi: 10.1080/00273171.2014.902298

Gorsuch, R. L. (1983). *Factor analysis* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum.

Gottlieb, G. (2001). *Individual development and evolution: The genesis of novel behavior*. Mahwah, NJ: Lawrence Erlbaum.

Granger, C. W. J. (1969). Investigating causal relations by econometric models and cross-spectral methods. *Econometrica, 37*(3), 424–438.

Grimm, K. J., Davoudzadeh, P., & Ram, N. (2015, in press). Longitudinal analysis. In N. Card (Ed.), *Developmental methodology. Society for Research in Child Development Monograph* (pp. 82–116). New York, NY: Wiley.

Hamaker, E. L. (2012). Why researchers should think "within-person": A paradigmatic rationale. In M. R. Mehl & T. S. Conner (Eds.), *Handbook of methods for studying daily life* (pp. 43–61). New York, NY: Guilford Publications.

Hamburg, M. A., & Collins, F. S. (2010). The path to personalized medicine. *New England Journal of Medicine*, *363*, 301–304. doi: 10.1056/NEJMp1006304

Harman, H. (1976). *Modern factor analysis* (3rd ed., revised). Chicago, IL: University Chicago Press.

Hatemi, J. A. (2012). Asymmetric causality tests with an application. *Empirical Economics*, *43*(1), 447–456. doi: 10.1007/s00181-011-0484-x

Henderson, C. R. (1975). Best linear unbiased estimation and prediction under a selection model. *Biometrics, 31*, 423–447.

Jöreskog, K. G. (1978). Structural analysis of covariance and correlation matrices. *Psychometrika, 43*(4), 443–477. doi: 10.1007/BF02293808

Jöreskog, K. G., & Sörbom, D. (1996). *LISREL8: User's reference guide*. Chicago, IL: Scientific Software International.

Kenny, D. A. (2005). Cross-lagged panel design. In B. S. Everitt & D. Howell (Eds.), *Encyclopedia of statistics in behavioral science*. New York, NY: Wiley. doi: 10.1002/9781118445112.stat06464

Kolmogorov, A. (1931). Über die analytischen Methoden in der Wahrscheinlichkeitsrechnung. *Mathematische Annalen*, *104*, 415–458.

Kuppens, P., Allen, N. B., & Sheeber, L.B. (2010). Emotional inertia and psychological maladjustment. *Psychological Science*, *21*, 984–991. doi: 10.1177/0956797610372634

Lickliter, R., & Honeycutt, H. (2009). Rethinking epigenesis and evolution in light of developmental science. In M. Blumberg, J. Freeman, & S. Robinson (Eds.), *Oxford handbook of developmental behavioral neuroscience* (pp. 30–47). New York, NY: Oxford University Press.

Littell, R. C., Milliken, G. A., Stroup, W. W., Wolfinger, R. D., & Schabenberger, O. (2006). *SAS for mixed models*. Cary, NC: SAS Institute.

Liu, S., Rovine, M. J., & Molenaar, P. C. M. (2012). Selecting a linear model for longitudinal data: Repeated measures analysis of variance, covariance pattern models and growth curve approaches. *Psychological Methods*, *17*(1), 15–30. doi: 10.1080/10705511.2012.726918

Lutkepohl, H. (2006). *New introduction to multiple time series analysis*. New York, NY: Springer.

McArdle, J. J., & Aber, M. S. (1990). Patterns of change within latent variable structural equation modeling. In A. von Eye (Ed.), *New statistical methods in developmental research* (pp. 151–224). New York, NY: Academic Press.

McArdle, J. J., & Epstein, D. (1987). Latent growth curves within developmental structural equation models. *Child Development, 58*, 110–133. doi: 10.2307/1130295

McArdle, J. J, & Hamagami, E. F. (1991). Modeling incomplete longitudinal and cross sectional data using latent growth structural models. In L. Collins & J. L. Horn (Eds.), *Best methods for the analysis of change* (pp. 276–304). Washington, DC: APA Press.

Meredith, W. (1993). Measurement invariance, factor analysis, and factorial invariance. *Psychometrika, 58*, 525–542.

Millsap, R. E. (2011). *Statistical approaches to measurement invariance*. New York, NY: Routledge.

Millsap, R. E., & Kwok, O. (2004). Evaluating the impact of partial invariance on selection in two populations. *Psychological Methods*, *9*(1), 93–115.

Millsap, R. E., & Meredith, W. (2004). *Factorial invariance: Historical trends and new developments*. Paper presented at the Factor Analysis at 100 Conference, L.L Thurstone Psychometric Laboratory, University of North Carolina.

Mitteness, L. S., & Nesselroade, J. R. (1987). Attachment in adulthood: Longitudinal investigation of mother–daughter affective interdependencies by P-technique factor analysis. *Southern Psychologist, 3,* 37–44.

Molenaar, P. C. M. (1985). A dynamic factor model for the analysis of multivariate time series. *Psychometrika, 50*, 181–202. doi: 10.1007/BF02294246

Molenaar, P. C. M. (1987). Dynamic assessment and adaptive optimisation of the therapeutic process. *Behavioral Assessment*, *9*, 389–416.

Molenaar, P. C. M. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement: Interdisciplinary Research and Perspectives, 2*, 201–218. doi: 10.1207/s15366359mea0204_1

Molenaar, P. C. M. (2010a). Testing all six person-oriented principles in dynamic factor analysis. *Development and Psychopathology*, *22*, 255–259. doi: 10.1017/S0954579410000027

Molenaar, P. C. M. (2010b). Note on optimization of individual psychotherapeutic processes. *Journal of Mathematical Psychology*, *54*, 208–213. doi: 10.1016/j.jmp.2009.04.003

Molenaar, P. C. M., & Campbell, C. G. (2009). The new person-specific paradigm in psychology. *Current Directions in Psychological Science*, *18*, 112–117. doi: 10.1111/j.1467-8721.2009.01619.x

Molenaar, P. C. M., Lerner, R., & Newell, K. M. (Eds.). (2014). *Handbook of developmental systems theory and methodology*. New York, NY: Guilford Press.

Molenaar, P. C. M., & Nesselroade, J. R. (2001). Rotation in the dynamic factor modeling of multivariate stationary time series. *Psychometrika, 66*(1), 99–107. doi: 10.1007/BF02295735

Molenaar, P. C. M., & Nesselroade, J. R. (2009). The recoverability of P-technique factor analysis. *Multivariate Behavioral Research, 44,* 130–141.

Molenaar, P. C. M., & Newell, K. M. (Eds.) (2010). *Individual pathways of change: Statistical models for analyzing learning and development*. Washington, DC: American Psychological Association.

Molenaar, P. C. M., Sinclair, K. O., Rovine, M. J., Ram, N., & Corneal, S. E. (2009). Analyzing developmental processes on an individual level using non-stationary time series modeling, *Developmental Psychology*, *45*(1), 260–271. doi: 10.1037/a0014170

Muthén, B., & Shedden, K. (1999). Finite mixture modeling with mixture outcomes using the EM algorithm. *Biometrics, 55*, 463–469. doi: 10.1111/j.0006-341X.1999.00463.x

Nagin, D. S. (1999). Analyzing developmental trajectories: A semiparametric group-based approach. *Psychological Methods, 4*, 139–157. doi: 10.1037/1082-989X.4.2.139

Nesselroade, J. R., & Jones, C. J. (1991). Multimodal selection effects in the study of adult development: A perspective on multivariate, replicate, single subject repeated measures. *Experimental Aging Research*, *11*, 21–27.

Nesselroade, J. R., Gerstorf, D., Hardy, S. A., & Ram, N. (2007). Idiographic filters for psychological constructs. *Measurement: Interdisciplinary Research and Perspectives*, *5*, 217–235. doi: 10.1080/15366360701741807

Nesselroade, J., McArdle, J., Aggen, S., & Meyers, J. (2002). Alternative dynamic factor models for multivariate time series. In D. M. Moscowitz & S. L. Hershberger (Eds.), *Modeling intraindividual variability with repeated measures data: Advances and techniques* (pp. 325–365). Mahwah, NJ: Lawrence Erlbaum Associates.

Nesselroade, J. R., & Molenaar, P. C. M. (1999). Pooling lagged covariance structures based on short, multivariate time series for dynamic factor analysis. In R. H. Hoyle (Ed.), *Statistical strategies for small sample research* (pp. 223–250). Thousand Oaks, CA: Sage Publications, Inc.

Ptasznik, A. (2011). The person in personal medicine: Moving consumers from periphery to centre of decision making. *Journal of Addiction and Mental Health*, *14*(3), 14–22.

Raiche, G., Riopel, M., & Blais, J. G. (2006). *Non graphical solutions for the Cattell's scree test*. Paper presented at the International Annual Meeting of the Psychometric Society, Montreal, Canada.

Rao, C. R. (1958). Some statistical methods for the comparison of growth curves. *Biometrics*, *14*, 1–17.

Reise, S. P., Widaman, K. F., & Pugh, R. H. (1993). Confirmatory factor analysis and item response theory: Two approaches for exploring measurement invariance. *Psychological Bulletin, 114*, 552–566.

Robinson, G. K. (1991). The BLUP is a good thing: the estimation of random effects. *Statistical Science*, *6*(1), 15–32. doi: 10.1214/ss/1177011926

Roebroeck, A., Formisano, E., & Goebel, R. (2005). Mapping directed influence over the brain using Granger causality and fMRI. *NeuroImage*, *25*, 230–242. doi: 10.1016/j.neuroimage.2004.11.017

Rovine, M. J., Molenaar, P. C. M., & Corneal, S. E. (1999). Analysis of emotional response patterns for adolescent stepsons using P-technique factor analysis. In R. K. Silbereisen & A. von Eye (Eds.), *Growing up in times of social change* (pp. 261–285). Berlin, Germany: De Gruyter.

Rovine, M. J., & Molenaar, P. C. M. (2000). A structural modeling approach to the random coefficients model. *Multivariate Behavioral Research, 35*(1), 51–88. doi: 10.1207/s15327906mbr3501_3

Rovine, M. J., & Walls, T. A. (2006). A multilevel autoregressive model to describe interindividual differences in the stability of a process. In J. L. Schafer & T. A. Walls (Eds.), *Models for intensive longitudinal data* (pp. 124–147). New York, NY: Oxford.

Schwartz, D., & Collins, F. S. (2007). Environmental biology and human disease. *Science*, *316*, 695–696.

Seth, A. K. (2008). Causal networks in simulated neural systems. *Cognitive Neurodynamics*, *2*(1), 49–64. doi: 10.1007/s11571-007-9031-z

Smith, L.B., & Thelen, E. (2003). Development as a dynamic system. *Trends in Cognitive Sciences*, *7*, 343–348.

Smith, S. M., Miller, K. L., Salimi-Korshidi, G., Webster, M., Beckman, C. F., Nichols, T. E., Ramsey, J. D., & Woolrich, M. V. V. (2011). Network modeling methods for fMRI. *NeuroImage*, *54*(2), 875–891. doi: 10.1016/j.neuroimage.2010.08.063

Sörbom, D. (1974). A general method for studying differences in factor means and factor structures between groups. *British Journal of Mathematical and Statistical Psychology*, *27*(2), 229–239. doi: 10.1111/j.2044-8317.1974.tb00543.x

Sterba, S. K., & Bauer, D. J. (2010). Matching method with theory in person-oriented developmental psychopathology research. *Development and Psychopathology*, *22*, 239–254. doi: 10.1017/S0954579410000015

Suls, J., Green, P., & Hillis, S. (1998). Emotional reactivity to everyday problems, affective inertia, and neuroticism. *Personality and Social Psychology Bulletin*, *24*, 127–136. doi: 10.1177/0146167298242002

Thurstone, L. L. (1947). *Multiple factor analysis*. Berkeley, CA: University of California Press.

Tucker, L. (1958). Determination of parameters of a functional relation by factor analysis. *Psychometrika, 23*, 19–23. doi: 10.1007/BF02288975

Vandenberg, R. J., & Lance, C. E. (2000). A review and synthesis of the measurement invariance literature: Suggestions, practices, and recommendations for organizational research. *Organizational Research Methods, 3*(1), 4–69. doi: 10.1177/109442810200500201

Voelkle, M. C., Brose, A., Schmiedek, F., & Lindenberger, U. (2014). Toward a unified framework for the study of between-person and within-person structures: Building a bridge between two research paradigms. *Multivariate Behavioral Research*, *49*(3), 193–213. doi: 10.1080/00273171.2014.889593

Walls, T. A., & Schafer, J. L. (2006). *Models for intensive longitudinal data*. New York, NY: Oxford University Press.

Wang, L., Hamaker, E. L., & Bergeman, C. (2012). Investigating interindividual differences in short term intraindividual variability. *Psychological Methods*, *17*(4), 567–581. doi: 10.1037/a0029317

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063–1070. doi: 10.1037/0022-3514.54.6.1063

Widaman, K. F., & Reise, S. P. (1997). Exploring the measurement invariance of psychological instruments: Applications in the substance use domain. In K. J. Bryant, M. Windle, & S. G. West (Eds.), *The science of prevention: Methodological advances from alcohol and substance abuse research* (pp. 281–324). Washington, DC: American Psychological Association.

Wood, P., & Brown, D. (1994). The study of intraindividual differences by means of dynamic factor models: Rationale, implementation, and interpretation. *Psychological Bulletin, 116,* 166–186. doi: 10.1037/0033-2909.116.1.166

Wright, S. (1934). The method of path coefficients. *Annals of Mathematical Statistics*, *5*, 161–215.

Zevon, M. A., & Tellegen, A. (1982). The structure of mood change: An idiographic/nomothetic analysis. *Journal of Personality and Social Psychology, 43*, 111–122. doi: 10.1037/0022-3514.43.1.111

CHAPTER 20

Configural Frequency Analysis for Research on Developmental Processes

ALEXANDER VON EYE and EUN-YOUNG MUN

INTRODUCTION 866
Introduction to Configural Frequency Analysis 868
CFA MODELS FOR DEVELOPMENTAL RESEARCH 877
CFA of Differences I 878
CFA of Differences II: Structural Zeros 881
Extensions 883
CFA of Trajectories: Typical and Atypical Development 884
Analyzing Series That Differ in Length 886
CFA of Pre–Post Designs—An Application of Confirmatory CFA 889
Treatment Effects in Designs with Control Group 892
Single-Subject Designs 895
CFA of Lags 900
CFA of Cross-Lagged Designs 905
Comparing Individuals' Trajectories 908
Predicting Events 912
PREDICTING END POINTS OF DEVELOPMENT 912
Predicting a Trajectory 915
DISCUSSION 916
Unique Characteristics of CFA 917
CFA and Translational Research 917
REFERENCES 919

INTRODUCTION

Developmental processes, either typical or atypical, are inherently about changes that occur over time in response to internal and external stimulation at the individual level. Recent development of statistical methods of analysis has resulted in a situation that is quite interesting and encouraging for researchers of developmental processes because both type and number of questions that can be answered has increased dramatically. This increased capacity allows researchers to answer scholarly questions that seemed intractable just a few years ago. Just think of dynamic modeling, modeling of trajectories and mixtures, the new models and methods for intensive longitudinal data, and for categorical or nonnormal data, or the new approaches to the analysis of causal hypotheses (for overviews, see Everitt & Howell, 2005; Laursen, Little, & Card, 2012; von Eye & Mun, 2013; von Eye & Wiedermann, 2013). The doors for sophisticated data analysis and, more important, for analysis that exactly tests the hypotheses that researchers ask are wide open.

[1]Color versions of Figures 20.1, 20.5–20.7 are available at http://onlinelibrary.wiley.com/book/10.1002/9781119125556

By the same token, however, this situation causes worry. A disconnect between theories of development and methodological application has also emerged. Theorists of person-oriented research (see Bergman & Magnusson, 1997; von Eye & Bergman, 2003; von Eye, Bergman, & Hsieh, 2015; see also Ch. 18) as well as idiographic psychology (Molenaar, 2004; Molenaar & Campbell, 2009) have suggested and shown that the results of typical statistical analysis are not applicable at the level of individuals. This is because most statistical methods perform analysis on aggregated data (e.g., means and covariances). A prominent example is the analysis of covariance matrices in structural equation modeling. These matrices contain variances in their diagonal cells and covariances in their off-diagonal cells. By definition, these aggregate measures indicate variability that is averaged over the individual cases included in a study. The cases themselves are considered random and, thus, replaceable (by other random cases).

Because of these characteristics, it is rare that any results from aggregated data do indeed apply to the individual. In longitudinal research, in particular dynamic modeling, very strict ergodicity conditions must be fulfilled for the results of aggregate-level, statistical inference to be applicable to the individual (see Loken, 2010; Molenaar, 2004; von Eye, Bergman, & Hsieh, 2015). Examples of such rigid conditions

include stationarity, that is, the data follow a stochastic process whose joint probability distribution is not sensitive to shifts in time and space. The parameters of such stochastic processes, for example, the mean and variance, will also be insensitive to shifts in time and space. Clearly, most developmental processes violate the stationarity condition. Note that we argue not that any data analytic techniques based on aggregate level data are not useful but that these characteristics necessitate that one needs to be aware of these assumptions and be cautious in interpreting results.

To be less confined and be truer to theories, what is needed, therefore, are methods of statistical data analysis that have the following two characteristics. First, they allow one to make statements about individuals or groups of individuals. Second, they do not require that improbable conditions for the process under study be met. *Configural frequency analysis* (CFA; Lienert & Krauth, 1975; von Eye, 2002; von Eye & Gutiérrez-Peña, 2004; von Eye, Mair, & Mun, 2010) is such a method. In this chapter, we introduce readers to CFA and give examples of questions that can be answered using this method. These questions differ from those that can be answered using standard methods of analysis. Instead of targeting relationships among variables, they target individual or groups of configurations, that is, individual profiles.

Before we discuss the technical aspects of CFA, its application and computation, we ask, however, where the disconnect between developmental theories and analytic practices has occurred in the field of developmental research. The answer to this question can be used to locate or develop statistical methods that do not suffer from the problems mentioned previously. To answer this question, we use the example of Eysenck's (1958) approach to the development of a personality theory (von Eye & Bogat, 2006). As is illustrated in Figure 20.1, Eysenck's approach involves proceeding through a hierarchy of steps.

At the lowest level of the hierarchy, indicated by the boxes at the bottom of the figure, researchers focus on what Eysenck terms *specific reactions*. These are behaviors that are observed for an individual, at a particular moment. For example, a child may act in defiance of his/her mother's instruction to clean up after playing with toys. Specific reactions are just observed and recorded. No evaluation or comparison is involved or intended. The next higher level in the hierarchy is that of *habitual reactions*. For example, the child tends to exhibit hostile behaviors toward the mother whenever instructed to clean up after playing. At the level of habitual reactions, ipsative comparisons (e.g., the same individual's behavior compared with that from a day or week before) but also comparisons with other individuals (e.g., different individuals at a given moment) can be made. In more general terms, at this level, statements are already made that go beyond specific observations. The third level is that of *personality characteristics such as perseverance, rigidity, or irritability*. At this level, statements are made that carry the researcher beyond a particular behavior observation, even beyond the individual that was observed. For example, one can talk about communication style preferences, regardless of the individuals who may or may not exhibit these preferences. At the highest level, *personality types* are defined. These types are based on grouping personality characteristics. In Eysenck's theory, we find the type of extraverted versus introverted personality characteristics, the type of stable versus labile personality characteristics, and the type of psychoticism.

Figure 20.1 shows where the disconnect occurs: between the levels of habitual reactions and personality characteristics (separated by the dotted line). Below the dotted line, descriptive or comparative statements are made about individuals. Above the dotted line, statements describe personality characteristics. These statements are abstract, and references to the individual are not necessarily implied. Standard statistical analysis relates personality characteristics to one another (cluster analysis of cases being among the few exceptions). In contrast, CFA analyzes individuals with particular profiles. Specifically, CFA creates all possible profiles for a set of categorical variables, and answers the question whether individual profiles were observed at different rates than expected from prior knowledge or theory. Analyses that focus on the information provided above the dotted line (variables) have been termed *variable oriented*. Analyses that focus on the information

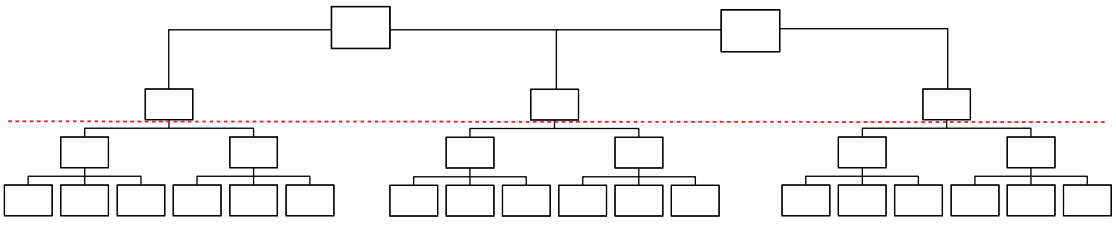


Figure 20.1 Constructing models of personality. See footnote 1. Source: Eysenck (1958).

provided below the dotted line (individuals) have been termed *person oriented* (see Bergman & Magnusson, 1997; von Eye, Bergman, & Hsieh, 2015; Ch. 18). CFA is regarded as among the prime methods of analysis for person-oriented research (Bergman, Magnusson, & El-Khouri, 2003). Although person-oriented research requires many other considerations, such as study design and assessment, this chapter focuses on CFA, a particular analytical approach that is useful in person-oriented research.

CFA proceeds from the person-oriented assumption that development is best described by patterns of characteristics (instead of variable relations). This is most relevant for research in developmental psychopathology, where we find that the development of pathological processes can be very specific to particular groups of individuals, causes, timing, and circumstances. This applies accordingly to translational and intervention research. These topics are taken up again later in this chapter.

This chapter is structured as follows. First, we introduce readers to CFA. We cover conceptual and technical aspects of CFA. This introduction will be selective. More detail can be found in von Eye (2002) and von Eye, Mair, and Mun (2010). After this introduction, we present models of CFA that are of importance for research in developmental psychopathology. Throughout, examples will be given with real data.

Introduction to Configural Frequency Analysis

CFA was proposed by Lienert in 1968 as a method that is particularly suited for clinical research and has since been developed further. CFA allows researchers to answer the question whether, given explicit expectations, individuals with certain profiles were observed at higher or lower rates than expected. The answers to this question can be used to provide support for a hypothesis or a theory. In addition, CFA can test whether the definition or the operationalization of a concept is valid. To give an example, consider a certain mood that is associated with depression. Individuals diagnosed as depressed often feel sad, anxious, hopeless, worthless, guilty, or irritable. In contrast, individuals diagnosed as depressed rarely feel happy or proud. Therefore, the null hypothesis stipulating that sad and happy moods can coexist in time within a person will, in an empirical study, be rejected. The two variables sadness and happiness are related such that the individual mood profile [sad, happy] is expected with below chance probability. Interestingly, it can also be discussed whether the profiles [not sad, happy], [not sad, unhappy], and [sad, unhappy] will be observed with above or below chance probability. In CFA, individual profiles are examined.

CFA is used to analyze cross-classifications of categorical variables. In this respect, CFA is comparable with log-linear modeling (Bishof, Fienberg, & Holland, 1975; von Eye & Mun, 2013), logistic regression (see Agresti, 1998), or latent class analysis (see Goodman, 1974). CFA differs, however, from these methods in that it does not attempt to explain the relations among variables. It allows one to examine individual cells of cross-classifications (also called *configurations*, *cells*, *patterns*, or *profiles*) with the question whether their occurrence frequencies differ from expectancy. Groups of cells can also be examined.

More generally, cross-classifications of categorical variables can be analyzed with specific aims in mind. Goodman (1984) listed the aims of examining

1. the joint distribution of the variables that span a cross-classification;
2. the association structure of these variables; and
3. the dependency structure of these variables. CFA adds a fourth aim. It asks whether individual cells or groups of cells deviate from the expectancy that is specified using a probability model. Later in this chapter, we describe in more detail how a CFA probability model is specified. Von Eye and Gutiérrez-Peña (2004) note that CFA is used to identify those *cells of a cross-classification where the action is*. In these cells, *local associations* (Havránek & Lienert, 1984) become apparent. That is, in particular cells, there is an association among the variables that span a cross-classification, as is manifested by above-chance or below-chance frequencies of observations. In other cells, observations are as frequent as expected with reference to the probability model (also called *base model*). Cells that contain more cases than expected are labeled as constituting a *CFA type*. Cells that contain fewer cases than expected are labeled as constituting a *CFA antitype*.

To illustrate these concepts, we present a first data example. The data in this example were collected in the context of the Mother–Infant Study (MIS; Bogat, Levendosky, DeJonghe, Davidson, & von Eye, 2004; Huth-Bocks, Levendosky, & Bogat, 2002). The study first assessed women in their last trimester of pregnancy and again at 2 months postpartum and followed up the women and their children every year until the children were 14 years old. The women in the study reported a range of experiences of intimate partner violence, from none to severe. Intimate partner violence was defined as

male violence toward a female partner. Research questions concerned factors that predict risk and resilience in women exposed to intimate partner violence and their children as well as aspects of the home environment (e.g., maternal mental health, parenting style); and individual child characteristics (e.g., temperament) that predict maladaptive socioemotional outcomes (e.g., aggression; for results, see, e.g., Bogat et al., 2004; Huth-Bocks, Levendosky, Bogat, & von Eye, 2004; www.msu.edu/∼mis/).

For the first data example, we use the variables posttraumatic stress disorder (PTSD) symptoms (P), Income (I), and Depression (D) observed at the first occasion, that is, in the last trimester of pregnancy. The variables were binned close to their median. For each of the variables, 1 indicates below the cutoff and 2 above the cutoff. Table 20.1 displays the CFA results for the $2 \times 2 \times 2$, P × I × D cross-classification. Without going into the technical details, which will be introduced later, we report that this table was analyzed under the base model of variable independence, standard normal z scores were used to evaluate the individual cells, and α was protected using the Holland–Copenhaver procedure.

The overall goodness-of-fit likelihood ratio X^2 for the base model of variable independence for the data in Table 20.1 is 45.60. For $df = 4$, this value suggests that the model is rejected ($p < 0.01$). This is the typical situation for a CFA that results in types and antitypes. In Table 20.1, we see two types and one antitype. The first type is constituted by configuration 1 2 1. These are women whose PTSD and Depression symptom scores were below the cutoff, and income was above the cutoff. Significantly more women showed this profile than expected with reference to the base model of variable independence. The second type, constituted by configuration 2 1 2, shows just the opposite profile. These are women with elevated PTSD and Depression symptom scores and Income below the cutoff. Again, significantly more women than expected showed this profile.

TABLE 20.1 First-Order CFA of the Cross-Classification of PTSD (P), Income (I), and Depression (D)

	Configuration				
PID	m	$\hat{m}$	Statistic	p	
111	28.00	37.317	−1.5251	.063612	
112	12.00	15.847	−.9663	.166938	
121	53.00	30.770	4.0076	.000031	Type
122	4.00	13.067	−2.5082	.006067	Antitype
211	37.00	42.703	−.8727	.191425	
212	37.00	18.134	4.4303	.000005	Type
221	28.00	35.211	−1.2152	.112144	
222	9.00	14.953	−1.5394	.061855	

The sole antitype in Table 20.1 is constituted by configuration 1 2 2. The women with this profile exhibit below-the-cutoff scores in PTSD, and above-the-cutoff scores in Income and Depression. This profile was observed four times, which is significantly below the expected frequency of about 13.

The results shown in Table 20.1 are typical of CFA results in a number of aspects. First, in Table 20.1, the largest cell does constitute a type and the smallest cell does constitute an antitype. This is not always the case. The main reason for this characteristic of CFA results is that CFA focuses on *discrepancies from expectation* instead of sheer size of cells (the base model of zero-order CFA is the only exception; Lienert & von Eye, 1989; von Eye, 2002). Therefore, even relatively small cells can contain more cases than expected, and relatively large cells can contain fewer cases than expected. Later in this chapter, Table 20.12 shows an example in which one of the smallest cells constitutes a type.

Second, CFA tables of types and antitypes are interpreted after the base model is rejected. However, we note that the rejection of a base model does not guarantee that types and antitypes exist. If a base model describes the data well, there will be no noteworthy discrepancies between observed and expected cell frequencies, and the search for types and antitypes that indicate the location of significant discrepancies is largely pointless. And finally, only a selection of cells (configurations) emerges as type or antitype constituting. The remaining cells do not statistically deviate from the base model.

In the following sections, we describe technical elements of CFA (for more detail, see von Eye, 2002; von Eye & Gutiérrez-Peña, 2004; von Eye, Mair, & Mun, 2010).

CFA: Technical Elements

We begin the description of technical elements of CFA with a discussion of CFA base models. These models are of crucial importance because the interpretation of CFA types and antitypes depends on the effects that are (not) included in the base model. In general, the base model includes all effects that are not of interest to the researcher. If the base model is rejected, at least some of the effects the researcher is interested in are bound to exist. Naturally, *different base models can lead to different expected cell frequencies* (Mellenbergh, 1996) and, thus, different patterns of types and antitypes. Therefore, the selection of base models is not arbitrary.

Definition of CFA Base Models. Most CFA base models are log-linear models (for base models that are not

log-linear, see von Eye, 2002). Therefore, expected cell frequencies for most CFA models can be estimated using the log-frequency model

$$\log \hat{m} = X\lambda$$

where $\hat{m}$ is the array of model frequencies, that is, frequencies that conform to the model specifications. X is the *design matrix* (also called *indicator* matrix). Its vectors reflect the CFA base model or, in other contexts, the log-linear model under study. λ is the vector of model parameters. These parameters are not of primary interest in frequentist CFA. Instead, CFA focuses on the discrepancies between the expected and the observed cell frequencies. In contrast to log-linear modeling, CFA is not applied with the goal to identify a model that describes the data sufficiently and parsimoniously. Rather, CFA is applied with the goal of identifying the configurations that contradict the base model. These configurations reflect the effects of interest.

CFA base models have three main characteristics (von Eye, 2004a):

1. They contain all effects that are *not* of interest to the researcher;
2. They reflect theoretical assumptions concerning the nature of the variables as either of equal status or grouped into predictors and criteria; and
3. Consider the sampling scheme under which the data were collected. In the following paragraphs, we discuss two of these three characteristics. We begin with the third.

In practically all CFA applications, the sampling scheme is either *multinomial* or *product-multinomial*. When the scheme is multinomial, cases can be placed in any cell of a cross-classification, with no constraints. Marginal probabilities of the data collected under this sampling scheme are neither a priori given nor determined. In contrast, when the sampling scheme is product-multinomial, marginal probabilities of the data collected using this sampling scheme are given a priori. So, when a study focuses on the effects of smoking, researchers may wish to implement a balanced design such that equal numbers of smokers and nonsmokers go into the table. In this case, no additional smokers can enter the study when this subsample is filled. From a data analytic perspective, the sampling scheme has no implications for parameter estimation. However, the number and type of base models that can be estimated are restricted under the product-multinomial sampling scheme (for more detail, see von Eye et al., 2010).

The first of these characteristics is most important for the interpretation of types and antitypes. A CFA base model contains all effects that are *not* of interest to the researcher. Consider two examples. The base model used for the analysis reported in Table 20.1 was the model of variable independence. That is, the base model included only the main effects of the three variables PTSD, Income, and Depression. This model prevails only if the three variables are indeed independent of one another. If, however, one or more of the three possible two-way interactions, or the three-way interaction among all three variables exist, the model is rejected. In other words, from the fact that the base model of variable independence is rejected, we conclude that interactions exist. From the emergence of types and antitypes and the existence of configurations that do not contradict the base model, we conclude that local associations exist (Havránek, & Lienert, 1984). Types and antitypes that result from the bae model of variable independence thus indicate that interactions exist and where the interactions exist.

For the second example, consider the base model of Prediction CFA (PCFA; Heilmann & Lienert, 1982; Heilmann, Lienert, & Maly, 1979; Lienert & Krauth, 1973a; von Eye, Mair, & Bogat, 2005; von Eye & Rovine, 1994). This base model includes two sets of effects. The first is defined by all possible effects on the predictor side. The model is thus saturated within the predictors. Types and antitypes cannot emerge just because predictors are related to one another. Second and in parallel, the base PCFA model is also saturated in the criterion variables. Therefore, types and antitypes cannot emerge just because criterion variables are related to one another. Third, the PCFA base model proposes independence between the two sets of variables: predictors and criterion variables. If this model is rejected, types and antitypes, by necessity, reflect relationships that link predictor variables with criterion variables. This characteristic is exemplified when we specify log-linear CFA base models, in the following paragraphs.

For the purposes of this chapter and in correspondence with the literature on log-linear modeling and CFA, we use two ways to specify a CFA base model. The first is the *log-linear notation*. Its main characteristic is that every main effect, interaction, special effect, and covariate are represented in the equation. Consider the base model for the example in Table 20.1. This model contains only the main effects of P, I, and D. The general log-linear model $\log \hat{m} = X\lambda$ is then, in this particular case,

$$\log \hat{m} = \lambda + \lambda^P + \lambda^I + \lambda^D$$

where $\hat{m}$ is the vector of expected cell frequencies (model frequencies), λ is the intercept, and the superscripted λs represent the main effects of the variables *P, I*, and *D*, respectively. In this particular case, each λ corresponds to one parameter. When effects require more than one parameter, which is the case when variables have more than two categories, subscripts are used to differentiate these categories.

To illustrate the PCFA base model, consider the two predictors, *P*1 and *P*2, and the two criterion variables, *C*1 and *C*2. The log-linear base model for these four variables is

$$\log \hat{m} = \lambda + \lambda^{P1} + \lambda^{P2} + \lambda^{C1} + \lambda^{C2} + \lambda^{P1,P2} + \lambda^{C1,C2}$$

The first line of this equation contains the main effects of all variables in the model. The second line contains the interaction between the two predictor variables and the interaction between the two criterion variables. All log-linear base models can be expressed using this notation.

In addition to the log-linear notation, we use the *bracket notation*. This notation includes each effect in a pair of brackets. For main effects, a bracket pair will include just one variable. For two-way interactions, the bracket pair includes two variables, and so forth. The base model of variable independence used for Table 20.1 contains three main effects. In bracket notation, this model is [*P*], [*I*], [*D*]. The PCFA base model is, for the last example, [*P*1], [*P*2], [*C*1], [*C*2], [*P*1, *P*2], [*C*1, *C*2].

Many CFA base models are hierarchical log-linear models. In these hierarchical models, the lower order relatives of higher order terms are implied. Therefore, these lower order terms can be omitted in the model specification. They are included in the model, but, in the specification, they are redundant. The base model of variable independence cannot be simplified further, because the main effects are the lowest order effects in a log-linear model. In contrast, the PCFA base model can be simplified to [*P*1, *P*2], [*C*1, *C*2]. In this chapter, to be consistent with the CFA literature, and because some CFA base models are not hierarchical, we specify base models so that every effect under consideration is made explicit.

We now ask which effects can be the causes for types and antitypes in the base model of variable independence. As we said before, these are effects that represent interactions. In the first data example, these are the effects [*P, I*], [*P, D*], [*I, D*], and [*P, I, D*], that is three two-way and one three-way interactions. If any of these effects exists, types and antitypes can emerge from the base model [*P*], [*I*], [*D*].

For the PCFA base model of the second example, the following effects may exist and cause types and antitypes to emerge:

- Two-way interactions: [*P*1, *C*1], [*P*1, *C*2], [*P*2, *C*1], and [*P*2, *C*2];
- Three-way interactions: [*P*1, *P*2, *C*1], [*P*1, *P*2, *C*2], [*P*1, *C*1, *C*2], and [*P*2, *C*1, *C*2];
- Four-way interaction: [*P*1, *P*2, *C*1, *C*2].

Each of these interaction terms contains at least one predictor and at least one criterion variable. Types from PCFA indicate which predictor configurations allow one to predict the occurrence of which criterion configuration. PCFA antitypes allow one to predict which criterion configuration cannot be expected to occur after which predictor configuration. Given the right context, causal inference may be possible. Types could then be interpreted as predictor configurations that cause criterion configurations to occur. Antitypes could then be interpreted as predictor configurations that prevent criterion configurations from occurring.

Classes of CFA Base Models

Two classes of log-linear CFA base models have been discussed: *global* models and *regional models* (von Eye, 1988, 2002). Global models share the characteristic that all variables have the same status. There are no predictors, no criterion variables, no mediators or moderators. This situation is comparable to standard exploratory factor analysis in which all variables have the same status as well.

There is a hierarchy of global CFA base models. Beginning at the bottom of the hierarchy, the first model is that of zero-order CFA (Lienert & von Eye, 1984, 1989), which is also called *Configural Cluster Analysis* (CCA). The base model of CCA is a null model. It assumes that no effects exist in the cross-classification of the variables under study. Types, which, here, are also called *configural clusters*, reflect agglomerations of cases in the data space. This characteristic makes zero-order CFA comparable to the cluster analytic methods that use spatial distance to cluster individual cases. Antitypes, which are also called *configural anticlusters i*n the context of CCA, reflect sectors of the data space that contain surprisingly small numbers of cases or none. These sectors of the data space are typically ignored in cluster analysis. The log-linear base model for CCA is $\log \hat{m} = \lambda$. In other words, the base model for CCA proposes a uniform distribution of cases.

On the next higher level of the hierarchy of global CFA base models, we find *first-order CFA*. This is the original model used by Lienert (1968). First-order CFA includes all

main effects but nothing else. It thus takes differences in marginal probabilities into account. Types and antitypes reflect local associations of first or higher order. To illustrate, consider the four variables *A, B, C*, and *D*. For these four variables, the first-order CFA base model is

$$\log \hat{m} = \lambda + \lambda^A + \lambda^B + \lambda^C + \lambda^D$$

The following interactions are not part of the first-order base model and can, therefore, cause types and antitypes to exist (in bracket notation):

- Two-way interactions [*A, B*], [*A, C*], [*A, D*], [*B, C*], [*B, D*], and [*C, D*];
- Three-way interactions [*A, B, C*], [*A, B, D*], [*A, C, D*], and [*B, C, D*]
- Four-way interaction [*A, B, C, D*].

The first data example in this chapter (Table 20.1) used first-order CFA as the base model. Most applications of CFA utilize first-order CFA as the base model, which takes only main effects into account. In many instances, first-order CFA base models are used to determine whether there are any effects. In most of the examples presented in this chapter, the first-order CFA base model is also used for this purpose.

On the next-higher level of the hierarchy of CFA base models, we find second-order CFA. This model assumes that two-way interactions exist. Types and antitypes can, therefore, emerge only if third- or higher order interactions exist. To illustrate second-order CFA, consider, again, the four variables *A, B, C*, and *D*. The second-order CFA base model for these variables is

$$\begin{aligned}\log \hat{m} = \lambda + \lambda^A + \lambda^B + \lambda^C + \lambda^D \\ +\lambda^{AB} + \lambda^{AC} + \lambda^{AD} + \lambda^{BC} + \lambda^{BD} + \lambda^{CD}\end{aligned}$$

The second line of this equation contains the two-way interactions that, in first-order CFA, may cause types and antitypes. The possible interactions that are not part of the second-order CFA base model can, therefore, cause types and antitypes to exist (in bracket notation) are

- Three-way interactions [*A, B, C*], [*A, B, D*], [*A, C, D*], and [*B, C, D*]; and the
- Four-way interaction [*A, B, C, D*].

Although rarely applied, second-order CFA can be of interest for a number of reasons. The most important of these is that it uses first-order relations as a basis for analysis as do factor analysis, multidimensional scaling, correspondence analysis, and some structural equation models. However, in contrast to these models, second-order CFA asks whether there exists systematic variability above and beyond what is explained by first-order interactions (i.e., all pairwise interactions). Factor analysis as well as the other methods on the list attempt to model the structure evident only in first-order relations. For an application of second-order CFA in psychopathological research, see von Eye and Lienert (1984).

Base models higher than second-order have not been discussed in detail in the CFA literature. However, higher order base models can easily be specified in the context of hierarchical log-linear models. Using the four variables *A, B, C*, and *D*, the third-order CFA base model is

$$\begin{aligned}\log \hat{m} = \lambda + \lambda^A + \lambda^B + \lambda^C + \lambda^D \\ +\lambda^{AB} + \lambda^{AC} + \lambda^{AD} + \lambda^{BC} + \lambda^{BD} + \lambda^{CD} \\ +\lambda^{ABC} + \lambda^{ABD} + \lambda^{ACD} + \lambda^{BCD}\end{aligned}$$

Using this model, only the four-way interaction [*A, B, C, D*] can be the cause of types and antitypes. In some of the paradoxes discussed in the statistics literature, for example, the Meehl's paradox (Meehl, 1950; von Eye, 2002), the highest order interaction carries the entire association in a cross-classification. To explore whether this is the case for a concrete data set, a CFA with a '*d*-1'st-order base model can be performed, where *d* denotes the number of variables. If types and antitypes still emerge from this base model, one can proceed and test hypotheses that are compatible with Meehl's paradox. This applies accordingly to Simpson's paradox (but not necessarily to other paradoxes; Krauth, 2005), and some of the recently discussed configural moderator models (von Eye & Mun, 2013).

In all global CFA base models, types and antitypes contradict the assumptions about associations that are made when estimating the expected cell frequencies. The resulting configurations stand out in the sense that they are observed at different rates from those that conform to the base model. An example of such a configuration is a pattern of symptoms that describes a nosological unit. These symptoms are not necessarily predictors of each other, and, conceptually, they cannot necessarily be grouped as dependent or independent variables, nor as mediators or moderators. For this reason, increasing numbers of CFA applications can be found in developmental psychopathology (e.g., Bogat, Levendosky, & von Eye, 2005; Martinez-Torteya, Bogat, von Eye, & Levendosky, 2009; von Eye & Bogat, 2006).

In contrast, *regional CFA base models* do distinguish groups of variables. Predictors are distinguished from

criterion variables, dependent variables from independent variables, covariates are treated differently than the variables that span the cross-classification, and mediator as well as moderator models can be tested. It is most important to note that most longitudinal CFA base models are regional models. A selection of these base models is explained later in this chapter. Therefore, we review only two examples in the present context.

The first example is that of Prediction CFA (PCFA). As discussed in previous sections of this chapter, the PCFA base model includes two sets of effects or variables. The first set includes all possible effects on the predictor side, rendering the model saturated in the predictors. This characteristic has the effect that types and antitypes cannot emerge because there are associations within the predictors. The PCFA base model is also saturated in the criterion variables. Therefore, types and antitypes cannot emerge because the criterion variables are related to each other. The third characteristic of the PCFA base model is that it proposes independence between predictor and criterion variables. If this base model is rejected, types and antitypes, by necessity, reflect relationships that link predictor variables to criterion variables.

The second example is that of Multigroup CFA. This CFA model also contains two groups of variables. The first group contains the variables that are used to compare the groups, that is, the *discriminator variables*. The Multigroup CFA base model is saturated in these variables. Therefore, types and antitypes (also called *discrimination types*) cannot emerge because the discriminator variables are related to each other. The second group of variables describes the groups. In most CFA applications, so far, this group contains only one variable, the grouping variable. For this variable, only the main effect is considered. This is done to take differences in group size into account. Interactions among the discriminator variables and the grouping variables are not part of the base model. Therefore, discrimination types and antitypes reflect such interactions, by necessity.

Consider, for example, the four variables *G, A, B*, and *C. G* is the grouping variable and *A, B*, and *C* are used to discriminate the groups. The multigroup CFA base model for these variables is

$$\log \hat{m} = \lambda + \lambda^G + \lambda^A + \lambda^B + \lambda^C + \lambda^{AB} + \lambda^{AC} + \lambda^{BC} + \lambda^{ABC}$$

Considering that this model is hierarchical, it could also have been expressed as [*G*], [*A, B, C*]. More examples of regional CFA models follow when we discuss models in more detail that are of particular interest in developmental research. Before we embark on the discussion and illustration of these models, we present, in the next section, a selection of significance tests that can be used to make type/antitype decisions.

Significance Testing in CFA

A large number of significance tests for CFA has been proposed. These tests differ in power, characteristics of the design under which they can be applied, their performance in samples of varying size, and whether they are exact or approximative. In this section, we present a selection of more popular tests. In addition, we present a test that can be applied when hypotheses are tested that involve groups of cells instead of individual cells.

The CFA Null Hypothesis. Each of the tests to be discussed and applied in this chapter can be used to test the CFA null hypothesis

$$H_0 : E[m_r] = m_r^*$$

where $E[m_r]$ is the expectancy for cell r, where r goes over all cells of a cross-classification, and m_r^* is the expected frequency for cell r under a base model. For the test of this null hypothesis, there are three possible outcomes: type, neither type nor antitype, and antitype. First, the cell constitutes a CFA type. In this case, the null hypothesis is rejected because

$$B_{N,\pi_r}(m_r - 1) \geq 1 - \alpha$$

where N is the sample size, π_r is the probability of cell r, and m_r is the observed frequency of cell r. In other words, in the first case, the null hypothesis is rejected because cell r contains more cases than expected. In the second case, the null hypothesis prevails because cell r contains as many cases as expected. In the third case, the null hypothesis is rejected because

$$B_{N,\pi_r}(m_r) \leq \alpha$$

In other words, in the third case, the null hypothesis is rejected because cell r contains fewer cases than expected under a base model. It constitutes an antitype.

CFA Tests for Individual Configurations. The first test to be presented here is the nonparametric binomial test. It can be employed under any sampling scheme (multinomial, product-multinomial sampling). Given m_r, the exact tail probability of m_r and more extreme frequencies is

$$B_r(m_r) = \sum_{j=a}^{l} \binom{N}{J} p^j q^{N-j},$$

where $q = 1 - p$, $a = 0$ if $m_r < m_r^*$ and $a = m_r$ if $m_r \geq m_r^*$, $l = m_r$ if $a = 0$, and $l = N$ if $a = m_r$. Usually, the value p is estimated from the sample. It is *a priori* known only in rare cases. As can be seen from the equation, the probability of each frequency is calculated and added to the total. There is no need to assume that a theoretical distribution is reasonably approximated. The binomial test is exact. This test is recommended when a sample is so small that the approximation properties of approximative tests are doubtful.

More powerful than the binomial test is the z test,

$$z = \frac{m_r - Np}{\sqrt{Npq}}$$

The z test can also be applied under any sampling scheme.

Among the most powerful tests is Lehmacher's (1981) normal approximation of the hypergeometric test,

$$z_{L,r} = \frac{m_r - m_r^*}{\sigma_r}$$

where σ_r is the square root of the exact variance, which can be calculated as

$$\sigma_r^2 = Np_r(1 - p_r - (N-1)(p_r - \widetilde{p}_r)),$$

and when $d = 3$ variables are studied, the values of p_r and $\widetilde{p}_r$ are estimated as

$$p_r = \frac{m_{i..}m_{.j.}m_{..k}}{N^2}$$

and

$$\widetilde{p}_r = \frac{(m_{i..} - 1)(m_{.j.} - 1)(m_{..k} - 1)}{(N-1)^d}$$

where d is the number of variables that span the cross-classification. For the present description of the values of p_r and $\widetilde{p}_r$, we used the case of $d = 3$. Lehmacher's test is powerful and tends to suggest types or antitypes even when they may not truly exist. To prevent this test from suggesting nonconservative type–antitype decisions, Küchenhoff (1986) recommended using a continuity correction for Lehmachers test, which involves reducing the difference in the numerator by 0.5. Lehmacher's test can be used only under a product-multinomial sampling scheme. That is, the marginal probabilities must be fixed for the test to be valid. Another constraint is that Lehmacher's test can be applied only under the base model of variable independence.

A test that is similarly powerful was recently proposed by von Eye and Mair (2008a): the *standardized Pearson residual*, z'_r. The test statistic is

$$z'_r = \frac{m_r - m_r^*}{\sqrt{m*_r(1 - h_r)}}$$

where h_r is the r-th diagonal element of the well-known *hat matrix, H*. This matrix is

$$H = W^{0.5}X(X'WX)^{-1}X'W^{0.5}$$

In this equation, W is a diagonal weight matrix that contains the expected frequencies, $m*_r$, in its diagonal. As the binomial test and the z test, the standardized Pearson residual can be used under any sampling scheme or base model.

Testing Groups of Configurations

The sum of the previous z statistics has an expectancy of $E(\Sigma z_r) = 0$ and a variance of t, where t is the number of terms (cells) in the summation. Now, if a group of t configurations is subjected to a CFA null hypothesis that includes the whole group, Stouffer's Z (Stouffer, Suchman, DeVinney, Star, & Williams, 1949) can be used, which is

$$Z = \left(\sum_{i=1}^{t} z_i\right) \Big/ \sqrt{t}.$$

Data Example for Stouffer's Z. The following data example illustrates the use of Stouffer's Z in CFA. For the example, we reanalyze the data in Table 94 in von Eye (2002). In a study on experimental psychoses, Lienert (1964) administered LSD50 to 65 students. When the effects of the drug set in, three symptoms were observed: Narrowed Consciousness (C), Thought Disturbance (T), and Affective Disturbance (A). Each symptom was scaled as either present (1) or absent (2). Table 20.2 displays the $2 \times 2 \times 2$ C × T × A cross-tabulation, along with the expected frequencies that were estimated under the base model of variable independence. We used the z test along with the Bonferroni-protected $\alpha = 0.00625$ (methods of α protection are discussed in the following section).

The overall goodness-of-fit likelihood ratio X^2 for the base model of variable independence for the data in Table 20.2 is 45.07. For $df = 4$, this value suggests that the model be rejected ($p < 0.01$). Still, none of the eight configurations constitutes a type or an antitype. When looking at the patterns in Table 20.2, we notice that the

TABLE 20.2 First-Order CFA of the Cross-Classification of Narrowed Consciousness (C), Thought Disturbance (T), and Affective Disturbance (A)

	Configuration			
CTA	m	$\hat{m}$	z	p
111	20.00	12.506	2.1193	.017034
112	1.00	6.848	−2.2348	.012715
121	4.00	11.402	−2.1921	.014185
122	12.00	6.244	2.3035	.010626
211	3.00	9.464	−2.1011	.017815
212	10.00	5.182	2.1162	.017164
221	15.00	8.629	2.1690	.015041
222	.00	4.725	−2.1738	.014862

bi-symptomatic configurations are all observed at low rates. These are the configurations 1 1 2, 1 2 1, and 2 1 1. We, therefore, ask, whether these three configuration constitute a *composite antitype* (also called *joint antitype*). To test the corresponding null hypothesis that they do not constitute a composite antitype, we take the z scores from Table 20.2, insert them into the formula for Stouffer's Z, and obtain

$$Z = \frac{-2.24 - 2.19 - 2.10}{\sqrt{3}} = -3.7689$$

This value suggests that the null hypothesis can be rejected. We conclude that the three bisymptomatic configurations, 1 1 2, 1 2 1, and 2 1 1, constitute a joint antitype. It is particularly unlikely that just two of the symptoms are observed.

Data Example for Comparison of CFA Tests. For the following example, we reanalyze data that were published by Magnusson in 1996. The data describe a sample of 70 male adolescents. To study the role played by physiological and psychological factors in criminal behavior, the three variables Committed a Crime (C), Hyperactivity (H), and Adrenaline level (A) were observed. Crime was coded: 1 = no crime committed, 2 = crime committed before the age of 18, and 3 = persistent crime. Hyperactivity and Adrenaline levels were both coded as 1 = low and 2 = high. Cutoffs were the means. Table 20.3 displays the 3 × 2 × 2 C × H × A cross-classification, along with the expected cell frequencies and the tail probabilities from the binomial test, the z-test, and the standardized Pearson residual. α was protected using the Holland–Copenhaver procedure (which is explained in the next section). The expected cell frequencies were estimated under the base model of variable independence.

The overall goodness-of-fit likelihood ratio X^2 for the base model of variable independence for the data in Table 20.3 is 47.14. For $df = 7$, this value suggests that the model is rejected ($p < 0.01$), and we expect types and antitypes to emerge. We now interpret the results in Table 20.3 from two perspectives. First, we discuss the substantive meaning of the type and antitype in this table. Then, we compare the three CFA significance tests.

As far as the analysis of crimes committed in adolescence is concerned, we note that of those 11 male adolescents who were grouped as persistent in their criminal behavior ($C = 3$), nine showed the pattern of high hyperactivity and low adrenaline. Pattern 3 2 1 constitutes a type. The antitype shows the same pattern (. 2 1), suggesting that with high levels of hyperactivity and low levels of adrenaline, it is particularly unlikely to be in the group of nonoffenders. In fact, not a single nonoffender showed this pattern, 1 2 1.

We also ask whether including all pairwise interactions would allow us to fully explain the frequency distribution in Table 20.3. The corresponding model is hierarchical. It is [*C, H*], [*C, A*], [*H, A*]. The results suggest that this model

TABLE 20.3 First-Order CFA of the Cross-Classification of Crime (C), Hyperactivity (H), and Adrenaline Level (A)

	Configuration							
CHA	m	$\hat{m}$	B	z	$p(z)$	z'	p(z')	
111	25.00	20.1367	.12564275	1.0838	.139235	2.1456	0.016	
112	18.00	16.0061	.32783713	0.4984	.309110	0.9069	0.182	
121	.00	5.4918	.00328220	−2.3435	.009553	−3.2172	0.001	Antitype
122	3.00	4.3653	.35776569	−0.6535	.256728	−0.8450	0.199	
211	2.00	5.6908	.06902106	−1.5472	.060912	−2.2438	0.012	
212	8.00	4.5235	.08160703	1.6346	.051067	2.1978	0.014	
221	2.00	1.5520	.46147235	0.3596	.359583	0.4067	0.342	
222	1.00	1.2337	.64975053	−0.2104	.416685	−0.2326	0.408	
311	1.00	4.8153	.04204248	−1.7387	.041046	−2.4868	0.006	
312	1.00	3.8276	.09856945	−1.4453	.074191	−1.9171	0.028	
321	9.00	1.3133	.00000666	6.7076	.000000	7.5137	0.000	Type
322	.00	1.0439	.34932965	−1.0217	.153461	−1.1204	0.131	

Note. B = Binomial test; z = z-test; z' = standardized Pearson residual test.

must be rejected also ($LR - X^2 = 11.05$; $df = 2$; $p = 0.004$). Although this model is significantly better than the original base model of variable independence ($\Delta X^2 = 36.09$; $\Delta df = 5$; $p < 0.01$), it cannot stand by itself.

As far as the comparison of the three CFA tests is concerned, we first see that, in this example, the z test is generally more powerful than the binomial test. As we said previously, we trust the binomial test more than the z test when a sample is small. Therefore, we interpreted the results from the binomial test. The standardized Pearson residual is much more powerful than the other two tests in this example. All configurations had smaller p values than under the other two tests.

Protection of α

CFA involves multiple testing. There are mainly two reasons why multiple tests in CFA (and in other methods of analysis, for example, ANOVA) can become problematic. The first is known as *capitalizing on chance*, the second lies in the *dependency of tests*. Only the first of a series of tests on the same data keeps the a priori specified α level. When more than one test is performed, the risk of making an α error increases, even if the tests are independent. For example, if, in the context of testing for CFA types and antitypes, each of the 27 cells of a $3 \times 3 \times 3$ table is examined under $\alpha = 0.05$, the probability of incorrectly rejecting two null hypotheses is 0.24. Performing multiple tests on one sample therefore includes a high risk of capitalizing on chance if no precautions are taken to protect α.

This situation gets even worse when tests are dependent. von Weber, Lautsch, and von Eye (2003) showed that, when the expected cell frequencies in a 2×2 table are estimated under a log-linear main effect model, that is, the model of variable independence, the first CFA test indeed keeps the a priori specified α level. However, the outcomes of the second, third, and fourth CFA tests in the 2×2 table are completely dependent upon the outcome of the first test. Similarly, Krauth (2003) showed that the number of possible patterns of types and antitypes increases as the size of a table increases, but that the tests in a CFA never become completely independent.

A number of procedures exists for the protection of α. The most conservative of these, the *Bonferroni adjustment*, is the most popular procedure. This procedure specifies (1) that the sum of all adjusted significance levels not exceed the nominal α, or $\Sigma_r \alpha_r \leq \alpha$, where r indexes the cells subjected to CFA tests; and (2) that the significance threshold be the same for each test, or $\alpha_r = \alpha^*$, for all r, where α^* is the adjusted threshold. The value of α^* that meets both of these conditions is $\alpha^* = \alpha/R$.

More efficient procedures have been proposed. Based on Holm's (1979) procedure, Holland and Copenhaver (1987) proposed a procedure that does not require that $\Sigma_r \alpha_r \leq \alpha$. In this procedure, the tail probabilities of the cell-wise test statistics are rank ordered. The smallest probability is given Rank 1. The protected value of α^* is then, for the i-th test,

$$\alpha_i^* = 1 - (1 - \alpha)^{\frac{1}{r-i+1}}$$

This procedure is clearly less restrictive than the Bonferroni protection. It is also (slightly) less restrictive than Holm's (1979) procedure (for comparisons of procedures for the protection of α, see Olejnik, Li, Supattathum, & Huberty, 1979; von Eye, 2002; for procedures for special cases, see von Eye, 2002).

The Five Steps of CFA

When performing a CFA, researchers proceed in five steps (von Eye, 2002).

1. *Selection of a base model and estimation of expected frequencies*. In general, a *CFA base model* is a chance model that takes those effects into account that are NOT of interest to the researcher. The base model is used to estimate the expected cell frequencies in the table under study that are then compared with the observed cell frequencies, cell-by-cell. If a deviation between an expected and an observed cell frequency is significant, it reflects, by necessity, effects that are of interest to the researcher.
2. *Selection of a concept of deviation from independence*. Some of the measures of deviation from a base model take into account marginal frequencies, others do not. The corresponding measures are called *marginal-dependent* and *marginal-free* (Goodman, 1991). Examples of marginal-dependent measure include the Φ-coefficient, which is a relative of Pearson's X^2. Φ quantifies the strength of the association between two dichotomous variables. The strength corresponds to the degree of deviation of the observed distribution from the base model of independence between these two variables. Examples of marginal-free measures include the odds ratio, θ. Marginal-dependent and marginal-free measures can give different appraisals of deviation from a base model. Marginal-free measures have been proposed for use in two-group CFA (von Eye, Spiel, & Rovine, 1995).
3. *Selection of a significance test*. Significance tests of the null hypothesis that types or antitypes do not exist differ in that some are exact, whereas others are

approximative. They also differ in statistical power and in the sampling schemes under which they can be employed. Simulation studies have shown that the tests that perform well under many conditions include Pearson's X^2, the z-test, and the exact binomial test. When sampling is product-multinomial, the best performing tests include Lehmacher's (1981) exact and approximative hypergeometric tests.

4. *Performing significance tests under protection of* α. In both exploratory and confirmatory application of CFA, typically, a large number of tests is conducted. This number of tests is generally smaller in confirmatory CFA than in exploratory CFA. In either case, when more than one significance test is performed, the significance level, α, needs to be protected. The routine method for α protection, the Bonferroni procedure, can suggest rather conservative decisions about the existence of types and antitypes. Therefore, Holm's (1979) procedure and even less restrictive methods that have been derived from it have gained in popularity.
5. *Interpretation of types and antitypes*. The interpretation of types and antitypes utilizes the following five sources of information.
   1. The *substantive meaning of a configuration*. This meaning is determined by the definition of the categories that constitute a configuration.
   2. The *base model*. For example, when the base model distinguishes between predictor and criterion variables, the interpretation of types and antitypes differs from the one without this distinction (Mellenbergh, 1996).
   3. The *concept of deviation from expectation* as marginal-dependent versus marginal free.
   4. The *sampling scheme* (e.g., multinomial, product-multinomial).
   5. *External information* that is used to discriminate among individuals in different cells in the cross-classification under study (from each other and from the configurations that constitute types or antitypes). The discrimination is not part of CFA itself. Instead, discrimination is performed in follow-up tests that are undertaken, for example, to establish external validity of CFA types and antitypes (see von Eye, Mair, & Mun, 2010). For discrimination, variables are used that were not part of CFA.

In the remainder of this chapter, we discuss and illustrate CFA models that are of particular interest in developmental research. The models are selected based on the developmental questions they allow the researcher to answer.

CFA MODELS FOR DEVELOPMENTAL RESEARCH

The questions asked in developmental research can concern any individual parameter of the population under study, and any combination of parameters. In the first two data examples of this chapter, we asked questions concerning local associations in cross sectional data. In the following sections, we focus on questions for longitudinal data. In general, CFA allows one to answer questions concerning typical trajectories (longitudinal types) and atypical trajectories (longitudinal antitypes). The trajectories can be defined with respect to means, characteristics of shapes of curves, variability of scores, trends, intervention effects, end points of development, the predictability of trajectories, lags, effects of covariates, mediation, moderation, causal hypotheses, and many other aspects of longitudinal data.

To answer some of the questions, ordinal data are needed, for other questions, data must be at the interval level. For many questions, nominal level data are sufficient. Viewed from a data characteristics perspective, given nominal, ordinal, or interval level data, some questions can be asked, but other questions cannot be asked. For example, for nominal level data, local associations and prediction patterns can be examined. However, questions concerning constancy and change in means cannot be asked using nominal-level data. In the following sections, we point to the properties that data must possess for certain questions to be tractable.

This part of the present chapter is structured as follows. We begin with *CFA of differences* and related methods. For the simplest CFA models in this section, ordinal-level data are sufficient. For more advanced models, interval or ratio scale-level data are required. In the following section, we model *shapes of trajectories*. The first two sections are closely related to each other. The third section covers pre–post designs. We show how this type of design can be analyzed with CFA. The role of control groups is discussed. The following section covers single subject designs. In the contexts of person-oriented (Bergman & Magnusson, 1997; von Eye & Bergman, 2003; von Eye, Bergman, & Hsieh, 2015; Ch. 18) and idiographic research (Molenear, 2004; Molenaar & Campbell, 2009), long series of data are collected for individuals. CFA models for such data are presented. Prediction and autoassociation models for shorter series of data follow in the next sections.

CFA of Differences I

In this section, we ask how CFA can be used to analyze trajectories of repeatedly observed variables. Specifically, we ask whether group differences in such trajectories can be ascertained.

Longitudinal, that is, intraindividual differences can be ascertained at all scale levels. At the nominal level, differences are described by switching from one category to another. At the ordinal level, longitudinal differences imply changing rank or moving up or down on an ordinal scale such as grade in school, or on a well-being scale. At the interval level, longitudinal differences imply that change can be measured in comparable, commensurate units. For example, an increase in intelligence can be by 5 IQ points. These points indicate the same amount of change, regardless where, on the IQ scale, the change is observed. At the ratio scale level, longitudinal differences can also be assessed in units of proportion. An improvement in processing speed from 4 to 2 seconds can be interpreted as an increase by 100%. In the following discussion of CFA methods for analyzing differences, we focus on differences on variables that are measured at least at the ordinal level.

The simplest way of measuring differences involves subtracting the subsequent observation, y_{i+1}, from the previous, y_i, that is, $\Delta y = y_{i+1} - y_i$. When the number of ranks or interval-level scores is small, the number of resulting differences will also be small, and the differences can be crossed with other differences or variables right away. When the number is large, researchers often resort to binning, even dichotomizing. The resulting number of cells in a cross-classification will then be much smaller with binning than without binning.

In the literature, there has been a discussion concerning the reliability of difference scores. Quite often, one finds the argument that the reliability of difference scores is deplorable. Unfortunately, this argument is not always well grounded. We present two counter arguments why. First, when scales are ratio scale level, differences can be highly reliable. Consider a car that travels at a speed of 50 mph. After braking to come to a standstill, the speed difference is 50 mph. This difference is perfectly reliable. This applies accordingly to other ratio scale measures used in developmental research, for example, blood pressure, alpha waves, reaction time, or number of words recalled, and to some interval-level measures and rankings. Second, Zimmerman (2009) showed that, even when test scores are used, the reliability of differences is not as low and anomalous when correlations among the true scores are used instead of correlations among the observed scores. Therefore, while we are fully aware of the potential information loss that can come with binning variables (see MacCallum, Zhang, Preacher, & Rucker, 2002; von Eye & Mair, 2011), we believe that difference scores and binned difference scores can be useful.

Most popular are *first differences*, that is, differences between time-adjacent measures, e.g., the difference of the second observation, y_2, from the first, y_1, $\Delta y = y_2 - y_1$. First differences are used for a number of reasons. The two most important ones are to (1) simply find out about change, and (2) to de-trend series of measures. The second reason is of importance when methods of time series analysis are employed that require the stationarity assumption. The first reason is of importance when individual change scores or change patterns are interpreted.

To calculate *second differences*, one subtracts adjacent first differences,

$$\Delta^2 y = \Delta y_{i+1} - \Delta y_i$$

for $i = 1, \ldots, m - 1$, with m being the number of first differences in the series. The superscript in this equation indicates the order of differences. Second differences are of interest (1) when changes in change, that is, acceleration or deceleration, are of interest, or (2) when the quadratic trend is to be taken out of a series of measures. Higher order differences such as third or fourth differences can be defined in an analogous way. Table 20.4 displays a scheme that shows how differences can be calculated.

In Table 20.4, the Y scores are the (repeated) measures to be modeled. The X scores are the points on the time axis used in repeated measures studies. Difference scores are most easily comparatively interpreted when the points on X are equidistant or, at the least, the distances are the same for every case in a study.

TABLE 20.4 Calculation of First Through Fourth Differences

Raw scores		Differences			
X	Y	ΔY first differences	$\Delta^2 Y$ second differences	$\Delta^3 Y$ third differences	$\Delta^4 Y$ fourth differences
x_0	y_0				
		Δy_0			
x_1	y_1		$\Delta^2 y_0$		
		Δy_1		$\Delta^3 y_0$	
x_2	y_2		$\Delta^2 y_1$		$\Delta^4 y_0$
		Δy_2		$\Delta^3 y_1$	
x_3	y_3		$\Delta^2 y_2$		
		Δy_3			
x_4	y_4				

Differences possess important characteristics that make them interesting for longitudinal research.

1. Differences can be used to estimate the degree of a polynomial that describes a series of measures. Specifically,
   a. the *n*-th differences of a polynomial of the *n*th degree are constant; this implies that, as soon as a series of differences is constant, the polynomial of corresponding degree will describe the series perfectly; and
   b. the differences of an order higher than *n* disappear; in other words, calculating differences of a degree higher than *n* is pointless.
2. Difference variables can be used to span cross-classifications. As we said before, when the number of differences is small, they can be directly crossed with other differences, with time-varying, and with time-constant (measured only once) variables.
   a. The method of differences can be used to identify scores that are suspected to be faulty. Scores that are faulty result in the existence of higher order differences when most lower order differences have long converged to very small values, or even disappeared. In addition, the magnitude of the error can be concluded from the magnitude of the differences.

To illustrate the method of differences, we first present an example with artificial data (von Eye, 2002), and then a real data example. For the artificial data example, we use the five consecutive *x*-values of 1 through 5 and select the five *y*-values {1, 3, 4, 3, 1}. For these scores, we calculate the first, second, and third differences. The three resulting curves of the differences are depicted in Figure 20.2, along with the curve of the raw scores.

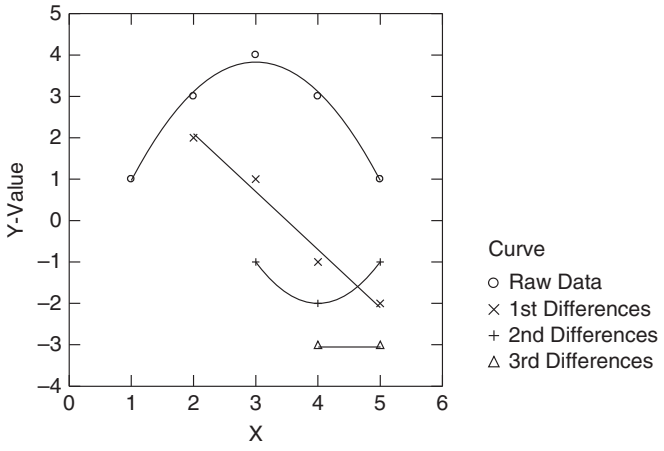


Figure 20.2 Method of differences.

Figure 20.2 shows that the raw data can be described as an inversely U-shaped function. Its slope changes from positive to negative (first differences). The change in slope is negative throughout and more pronounced around the middle of the curve (second differences). The third differences are constant, suggesting that the raw data can be perfectly described by using a third-order polynomial.

Data Example

For the real-data example, we use data that Finkelstein, von Eye, and Preece (1994) collected on the development of aggressive behavior. The authors administered a questionnaire concerning aggressive behavior to 67 adolescent girls and 47 boys at three points in time. The time intervals were 2 years apart. The questionnaire addressed the four dimensions of aggression: Aggressive Impulse, Aggression-Inhibitory Response, Verbal Aggression against Adults, and Physical Aggression against Peers. In addition, physical pubertal development was assessed using Tanner scores. In the following analyses, we use the data on physical aggression against peers (PAAP). Three scores existed per person, PAAP83, PAAP85, and PAAP87, where the numbers indicate the years in which the data were collected. When the data were collected, the respondents were, on average, 11, 13, and 15 years of age, respectively. In the following example, we ask whether the developmental change in self-rated physical aggression against peers is different for boys and girls. We perform the following three steps.

1. Calculate the differences PADIF1 = PAAP85 – PAAP83 and PADIF2 = PAAP87 – PAAP83;
2. Dichotomize the differences; here, we dichotomize the differences at their grand mean; the resulting split will not lead to groups that are exactly the same in size (the distributions of the difference scores have slightly elevated skewness); however, information about the mean is conserved;
3. Perform 2-group CFA with Gender as the grouping variable.

Descriptive statistics for the difference scores PADIF1 and PADIF2 appear in Table 20.5.

After dichotomization, both variables were coded as 1 = below the mean and 2 = above the mean. Gender was coded as 1 = female and 2 = male. Crossed, the three variables span the 2 × 2 × 2 Gender (G) × PADIF1 (P1) × PADIF2 (P2) cross-classification. We analyze this table using two CFA base models. The first is the two-group base model, the second is the standard base model of first-order CFA.

TABLE 20.5 Descriptive Statistics for the Variables PADIF1 = PAAP85 − PAAP83 and PADIF2 = PAAP87 − PAAP85

	PAAPDIF1	PAAPDIF2
N of Cases	114	114
Minimum	1.000	1.000
Maximum	2.000	2.000
Arithmetic mean	1.561	1.482
Standard deviation	0.498	0.502

Similar to the PCFA base model, the two-group model proposes that (1) the model is saturated in the variables used to discriminate between the two groups, and (2) the grouping variable is independent of the discriminator variables. In the present example, the two-group base model is

$$\log \hat{m} = \lambda + \lambda^{G} + \lambda^{P1} + \lambda^{P2} + \lambda^{P1,P2}$$

This model can be rejected only if the grouping variable, Gender, is associated with the discriminator variables, *P*1 and *P*2. Specifically, this model can be rejected if one or more of the following interactions exist: [*G, P*1], [*G, P*2], and [*G, P*1, *P*2].

This base model has an interesting characteristic. It makes the association between the variables *P*1 and *P*2 part of the model. Considering that these two variables describe temporal elements of the development of physical aggression against peers, this association reflects the serial dependency of *P*1 and *P*2. Types and antitypes cannot, therefore, emerge just because *P*1 and *P*2 show an autoassociation (comparable to autocorrelation).

The second base model is that of first-order CFA,

$$\log \hat{m} = \lambda + \lambda^{G} + \lambda^{P1} + \lambda^{P2}$$

This model contains only the main effects of the variables that span the cross-classification of *G*, *P*1, and *P*2. Therefore, the autoassociation of *P*1 and *P*2 can be among the reasons why this model is rejected (if it is rejected). For the type–antitype decisions under both base models, we use the normal approximation of the binomial test and the Holland–Copenhaver procedure of α protection.

Table 20.6 displays the 2 × 2 × 2 Gender (G) × PADIF1 (P1) × PADIF2 (P2) Cross-Classification along with the results of two-group CFA. The overall goodness-of-fit likelihood ratio X^2 for the base model of two-group CFA for the data in the table is 12.58. For $df = 3$, this value suggests that the model is rejected ($p = 0.006$), and we expect that discrimination types and antitypes may exist.

The results in Table 20.6 suggest that the two gender groups differ in two profiles. Significantly more male than female respondents display the trajectory of individuals who consistently change to self-perceived ever lower levels of physical aggression against peers. The second discrimination type suggests that significantly more female than male respondents change to self-perceived, ever higher levels of physical aggression against peers. The remaining two comparisons—both concern up-and-down patterns of change—were observed about as often as expected under the base model.

TABLE 20.6 Two-Group CFA of the Gender (G) × PADIF1 (P1) × PADIF2 (P2) Cross-Classification, with Gender as the Grouping Variable

	Configuration			
P1 P2 G	*m*	*z*	*p*	Type?
111	8.00			
112	16.00	−2.849	.002190	Discrimination type
121	17.00			
122	9.00	.780	.217801	
211	19.00			
212	16.00	−.648	.258591	
221	23.00			
222	6.00	2.602	.004632	Discrimination type

The two types also come with significant odds ratios. Specifically, males are 1.337 times more likely than females to show trajectory pattern 1 1 ($z = -2.474$; $p = 0.007$), and females are 1.273 times more likely than females to show trajectory pattern 2 2 ($z = 2.520$; $p = 0.006$).

From a data analytic perspective, it is important to point to the fact that two-group CFA involves only half the number of tests as the corresponding standard CFA. Therefore, the protected α-level is less restrictive in two-group CFA than in standard one-group CFA. Consider the example in Table 20.6. In this table, four tests were performed instead of eight tests that are needed in standard, first-order CFA. One implication of a less restrictive, protected α is that types and antitypes are more likely to be detected. Table 20.7 displays the results from first-order CFA that was performed using the same significance test and α protection as for the two-group CFA in Table 20.6.

The overall goodness-of-fit likelihood ratio X^2 for the base model of first-order CFA for the data in Table 20.7 is 13.09. For $df = 4$, this value suggests that the model is rejected ($p = 0.011$). This result leads to the expectation that types and antitypes may exist. However, as is evident from Table 20.7, none of the configurations deviates significantly from expectation, when the stricter protected α is used.

In an add-on step of the analysis of the data in Tables 20.6 and 20.7, we ask which log-linear model could

TABLE 20.7 First-Order CFA of the Gender (G) × PADIF1 (P1) × PADIF2 (P2) Cross-Classification

	Configuration			
P1 P2 G	m	$\hat{m}$	z	$p(z)$
111	8.00	15.209	−1.9856	.023538
112	16.00	10.669	1.7144	.043226
121	17.00	14.11	.8011	.211539
122	9.00	9.945	−.3138	.376848
211	19.00	19.467	−.1162	.453743
212	16.00	13.656	.6761	.249482
221	23.00	18.147	1.2424	.107053
222	6.00	12.730	−2.0013	.022679

be used to adequately describe the frequency distribution. We find that the model $\log \hat{m} = \lambda + \lambda^G + \lambda^{P1} + \lambda^{P2} + \lambda^{G,P2}$ explains the data very well ($\mathrm{LR} - X^2 = 4.398$; $df = 3$; $p = 0.222$). This model suggests that there is an association between Gender and change in Physical Aggression Against Peers between the ages of 13 and 15. This result is plausible and easy to interpret. However, the specifics of the results, in particular, the findings that (1) male adolescents are more likely than female adolescents to exhibit a repeated reduction in self-perceived physical aggression and that (2) repeated increases in aggression are comparatively more likely in female adolescents can most easily be obtained using CFA.

In sum, two-group CFA of trajectories shows, in the present example, a typical pattern of CFA results. A selection of configurations allows one to discriminate between the comparison groups. Other configurations are observed as often as expected, in both comparison groups. In addition, two-group CFA of differences has the interesting characteristic that the analyzed cross-classification is much smaller than the cross-classification of the original variables. Consider a study in which an ordinal variable with five ranks is observed three times. Crossing these observations results in a table with $5^3 = 125$ cells. Creating first differences reduces the number of variables from 3 to 2. Binning the differences into three categories such as change toward higher ranks, lower ranks, and no change, and crossing the two resulting change variables results in a table with $3 \times 3 = 9$ cells, a savings of 92.8%. Naturally, this savings goes hand in hand with loss of information. However, it provides the researcher with the opportunity to capture interesting parts of the original information and to make them the focus of data analysis, or to include additional information such as covariates. In the next section, we discuss a priori probabilities in the analysis of differences (von Eye, 2002; von Eye, Mair, & Mun, 2010; von Eye & Mun, 2007, 2012).

CFA of Differences II: Structural Zeros

So far in this chapter and, in most applications of CFA, researchers proceed from the assumption that the variables under study can be completely crossed. That is, each category of every variable can be observed under each category of every other variable. When difference variables as the ones discussed in the last section are crossed with covariates or difference variables that are based on other variables, this assumption is practically always met. However, there is one interesting exception. This exception concerns crossing difference variables of different order of the same original variable with each other.

Consider, for example, a variable that is observed three times. For the resulting three measures, two measures of first differences can be calculated (Δ_1^1 = Time 2 – Time 1, and Δ_2^1 = Time 3 – Time 2) and one measure of second differences ($\Delta_2^2 = \Delta_2^1 - \Delta_1^1$). Now, let the difference measures be dichotomized at zero (this is also called *sign-transformed*). The resulting scores indicate whether the differences indicate an upswing or a downswing of differences. Let the original variable also be dichotomized. Crossed, these three indicators span a $2 \times 2 \times 2$ contingency table, with cell indices 0 0 0, 0 0 1, 0 1 0, 0 1 1, 1 0 0, 1 0 1, 1 1 0, and 1 1 1.

Of these eight cells, two are logically impossible and therefore, cannot contain any case at all. These are the cells with patterns 0 1 0 and 1 0 1, where the order of indices is original variable—first differences—second differences. The first of these patterns suggests a decrease that is followed by an increase and, overall, a negative quadratic trend. This pattern would reflect a ∪-shaped curve that is, simultaneously, ∩-shaped. The second pattern indicates an initial increase that is followed by a decrease in tandem with an overall positive quadratic trend. This pattern would reflect a ∩-shaped curve that is, simultaneously, ∪-shaped. Neither is logically possible. Because these shapes are contradictory, configurations 0 1 0 and 1 0 1 must be declared *structural zeros*.

Structural zeros are different from observed zeros. When, in a sample, a pattern was not observed but, theoretically, maybe in a larger sample, there could be cases that exhibit this pattern, the zero is called a *sampling zero*. If, however, it is impossible to observe a particular pattern, mostly for logical and design reasons, the zero is structural. These two types of zeros are treated differently when it comes to estimating expected cell frequencies. Sampling zeros are treated just as the other frequencies. An expected value is estimated and the zero is compared with the estimate (exceptions exist when there are so many

zeros that parameter estimation is impeded). In contrast, cells that contain structural zeros must be blanked out. For these cells, no expected frequency is estimated, and there is no comparison of observed with expected frequencies.

Von Eye and Mun (2007) presented the following definition of impossible patterns in the analysis of categorized differences: *Combinations of sign patterns are impossible* if lower order and higher order signs suggest different orientations of a series of measures. This applies to patterns of difference signs of any order.

The authors proposed a procedure with which all candidate patterns for contradictory descriptions of orientation can be identified. Candidates for a search are all patterns that indicate a change in orientation from *up* to *down* and vice versa. Patterns that show no change do not need to be considered. Now, let Δ_j^1 and Δ_{j+1}^1 be the first differences between the adjacent measure pairs x_{j+1} and x_j, and x_{j+2} and x_{j+1}, respectively, for $j = 1, \ldots, I-1$, and Δ_k^2 the corresponding second difference, that is, $\Delta_{k+1}^2 - \Delta_k^2$. Let sgn Δ_j^1, sgn Δ_{j+1}^1, and sgn Δ_k^2 be the signs of these differences. The following two steps are performed to identify the structural zeros.

Step 1: Compare each pair of sgn Δ_j^1 and sgn Δ_{j+1}^1 with the corresponding sgn Δ_k^2. If both sgn $\Delta_j^1 \neq$ sgn Δ_{j+1}^1 and sgn $\Delta_{j+1}^1 \neq$ sgn Δ_k^2, then the pattern is impossible. For example, if the sign pattern + − at a given difference level is combined with sign + at the next higher level, the sign pattern is impossible. The first differences would indicate a negative orientation, whereas the second difference would indicate a positive orientation. Accordingly, the sign pattern − + at a given level cannot be combined with the sign − at the next higher level. If a sign pair is classified as impossible, proceed to the next sign pattern. If a sign pattern is impossible, all other patterns that could be combined with the impossible pattern are impossible also.

Step 2: Proceed to the next pair of signs, and start over, at Step 1. Continue until all patterns of signs are examined.

This procedure can be applied to sign patterns of all levels. If a researcher decides to skip levels, the procedure can be adjusted. For example, pattern + − at level k cannot go with the sign – at level $k + 2$.

To give an example of a possible pattern, let k indicate first differences and let $k + 2$ indicate third differences. In this situation, consider the sign pattern + − + for first differences and + for third differences. The first signs indicate an initial increase that is followed by a decrease, and then an increase. The third difference indicates an increase in acceleration. To give an example of an impossible pattern, consider the series 9, 15, 10, 6. For this sequence, the first differences sign pattern is + − −, the second differences sign pattern is − +, and the corresponding third difference has the sign +, thus indicating a ∩-shaped trend. For this series, a negative sign for the third difference is impossible. A complete example with an artificial data set is given by von Eye and Mun (2007). A real-data example follows.

Data Example

In the data example in the following paragraphs, we use the data on adolescent development of aggression again that we used previously for the example in Table 20.6. For the earlier example, we had analyzed the first differences of Physical Aggression Against Peers. Here, we analyze first and second differences. Specifically, we create a 2 × 2 × 2 table by crossing PADIF1 = PAAP85 − PAAP83 (abbreviated by P1), PADIF2 = PAAP87 − PAAP85 (abbreviated by P2), and P2DIF = PADIF2 − PADIF1 (abbreviated by ΔP). The resulting cross-classification contains two structural zeros. The first is in cell 1 2 2. It only makes sense that nobody shows this pattern. The pattern would describe a ∪-shaped curve that is, simultaneously, ∩-shaped. The second structural zero is found in cell 2 1 2. This pattern describes a ∩-shaped curve that is, simultaneously, ∪-shaped. Neither is logically possible.

The 2 × 2 × 2 P1 × P2 × ΔP cross-classification is analyzed using two CFA base models. The first is the log-linear main effect model of variable independence,

$$\log \hat{m} = \lambda + \lambda^{P1} + \lambda^{P2} + \lambda^{\Delta P}$$

In this model, no provision is taken for the structural zeros. Therefore, the expected frequencies for Cells 1 2 2 and 2 1 2 will be greater than zero. The second base model declares these two cells structural zeros. The model is

$$\log \hat{m} = \lambda + \lambda^{P1} + \lambda^{P2} + \lambda^{\Delta P} + \lambda^{s1} + \lambda^{s2},$$

where the $s1$ and the $s2$ in the superscript represent the first and the second vectors that indicate structural zeros. Under this base model, the expected frequencies for Cells 1 2 2 and 2 1 2 will be zero. Table 20.8 displays the results of the first-order CFA under the first base model. Table 20.9 displays the results of first-order CFA under the second base model. For both CFA runs, we use the z test and protect α using the Holland–Copenhaver procedure.

The overall goodness-of-fit likelihood ratio X^2 for the base model of first-order CFA for the data in Table 20.8 is 85.72. For $df = 4$, this value suggests that the model is rejected ($p < 0.01$), and we anticipate that types and antitypes emerge. The table shows two types and two

TABLE 20.8 First-Order CFA of the P1 × P2 × ΔP Cross-Classification; Structural Zeros Not Taken into Account

	Configuration				
P1 P2 ΔP	m	$\hat{m}$	z	$p(z)$	Type/antitype?
111	10.00y	14.074	−1.0859	.138771	
112	14.00	11.804	.6393	.261317	
121	.00	13.119	−3.6221	.000146	Antitype
122	26.00	11.003	4.5210	.000003	Type
211	35.00	18.014	4.0020	.000031	Type
212	.00	15.109	−3.8870	.000051	Antitype
221	17.00	16.793	.0505	.479843	
222	12.00	14.084	−.5554	.289314	

TABLE 20.9 First-Order CFA of the P1 × P2 × ΔP Cross-Classification; Structural Zeros Taken into Account

	Configuration				
P1 P2 ΔP	m	$\hat{m}$	z	$p(z)$	Type/antitype?
111	10.00	17.821	−1.8526	.031969	
112	14.00	17.354	−.8051	.210380	
121	.00	.000	–	–	
122	26.00	14.825	2.9022	.001853	Type
211	35.00	23.825	2.2894	.011029	
212	.00	.000	–	–	
221	17.00	20.354	−.7434	.228617	
222	12.00	19.821	−1.7567	.039488	

antitypes. The first type is constituted by cell 1 2 2. This pattern describes a curve that shows an initial decrease that is followed by an increase, with positive acceleration (positive quadratic trend). 11 cases were expected to exhibit this pattern, but 26 did exhibit it, a significant discrepancy. The second type is constituted by cell 2 1 1. It shows just the opposite pattern. Instead of the expected 18, 35 adolescents showed an initial increase in physical aggression that was followed by a decrease, with negative acceleration.

For the present purposes, the two antitypes are most interesting. The first, constituted by cell 1 2 1, indicates that not a single adolescent showed a decrease that is followed by an increase which, at the same time, indicates a negative quadratic trend. The second, constituted by cell 2 1 2 indicates that no one showed an initial increase, followed by a decrease together with an overall positive quadratic trend. It only makes sense that nobody showed these patterns. The first antitype would be a ∪-shaped curve that is, simultaneously, ∩-shaped. The second antitype would be a ∩-shaped curve that is, simultaneously, ∪-shaped. Neither is logically possible. Because of this pattern of contradictory shapes, the Cells 1 2 1 and 2 1 2 must be declared structural zeros. The second CFA base model considered here takes the characteristic of Cells 1 2 1 and 2 1 2 as structural zeros into account. Results appear in Table 20.9.

The overall goodness-of-fit likelihood ratio X^2 for the base model of first-order CFA for the data in Table 20.9 is 20.40. For $df = 2$, this value suggests that the model is rejected ($p < 0.01$). This model is significantly better than the one that does not take the structural zeros into account ($\Delta X^2 = 65.32$; $\Delta df = 2$; $p < 0.01$), but it cannot stand by itself. Therefore, we anticipate that types and antitypes emerge. The table shows that only one of the two types from Table 20.8 reemerges. It is constituted by cell 1 2 2.

We conclude that, in the present example, taking into account the structural zeros does not lead to a fitting model that is more parsimonious than the saturated model. Each of the structural zeros reduces the degrees of freedom by one. In the present example, there is one degree of freedom left to invest before we reach the saturated model (which is not more parsimonious than the data themselves). Including the interaction between the two first difference-variables results in a well-fitting model (likelihood ratio $X^2 = 1.517$; $df = 1$; $p = 0.218$), and the interaction parameter is significant. However, as before, the details of the interaction can be identified and interpreted in particular with CFA methods.

Extensions

Two extensions of the method of differences are particularly of note. The first concerns possible a priori probabilities. As shown by von Eye (2002), differences of any order come with a priori probabilities. Consider a series of three ordinal-level scores that describe the development of depression, with 1 indicating no depression, 2 indicating elevated depression, and 3 indicating clinical depression. For three observation points, the six sequences 1 2 3, 1 3 2, 2 1 3, 2 3 1, 3 1 2, and 3 2 1 are possible. These sequences are equally likely. Calculating, however, the first differences for the six sequences, one obtains the sign patterns + +, + −, − +, + −, − +, and − −. These patterns are not equally likely. Specifically, the probabilities are $p(++) = 0.167$, $p(+-) = 0.333$, $p(-+) = 0.333$, and $p(--) = 0.167$. Similarly, if differences are scored so that *no change* can be part of the resulting pattern, the possible patterns come with a priori probabilities that are not uniform. Uniformly distributed difference patterns can result only for continuous variables. This applies accordingly to higher order differences. Estimating expected frequencies without taking a priori probabilities into account can cause severely biased estimates for type and antitype patterns or, in log-linear modeling, severely biased parameter estimates.

A second extension of CFA or log-linear models for difference scores concerns the inclusion of additional

TABLE 20.10 First-Order CFA of the Gender × P1 × P2 × ΔP Cross-Classification; Structural Zeros Taken into Account

Configuration					
G P1 P2 ΔP	m	$\hat{m}$	z	$p(z)$	Type/antitype?
1111	4.00	10.474	−2.0003	.022733	
1112	4.00	10.199	−1.9411	.026121	
1121	.00	–	–	–	
1122	17.00	8.713	2.8074	.002497	Type
1211	19.00	14.003	1.3355	.090860	
1212	.00	–	–	–	
1221	14.00	11.962	.5891	.277886	
1222	9.00	11.649	−.7761	.218833	
2111	6.00	7.347	−.4970	.309595	
2112	10.00	7.155	1.0637	.143723	
2121	.00	–	–	–	
2122	9.00	6.112	1.1681	.121391	
2211	16.00	9.823	1.9710	.024364	
2212	.00	–	–	–	
2221	3.00	8.392	−1.8612	.031359	
2222	3.00	8.172	−1.8092	.035213	

variables or covariates. For example, given the results in Tables 20.6 and 20.9, one can ask whether the type/antitype pattern found in Table 20.9 is gender specific. Table 20.10 displays the results for this question, with structural zeros taken into account. To make these results comparable with the ones in Table 20.9, the z test was used again, along with the Holland–Copenhaver procedure for α protection.

The overall goodness-of-fit likelihood ratio X^2 for the base model of first-order CFA for the data in Table 20.10 is 33.55. For $df = 7$, this value suggests that the model is rejected ($p < 0.01$). Only one type results. It is constituted by Pattern 1 1 2 2, suggesting that females are significantly more likely than expected to initially show a decrease in aggression, that is followed by an increase, with an overall positively accelerated trend. For male adolescents, the same pattern was observed at a rate that did not differ from expectancy.

Candidates for covariates include time-invariant variables such as gender, once-observed variables such as living conditions, and repeatedly observed variables such as diagnoses, or physiological measures of development.

CFA of Trajectories: Typical and Atypical Development

In person-oriented (Bergman & Magnusson, 1997; von Eye & Bergman, 2003; von Eye, Bergman, & Hsieh, 2015; Ch. 18) and idiographic research (Molenaar, 2004; Molenaar & Campbell, 2009), it has been shown that, to be able to replace individual series of repeated observations with cross sectional data from many individuals and to draw inference conclusions as if data came from repeated observations, the data must be stationary. This condition is rarely reasonable and fulfilled in developmental processes such as learning or in temporal etiological processes. Therefore, it has been recommended that data not be aggregated across individuals. Instead, parameters are to be estimated first for longitudinal data, at the level of the individual. Aggregation can then be performed based on parameter profiles.

In this section, we present a CFA method that does exactly this, in three steps, for interval-level or ratio scale-level variables. In a first step, polynomials are estimated for each individual. These polynomials describe the trajectory of development. At least two consecutive interval- or ratio scale-level measures are needed. The method is more fruitful, however, when a series consists of three or more measures. In its second step, the polynomial parameters are binned, that is, reduced to a smaller number of binned parameters that are then crossed for CFA or log-linear analysis. In the third step, the parameter ranges are crossed with each other or with other variables and then subjected to CFA (von Eye, 2002; von Eye & Nesselroade, 1992).

There are many forms of polynomials (Abramowitz & Stegun, 1972). Using an approach that is easily related to regression analysis, a series of scores y_j can be approximated by the polynomial

$$\hat{y} = f(x) = P_I(x) = b_0x^0 + b_1x^1 + b_2x^2 + \ldots + b_Ix^I = \sum_{i=0}^{I} b_ix^i$$

where the x are either the scores of a predictor variable that is observed at the same observation points as y, or scores that describe the observation points (1, 2, ...). The number I is called the *degree of the polynomial* or the *order of the polynomial*.

In statistical data analysis, polynomials are well known and often used. For example, a standard regression analysis which fits a straight line to the data uses P1(x), that is, a first-order polynomial. Quadratic regression slopes are also often considered. In repeated measures analysis of variance with polynomial contrasts, polynomials up to order I can be estimated for I + 1 repeated measures. Polynomials are attractive tools for the description of series of measures, for a number of reasons.

1. Any series of scores can be perfectly described using a polynomial. Note, however, that estimating a polynomial that goes exactly through the observed data points is not advisable, because practically all observed

data points come with measurement error and the polynomial parameter estimates must not be affected by this error. However, this characteristic of polynomials gives researchers the option of adjusting the degree of the estimated polynomial as needed. Typically, the degree of the estimated polynomial is much below the number of observation points.

2. The coefficients of orthogonal polynomials do not need to be calculated (in particular when the points on x are equidistant). They are either part of software already, or they can be found on the Internet (e.g., www.watpon.com/table/polynomial.pdf; for detail on the estimation of polynomial coefficients, see Abramowitz & Stegun, 1972).
3. When orthogonal polynomials are used, the polynomial parameters are independent of each other. Therefore, polynomial elements of any degree can be removed from an equation without affecting other parameters. The remaining parameters do not need to be reestimated.
4. Polynomial approximation of series of scores is attractive in particular when the series are relatively short, as is the case in most social and behavioral science applications. Among the main reasons for this are that (1) a polynomial can be devised so that it approximates the observed series as closely as desired, and that (2) polynomial parameters have the same interpretation as standard regression parameters. For example, the intercepts have the same interpretation and the parameter for the first-order polynomial is equivalent to the standard regression slope.

On the downside, we find that

1. Although any series of scores can perfectly be approximated with polynomials, certain patterns are hard to approximate in the sense that polynomials of high orders may be needed for a satisfactory description. Among those patterns are cyclical series of scores (orthogonal trigonometric polynomials can be useful here) and series that approach an asymptote.
2. In many instances, the estimated polynomials cannot be used for purposes of extrapolation. Any polynomial approximates infinity, on both ends. Therefore, polynomials are valid only for the series used for estimation.

In most applications, researchers use families of orthogonal polynomials instead of standard polynomials. In these families, the element of order I is a polynomial of order I itself. Specifically,

$$f(x) = a_0\phi_0(x) + a_1\phi_1(x) + a_2\phi_2(x) + \ \ldots\ + a_I\phi_I(x) = \sum_{i=0}^{I} a_i\phi_i(x)$$

where $\phi_i(x)$ is an i-th order polynomial. The coefficients a_i are the estimated weights. They are comparable to the weights estimated in regression analysis (and can be estimated using least squares methods). This system of polynomials is orthogonal over the interval a $\leq x \leq b$, with respect to the weight function $w(x)$ if

$$\int_a^b w(x)P_n(x)P_m(x)dx = 0$$

where $P_n(x)$ and $P_m(x)$ are polynomials of degree n and m, respectively, and $n \neq m$. At the level of individual polynomials, the two polynomials $\phi_i(x)$ and $\phi_j(x)$ are orthogonal to each other if

$$\sum_{k=1}^{n} \phi_i(x_k)\phi_j(\mathrm{x}_k) = 0$$

where n is the number of points on x, and $i \neq j$.

Data Example

In the following example, we reanalyze data published by von Eye and Nesselroade (1992; von Eye, 2002). 42 college students were presented a state anxiety questionnaire four times (Nesselroade, Pruchno, & Jacobs, 1986). Each time, all respondents completed both parallel forms, A and B, of the questionnaire. This way, two series of four repeated measures resulted for each student. Von Eye and Nesselroade approximated each of these series using the second order orthogonal polynomial

$$y = \overline{y} + b_1 t + b_2 t^2$$

where $\overline{y}$ is the arithmetic mean of the scores in the series, and t counts the equidistant observation points (t = 1, 2, 3, 4). For each student, one set of parameters was estimated for each of the two series. For example, the first student's parameter estimates were $\overline{y} = 37.8$, $b_1 = 1.0$, and $b_2 = -0.8$ for parallel form A, and $\overline{y} = 38.3$, $b_1 = 0.95$, and $b_2 = -0.6$ for parallel form B. These estimates suggest that this student's scores increase with time (*linear trend*, *slope*, b_1). However, the negative quadratic trend b_2 (also called

curvature) suggests that toward the end this student's scores increase less rapidly or even become lower, countering the linear trend. This applies to both parallel forms of the instrument.

For the present re-analyses, we use the mean and the slope. The means were dichotomized at their medians, with 1 indicating above the median and 2 indicating below the median. The estimates for the slopes, b_1, were dichotomized at zero. Thus, 1 indicates a positive trend and 2 indicates a negative trend. Positive trends indicate increasing state anxiety, negative trends suggest decreasing state anxiety. Thus, zero is a natural cutoff point. The four variables A_0, A_1, B_0, and B_1 resulted from dichotomizing the parameter estimates. A_0 and B_0 represent the means of the two parallel test forms, and A_1 and B_1 represent the two linear slopes. Crossed, these four variables form the $2 \times 2 \times 2 \times 2$ $A_0 \times A_1 \times B_0 \times B_1$ contingency table.

We use first-order CFA for the analysis of this cross-classification (Table 20.11). Because of the relatively small sample size, we use the binomial test and protect α using the Holland–Copenhaver method. Table 20.11 displays the results. For the base model, we obtain an overall goodness-of-fit likelihood-ratio $X^2 = 57.56$. For $df = 11$, this value suggests that the base model must be rejected ($p < 0.01$), and we have reason to expect types and antitypes to emerge.

First-order CFA suggests the existence of two types (note that von Eye, 2002, used Anscombe's z test instead of the binomial test, and found an antitype in addition; it was constituted by cell 2 2 1 1). The types suggest that the parallel forms of the test perform as expected. The first type, constituted by configuration 1 1 1 1, describes those students who scored above the median in both parallel test forms, and showed a linear increase in state anxiety over the four measurement occasions, also in both parallel test forms. Instead of the expected about 3 respondents, 10 displayed this pattern. The second type, constituted by configuration 2 2 2 2, contains individuals who scored below the median in both parallel test forms and exhibited a trend toward lower scores over the four occasions, in both parallel test forms. Only about 2 individuals had been expected to show this pattern, but 10 did show it. The two configurations 1 1 1 1 and 2 2 2 2, thus, describe *typical* (or *prevailing*) developmental patterns. In this example, there are no *atypical developmental patterns*, which would be indicated by antitypes. However, readers are encouraged to test whether patterns that indicate that the two parallel forms result in diverging statements constitute a composite antitype.

TABLE 20.11 First-order CFA of Means and Linear Trends of Two State Anxiety Questionnaire Parallel Forms

Configuration				
$A_0A_1B_0B_1$	m	$\hat{m}$	$p(z)$	Type/antitype?
1111	10.00	3.0967	.00077668	Type
1112	.00	2.1057	.11528525	
1121	1.00	2.5581	.26593006	
1122	.00	1.7395	.16921897	
1211	5.00	3.7486	.31969519	
1212	4.00	2.5490	.24977930	
1221	.00	3.0967	.04008495	
1222	1.00	2.1057	.37085783	
2111	2.00	3.0967	.39276833	
2112	1.00	2.1057	.37085783	
2121	5.00	2.5581	.11035198	
2122	.00	1.7395	.16921897	
2211	.00	3.7486	.01971179	
2212	1.00	2.5490	.26776931	
2221	2.00	3.0967	.39276833	
2222	10.00	2.1057	.00003352	Type

As always, after performing a CFA, we ask whether there is a log-linear model that explains the frequency distribution in the cross-classification in Table 20.11. The model $[A_0]$, $[A_1]$, $[B_0]$, $[B_1]$, $[A_0, B_0]$, $[A_1, B_1]$ describes the data well ($\mathrm{LR} - X^2 = 12.79$; $df = 11$; $p = 0.172$). The two interactions suggest that the means as well as the linear slopes are associated with the other. This model supports the interpretation that the two forms of the state anxiety instrument are parallel. CFA suggests that this result is carried in particular by patterns 1 1 1 1 and 2 2 2 2.

Extensions of the method of polynomial configural analysis are straightforward. For example, one can ask whether groups of individuals differ in characteristics of their trajectories; one can ask whether covariates allow one to come to more differentiated conclusions; one can ask whether once-observed variables play a role; or one can ask whether characteristics of trajectories can be predicted, at the level of configurations, from other variables such as age or gender. Later in this chapter, we discuss CFA methods to predict trajectories.

Analyzing Series That Differ in Length

Thus far, the series of scores that we subjected to CFA shared the characteristic that they were all equal in length. However, there are many situations in which series differ in length. Apart from missing data which are dealt with using different tools, series can differ in length, for example, because clients need different numbers of therapy sessions, learners need different numbers of trials before they reach a criterion, workers are employed for different numbers of years before they retire, chess players need different

numbers of moves before a problem is solved, or students take different numbers of semesters before they complete their degree.

In all these and other cases, estimating and imputing missing data makes no sense, and neither does using such methods as full information maximum likelihood (FIML) estimation. These and other methods of missing data handling require the assumption that missing data could have been available to the analyst. In the sample cases, however, data do not and cannot exist because the process has come to an end (see the distinction between zeros, missing data, and nonexisting data; von Eye, 1989). Different methods are, therefore, needed to deal with the situation in which series of scores differ in length.

Lienert and von Eye (1986) proposed methods to characterize the characteristics of series of different length so that they can be analyzed with CFA. The principle behind these methods is that every feature of a series can be scaled, including length itself. For example, consider a sample of series of measures, the shortest of which includes k scores. For this series, polynomials can be estimated up to an order of $I = k - 1$. Now, because all other series include k scores or more, polynomials of order $I = k - 1$ can also be estimated for these series. The series can, therefore, be compared based on these polynomials. Technically, this procedure is the same as the one described in the last section, and will not be repeated here.

Similarly, when longer series are modeled using trigonometric functions, as is done in spectral analysis, spectral coefficients can be used for comparisons for the wave lengths that are applicable to the shortest of the comparison series. CFA methods for the analysis of coefficients from spectral analysis have not been discussed, so far. This possibility should be pursued in more detail in future work.

Here, we review and exemplify the methods proposed by Lienert and von Eye (1986). The authors proposed describing series of ordinal or higher level scores, regardless of length, with respect to the following three characteristics:

1. *Trend*. Series can be coded based on whether trends exist. The original method proposed by Lienert and von Eye (1986) focused on linear trends. However, depending on scale level and length of the shortest series, not only change in the linear trend (quadratic, second order polynomial), but also change in curvature (cubic, third order polynomial) and higher order components can be considered as well. Naturally, the intercept or mean can also be made part of the CFA analysis. A special case is what is called the *monotonic trend* of a series of length k. For ordinal variables, trends in changes in ordinal scores or ranks can be considered. A series is said to exhibit a monotonic trend if the inequality $y_{i+1} \geq y_i$ holds for every $i = 1, \ldots, k - 1$. This characteristic is known as *weak monotonicity*. This applies accordingly for $y_{i+1} \leq y_i$. If this inequality is violated just once, the series is said to not display a monotonic trend. Monotonicity is interesting because the trend it describes is not necessarily linear. Suppose the differences between time-adjacent measures fulfill the inequality but decrease over time, the curve approximates an asymptote but still is monotonic. Similarly, if the differences increase systematically with x, the curve is monotonic but can look similar to a quadratic curve. Finally, if the differences vary unsystematically, the curve zigzags its way to higher scores. The minimum number of scores needed for this criterion to be applicable is $k = 2$.
2. *Early completion*. In many studies, a series terminates when a particular criterion is reached. Examples of such criteria include recall rates in learning experiments and behavior thresholds in psychotherapy. Here, at least two options can be considered. First, one can specify a threshold and code a series asking whether the threshold was reached or not. Second, one can create an ordinal variable that provides a binning of the lengths of the series. The minimum number of scores needed for the early completion criterion to be applicable is 1 (e.g., a participant in a learning experiment reaches a 100% recall rate after the first trial).
3. *Qualitative characteristics*. In addition to the two criteria just discussed, any other characteristic of a curve can be considered. Examples of such characteristics include the number of errors in a problem solving task, the type of therapy used to cure a patient, the lengths of the intervals between therapy sessions, or the number of comorbid conditions that improved over the course of a treatment.

For use in CFA, the scores that describe these and other characteristics are binned, if needed, and the resulting categorical variables are crossed. The cross-classification from this procedure can then be analyzed using, for example, log-linear modeling, latent class analysis, or CFA.

Data Example

For the following example, we use data from the MIS (Bogat et al., 2004; Huth-Bocks et al., 2002; see first data example). The study assessed a sample of 206 women in their last trimester of pregnancy and at 2 months postpartum, and the women and their children every year until

the children were 14 years old. The women in the study reported a range of intimate partner violence experiences, from none to severe.

For the following data example, we use the variables Depression (*D*), Anxiety (*A*), and PTSD (*P*). We use data from the first three observation points, that is, the first three years of observation, and ask whether trends as measured in terms of weak monotonicity of the three variables are related to each other. In other words, we ask whether the three variables develop in unison. Most important from the perspective of analyzing series that differ in length is that, after three years, a portion of the original participants had left the study already. Reasons for dropping out include death of child (three instances), loss of custody, and moving away. Still, we include data from the entire sample of 206. For each of the participants, weak monotonicity could be evaluated, based on the procedure outlined before.

The three variables were coded as 1 if the criterion of weak monotonicity for a decline in scores was fulfilled and as 2 if the criterion was not fulfilled. In other words, the respondents were given a 1 if, for both comparisons of time-adjacent scores, the later score was below the earlier score. For respondents who dropped out after the second wave of data collection, only one comparison was used to determine the coding. This was done for all three variables. Using the coded complete data, we estimate two CFA base models. For both models, we use the normal approximation of the binomial test and the Holland–Copenhaver procedure for α protection. The first base model is that of standard first-order CFA of variable independence. The second is that of two-group CFA. The first model is

$$\log \hat{m} = \lambda + \lambda^D + \lambda^A + \lambda^P$$

and the second model is

$$\log \hat{m} = \lambda + \lambda^D + \lambda^A + \lambda^P + \lambda^{D,A}$$

The second model was estimated to determine whether respondents with a monotonic decrease in PTSD symptoms differ from those with a nonmonotonic development of PTSD symptoms in the development of Depression and Anxiety. To prevent estimation problems that the zero frequency in cell 2 1 1 could cause for the second of these models, we invoke the delta option for both models with $\Delta = 0.01$.

For the first model, we calculate an overall goodness-of-fit likelihood-ratio of $X^2 = 14.99$. For $df = 4$, this value leads to the rejection of the base model ($p < 0.01$) and we anticipate that types and antitypes may emerge. Table 20.12 displays results from first-order CFA.

TABLE 20.12 First-order CFA of the Cross-Classification of Monotonic Trend Information of Three Data Waves of Depression (*D*), Anxiety (*A*), and PTSD (*P*)

Configuration					
D A P	*m*	$\hat{m}$	*z*	*p*(*z*)	Type/antitype?
111	4.01	.494	5.0096	.000000	Type
112	5.01	3.951	.5382	.295236	
121	4.01	4.617	−.2858	.387507	
122	33.01	36.938	−.7131	.237895	
211	.01	1.728	−1.3126	.094664	
212	11.01	13.827	−.7843	.216445	
221	15.01	16.160	−.2981	.382828	
222	135.01	129.284	.8219	.205570	

First-order CFA shows an interesting result. Only one type resulted. This type describes the women who show a decline in the scores of all three variables. Only four, but still significantly more women than expected showed this pattern. None of the other patterns deviates significantly from expectancy.

The two-group CFA base model was estimated under the assumption that women who show monotonic decrease in PTSD may differ in levels of Depression and Anxiety from those women who do not show a monotonic decrease in PTSD. The base model for this two-group CFA can be rejected only if one or more of the following interactions exist: [*D, P*], [*A, P*], and [*D, A, P*]. Each of these interactions includes variable *P*, that is, PTSD. For this base model, we obtain the overall goodness-of-fit likelihood-ratio of $X^2 = 9.178$. For $df = 3$, this value leads to the rejection of the base model ($p = 0.027$), and we anticipate that discrimination types may emerge. Table 20.13 displays results from two-group CFA.

TABLE 20.13 Two-Group CFA of the Cross-Classification of Monotonic Trend Information of Three Data Waves of Depression (*D*), Anxiety (*A*), and PTSD (*P*), with PTSD as the Grouping Variable

Configuration				
D A P	*m*	statistic	*p*	Type?
111	4.01			
112	5.01	3.255	.000567	Discrimination Type
121	4.01			
122	33.01	−.063	.474961	
211	.01			
212	11.01	−1.197	.115600	
221	15.01			
222	135.01	−.832	.202799	

Two-group CFA presents one discrimination type. This type results from the comparison of cell 1 1 1 with cell 1 1 2. It suggests that significantly more women whose PTSD symptom scores systematically decrease show Pattern 1 1 than women whose PTSD scores do not decrease. In other words, women whose PTSD symptom scores show a monotonic decrease are significantly more likely to also show monotonic decreases in their depression and anxiety symptom scores than women whose PTSD symptom scores do not show a monotonic decrease.

On a methodological note, this result may sound implausible, when one considers that Pattern 1 1 2 is exhibited by five women but Pattern 1 1 1 only by four. However, Tables 20.12 and 20.13 also show that, in general, far more women fail to fulfill the weak monotonicity criterion than fulfilling it. From this perspective, the number five is far fewer than one would expect based on the ratios found for the other three comparison patterns.

A similar result is found when the odds ratio is calculated for cells 1 1 1 and 1 1 2 compared with all other cells combined. This odds ratio is 7.54 (log odds ratio = 2.019). This value indicates that the rate of women with Pattern 1 1 1 is more than twice as often found than women with Pattern 1 1 2 when the aggregate of all other patterns is used for a reference ($p < 0.01$).

Extensions

The approach to analyzing series that differ in length can be extended in several ways. First, one can include once-measured variables and time-invariant measures in the base model. Examples of such measures are grouping variables or information that remains unchanged such as the year of birth of respondents. Second, other characteristics of the series under study can be taken into account. For example, one can ask whether the score of a variable at the first occasion is predictive of trends, completion, or qualitative characteristics of a series. Later in this chapter, the predictive value of a score at the beginning of a series will be made part of a CFA prediction model.

CFA of Pre–Post Designs—An Application of Confirmatory CFA

In evaluation studies and in the study of effects of experiments or interventions, researchers typically follow a priori planned experimental designs. Hypotheses compatible with these designs are subjected to statistical testing, and detailed hypotheses are pursued, for example, by way of planned contrasts or between-group post hoc mean comparisons. In many cases, the dependent variables in such research are metrical, that is, measured at the interval or ratio scale level. In other cases, however, the dependent variables are frequency variables such as the number of correctly reproduced syllables or the number of times a person diagnosed with obsessive-compulsive disorder washes his or her hands. Both the number of syllables a person is able to reproduce and the number of hand washes may depend on intervention procedures. In these and similar cases, CFA is the method of choice for data analysis.

In the examples of CFA application used thus far, the method was employed in an *exploratory* context. When effects of experiments or intervention are examined, precise hypotheses often exist. In this situation, *confirmatory* application of CFA is in order. In confirmatory CFA, a priori specified hypotheses are tested. In other words, in confirmatory CFA, researchers determine before data collection which configurations are subjected to CFA tests. The number of these configurations is smaller than the number examined in exploratory CFA. Therefore, the procedures implemented to protect α need to take into account only this number instead of the total number of cells in the cross-classification. This difference in the number of tests has an important implication for the testing procedure in CFA: the significance threshold α^* will be less extreme.

Let t' be the number targeted in confirmatory CFA and t the total number of cells, with $t' < t$. Then, we obtain, in confirmatory CFA, for the Bonferroni-protected $\alpha'^* = \alpha/t'$ and, in exploratory CFA, the Bonferroni-protected $\alpha^* = \alpha/t$, with $\alpha'^* > \alpha^*$. This indicates that investing information in the specification and selection of hypotheses results in a less extreme significance threshold. This effect is known from the differences in significance thresholds between one-sided and two-sided testing, where investing information in the derivation of one-sided tests also results in less extreme significance thresholds.

Consider, for example, a cross-classification with 27 cells. The Bonferroni-protected α^* for this table is, in exploratory application of CFA, $\alpha^* = 0.05/27 = 0.0019$. If, in confirmatory research, only five cells are examined, the protected α'^* is $\alpha'^* = 0.05/5 = 0.01$. In this example, the difference between the protected α in exploratory and in confirmatory research is a factor of 5.4. That is, the protected α is, in exploratory research, 5.4 times more extreme than in confirmatory research. Types and antitypes are, therefore, more likely to emerge in confirmatory research than in exploratory research.

The rest of the testing procedure is unchanged from standard, exploratory CFA testing. The cells that were selected for testing are either tested using a constant significance threshold (as is the case in the Bonferroni procedure)

or the test statistics of these cells are ranked in descending order and tested until either the first discrepancy between the observed and the expected cell frequency fails to be significant or the number of a priori-specified cells is exhausted (Holm or Holland–Copenhaver procedures).

Treatment Effects in Designs with No Control Group

In the following sections, we present two data examples (Lienert & Straube, 1980; von Eye, 2002). The first example involves the examination of effects of neuroleptic drugs on inpatients diagnosed with schizophrenia, with no control group. The drugs were administered in a sample of 75 patients over a period of 2 weeks. At the beginning and end of this 2-week period, the patients were also administered the Brief Psychiatric Rating Scale (Overall & Gorham, 1962). Three of the 17 symptoms captured by this instrument are used in the following analyses: W = emotional withdrawal; T = thought disturbances; and H = hallucinations. Each of the symptoms was scaled as either present (+) or absent (−). Table 20.14 displays the data, along with the CFA results. We analyze these data in three different ways. The first is first-order, confirmatory CFA. For this CFA, we use the z test and the Holland–Copenhaver procedure for α protection.

The data in Table 20.14 are arranged in a somewhat unusual fashion. Instead of presenting all possible combinations of before–after treatment symptoms, we created an ordinal variable with the categories 3 symptoms observed (indicated by + + +), two symptoms observed (indicated by + + −, + − +, and − + +), one symptom observed (indicated by + − −, − + −, and − − +), and no symptom observed (− − −). This variable shows that, in this data example, we are interested in the number of symptoms instead of the individual symptoms.

TABLE 20.14 Evaluation of Treatment of Schizophrenics with Neuroleptic Drugs in a Pre–Post Study

Number of symptoms before treatment	Configurations	Number of symptoms after treatment (expected number in *italics*)				Totals
	WTH	3 + + +	2 + + − + − + − + +	1 + − − − + − − − +	0 − − −	
3	+ + +	1 *1.60*	10 *5.20*	4 *6.00*	0 *2.20*	15
2	+ + − + − + − + +	6 *4.05*	11 *13.17*	17 *15.20*	4 *5.57*	38
1	+ − − − + − − − +	1 *1.71*	4 *5.55*	7 *6.40*	4 *2.35*	16
0	− − −	0 *0.64*	1 *2.08*	2 *2.40*	3 *0.88*	6
Totals		8	26	30	11	75

The overall goodness-of-fit likelihood-ratio for the base model of variable independence is $X^2 = 17.79$. For $df = 9$, this value leads to the rejection of the base model ($p = 0.038$), and we anticipate that CFA discrimination types may emerge. However, the tail probability, for none of the 16 tests, exceeds the threshold dictated by this exploratory analysis. Under standard conditions of exploratory application of CFA we would, therefore, conclude that the first and the second observations are independent of each other and that the neuoleptic drugs have no systematic effect.

Having a hard time believing this, Lienert and Straube (1980) proposed using two additional methods of analysis for data as the ones presented in Table 20.14. The first involves using the *diagonal-half sign test*. This test can be used to compare the aggregate of the frequencies above the diagonal with the aggregate of the frequencies below the diagonal. For this test, the frequencies in the cells above the main diagonal (from upper left to lower right) in Table 20.14 are summed, and so are the frequencies below the main diagonal. Let the first sum be called b, and the second sum w. The patients in the upper triangle are those who moved, under the drug, from more to fewer symptoms. The patients in the lower triangle are those who moved from fewer to more symptoms. The diagonal-half sign test for the comparison of b with w is

$$z = \frac{b - w}{\sqrt{b + w}}$$

The test statistic z is approximately normally distributed. As an alternative, for small samples, the binomial test can be considered, which is

$$B(h) = \sum_{j=0}^{h} (N) p^j q^{N-j}$$

where, in the present case, $p = q = 0.5$, $N = b + w$, and h is the lesser of b and w. To illustrate the z test, we use the data in Table 20.14. For the upper triangle, we obtain $b = 10 + 4 + \ldots + 4 = 39$. For the lower triangle, we obtain $6 + \ldots + 2 = 14$. Inserting into the formula for z results in

$$z = \frac{39 - 14}{\sqrt{39 + 14}} = 3.434$$

The corresponding tail probability is 0.0003. From this result, we conclude that the neuroleptic drugs reduce the number of symptoms in schizophrenic inpatients.

The second method of analysis proposed by Lienert and Straube (1980) allows one to test more detailed hypotheses than the one concerning general change in the number of symptoms. One can create a preintervention × postintervention cross-tabulation for each symptom and analyze the resulting $I \times I$ table using the Bowker test (1948; von Eye & Spiel, 1996), where I represents the number of categories, or the McNemar test (McNemar, 1947), when $I = 2$. The test statistic for the Bowker test is

$$X^2 = \frac{\sum_i \sum_j (m_{ij} - m_{ji})^2}{m_{ij} + m_{ji}}$$

for $i > j$ and $i, j = 1, \ldots, I$. This test statistic is approximately distributed as χ^2 with $df = (I)$. When $I = 2$, one can use the McNemar test which has the same form but simplifies to

$$X^2 = \frac{(m_{12} - m_{21})^2}{m_{12} + m_{21}}$$

This statistic is approximately distributed as χ^2 with $df = 1$. When cell frequencies are small, the continuity-corrected equation

$$X^2 = \frac{(\mid m_{12} - m_{21} \mid -0.5)^2}{m_{12} + m_{21}}$$

is recommended. Von Eye (2002) presents the following application example for this equation. In the example, the question is asked whether the neuroleptic drug reduces the number of hallucinations in Lienert and Straube's (1980) neuroleptic drug treatment study in particular. The cell frequencies for hallucinations are $++ = 8$, $+- = 21$, $-+ = 9$, and $-- = 32$, where $+$ indicates presence and $-$ indicates absence of a symptom. For these values one obtains

$$X^2 = \frac{(21 - 9)^2}{21 + 9} = 4.80$$

For $df = 1$, the probability of this value is $p = 0.0285$. We, therefore, reject the null hypothesis that the neuroleptic drug treatment leads to no better than random changes in the number of hallucinations.

An interesting alternative to the tests presented in this section can be found in the literature on rater (dis)agreement. In the present context, we ask whether a shift can be ascertained that manifests in a larger number of cases than expected in the upper or lower triangle of the cross-classification of the repeated observations. This pattern corresponds with the pattern of disagreement in which one rater systematically uses higher ratings than the other. Von Eye and von Eye (2005) proposed coefficients for this case. These coefficients are derivatives of Cohen's (1960) κ (kappa) and can therefore be interpreted as proportionate reduction in error (PRE) coefficients. Focusing on the upper triangle of an agreement matrix, one can ask whether disagreement differs from expectation. To answer this question, we need to define a base model. A first option is that we define this model in a fashion similar to the base model for first-order CFA, that is, as a model of rater independence. The same model is used for Cohen's κ. A second option is to define the base model as the null model, as for Brennan and Prediger's (1981) κ_n. Using these base models, one can define coefficients of disagreement or, as in the present context, temporal shifts in symptoms, for the upper triangle of an agreement table.

A coefficient of disagreement in the upper triangle of an agreement table can be defined in a fashion analogous to Cohen's κ. The probability of disagreement in the upper triangle of the cross-classification, that is, the probability that Rater B (columns) selects higher scores than Rater A (rows) or the probability that fewer symptoms are diagnosed at Time 2 than at Time 1 is

$$\theta_1^t = \sum_{i<j} p_{ij} = 1 - \sum_i p_{ii} - \sum_{i>j} p_{ij}$$

where the superscript indicates that we consider the disagreement cells in the upper triangle. Under the base model of independence of the first from the second observation (or the base model of rater independence), the expected probability of finding cases in the upper triangle in the upper triangle is

$$\theta_2^t = \sum_{i<j} p_{i.} p_{.j} = 1 - \sum_i p_{i.} p_{.i} - \sum_{i>j} p_{i.} p_{.j}$$

Using θ_1^t and θ_2^t, one can define a κ-like PRE coefficient as

$$\kappa^t = \frac{\theta_1^t - \theta_2^t}{1 - \theta_2^t}$$

This coefficient also has a range of $-\infty < \kappa^t \leq 1$ (for specifics on range and special cases, see von Eye & von Eye, 2005).

To define a coefficient κ_n^t that is analogous to Brennan and Prediger's (1981) coefficient κ_n, we select the null model as the base model. This model is also used in CFA of zero

order. For θ_1^t, we use the same definition as above. For the expected probability, θ_2^t, we obtain

$$\theta_{n,2}^t = \binom{I}{2}\frac{1}{I^2} = \frac{I-1}{2\mathrm{I}}$$

and, thus,

$$\kappa_n^t = \frac{\theta_1^t - \theta_{n,2}^t}{1-\theta_{n,2}^t}$$

This coefficient has a range of $-\infty < \kappa_n^t \leq 1$ (again, for specifics on range and special cases, see von Eye & von Eye, 2005).

There are three major differences between the diagonal-half sign test proposed by Lienert and Straube (1980) and the PRE coefficients proposed by von Eye and von Eye (2005). First, the PRE coefficients can always be interpreted in units of proportionate reduction in error. That is, the PRE coefficients tell the user the increase in the percentage of cases that can be found in the upper triangle in comparison to the expected percentage. Second, the user can select from a number of base models. These models are selected with the goals of CFA in mind. Third, the effects of covariates can be made part of the analysis by estimating the expected frequencies using the model $\log \hat{m} = X\lambda + X^c\lambda^c$ instead of $\log \hat{m} = X\lambda$, where the extended model includes, in its second term, the covariate(s). The rest of the calculations remains unchanged from the case with no covariates.

To illustrate the PRE coefficients, we reanalyze the data example in von Eye and von Eye (2005; Fennig, Craig, Tananberg-Karant, & Bromet, 1994; Schuster & Smith, 2002). The data describe psychiatric diagnoses of the same 223 patients, once made by practicing psychiatrists and once in a research context. The goal of the original analyses was to determine whether there is agreement between the diagnoses provided by the service facility and research teams. The patients were rated on an ordinal 4-point severity scale with the categories 1 = severe psychosis, 2 = average severity psychosis, 3 = mild psychosis, and 4 = no clinical diagnosis. Table 20.15 gives the cross-classification of the facility with the research diagnoses.

The frequency distribution in Table 20.15 shows that the two diagnoses coincide in most cases. In each row of the cross-classification, the agreement cell (diagonal cell) contains the largest number of cases. Also in each row, the agreement cell contains more cases than expected under the base model of independent diagnoses. This is also indicated by the coefficients discussed by von Eye and von Eye (2005).

TABLE 20.15 Cross-Classification of Facility and Research Diagnoses for 223 Psychiatric Patients

Facility diagnosis	Research diagnosis								
	1		2		3		4		Totals
1	40	*18.95*	6	*13.41*	4	*11.08*	15	*21.57*	65
2	4	*10.20*	25	*7.22*	1	*5.96*	5	*11.61*	35
3	4	*10.49*	2	*7.43*	21	*6.14*	9	*11.95*	36
4	17	*25.36*	13	*17.95*	12	*14.83*	45	*28.87*	87
Totals	65		46		38		74		223

Note: Expected cell frequencies in *Italics* (base model of first-order CFA). Agreement cells underlined.
Source: Fennig et al. (Fennig et al., 1994)

Looking at the upper triangle, we obtain $\kappa^t = -0.24$. This value suggests that the proportionate reduction in these error cells is 24.14%. In other words, the upper triangle contains 24.14% fewer cases than one would expect under the assumption of independence of the two diagnoses. Proceeding from the null model, that is, the model that does not take the differences in the marginal probabilities into account, we use the expected frequency of 223/16 = 13.9375, for each cell. For the κ_n equivalent, the value of $\kappa_n^t = -0.31$ indicates that 31.30% discrepant diagnoses are made less than expected under the assumption of a uniform diagnoses distribution. The coefficient of raw disagreement for the upper triangle (this is just the proportion of cases in the upper triangle) of the disagreement cells is $ra^t = 0.18$. None of these values is significant. For example, $ra^t = 0.18$ comes with a z score of 0.10 and $p = 0.46$ (for this significance test, see von Eye, Schauerhuber, & Mair, 1997).

For comparison, we also calculate the test discussed by Lienert and Straube (1980). We obtain $w = 6 + 4 + \ldots + 9 = 40$, $b = 52$, and $z = \frac{40-52}{\sqrt{40+52}} = -1.25$. The tail probability for this value is 0.11, thus indicating that there is only an insignificant shift toward more severe ratings in the facility as compared to the research unit.

Apart from indicating nonsignificant effects, this example shows that the various coefficients are sensitive to different characteristics in the data table. Lienert and Straube's test responds to shifts toward to upper or lower triangle. Von Eye and von Eye's κ equivalents compare the observed frequencies in sectors of the cross-classification with expectancies that reflect assumptions concerning marginal probabilities and, thus, effects in the table.

Treatment Effects in Designs with Control Group

Experimental designs without a control group are deemed less informative or more biased than designs with control group. However, designs without a control group can still

TABLE 20.16 $2 \times 2 \times 2$ Table for the Comparison of Pattern Shift in Two Groups

	Posttreatment patterns treatment group		
Pretreatment patterns	A	B	Totals
A	m_{111}	m_{112}	$m_{11.}$
B	m_{121}	m_{122}	$m_{12.}$
Totals	$m_{1.1}$	$m_{1.2}$	$m_{1..}$
	Control group		
	A	B	
A	m_{211}	m_{212}	$m_{21.}$
B	m_{221}	m_{222}	$m_{22.}$
Totals	$m_{2.1}$	$m_{2.2}$	$m_{2..}$
Sample Totals	$m_{..1}$	$m_{..2}$	N

TABLE 20.17 2×2 Table for the Comparison of Pattern Shift in Two Groups

Patterns	Comparison groups treatment	Control	Totals
m_{112} versus m_{212}	m_{112}	m_{212}	$m_{212} + m_{212}$
all others combined	$m_{111} + m_{121} + m_{122}$	$m_{211} + m_{221} + m_{222}$	$N - m_{212} + m_{212}$
Totals	$m_{1..}$	$m_{2..}$	$N = m_{1..} + m_{2..}$

provide information about whether or not treatment effects can be expected at all. If, in a design without a control group, results suggest that effects may exist, controlled studies can be undertaken (if they are not undertaken from the very start). Randomized clinical trials (RCTs) are necessary for a number of reasons. One is to compare treatments with each other. For example, a new form of psychotherapy is compared with an established one. Most important, in particular in psychotherapy research, RCTs are needed to distinguish spontaneous recovery, assessment reactivity, or expectancy effects from treatment effects.

CFA can be used to compare experimental groups and control groups with two-group CFA, or, when there are more than two groups, with multigroup CFA. Here, we discuss methods that allow one to compare two groups in regard to the change from one configuration to another. To explain the scenario, consider one treatment group and one control group. For both groups, Patterns A and B are observed before and after the intervention administered to the treatment group. Table 20.16 schematizes the data situation.

The goal of analysis is the comparison of the control with the treatment group in a particular change pattern. In the example in Table 20.16, there are only two change patterns, the change from $m_{.11}$ to $m_{.12}$ and from $m_{.21}$ to $m_{.22}$, where, in the table as well as here, in the text, a dot indicates the variable aggregated across. The null hypothesis for the present comparison is that the comparison groups do not differ in their change rates. Specifically, we ask whether m_{112} differs from m_{212}, or whether m_{121} differs from m_{221}. The answer to these questions is given on the basis of 2×2 cross-classifications. Table 20.17 displays this cross-classification for the first comparison.

In Table 20.17, the notation for the cell frequencies carries over from Table 20.16. Table 20.17 shows a 2×2 cross-classification in which Frequency m_{112} is compared with m_{212}, in the context of the rest of the treatment and control group samples. Tables as the one in Table 20.17 can be analyzed with marginal-dependent measures such as Pearson's X^2 or with marginal-free measures as the odds ratio (for the distinction between marginal-dependent and marginal-free measures, see Goodman, 1991, or von Eye, Spiel, & Rovine, 1995).

Data Example

In the following example, we reanalyze the data presented by Martinez-Torteya, Bogat, von Eye, Levendosky, and Davidson (Martinez-Torteya et al., 2009). The data were collected in the context of the MIS (//www.msu.edu/~mis/). As was indicated before, the study first assessed a sample of 206 women in their last trimester of pregnancy and at 2 months postpartum, and the women and their children every year until the children were 14 years old. The women in the study reported a range of experiences of intimate partner violence, from none to severe. In the example, we focus on the 93 women who reported one or more episodes of partner violence during the second year of their child's life. We ask whether frequency of violence (F) experienced during the second year of their child's life is predictive of Depression (D) and PTSD (P) in the same year. Each of these three variables was dichotomized at the median, and coded such that the value of 1 indicates a score below the median and 2 indicates a score above the median. Table 20.18 displays the $2 \times 2 \times 2$ $D \times P \times F$ cross-classification along with CFA results. We first analyze these data using a standard first-order CFA with the base model $\log \hat{m} = \lambda + \lambda^D + \lambda^P + \lambda^F$. Then, we ask whether particular comparison patterns stand out. For the CFA tests, we use the binomial approximation of the normal distribution and the Holland–Copenhaver procedure for α protection.

The overall goodness-of-fit likelihood-ratio for the base model of variable independence is $X^2 = 44.15$. For $df = 4$, this value leads us to reject of the base model ($p < 0.01$), and we anticipate that types and antitypes may

TABLE 20.18 Cross-Classification of Depression (*D*), PTSD (*P*), and Frequency of Violence (*F*)

Configuration					
DPF	m	$\hat{m}$	z	$p(z)$	Type/antitype?
111	22.00	10.989	3.5374	.000202	Type
112	9.00	12.237	−.9930	.160343	
121	3.00	10.302	−2.4125	.007922	Antitype
122	11.00	11.472	−.1490	.440789	
211	14.00	11.721	.7120	.238228	
212	3.00	13.053	−3.0011	.001345	Antitype
221	5.00	10.989	−1.9238	.027191	
222	26.00	12.237	4.2218	.000012	Type

Note: Expected cell frequencies are estimated under the base model of first-order CFA.

emerge. Indeed, Table 20.18 shows two types and two antitypes. The first type is constituted by configuration 1 1 1. These are women who report below-average frequencies of violence, along with below-average scores in Depression and PTSD. The second type is constituted by just the opposite pattern, 2 2 2. These are women who report above average frequencies of violence, along with above average Depression and PTSD. The two antitypes are constituted by Patterns 1 2 1 and 2 1 2, indicating that it is very unlikely that low frequencies of violence lead to high levels of depression but low levels of PTSD or high levels of depression but low levels of PTSD, in the presence of intimate partner violence.

To illustrate the application of the scheme shown in Table 20.17, we now consider the two levels of violence the comparison groups. The variables Depression and PTSD are crossed to form the patterns in which the two treatment groups are compared. It is important to note that the analysis we are about to illustrate is not just an extension of first-order CFA. In terms of CFA base models, the following analysis represents a 2-group CFA that is saturated in the comparison variables. Specifically, the expected frequencies are estimated using the first-order base model

$$\log \hat{m} = \lambda + \lambda^D + \lambda^P + \lambda^F + \lambda^{DP}$$

The main reason for using this base model is that types and antitypes (or discrimination types) can no longer appear just because, maybe, the discrimination variables Depression and PTSD are associated. The two treatment groups can differ in a pattern only if one or both of the discrimination variables is related to the grouping variable. We insert, for instance, Pattern $D = 1$ and PTSD = 1 into a scheme as it is shown in Table 20.17 and obtain the cross-classification in Table 20.19.

TABLE 20.19 2×2 Table for the Comparison of Two Groups in a Two-Variable Pattern

	Violence Frequency		
Patterns	Below Average	Above Average	Totals
m_{111} versus m_{211}	$m_{111} = 22$	$n_{211} = 9$	$m_{111} + m_{211} = 31$
all others combined	22	40	62
Totals	44	49	$N = 93$

The Pearson X^2 for the data in Table 20.19 is 10.44 ($df = 1$; $p < 0.01$). This result suggests that below-average frequency of violence is predictive of higher frequencies of low Depression and PTSD than above average violence.

Two points concerning the method used for analysis must be discussed, in this context. First, it is important to note that this result is not just a reformulation of the description of the first type in Table 20.18. The type suggests that the pattern low frequency of Violence–low Depression–low PTSD is *more likely than expected*. Here, we can make a comparative statement in which we contrast the two violence groups.

Second, the MIS study oversampled, on purpose, women who were victims of violence. Therefore, the marginal frequencies in Table 20.19 (as well as the univariate frequencies for tables that use the MIS data) may not reflect population characteristics. X^2 measures are known to be marginal-dependent. Thus, the result from the 2×2 cross-classification in Table 20.19 may be distorted or biased. A measure that is marginal-free is the *odds ratio*. Consider a 2×2 table with the cell frequencies m_{11}, m_{12}, m_{21}, and m_{22}. A cross-classification of this type (parallel to the one displayed in Table 20.17) is given in Table 20.20.

Table 20.20 contains the information for four odds and one odds ratio (see von Eye & Mun, 2013):

1. $\Omega_1 = p_{11}/p_{12}$: the odds of a diagnosis of the undesirable outcome versus no such undesirable outcome under treatment;
2. $\Omega_2 = p_{21}/p_{22}$: the odds of a diagnosis of the undesirable outcome versus no such undesirable outcome without treatment;

TABLE 20.20 Cross-Classification of Treatment and Outcome Pattern and Cell Frequencies

	Diagnosis of undesirable outcome	
Treatment	Yes	No
Yes	m_{11}	m_{12}
No	m_{21}	m_{22}

3. $\Omega_3 = p_{11}/p_{21}$: the odds of a diagnosis of the undesirable outcome in the treatment versus the comparison groups;
4. $\Omega_4 = p_{12}/p_{22}$: the odds of a diagnosis of no undesirable outcome in the treatment versus the comparison groups.

Each of these odds has an easy, natural interpretation, and so does the odds ratio that relates these odds to each other. Consider $\theta^T = \Omega_1/\Omega_2$. This odds ratio can be used to test the hypothesis that respondents in the treatment group are more likely to be diagnosed with the undesirable outcome than respondents who were not exposed.

It is interesting to note that there are more odds ratios that could be considered for this 2 × 2 table. However, because they all use the same information, they all have the same interpretation. Specifically, we obtain

$$\theta = \frac{\frac{p_{11}}{p_{12}}}{\frac{p_{21}}{p_{22}}} = \frac{\frac{p_{11}}{p_{21}}}{\frac{p_{12}}{p_{22}}} = \frac{p_{11}p_{22}}{p_{12}p_{21}}$$

As far as the odds ratio is concerned, one can formulate the hypothesis that the odds ratio $\theta^T = \Omega_1 / \Omega_2$ is greater than one in the population, where the superscript T indicates that the odds ratio contrasts the treatment and the comparison groups. To test this hypothesis, one can divide the logarithm (base e) of the odds ratio by its standard error (se), which can be estimated by

$$se(\log \hat{\theta}) = \sqrt{\frac{1}{m_{11}} + \frac{1}{m_{12}} + \frac{1}{m_{21}} + \frac{1}{m_{22}}}$$

The test statistic for the logarithm of the odds ratio is

$$z_\theta = \frac{\log \hat{\theta}}{se(\log \hat{\theta})}.$$

This statistic is approximately normally distributed. Applied to the data example in Table 20.19, one obtains $\hat{\theta} = 4.44$, $\log \hat{\theta} = 1.49$; $\text{se}(\log \hat{\theta}) = 0.48$; $z = 3.13$; and $p < 0.01$. We note that, in this example, the X^2 and the odds ratio analyses suggest the same conclusion. If the present X^2 result is biased, the distortion has no effect on the present result.

Single-Subject Designs

Why would one conduct research using single-subject designs when generalization of results is the main goal of scholarly activity? There are many answers to this question. Here, we focus on two answers that are of interest from a person-oriented perspective. First, in person-oriented (Bergman & Magnusson, 1997; von Eye & Bergman, 2003; von Eye, Bergman, & Hsieh, 2015) and idiographic research (Molenaar, 2004; Molenaar & Campbell, 2009; von Eye, 2004b), it has been shown that results that are based on longitudinal aggregated raw data can be trusted only under rare and unrealistic conditions such as ergodicity. What needs to be done is (1) estimation of parameters at the level of individuals and (2), if needed, aggregation of cases based on their parameters. If this research strategy is followed, one needs longitudinal single-subject designs instead of cross sectional studies.

Second, researchers can be interested in an individual in his or her own right, in a treatment study in which treatment can be extensive, or when random assignment can be ethically difficult. There can be a situation in which an individual can be different from other individuals to a degree that aggregation would bias the aggregation of parameter estimates beyond the tolerable. On the other extreme there may be an individual that is known to belong to a population and researchers are interested in how the individual responds to a new form of treatment.

A number of single-subject designs has been developed for CFA (von Eye, 2002; von Eye et al., 2010). Here, we begin with *single-subject CFA*. This is followed by *CFA of runs* and *cross-lagged CFA*.

Single-Subject CFA

When a CFA is considered for multidimensional data that stem from a single individual, the resulting cross-classification that is being analyzed does not differ in form from the cross-classification of variables across multiple individuals. Single subject data are different however, in that they were collected over multiple occasions. Consider the relationships between the three variables A, B, and C, observed over n occasions for one individual. The $A \times B \times C$ cross-classification has n entries. It allows one to test hypotheses concerning the association structure of the three variables, for that particular individual. It also allows one to search for types and antitypes that contradict a particular base model, also for the particular individual.

Data Example

To illustrate this approach, we use data from an intensive longitudinal study of self-identified alcoholics (Perrine, Mundt, Searles, & Lester, 1994). A sample of alcoholic adult men provided every morning information about their drinking the day before, as well as their subjective ratings of mood, health, and quality on the day of the interview. Here, we ask whether there are associations among mood (M) of

Respondent 3000, his subjective reports of health (H) and beer drinking (B). Data assessed from 734 consecutive days were available. Mood was rated on a 10-point Likert scale with 1 indicating "terrible mood" and 10 indicating "just wonderful." Respondent 3000 rarely endorsed the low and the high end points on this scale. Therefore, scores 4 and below were combined into a single category. Similarly, this respondent nearly never used scores 7 through 10. Therefore, scores 7–10 were also combined into a single category. The resulting scale thus ranged from 1 to 4 with 1 indicating average mood or below, and 4 indicating good or better mood. Similarly, the range of health ratings was condensed from a 10-point Likert scale to the values 1–4. The resulting scale values indicate 1 = perception of average health or below through 4 = perception of good to excellent health. The range of the numbers of beer consumed by Respondent 3000 was wide, with the extremely high numbers rarely reported. Therefore, this scale was also condensed to range from 1 = no beer consumed through 3 = three or more beers consumed in one day.

For the analysis of the resulting $4 \times 4 \times 3$ H × M × B cross-classification, we use the z-test and the Holland–Copenhaver procedure of α protection. The first base model we use is that of first-order CFA,

$$\log \hat{m} = \lambda + \lambda^H + \lambda^M + \lambda^B$$

This model can be rejected when any combination of the interactions [*B, M*], [*B, H*], [*M, H*], [*B, M, H*] exists, that is, any interaction at all.

For the base model of first-order CFA, we obtain a $\mathrm{LR} - X^2 = 89.60$ ($df = 39$; $p < 0.01$). Therefore, we anticipate that types and antitypes emerge. Table 20.21 summarizes the CFA results.

The results in Table 20.21 suggest that only one type exists and no antitype. This type indicates that comparatively low ratings of mood and health co-occur with low consumption of beer more often than expected. This result reflects an extreme case of a *local association* (Havránek & Lienert, 1984). The associations in this table of 48 cells are carried by just one cell. One could argue that cell 1 1 2 also makes a contribution. However, under the restrictions of α protection, this contribution is nonsignificant if this cell is examined by itself. If one tests whether cells 1 1 1 and 1 1 2 constitute a *composite type*, one obtains a Stouffer $Z = 6.93$, which suggests a strong deviation from the independence model for this pair of cells. This composite type suggests that health and mood ratings are at their lowest when Respondent 3000 consumed two beers or less on the day before the interview.

TABLE 20.21 First-Order CFA of the Cross-Classification of Numbers of Subjective Health (H), Mood (M), and Beer Consumed (B), for Respondent 3000

	Configuration				
HMB	m	$\hat{m}$	z	$p(z)$	Type/antitype?
111	11.00	1.823	6.7972	.000000	Type
112	21.00	11.089	2.9762	.001459	
113	38.00	42.837	−.7391	.229927	
121	7.00	5.469	.6549	.256278	
122	38.00	33.267	.8205	.205954	
123	110.00	128.512	−1.6330	.051236	
131	.00	3.038	−1.7430	.040665	
132	15.00	18.482	−.8099	.208996	
133	77.00	71.396	.6633	.253577	
141	.00	.820	−.9057	.182548	
142	5.00	4.990	.0044	.498231	
143	19.00	19.277	−.0630	.474865	
211	.00	.898	−.9477	.171650	
212	8.00	5.463	1.0853	.138893	
213	12.00	21.105	−1.9819	.023747	
221	3.00	2.694	.1863	.426105	
222	11.00	16.390	−1.3313	.091542	
223	77.00	63.314	1.7200	.042715	
231	1.00	1.497	−.4061	.342351	
232	5.00	9.105	−1.3605	.086831	
233	43.00	35.174	1.3195	.093503	
241	.00	.404	−.6357	.262482	
242	1.00	2.458	−.9302	.176141	
243	7.00	9.497	−.8103	.208888	
311	.00	.631	−.7942	.213533	
312	5.00	3.837	.5936	.276405	
313	14.00	14.823	−.2139	.415319	
321	.00	1.892	−1.3756	.084468	
322	16.00	11.512	1.3228	.092951	
323	47.00	44.470	.3793	.352224	
331	.00	1.051	−1.0253	.152603	
332	1.00	6.395	−2.1335	.016442	
333	24.00	24.706	−.1420	.443541	
341	.00	.284	−.5328	.297093	
342	1.00	1.727	−.5531	.290106	
343	10.00	6.671	1.2891	.098681	
411	.00	.572	−.7563	.224736	
412	2.00	3.480	−.7932	.213836	
413	9.00	13.442	−1.2115	.112856	
421	2.00	1.716	.2168	.414167	
422	11.00	10.439	.1737	.431041	
423	38.00	40.325	−.3661	.357140	
431	.00	.953	−.9764	.164439	
432	4.00	5.799	−.7472	.227484	
433	30.00	22.403	1.6051	.054234	
441	.00	.257	−.5073	.305958	
442	2.00	1.566	.3470	.364301	
443	9.00	6.049	1.2000	.115073	

We also ask whether the pairwise associations that involve mood, that is, [*B, M*], [*M, H*], allow us to explain the data. To answer this question, we estimate the log-linear model

$$\log \hat{m} = \lambda + \lambda^H + \lambda^M + \lambda^B + \lambda^{BM} + \lambda^{MH}$$

and a multinomial logit model. This model describes the data well. Specifically, we obtain a LR $- X^2 = 33.87$ ($df = 24$; $p = 0.251$). This model clearly stands by itself. It is also significantly better than the base model of first-order CFA (ΔLR $- X^2 = 55.73$; $\Delta df = 15$; $p < 0.01$). Both interactions are significant. Similarly, a multinomial logit model comes with an X^2 of 53.54, which indicates that beer consumption and subjective health can be used as predictors of mood ($df = 15$; $p < 0.01$).

This result illustrates again the difference between variable-oriented and person-oriented research. The former allows one to state that, in the present example, mood is associated with both beer consumption and subjective health ratings. The person-oriented results, however, suggest that (1) comparatively low beer consumption goes hand in hand with comparatively poor mood and low health ratings and that (2) otherwise the three variables are independent of one another.

Although interesting, the approach to single subject CFA just described lacks a developmental perspective in the sense that possible change over time is aggregated over. Therefore, we present, in the next example an approach to CFA that takes the longitudinal nature of data into account.

TABLE 20.22 Prediction CFA of the Cross-Classification of Numbers of Beer Consumed (*B*) and Subjective Health (*H*), over the First and the Second Halves of the Observation Period (*P*); for Respondent 3004

	Configuration			
BHP	m	$\hat{m}$	z	Type/antitype?
111	1.000	0.496	0.716	
112	0.000	0.504	−0.710	
121	0.000	0.496	−0.704	
122	1.000	0.504	0.698	
131	3.000	66.452	−7.784	Antitype
132	131.000	67.548	7.720	Type
211	1.000	0.496	0.716	
212	0.000	0.504	−0.710	
221	0.000	0.496	−0.704	
222	1.000	0.504	0.698	
231	0.000	14.381	−3.792	Antitype
232	29.000	14.619	3.761	Type
311	2.000	1.488	0.420	
312	1.000	1.512	−0.417	
321	1.000	0.992	0.008	
322	1.000	1.008	−0.008	
331	3.000	15.373	−3.156	Antitype
332	28.000	15.627	3.130	Type
411	19.000	20.332	−0.295	
412	22.000	20.668	0.293	
421	3.000	1.984	0.722	
422	1.000	2.016	−0.716	
431	331.000	241.014	5.796	Type
432	155.000	244.986	−5.749	Antitype

Data Example

We use data from the study on the development of alcoholics again. Instead of Respondent 3000, we now use the answers from a different participant, Respondent 3004. He provided data on 742 consecutive days. To keep the example simple, we ask whether the relationship between beer consumption (B) and subjective health (H) varies from the first to the second half of the observation period (P). Each variable was coded so that small numbers indicate low levels and high numbers indicate high levels (or perceptions indicating good levels).

This question can be answered using a base model that has two characteristics. First, the observation period is proposed to be independent of beer consumption and subjective health. Second, beer consumption is proposed to be associated with health rating. The base model is that of a Prediction CFA,

$$\log \hat{m} = \lambda + \lambda^B + \lambda^H + \lambda^P + \lambda^{BH}$$

This model can be rejected only if one or more of the following interactions exist: [*B, P*], [*H, P*], [*B, H, P*]. Each of these interactions contains the variable that indicates the first versus the second half of the observation period. If one or more of these interactions exist, the association between beer consumption and health varies over the two observation periods. For the following Prediction CFA, we use the z test and the Holland–Copenhaver procedure for α-protection. Table 20.22 displays results of CFA.

The results in Table 20.22 suggest that four pairs of prediction type/antitype exist. Each of these pairs suggests that a pattern that stands out in the first half of the observation period also stands out in the second half, but for the opposite reason. Specifically, the first antitype, constituted by configuration 1 3 1, suggests that it is very unlikely that, for this respondent, very low levels of beer consumption together with subjective ratings of average to good health are reported during the first half of the observation period. More than 66 daily instances of this pattern are expected, but it was found only for 3 days. This pattern reverses dramatically in the second half of the period. More than 67 instances of the pattern of very low levels of beer consumption and average to good health are expected, but 131 instances were found. Configuration 1 3 2 thus constitutes a prediction type.

Similarly, the second antitype, constituted by configuration 2 3 1, suggests that low beer consumption in combination with average or better subjective health is unlikely for the first half of the observation period. It constitutes a prediction antitype. In the second half, however,

this pattern is particularly likely, and constitutes a prediction type. Similarly, Pattern 3 3 (average beer consumption in tandem with subjectively average or better health) is unlikely in the first half (three instances observed versus over 15 expected) but very likely in the second half of the observation period (28 instances observed versus over 15 expected).

For Pattern 4 3, the relationship is reversed. This pattern indicates above average beer consumption in tandem with subjectively above average or better health, can be predicted to occur more often than expected for the first half of the study (331 observed versus 241 expected instances) but less often than expected in the second half (155 observed versus 244 expected instances). Pattern 4 3 is also the most frequent of all patterns for this respondent.

If the temporal aspect of the observations is not taken into account, that is, after aggregating responses over time, the $B \times H$ cross-classification (aggregated over the two periods of time; not shown here) would have contained just one type. This type suggests that drinking small amounts of beer on any given day goes hand in hand with average or better self-reported health. This type can, evidently, not be generalized over time. In the second half of the observation period, it transmutes into an antitype.

CFA of Runs

Published for the first time in the context of CFA of intensive longitudinal data, CFA of runs focuses on particular characteristics of series of data. Using runs, researchers answer the question whether there is randomness in a series of data. For a definition of *runs*, consider a series of observations. A run is defined as a pattern in this series. In general, a run is (1) an uninterrupted series of bits that share one or more characteristics that is (2) bounded on both sides by bits of different characteristics. In the following paragraphs, we give a selection of examples (for the well-known runs tests, see Stevens, 1939; Swed & Eisenhart, 1943; Wald & Wolfowitz, 1940; for runs in the context of CFA and an algorithmic perspective, see von Eye et al., 2010). The definitions involve single scores, but they can also involve two or more scores. For single scores, a run can be defined as

1. The *length*, $k > 1$, *of uninterrupted series of scores of the same value*. This is the classical definition of a run. For example, the series 1 2 3 3 3 4 4 5 6 7 contains two runs. The first involves the three 3s, that is $k_1 = 3$. The second involves the two 4s, that is $k_2 = 2$, where the subscripts index the runs. This definition of runs requires no more than nominal level scales.
2. The *length*, $k > 1$, *of an uninterrupted series of scores of increasing value*. The series 8 9 9 3 4 5 6 contains 2 runs. The first involves the first two scores, and the second involves the last four scores. For runs of series of increasing scores, one needs scales that are at least ordinal.
3. The *length*, $k > 1$, *of an uninterrupted series of scores of decreasing value*. As for runs of decreasing scores, runs of decreasing scores require no more than ordinal scales.
4. The *length*, $k > 1$, *of an uninterrupted series of scores within a pre-specified range*. For this definition, at least ordinal information is needed.
5. For pairs of scores, a run can also be defined as
6. The *length*, $k > 3$, *of an uninterrupted series of scores that go up-and-down* or *down-and-up* (Wallis & Moore's, 1941, *runs up-and-down* test). The series 1 2 5 4 6 7 6 9 contains two up-and-down runs. The first run contains the values 2, 5, and 4, and the second contains the values 6, 7, and 6. The series also contains two down-and-up runs. The first contains the values 5, 4, and 6. The second contains the values 7, 6, and 9. To describe the runs of up-and-down or down-and-up series of scores, one needs at least ordinal information.
7. The *length*, $k > 1$, *of an uninterrupted series of pairs of scores in which one particular score follows the other particular score*. Consider, for example, the series 4 1 0 1 0 1 0 6 7 8. This series contains one run of length $k = 3$ of 1s and 0s that follow each other.

More complex definitions of runs are conceivable. As was illustrated for the case of up-and-down and down-and-up runs, a series can contain runs that follow more than one definition. In most applications of runs tests, however, just one definition is used.

For use in CFA, runs information from different variables can be crossed directly, if the number of different runs is limited. This is the case when the series under study are relatively short. When series are long, many runs can result that differ in length. Therefore, for long series, researchers either create classes of runs, e.g., 'did not drink hard liquor for a week' or 'depression did not relapse for one month', or they focus on particular runs such as the longest.

The questions that CFA allows one to answer concerning runs information differ from the questions asked when runs tests are applied. With runs tests, one asks whether a series of scores or observations is random in nature. If the corresponding null hypothesis is rejected, one can entertain hypotheses concerning the processes that cause deviations from randomness. In CFA, one asks whether runs information for one variable is related to runs information

from other variables or to other variables, in general. The answers to these questions are given in terms of local deviations from CFA base models, that is, in terms of types and antitypes.

Data Example

For the following example, we use data from the Finkelstein, et al. (1994) study on the development of aggressive behavior. The authors administered a questionnaire concerning aggressive behavior to 67 adolescent girls and 47 boys at three points in time. The time intervals were two years each. The questionnaire addressed the four measures of aggression: Aggressive Impulse, Aggression-Inhibitory Response, Verbal Aggression against Peers, and Physical Aggression against Peers. In the following analyses, we use the data on physical aggression against peers (PAAP), verbal aggression against adults (VAAA), and aggressive impulses (AI). For each of the variables, three scores are available, PAAP83, PAAP85, and PAAP87, VAAA83, VAAA85, and VAAA87, and AI83, AI85, and AI87, where the numbers indicate the years in which the data were collected. When the data were collected in those years, the respondents were 11, 13, and 15 years of age, respectively. In the following example, we look at *uninterrupted series of scores of decreasing value*. The question we ask concerns the decrease in aggression in adolescents. Specifically, we ask whether there are local associations among patterns of steady decrease in the three aggression variables: physical aggression against peers (PAAP), verbal aggression against adults (VAAA), and aggressive impulses (AI).

For three observation points, there can only be increasing- or decreasing-score runs of length $k = 2$. In the present example, a run of $k = 2$ indicates a decrease in scores from 1983 to 1985 and again from 1985 to 1987. The run variables were coded as follows:

- physical aggression against peers (PAAP): the run variable for PAAP, PR, was coded as
  - PR = 1 if there was no run of decreases in PAAP scores, and
  - PR = 2 if there was a run (two consecutive decreases);
- verbal aggression against adults (VAAA): the run variable for VAAA, VR, was coded as
  - VR = 1 if there was no run of decreases in VAAA scores, and
  - VR = 2 if there was a run (two consecutive decreases);
- aggressive impulses (AI): the run variable for AI, AR, was coded as
  - AR = 1 if there was no run of decreases in AI scores, and
  - AR = 2 if there was a run (two consecutive decreases).

The 2 × 2 × 2 PR × VR × AR cross-classification can be approached with two questions. First, we ask whether there are any local associations at all. This question can be answered with the base model of first-order CFA,

$$\log \hat{m} = \lambda + \lambda^{PR} + \lambda^{VR} + \lambda^{AR}$$

This base model can fail if any interaction exists. Second, we can also ask whether association patterns are specific to adolescents who differ in their patterns of change in aggressive impulses. This question can be answered using the following base model of 2-group CFA,

$$\log \hat{m} = \lambda + \lambda^{PR} + \lambda^{VR} + \lambda^{AR} + \lambda^{PR,VR}.$$

This base model can be rejected only if interactions exist that link the run variables for physical aggression against peers and verbal aggression against adults on one side with runs of aggressive impulses on the other. These are the interactions [PR, AR], [VR, AR], and [PR, VR, AR].

Table 20.23 presents results for the base model of first-order CFA, that is, the model of variable independence. Table 20.24 presents results for two-group CFA. For both CFAs, we use the normal approximation of the binomial test and protect α using the Holland–Copenhaver procedure.

The overall goodness-of-fit likelihood-ratio for the base model of variable independence is $X^2 = 13.93$. For $df = 4$, this value leads us to reject the base model ($p < 0.01$), and we anticipate that types and antitypes may emerge. However, in this example, we encounter the rare case, in which no type or antitype emerges although the base model is rejected. Inspecting the frequency distribution in Table 20.23, we notice that, for three of the four cell pairs . . 1/. . 2, cell . . 1 contains (numerically) more

TABLE 20.23 First-Order CFA of the PR × VR × AR Cross-Classification

	Configuration				
PR VR AR	m	$\hat{m}$	z	$p(z)$	Type/antitype?
111	45.00	38.078	1.3746	.084620	
112	6.00	11.817	−1.7874	.036937	
121	18.00	16.870	.2981	.382811	
122	3.00	5.235	−1.0002	.158601	
211	15.00	22.212	−1.7054	.044063	
212	13.00	6.893	2.3996	.008207	
221	9.00	9.841	−.2804	.389594	
222	5.00	3.054	1.1288	.129500	

TABLE 20.24 2-Group CFA of the PR × VR × AR Cross-Classification, with AR as the Grouping Variable

	Configuration			
PR VR AR	*m*	Statistic	*p*	Type?
111	45.00			
112	6.00	2.693	.003537	Discrimination type
121	18.00			
122	3.00	1.122	.131014	
211	15.00			
212	13.00	−3.259	.000559	Discrimination type
221	9.00			
222	5.00	−1.130	.129145	

cases than expected but cell . . 2 contains (numerically) fewer cases than expected. We, therefore, conduct the 2-group CFA and expect that discrimination types emerge. Table 20.24 displays results.

The overall goodness-of-fit likelihood-ratio for the base model of 2-group CFA is $X^2 = 13.72$. For $df = 3$, this X^2 value leads to the rejection- of the base model ($p < 0.01$), and we anticipate that discrimination types may emerge. The fit of this model is only marginally better than the fit of the model of variable independence. We conclude that the interaction between the runs of physical aggression against peers and verbal aggression against adults is weak, and makes no contribution to the explanation of the frequency distribution in Tables 20.23 and 20.24.

The results in Table 20.24 show that two-group CFA identified two discrimination types. The first is constituted by configuration pair 1 1 1/1 1 2. It suggests that those adolescents who show inconsistent increases/decreases in aggressive impulses (Pattern . . 1) are more likely than those adolescents who show consistent decreases in aggressive impulses (Pattern . . 2) to also show inconsistent patterns of decreases/increases in verbal aggression against adults and physical aggression against peers. The first group contains 45 respondents, the second group contains 6 respondents. This ratio is more extreme than the group ratio, which is 74 to 27. The corresponding log odds ratio is 1.32 (se = 0.51; $z = 2.59$; $p < 0.01$).

The second discrimination type is constituted by configuration pair 2 1 1/2 1 2. This discrimination type suggests that those adolescents who show inconsistent increases/decreases in aggressive impulses (Pattern . . 1) are more likely than those adolescents who show consistent patterns of decreases in aggressive impulses (Pattern . . 2) to show consistent patterns of decreases only in verbal aggression against adults, but not in physical aggression against peers. The first group contains 15 respondents, the second group contains 13 respondents. This ratio is less extreme than the group ratio, which is 74 to 27. The corresponding log odds ratio is −1.50 ($se = 0.48$; $z = -3.12$; $p < 0.01$).

From the perspective of conducting research with CFA, we conclude:

1. Significant discrepancies between a base model and the observed frequency distribution are a necessary but not a sufficient condition for types and antitypes to emerge. Types and antitypes require, in addition, that the discrepancies between observed and expected cell frequencies are particularly strong in a few cells instead of being distributed more evenly over all cells in a table. Evidently, the distribution of discrepancies in Table 20.23 was not extreme enough.
2. Two-group CFA is able to find discrimination types in particular when the model–data discrepancies in corresponding cells go in opposite directions. This applies even when other models of CFA do not suggest that types or antitypes exist. As we said before, in this chapter, patterns of types and antitypes depend on the base model. Different base models can result in different patterns of types and antitypes (and come with different interpretations; Mellenbergh, 1996).
3. Depending on the number of groups to be compared, the number of tests performed in multigroup CFA is only a fraction of the number performed in other CFA models. Therefore, the protected α is less extreme and, thus, less prohibitive for the detection of discrimination types.

CFA of Lags

In longitudinal research, one can distinguish between *lagged values* and *current values*. Lagged values are the values of a variable from a different period. Current values are from the last observation point in time. Lags are measured in units of time (e.g., days or years), or in experimental units (e.g., trials). In the simplest case, one variable is related to itself over time. For example, one can ask whether the number of technical problems in cars produced on a Monday are predictive of problems for cars produced the following Monday, or whether the number of beers an alcoholic individual consumes on a Friday is predictive of the number of beers consumed the following Friday.

Creating data sets for the analysis of lags is relatively straightforward. All one needs to do is copy the series of scores and paste it back in with the lag of interest, shifting it by this lag. Table 20.25 displays the resulting arrangement for lags 1, 2, and 3.

TABLE 20.25 Series with Lags 1, 2, and 3

Time	Original scores	Scores with lag 1	Scores with lag 2	Scores with lag 3
1	x_1	–	–	–
2	x_2	x_1	–	–
3	x_3	x_2	x_1	-
4	x_4	x_3	x_2	x_1
5	x_5	x_4	x_2	x_2
.	.	.	.	.
.	.	.	.	.
.	.	.	.	.
$n-1$	x_{n-1}	x_{n-2}	x_{n-3}	x_{n-4}
n	x_n	x_{n-1}	x_{n-2}	x_{n-3}

TABLE 20.26 Cross-Classification of a Series of Scores with Itself, for a Lag of *k*

	Lag k series			
Original series	$i = 1$	$i = 2$	$i = 3$	Totals
$i = 1$	m_{11}	m_{12}	m_{13}	$m_{1.}$
$i = 2$	m_{21}	m_{22}	m_{23}	$m_{2.}$
$i = 3$	m_{31}	m_{32}	m_{33}	$m_{3.}$
Totals	$m_{.1}$	$m_{.1}$	$m_{.1}$	$m_{.n} = N - k$

In Table 20.25, the subscripts of the x-scores indicate the observation point. The subscripts for the lagged series show that, with increasing lags, the number of scores that can directly be compared decreases by k, the size of the lag.

When scores are categorical in nature, lagged series can be crossed with each other and subjected to methods of categorical data analysis, for example, log-linear modeling, logit modeling, or CFA. Table 20.26 displays a sample cross-classification for a variable with $I = 3$ categories.

The frequencies in the diagonal cells in Table 20.26, which are the cells with subscripts ii, indicate the number of times a category of the original series is followed by an observation of the same category, for a lag of k. The frequencies in the off-diagonal cells, which are the cells with subscripts ij, with $i \neq j$, indicate the frequency with which an observation of Category i is followed by the observation of Category j, for a lag of k.

Extensions of the approach shown in Table 20.26 can be achieved, for example, by taking stratification variables or covariates into account (gender, age, ethnic background, etc.) or by increasing the number of lagged variables. Cross-classifications such as illustrated in Table 20.26 can be analyzed using well-known methods of categorical data analysis. It is interesting to note that rater agreement tables have the exact same form as the one given in Table 20.26. Therefore, the same methods can be used for analysis (see von Eye & Mun, 2005). These methods include the estimation of coefficients of agreement and the estimation of manifest or latent variable models that reflect hypotheses concerning the pattern of agreement or disagreement (von Eye & von Eye, 2005).

The best-known coefficient of rater agreement is Cohen's κ (kappa), which is estimated from an $I \times I$ cross-classification as the one given in Table 20.26 by

$$\kappa = \frac{N\sum_{i=j} m_{ij} - \sum_{i=j} m_{i.}m_{.j}}{m^2 - \sum_{i=j} m_{i.}m_{.j}}$$

The interpretation of κ can proceed as for all PRE (proportionate reduction in error) coefficients. Specifically, κ can be interpreted as proportion of agreement beyond chance.

Data Example

For the following data example, we use data from a study of self-identified alcoholic men again (Perrine et al., 1994). We use the data from Respondent 3053 who answered the telephone survey on 800 consecutive days. The following analyses focus on the numbers of beer (B) and liquor shots (L) consumed on a given day and on mood (M) on the following day. Mood was rated on a 10-point Likert scale with 1 indicating "terrible mood" and 10 indicating "just wonderful." Respondent 3053 tended to not use the lower end points of the mood scale. Therefore, scores 4 and below were combined into a single category of 1. In addition, this respondent used scores greater than 6 rarely. Therefore, scores 6 through 10 were coded as 3. The resulting scale thus ranged from 1 to 3 with 1 indicating average mood or below, and 3 indicating good or better mood. Similarly, the range of shots of liquor consumed was condensed to include the values of 1 = no liquor consumed and 2 = 1 or more shots of liquor consumed. This respondent almost never had more than 2 shots of liquor on any given day. The range of the numbers of beer consumed by Respondent 3053 was narrow also, but he drank beer almost every day. This beer response was, therefore recoded to have values 1, 2, and 3, with 1 = one or no beer consumed through 3 = three or more beers consumed in one day.

In the following paragraphs, we ask three questions that can be answered using the data thus created.

1. Can the number of beers consumed on consecutive days be predicted?
2. Can the number of shots of liquor consumed on consecutive days be predicted?
3. Is the mood on one day predictive of the mood on the following day?
4. Is the mood on one day predicted by the numbers of beer and shots of liquor the day before?

TABLE 20.27 Cross-Classification of Beer Consumption for a Lag of One Day (Respondent 3053)

		Beer consumption lag 1			
		1	2	3	Total
Beer consumption	1	296	24	161	481
	2	30	2	15	47
	3	156	21	95	272
	Total	482	47	271	800

TABLE 20.28 Cross-Classification of Liquor Consumption for a Lag of One Day (Respondent 3053)

		Liquor consumption lag 1		
		1	2	Total
Liquor consumption	1	531	110	641
	2	110	35	145
	Total	641	145	786

The first three questions are answered using CFA as well as methods otherwise employed in the analysis of rater agreement. Specifically, considering that the tables to be analyzed are all of the form $I \times I$, we use first-order CFA for questions 1 and 3, Cohen's κ for questions 1, 2, and 3, and the odds ratio for question 2. To answer question 4, we use CFA and log-linear modeling.

Question 1: Beer consumption over a lag of one day. The cross-classification of the ordinal beer consumption scale for Respondent 3053 is given in Table 20.27.

Table 20.27 shows an interesting marginal frequency pattern. Regardless of the number of beers the respondent consumed, the first observation day is often followed by a day during which the respondent consumed only a small number of beers or none. This becomes evident by comparing the frequencies in the first column with the frequencies in the second and third columns. This pattern implies that the number of beers consumed is not strongly related to the number of beers consumed on the following day. Accordingly, the Pearson X^2 for this 3×3 table suggests a nonsignificant association ($X^2 = 3.21$; $df = 4$; $p = 0.52$). First-order CFA of the frequency distribution in Table 20.27 (not shown here) identified no types or antitypes. This is expected from the nonsignificant X^2.

Considering that the diagonal contains the largest number of cases only in the first row/column, we also expect a small and nonsignificant κ. Indeed, the $\kappa = 0.02$ indicates that the diagonal contains only 2% more cases than expected under the assumption of independence between beer consumption on one day and the next. This value is also nonsignificant ($z = 0.66$; $p = 0.51$).

Question 2: Liquor consumption over a lag of one day. The cross-classification of the ordinal liquor consumption scale for Respondent 3053 is given in Table 20.28.

Liquor consumption of respondent 3035 is not associated with itself from one day to the next. The Pearson X^2 for this 2×2 table suggests a nonsignificant association ($X^2 = 3.83$; $df = 1$; $p = 0.05$). As for beer consumption, the diagonal contains the largest cell frequency only for one cell, here in Row 1/Column 1. Also as for Table 20.27, we therefore expect κ to be small and nonsignificant. Indeed, the $\kappa = 0.07$ indicates that the diagonal contains only 7% more cases than expected under the assumption of independence between liquor consumption on one day and the next. This value is nonsignificant ($z = 1.82$; $p = 0.07$). Similarly, the odds ratio of $\theta = 1.54$ suggests that the association of liquor consumption on consecutive days is nonsignificant ($\log \theta = 0.43$; $z = 1.95$; $p = 0.052$).

Excursus: on the dependency of types and antitypes in 2×2 tables. Interestingly, it does not make much sense to perform a first-order CFA on a 2×2 cross-classification as the one given in Table 20.28. Von Weber, Lautsch, and von Eye (2003) showed that the CFA tests in 2×2 tables are completely dependent, and therefore the result of the first CFA test fully determines the results of the remaining three tests (for a discussion of type/antitype patterns in larger tables, see Krauth, 2003). To illustrate the dependency of cell-wise CFA tests in 2×2 tables, we consider the cross-classification shown in Table 20.29.

Suppose we employ the base model of variable independence. Under this base model, $m_{1.} = \hat{m}_{1.}$, $m_{2.} = \hat{m}_{2.}$, $m_{.1} = \hat{m}_{.1}$, and $m_{.2} = \hat{m}_{.2}$. Now suppose also that CFA identifies a type for cell 1 1, that is, $m_{11} - \hat{m}_{11} > 0$. We set $m_{11} - \hat{m}_{11} =$ d. Because $m_{11} + m_{12} = m_{1.} = \hat{m}_{11} + \hat{m}_{12} = \hat{m}_{1.}$, we obtain $m_{12} - \hat{m}_{12} = -$d. Accordingly, we find that $m_{21} - \hat{m}_{21} = -d$, and $m_{22} - \hat{m}_{22} = d$.

In words, in 2×2 tables for which the expected cell frequencies are estimated using the log-linear main effect

TABLE 20.29 2×2 Cross-Classification with Observed Frequencies m_{ij} and Expected Frequencies $\hat{m}_{ij}$

	Observed and expected cell frequencies		Totals
	m_{11}	m_{12}	$m_{1.}$
	$\hat{m}_{11}$	$\hat{m}_{12}$	$\hat{m}_{1.}$
	m_{21}	m_{22}	$m_{2.}$
	$\hat{m}_{21}$	$\hat{m}_{22}$	$\hat{m}_{2.}$
Totals	$m_{.1}$	$m_{.2}$	$m_{..} = N$
	$\hat{m}_{.1}$	$\hat{m}_{.2}$	N

model, there is no space for independent CFA types and antitypes. Under the main effect model (which is also the model used for X^2 testing), there is only one degree of freedom left. Therefore, if the difference between the observed and the expected cell frequency in cell 1 1 suggests the existence of a type, cells 1 2 and 2 1 will, by necessity, constitute antitypes, and cell 2 2 will, also by necessity, constitute a second type. Accordingly, if cell 1 1 constitutes an antitype, Cells 1 2 and 2 1 constitute types, and cell 2 2 constitutes a second antitype. In one word, if a type–antitype decision is made for any cell in a 2×2 cross-classification in first-order CFA, the type–antitype decisions in the remaining three cells are completely redundant. Therefore, we did not perform a first-order CFA on Table 20.28.

Question 3: Mood over a lag of one day. The 3×3 cross-classification of the ordinal mood scale for Respondent 3053 is given in Table 20.30.

Table 20.30 also shows an interesting pattern. The marginal frequencies of mood ratings are identical on consecutive days. Pearson's X^2 for this 3×3 table suggests a significant association ($X^2 = 86.18$; $df = 4$; $p < 0.01$). Only one of the diagonal cells is the largest of its row/column. This is cell 3 3. Considering that this cell frequency is the largest of the entire table, by far, we anticipate a significant κ that is larger than those for the beer and liquor consumption data. Indeed, the $\kappa = 0.22$ indicates that the diagonal contains 22% more cases than expected under the assumption of independence between mood on one day and the next. This value is significant ($z = 5.03$; $p < 0.01$).

Considering that the value of κ, although significant, is small, and that the distribution in Table 20.30 is very uneven, we now perform a first-order CFA. The base model for this analysis is that of independence of the two mood ratings, M and $M1$. The model is

$$\log \hat{m} = \lambda + \lambda^{M} + \lambda^{M1}$$

If this model is rejected, the two mood ratings must be related to each other. Types and antitypes tell us where local associations exist. Table 20.31 displays CFA results. We use the z test and the Holland–Copenhaver procedure of α protection.

TABLE 20.30 Cross-Classification of Mood Ratings for a Lag of One Day (Respondent 3053)

		Mood lag 1			
		1	2	3	Total
Mood	1	3	8	9	20
	2	6	11	38	55
	3	11	36	663	710
	Total	20	55	710	785

TABLE 20.31 First-order CFA of Mood Ratings on Consecutive Days (Respondent 3053)

	Configuration				
M M1	m	$\hat{m}$	z	p	Type/antitype?
11	3.00	.510	3.4888	.000243	Type
12	8.00	1.401	5.5744	.000000	Type
13	9.00	18.089	−2.1371	.016297	
21	6.00	1.401	3.8849	.000051	Type
22	11.00	3.854	3.6405	.000136	Type
23	38.00	49.745	−1.6653	.047929	
31	11.00	18.089	−1.6668	.047776	
32	36.00	49.745	−1.9488	.025657	
33	663.00	642.166	.8222	.205492	

The overall goodness-of-fit likelihood-ratio for the base model of independence of the mood ratings on consecutive days is $X^2 = 54.11$. For $df = 4$, this value leads us to reject the base model ($p < 0.01$), and we anticipate types and antitypes to emerge. Interestingly, only types emerge but no antitypes.

The first type is constituted by cell 1 1. This cell contains the smallest number of ratings. Still, it constitutes a type. It indicates that, although Respondent 3053 rarely indicates average to low mood on any given day, on two consecutive days, he reports average to low mood more often than expected. The second type is constituted by cell 1 2, the third-smallest cell in the table. This type signifies that although an improvement in mood from average/low to slightly above average is rare, this occurs more often than expected. The same applies to the reverse change, from slightly above average mood down to average mood (configuration 2 1). This pattern is also rare but reported more often than expected. The fourth type, constituted by configuration 2 2, suggests that two consecutive days for which Respondent 3053 reports slightly above average mood occur more frequently than expected.

The most frequent pattern (84.5%), for Respondent 3053, is 3 3. Respondent 3053 reports that his mood is above average on any two consecutive days for 84.5% of the times. We conclude that this respondent is remarkably stable and in a good mood. The observed frequency of 663 is only minimally greater than the frequency that was expected for this pattern (642.17). Therefore, configuration 3 3 does not constitute a type.

Question 4: Is the mood on one day predicted by the numbers of beer and shots of liquor the day before? It is often assumed that alcohol consumption affects mood (First & Tasman, 2004). Here, we ask whether there

are, for Respondent 3053, short-term predictive relationships of beer and liquor consumption to mood. To answer this question, we apply three methods and compare their results. First, we employ log-linear modeling. The results from this approach tell us whether there are interactions among the variables beer consumption (B), liquor consumption (L), and mood on the following day ($M1$), in general. Second, we estimate a log-linear model. The results from this model tell us whether these interactions are compatible with the hypothesis that beer and liquor consumption affects mood on the following day. Third, we perform a first-order and a prediction CFA. The results from these approaches tell us where local associations exist, if they exist. The $B \times L \times M1$ cross-classification is given in Table 20.32.

Results from Log-Linear Modeling

To create a base model that can be used for comparison, we first estimate the model of variable independence. This is the same model that will be used for first-order CFA. The overall goodness-of-fit likelihood-ratio for the base model of independence of alcohol consumption and mood ratings on the following day is $X^2 = 33.34$. For $df = 12$, this value leads us to the reject the base model ($p < 0.01$) (and we anticipate types and antitypes to emerge; see below). We then ask whether beer and liquor consumption are related to each other to the degree that this model–data discrepancy could be explained. The corresponding model is

$$\log \hat{m} = \lambda + \lambda^B + \lambda^L + \lambda^{M1} + \lambda^{B,L}$$

TABLE 20.32 First-Order CFA of Beer and Liquor Consumption and Mood Ratings on the Following Day (Respondent 3053)

Configuration					
$B\ L\ M1$	m	$\hat{m}$	z	p	Type/antitype?
111	6.00	9.777	−1.2079	.113546	
112	26.00	26.886	−.1709	.432148	
113	338.00	349.521	−.6162	.268874	
121	3.00	2.198	.5410	.294238	
122	6.00	6.044	−.0180	.492831	
123	94.00	78.574	1.7402	.040911	
211	1.00	.971	.0289	.488457	
212	4.00	2.672	.8127	.208181	
213	26.00	34.730	−1.4814	.069247	
221	2.00	.218	3.8123	.000069	Type
222	1.00	.601	.5154	.303140	
223	13.00	7.808	1.8583	.031565	
311	7.00	5.581	.6007	.274008	
312	15.00	15.347	−.0887	.464679	
313	222.00	199.515	1.5919	.055707	
321	1.00	1.255	−.2273	.410092	
322	3.00	3.450	−.2424	.404252	
323	22.00	44.852	−3.4122	.000322	Antitype

This is also the base model for the Prediction CFA that will be performed below. The overall goodness-of-fit likelihood ratio for this model of independence of alcohol consumption and the mood ratings on the following day, which incorporates the hypothesis that the two forms of alcohol consumption are related to each other, is $X^2 = 7.71$. For $df = 10$, this value leads us to the retain the model ($p = 0.66$) (and we anticipate that no prediction types and antitypes emerge). From the signs of the estimates of the interaction parameter, we conclude that the relationship between beer and liquor consumption is, for respondent 3053, negative, indicating that the more beer the respondent consumes, the less liquor he consumes, and vice versa.

From this result, we conclude that, over a lag of one day, there is no relationship of beer and liquor consumption to mood on the following day. Based on this conclusion, we also expect that the log-linear model will suggest no predictive relationship. The overall goodness-of-fit likelihood-ratio for the log-linear model is $X^2 = 28.19$. For $df = 6$, this value leads us to reject the null hypothesis of no relationship between predictors (beer or liquor consumption) and criterion (mood on the following day) ($p < 0.01$). None of the parameters of the effects that link the predictors and the criterion is significant. The Nagelkerke R^2 – equivalent is 0.01. This also indicates that close to nothing of the variability in the $B \times L \times M1$ cross-classification is explained by the model.

A log-linear model that includes the interactions between beer and mood on the following day and liquor and mood on the following day, that is, the model

$$\log \hat{m} = \lambda + \lambda^B + \lambda^L + \lambda^{M1} + \lambda^{B,M1} + \lambda^{L,M1}$$

also fails to describe the data well. The $B \times L$ interaction is needed. Evidently, the two-way interaction between beer and liquor consumption is not sufficient to explain the variability of the data that exists in Table 20.32 beyond the main effects.

Results from CFA

The results from log-linear modeling make us expect that first-order CFA will identify types and antitypes. However, the same results make us also expect that these types and antitypes only indicate where liquor and beer consumption are locally associated. As soon as the interaction between beer and liquor consumption is included in the base model, all types and antitypes can be expected to disappear. This would, again, suggest that there is no relationship between alcohol consumption and mood on the following day.

Table 20.32 displays the results from first-order CFA (the base model for this CFA was given previously). For both this run and the following Prediction CFA, we use the z-test and the Holland–Copenhaver procedure of α protection.

The overall goodness-of-fit for the base model of first-order CFA is the same as for the log-linear model of variable independence discussed previously. The base model is rejected. However, the results in Table 20.32 are, from a CFA perspective, not overly rich. We find only one type, for a cell that contains only 2 ratings, and one antitype. When the corresponding expected frequency is small, we should interpret the findings of types and antitypes quite cautiously. Specifically, as soon as an expected cell frequency is below 0.5, we do not interpret a type. Then what is left in this table is the sole antitype that is constituted by configuration 3 2 3. It indicates that, significantly less often than expected, Respondent 3053 consumed above average quantities of beer together with liquor and was in a positive mood on the next day.

From the log-linear modeling results, we already know that Respondent 3053 tended to drink predominantly either beer or liquor on drinking occasions but not both, and that his drinking had no effect on his mood on the next day. Therefore, we expect the type and the antitype to disappear when the beer × liquor interaction is taken into account. This is indeed the case (table of results not shown here).

To provide closure on the substantive part of this example, we now ask whether alcohol consumption has effects on the mood as rated for the same day. We find almost the same results. The log-linear model of variable independence is rejected ($LR - X^2 = 30.25$; $df = 12$; $p < 0.01$), and the same antitype emerges. The model that takes the beer × liquor consumption into account describes the data very well ($LR - X^2 = 3.77$; $df = 10$; $p = 0.96$), and no type or antitype emerges. We conclude that, for this respondent, there is no short-term relationship between alcohol consumption and mood.

CFA of Cross-Lagged Designs

Cross-lagged designs allow researchers to test hypotheses about the temporal relationships between two or more repeatedly observed variables. Consider the two variables, X and Y, each observed twice, that is, the four realizations $X1$, $X2$, $Y1$, and $Y2$. Figure 20.3 displays the relationships that are usually estimated.

All possible two-way relationships can be arranged in the form of a correlation matrix, as in Table 20.33.

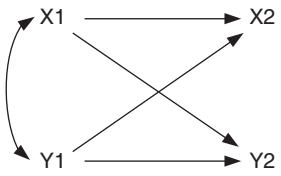


Figure 20.3 Cross-lagged relationship.

TABLE 20.33 Correlation Matrix for *X*1, *X2*, *Y*1, and *Y*2

	Variables		
Variables	*X*1	*X*2	*Y*1
*X*2	$r_{X2\,X1}$		
*Y*1	$r_{Y1\,X1}$	$r_{Y1\,X2}$	
*Y*2	$r_{Y2\,X1}$	$r_{Y2\,X2}$	$r_{Y2\,Y1}$

Five of the six relationships listed in Table 20.33 are included in Figure 20.3. The only one missing is the one between $X2$ and $Y2$. This relationship was omitted to illustrate that cross-lagged models do not necessarily involve all possible two-way relationships.

In standard, aggregate-level, variable-oriented data analysis, the paths in Figure 20.3 are estimated as regression coefficients. The paths between $X1$ and $X2$ as well as between $Y1$ and $Y2$ are called *autoregressions*, and the paths between $X1$ and $Y2$ as well as between $Y1$ and $X2$ are *cross-lagged regressions*. The relationships between $X1$ and $Y1$ as well as between $X2$ and $Y2$ are typically estimated as correlations (they could be regressions also).

These paths and correlations allow one to test hypotheses concerning

1. The *contemporaneous* associations between $X1$ and $Y1$ as well as between $X2$ and $Y2$; and
2. The *lagged* associations between $X1$ and $Y2$ as well as between $Y1$ and $X2$.

For both manifest and latent variable models, structural modeling is most suitable for estimation. Occasionally, regression analysis is still applied for manifest variable models.

One characteristic of such models that is of importance here is that all relationships are expressed at the level of aggregated raw data. That is, all relationships are expressed within a framework of variable-oriented research. In the present context, we take a person-oriented perspective for the analysis of categorical variables. That is, we do not assume that all categories of all variables carry a relationship. Instead, using the concept of *local associations* (Havránek & Lienert, 1984), we assume that, in many

cases, only some of the categories of a variable carry an association. Under this assumption, CFA is the method of choice. Therefore, we present here, for the first time, a CFA approach to cross-lagged analysis. This approach is an extension of the CFA approach to lagged relationships that was discussed and illustrated in the last section (von Eye et al., 2010).

To develop the base model for cross-lagged CFA of categorical data, we use the general definition of CFA base models. This definition states that a CFA base model contains all relationships that are *not* of interest to the researchers. If this model is rejected and types and antitypes emerge, they necessarily reflect the cross-lagged relationship under study.

To illustrate, consider the model in Figure 20.3. This model contains five of the six possible bivariate relationships. These must, therefore, not be included in the base model. In contrast, the remaining two-way relationship (the one between $X2$ and $Y2$) must be included. None of the four possible three-way interactions and the four-way interaction are part of the tested hypotheses. Therefore, they are included in the base model. In log-linear modeling notation, the nonhierarchical base model for cross-lagged CFA is

$$\begin{aligned}\log \hat{m} = \lambda &+ \lambda^{X1} + \lambda^{X2} + \lambda^{Y1} + \lambda^{Y2} + \lambda^{X2,Y2} \\ &+ \lambda^{X1,X2,Y1} + \lambda^{X1,X2,Y2} + \lambda^{X1,Y1,Y2} + \lambda^{X2,Y1,Y2} \\ &+ \lambda^{X1,X2,Y1,Y2}\end{aligned}$$

As with all other CFA base models, if any of the interactions not included in the model exist, the model is rejected and resulting types and antitypes indicate the configurations that carry the relationship. Also, as was illustrated before, the rejection of a model is a necessary condition but no guarantee that types and antitypes emerge.

It is interesting to note that the rejection of the base model of cross-lagged CFA gives no indication concerning which of the hypothesized relationships exist. Therefore, we recommend considering one of two strategies. The first involves estimating a categorical variable path model, in particular when there are more than two observation points. This can be achieved with Mplus (Muthén & Muthén, 2010) or with a specialized program for categorical data analysis such as Lem (Vermunt, 1997). The methods of functional CFA can also be used (von Eye & Mair, 2008a, 2008b, 2008c), which allow one to identify the configurations that carry the two-way interactions of the cross-lagged model.

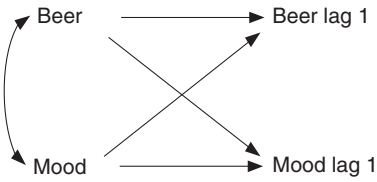


Figure 20.4 Cross-lagged relationship between beer consumption and mood.

Data Example

In the following example, we continue the analysis of the one-lag data of Respondent 3053 from the study of self-identified alcoholic men (Perrine et al., 1994). We ask whether, from one day to the next, beer consumption and mood are related to each other so that it can be described by a cross-lagged model such as the one in Figure 20.3. In this example, the model would be as shown in Figure 20.4.

We approach this model first from the variable-oriented perspective. We estimate a log-linear model that includes all the variable relations indicated in the graph. That is, we estimate the model

$$\begin{aligned}\log \hat{m} = \lambda &+ \lambda^{B} + \lambda^{B1} + \lambda^{M} + \lambda^{M1} \\ &+ \lambda^{B,M} + \lambda^{B,B1} + \lambda^{B,M1} + \lambda^{M,B1} + \lambda^{M,M1}.\end{aligned}$$

In this section, we use the same abbreviations as in the last. That is, B = beer consumption, $B1$ = beer consumption on the following day, M = self-rated mood, $M1$ = self-rated mood on the following day. This is not the CFA base model. Therefore, we hope that this model describes the data well.

If this is the case, we perform the CFA of the cross-lagged model described previously, which includes the terms that are not of interest. The base model for this CFA is

$$\begin{aligned}\log \hat{m} = \lambda &+ \lambda^{B} + \lambda^{B1} + \lambda^{M} + \lambda^{M1} + \lambda^{B1,M1} \\ &+ \lambda^{B,B1,M} + \lambda^{B,B1,M1} + \lambda^{B,M,M1} + \lambda^{B1,M,M1} \\ &+ \lambda^{B,B1,M,M1}\end{aligned}$$

If this cross-lagged base model is rejected, resulting types and antitypes indicate the interactions that are not specified in this model, which is nonhierarchical. We will know which of the interactions exist because, in this example, we estimate the log-linear model before we perform CFA. The types and antitypes will tell us where, in the cross-classification, these effects become manifest.

In the example in the last section, we used the three-category variables B and $B1$, and M and $M1$. Crossed,

these variables span a 3 × 3 × 3 × 3 cross-classification with 81 cells. For about 800 observations, this table is not too large. However, due to the very uneven frequency distribution, many cells were empty. Sparseness was so extreme that the programs we use were unable to estimate the models. Therefore, we further combined categories of the variables. Specifically, we now use for B and $B1$ the categories 1 = two or fewer beers consumed and 2 = three or more beers consumed in one day. For M and $M1$, we use the categories 1 = good mood or below, and 2 indicating better mood.

For the log-linear model, we obtain an overall goodness-of-fit likelihood-ratio of $X^2 = 7.11$. For $df = 6$, this value leads us to retain the model ($p = 0.31$), and we conclude that the higher order interactions are not needed to explain the frequency distribution shown in Table 20.34. We ask which of the five two-way interactions are significant. Table 20.35 displays the parameter estimates.

Table 20.34 shows an interesting pattern of results. Although the model, overall, describes the data well, the hypothesis that the relationships among the variables that span the cross-classification in Table 20.35 can be described by a cross-lagged model must be rejected. Of the five bivariate relationships included in this model, only one is significant. It is the one between mood on a given day and mood on the following day. We conclude that, for Respondent 3053, mood is predictive of itself over a lag of one day. In contrast, the quantities of beer consumed are hard to predict, even from one day to the next, and there seems to be no relationship between mood and beer consumption. This applies both to the contemporaneous and the lagged beer–mood and mood–beer relationships. Drinking does not seem to affect the mood of this respondent, and vice versa.

TABLE 20.34 Cross-Lagged CFA of the $B \times M \times B1 \times M1$ Cross-Classification

Configuration					
*B M B*1 *M*1	*m*	$\hat{m}$	*z*	*p*	Type/antitype?
1111	14.00	4.690	4.299	<α*	Type
1112	16.00	19.927	−.673	.31809	
1121	7.00	1.348	4.867	<α*	Type
1122	11.00	17.586	−1.570	.116322	
1211	21.00	25.154	−0.828	.283164	
1212	296.00	292.911	0.181	.392460	
1221	7.00	12.814	−1.624	.106711	
1222	148.00	146.571	0.118	.396174	
2111	3.00	1.571	1.140	.208307	
2112	15.00	20.814	−1.274	.177199	
2121	4.00	0.911	3.238	.002109	Type
2122	5.00	9.154	−1.373	.155438	
2211	12.00	18.586	−1.528	.124141	
2212	145.00	139.348	0.479	.355703	
2221	7.00	9.927	−0.929	.259121	
2222	79.00	69.690	1.115	.214263	

TABLE 20.35 Parameter Estimates for Log-Linear Cross-Lagged Model

Effect	Lambda	Std err	*z*	Exp (lambda)	Wald	*df*	*p*
Main	2.9035			18.2371			
B	−0.3041	0.0746	4.078	0.7378	16.63	1	0.000
M	0.7789	0.0753	−10.344	2.1791	107.00	1	0.000
B1	−0.3067	0.0643	4.767	0.7358	22.73	1	0.000
M1	0.7933	0.0734	−10.803	2.2107	116.71	1	0.000
Two–way interactions							
B M	−0.0220	0.0666	−0.330	0.9783	0.11	1	0.742
B B1	0.0211	0.0395	0.535	1.0213	0.29	1	0.593
M B1	−0.0248	0.0633	−0.392	0.9755	0.15	1	0.695
B M1	0.0010	0.0671	0.014	1.0010	0.00	1	0.989
M M1	0.5341	0.0706	7.562	1.7059	57.18	1	0.000

To identify the details of the relationship between mood on two consecutive days, we perform a CFA on the 2 × 2 × 2 × 2 B × M × B1 × M1 cross-classification. Considering that we already know that the M × M1 interaction exists, there is no need to create a reference model and estimate a first-order CFA. Instead, we estimate the nonhierarchical base model for cross-lagged CFA shown previously right away. If types and antitypes emerge, they will indicate the location of the association between the mood ratings on consecutive days, in this table. Table 20.35 displays the results of cross-lagged CFA. We use the z test and the Holland–Copenhaver procedure for the protection of α.

Table 20.35 suggests that, in the B × M × B1 × M1 cross-classification, three types exist and no antitype. The first type is constituted by configuration 1 1 1 1. It indicates that, more often than expected for the cross-lagged base model, average mood on one day is predictive of average mood on the following day. Please notice that we interpret the four types with reference to mood only. Neither of the two beer consumption variables is related to any of the other variables based on the log-linear modeling results. Therefore, we focus on mood. The second type is constituted by configuration 1 1 2 1. This type has the same interpretation as the first. The variable that changes score is B1. This variable is not taken into account when this type is interpreted. This applies accordingly for the third type. It is constituted by configuration 2 1 2 1. Again, it suggests that average mood is predictive from one day to the next. In all, we conclude that, for Respondent 3053, average or worse mood on a given day tends to carry over to the following day. The amount of beer consumed is not predictive of mood. Note that we cannot say that positive mood carries over as well. This result shows that it is not mood in

general that is autocorrelated over time, but it is average or negative mood that lingers over time, for this participant.

This is not the modal CFA result but, as can be seen in the examples given in this chapter, the largest cells do not always constitute types, and the smallest do not always constitute antitypes. This is the case only in zero-order CFA (Lienert & von Eye, 1989; von Eye, 2002). The largest configurations in Table 20.35 are 1 2 1 2 (small quantities of beer consumed in a good mood on both consecutive days), 1 2 2 2 (increasing quantities of beer consumed in a good mood on two consecutive days), and 2 2 1 2 (decreasing quantities of beer consumed in a good mood on two consecutive days). These configurations again illustrate that, for Respondent 3053, the quantity of beer that he consumes seems to be unrelated to his typically elevated good mood.

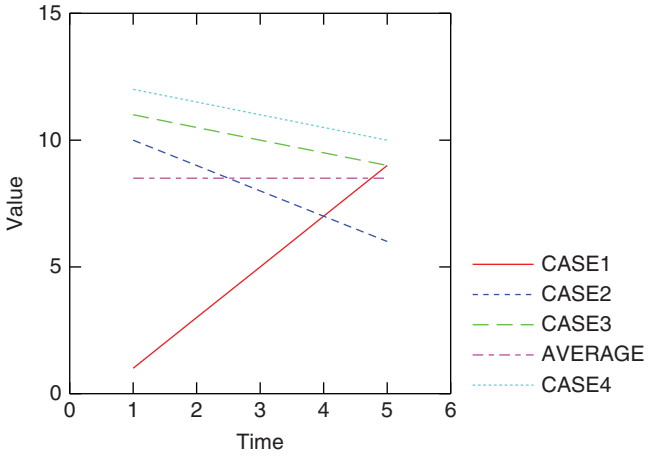


Figure 20.5 Individual and averaged developmental trajectories. See footnote 1.

Comparing Individuals' Trajectories

Comparing individuals' trajectories or, in different words, assessing interindividual differences in intraindividual change lies in the heart of person-oriented and idiographic research. Therefore, it is an important domain of data analysis with CFA. One of the main reasons for the emphasis on interindividual differences in intraindividual change is that aggregate-level descriptions of trajectories come with the high risk of missing out on developmental patterns that do not follow the overall trend. It has been repeatedly shown in the developmental literature (e.g., Baltes, Reese, & Nesselroade, 1977) and person-oriented research (von Eye & Bergman, 2003; von Eye et al., 2015) that aggregate-level estimation of parameters of developmental trajectories can miss the development of individuals completely. Here, we present two additional examples. The first uses artificial data. The second uses real data.

For the first example, consider four individuals diagnosed with obsessive-compulsive personality disorder. The therapist records the number of times they wash their hands. Whereas three of the individuals improve over the course of the therapy, one individual reports worsening symptoms. This is depicted in Figure 20.5.

Figure 20.5 shows that Cases 1, 2, and 3 improve but Case 4 gets worse. The figure also shows that the four patients, on average, do not change their behavior at all. So, based on the average trajectory, the therapy can be considered ineffective. Based on the individual trajectories, the therapy can be concluded to have the desired effects in three of the four cases.

In the real data-example, we compare the autocorrelograms of two cases from the Perrine et al. (1994) study of self-identified alcoholic men. Autocorrelograms depict the correlations among series of scores with increasing lags. The correlation for a lag of 1 is the correlation between the scores from any two consecutive days. The correlation between scores for a lag of 49 is the correlation between series of scores that are seven weeks apart. In the study of self-identified alcoholics, some of the respondents provided the researchers with responses for about 800 consecutive days. Therefore, we can compare the autocorrelograms of two respondents for longer lags, here up to $k = 50$. We use the beer consumption data from Respondents 3050 and 3053. All participants of this study started on a Sunday. Therefore, the autocorrelation patterns for these two respondents are directly comparable.

We use raw data. Specifically, the correlations are based on scales that range from 0 (no beer consumed) to 10 (10 or more beers consumed on one day). Figure 20.6 displays the autocorrelogram of Respondent 3050 in the first panel, and the autocorrelogram for Respondent 3053 in the second panel.

Not surprisingly, the two respondents differ in their autocorrelograms. Respondent 3050 shows a distinct weekday-related pattern with strong (about $r = 0.50$) positive correlations for Sundays. These correlations are significant even over a span of 7 weeks. The correlations for the days from Monday through Saturday are weak and negative. The rank order of the correlations is, over the lags, almost invariant, with the strongest negative correlations found for Wednesdays and the weakest for Saturdays.

The autocorrelation pattern for Respondent 3053 is also consistent over the lags, but the correlations decrease to the extent that, toward the longer lags, there is no correlation. The pattern is also weekday-related, with positive

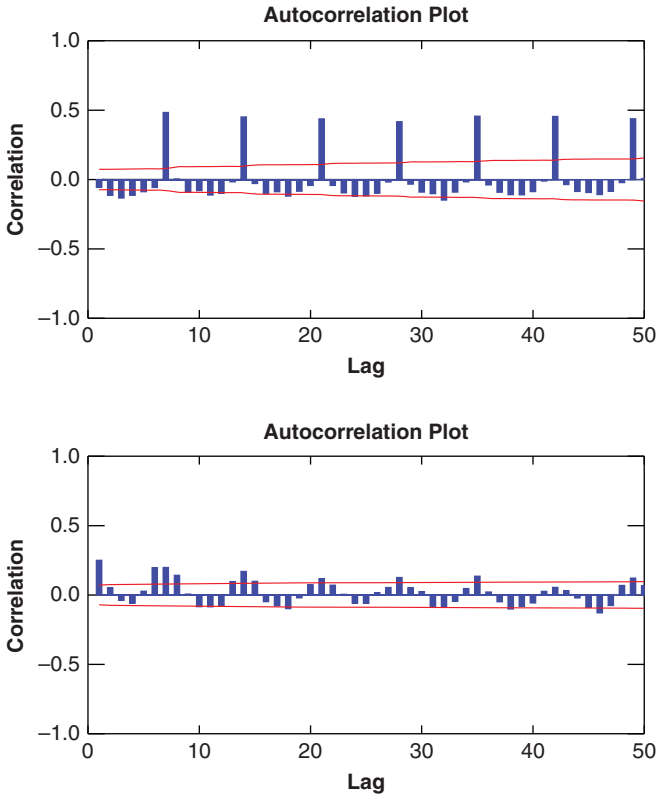


Figure 20.6 Autocorrelograms for lags between 1 and 50 for respondent 3050 (Panel 1) and 3053 (Panel 2). See footnote 1.

correlations for Saturdays, Sundays, and Mondays, and negative correlations for the other days. As for Respondent 3050, the positive correlations are stronger than the negative correlations.

In sum, the autocorrelation patterns of Respondents 3050 and 3053 show similarities in that they are weekday-related, but they also show dissimilarities in the magnitude of the correlations and the days for which positive and negative correlations are measured. In Figure 20.7, we display the autocorrelation of the aggregate of the quantities of beer that the two respondents have consumed over a span of about 800 consecutive days.

Clearly, the autocorrelation pattern for the aggregate fails to represent either individual autocorrelogram properly. Most prominent are three differences. First, it shows significant positive correlations for five of the seven days of the week. For Respondent 3050, there were six, and for Respondent 3053, there were four negative correlations for the days of the week. Second, and in correspondence with the first difference, the autocorrelogram for the aggregate shows practically no negative correlations. In contrast, for Respondent 3050, the negative correlations cease to be significant only after a lag of five weeks. For Respondent

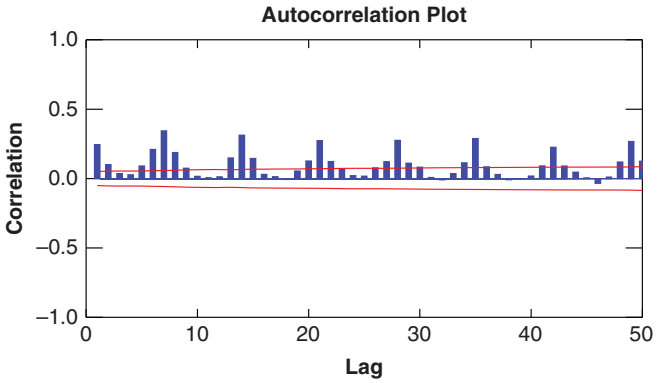


Figure 20.7 Autocorrelogram for the aggregate of respondents 3050 and 3053. See footnote 1.

3053, the negative correlations are not as strong, but there is one significant and negative correlation each for the Wednesday of the lags of six and seven weeks. Third, the autocorrelogram overestimates the magnitude of the correlations for Respondent 3053 and underestimates the correlations for Respondent 3050. This is not a surprise for aggregate statements. However, in this example, the aggregate suggests significant positive correlations for both respondents when they are nonsignificant or negative.

In the following paragraphs, we study interindividual differences in intraindividual development between Respondents 3050 and 3053 (von Eye & Mun, 2012). We take both a variable-oriented and a person-oriented perspective. For the former, we estimated a log-linear model. From the person-oriented perspective, we use CFA to answer the question concerning differences. To be able to study differences in developmental trajectories, we need a cross-classification with the following characteristics:

1. It must allow researchers to describe change at the level of the individual. Cross-classifications of the kind used for the analysis of lags, for cross-lagged analysis, and turnover tables for individuals are suitable. Based on each of these, statements are possible that relate behavior at one or more earlier points in time to behavior at one or more later points in time. These statements describe trajectories or, in more general terms, developmental change.
2. In the simplest case, we have lagged information for one variable, for two individuals. Let the variable that indicates the respondents be labeled by ID, the original observed series be labeled by O, and the lagged series by K. Then, the reference model of first-order CFA is

$$\log \hat{m} = \lambda + \lambda^{ID} + \lambda^{O} + \lambda^{K}$$

3. Cross-classifications must be available for at least two individuals. Here, it is important that cross-classifications are created that make respondents comparable. An example would be to create lagged data for series that (1) start at the same moment (2) are equal in length, and (3) have the same lag.

If the model of first-order CFA is rejected, types and antitypes suggest any kind of local associations between the variables *ID*, *O*, and *K*. More specific and pertinent to longitudinal research are the following two models. The first allows one to answer the question whether the respondents labeled by ID differ in their lag structure. The base model for this question includes the association between *O* and *K*, but proposes independence of *ID*, *O*, and *K*, or

$$\log \hat{m} = \lambda + \lambda^{ID} + \lambda^{O} + \lambda^{K} + \lambda^{O,K}$$

This model will be rejected if any of the interactions [*ID*, *O*], [*ID*, *K*], and [*ID*, *O*, *K*] exist. Types and antitypes from this model suggest that the respondents differ either in their development over time (two-way interactions of *ID* with *O* or *K*) or in the development of their patterns of constancy and change, over a lag of *K* (three-way interaction $ID \times O \times K$). In its structure, this base model is identical to the base model of two-group CFA or prediction CFA, both of which have been discussed already in this chapter (von Eye, 2002).

The second developmental model also takes into account that the two respondents may differ in their temporal pattern of behavior both in the original and the lagged string. It is the model

$$\log \hat{m} = \lambda + \lambda^{ID} + \lambda^{O} + \lambda^{K} + \lambda^{O,K} + \lambda^{ID,O} + \lambda^{ID,K}$$

If this model is rejected, only the interaction [*ID*, *O*, *K*] can exist. Types and antitypes from this base model suggest that the two respondents differ in their pattern of constancy and change over a lag of size *K*.

Data Example

In the following illustration, we resume the analysis of the alcohol consumption data of Respondents 3050 and 3053. Specifically, we examine the differences between the developmental trajectories of beer consumption, and the effects that beer consumption has on mood on the day that follows the first beer consumption. We use a lag of one day. Thus, we have four variables that span the cross-classification: (1) *B* and *B*1, the original beer consumption variable and the same variable, with a lag of one day; these variables are coded as before, with 1 indicating 0–1 beer consumed, 2 indicating 2 beers consumed, and 3 indicating 3 or more beers consumed on one day; (2) *M1*, mood on the day after the first beer consumption; this variable is coded into the two categories 1 = low or average mood and 2 = good mood or better; (3) *ID* with the categories 3050 and 3053. Together, these four variables span a $3 \times 3 \times 2 \times 2$ $B \times B1 \times M1 \times ID$ cross-classification with 36 cells. We approach this table with the following CFA base models. First, we estimate the base model of variable independence, that is, the base model of first-order CFA,

$$\log \hat{m} = \lambda + \lambda^{B} + \lambda^{B1} + \lambda^{M1} + \lambda^{ID}$$

If this model is rejected, we know that local associations exist, and we can estimate more specific developmental models. Of the many models that could be considered, we consider the hierarchical two-group model

$$\begin{aligned}\log \hat{m} = \lambda &+ \lambda^{B} + \lambda^{B1} + \lambda^{M1} + \lambda^{ID} \\ &+ \lambda^{B,B1} + \lambda^{B,M1} + \lambda^{B1,M1} \\ &+ \lambda^{B,B1,M1}\end{aligned}$$

This model is saturated in the variables *B*, *B*1, and *M*1. Therefore, types and antitypes cannot emerge because these variables might be associated with each other. This model can be rejected only if there is an interaction between *ID* and one or more of the other variables, that is, if the two respondents differ in their developmental trajectories. For both CFA runs, we use the normal approximation of the binomial test along with the Holland–Copenhaver procedure for the protection of α. In addition, to prevent convergence problems from resulting in estimates that cannot be trusted, we invoke the delta option with $\Delta = 0.01$ in both runs. Invoking the delta option has the effect that a small constant, the delta value, is added to each cell. To prevent this option from artificially increasing the sample, this constant is usually chosen to be small. In the present example, invoking $\Delta = 0.01$ results in an increase in the number of observations of $36 \cdot 0.01 = 0.36$, that is, not even half an observation. Table 20.36 displays the results of first-order CFA, and Table 20.37 shows the results of two-group CFA.

The overall goodness-of-fit likelihood-ratio X^2 of 401.90 leads us to reject the base model of first-order CFA ($df = 29$; $p < 0.01$), and we anticipate that types and antitypes emerge. Table 20.36 shows that four types and five antitypes emerge. From both, the large and significant likelihood-ratio X^2 and the types and antitypes, we conclude that local associations do exist. However, at this

TABLE 20.36 First-order CFA of the Cross-Classification of *B*, *B*1, *M*1, and *ID*

Configuration					
*B B*1 *M*1 *ID*	*m*	$\hat{m}$	*z*	*p*	Type/antitype?
1111	5.01	20.631	−3.4627	.000267	Antitype
1112	27.01	21.920	1.0951	.136741	
1121	518.01	361.289	9.4455	.000000	Type
1122	255.01	383.869	−7.6090	.000000	Antitype
1211	.01	1.661	−1.2818	.099965	
1212	2.01	1.765	.1847	.426752	
1221	41.01	29.088	2.2320	.012809	
1222	22.01	30.906	−1.6168	.052964	
1311	.01	6.361	−2.5234	.005811	
1312	12.01	6.759	2.0245	.021458	
1321	65.01	111.395	−4.5655	.000002	Antitype
1322	147.01	118.357	2.7429	.003045	
2111	1.01	1.678	−.5162	.302860	
2112	6.01	1.783	3.1670	.000770	Type
2121	39.01	29.392	1.7915	.036604	
2122	24.01	31.229	−1.3053	.095899	
2211	.01	.135	−.3404	.366774	
2212	.01	.144	−.3525	.362218	
2221	.01	2.366	−1.5330	.062637	
2222	2.01	2.514	−.3183	.375126	
2311	.01	.517	−.7056	.240224	
2312	2.01	.550	1.9696	.024444	
2321	2.01	9.062	−2.3497	.009395	
2322	13.01	9.629	1.0932	.137160	
3111	1.01	6.317	−2.1160	.017171	
3112	13.01	6.712	2.4362	.007422	
3121	62.01	110.632	−4.8009	.000001	Antitype
3122	142.01	117.547	2.3491	.009408	
3211	.01	.509	−.6993	.242188	
3212	2.01	.540	1.9994	.022781	
3221	1.01	8.907	−2.6539	.003979	
3222	18.01	9.464	2.7867	.002663	
3311	.01	1.948	−1.3894	.082360	
3312	11.01	2.070	6.2189	.000000	Type
3321	1.01	34.111	−5.7323	.000000	Antitype
3322	84.01	36.243	8.0310	.000000	Type

TABLE 20.37 Two-Group CFA of the Cross-Classification of *B*, *B*1, *M*1, and *ID* With *ID* as the Grouping Variable

Configuration				
*B B*1 *M*1 *ID*	*m*	Statistic	*p*	Type?
1111	5.01			
1112	27.01	−3.758	.000086	Discrimination Type
1121	518.01			
1122	255.01	14.710	.000000	Discrimination Type
1211	.01			
1212	2.01	−1.366	.086021	
1221	41.01			
1222	22.01	2.692	.003556	Discrimination Type
1311	.01			
1312	12.01	−3.371	.000374	Discrimination Type
1321	65.01			
1322	147.01	−5.598	.000000	Discrimination Type
2111	1.01			
2112	6.01	−1.812	.035002	
2121	39.01			
2122	24.01	2.177	.014753	
2211	.01			
2212	.01	.004	.498290	
2221	.01			
2222	2.01	−1.366	.086021	
2311	.01			
2312	2.01	−1.366	.086021	
2321	2.01			
2322	13.01	−2.736	.003112	Discrimination Type
3111	1.01			
3112	13.01	−3.107	.000944	Discrimination Type
3121	62.01			
3122	142.01	−5.557	.000000	Discrimination Type
3211	.01			
3212	2.01	−1.366	.086021	
3221	1.01			
3222	18.01	−3.791	.000075	Discrimination Type
3311	.01			
3312	11.01	−3.226	.000627	Discrimination Type
3321	1.01			
3322	84.01	−8.981	.000000	Discrimination Type

point, we do not interpret the types and antitypes. They do not necessarily reflect between-respondent differences in the effects that beer consumption on one day has on beer consumption and mood on the next day. Therefore, we proceed immediately to the two-group model specified previously.

The overall goodness-of-fit likelihood-ratio X^2 of 368.756 leads us to reject the base model of two-group CFA ($df = 17$; $p < 0.01$). This model is significantly better than the base model of first-order CFA ($\Delta X^2 = 33.14$; $\Delta df = 12$; $p = 0.001$). However, there are significant model–data discrepancies, and we anticipate that types and antitypes emerge. Table 20.37 shows that 11 discrimination types emerge. To illustrate interpretation, we discuss the three most extreme discrimination types. These are the ones with the largest test statistics.

The most extreme discrimination type is constituted by configuration 1 1 2 .. Respondent 3050 reported 518 times, and Respondent 3053 reported 255 times that they consumed 0–1 beer on two consecutive days and were in good mood or better, on the second day. Although these numbers are large for both respondents, Respondent 3050 reported this pattern significantly more often than Respondent 3053.

The second-largest discrepancy was found for configuration 3 3 2 ., that is for three or more beers consumed on two consecutive days in tandem with good mood or better on the second day. Respondent 3050 reported this pattern once. In contrast, Respondent 3053 reported it 84 times.

The third most extreme pattern is 1 3 2 .. This pattern was found 65 times for Respondent 3050 and 147 times for Respondent 3053. The pattern describes an increase from

consuming 0–1 beer on one day to 3 beers or more on the following day, along with good mood or better on the second day.

Readers are invited to interpret the other discrimination types. Each of the discrimination types describes constancy or change in beer drinking from one day to the next, that go hand in hand with one of two mood levels on the second day. In all, two-group CFA shows that the two respondents differ greatly and in many configurations in constancy and change in beer consumption and mood.

Taking the variable-oriented perspective, we ask whether the association pattern of the four variables, B, $B1$, $M1$, and ID can be explained by a log-linear model. A model that describes the $B \times B1 \times M1 \times ID$ cross-classification parsimoniously and well includes all main effects and the three two-way interactions [B, ID], [$B1$, ID], and [$M1$, ID], that is, the model

$$\log \hat{m} = \lambda + \lambda^{B} + \lambda^{M1} + \lambda^{M1} + \lambda^{B,ID} + \lambda^{B1,ID} + \lambda^{M1,ID}$$

The likelihood-ratio X^2 for this model is 26.30 (df = 24; $p = 0.49$). This model suggests that the two respondents differ in all three discrimination variables, B, $B1$, and $M1$. It also suggests that there is no interaction among the discrimination variables. Based on our earlier analyses of the beer drinking data of Respondents 3050 and 3053, this result is not surprising. We had already found that, for these two individuals, mood seems to be unrelated to beer drinking. Beer drinking on one day is associated with beer drinking on the following day, but this relationship is not sufficient for an explanation of the frequency distribution of the $B \times B1 \times M1 \times ID$ cross-classification.

Extensions and Other Base Models

Depending on the aims of research, more base models are conceivable. For example, the model that includes only the two-way interactions of discriminating variables with the grouping variables, can be considered. This model would allow one to determine whether respondents differ in the main effects of the discriminating variables as well as in their interactions. This way, a model can be specified that includes person-specific marginal distributions and developmental trajectories. This model is most useful if researchers wish to determine the exact differences in developmental trajectories between respondents.

Another class of models of interest takes situational elements into account. Examples of such elements include therapy sessions, life events, the location of events, or characteristics of the people involved. A model that considers one or more of these variables can lead to *conditional types and antitypes*, that is, types and antitypes that relate developmental changes to events.

A third class of models involves estimating CFA separately for each respondent or groups of respondents. With these models, respondent-specific types and antitypes are identified. With models such as the two-group model illustrated in Table 20.37, differences between respondents' configurations can be studied. When respondents differ in a configuration, it is not implied that this configuration also constitutes a type or an antitype for either of the comparison individuals.

A fourth class of models would take covariates into account. These covariates can be once-observed variables such as gender or number of years of formal school training. However, they could also be repeatedly observed covariates such as topic covered in individual therapy sessions. The hypotheses that are asked in the context of a cross-lagged model can then be made conditional on categories of covariates.

Predicting Events

Prediction CFA (P-CFA) relates configurations on the predictor side to configurations on the outcome side. In this section, we discuss models of P-CFA that use specific outcomes. For example, one can ask whether configurations exist that make therapy success particularly likely, or whether sequences of events or trajectories exist that allow one to predict that an adolescent engages in using illicit drugs. In other contexts, one can ask whether patterns of characteristics allow one to predict which an applicant is tended a job offer, or that a student gets admitted to graduate school. In each of these examples, a particular event, such as the end point of a developmental process, is targeted. Alternatively, one can ask whether a particular starting pattern allows one to predict a particular trajectory. In this case, a series of events is predicted instead of a point in development. In the next section, we discuss CFA base models for the prediction of end points. This is followed by a discussion of CFA base models for the prediction of trajectories.

PREDICTING END POINTS OF DEVELOPMENT

As is the case throughout the entire chapter, we deal with categorical outcomes. Frequently analyzed are dichotomous outcomes such as accepted versus rejected marriage proposals or successful versus unsuccessful job applications. However, outcomes can also be multicategorical,

for example, when profiles of symptoms are used to come to a conclusion about a psychiatric diagnosis, or when a child's attachment is characterized as secure, anxious-ambivalent, anxious-avoidant, or disorganized.

To derive the base model for the configural prediction of an end point, let the descriptors of the end point be labeled by $E_1, E_2, \ldots, E_m$, and the predictors, which can be preceding or concurrent events, by $P_1, P_2, \ldots, P_m$. Then, the P-CFA base model that allows one to determine which category of the end point is more (or less) likely to occur than chance, given a particular predictor profile, includes the following terms:

1. Main effects of all variables;
2. All possible interactions among the predictor variables; and
3. All possible interactions among the outcome variables (if the end point is a profile instead of just a category); and

The corresponding log-linear base model is, thus, saturated on the predictor side as well as on the outcome side. In addition, it proposes independence of predictors and outcome variables. In the present context, we focus on outcomes that can be represented by just one variable. Multivariate outcomes can be treated in an analogous fashion. The log-linear base model for configural prediction of an outcome is

$$\log \hat{m} = \lambda + \sum_i \lambda^{E_i} + \sum_{ij} \lambda_{ij}^{E_i E_j} + \ldots + \sum_k \lambda_k^{P_k} + \sum_{kl} \lambda_{kl}^{P_k P_l} \ldots$$

where the double subscripted terms indicate two-way interactions, and the . . . indicate that all higher order interactions among the predictors and among the outcome variables are also taken into account. This model can be rejected only if relationships between predictors and outcomes exist. In other words, if this model is rejected and types and if antitypes emerge, relationships among the outcomes and the predictors must exist. In longitudinal research, predictors often are series of antecedents or series of repeatedly observed measures that are taken before the outcome occurred.

Data Example

For the following data example, we analyze data from the MIS (Bogat et al., 2004; www.msu.edu/~mis/). We use the data from the fifth year of the study. We ask whether a diagnosis of maternal clinical-level symptoms 5 years after the birth of a child can be predicted from a pattern of intimate partner violence in the fifth year and the three years before. Four variables go into the analysis. The first three are *DV*3, *DV*4, and *DV*5. These variables indicate the status of a respondent as victim (coded 2) versus not victim (coded 1) of violence that was perpetrated by an intimate partner. The fourth variable is *SX*5, the diagnosis of clinical-level psychological problems (coded 2) versus no clinical-level problems (coded 1). Crossed, these four variables span the $2 \times 2 \times 2 \times 2$ *DV*3 × *DV*4 × *DV*5 × *SX*5 cross-classification shown in Tables 20.38 and 20.39.

TABLE 20.38 First-Order CFA of the *DV*3 × *DV*4 × *DV*5 × *SX*5 Cross-Classification

Configuration					
*DV*3 *DV*4 *DV*5 *SX*5	*m*	$\hat{m}$	*z*	*p*	Type/antitype?
1111	61.00	29.947	5.6745	.000000	Type
1112	5.00	7.103	−.7890	.215054	
1121	7.00	17.428	−2.4979	.006246	
1122	3.00	4.134	−.5576	.288572	
1211	17.00	27.278	−1.9680	.024537	
1212	3.00	6.470	−1.3642	.086259	
1221	15.00	15.875	−.2196	.413077	
1222	1.00	3.765	−1.4251	.077068	
2111	16.00	21.658	−1.2158	.112034	
2112	1.00	5.137	−1.8252	.033982	
2121	4.00	12.604	−2.4236	.007684	
2122	4.00	2.989	.5845	.279457	
2211	14.00	19.728	−1.2896	.098588	
2212	5.00	4.679	.1483	.441033	
2221	22.00	11.481	3.1044	.000953	Type
2222	15.00	2.723	7.4398	.000000	Type

TABLE 20.39 Predicting SX5 from the Serial Pattern of *DV* in the *DV*3 × *DV*4 × *DV*5 × *SX*5 Cross-Classification

Configuration					
*DV*3 *DV*4 *DV*5 *SX*5	*m*	$\hat{m}$	*z*	*p*	Type/antitype?
1111	61.00	53.347	1.0478	.147371	
1112	5.00	12.653	−2.1514	.015721	
1121	7.00	8.083	−.3809	.351641	
1122	3.00	1.917	.7821	.217075	
1211	17.00	16.166	.2075	.417819	
1212	3.00	3.834	−.4260	.335046	
1221	15.00	12.933	.5749	.282689	
1222	1.00	3.067	−1.1804	.118918	
2111	16.00	13.741	0.6094	.271121	
2112	1.00	3.259	−1.2514	.105402	
2121	4.00	6.466	−.9699	.166051	
2122	4.00	1.534	1.9915	.023212	
2211	14.00	15.358	−.3464	.364519	
2212	5.00	3.642	.7113	.238453	
2221	22.00	29.907	−1.4458	.074115	
2222	15.00	7.093	2.9688	.001495	Type

We analyze this table under two CFA base models. The first is the first-order CFA base model of variable independence,

$$\log \hat{m} = \lambda + \lambda^{DV3} + \lambda^{DV4} + \lambda^{DV5} + \lambda^{SX5}$$

This model can result in types and antitypes when any interactions exist. This includes interactions among the intimate partner violence indicators. These interactions are not of interest in the present context. Here, we ask whether patterns of the $DV3 \times DV4 \times DV5$ subtable are predictive of $SX5$. Therefore, we also estimate the P-CFA–type model

$$\log \hat{m} = \lambda + \lambda^{DV3} + \lambda^{DV4} + \lambda^{DV5} + \lambda^{SX5} + \lambda^{DV3,DV4} + \lambda^{DV3,DV5} + \lambda^{DV4,DV5} + \lambda^{DV3,DV4,DV5}$$

This model can be rejected only if interactions exist that link DV to SX. For both CFA runs, we use the z test and the Holland–Copenhaver procedure for the protection of α. Table 20.38 shows the results of first-order CFA, and Table 20.39 shows the results of CFA of the prediction of end points.

The overall goodness-of-fit likelihood-ratio X^2 of 94.60 leads us to reject the base model of variable independence ($df = 11$; $p < 0.01$). We, therefore, anticipate that types and antitypes emerge. Table 20.38 shows three types and no antitype. The first type is constituted by configuration 1 1 1 1. This is the modal configuration. 61 women indicated that they did not experience intimate partner violence at any time during the observation period considered for the present analysis, and they were not diagnosed as suffering from clinical-level symptoms. Only about 30 had been expected to show this pattern. We conclude that it is most likely that no clinical-level symptoms are diagnosed when women are not victimized over a span of three years, when their children are 2–5 years old. The second type is constituted by configuration 2 2 2 1. This type indicates that it can be predicted for more women than expected under the assumption of variable independence that no clinical-level symptoms are reported even if they suffer from consistent victimization, over a span of three years. In turn, the third type, constituted by configuration 2 2 2 2, suggests that more women than expected do show clinical-level symptoms. The last two types, seemingly contradictory, can be aggregated to constitute the *aggregate type* 2 2 2 ., where the dot indicates the variable aggregated over. The aggregate type suggests that more women than expected are victims of intimate partner violence in each of the three years considered here. Interestingly, the following analysis shows that no additional information than the violence pattern is needed to discriminate between the individuals with the profiles 2 2 2 1 and 2 2 2 2.

As said previously, the three types found in the $DV3 \times DV4 \times DV5 \times SX5$ cross-classification shown in Table 20.38 may simply reflect interactions among the DV measures. Therefore, we include these interactions in the base model. If types or antitypes still emerge, they reflect interactions among the DV measures and $SX5$, by necessity. The results for the P-CFA that takes all DV interactions into account are presented in Table 20.39.

The overall goodness-of-fit likelihood-ratio X^2 of 26.06 leads us to reject the base model that includes all interactions among the DC variables ($df = 7$; $p < 0.01$). We, therefore, anticipate that types and antitypes emerge. Table 20.39 shows one type and no antitype. The expected cell frequencies in Table 20.39 are, overall, much closer to the observed cell frequencies in Table 20.38. Considering that more effects and more complex effects were included in this base model, this is not surprising. The P-CFA base model for the prediction of an end point is significantly better than the base model of first-order CFA ($\Delta X^2 = 68.54$; $\Delta df = 4$; $p < 0.01$). Still, there is one significant model–data discrepancy, for configuration 2 2 2 2. This configuration—it constitutes a type—suggests that it is significantly more likely than expected that a woman who experiences intimate partner violence consistently over a span of 3 years, when her child is between 2 and 5 years old, is diagnosed with clinical-level symptoms. This applies even when all possible interactions among the DV measures across the three years are made part of the base model. The other two types that were found in first-order CFA disappeared, thus indicating that these types reflect interactions among the DV measures instead of interactions of DV measures with $SX5$.

To round out the analyses in which we predict the end point of clinical-level symptoms from a sequence of experiences of DV, we adopt a variable-oriented perspective and first estimate a path model. In this model, we predict $DV4$ from $DV3$, $DV5$ from $DV4$, and $SX5$ from $DV3$, $DV4$, and $DV5$. This model fails to describe the data ($\mathrm{LR} - X^2 = 8.34$; $df = 2$; $p = 0.015$). We, therefore, search for a parsimonious log-linear model. The model that includes all two-way and the three-way interactions among the DV measures and the two-way interactions $DX3 \times SX5$ and $DX5 \times SX5$ describes the data well ($\mathrm{LR} - X^2 = 7.45$; $df = 5$; $p = 0.19$). Each of these interactions is significant and interpretable. However, this model cannot be used to state that three consecutive years of intimate partner violence are predictive of

clinical-level psychological symptoms. For this statement, one needs CFA.

Predicting a Trajectory

There are many theories in developmental research that conceptualize turning points in development. Examples of such turning points can be found in life events. For example, when basketball player Len Bias died in 1986, at age 22, from cardiac arrhythmia that was induced by a cocaine overdose, a number of late adolescent cocaine users discontinued using the drug (Bachman, Johnston, O'Malley, & Schulenberg, 1996). On the other end of trajectories of drug use, whereas some individuals are hooked after initiation, many others do not adopt the habit of consistent and long-term drug use. Therefore, based on these and other examples, one can ask whether trajectories of development can be predicted from a particular point in time, event, or pattern of conditions.

To develop the CFA base model for the prediction of a trajectory, we proceed as in the last section (von Eye, Mair & Mun, 2010). The model we develop is also a variant of P-CFA. Let the descriptors of the potential turning point be labeled by $P_1, P_2, \ldots, P_i$, and the trajectory indicators by $Y_1, Y_2, \ldots, Y_m$. Then, the P-CFA base model that allows one to determine which trajectory is more (or less) likely to follow the potential turning point includes the following terms:

1. Main effects of all variables;
2. All possible interactions among the outcome variables; and
3. All possible interactions among the predictor variables; these are the variables that describe the potential turning point; if the turning point is described by just one variable, the main effect of this variable is included in the model (Term 1).

The corresponding log-linear base model is, thus, saturated on the side of the outcome variables as well as on the side of the variables that describe the turning point. In the present context, we focus on potential turning points that can be represented by just one variable. Multivariate potential turning points can be treated in an analogous fashion. The log-linear base model for configural prediction analysis of a potential turning point is

$$\log \hat{m} = \lambda + \sum \lambda_i^P + \sum_k \lambda_k^{Y_k} + \sum_{kl} \lambda_{kl}^{Y_k Y_l} \ldots$$

where the double subscripted terms indicate two-way interactions, and the ... indicates that all higher order interactions among variables that describe the outcome trajectory are also taken into account. This model can only be rejected if relationships between descriptors of the potential turning point and the outcome, that is, the trajectory, exist. In other words, if this model is rejected and types and antitypes emerge, relationships among the trajectory and the turning point must exist.

Data Example

For the data example of predicting a trajectory, we use the intimate partner violence data again (MIS; Bogat et al., 2004). Here, we ask whether violence experienced during the last trimester of a pregnancy is predictive of the trajectory of patterns of clinical-level psychological problems 3, 4, and 5 years after the birth of the child. We use the following variables: Violence during the last trimester of pregnancy, *DV*1, coded as 1 = no, and 2 = yes, and the indicators of maternal clinical-level problems *SX*3, *SX*4, and *SX*5, all coded as 1 = no and 2 = yes. We analyze the $2 \times 2 \times 2 \times 2$ cross-classification of these four variables under two models. To have a reference, we first estimate the first-order CFA base model of variable independence,

$$\log \hat{m} = \lambda + \lambda^{DV1} + \lambda^{SX3} + \lambda^{SX4} + \lambda^{SX5}$$

If this model is rejected, interactions among the four variables that span the table exist. To determine whether the interactions among *DV*1 and the trajectories constitute types and antitypes, we also estimate the P-CFA model of the prediction of trajectories,

$$\log \hat{m} = \lambda + \lambda^{DV1} + \lambda^{SX3} + \lambda^{SX4} + \lambda^{SX5} + \lambda^{SX3,SX4} + \lambda^{SX3,SX5} + \lambda^{SX4,SX5} + \lambda^{SX3,SX4,SX5}$$

If this model is rejected and types or antitypes emerge, *DV* during pregnancy is predictive of particular trajectories of clinical-level psychological symptoms. For both CFA runs, we use the normal approximation of the binomial test and protect α using the Holland–Copenhaver procedure. Table 20.40 summarizes the CFA results for the CFA base model of variable independence.

The overall goodness-of-fit likelihood-ratio X^2 of 80.48 leads us to reject the base model of variable independence ($df = 11$; $p < 0.01$), and we expect types and antitypes to emerge. Table 20.40 shows that first-order CFA identifies two types and no antitype. The first type, constituted by

TABLE 20.40 First-Order CFA of the $DV1 \times SX3 \times SX4 \times SX5$ Cross-Classification

Configuration					
*SX*3 *SX*4 *SX*5 *DV*1	m	$\hat{m}$	z	p	Type/antitype?
1111	59.00	36.662	4.0990	.000021	Type
1112	59.00	55.232	.6002	.274195	
1121	2.00	8.696	−2.3235	.010075	
1122	7.00	13.100	−1.7456	.040440	
1211	7.00	14.612	−2.0712	.019168	
1212	13.00	22.013	−2.0408	.020633	
1221	2.00	3.466	−.7944	.213468	
1222	10.00	5.221	2.1204	.016985	
2111	1.00	7.840	−2.4940	.006316	
2112	6.00	11.811	−1.7450	.040494	
2121	2.00	1.859	.1036	.458747	
2122	2.00	2.801	−.4822	.314823	
2211	2.00	3.125	−.6414	.260634	
2212	9.00	4.707	2.0033	.022575	
2221	2.00	.741	1.4652	.071430	
2222	10.00	1.116	8.4320	.000000	Type

TABLE 20.41 2-Group CFA of the $DV1 \times SX3 \times SX4 \times SX5$ Cross-Classification with *DV*1 as the Grouping Variable

Configuration				
*SX*3 *SX*4 *SX*5 *DV*1	m	z	p	Type?
1111	59.00			
1112	59.00	3.595	.000162	Discrimination type
1121	2.00			
1122	7.00	−1.109	.133726	
1211	7.00			
1212	13.00	−.472	.318352	
1221	2.00			
1222	10.00	−1.697	.044858	
2111	1.00			
2112	6.00	−1.410	.079339	
2121	2.00			
2122	2.00	.417	.338339	
2211	2.00			
2212	9.00	−1.515	.064948	
2221	2.00			
2222	10.00	−1.697	.044858	

configuration 1 1 1 1, suggests that women who do not suffer from intimate partner violence during pregnancy show, more often than expected, no clinical-level psychological symptoms in years 3, 4, and 5 after the birth of the child. In parallel, the second type, which is constituted by configuration 2 2 2 2, suggests that women who did suffer from intimate partner violence during pregnancy show clinical-level psychological symptoms in years 3, 4, and 5 after the birth of the child at higher rates than expected.

These two types are plausible and easy to interpret. However, they can, in theory, be the results of interactions among *DX*3, *DX*4, and *DX*5. Therefore, we estimate the model of prediction of trajectories that was specified previously. For the present purposes, we do not simply ask whether types and antitypes emerge (readers are invited to confirm that, although this base model is also rejected, no types or antitypes emerge; the overall goodness-of-fit LR-X^2 for this base model is 17.26; $df = 7$; $p = 0.02$). Instead, we ask whether the women who do versus do not suffer from intimate partner violence during pregnancy differ in the occurrence rates of particular trajectories. In other words, we analyze the data using the aforementioned base model from the perspective of a 2-group analysis. Table 20.41 summarizes the results.

The results in Table 20.41 indicate that victims of intimate partner violence differ from nonvictims in particular in the occurrence rate of a trajectory of no clinical-level psychological problems, that is, in configuration 1 1 1 .. This pattern is significantly more likely in the group of nonvictims than in the group of victims. The table gives equal observed cell frequencies, and one might conclude that the occurrence rates are the same. However, one has to take into account that the group of victims is, in the present sample, larger than the group of nonvictims (116 victims of intimate partner violence vs. 77 nonvictims), and that the estimation of the expected cell frequencies takes this proportion as well as the possible interactions among the three *SX* measures into account. The expected cell frequency for the nonvictims is 47.08 (below the observed frequency), and the expected cell frequency for the victims is 70.92 (above the observed frequency).

We conclude that the base model of CFA for the prediction of a trajectory is not only significantly better than the base model of first-order CFA ($\Delta X^2 = 63.22$; $\Delta df = 4$; $p < 0.01$), it also allows one to identify a difference in occurrence rate of the trajectory of no clinical-level psychological symptoms. The difference goes in the expected direction: nonvictims of intimate partner violence show this pattern significantly more often than victims.

DISCUSSION

This chapter introduces CFA as a method for the analysis of developmental processes, and illustrates various applications using real data. There are several unique aspects that distinguish CFA from more variable-oriented approaches.

We first discuss these differences broadly and then move to implications of these characteristics more specifically for translational research.

Unique Characteristics of CFA

The first characteristic that sets CFA apart from other approaches to research in developmental psychopathology is that CFA allows one to answer the same questions, but with individuals in mind instead of variables. Findings from variable-oriented research are difficult to translate into prevention or intervention practices given that effects of covariates cannot be isolated or removed in an individual. Similarly, it is difficult to estimate the cost-effectiveness of prevention and intervention programs based on variable-oriented research because of the co-occurrence of problems and associated contextual conditions observed in individuals. It is not surprising that cost-effectiveness of well-known prevention/intervention programs are gauged per individual, not per problem or desirable outcome (see also Mun, Bates, & Vaschillo, 2010). Likewise, when clinical practitioners make observations that can shed new light on etiological pathways, CFA can be utilized to see if the new discovery can be found in other populations or settings before more controlled, variable-oriented experimental studies can be conducted.

Based on CFA, results are stated in terms of profiles (configurations) that were observed more often or less often than expected and, therefore, contradict a particular hypothesis. This hypothesis is formulated such that the effects the researchers are interested in must exist when the hypothesis is rejected, for the individuals who exhibit a particular profile. The CFA base model incorporates these hypotheses. When using CFA, one typically hopes that the base model is rejected and types and antitypes emerge that illuminate the phenomenon of interest. If this is the case, the hypotheses of interest prevail. In general, the probability with which particular profiles occur allows one to make decisions about the hypotheses that are reflected in the CFA base model. This way, one of the chief goals of psychological research can be reached, that is, the goal of making statements about people.

Second, researchers who use CFA do not assume that variable relationship always include all categories of a variable. Instead, they assume that particular effects exist in particular sectors of the data space, for select groups of cases. In other words, CFA is more attuned to detecting subgroups than variable-oriented methods in which analysis for interactions or subgroups need to be explicitly specified. The difference between the two broad approaches is that in the former, finding subgroups or unusual patterns, via detecting types and antitypes, is the goal of application, but the opposite is true in the latter. Thus, for research in developmental psychopathology where the existence of subgroups can provide important clues to the etiology, treatment, and prognosis of a problem, CFA is a user-friendly, analytic tool.

A third major difference between CFA and other methods of analysis is that CFA allows one to make statements about sectors of the data space that are comparatively sparsely populated or even empty. This is done in the form of CFA antitypes. Talking about sectors in the data space that contain fewer cases than expected based on theory or prior knowledge can be of utmost importance in many research domains. Consider, for example, research on the effects of neuroleptic drugs. Some of these drugs are known to come with high risks of lowered life expectancy, extrapyramidal effects on motor control, or decrease in brain volume. Imagine a new drug that is being tested in patients. One domain of comparison with existing drugs concerns these side effects. An antitype for the new drug could indicate that, in comparison with existing drugs, certain side effects are particularly unlikely in a group of patients with a particular profile.

CFA and Translational Research

As discussed in the previous section, and illustrated in the examples in this chapter, application of CFA proceeds under the assumption that relations among variables are not necessarily valid across the entire range of admissible scores (when variables are metric) or all categories (when variables are categorical). As implied by this assumption, researchers use CFA to create statements that apply to individuals who exhibit a particular profile, that is, a particular pattern of scores or categories.

Interestingly, this assumption seems to closely correspond with the language used to describe results of developmental psychopathological research. For example, we read, in the context of research on the long-term effects of insecure attachment, that "one such outcome may be conduct disorder, in a child who has the genetic and neurobiological diathesis, who has an insecure representational model of the self, and who faces extremes and additional stress in the form of a violent home and/or community environment in conjunction with minimal support or nurturance from caregivers" (Cicchetti & Toth, 2008, p. 18; see also Richters & Cicchetti, 1993). This statement describes a profile of scores and categories. Children covered by this statement have four characteristics. They (1) possess the

genetic and neurobiological diathesis; (2) have an insecure representational model of the self; (3) face extreme and additional stress in the form of a violent home and/or community environment; and (4) face extreme and additional stress because of minimal support or nurturance from caregivers.

Regardless of whether some or all of these characteristics are necessary, or some or all are sufficient for the development of conduct disorder, it is important to realize that this pattern of characteristics, that is used to predict conduct disorder cannot be created using such methods as regression analysis, hierarchical linear modeling, or, more generally, structural modeling (the disconnect discussed in the introduction). Each of these methods creates statements on variable relations that include the entire range of scores of categories. In the previous example profile, only a selection of scores and categories is used. This implies either that no statement is made about the remaining scores or categories or that the outcome will not be observed when the remaining scores or categories are observed. In brief, this statement applies to a selection of individuals only.

Relating this basic characteristic of CFA to the desideratum of translational research, we note that this type of research is designed to "address how basic behavioral processes inform the diagnosis, prevention, treatment, and delivery of services for mental illness . . . ", or vice versa (National Advisory Mental Health Council, 2000, p. iii). It is one of the key characteristics of CFA that it suggests that these processes are not the same for individuals who differ in behavioral and neuropsychological profiles, or who live under different conditions. CFA also suggests that translational research, as well as the delivery of services must be tailored to individuals or subpopulations, that is, those who can benefit in particular from services. For example, dose–response models must be created that take characteristics of clients into account, type, and delivery of therapy must be tailored to the specific pattern of illness, or the combination of drugs and psychotherapy must take the interplay between diagnosis and other patient characteristics into account. The same drug, the same dose, and the same type of psychotherapy can have effects that are specific to the client in need of help, but also his or her living conditions, age or partner situation.

The statistical method of CFA is uniquely suited to point researchers as well as applied scholars to those patterns that are most promising, but also to those patterns that are least promising for intervention measures. The methods is, therefore, of great interest for basic, translational, and applied research.

Limitations

In spite of all the encouraging characteristics of CFA, there are limitations. Here, we discuss three most important ones. First, to perform CFA when variables are metric, researchers often resort to reducing the number of possible values to a small, manageable number of categories. The reason for this is obvious. Crossing variables and examining individual profiles would result in exorbitantly large numbers of profiles, tables of sizes that can no longer be analyzed, and most profiles would not be observed at all or observed just once. In other words, to enable researchers to analyze profiles statistically, the number of categories must be small, and the sample of cases must be large.

Naturally, when the number of possible values is reduced, information is bound to disappear. This issue has been discussed at length in the methodological literature (e.g., MacCallum et al., 2002). However, we maintain that (1) researchers do not always have a choice when they analyze data with CFA, log-linear models, or latent class analysis, (2) not always will this loss of information result in distorted results (for an example, see von Eye & Mair, 2011), and (3) because of measurement error, dichotomization of metric response variables can provide even better, unbiased results by reducing errors in measurement (Shentu & Xie, 2010). Likewise, cutting-edge medical research is typically conducted using dichotomized or binary outcomes. Results from this type of variables can be easier to communicate with nonacademic, important stake holders (Mun et al., 2010).

Outlook. Methods of CFA are still in need of further development. Although visible progress has been made (e.g., von Eye et al., 2010), there are still domains that CFA has yet to enter. Two domains are of note. The first domain is that of causality research. Recently, considerable progress has been made in the development of statistical methods that allow one to test hypotheses that are compatible with assumptions of causal relations (see Dodge & Rousson, 2001; von Eye & DeShon, 2012; von Eye & Wiedermann, 2013). The development of CFA methods in this domain is at its beginning (von Eye & Brandtstädter, 1997, 1998; von Eye, Wiedermann, & Mun, 2013). The most important aspect of these developments is that the idea that carries CFA, that not all categories of variables are necessarily involved in relations, be translated into the domain of causal relations.

Second, this idea needs to be translated into the universe of latent variables. To date, there exist no configural approaches to latent variables analysis. One would hope that CFA models be developed that allow researchers to

analyze composite variables without inflating the table under study beyond the tractable.

Value Added

To conclude this chapter, we summarize the value added by CFA.

1. CFA allows researchers to make statements about people instead of variables.
2. These statements are valid for particular groups of individuals, but not for others.
3. These statements are based on selections of categories, not all categories of variables.
4. CFA allows one to test very specific hypotheses.
5. CFA allows one to identify typical developmental trajectories and atypical developmental trajectories. This option is of particular importance in research on the development of pathological behavior.

REFERENCES

Abramowitz, M., & Stegun, I. A. (1972). *Handbook of mathematical functions*. New York, NY: Dover.

Agresti, A. (1998). *An introduction to categorical data analysis*. New York, NY: Wiley.

Bachman, J. G., Johnston, L. D., O'Malley, P. M., & Schulenberg, J. (1996). Transitions in drug use during late adolescence and young adulthood. In J. A. Graber, J. Brooks-Gunn, & A. C. Petersen (Eds.), *Transitions through adolescence: Interpersonal domains and contexts* (pp. 111–140). Hillsdale, NJ: Lawrence Erlbaum Associates.

Baltes, P. B., Reese, H. W., & Nesselroade, J. R. (1977). *Life-span developmental psychology: Introduction to research methods*. Monterey, CA: Brooks/Cole.

Bergman, L. R., & Magnusson, D. (1997). A person-oriented approach in research on developmental psychopathology. *Development and Psychopathology*, *9*, 291–319. doi: 10.1017/S095457949700206X

Bergman, L. R., Magnusson, D., & El-Khouri, B. M. (2003). *Studying individual development in an interinidividual context: A person-oriented approach*. Mahwah, NJ: Erlbaum.

Bishop, Y. M. M., Fienberg, S. E., & Holland, P. W. (1975). *Discrete multivariate analysis*. Cambridge, MA: MIT Press.

Bogat, G. A., Levendosky, A. A., DeJonghe, E., Davidson, W. S., & von Eye, A. (2004). Pathways of suffering: The temporal effects of domestic violence on women's mental health. *Maltrattamento e abuso all'infanzia*, *6*, 97–112.

Bogat, G. A., Levendosky, A. A., & von Eye, A. (2005). The future of research on intimate partner violence (IPV): Person-oriented and variable-oriented perspectives. *American Journal of Community Psychology*, *36*, 49–70. doi: 10.1007/s10464-005-6232-7

Bowker, A. H. (1948). A test for symmetry in contingency tables. *Journal of the American Statistical Association*, *43*, 572–574. doi: 10.2307/2280710

Brennan, R. L., & Prediger, D. J. (1981). Coefficient λ: Some uses, misuses, and alternatives. *Educational and Psychological Measurement*, *41*, 687–699.

Cicchetti, D., & Toth, S.L. (2009). The past achievements and future promises of developmental psychopathology: The coming of age of a discipline. *Journal of Child Psychology and Psychiatry*, *50*, 16–25. doi: 10.1111/j.1469-7610.2008.01979.x

Cohen, J. (1960). A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*, *20*, 37–46. doi: 10.1177/001316446002000104

Dodge, Y., & Rousson, V. (2001). On asymmetric properties of the correlation coefficient in the regression setting. *American Statistician*, *55*, 51–54. doi: 10.1198/000313001300339932

Everitt, B. S., & Howell, D. C. (Eds.). (2008). *Encyclopedia of statistics in behavioral science*. Chichester, UK: Wiley.

Eysenck, H. J. (1952). *The scientific study of personality*. London, UK: Routledge & Paul. doi: 10.1111/j.2044-8317.1953.tb00132.x

Fennig, S., Craig, T. J., Tananberg-Karant, M., & Bromet, E. J. (1994). Comparison of facility and research diagnoses in first admission psychotic patients. *American Journal of Psychiatry*, *151*, 1423–1429.

Finkelstein, J. W., von Eye, A., & Preece, M. A. (1994). The relationship between aggressive behavior and puberty in normal adolescents: A longitudinal study. *Journal of Adolescent Health*, *15*, 319–326. doi: 10.1016/1054-139X(94)90605-X

First, M. B. & Tasman, A. (2004). *DSM-IV-TR mental disorders: Diagnosis, etiology and treatment*. Chichester, UK: Wiley.

Goodman, L. A. (1974). Exploratory latent structure analysis using both identifiable and unidentifiable models. *Biometrika*, *61*, 215–231. doi: 10.2307/2334349

Goodman, L. A. (1984). *The analysis of cross-classified data having ordered categories*. Cambridge, MA: Harvard University Press. doi: 10.2307/2531978

Goodman, L. A. (1991). Measures, models, and graphical displays in the analysis of cross-classified data. *Journal of the American Statistical Association*, *86*, 1085–1111. doi: 10.2307/2290528

Granger, C. W. J. (1969). Investigating causal relations by econometric models and cross-spectral methods. *Econometrica*, *37*, 424–438. doi: 10.2307/1912791

Havránek, T., & Lienert, G. A. (1984). Local and regional versus global contingency testing. *Biometrical Journal*, *26*, 483–494. doi: 10.1002/bimj.4710260504

Heilmann, W.-R., & Lienert, G. A. (1982). Predictive configural frequency analysis evaluated by simultaneous Berchtold-corrected fourfold X2-tests. *Biometrical Journal*, *24*, 723–728. doi: 10.1002/bimj.4710240714

Heilmann, W.-R., Lienert, G. A., & Maly, V. (1979). Prediction models in configural frequency analysis. *Biometrical Journal*, *21*, 79–86. doi: 10.1002/bimj.4710210110

Holland, B. S., & Copenhaver, M. D. (1987). An improved sequentially rejective Bonferroni test procedure. *Biometrics*, *43*, 417–423. doi: 10.2307/2531823

Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics*, *6*, 65–70.

Huth-Bocks, A. C., Levendosky, A. A., & Bogat, G. A. (2002). The effects of domestic violence during pregnancy on maternal and infant health. *Violence and Victims*, *17*, 169–185. doi: 10.1891/vivi.17.2.169.33647

Huth-Bocks, A. C., Levendosky, A. A., Bogat, G. A., & von Eye, A. (2004). The impact of maternal characteristics and contextual variables on infant–mother attachment. *Child Development*, *75*, 480–496. doi: 10.1111/j.1467-8624.2004.00688.x

Krauth, J. (2003). Type structures in CFA. *Psychology Science*, *45*, 217–222.

Krauth, J. (2005). Paradoxes in multidimensional contingency tables. What does this mean for CFA? *Psychology Science*, *47*, 304–314.

Küchenhoff, H. (1986). A note on a continuity correction for testing in three-dimensional configural frequency analysis. *Biometrical Journal*, *28*, 465–468.

Laursen, B., Little, T. D., & Card, N. A. (Eds.) (2012). *Handbook of developmental research methods*. New York, NY: Guilford Press.

Lehmacher, W. (1981). A more powerful simultaneous test procedure in configural frequency analysis. *Biometrical Journal*, *23*, 429–436. doi: 10.1002/bimj.4710230503

Lienert, G.A. (1964). *Belastung und Regression [Stress and regression]*. Meisenheim am Glan: Hain.

Lienert, G. A. (1968). *Die "Konfigurationsfrequenzanalyse" als Klassifikationsmethode in der klinischen Psychologie* [*Configural frequency analysis as a classification method in clinical psychology*]. Paper presented at the 26. Kongress der Deutschen Gesellschaft für Psychologie in Tübingen.

Lienert, G. A., & Krauth, J. (1973). Die Konfigurationsfrequenzanalyse als Prädiktionsmodell in der angewandten Psychologie [Configural frequency analysis—A prediction model for applied psychology]. In H. Eckensberger (Ed.), *Bericht über den 28. Kongress der Deutschen Gesellschaft für Psychologie in Saarbrücken 1972* (pp. 219–228). Göttingen, Germany: Hogrefe.

Lienert, G. A., & Krauth, J. (1975). Configural frequency analysis as a statistical tool for defining types. *Educational and Psychological Measurement*, *35*, 231–238. doi: 10.1177/001316447503500201

Lienert, G. A., & Straube, E. (1980). Die Konfigurationsfrequenzanalyse XI. Strategien des Symptom-Konfigurations-Vergleichs vor und nach einer Therapie [Configural frequency analysis XI. Strategies for the comparison of symptoms and sundromes before and after a therapy]. *Zeitschrift für Klinische Psychologie und Psychotherapie*, *28*, 110–123.

Lienert, G. A., & von Eye, A. (1984). Multivariate Änderungsbeurteilung mittels Inkrementen- Konfigurationsclusteranalyse [Analyzing multivariate change using configural frequency analysis of increments]. *Psychologische Beiträge*, *26*, 363–371.

Lienert, G. A., & von Eye, A. (1986). Nonparametric two-sample CFA of incomplete learning curves. In F. Klix & H. Hagendorf (Eds.), *Human memory and cognitive capabilities* (pp. 123–138). New York, NY: Elsevier.

Lienert, G. A., & von Eye, A. (1989). Die Konfigurationsclusteranalyse als Alternative zur KFA [Configural cluster analysis—An alternative to configural frequency analysis]. *Zeitschrift für Klinische Psychologie, Psychopathologie, und Psychotherapie*, *37*, 451–457.

Loken, E. (2010). Person-specific analysis of the dynamics of weight change. In P. C. M. Molenaar & K. M. Newell (Eds.), *Individual pathways of change: Statistical models for analyzing learning and development* (pp. 9–22). Washington, DC: American Psychological Association. doi: 10.1037/12140-001

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods*, *7*, 19–40. doi: 10.1037//1082-989X.7.1.19

Magnusson, D. (1997). (Ed.). *The life-span development of individuals: Behavioral, neurobiological, and psychosocial perspectives: A synthesis*. New York, NY: Cambridge University Press.

Martinez-Torteya, C., Bogat, G. A., von Eye, A., & Levendosky, A. A. (2009). Resilience among children exposed to domestic violence: The role of protective and vulnerability factors. *Child Development*, *80*, 562–577. doi: 10.1111/j.1467-8624.2009.01279.x

McNemar, Q. (1947). Note on the sampling error of the difference between correlated proportions or percentages. *Psychometrika*, *12*, 143–157. doi: 10.1007/BF02295996

Meehl, P. E. (1950). Configural scoring. *Journal of Consulting Psychology*, *14*, 165–171. doi: 10.1037/h0058049

Mellenbergh, G. J. (1996). Other null model, other (anti)type. *Applied Psychology: An International Review*, *45*, 329–330. doi: 10.1111/j.1464-0597.1996.tb00772.x

Molenaar, P. C. M. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology—This time forever. *Measurement: Interdisciplinary Research and Perspectives*, *2*, 201–218. doi: 10.1207/s15366359mea0204_1

Molenaar, P. C. M., & Campbell, C. G. (2009). The new person-specific paradigm in psychology. *Current Directions in Psychology*, *18*, 112–117. doi: 10.1111/j.1467-8721.2009.01619.x

Mun, E. Y., Bates, M. E., & Vaschillo, E. (2010). Closing the gap between person-oriented theory and methods. *Development and Psychopathology*, *22*(2), 261–271. doi: 10.1017/S0954579410000039.

Muthén, L. K., & Muthén, B. O. (2010). *Mplus user's guide* (6th ed.). Los Angeles, CA: Muthén & Muthén.

National Advisory Mental Health Council (2000). *Translating behavioral science into action: Report of the National Advisory Mental Health Council's Behavioral Science Workgroup (00-4699)*. Bethesda, MD: National Institutes of Health.

Nesselroade, J. R., Pruchno, R., & Jacobs, A. (1986). Reliability and stability in the measurement of psychological states: An illustration with anxiety measures. *Psychologische Beiträge*, *28*, 252–264.

Olejnik, S., Li, J., Supattathum, S., & Huberty, C. J. (1997). Multiple testing and statistical power with modified Bonferroni procedures. *Journal of Educational and Behavioral Statistics*, *22*, 389–406. doi: 10.2307/1165229

Overall, J. E., & Gorham, D. R. (1962). The brief psychiatric rating scale. *Psychological Reports*, *10*, 799–812. doi://10.2466/PR0.10.3.799-812

Perrine, M. W., Mundt, J. C., Searles, J. S., & Lester, L. S. (1995). Validation of daily self-report consumption using interactive voice response (IVR) technology. *Journal of Studies on Alcohol and Drugs*, *56*, 487–490.

Richters, J. E., & Cicchetti, D. (1993). Mark Twain meets DSM-III-R: Conduct disorder, development, and the concept of harmful dysfunction. *Development and Psychopathology*, *5*, 5–29. doi: 10.1017/S0954579400004235

Schuster, C., & Smith, D. A. (2002). Indexing systematic rater agreement with a latent class model. *Psychological Methods*, *7*, 384–395. doi: 10.1037/1082-989X.7.3.384

Shentu, Y., & Xie, M. (2010). A note on dichotomization of continuous response variable in the presence of contamination and model misspecification. *Statistics in Medicine*, *29*(21), 2200–2214. doi: 10.1002/sim.3966

Stevens, W. L. (1939). Distribution of groups in a sequence of alternatives. *Annals of Eugenics*, *9*, 10–17. doi: 10.1111/j.1469-1809.1939.tb02193.x

Stouffer, S. A., Suchman, E. A., DeVinney, L. C., Star, S. A., & Williams, Jr., R. M. (1949). *Studies in social psychology in World War II: The American soldier: Vol. 1, Adjustment during army life*. Princeton, NJ: Princeton University Press.

Swed, F. S., & Eisenhart, C. (1943). Tables for testing randomness of grouping in a sequence of alternatives. *Annals of Mathematical Statistics*, *14*, 66–87. doi: 10.1214/aoms/1177731494

Vermunt, J. K. (1997). *Lem: A general program for the analysis of categorical data*. Unpublished program manual, Tilburg University.

von Eye, A. (1988). The general linear model as a framework for models in configural frequency analysis. *Biometrical Journal*, *30*, 59–67. doi: 10.1002/bimj.4710300110

von Eye, A. (1989). Zero-missing-non existing: A conceptualization of the missing data problem in longitudinal research with categorical data solutions. In M. Brambring, F. Lösel, & H. Skowronek (Eds.),

Children at risk: Assessment and longitudinal research (pp. 336–355). New York, NY: De Gruyter.

von Eye, A. (2002). *Configural frequency analysis—Methods, models, and applications*. Mahwah, NJ: Lawrence Erlbaum.

von Eye, A. (2004a). Base models for configural frequency analysis. *Psychology Science*, *46*, 150–170.

von Eye, A. (2004b). The treasures of Pandora's box. *Measurement: Interdisciplinary Research and Perspectives*, *2*, 244–247.

von Eye, A., & Bergman, L.R. (2003). Research strategies in developmental psychopathology: Dimensional identity and the person-oriented approach. *Development and Psychopathology*, *15*, 553–580. doi: 10.1017/S0954579403000294

von Eye, A., Bergman, L. R., & Hsieh, C.-A. (2015). Person orientation. In W. R. Overton & P. C. M. Molenaar (Eds.). *Handbook of child development: Vol. 1. Theory and methods*. New York, NY: Wiley.

von Eye, A., & Bogat, G.A. (2006). Person orientation—Concepts, results, and development. *Merrill Palmer Quarterly*, *52*, 390–420. doi: 10.1353/mpq.2006.0032

von Eye, A., & Brandtstädter, J. (1997). Configural frequency analysis as a searching device for possible causal relationships. *Methods of Psychological Research—Online*, *2*, 1–23.

von Eye, A., & Brandtstädter, J. (1998). The wedge, the fork, and the chain—Modeling dependency concepts using manifest categorical variables. *Psychological Methods*, *3*, 169–185. doi: 10.1037/1082-989X.3.2.169

von Eye, A., & DeShon, R. P. (2012). Directional dependency in developmental research. *International Journal of Behavior Development*, *36*, 303–312. doi: 10.1177/0165025412439968

von Eye, A., & Gutiérrez-Peña, E. (2004). Configural frequency analysis—The search for extreme cells. *Journal of Applied Statistics*, *31*, 981–997. doi: 10.1080/0266476042000270545

von Eye, A., & Lienert, G. A. (1984). Die Konfigurationsfrequenzanalyse XX. Typen und Syndrome zweiter Ordnung [Configural Frequency Analysis XX. Second order types and syndromes]. *Zeitschrift für Klinische Psychologie*, *Psychopathologie und Psychotherapie*, *32*, 345–355.

von Eye, A., & Mair, P. (2008a). Functional Configural Frequency Analysis: Explaining types and antitypes. *Bulletin de la Société des Sciences Médicales, Luxembourg*, *144*, 35–52.

von Eye, A., & Mair, P. (2008b). A functional approach to configural frequency analysis. *Austrian Journal of Statistics*, *37*, 161–173.

von Eye, A., & Mair, P. (2008c). A functional approach to configural frequency analysis—Computational issues. In M. Stemmler, E. Lautsch, & D. Martinke (Eds.), *Configural frequency analysis (CFA) and other nonparametric statistical methods: Gustav A. Lienert Memorial Issue* (pp. 84–94). Lengerich: Pabst.

von Eye, A., & Mair, P. (2011). On the effects of dichotomizing information. In A. A. Hernández, & J. G. Hernández (eds.), *Memoria del XXV Foro Nacional de Estadística* (pp. 11–19). Aguascalientes, Ags., Mexico: Instituto Nacional de Estadística y Geografía.

von Eye, A., Mair, P., & Bogat, G.A. (2005). Prediction models for configural frequency analysis. *Psychology Science*, *47*, 342–355.

von Eye, A., Mair, P., & Mun, E.-Y. (2010). *Advances in configural frequency analysis*. New York, NY: Guilford Press.

von Eye, A., & Mun, E.Y. (2005). *Modeling rater agreement—Manifest variable approaches*. Mahwah, NJ: Lawrence Erlbaum.

von Eye, A., & Mun, E.-Y. (2007). A note on the analysis of difference patterns—Structural zeros by design. *Psychology Science*, *49*, 14–25.

von Eye, A., & Mun, E.-Y. (2012). Interindividual differences in intraindividual change in categorical variables. *Psychological Test and Assessment Modeling*, *54*, 151–167.

von Eye, A., & Mun, E.-Y. (2013). *Log-linear modeling—Concepts, interpretation and applications*. New York, NY: Wiley. doi: 10.1002/9781118391778

von Eye, A., & Nesselroade, J. R. (1992). Types of change: Application of configural frequency analysis to repeated observations in developmental research. *Experimental Aging Research*, *18*, 169–183. doi: 10.1080/03610739208260357

von Eye, A., & Rovine, M. J. (1994). Non-standard log-linear models for orthogonal prediction configural frequency analysis. *Biometrical Journal*, *36*, 177–184. doi: 10.1002/bimj.4710360210

von Eye, A., Schauerhuber, M., & Mair, P. (2007). Significance tests for the measure of raw agreement. *InterStat*. Retrieved from http://interstat.statjournals.net/YEAR/2007/abstracts/0701001.php

von Eye, A., & Spiel, C. (1996). Standard and non-standard log-linear symmetry models for measuring change in categorical variables. *American Statistician*, *50*, 300–305. doi: 10.2307/2684924

von Eye, A., Spiel, C., & Rovine, M. J. (1995). Concepts of nonindependence in configural frequency analysis. *Journal of Mathematical Sociology*, *20*, 41–54. doi: 10.1080/0022250X.1995.9990151

von Eye, A., & von Eye, M. (2005). Can one use Cohen's kappa to examine disagreement? *Methodology*, *1*, 129–142. doi: 10.1027/1614-2241.1.4.129

von Eye, A., & Wiedermann, W. (2013). On direction of dependence in latent variable contexts. *Educational and Psychological Measurement*, *74*, 5–30. doi: 10.1177/0013164413505863

von Eye, A., Wiedermann, W., & Mun, E.-Y. (2013). Granger causality—Statistical analysis under a configural perspective. *IPBS: Integrative Psychological & Behavioral Science*, *47*, 1–21. doi: 10.1007/s12124-013-9243-1

von Weber, S., Lautsch, E., & von Eye, A. (2003). On the limits of Configural Frequency Analysis: Analyzing small tables. *Psychology Science*, *45*, 339–354.

Wald, A., & Wolfowitz, J. (1940). On a test whether two alternatives are from the same population. *Annals of Mathematical Statistics*, *11*, 147–162.

Wallis, W. A., & Moore, G. H. (1941). A significance test for time series analysis. *Journal of the American Statistical Association*, *20*, 257–267. doi: 10.2307/2279616

Zimmerman, D. W. (2009). The reliability of difference scores in populations and samples. *Journal of Educational Measurement*, *46*, 19–42. doi: 10.1111/j.1745-3984.2009.01067.x

CHAPTER 21

Moderation and Mediation in Interindividual Longitudinal Analysis

JENNIFER L. KRULL, JEEWON CHEONG, MATTHEW S. FRITZ, and DAVID P. MACKINNON

INTRODUCTION 922
THE ROLE OF MODERATION AND MEDIATION ANALYSES IN DEVELOPMENTAL PSYCHOPATHOLOGY RESEARCH 923
ECLS-K DATA EXAMPLES 924
BASIC MODERATION 925
BASIC MEDIATION MODEL 928
COMBINING BASIC MEDIATION AND MODERATION 931
LONGITUDINAL DATA 933
MULTILEVEL MODELING 934
Moderation in Multilevel Models 936
Mediation in Multilevel Models 937
MULTILEVEL MODELING APPROACHES FOR LONGITUDINAL DATA 939
Multilevel Growth Modeling 939
Multilevel Exponential Decay Model 940
STRUCTURAL EQUATION MODELING 941
Mediation in SEM 941
Moderation in SEM 941
STRUCTURAL EQUATION MODELS FOR LONGITUDINAL DATA 942
Autoregressive Model 942
Latent Growth Curve Model 943
Latent Change Model 944
Exponential Decay Structural Equation Model 945
MODELS FOR MODERATION AND MEDIATION IN LONGITUDINAL DATA 946
Lag as Moderator Model 946
Multilevel Growth Models with Time-Varying Covariate Interactions 948
Mediation Change over Time Model 950
Longitudinal Mediation in Autoregressive Panel Models 953
Latent Growth Curve Mediation Models 958
Estimating LGC Mediation as a Multilevel Model 961
Autoregressive Latent Trajectory Mediation Models 963
Latent Change Score Models for Moderation and Mediation 964
Moderation and Mediation in Exponential Decay Models 967
Moderated Mediation in SEM 970
MODERN CAUSAL INFERENCE AND LONGITUDINAL MEDIATION MODELS 973
Potential Outcomes Model 974
Comparison of Traditional and Potential Outcome Mediation Analysis 975
Moderation in the Potential Outcome Model 976
Potential Outcome Mediation Model with Longitudinal Data 976
Inverse Probability Weighting with Longitudinal Data 977
Causal Estimators for the ECLS-K Data Set 978
FUTURE DIRECTIONS 978
REFERENCES 980

INTRODUCTION

In 1986, Reuben Baron and David Kenny published their classic article on the distinction between mediator and moderator variables in social psychological research (Baron & Kenny, 1986). This particular paper has been incredibly influential on the way data have been analyzed in the social sciences and, according to the Web of Science, has been cited more than 24,000 times to date. In fact, this is the most highly cited paper written by any social psychologist (Nosek et al., 2010), and its citation count is still climbing by more than 1000 each year. This particular article has even spawned its own spinoff literature, with a number of recent articles including Baron and Kenny in their titles, specifically written in response to and elaborating upon the initial Baron and Kenny work, including *Reconsidering Baron and Kenny* (Zhao, Lynch, & Chen, 2010), *Reflections on the Baron and Kenny Model of Statistical Mediation* (Pardo & Román, 2013), and two additional works with *Beyond Baron and Kenny* in their titles (Hayes, 2014; Krause et al., 2010).

Perhaps the popularity and influence of this particular paper is due to that fact that it strives to distinguish between *mediation* and *moderation*, two "often-confused

functions of third variables" (Baron & Kenny, 1986, p. 1173), both conceptually and in terms of the analyses necessary to test them. Both mediation and moderation involve systems of three variables. Each has an initial or focal predictor variable (which we will refer to as *X*) and an outcome or criterion variable (which we will refer to as *Y*). In addition, there is a third variable—the mediator (*M*) or moderator (*Z*)—involved in the system. Baron and Kenny (1986) state that a moderator "is a qualitative . . . or quantitative . . . variable that affects the direction and/or strength of the relation between an independent or predictor variable and a dependent or criterion variable" (p. 1174) and that it "partitions a focal independent variable into subgroups that establish its domains of maximal effectiveness in regard to a given dependent variable" (p. 1173). In other words, a moderator affects the association between two variables such that the impact of the predictor on the criterion varies depending on the level or value of the moderator (Holmbeck, 1997). Baron and Kenny (1986) stated that a mediator, on the other hand, "represents the generative mechanism through which the focal independent variable is able to influence the dependent variable of interest" (p. 1173). The key features defining such a mediator model are that (1) the predictor influences the mediator, and (2) the mediator, in turn, influences the outcome variable (Holmbeck, 1997; MacKinnon & Dwyer, 1993).

In the nearly 30 years since the publication of the Baron and Kenny article, the analytic literatures on both mediation and moderation have continued to grow. For example, recommendations for the post hoc probing of moderated effects have been extensively developed (Aiken & West, 1991), as have optimal methods for statistical testing and finding confidence intervals for mediated effects (MacKinnon, Fritz, Williams, & Lockwood, 2007; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2004, 2008). In addition, the collection of longitudinal data has become more prevalent as new analytic approaches well-suited to such data have been developed and researchers have come to realize the benefit of investigating how individuals change across time (Willett, 1997). In this chapter, we will discuss how both mediation and moderation can be explored in longitudinal data in which the focal predictors, outcomes, and moderators and mediators are repeatedly measured over time.

We will begin by discussing how the concepts of mediation and moderation feature in developmental psychopathology research and outlining the typical analysis methods used to identify moderation and mediation in cross sectional data. Because these methods and their application in particular literatures has been covered extensively elsewhere (e.g., Dearing & Hamilton, 2006; Frazier, Tix, & Baron, 2004; Gogineni, Alsup, & Gillespie, 1995; Rose, Holmbeck, Coakley, & Franks, 2004) our discussion will be relatively brief. Then we will explain the possible benefits of collecting longitudinal observations and discuss two analytic frameworks—multilevel modeling and structural equation modeling—that are often used to model longitudinal data. Then we will bring these together and discuss in detail various models designed to test moderation and mediation hypotheses in longitudinal data, including a lag-as-moderator approach, *interactions* between time and time-varying covariates in multilevel models, a mediation *change over time* model, autoregressive and latent growth models (as well as a combination of the two), a latent change score approach, and an exponential decay model. This is followed by a discussion of how modern methods of determining causal inference may be applied and extended in the longitudinal context. Finally, we conclude with a section on promising directions for future research on mediation and moderation in longitudinal data.

THE ROLE OF MODERATION AND MEDIATION ANALYSES IN DEVELOPMENTAL PSYCHOPATHOLOGY RESEARCH

Several of the foundational ideas in developmental psychopathology involve the types of associations that can be appropriately tested with models that include moderated and mediated effects. A primary goal—if not *the* primary goal—of developmental science is to understand how processes unfold over time, and it is precisely this idea of *process* that guides methodology designed to detect mediation. In fact, one of the earliest presentations of methodology to detect longitudinal mediation effects described the procedure as process analysis (Judd & Kenny, 1981). The Judd and Kenny article is particularly important as it describes the importance of longitudinal mediation relations, while the Baron and Kenny article does not. The often used geographic analogy of *developmental pathways* implies that earlier variables affect later variables in series and readily maps onto mediational logic where initial variables predict mediators that, in turn, predict outcomes. The sort of sequential effects in a mediation analysis could also be used to model the spreading influence across domains or systems in developmental cascades (Masten & Cicchetti, 2010) and chain

reactions (Rutter, 1999). In addition, mediation analyses can be useful in exploring how interventions designed to change or prevent maladaptive behavior achieve their effects. Intervention versus control is the initial variable, constructs that are targeted by the intervention are the mediators, and the behavior is the outcome in such an analysis.

The idea that contextual effects influence individual development is a natural fit for a moderation framework, which can model how characteristics of the individual interact with aspects of the physical and social environment to produce adaptive and maladaptive behavior. The emphasis on risk and resilience that features prominently in the developmental psychopathology literature also suggests an analytic framework involving moderator variables. Vulnerabilities and protective factors from multiple domains serve as moderators and interact with stressors to produce a range of behavioral outcomes. Any particular outcome is probably not a simple function of a single isolated factor but rather the result of a combination of multiple biological, social/cultural, and psychological factors, and a framework that can accommodate such interactions is necessary to model such influences appropriately. The concept of multifinality—the idea that a given risk factor could lead to a variety of different outcomes depending on other aspects of the system (Cicchetti & Rogosch, 1996)—is necessarily going to involve some moderator variables that determine the manner in which the risk factor is ultimately expressed behaviorally.

Indeed, modeling a process as complex as human development may ultimately require models that combine aspects of both mediation and moderation. Viewing the individual as a complex system with variables at multiple levels exerting mutual and potentially reciprocal effects might lead to models that examine the effects of change at one level of the system on mediators at other levels that ultimately affect behavioral outcomes. One might also test whether the effects of focal variables at one level might depend upon the state of moderator variables at other levels. Adding to this the longitudinal aspect of processes unfolding over time results in an incredibly intricate framework of influences that will require careful consideration, clever study design, and sophisticated analytical tools to explore and ultimately understand. The focus of this chapter is to provide developmental psychopathology researchers with information about analytic techniques involving mediation and moderation in longitudinal data that might prove useful in understanding how developmental processes result in adaptive and maladaptive behavior.

ECLS-K DATA EXAMPLES

The various moderation and mediation analyses described in this chapter will be illustrated with examples drawn from the Early Childhood Longitudinal Study–Kindergarten Cohort (ECLS-K) data. This data set includes a wealth of information on a nationally representative sample of children who entered kindergarten in the 1998–1999 school year and were followed through the eighth grade. ECLS-K participants were selected via a multistage probability sampling design (U.S. Department of Education, National Center for Educational Statistics, 2009). One hundred geographical areas consisting of counties or groups of counties were initially selected, and a total of 1,280 public and private schools were sampled from within these areas. A target of approximately 24 kindergarteners enrolled at each school were then selected for the study. In total, the original sample at kindergarten fall included 21,260 children.

Study participants were assessed at kindergarten fall and spring, first grade fall and spring, third grade spring, fifth grade spring, and eighth grade spring. Because the first grade fall assessment involved only a small subsample of participants and many of the measures of interest here were not assessed at eighth grade spring, we focus on the remaining five assessments. For analyses in which equally spaced time points are desirable, we will focus on the first through fifth grade assessments, which occurred every 2 years. At each assessment, data were gathered from children, parents, and teachers. Our illustrative analyses utilize demographic information, child academic assessments, and teacher questionnaire data.

To further unify the illustrative analyses, we will focus on mediation and moderation effects involving attention-deficit/hyperactivity disorder (ADHD). A number of research efforts have examined ADHD in the ECLS-K data set (e.g., Barnard-Brak & Brak, 2011; Morgan, Staff, Hillemeier, Farkas, & Maczuga, 2013; Schneider & Eisenberg, 2006; Stone, Brown, & Hinshaw, 2010). Several of these have focused primarily on diagnoses of ADHD within the sample. We focus instead on reported ADHD symptom levels, which provide different (and, some would argue, better) information than diagnosis indicators (Currie & Stabile, 2006, 2009; Fletcher & Wolfe, 2008).

A modified version of the Social Rating Scale (adapted by ECLS-K with permission; Gresham & Elliott, 1990) was administered to teachers at each time point to assess children's social and behavioral functioning in the classroom. Four of the five subscales of the Social Rating Scale are used in our illustrative analyses: Approaches to Learning

(LEARN, six items), Externalizing Behavior (EXTERN, five items), Interpersonal Skills (INTERP, five items), and Internalizing Behavior (INTERN, four items). These subscales are highly reliable, with split half reliabilities ranging from .76 (EXTERN at third grade spring) to .92 (INTERN at fifth grade spring; Tourangeau, Nord, Lê, Sorongon, & Najarian, 2009). A detailed examination of the associations between these subscales and ADHD diagnosis measures in the ECLS-K data set showed that approaches to learning and externalizing behavior were the strongest predictors of diagnosis (Elder, 2010). The Approaches to Learning subscale includes items that rate the child's attentiveness, task persistence, eagerness to learn, learning independence, flexibility, and organization. The Externalizing Behavior subscale includes items that indicate the frequency with which a child argues, fights, gets angry, acts impulsively, and disturbs ongoing activities. Thus, we will use these particular variables as proxies for the inattentive (LEARN) and impulsive/hyperactive (EXTERN) symptoms of ADHD.

The ECLS-K data includes a number of academic performance measures. We utilize the IRT theta estimates for age-graded reading (READ) and mathematics (MATH) performance. The reliability of these scores ranged from .91 (for math at kindergarten fall) to .96 (for reading at kindergarten spring; Tourangeau et al., 2009).

Our illustrative analyses in the ECLS-K data will examine a number of moderation and mediation effects involving ADHD that have been previously identified in the literature. For example, we will examine how the association between ADHD symptoms and academic performance changes over time (or, alternately, as children grow older; Frazier, Youngstrom, Glutting, & Watkins, 2007; Kawabata, Tseng, & Gau, 2012). We will also examine potential moderating effects of gender on the association between ADHD and negative academic and social effects (Gaub & Carlson, 1997; Gershon & Gershon, 2002; Lahey et al., 2007; Lee, Lahey, Owens, & Hinshaw, 2008; Owens, Hinshaw, Lee, & Lahey, 2009).

Our primary mediation example is drawn from a recent article examining possible mediators of the association between ADHD and depression (Humphreys et al., 2013). Specifically, we will examine whether interpersonal skills and academic performance in reading and math mediate the association between our two ADHD symptom measures and depression. Such a mediated effect has been suggested as an explanation for the comorbidity between ADHD and depression that fits within a developmental psychopathology perspective (Drabick & Kendall, 2010). ADHD may lead to difficulties in social and academic functioning, and over time, failure to achieve success in these areas may lead to depression. Interpersonal skills and depression will be measured by the appropriate subscales of the Social Rating Scale. The Interpersonal Skills subscale (INTERP) rates a child's skill in forming and maintaining friendships, getting along with people who are different, comforting or helping others, expressing feelings and ideas in positive ways, and showing sensitivity to the feelings of others. The Internalizing Behavior subscale (INTERN) includes items rating the apparent presence of anxiety, loneliness, low self-esteem, and sadness. Admittedly, this is a confounded measure of depression, but it is the best proxy for this construct in the public use data set.

Because we wish to focus the reader's attention on similarities and differences between different methods of estimating longitudinal moderation and mediation, our analyses of the ECLS-K data are somewhat oversimplified. In particular, they do not address the complexly multilevel nature of the data, and they do not include sampling weights. Our analyses of the longitudinal ECLS-K data incorporate the nesting of multiple observations over time within individuals, but they do not explicitly model higher levels of nesting, for example individuals nested within classrooms, classrooms nested within schools, and schools nested within the counties that make up the primary sampling units of the study. Moreover, we did not make use of the multiple weights included in the ECLS-K data set designed to adjust for oversampling of Asians and Pacific Islanders and survey nonresponse (Tourangeau et al., 2009) to produce estimates that characterize the U.S. population of kindergarteners from the 1998–1999 cohort. As a consequence, similarity among students within classes and schools may produce some downward bias in the standard errors reported here, and our results are not necessarily representative of the whole of the U.S. population of students.

BASIC MODERATION

A situation in which the effect of one predictor varies depending on the value of another defines moderation. Moderation hypotheses are common in the social sciences and can be tested using a number of different frameworks, including one-way analysis of variance (ANOVA), regression, and structural equation modeling. ANOVA-based methods for testing moderation were developed prior to those in other areas; however, these methods are appropriate only for categorical predictors. Application of ANOVA-based methods to continuous predictors requires artificial dichotomization of the continuum into high and low groups, which is not ideal because it unnecessarily

reduces the power to detect the moderated effect and because it can produce spurious main and moderated effects under certain conditions (Maxwell & Delaney, 1993). Hence we will focus here on moderation within the regression framework.

The most typical approach to testing moderation effects with regression simply represents the synergistic effect between two predictors as a cross-product term and includes the cross-product in a regression equation simultaneously with its lower order component vectors. If we wish to test whether the effect of a predictor variable X on an outcome variable Y is moderated by Z, we would fit an equation:

$$Y = b_0 + b_1X + b_2Z + b_3XZ + e \qquad (21.1)$$

and examine the test of the b_3 coefficient to determine whether the effect of X on Y is moderated as predicted. The analysis is carried out in the same way regardless of whether X and Z are continuous measures or measured dichotomies. If one or more of the predictors is a categorical variable with more than two levels, a series of coded vectors will be required to represent it, and the test of the moderated effect will involve more than a single vector. In such a case, each vector coding the first predictor will need to be multiplied by each vector coding the second predictor, and the whole set of cross-products should be tested using a hierarchical regression approach to determine the R^2 change associated with the addition of the cross-products to the model (Cohen, Cohen, West, & Aiken, 2003).

Because cross-product vectors created to represent moderation effects are often highly collinear with their component vectors, it is typically recommended that the researcher center continuous predictor variables at their means prior to creating the cross-product. This will reduce the correlation between the vectors by eliminating what is known as nonessential collinearity (Aiken & West, 1991) among the predictor vectors, while leaving the associations between the predictors and the outcome unaltered. This can improve estimation as well as provide meaningful interpretations for lower order coefficients in the regression analysis. For example, if we left both X and Z in their raw metric in equation 21.1, the intercept of equation 21.1 would represent expected Y values for a subject with zeros on both the X and Z measures. Zero might not represent a particularly meaningful value on the X and Z scales, and it might actually be off-scale (e.g., if X and Z were 1–7 Likert-type measures). Similarly, the b_1 coefficient in equation 21.1 would represent the expected change in Y associated with a one unit increase in X when $Z = 0$ and the b_2 coefficient would represent the expected change in Y associated with a one unit increase in Z when $X = 0$. If we were to center each of the two predictors around their respective means, however, and create the cross-product from these centered variables, the intercept will represent the expected outcome for an "average" subject with mean values on both X and Z, the b_1 coefficient would represent change associated with X when Z is at its mean, and b_2 would represent change associated with Z when X is at its mean.

In the ECLS-K data, we tested whether the association between teachers' ratings of ADHD symptoms (LEARN and EXTERN, the predictors) and interpersonal skill (INTERP), reading (READ) and math performance (MATH), and internalizing behavior (INTERN) outcomes at kindergarten fall might be moderated by child gender (GENDER). The ADHD symptom predictors were centered at their means, and GENDER was coded such that males = 0 and females = 1. Regression results are reported in Table 21.1. As the coefficient b_1 indicates the association between the ADHD symptom predictor and the outcome at GENDER = 0 (male), the second column of Table 21.1 was labeled as the ADHD symptom effect for males (e.g., b_1 $\text{LEARN}_{\text{Male}}$). The coefficient b_2 in the third column (e.g., b_2 Gender) represents the difference between females and males in the outcome variable at the mean of the ADHD symptoms.

The fourth column of Table 21.1 presents the tests of the b_3 interaction coefficients in each of the various analyses. Only 2 of the 8 interactions were significant. Specifically, LEARN and EXTERN interacted with GENDER to predict READ and INTERP, respectively. These significant coefficients associated with the cross-product terms indicate that, in fact, the association between attentive symptoms and reading performance and the association between impulsive/hyperactive symptoms and interpersonal skill ratings is different for boys and girls. Both effects are slightly stronger for girls than for boys.

Higher order interactions involve synergistic effects among three or more variables. For example, we might predict that an XZ interaction is significant at only one of the two levels of a dichotomous variable W. Or if W is a continuous variable, we might hypothesize that the XZ interaction becomes more pronounced at higher levels of W. We would fit

$$Y = b_0 + b_1X + b_2Z + b_3W + b_4XZ + b_5XW + b_6ZW + b_7XZW + e \qquad (21.2)$$

TABLE 21.1 Coefficients and Standard Errors for Moderation Models

Outcome	b_1 $LEARN_{Male}$	b_2 GENDER	b_3 Interaction	$LEARN_{Female}$
INTERP	0.6475***	0.0395***	0.0063	0.6538***
	(.0074)	(0.0072)	(0.0108)	(0.0079)
READ	0.2966***	−0.0002	0.0314**	0.3280***
	(0.0077)	(0.0075)	(0.0112)	(0.0082)
MATH	0.3257***	−0.0816***	−0.0051	0.3206***
	(0.0068)	(0.0066)	(0.0099)	(0.0072)
INTERN	−0.2935***	0.0384***	0.0073	−0.2862***
	(0.0079)	(0.0078)	(0.0116)	(0.0084)
	b_1 $EXTERN_{Male}$	b_2 GENDER	b_3 Interaction	$EXTERN_{Female}$
INTERP	−0.5217***	0.0705***	−0.0541***	−0.5757***
	(0.0083)	(0.0083)	(0.0132)	(0.0103)
READ	−0.1095***	0.0551***	−0.0038	−0.1132***
	(0.0082)	(0.0081)	(0.0129)	(0.0100)
MATH	−0.1266***	−0.0289***	−0.0013	−0.1279***
	(0.0074)	(0.0074)	(0.0117)	(0.0091)
INTERN	0.2324***	0.0203*	−0.0116	0.2208***
	(0.0080)	(0.0080)	(0.0127)	(0.0099)

and examine the test of the b_7 coefficient associated with the three-way interaction to determine whether the interactive effect of XZ, in fact, varies depending on the value of W. Tests of whether the two gender interactions identified above might differ for families with varying incomes in the ECLS-K data found no significant three-way interactions when income was represented continuously (using the natural log of the WKINCOME variable) or dichotomously (using a median split on the WKINCOME variable), suggesting that the interactive effects remained stable across the range of income.

Significant interactions are typically followed by post hoc tests of simple effects. These examine the effect of one variable at particular values of the other. When both variables involved in the interaction are categorical, the post hoc analyses involve splitting the sample into groups defined by the levels of one variable and examining the effect of the other variable within each of these groups. Tests are typically conducted using model sums of squares produced in the group-specific analyses and residual sums of squares from the full model in which the interaction was initially estimated (Pedhazur, 1997). If one variable is continuous and the other categorical, the data splitting approach could be used to examine the effect of the continuous predictor within the various groups defined by the levels of the categorical variable, or the effect of the categorical variable could be examined at particular values of the continuous predictor. The Pothoff extension of the Johnson-Neyman technique can be used to find the ranges of the continuous predictor where the categorical variable has a significant effect, or specific values on the continuum can be selected at which to test the significance of the categorical distinction using a recentering strategy. Both approaches are discussed in detail in Aiken and West (1991).

When both variables involved in the interaction are continuous, the appropriate post hoc analyses involve testing the simple slope of one variable at particular values of the other. Ideally the simple slopes would be calculated and tested at meaningful points along the continuum of the other continuous variable. Often simple effects are simply examined at the mean and values ±1 standard deviation from the mean of a variable, which might be considered average, high, and low levels of the construct. Some care should be taken with this approach, however, as it is possible that one of the extreme values might fall outside the range of actual values if the variable is highly skewed.

The various terms in equation 21.1 can be regrouped as

$$Y = (b_0 + b_2Z) + (b_1 + b_3Z)X + e \qquad (21.3)$$

to show that that $b_1 + b_3Z$ provides an estimate of the simple slope of Y on X at a particular value of Z. The coefficient b_1 estimates the simple slope of Y on X when $Z = 0$, which will be at the mean of Z if centered predictors were used in the regression analysis. Values of Z (or centered Z, if centered predictor variables were used) can simply be substituted into equation 21.3 to calculate simple slopes at high and low Z values. The standard error of these simple slopes can be calculated using the typical equation for a linear combination of coefficient values:

$$SE_{b1+b3} = \sqrt{s_{b1}^2 + 2Zs_{b1,b3} + Z^2s_{b3}^2} \qquad (21.4)$$

where s^2_{b1} is the sampling variance of b_1, s^2_{b3} is the sampling variance of b_3, and $s_{b1,b3}$ is the sampling covariance between b_1 and b_3 (Aiken & West, 1991). The matrix of sampling covariances of the coefficient estimates can be obtained from most statistical programs that estimate ordinary least squares (OLS) regression. The square roots of the diagonal elements of this matrix are the standard errors of the coefficients reported in typical regression output.

We examined simple effects associated with the previously identified significant gender interactions in the ECLS-K data using a recoding strategy. Examination of the lower order coefficient for b_1 in Table 21.1 shows that the effect of approaches to learning on reading performance for boys (i.e., when GENDER = 0) is 0.2966, which is significant at $p < .0001$. This indicates that for every 1-point increase in the approaches to learning subscale score, we should expect a 0.2966 point increase in reading performance. We can then calculate the effect for girls by combining the b_1 and b_3 coefficients: $b_1 + b_3 = 0.2966 + 0.0314 = 0.3280$. So for girls, for every 1-point increase in the approaches to learning subscale score, we should expect a 0.328 point increase in reading performance. The significance of this value could be tested by computing the standard error of the linear combination $b_1 + b_3$ as described in equation 21.2. Alternately, GENDER could be recoded such that females = 0 and males = 1, the cross-product recomputed and the analysis rerun to produce an estimate of the LEARN effect for girls as the b_1 coefficient, shown in column 5 of Table 21.1. The standard error of this simple effect for girls is .0082 and the effect is significant at $p < .0001$. Similarly, we see that the simple effect of externalizing on interpersonal skill is –0.5217 for boys and –0.5757 for girls, both significant at $p < .0001$.

BASIC MEDIATION MODEL

The basic framework of a single mediator model at a single time point (i.e., a cross sectional mediation model) is shown in the two panels of Figure 21.1. The top panel shows the path diagram for a single predictor variable *X* and its effect on an outcome variable *Y*. The coefficient *c* on the path linking *X* to *Y* represents the total effect of the predictor on the outcome variable. The lower panel of Figure 21.1 shows the path diagram for this association when including the mediator variable. The coefficient *a* represents the effect of the predictor *X* on the mediator *M*, and the coefficient *b* represents the effect of the mediator *M* on the outcome *Y*,

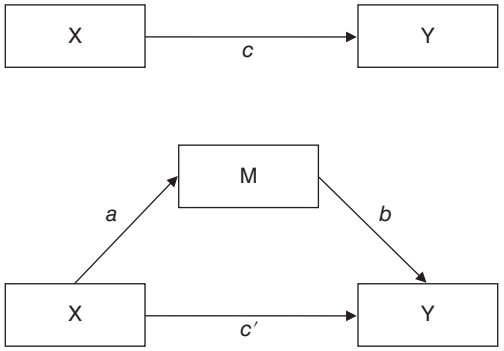


Figure 21.1 Path diagrams for the mediation model.

over and above the effect of the predictor. The coefficient c' on the path linking *X* to *Y* represents the direct effect of the predictor on the outcome variable, over and above the effect of the mediator.

The estimates of the path coefficients in these diagrams can be straightforwardly obtained through a series of three OLS regression analyses or equivalently through fitting two structural equation models to the manifest variables. The regression model corresponding to the top panel of Figure 21.1 would be

$$Y = b_0 + cX + e \tag{21.5}$$

The coefficient associated with the *X* predictor in this equation estimates the *c* path coefficient representing the total effect of *X* on *Y*. Two regression analyses are necessary to produce estimates of the coefficients in the lower panel of Figure 21.1. The first of these predicts the mediator variable from the initial predictor:

$$M = b_0 + aX + e \tag{21.6}$$

The coefficient associated with the *X* predictor in this equation estimates the *a* path coefficient linking the predictor to the mediator. The second regression equation predicts the outcome variable from the predictor and the mediator simultaneously:

$$\mathrm{Y} = b_0 + c'X + b\,M + e \tag{21.7}$$

The coefficient associated with the *X* predictor in this equation estimates the c' path coefficient representing the direct effect of *X* on *Y*. This is the effect of the predictor on the outcome that *cannot* be attributed to the mediator. The coefficient associated with the *M* mediator in this equation

estimates of the b path coefficient linking M to Y, over and above the effect of X.

Baron and Kenny's (1986) steps approach to testing mediation specifies a number of conditions that must be met to establish mediation:

1. The independent variable must affect the mediator.
2. The predictor must affect the outcome.
3. The mediator must affect the outcome.
4. The effect of the predictor on the outcome must be less when the mediator is present in the analysis than when it is absent.

Each of these steps involves coefficients from the three regression equations specified above. Step 1 requires examination of the significance of the a path coefficient from equation 21.6. Step 2 requires examination of the significance of the c path coefficient for the total effect from equation 21.5. Step 3 requires examination of the significance of the b path in equation 21.7. Then Step 4 involves comparing the c and c' coefficients from equations 21.5 and 21.7. If $|c'| < |c|$, the final step has been met, and according to this approach, mediation has been identified. Once mediation has been established, the significance of the c' coefficient in equation 21.7 can be examined to determine whether mediation is complete (i.e., no significant direct effect once the mediator has been taken into account) or partial (a significant direct effect persists).

Two point estimates of the mediated or indirect effect can be produced from the set of coefficient estimates in equations 21.5–21.7. The first such point estimate is that implied by Step 4 of the Barron and Kenny approach: $c - c'$. Using this difference as a point estimate of the mediated effect makes intuitive sense: when the effect of X on Y not due to the mediator (i.e., the direct effect or the c' estimate) is subtracted from the total effect of X on Y (i.e., the c estimate), the difference logically must be the effect of X on Y that is due to the mediator. A second point estimate of the mediated effect is the product of the a and b path coefficients. Using this product as a point estimate of the mediated effect makes intuitive sense for this function as well: if the a path coveys the influence of X on M and the b path similarly conveys the influence of M on Y, their product naturally conveys the influence of X (at the beginning of the system) to Y (at the end of the system). With simple single-level data and continuous M and Y measures, these two point estimates are algebraically equivalent (MacKinnon, Warsi, & Dwyer, 1995).

Several researchers have pointed out flaws in the steps approach to mediation. The first of these is that Step 2, which specifies that the total effect of X on Y must be significant, is not strictly necessary (MacKinnon et al., 2002; Zhao et al., 2010) for a mediated or indirect effect to be present. There are situations where the direct and indirect effects might have different signs (inconsistent mediation or suppression, see MacKinnon, Krull, & Lockwood, 2000), which might combine to produce a nonsignificant total effect that would not meet the criteria for Step 2. Such a situation may also prove problematic for Step 4, which specifies that the direct effect must be smaller in magnitude than the total effect. Consider, for example, a situation in which $a = 2$, $b = -1$, and $c' = 2$. The total effect c would be equal to the sum of the indirect effect ab and direct effect c'. This would be $c = ab + c' = 2 * -1 + 2 = 0$, and the condition of Step 2 would not be met. Moreover, in this case $|c'| > |c|$, as $2 > 0$, and the condition of Step 4 would also not be met, and a researcher applying the steps approach would erroneously conclude that no mediation was present.

Another apparent difficulty of strictly applying the steps approach is that at Step 4, any difference in the appropriate direction could be deemed evidence of mediation, but given ever-present sampling variability, a stricter criterion requiring a significant difference seems more appropriate. An approximate standard error can be derived via first-order Taylor series expansion or the multivariate delta method (MacKinnon, 2008):

$$s_{ab} = \sqrt{b^2 s_a^2 + a^2 s_b^2} \tag{21.8}$$

where s_a^2 and s_b^2 are the sampling variances of the regression estimates of a and b. These can be obtained by squaring the standard errors of a and b produced in all standard regression output or by examining the diagonal elements of the co/variance matrix of the estimates which can generally be requested as additional output in statistical software for regression estimation. This particular form of the standard error of the mediated effect is often referred to as the Sobel standard error (Sobel, 1982, 1986). Baron and Kenny (1986) suggested a slightly more complex approximate standard error, which can be derived via second order Taylor series expansion and represents the exact variance under the condition of independence:

$$s_{ab} = \sqrt{b^2 s_a^2 + a^2 s_b^2 + s_a^2 s_b^2} \tag{21.9}$$

A statistical test of the mediated effect can be conducted by dividing either the ab or the equivalent $c - c'$ estimate of

the mediated effect by the selected approximate standard error. This should be distributed as a z in large samples, and thus values more extreme than ±1.96 would be considered significantly different from zero at $p < .05$. However, this large sample z test assumes that the product ab is normally distributed, and the ab estimate of mediation generally is not (MacKinnon et al., 2007). This test has been found to be overly conservative (i.e., to have low power) in simulation studies (MacKinnon et al., 2002). This may or may not be problematic, depending on the particular circumstances in which the test is employed. If a researcher uses this test in an existing data set and finds a significant mediated effect, the fact that the test is conservative and therefore somewhat underpowered does not diminish the significance of the finding in any way, although a confidence interval formed around the significant point estimate of the mediated effect will still be too wide and indicate more uncertainty in the value than is truly warranted. In contrast, a nonsignificant result might indicate a null effect or might simply be the result of an underpowered test. In addition, using this test to determine the necessary number of subjects for a future study in an a priori power analysis would result in an unnecessarily large sample size recommendation.

Because of this distributional difficulty, several alternative approaches have been suggested. One such alternative involves looking not at the distribution of ab, but at the product $z_a z_b$, where z_a and z_b are z scores formed as the ratios of the coefficient estimates to their standard errors. The $z_a z_b$ product is known to have a complex nonnormal distribution, but values obtained in such an analysis can be compared to tabled values (Meeker, Cornwell, & Aroian, 1981) and asymmetric confidence limits can be obtained from software written especially for this purpose (MacKinnon et al., 2007; Tofighi & MacKinnon, 2011). An alternate approach involves a resampling technique known as bootstrapping. Bootstrapping involves repeated resampling with replacement (Efron & Tibishirani, 1993). For example, if a researcher is bootstrapping cases from a sample of 100, many (typically over 1000) new samples of 100 cases are drawn from the original sample. These samples may differ from the original in that a given case may appear more than once in each bootstrap sample. Then the statistic of interest may be calculated for each bootstrap sample. These are ordered and values of the statistic associated with the desired percentiles (often the 2.5th and 97.5th for a 95% confidence interval) define the lower and upper limits of a confidence interval. This procedure may be refined using a procedure known as bias correction to adjust for the fact that the statistic obtained in the original analysis may not fall at the 50th percentile of the distribution of bootstrap values (Efron & Tibishirani, 1993). In the case of a mediated effect, the cases themselves are resampled and ab estimates produced from regressions involving X, Y, and M in each of the new bootstrap samples are obtained (MacKinnon et al., 2004; Preacher & Hayes, 2004).

The simple single-mediator model in Figure 21.1 can be expanded to multivariate situations that include multiple predictors and multiple mediators. With multiple predictor and mediator variables, it is common to run an initial set of analyses examining mediated effects of each predictor–mediator pairing individually, then to run a combined model to examine the *unique* effects of each variable over and above the others. For example, if we were to expand Figure 21.1 to include three mediators, equation 21.5 examining the effect of the single predictor on the single outcome would remain unchanged, producing a single estimate of the total effect c. The X to M associations examined with equation 21.6 would require three separate regression equations, each predicting a single mediator from the predictor X, producing three unique a coefficients, a_1, a_2, and a_3. Equation 21.7 would need to include all three mediators simultaneously along with the predictor to produce three unique b coefficients b_1, b_2, and b_3 and a single estimate of the direct effect c'. The $c - c'$ point estimate of mediation in this case represents the total mediated effect and the a_1b_1, a_2b_2, and a_3b_3 point estimates represent specific mediated effects unique to each of the three mediators.

A set of cross sectional mediation analyses was conducted in the ECLS-K data. These analyses tested whether the effect of ADHD symptoms on depression was mediated by academic performance and interpersonal skill at the initial kindergarten fall assessment. Specifically, teacher ratings of approaches to learning and externalizing behavior were treated as initial predictor variables, theta scores on math and reading and teacher ratings of interpersonal skill served as mediators, and teacher ratings of internalizing behavior were the outcome. Regression equations similar to those in equations 21.5 to 21.7 were first fitted to each predictor–mediator pair separately, then all predictors and mediators were entered simultaneously into a multiple mediator model. The results of these analyses are summarized in Table 21.2.

The third and fourth columns of Table 21.2 present a and b coefficient estimates, with their standard errors below in parentheses. The results of these analyses show that both approaches to learning and externalizing behaviors significantly predict each of the three mediators, and each of the three mediators significantly affects the internalizing

TABLE 21.2 Coefficients, Standard Errors, Tests of Mediation, and Bootstrap Confidence Intervals for Single- and Multiple-Mediator Models

		Single-mediator models				Multiple-mediator models			
X	M	*a*	*b*	Med(*Z* test)	99% bootstrap CI: LL, UL	*a*	*b*	Med (*Z* test)	99% bootstrap CI: LL, UL
LEARN	INTERP	0.6564***	−0.1515***	−0.0994***	−0.1145,	0.5213***	−0.1282***	−0.0668***	−0.0808,
		(0.0053)	(0.0083)	(0.0055)	−0.0842	(0.0058)	(0.0088)	(0.0046)	−0.0550
LEARN	READ	0.3115***	−0.0234**	−0.0073**	−0.0147,	0.3427***	0.0042	0.0014	−0.0085,
		(0.0055)	(0.0081)	(0.0025)	−0.0014	(0.0064)	(0.0115)	(0.0039)	0.0112
LEARN	MATH	0.3113***	−0.0422***	−0.0131***	−0.0212,	0.3405***	−0.0626***	−0.0213***	−0.0334,
		(0.0049)	(0.0091)	(0.0028)	−0.0072	(0.0056)	(0.0130)	(0.0044)	−0.0094
EXTERN	INTERP	−0.5545***	−0.2348***	0.1302***	0.1183,	−0.2755***	−0.1282***	0.0353***	0.0280,
		(0.0064)	(0.0073)	(0.0043)	0.1411	(0.0060)	(0.0088)	(0.0025)	0.0433
EXTERN	READ	−0.1197***	−0.1315***	0.0157***	0.0137,	0.0638***	0.0042	0.0003	−0.0016,
		(0.0062)	(0.0076)	(0.0012)	0.0199	(0.0067)	(0.0115)	(0.0007)	0.0023
EXTERN	MATH	−0.1227***	−0.1699***	0.0208***	0.0165,	0.0596***	−0.0626***	−0.0037***	−0.0063,
		(0.0056)	(0.0084)	(0.0014)	0.0236	(0.0059)	(0.0130)	(0.0009)	−0.0017

outcome. The *ab* point estimates and the Sobel standard errors of each mediated effect appear in the fifth column of the table. When examined in separate analyses, each of these mediated effects is significant. In this particular case, the bootstrap 99% confidence intervals in the sixth column of the table are very close to those that would be computed using the normal theory and Sobel standard error provided in the previous column. The fact that zero is not included in any of these confidence intervals indicates significance at $p < .01$ and corresponds to the *z* test results.

The remaining columns of the table present the results of a multiple predictor–multiple mediator analysis in which all predictors and mediators were analyzed simultaneously. A path diagram for this particular model is shown in Figure 21.2. The two initial predictor variables, approaches to learning and externalizing behavior, are highly negatively correlated ($r = –.50$, $p < .0001$). The two academic mediators are strongly positively correlated (.77, $p < .0001$), and these two measures are moderately positively correlated with interpersonal skill (.27 for math and .25 for reading, both *p*s < .0001). The *a* path estimates representing the unique effects of each of these predictors (over and above the others) on each of the three mediators appear in column 7 of Table 21.2, and all six of these remain significant, though the effects of externalizing behavior seem somewhat attenuated (when compared with the separate analyses reported in column 3 in the table), and the effects of externalizing on reading and math have, in fact, changed sign. The *b* path estimates representing unique effects of a particular mediator are provided in column 8. When all variables are in the model simultaneously, the path representing the unique effect of reading on internalizing is not significant (over and above the effects of math, interpersonal skill, approaches to learning, and externalizing behavior). The *b* paths from interpersonal skill and math to internalizing have also reversed sign in the analyses that simultaneously include the externalizing predictor. The *z* tests of point estimates of the mediated effects in column 9 and the bootstrap confidence intervals in column 10 indicate that the effects of approaches to learning and externalizing behavior on internalizing behavior are significantly—and uniquely—mediated by math and interpersonal skill but not reading.

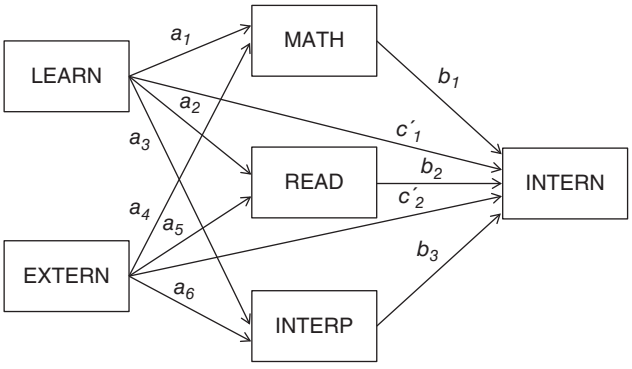


Figure 21.2 Path diagram for the two-predictor, three-mediator in the ECLS-K data.

COMBINING BASIC MEDIATION AND MODERATION

Several researchers have described approaches that combine moderation and mediation into a single framework to examine more complex associations among sets of variables (e.g., Edwards & Lambert, 2007; Fairchild & MacKinnon, 2009; Hayes, 2013; MacKinnon, 2008; Muller, Judd, & Yzerbyt, 2005; Preacher, Rucker, & Hayes, 2007). Such approaches typically consider situations involving both

moderated mediation and mediated moderation. Moderated mediation, or moderation of an indirect effect (Fairchild & MacKinnon, 2009), is moderation of the *ab* estimate of a mediated effect by a fourth variable, *Z*. *Z* could exert its moderating influence on the *a* path from *X* to *M*, on the *b* path from *M* to *Y*, or both (though it should be noted that circumstances exist when moderation of both paths does not necessarily imply moderation of the *ab* product). Mediated moderation, or the mediation of a moderator effect (Fairchild & MacKinnon, 2009), involves an exploration of the mechanism through which an interaction between two predictors *X* and *Z* (i.e., the *XZ* cross-product) exerts its effect on *Y*.

The framework described in Fairchild and MacKinnon (2009) attempts to unify the various proposed approaches by providing a single comprehensive presentation that subsumes the specific differing approaches as special cases. Figure 21.3 presents the path diagram for this combined framework. The regression equations necessary to produce the various path coefficients are straightforward generalizations of each of the three regression equations necessary for a simple mediation analysis. For example, equation 21.5 in which the outcome is regressed on the focal predictor becomes

$$Y = b_0 + c_1 X + c_2 Z + c_3 XZ + e \tag{21.10}$$

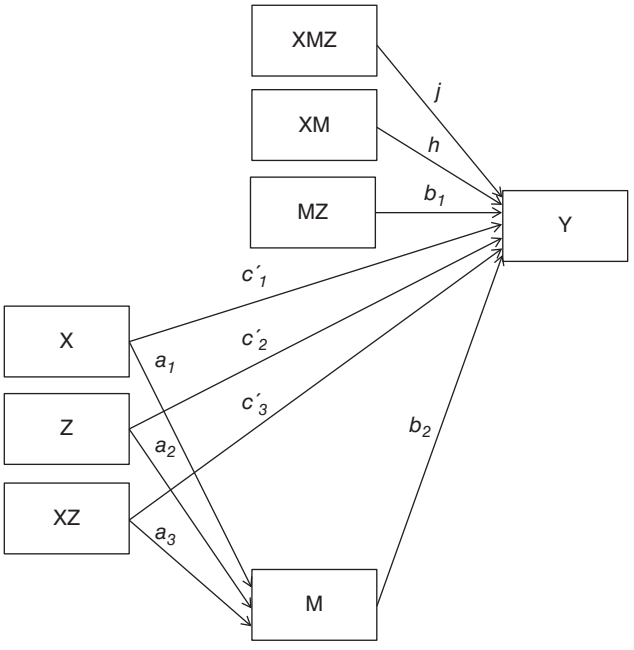


Figure 21.3 Path diagram of the Fairchild and MacKinnon (2009) approach to moderated mediation and mediated moderation.

The *c* coefficients in this equation thus provide total effect analogs for each of two predictors, *X* and *Z*, as well as their interaction *XZ*. The coefficient c_1 is the effect of *X* on *Y* when $Z = 0$, the coefficient c_2 is the effect of *Z* on *Y* when $X = 0$, and the coefficient c_3 is the interaction of *X* and *Z* on the outcome. Typically, all predictor variables in such an analysis would be centered around their means prior to estimation, which would make the c_1 and c_2 coefficients the average effects of *X* and *Z* on the outcome, respectively.

The generalization of equation 21.6 is

$$M = b_0 + a_1 X + a_2 Z + a_3 XZ + e \tag{21.11}$$

This equation provides *a* path coefficient estimates for *X*, *Z*, and their interaction. The coefficient a_1 is the average effect of *X* on the mediator *M*, the coefficient a_2 is the average effect of *Z* on the mediator, and the coefficient a_3 is the interaction of *X* and *Z* on the mediator variable.

The generalization of equation 21.7 is

$$Y = b_0 + c'_1 X + c'_2 Z + c'_3 XZ + b_1 M + b_2 MZ + hXM + jXMZ + e \tag{21.12}$$

The c' coefficients in this equation provides direct effect analogs for *X*, *Z*, and their interaction *XZ*. The coefficients c'_1 and c'_2 are the average direct effects of *X* and *Z* on the outcome, and the coefficient c'_3 is the direct effect of the *XZ* interaction on the outcome. The b_1 coefficient is the average effect of *M* on *Y*, and the remaining paths b_2, *h*, and *j* estimate possible moderation of this effect by *Z*, *X*, and the *XZ* interaction, respectively.

Moderation of an indirect effect, in this framework, would involve the a_3 or b_2 coefficients. Fairchild and MacKinnon (2009) suggested that a joint test of these two coefficients may be acceptable to identify moderated mediation (i.e., mediation is deemed significant if both the *a* and *b* paths are significant). When the moderator is dichotomous, this effect can be more rigorously tested by calculating the *ab* point estimate of the mediated effect within each group and testing the significance of the difference between them using a pooled standard error. Details of this procedure as well as that for a continuous moderator are discussed in Fairchild and MacKinnon (2009). Mediation of a moderator effect could be estimated as the $a_3 b_1$ product.

Table 21.3 presents the results of six analyses examining whether (1) the mediated effects of ADHD symptoms on internalizing behavior through academic performance and

TABLE 21.3 Coefficients, Standard Errors, and Tests of Moderated Mediation and Mediated Moderation

X	*M*	a_3	b_2	b_1	Med_{Male}	Med_{Female}	Moderated mediation test	Mediated moderation test
LEARN	INTERP	0.0018	−0.0239	−0.1551***	−0.0948***	−0.1051***	0.0103	−0.0003
		(0.0102)	(0.0084)	(0.0081)	(0.0078)	(0.0078)	(0.0110)	(0.0016)
LEARN	READ	0.0324**	−0.0365*	−0.0281***	−0.0033	−0.0122**	0.0089†	−0.0009*
		(0.0109)	(0.0165)	(0.0082)	(0.0034)	(0.0038)	(0.0051)	(0.0004)
LEARN	MATH	−0.0042	−0.0260	−0.0407***	−0.0086*	−0.0171***	0.0086	0.0002
		(0.0097)	(0.0176)	(0.0088)	(.0040)	(0.0044)	(0.0060)	(0.0004)
EXTERN	INTERP	−0.0545***	−0.0326*	−0.2399***	0.1164***	0.1450***	−0.0286***	0.0131***
		(0.0128)	(0.0142)	(0.0071)	(0.0058)	(0.0064)	(0.0086)	(.0.0031)
EXTERN	READ	−0.0031	−0.0394*	−0.1401***	0.0127***	0.0171***	−0.0044†	0.0004
		(0.0127)	(0.0155)	(0.0078)	(0.0015)	(0.0020)	(0.0025)	(0.0018)
EXTERN	MATH	−0.0009	−0.0384*	−0.1719***	0.0191***	0.0246***	−0.0055†	0.0002
		(0.0116)	(0.0164)	(0.0082)	(0.0019)	(0.0025)	(0.0030)	(0.0020)

interpersonal skill might be moderated by gender, and (2) the moderation effect of gender on the association between ADHD symptoms and internalizing might be mediated by academic performance and/or interpersonal skill. The a_3 and b_2 coefficients that suggest moderated mediation appear in columns 3 and 4, and the statistical test appears in column 8. The joint test would suggest moderation of the mediated effect by gender for the effect of LEARN on INTERN through READ and the effect of EXTERN on INTERN through INTERP. The test of the difference between the *ab* estimates of mediation for boys and girls indicates that the latter effect is significant at $p < .001$, but the former is only marginally significant. The a_3 and b_1 coefficients that comprise the a_3b_1 estimate of mediated moderation appear in columns 3 and 5 of Table 21.3, and the test of mediated moderation is provided in column 9. These results indicate that the interactive effect of LEARN and GENDER on INTERN is mediated by reading performance, and the interactive effect of EXTERN and GENDER on INTERP is mediated by interpersonal skill.

LONGITUDINAL DATA

The discussion of methods to detect mediation and moderation above are specific to cross sectional data. The introduction of longitudinal data, with repeated measurements of the same variables over time, provides several benefits that may help to elucidate the associations of interest to a researcher. Many of these are discussed in MacKinnon (2008), with attention to how these are relevant to mediated effects in particular. First, longitudinal data provide information about the temporal precedence of the variables involved in the association. This is especially salient when the researcher wishes to establish causal pathways in a mediation model. Causes must precede their effects, and mediation models necessarily involve either implicit or explicit causal claims. In cross sectional data, if the initial predictor is randomly assigned (e.g., randomization to treatment and control groups), then logic dictates that *X* precedes *M* and *Y* in the model. For nonrandomly assigned variables and for the *M* to *Y* relation, however, it is often difficult to establish the ordering of the variables, and thus it is common to acknowledge the possibility of reverse or reciprocal causation in such situations. With repeated measurements of the various constructs in longitudinal data, however, the researcher can explore lagged associations and changes in variables over time. If *X* at Time 1 predicts *M* at Time 2, which in turn predicts *Y* at Time 3, it is more likely that the earlier measures cause the later measures than vice versa (though it is still possible that some third variable causes both the earlier and later measures). Testing moderation does not necessarily involve ordering variables as in a mediation model, but here still it can be useful to explore lagged associations and change over time. If an interaction between two variables at Time 1 predicts an outcome at a later time point or change in the outcome over time, this makes a stronger argument for possible causality than examination of all variables cross sectionally.

A second benefit of the repeated measurement of constructs in longitudinal data is that it allows the researcher to disentangle processes occurring within and between individuals. In cross sectional data, the only differences that can be detected are those between different individuals at the single time point. With two or more observations, however, it is possible to calculate intraindividual change, as well as differences between individuals. It may well be that the predictors of individual change are quite different from the

predictors of differences between individuals, hence disentangling the within-person and between-person effects will provide a more fully accurate model of how real-world processes operate to produce their effects on behavior.

A third benefit of longitudinal data is that having repeated measurements of variables allows the researcher to rule out some possible alternative explanations for the effect of one variable on another. In cross sectional data, it is possible to argue that virtually any association between two variables is, in fact, due to an unmeasured and omitted third variable that affects them both. With longitudinal data, however, a researcher can covary previous measures of an outcome construct when predicting the current measure of that construct. This sort of autoregressive effect essentially removes the influence of any stable person characteristics that might influence the current outcome measure, as each individual serves as his or her own control. While one could still argue that some characteristic of the individual influences both the Time 1 and Time 2 measurement of a construct, it would be difficult to assert that this stable characteristic influences change in the construct over time. However, it is still possible that some time-varying confound provides a reasonable alternative account of a significant effect, so though use of a longitudinal design guards against some kinds of third variable explanations, it is not a universal panacea.

Another benefit of longitudinal data was noted by Gollob and Reichardt (1991), who point out that the effect that one variable has on another may not be instantaneous. It may take some time for certain types of variables to exert their effects, which cross sectional data may not be able to capture. Examining lagged associations in longitudinal data or changes over time may be more illuminating in such cases.

The concepts of stability, stationarity, and equilibrium are helpful in describing longitudinal data, longitudinal relationships, and the models used to examine them. Stability refers to the extent to which some feature of the data stays the same across time. That feature may be a variable mean, a rank ordering of variable values, a trend over time, or a periodic process. Stationarity refers to the extent to which relations between variables stay the same across time. Equilibrium refers to the extent to which the pattern of variances and covariances among variables stays the same over time. Equilibrium is related to both stability and stationarity, but a stable and stationary model is not necessarily at equilibrium (Cole & Maxwell, 2003). A cross sectional model can accurately assess relations among variables only after equilibrium has been reached (Maxwell & Cole, 2007). In longitudinal data, stability can be assessed by measuring the dependency of an observation at a given time point on its value at the previous time point; stationarity can be evaluated by assessing invariance of relations among variables across time points; and some information about equilibrium can be obtained by examining the variance–covariance matrix among the variables across time points (MacKinnon, 2008).

The analysis of longitudinal data requires an analytic framework that can accommodate the unique features of such a data structure, most importantly the dependence that arises from the within-person stability of repeated measurements of an outcome measure assessed at multiple time points. Such dependence violates assumptions regarding independent errors that are necessary for many traditional statistical model techniques (e.g., GLM), resulting in downwardly biased standard errors, inflated test statistics, and inflation of the Type I error rate when such methods are improperly applied to longitudinal data. Two alternative analytic frameworks—multilevel modeling and structural equation modeling—are able to appropriately model the within-person similarity and thus are frequently used to analyze data from developmental studies. Below we briefly discuss these two frameworks, how cross sectional moderation and mediation are incorporated into each, and what types of models are commonly used to analyze longitudinal data with each approach.

MULTILEVEL MODELING

When data are nested or clustered, application of standard regression analysis techniques can be problematic. For example, when individual subjects are nested within groups, it is quite possible that outcome measures for individuals within the same group may be more similar than a random grouping of individuals would be. This similarity could arise from a number of different sources, including shared group experiences (e.g., students within the same elementary school classroom all share the same teacher), interaction among group members (e.g., students within the classroom talk to each other, which might result in more similar attitudes), or nonrandomly distributed background variables (e.g., students within a school may all live in the surrounding neighborhood, which may be relatively homogeneous in terms of socioeconomic status). Such data would have a positive intraclass correlation (ICC). Such within-group similarity (or between-group mean differences) would not be modeled within a single-level regression analysis, resulting in downwardly biased standard errors and overly large

test statistics, ultimately increasing the probability of making a Type I error (alpha inflation) and concluding that a significant effect exists more often than warranted (Barcikowski, 1981). Multilevel models—also known as mixed models (Laird & Ware, 1982), random coefficient models (Longford, 1993), or hierarchical linear models (Raudenbush & Bryk, 2002)—are explicitly designed to accommodate such nested data structures.

A multilevel model can be conceptualized as a series of models at the various levels of the data. For students nested within schools, the lower level (L1) model for students would be specified as

$$\text{L1:} \qquad Y_{ij} = \beta_{0j} + \beta_{1j}X_{ij} + r_{ij} \tag{21.13}$$

where Y_{ij}, X_{ij}, and r_{ij} are the outcome score, predictor score, and residual, respectively, for individual i within school j. β_{0j} and β_{1j} are intercept and slope values for school j. Thus, one can think of this part of the model as a series of regressions within each of the j groups.

Each of the coefficients of the L1 model is treated as a criterion measure in higher or upper level (L2) models. In the students within schools example, these would be specified at the school level:

$$\begin{aligned} \text{L2:} \qquad \beta_{0j} &= \gamma_{00} + u_{0j} \\ \beta_{1j} &= \gamma_{10} + u_{1j} \end{aligned} \tag{21.14}$$

The β_{0j} intercept for school j is a function of the overall intercept for the sample (γ_{00}) plus a group-specific deviation u_{0j}. Similarly the β_{1j} slope of Y on X is a function of the overall slope for the sample (γ_{10}) plus a group specific deviation u_{1j}. Including error terms at both L1 (r_{ij}) and L2 (the u's) appropriately models the within-group similarity, thus providing accurate tests of significance of predictor effects.

The multiple equations at L1 and L2 may be algebraically combined into a single equation:

$$Y_{ij} = \gamma_{00} + \gamma_{10}X_{ij} + u_{0j} + u_{1j}X_{ij} + e_{ij} \tag{21.15}$$

The single-equation form of the model is typically estimated using an iterative procedure that alternates between generalized least squares estimates of fixed effects (γ_{00} and γ_{10}) and maximum likelihood estimates of the variance components. The L2 u error terms are assumed to be distributed $\sim \boldsymbol{N}(\mathbf{0}, \boldsymbol{\tau})$, and thus the variance components include τ_{00} (the variance of u_{0j}), τ_{11} (the variance of u_{1j}), τ_{01} (the covariance between u_{0j} and u_{1j}), as well as σ^2 (the variance of r_{ij}). Estimating the combined equation rather than performing the two-step procedure implied by the multiple equation form (known as slopes and intercepts-as-outcomes; see Burstein & Miller, 1980) allows for more appropriate weighting of large versus small groups in the analysis and permits estimation even when some groups are rank deficient (i.e., when some groups contain too few individuals to run the within-group regression analysis). This particular analysis, also known as a random coefficient regression model (Raudenbush & Bryk, 2002), essentially describes the distribution of intercepts and slopes across groups, estimating an average intercept γ_{00}, the variability of intercepts τ_{00}, an average slope γ_{10}, and the variability of slopes τ_{11}. These averages are not the simple arithmetic averages that one could obtain from the two-step procedure but rather an Empirical Bayesian weighting that shrinks less reliable estimates (i.e., those from smaller groups) toward common group estimates to obtain a more representative measure of central tendency.

Another common application of multilevel modeling involves repeated measures data where the same constructs are assessed on multiple occasions for a set of individuals. In this scenario, L1 is the repeated measures (within-person) level, and L2 is the individual (between-person) level. The multiple outcome measurements provided by a given individual are more likely to be similar than a random grouping of measurements, and thus we would expect a positive ICC value. When the repeated measurements are considered simple replications and no systematic trend across time is expected, the multilevel framework discussed above can be readily adapted. Such analyses are common, for example, in daily diary studies (see Bolger & Laurenceau, 2013) where days are nested within individuals. Often no trends are necessarily expected across the relatively brief spans of such studies; the multiple days are included simply to obtain many measures of predictor and outcome variables and to allow the researcher to distinguish between within- and between-person effects.

The data for a multilevel analysis of repeated measures is typically organized as a long file with one observation for each of the assessments. The multiple-equation form of the model would be

$$\begin{aligned} \text{L1:} \qquad Y_{ti} &= \pi_{0i} + \pi_{1i}X_{ti} + e_{ti} \\ \text{L2:} \qquad \pi_{0i} &= \beta_{00} + r_{0i} \\ \pi_{1i} &= \beta_{10} + r_{1i} \end{aligned} \tag{21.16}$$

One might think of the first equation as a regression across timepoints t for each individual i, providing individual-specific intercept and slope coefficients that serve as outcomes in the individual-level L2 equations.

The single-equation form of the model is

$$Y_{ti} = \beta_{00} + \beta_{10}X_{ti} + r_{0i} + r_{1i}X_{ti} + e_{ti} \tag{21.17}$$

This model provides estimates of the overall expected value of the outcome measure when the X predictor is equal to zero (β_{00}), the variance in this value across individuals ($\tau_{00} = \text{var}(r_{0i})$) the overall association between X and Y (β_{10}), the variance in this association across individuals ($\tau_{11} = \text{var}(r_{1i})$), as well as the residual variance among time points within an individual ($\sigma^2 = \text{var}(e_{ij})$).

Moderation in Multilevel Models

The aforementioned multilevel equations could be expanded to accommodate a higher level predictor of both intercepts and slopes. This would be a group-level predictor in the scenario with individuals nested within groups and an individual-level predictor in the repeated measures scenario. Going forward with the repeated measures case, the lower level equation would remain unchanged:

$$\text{L1:} \qquad Y_{ti} = \pi_{0i} + \pi_{1i}X_{ti} + e_{ti} \tag{21.18}$$

and each of the L2 equations would be expanded to incorporate the individual-level predictor:

$$\begin{aligned} \text{L2:} \qquad \pi_{0\text{i}} &= \beta_{00} + \beta_{01}W_i + r_{0i} \\ \pi_{1i} &= \beta_{10} + \beta_{11}W_i + r_{1i} \end{aligned} \tag{21.19}$$

The single-equation form of the model becomes

$$Y_{ti} = \beta_{00} + \beta_{01}W_i + \beta_{10}X_{ti} + \beta_{11}W_iX_{ti} + r_{0i} + r_{1i}X_{ti} + e_{ti} \tag{21.20}$$

This model estimates four fixed effects. The β_{00} estimate is the expected value of the outcome when both the L1 and L2 predictors are equal to 0. The β_{01} estimate is the effect of the individual-level predictor W on the outcome when the repeated measures predictor X is equal to 0 (i.e., the simple effect of W). The β_{10} estimate is the overall effect of the repeated measures predictor X on the outcome when the individual-level predictor W is equal to 0 (i.e., the simple effect of X). The β_{11} estimate is the cross-level interaction between the repeated measures predictor X and the individual level predictor W. Thus this estimate represents (1) the extent to which the association between X and Y changes for each unit increase in W, and/or (2) the extent to which the association between W and Y changes for each unit increase in X. Cross-level interactions are not entirely unique to multilevel models, but they arise most naturally within the multiple equation form within this framework. The t test of the β coefficient associated with the cross-product (i.e., β_{11}) is thus often an appropriate test of moderation when predictor and moderator variables are assessed at different levels of a hierarchical data structure.

In addition to cross-level interactions, multilevel models can accommodate single-level interactions at either the lower or the higher level of the data structure. The techniques involved are essentially identical to the cross-product methods used in single-level regression models. Moderation of the effect of a repeated measures variable by another repeated measures variable would require expanding the L1 equation to include the second variable and the L1 cross-product vector:

$$\text{L1:} \quad Y_{ti} = \pi_{0i} + \pi_{1i}X_{ti} + \pi_{2i}Z_{ti} + \pi_{3i}X_{ti}Z_{ti} + e_{ti} \tag{21.21}$$

and additional L2 equations would need to be specified for the two new L1 coefficients:

$$\begin{aligned} \text{L2:} \qquad \pi_{2i} &= \beta_{20} + r_{2j} \\ \pi_{3i} &= \beta_{30} + r_{3j} \end{aligned} \tag{21.22}$$

The β_{20} coefficient represents the simple effect of the Z predictor when X equals 0, and the test of the β_{30} coefficient tests the overall moderated effect. Note that if an L2 predictor was added to the L3 equation for π_{3i}, the associated coefficient would represent the three-way interaction among L1 X, L1 Z, and the L2 predictor. The fact that this is a three-way interaction would be apparent in the single-equation form of the model.

Single-level moderation at L2 would involve expanding (at least) the first of the L2 equations (which predicts the L1 intercept) to include the second individual-level predictor and the cross-product vector:

$$\text{L2:} \quad \pi_{0i} = \beta_{00} + \beta_{01}W_i + \beta_{02}Z_i + \beta_{03}W_iZ_i + r_{0i} \tag{21.23}$$

The β_{02} coefficient represents the simple effect of Z when W equals 0, and the test of the β_{03} coefficient tests the moderated effect. Note that if the individual-level Z predictor and the WZ cross-product are added to other L2 equations

(which predict L1 slope coefficients associated with particular L1 variables) the associated coefficients would represent three-way interactions among L2 *W*, L2 *Z*, and the particular L1 variable whose L1 parameter estimate serves as the outcome for the L2 equation. Calculation, testing, and plotting simple slopes for interactions in multilevel models is carried out in exactly the same way as in single-level regression models.

The issue of centering, which features prominently in discussions of interactions within the single-level regression framework, is no less important when considering multilevel models. Centering serves the same two purposes, providing meaningful interpretations of lower order coefficients and increasing the stability of model estimation. In fact, the latter probably provides greater benefit in multilevel models than in single-level regression analyses, as estimation is much more difficult in the multilevel case. When considering an interaction between two L2 predictor variables, centering is essentially carried out just as in single-level regression: subtracting the mean of each L2 predictor from each observation of that predictor and creating the cross-product from these centered variables reduces nonessential collinearity and allows interpretation of lower order effects of one variable at the mean of the other. One slight wrinkle does emerge: the analyst must decide whether to calculate the mean of the L2 variable to use in the centering as a simple unweighted function, counting each L2 score equally, or as a weighted function which adjusts for the number of L1 units within each L2 unit (i.e., individuals within groups or observations within individuals). Of course, there will be no difference between the two when the number of L1 units per L2 unit is identical across all groups/individuals.

When one or both of the variables involved in the interaction is at L1, however, centering becomes a slightly more complex issue. Consider first the situation with individuals nested within groups. Kreft, de Leeuw, and Aiken (1995) discussed two possible means to use in centering L1 predictors. Centering around the grand mean (CGM) simply involves subtracting the single grand mean value of the L1 predictor from each predictor observation. Centering within contexts (CWC) involves subtracting the mean of each group from the predictor observations within each group. Thus while GGM is a simple linear transformation of the raw variable score, CWC is not. Both provide meaningful (though different) zero points at which to interpret lower order coefficients, and both confer some computational advantage, though this is typically greater for CWC, which orthogonalizes effects at L1 and L2. Additional discussion of centering issues for multilevel models with individuals nested within groups can be found in Hofmann and Gavin (1998) and Enders and Tofighi (2007). Repeated measurements of time-varying predictors can be centered with CGM or CWC analogs that subtract the mean value of the L1 predictor for the entire sample or a person-specific mean from each observed score. Hoffman and Stawski (2009) explain how such centered L1 variables can be entered into multilevel models that simultaneously include the means as L2 predictors to disentangle between-person and within-person effects in repeated measures data.

Mediation in Multilevel Models

Mediation in multilevel models is tested in essentially the same matter as for single-level regression models. Each of the three regression models necessary for producing the coefficients for a mediation analysis may be recast as a multilevel model, including an L2 intercept error term to appropriately model clustering of individuals within groups or repeated measures within individuals. Details are provided in Krull and MacKinnon (1999, 2001). The multiple equation forms of such models would differ depending on the level of measurement of *X* and *M*. Krull and MacKinnon (2001) adopted a model naming convention related to the sequential ordering of variables in a mediation model, where $X \rightarrow M \rightarrow Y$. In a $1 \rightarrow 1 \rightarrow 1$ model, the *X*, *M*, and *Y* variables would each be measured at the lower level of the data hierarchy. So the initial variable, the mediator, and the outcome variable would all be characteristics of individuals in multilevel data with individuals nested within groups or time-varying characteristics measured on multiple occasions over a period of time in multilevel data with repeated measures nested within individuals. In a $2 \rightarrow 1 \rightarrow 1$ model the initial predictor would be a L2 variable, while the mediator and outcome would be L1 constructs. In a $2 \rightarrow 2 \rightarrow 1$ model, both the initial predictor and mediator are measured at L2, while the outcome is at L1. Models including a link from a lower level to a higher level are not estimable within a multilevel modeling framework, but these could potentially be handled within a multilevel structural equation model (Preacher, Zyphur, & Zhang, 2010).

Fitting a $2 \rightarrow 2 \rightarrow 1$ mediation model would involve running a regression model on L2 data to obtain an estimate of the *a* path linking predictor to mediator and a multilevel model of the form

$$\begin{aligned} \text{L1:} \quad & Y_{ij} = \beta_{0j} + e_{ij} \\ \text{L2:} \quad & \beta_{0j} = \gamma_{00} + c' X_j + b M_j + u_{0j} \end{aligned} \tag{21.24}$$

to obtain an estimate of the *b* coefficient. These would be multiplied to produce the *ab* estimate of the mediated effect, and this can be tested using the Sobel standard error described earlier for single-level mediation models or more precisely by developing bootstrap confidence intervals (Pituch, Stapleton, & Kang, 2006).

For a $2 \rightarrow 1 \rightarrow 1$ model, the multilevel model equations for obtaining the *a* path are

$$\begin{aligned} &\text{L1:} \quad \mathrm{M}_{ij} = \beta_{0j} + e_{ij} \\ &\text{L2:} \quad \beta_{0j} = \gamma_{00} + \mathrm{a}X_j + u_{0j} \end{aligned} \tag{21.25}$$

And the equations for the *b* path are

$$\begin{aligned} &\text{L1:} \quad \mathrm{Y}_{ij} = \beta_{0j} + \beta_{1j}M_{ij} + e_{ij} \\ &\text{L2:} \quad \beta_{0j} = \gamma_{00} + c\prime X_j + u_{0j} \\ &\qquad\quad \beta_{1j} = b \end{aligned} \tag{21.26}$$

Note the similarity in form for the *b* path in the $2 \rightarrow 2 \rightarrow 1$ model and the *a* path in the $2 \rightarrow 1 \rightarrow 1$ model, both of which involve $2 \rightarrow 1$ links.

For a $1 \rightarrow 1 \rightarrow 1$ model, the multilevel equations for the *a* path are

$$\begin{aligned} &\text{L1:} \quad \mathrm{Y}_{ij} = \beta_{0j} + \beta_{1j}X_{ij} + e_{ij} \\ &\text{L2:} \quad \beta_{0j} = \gamma_{00} + u_{0j} \\ &\qquad\quad \beta_{1j} = a \end{aligned} \tag{21.27}$$

And the multilevel equations for the *b* path are

$$\begin{aligned} &\text{L1:} \quad \mathrm{Y}_{ij} = \beta_{0j} + \beta_{1j}X_{ij} + \beta_{2j}M_{ij} + e_{ij} \\ &\text{L2:} \quad \beta_{0j} = \gamma_{00} + u_{0j} \\ &\qquad\quad \beta_{2j} = b \end{aligned} \tag{21.28}$$

Again, note the similarity between the multilevel equations for these paths and those for the *b* path in the $2 \rightarrow 1 \rightarrow 1$ model, which also involves a $1 \rightarrow 1$ link. Equations for producing *c* path estimates are not provided here but can be found in Krull and MacKinnon (2001).

Two complications arise when working with multilevel rather than single-level equations for estimating the various paths in a mediation model. The first is that the two alternate point estimates of the mediated effect, *ab* and $c - c'$, are generally not identical in the multilevel case. The reasons for this are discussed in Krull and MacKinnon (1999). The differences are usually quite small and become smaller with increasing sample size. The second complication is unique to the $1 \rightarrow 1 \rightarrow 1$ model. The previously specified equations allow for random intercepts to accommodate within group similarity, but slopes of all other L1 coefficients are specified as fixed. It may, in fact, be that the effects of the initial predictor on the mediator (i.e., the *a* path) or the effect of the mediator on the outcome (i.e., the *b* path) vary across L2 units. This would mean that different groups have different *a* and/or *b* paths in multilevel data with individuals nested within groups or that different individuals have different *a* and/or *b* paths in multilevel data with repeated measures nested within individuals. To appropriately model such data, it will be necessary to include random errors on the L2 equations predicting the L1 slope coefficients.

If it should be the case that both the *a* path and the *b* path in a $1 \rightarrow 1 \rightarrow 1$ model randomly vary across L2 units, it is possible that these *a* and *b* coefficients might covary. In this case, the point estimate of the mediated effects is not equal to *ab* but rather

$$Med = ab + \sigma_{ab} \tag{21.29}$$

which involves the covariance between the *a* and *b* estimates (Kenny, Korchmaros, & Bolger, 2003). Moreover, the approximate standard error is not equal to equation 21.8 but rather

$$s_{Med} = \sqrt{b^2\sigma_a^2 + a^2\sigma_b^2 + \sigma_a^2\sigma_b^2 + 2ab\sigma_{ab} + \sigma_{ab}^2} \tag{21.30}$$

which involves the covariance between the *a* and *b* estimates as well as the sampling variance of the covariance estimate (Kenny et al., 2003).

Finding an estimate of the covariance between the *a* and *b* estimates is challenging because these are typically produced in two completely separate analyses. Bauer, Preacher, and Gil (2006) presented a method that fits both the *a* and *b* paths in a single analysis, which allows the covariance and its corresponding sampling variance to be estimated. Their approach involves stacking the data so that each L1 unit has two observations with identical predictors and creating a generic outcome variable. The outcome variable is set equal to the *M* variable on the first observation for each L1 unit, and the outcome variable is set equal to the *Y* variable on the second observation. Then two selection variables (S_m and S_y) are created to distinguish between the first ($S_m = 1,\ S_y = 0$) and second

($S_m = 0,\ S_y = 1$) observations for each case. A multilevel model with a L1 equation of the form:

$$\begin{aligned} \text{L1: } Z_{ti} &= S_{mti}(1 + a_i X_{ti}) + S_{yti}(1 + b_i M_{ti} + c_i' X_{ti}) + e_{Zti} \\ &= S_{mti} + a_i S_{mti} X_{ti} + S_{yti} + b_i S_{yti} M_{ti} \\ &\quad + c_{ti}' S_{yti} X_{ti} + e_{Zti} \end{aligned} \tag{21.31}$$

is then fit to the stacked data, and each of the five L1 coefficients is allowed to vary randomly at L2. This model essentially fits multilevel models for the *a* path and the *b* and *c*′ paths simultaneously. To model the error variance appropriately, it will be necessary to allow the estimation of two separate values, corresponding to the observations with different values on one of the two indicator variables. Further details on how to implement the Bauer et al. (2006) method in a number of statistical software programs can be obtained from the website of the article's first author. More information on implementation of the model in the Mplus programming language is also available (MacKinnon, 2008; Preacher et al., 2010). The multilevel mediation model is summarized in MacKinnon and Valente (2014) and a Bayesian formulation for the multilevel mediation model is in Yuan and MacKinnon (2009).

MULTILEVEL MODELING APPROACHES FOR LONGITUDINAL DATA

The dominant multilevel model for the analysis of longitudinal data is the multilevel growth model (Hox, 2010; Raubenbush & Bryk, 2002; Singer & Willett, 2003). Growth in this sense refers to any sort of change over time, including increasing values, decreasing values, or trends that change direction. For longitudinal data that involve an intervention delivered at a particular point in time, an exponential decay model might provide a useful alternative that is especially well-suited for examining the change that follows the delivery of the intervention. Both approaches are discussed in detail below.

Multilevel Growth Modeling

A multilevel growth model is very similar to the two-level model for repeated measurements nested within individuals shown in equations 21.16 and 21.17. The primary difference is that the growth model includes at least one variable representing time at L1. If a single time variable is linearly coded and no covariates are included in the model, the multiple equation specification for the model would be

$$\begin{aligned} \text{L1:} \quad & Y_{ti} = \pi_{0i} + \pi_{1i} time_{ti} + e_{ti} \\ \text{L2:} \quad & \pi_{0i} = \beta_{00} + r_{0i} \\ & \pi_{1i} = \beta_{10} + r_{1i} \end{aligned} \tag{21.32}$$

where $time_{ti}$ is the time relevant variable. Each individual-specific growth parameter π is a function of the overall estimate of the growth parameter β in the sample and individual specific deviation r from that value. The single-equation form of the model is

$$Y_{ti} = \beta_{00} + \beta_{10} time_{ti} + r_{0i} + r_{1i} time_{ti} + e_{ti} \tag{21.33}$$

β_{00} estimates is the overall intercept, which is the expected value of the outcome variable when time is at its zero point. β_{10} estimates the overall linear slope estimate. The τ matrix contains the variances and covariances of the L2 errors *r*, and thus τ_{00} is the variance of individual-specific intercepts, τ_{11} is the variance of individual-specific slopes, and τ_{01} is the covariance between them. This particular parameter estimate is more often interpreted in growth models than in other multilevel models that do not involve time, because it can indicate whether individuals are becoming more similar (compensatory growth) or different (fan-spread growth) in their outcome variable scores.

More complex forms of growth can be modeled by adding additional variables coding time information to the L1 model. For example, higher order polynomial trends can be fitted by adding powered functions of the linear time variable. Piecewise models can also be fitted by adding additional coded vectors to produce models that estimate either (1) the slopes of the different time segments, or (2) one slope and the difference between slopes (Raudenbush & Bryk, 2002). Both individual and time-varying covariates can be added to the model. Individual covariates are added to the L2 specification as predictors of intercepts and/or slopes. For example, a gender variable could be added to the L2 equations in 21.32 to test whether intercepts and slopes of the outcome variable differed for males and females. A time-varying covariate could be added to the L1 specification either to adjust for its effect when estimating growth or to examine its effect over and above growth. A decision would need to be made about whether the effect of this variable would vary across individuals (in which case the L2 equation associated with this variable

would include an r error term) or would be assumed fixed (no L2 error term).

Most information relevant to multilevel models in general applies equally to multilevel growth models, with just a couple of notable exceptions. First, the time variable in a growth model is typically "centered" on a meaningful value—often the initial time point of the study—rather than on the mean per se. Centering time at its mean would simply put the zero point in the middle of the study period, which may not be the most substantively meaningful point at which to evaluate effects. Second, typical independence and homoscedasticity assumptions about L1 errors may not be realistic in longitudinal data. L1 error variances may increase or decrease over time, and values measured at later time points may depend on previous values measured at earlier time points. Most multilevel modeling software includes options for specifying heteroscedastic and autoregressive error structures that might be better suited for growth models.

Multilevel Exponential Decay Model

In considering models for longitudinal data, a primary issue is the shape of the trajectory across time. As discussed by Fritz (2014), often when researchers encounter data that exhibit any nonlinearity, a quadratic growth model is chosen without regard for the implications to the underlying variables or theory. For longitudinal psychological data, especially following an intervention or treatment, Fritz suggests that an exponential decay model has several advantages over a quadratic model. First, the exponential decay function is more realistic for many variables in psychology because it approaches an asymptote. One can imagine many scenarios where an individual is given a treatment, causing a change in the individual's underlying construct, which slowly returns to the individual's pretreatment level over time. At the same time, the individuals in the control group remain unchanged, a scenario illustrated in Figure 21.4. If the treatment had a lasting effect on the individual, the individual's score would still likely decrease across time, but not back to his or her pretreatment level (i.e., the construct would asymptote at a different value than the pretreatment level). While a quadratic growth model can approximate the exponential decay present in the figure, a quadratic function at some point must reach a minimum and then begin to grow towards infinity, an unlikely scenario given most variables in psychology. For this reason, many researchers have already used an exponential decay function (e.g., Blozis, 2004, 2007; Blozis, Conger, & Harring, 2007; Browne, 1993; Cudeck, 1996).

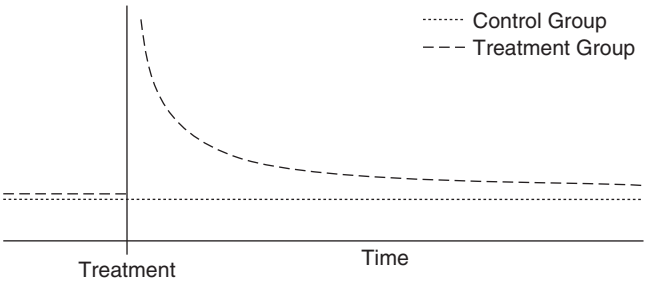


Figure 21.4 Illustration of a treatment group decaying exponentially across time while the control group remains constant.

Another reason for choosing an exponential decay model is that exponential decay is a parsimonious function requiring the estimation of three parameters:

$$Y_i = \beta_2 + (\beta_0 - \beta_2)\exp[\beta_1 t] + r_i \tag{21.34}$$

where t is time, β_0 is the initial state value at $t = 0$, β_2 is the asymptote value as time goes to infinity, and β_1 is the rate of change value, which is constrained to be less than zero to ensure that the asymptote exists (Pinheiro & Bates, 2000). While a quadratic model also contains three parameters, the interpretations of the coefficients from the exponential decay model are likely to be more useful to a researcher than those from a quadratic model.

Analyzing longitudinal data using an exponential decay model requires the use of a nonlinear multilevel model. Using notation from Raudenbush and Bryk (2002; see also Davidian & Giltinan, 1995), the exponential decay model is

$$\begin{aligned}
\text{L1:} \quad & Y_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i})\exp[\beta_{1i} t] + r_{ti} \\
\text{L2:} \quad & \beta_{0i} = \gamma_{00} + u_{0i} \\
& \beta_{1i} = \gamma_{10} + u_{1i} \\
& \beta_{2i} = \gamma_{20} + u_{2i}
\end{aligned} \tag{21.35}$$

where $time_i$, β_{0i}, β_{1i}, and β_{2i} have the same interpretation as in equation 21.34, r_{ti} are the Level 1 residuals, and the u_{ji}'s are random effects distributed $\sim \boldsymbol{N}(\mathbf{0}, \boldsymbol{\tau})$. As in linear multilevel models, time-varying predictors can be added at L1, while time-invariant predictors are added at L2. The inclusion of L2 predictors may be especially interesting for exponential decay because they test whether variance in the initial value, rate of change, and asymptote values can be predicted by another variable. Two of the most widely used software packages for estimating nonlinear multilevel

models in the social sciences are NLME (R Development Core Team, 2013) and SAS PROC NLMIXED (SAS Institute, 2014), although many other statistical packages for linear multilevel models also allow the estimation of nonlinear multilevel models.

STRUCTURAL EQUATION MODELING

Structural equation modeling is a multivariate analytic technique for exploring patterns of association among variables. A typical structural equation model (SEM) has two components: (1) a measurement model that specifies how observed indicator variables are related to unobserved latent constructs; and (2) a structural model that specifies how the various observed and latent variables are related to one another. Structural equation modeling employs a matrix representation to specify a number of simultaneous equations that denote the associations among observed and latent variables, and the model can be fitted using estimation methods that maximize the fit between the observed covariance matrix of the variables and the covariance matrix implied by the model. In depth treatment of this topic can be found in a number of texts, including Bollen (1989) and Kline (2011).

Diagrams are often used to depict the variables and relations specified in an SEM. By convention, observed variables are typically represented as squares or rectangles, and latent variables are typically represented as circles or ovals. Single-headed arrows represent directional causal relations between variables and double-headed arrows represent nondirectional correlational associations. The estimates produced in an SEM typically include factor loadings relating observed indicators to latent factors, path coefficients relating variables and factors to one another, variances and covariances of errors/disturbance terms. It is also possible to estimate means of latent variables, although these parameters are not typically part of standard SEM estimation. Mean structure can be indicated in SEM diagrams by arrows from a triangle representing a constant value of 1, though often such effects are not noted in the diagrams. In addition to parameter estimates, SEM programs provide a number of absolute and relative fit indices that allow researchers to examine how well the proposed model fits the data. These include a chi-square test of overall model fit, the root mean square error of approximation (RMSEA; Steiger, 1990), and the comparative fit index (CFI; Bentler, 1990). Particular aspects of the model can be tested by examining the size of the estimated coefficients relative to their standard errors or by comparing the fit of a series of models in which specific parameters, originally freely estimated, are systematically constrained.

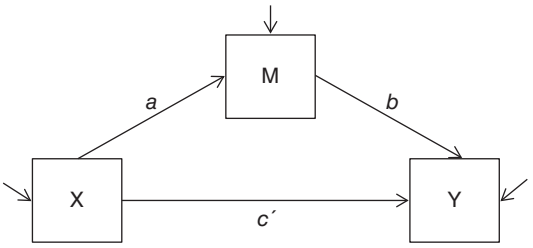


Figure 21.5 SEM mediation model for manifest *X*, *M*, and *Y* variables.

Mediation in SEM

Specification and estimation of mediation in SEM is straightforward. Figure 21.5 depicts a simple SEM for a single mediator model. All three variables in this model are observed, and thus this particular SEM is a simple path analysis and does not include a measurement model. Fitting the model produces estimates of the *a* and *b* path coefficients and their standard errors. These can be used to test mediation in much the same way as with regression analysis. Some SEM programs offer specific options for testing mediated (or indirect) effects, using either delta standard error method tests or bootstrapping methods.

If variables are measured with multiple indicators, SEM can be used to examine mediation among the underlying latent constructs. A measurement model can be specified to define the initial, mediator, and outcome factors from their various observed indicator variables (as shown in Figure 21.6), and the structural model specifies the appropriate paths between the latent constructs to test the mediated effect. The latent constructs are theoretically error-free, and the path coefficients will not suffer from the attenuation that occurs in estimated associations between observed measurements. Thus the mediated effects among latent constructs should be stronger and more easily detected than mediation among observed variables.

Moderation in SEM

There are a number of different approaches for examining moderation within an SEM. If the moderating variable is observed and categorical, the typical method involves fitting a multiple group model (Jöreskog, 1971). Such a model essentially fits a separate SEM within each level of the categorical moderator. For example, to examine

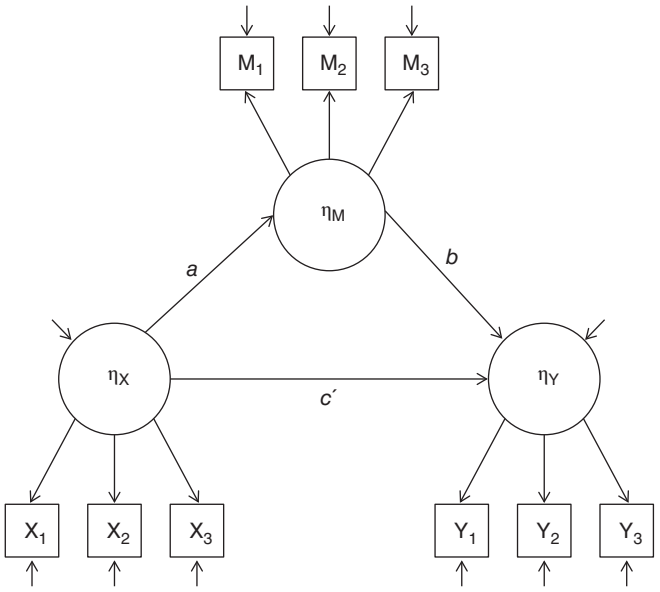


Figure 21.6 Mediation model with latent *X, M*, and *Y* variables.

whether the association between impulsive/hyperactive symptoms and interpersonal skill is moderated by gender, an SEM that allows different coefficients for the EXTERN → INTERP path would be fitted. The chi-square value for this model would be compared with that from a nested model that constrained the coefficients in the two groups to be equal. A significant chi-square difference would indicate that model fit improves when the two coefficients differ, supporting the hypothesis that the association is moderated by gender.

If the two variables involved in the moderated effect are observed, but neither is categorical, a multiplicative cross-product variable can be created and entered as an additional observed variable in the SEM. This cross-product variable then (1) is allowed to covary with the two component variables from which it was produced, and (2) predicts the outcome variable as shown in Figure 21.7. A significant path coefficient from the cross-product variable to the outcome measure would indicate moderation.

A moderated effect involving latent variables is somewhat more difficult to model. The most common approach to representing a latent variable interaction involves specifying an additional latent variable—essentially the cross-product of the two latent variables of interest—with cross-products of the original variable indicators serving as indicators of the interaction construct. This latent cross-product variable represents the interactive effect on the outcome measure when fitted with the two original latent variables simultaneously in the model. Specification

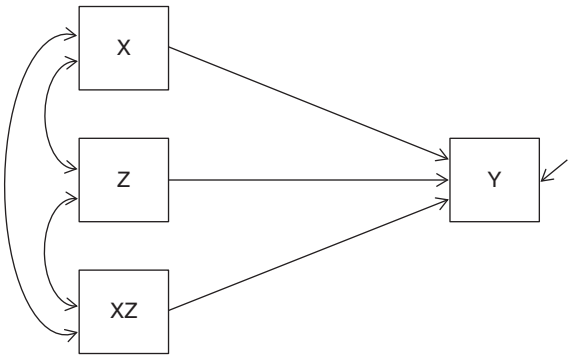


Figure 21.7 An SEM with an interaction between manifest variables.

and fitting of this model can be difficult, as it may involve complex nonlinear constraints, include many highly correlated variables, and violate normality assumptions. A number of different methods have been proposed to address these issues, but no clear consensus as to the best method for analyzing latent variable interactions has emerged. The various issues and proposed solutions are summarized in Kline (2011, Ch. 12).

STRUCTURAL EQUATION MODELS FOR LONGITUDINAL DATA

When longitudinal data are to be used in an SEM, the data set is typically organized in wide format (as opposed to the long format necessary for multilevel modeling), with one line of data per individual subject and multiple uniquely named variables representing the repeated measures of each construct. A number of different types of SEMs can be used to model longitudinal data, including autoregressive panel models, latent growth models, and latent change models. It is also possible to fit an exponential decay model within the SEM framework.

Autoregressive Model

In an autoregressive panel model (Duncan, 1969; Humphreys, 1960; Jöreskog, 1979; Wiley & Wiley, 1970), a measure of a given construct at a particular time point is presumed to be a function of value of that construct at the previous time point. The top series in Figure 21.8 illustrates an autoregressive model for an X variable, and the lower series illustrates a similar model for a Y variable. The arrows from time t to $t + 1$ within each series (i.e., X_1 to X_2 and X_2 to X_3; Y_1 to Y_2 and Y_2 to Y_3) are the autoregressive coefficients representing stability

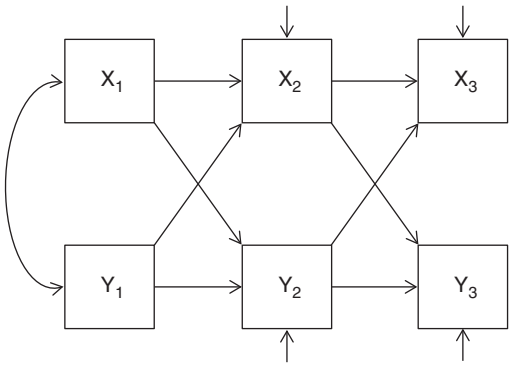


Figure 21.8 Autoregressive model with cross-lagged paths.

over time. Such models can be estimated with a series of OLS regression models or in a single simultaneous SEM. Autoregressive models for particular variables can be combined to form more complex models. For example, Figure 21.8 includes two types of cross-lagged paths: (1) paths from earlier measures of *X* to later measures of *Y*, and (2) paths from earlier measures of *Y* to later measures of *X*. Testing such a cross-lagged model—usually by fitting a series of SEMs that constrain particular sets of paths to be equal to zero or equal to each other (see Martens & Haase, 2006)—can allow a researcher to determine whether one set of effects leads the other or if there is reciprocal causality between the two constructs.

Latent Growth Curve Model

Another common SEM for longitudinal data is the latent growth curve (LGC) model (Duncan, Duncan, & Strycker, 2006). In such a model, multiple measurements of a given construct over time serve as indicator variables for latent growth factors. While typical SEM measurement models estimate the factor loadings that relate the latent variable to the observed indicators, the loadings in a latent growth model are fixed to define the shape of the growth trajectories. For example, Figure 21.9 presents a linear growth trajectory modeled based on three measurement time points. The factor loadings on the intercept factor are set to 1. The factor loadings on the linear slope factor are set to represent the amount of time elapsed since the first measurement occasion while setting the loading for the time point represented by the intercept factor to 0. Therefore, in the ECLS-K data, if we model linear growth from the kindergarten fall to fifth grade spring with kindergarten fall as the intercept, the first loading would be set equal to zero (essentially erasing the arrow) for the kindergarten fall assessment and the second loading would be set equal to .5, indicating that, on average, half of a year has elapsed

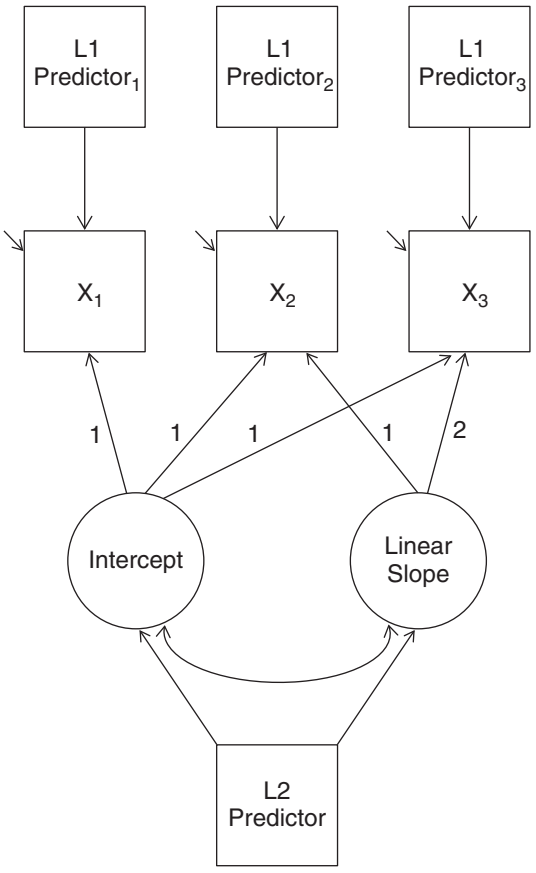


Figure 21.9 SEM latent growth curve model with L1 and L2 predictions.

at kindergarten spring. The other loadings would be set equal to 1.5, 3.5, and 5.5 to represent elapsed time at the first, third, and fifth grade spring assessments. If we model a quadratic growth trajectory, another growth factor, a quadratic growth factor, is added to the linear growth model in Figure 21.9, with the factor loadings equal to the squared linear values: 0, .25, 2.25, 12.25, and 30.25.

Estimating an SEM for a latent growth model produces means and variances for each of the latent growth factors. The means describe the normative growth trajectory. In a linear growth model, means of the intercept and linear growth factors would represent the expected value of the construct at the initial time point and the linear growth rate, respectively. The estimated variances of the growth factors represent differences between individuals in intercepts and linear rates of change. The covariance between intercepts and linear slopes is also estimated, along with an estimate of the error variance associated with the measurement of the outcome variable at each time point.

Both time-varying and individual (invariant over time) covariates can be added to a latent growth model.

Individual covariates are added as predictors of the latent growth factors, as shown in the lower portion of Figure 21.9. A time-varying covariate measured at a given time point can be added as a predictor of the outcome construct at that time point, as shown in the upper portion of Figure 21.9. Alternately, a researcher could let a time-varying covariate measured at an earlier time point predict the outcome construct measured at later time point, introducing an aspect of temporal precedence and a prospective lagged effect.

SEM and multilevel models can produce equivalent estimates of growth model parameters (Curran, 2003). However, the two modeling frameworks differ somewhat in their defaults for model specification and estimation, and thus results from the two approaches may initially appear to differ. Specifically, multilevel models estimate a single L1 variance component (assuming homogenous error variances across time points), while SEMs usually estimate time point-specific error variances. Also, multilevel programs will usually constrain negative variance estimates to zero, while SEM programs will allow the negative estimate. However, if program defaults are appropriately changed to reflect common settings, results produced with multilevel models and SEMs will be essentially identical.

Latent Change Model

Another type of latent variable model for investigating longitudinal data is the latent change score (LCS) model, also referred to as the latent difference score model (Ferrer & McArdle, 2003; McArdle, 2001; McArdle & Hamagami, 2001; McArdle & Nesselroade, 2003). An example of a four-wave LCS model is shown in Figure 21.10, where the η variables represent the error-free true score for each measurement wave, the Δ variables represent the latent change scores, and the ξ_1 variable is a second-order slope variable that represents the change in the latent change scores over time or, in other words, the change in the rate of change in score over time. Because each η variable is represented by only one observed score, the paths between all of the observed Y variables and the η variables are constrained to be equal to 1. A measurement model can be added to the LCS model to incorporate multiple indicators; Figure 21.11 shows an example of a four-wave multiple-indicator LCS model.

McArdle and Hamagami (2001) described the Δ variables as being a rate of change for a given time lag such that for a time lag of 1,

$$\Delta_t = \eta_t - \eta_{t-1} \tag{21.36}$$

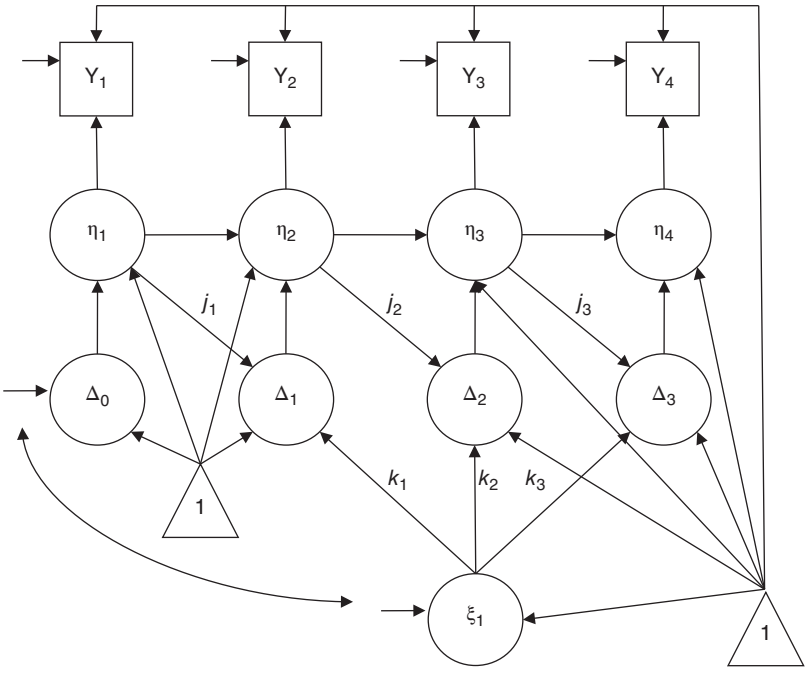


Figure 21.10 A four-wave latent change score model.

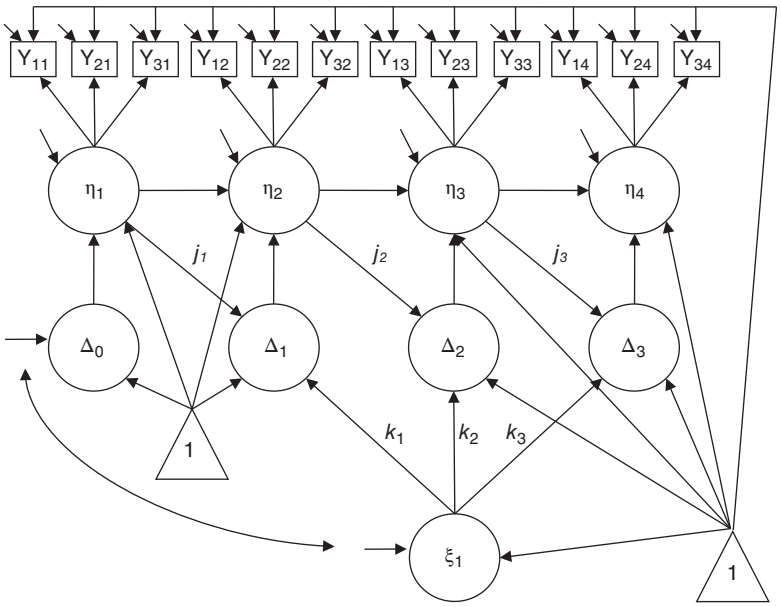


Figure 21.11 Multiple-indicator latent change score model.

except for Δ_0. An individual latent change score is then equal to

$$\Delta_t = k_t\xi_1 + j_t\eta_{t-1} \tag{21.37}$$

where j_t is the loading between the latent change score at time t to the latent construct score at time $t - 1$, and k_t is the loading of the latent change score on the ξ_1 variable. Assuming equally spaced measurement, the values of k_t are usually constrained to be equal across waves, as are the values of j_t, but unequal spacing of measurements can be taken into account by adjusting these constraints to reflect the actual spacing. In addition, Ferrer and McArdle (2003) described a cross-lagged version of the LCS model where the change score at time t predicts the observed score at time $t + 1$.

Equation 21.37 shows that the latent change score Δ_t is proportional to the score at the previous time point, $j_t\eta_{t-1}$, and the latent slope, $k_t\xi_1$. That is, an individual's change score at time t is a function of the overall change across time and the individual's score at the previous time point, which is why McArdle and Hamagami (2001) called this model a dual change score model. Constraining either the j paths, k paths or both to be equal to 0 changes the interpretation of the model. For instance, if the k paths are set to 0, the model becomes a proportional change score model because an individual's change score at time t only depends on their score at the previous time point, which will not be the same for each individual or time point. Alternatively if the j paths are set to 0, the model becomes a constant change score model because an individual's score at time t only depends on the latent slope, which is the same for all individuals across all time points. If the k and j paths are both set to 0, then the latent change score is not proportional to the previous score or related to the latent slope. When this occurs, there is no change in the construct scores across time, so the model becomes a no-change score model.

Much like a linear LGC model, the mean of Δ_0 can be considered as a latent intercept because it is the average score across individuals at baseline and the mean of ξ_1 can be considered a latent slope because it is the average change in change scores across time. Overall model fit can be determined by typical SEM fit indices such as the chi-square goodness of fit test, the RMSEA, and the CFI. Stationarity in the LCS model can be investigated by comparing models that constrain the autoregressive paths, j paths, and k paths of the model to be equal across waves with models where the constraint is lifted, using a series of nested model chi-square change tests.

Exponential Decay Structural Equation Model

A latent variable version of the exponential decay nonlinear mixed effects model can be specified using the general method for nonlinear latent variable growth curve models described by Browne (1993; see also Blozis, 2004, 2007;

Blozis et al., 2007). The nonlinear LGC model for exponential decay is similar to the LGC model and is of the form

$$\mathbf{X} = \boldsymbol{\tau}_{\mathbf{x}} + \boldsymbol{\Lambda}_{\mathbf{x}}\boldsymbol{\xi} + \boldsymbol{\delta} \tag{21.38}$$

where the vector $\boldsymbol{\xi}$ contains the latent growth parameters, the paths in the loading matrix $\boldsymbol{\Lambda}_{\mathbf{x}}$ are constrained to specific values, and the $\boldsymbol{\tau}_{\mathbf{x}}$ vector contains the means of the observed variables. Also present is the vector $\boldsymbol{\kappa}$, which contains the means of the latent variables, in this case the growth parameters. The difference between a linear LGC model and one for exponential decay is in the interpretation of the means of the latent variables and in the values of the constrained path loadings in the $\boldsymbol{\Lambda}_{\mathbf{x}}$ matrix.

To construct an exponential decay LGC model using Browne's (1993) method, illustrated in Figure 21.12, the loadings in the $\boldsymbol{\Lambda}_{\mathbf{x}}$ matrix must be set equal to the first partial derivatives of the exponential decay function

$$\lambda_{0t} = \frac{d}{d\beta_0} f(t, \boldsymbol{\theta}) = Exp[t\beta_0] \tag{21.39}$$

$$\lambda_{1t} = \frac{d}{d\beta_1} f(t, \boldsymbol{\theta}) = (\beta_0 - \beta_2) t Exp[t\beta_1] \tag{21.40}$$

$$\lambda_{2t} = \frac{d}{b\beta_2} f(t, \boldsymbol{\theta}) = 1 - Exp[t\beta_2] \tag{21.41}$$

where

$$f(t, \boldsymbol{\theta}) = \beta_2 - (\beta_0 - \beta_2) Exp[t\beta_1]$$

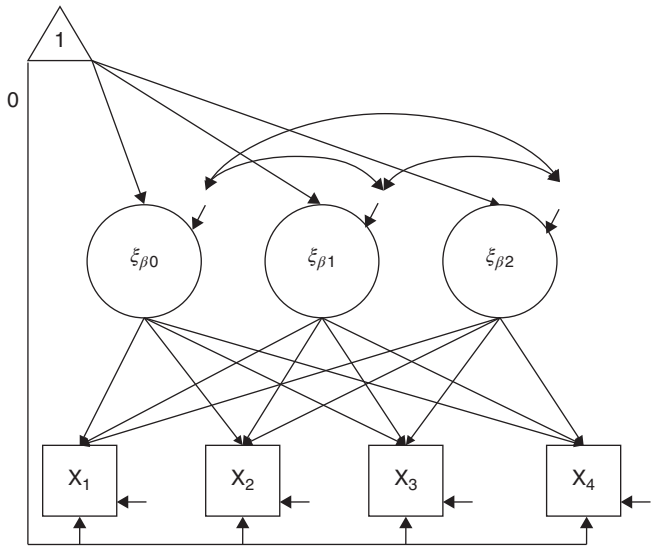


Figure 21.12 A four-wave exponential decay latent growth curve model.

This defines the vector of latent variables to be $\boldsymbol{\xi} = [\xi_{\beta 0} \ \xi_{\beta 1} \ \xi_{\beta 2}]'$ and the vector of means for the latent variables to be $\boldsymbol{\kappa} = [\beta_0 \ \beta_1 \ \beta_2]'$ where β_0, β_1, and β_2 have the same interpretations as in equation 21.34.

The latent exponential decay model created using the Browne (1993) method has two problems that arise during estimation. The first is that the mean of one of the latent variables must be constrained to be equal to zero. The second problem is that, as shown in equations 21.39 to 21.41, to constrain the loadings in the $\boldsymbol{\Lambda}_{\mathbf{x}}$ matrix correctly, the values of the means of the latent variables must already be known prior to estimating the model. Both of these problems may be circumvented, however, by setting β_1 equal to 0 and then simultaneously estimating the path loadings and the means of the latent variables using nonlinear constraints, for example using the MODEL CONSTRAINT command in Mplus.

MODELS FOR MODERATION AND MEDIATION IN LONGITUDINAL DATA

The next section of the chapter describes several models that incorporate mediation and moderation into the analysis of longitudinal data. Most employ the multilevel and SEM frameworks for model specification and estimation. Some of these approaches (e.g., the lag as moderator model, time-varying covariate interactions within multilevel models, and the mediation over time model) make use of the repeated measurements to explore how certain effects and associations change over time. Other approaches, especially those examining mediation using the SEM framework, focus more on the inherently time-ordered aspect of longitudinal data to establish the temporal precedence of effects necessary to make a causal argument about how predictor variables exert their effects on the outcomes of interest.

Lag as Moderator Model

Selig, Preacher, and Little (2012) developed a moderator model unique to longitudinal data. Specifically, they propose the lag as moderator (LAM) approach to examine time-dependent associations. It is possible that the strength of the association between two variables assessed at different occasions will depend on the length of time elapsed between the occasions. Their presentation assumes just a single measurement of two variables for each subject, but variability of the time lag between the two measurements across subjects.

The general approach involves using the length of the lag between measurement occasions as a predictor in a regression model. If the focal variables are X (predictor) and Y (criterion), the model would include X, the lag, and the interaction between X and the lag as predictors:

$$\hat{Y} = b_0 + b_1 X + b_2\, Lag + b_3 \tag{21.42}$$

In this equation, the b_1 coefficient represents the association between X and Y when Lag = 0 (i.e., simultaneous estimation), the b_2 coefficient represents the expected change in Y for a 1 unit increase in the length of the lag, and b_3 represents the linear change in the association between X and Y that accompanies a 1 unit increase in the length of the lag. Strategic centering of the lag variable can change the meaning of the b_1 coefficient to test the association between X and Y if a particular lag is believed to be particularly important.

Selig et al. pointed out that in many cases, change in the X–Y association as a function of the lag will not be linear. This nonlinearity might be captured with a straightforward polynomial specification (here, a quadratic form):

$$\hat{Y} = b_0 + b_1 X + b_2 Lag + b_3 Lag^2 + b_4 XLag + b_5 XLag^2 \tag{21.43}$$

or with a more complex nonlinear parameterization:

$$\hat{Y} = b_0 + \left[\alpha_{Y \cdot X} - \left(\alpha_{Y \cdot X} - \alpha_0\right)\left(\frac{Lag}{\alpha_{Lag}} - 1\right)^2\right] X \tag{21.44}$$

Though the latter expression requires nonlinear regression techniques for the estimation of its parameters, it provides estimates with more meaningful interpretations than those of the quadratic model (Cudeck & du Toit, 2002). α_0 is the expected association between X and Y when Lag = 0, α_{Lag} is the value of the lag at which the maximal X-Y association is obtained, and $\alpha_{X \cdot Y}$ is that maximum association. Alternately, an exponential model might be specified:

$$\hat{Y} = b_0 + (ae^{bLag})X \tag{21.45}$$

to represent a functional relationship that approaches an asymptote rather than symmetric form (i.e., any increase to a maximum must necessarily be followed by a decrease) implied by a quadratic specification.

Selig et al. provide an empirical example illustrating the LAM approach using variables from the Early Head Start Research and Evaluation study (Department of Health and Human Services, Administration for Children and Families, 1996–2001; available from the Inter-university Consortium for Political and Social Research website: www.icpsr.org/). As is often the case, the original intention was to collect the observational data at a single fixed lag; however, practical difficulties resulted in variation in time elapsed between measurement occasions across subjects. The LAM analysis examined effect of lag length on the association between the quality of stimulation provided to a child in his or her home at approximately 14 months and a measure of cognitive, language, and social development at approximately 24 months of age. Though the nominal lag between the 14-month and the 24-month observations was 10 months (and, in fact, the average lag was 10.08 months), there was considerable variability in the actual length of the lag, which ranged from 2.95 to 16.98 months (SD = 1.74 months).

Initial examination of the data consisted of a series of scatterplots windowing different lag lengths and estimation of a regression slope within each window. A plot of these slope values against the average lag for each window showed a clear decrease in the strength of association with longer lags. To capture this change, Selig et al. fit a linear and an exponential LAM model, and both showed significant lag moderation. The two models seemed to fit the data equally well for the range of lags actually present in the data, but the authors pointed out that for even longer lags the linear model predicts a negative association between the two variables while the exponential model predictions approach a lower asymptote of zero. They cautioned, however, that their example data did not involve random assignment of the lag length, and thus interpretations must be circumspect.

We used the LAM model to examine the effect of the lag between the measurement of ADHD symptoms at kindergarten fall and measurement of interpersonal skills, academic performance, and internalizing behavior at kindergarten spring. The lag between the two assessment occasions ranged from 3.8 to 9.3 months, with an average of 6.1 months. Linear and quadratic effects of lag length were tested. Only two interactions between predictor values and lag length emerged, both linear. The negative association between kindergarten fall EXTERN and kindergarten spring INTERP was stronger for shorter than for longer lags ($b_3 = 0.3340$, SE = 0.1177, $p < .01$), and the positive association between kindergarten fall EXTERN and kindergarten spring INTERN was stronger for longer than for shorter lags ($b_3 = 0.2268$, SE = 0.1063, $p < .05$).

Selig et al.'s LAM approach was designed for a very specific data structure, with only two observations of

measurement per subject, each variable assessed at only one of the measurement occasions, and variation in the lag between measurement occasions across subjects either produced intentionally (e.g., through random assignment of lag lengths) or as the result of vagaries in the implementation of a fixed-lag design. The single-level regression estimation that they recommend, whether linear or nonlinear, requires a single outcome observation for each subject. It should be possible, however, to adapt their framework for estimation as a multilevel model, which will widen the range of data structures to which the LAM approach might be applied. Moreover, such applications would convert what Selig et al. acknowledge as an interindividual approach to what is often an intraindividual question into an appropriately matched intraindividual analysis.

When the predictor variable is time-invariant but the criterion is assessed at multiple later time points, each subject shows intraindividual variation in the predictor-criterion lag. The single equation form of a multilevel model would closely parallel those specified by Selig et al., simply adding L2 error terms to model the potential unmeasured similarity among the repeated criterion observations obtained from a given individual. At L1 (the repeated measures level), the equation would be

$$Y_{ti} = \beta_{0i} + \beta_{1i}\text{Lag}_{ti} + e_{ti} \tag{21.46}$$

and at L2 (the individual level), we would have

$$\begin{aligned}\beta_{0i} &= \gamma_{00} + \gamma_{01}X_i + u_{0i}\\ \beta_{1i} &= \gamma_{10} + \gamma_{11}X_i + u_{1i}\end{aligned} \tag{21.47}$$

The γ_{11} coefficient captures the lag moderation effect in this model.

Results of fitting such models in the ECLS-K data are provided in Table 21.4. These models predicted kindergarten spring through fifth grade spring assessments of interpersonal skills, academic performance, and internalizing behavior from kindergarten fall measures of ADHD symptoms. Initial models included interactions between ADHD symptoms and both linear and quadratic lag variables. If the interaction between the ADHD symptoms variable and the quadratic lag was not significant, the model was refitted with only the interaction involving the linear lag. Most of the final models included significant interactions with the quadratic lag function; however, the model predicting READ from EXTERN included only an interaction with linear lag. All of the models indicated that the strongest effects of the kindergarten fall ADHD symptoms variables were the contemporaneous ones on the outcome variables at kindergarten fall with Lag = 0. The magnitude of the association between kindergarten fall EXTERN and READ declined linearly through the fifth grade spring assessment. For the other predictor/outcome combinations, the magnitude of the association declined quickly at the kindergarten spring and first grade spring assessments and gradually leveled off. Some of the models, in fact, indicate a reversal of the original trend near the fifth grade assessment, but this seems more likely an artifact of the necessary symmetry in quadratic models than a real pattern in the data, suggesting that exponential models might be a more appropriate choice for modeling some of these particular associations.

TABLE 21.4 Coefficients and Standard Errors for Multilevel LAM Models

Predictor	Outcome	Simple effect at Lag = 0	Predictor x Lag	Predictor x Lag^2
LEARN	INTERP	0.6204***	−0.1982***	0.0261***
		(0.0050)	(0.0060)	(0.0011)
LEARN	READ	0.3262***	−0.0451***	0.0029***
		(0.0052)	(0.0030)	(0.0005)
LEARN	MATH	0.3231***	−0.0484***	0.0059***
		(0.0046)	(0.0025)	(0.0004)
LEARN	INTERN	−0.2690***	0.0665***	−0.0103***
		(0.0052)	(0.0054)	(0.0010)
EXTERN	INTERP	−0.5390***	0.0866***	−0.0105***
		(0.0060)	(0.0066)	(0.0012)
EXTERN	READ	−0.1469***	0.0146***	NS
		(0.0061)	(0.0013)	
EXTERN	MATH	−0.1242***	0.0146***	−0.0021***
		(0.0054)	(0.0027)	(0.0005)
EXTERN	INTERN	0.2084***	−0.0508***	0.0078***
		(0.0057)	(0.0059)	(0.0011)

Multilevel Growth Models with Time-Varying Covariate Interactions

The multilevel growth model presents an interesting opportunity for examining a particular kind of moderation in longitudinal data. This is moderation of the effect of a L1 predictor variable on a L1 outcome variable by time. That is, one can test whether the association between repeatedly measured predictor and outcome constructs changes systematically over the course of a longitudinal study. Many theories would predict change in the strength of association between two variables during the course of development or when transitioning from one developmental stage to another. Judicious coding of time trends and modeling interactions between predictors and the time-related variables will allow such effects to be directly tested.

For example, consider a simple linear multilevel growth model

$$\begin{aligned} \text{L1:} \quad & Y_{ti} = \beta_{0i} + \beta_{1i} time_{ti} + e_{ti} \\ \text{L2:} \quad & \beta_{0i} = \gamma_{00} + u_{0i} \\ & \beta_{1i} = \gamma_{10} + u_{1i} \end{aligned} \tag{21.48}$$

Combined:

$$Y_{ti} = \gamma_{00} + \gamma_{10} time_{ti} + u_{0i} + u_{1i} time_{ti} + e_{ti}$$

This model estimates the linear trend in the outcome variable over time. If a researcher is interested in the effect of a time point-specific predictor variable (X_{ti}, a time-varying covariate), it can be added to the L1 specification

$$\text{L1:} \quad Y_{ti} = \beta_{0i} + \beta_{i1} time_{ti} + \beta_{2i} X_{ti} + e_{ti} \tag{21.49}$$

with an associated L2 equation

$$\text{L2:} \quad \beta_{2i} = \gamma_{02} \tag{21.50}$$

Combined:

$$Y_{ti} = \gamma_{00} + \gamma_{10} time_{ti} + \gamma_{02} X_{ti} + u_{0i} + u_{1i} time_{ti} + e_{ti} \tag{21.51}$$

With this specification, the time trend (γ_{01}) is adjusted for variation in the time-varying predictor and γ_{02} represents the effect of the predictor over and above the time trend.

Now consider adding the interaction between the L1 predictor and the linear time variable:

$$\begin{aligned} \text{L1:} \quad & Y_{ti} = \beta_{0i} + \beta_{i1} time_{ti} + \beta_{2i} X_{ti} \\ & \qquad + \beta_{3i} X_{ti} time_{ti} + e_{ti} \\ \text{L2:} \quad & \beta_{3i} = \gamma_{30} \end{aligned} \tag{21.52}$$

Combined:

$$\begin{aligned} Y_{ti} = {} & \gamma_{00} + \gamma_{10} time_{ti} + \gamma_{02} X_{ti} + \gamma_{03} X_{ti} time_{ti} + u_{0i} \\ & + u_{1i} time_{ti} + e_{ti} \end{aligned}$$

As Singer and Willett (2003, p. 171) noted, the coefficient associated with the interaction between a time-varying predictor and the time variable (here, γ_{03}) can be interpreted in two ways. One interpretation is that the rate of change over time in the outcome variable depends on the level of the time-varying predictor. The second interpretation, and the one that we will focus on here, is that the effect of the time-varying predictor on the outcome changes over time. The equation above specifies linear change in the association between the time-varying predictor and the outcome, but (as discussed in the original Baron and Kenny (1986) paper), a more (e.g., quadratic) or less (e.g., step) complex function could be used to model a hypothesized pattern of change.

Hedeker (2004) discussed such an effect in the context of a longitudinal psychiatric study originally described in Reisby et al. (1977). This study examined the association between imipramine and desipramine plasma levels (the time-varying predictors) and clinical response (the outcome variable) over time in a sample of depressed inpatients. Because the effectiveness of antidepressants is not immediate but develops over time, an exploration of such moderated effects seems warranted. The model included a linear time trend, main effects of the imipramine and desipramine plasma levels, and interactions between linear time and the plasma levels. The model including the two interactions fit better than the model without the interactions. Examination of specific coefficients showed that the imipramine level by time interaction was not significant in this sample, but the desipramine interaction was significant, indicating that, in fact, the effect of this particular antidepressant on clinical response became more pronounced over the course of the 5-week study.

Table 21.5 reports the results of analyses in the ECLS-K data that included interactions between time-varying predictors and time. In particular, we explored whether the effects of the two ADHD symptoms variables on interpersonal skill, academic performance, and internalizing behavior change between fall of kindergarten and spring of fifth grade. Multilevel growth models incorporating an interactions between an ADHD symptoms variable and functions of the time variable were fitted for each possible pairing of LEARN and EXTERN with INTERP, MATH, READ, and INTERN. In each model, the base growth curve for the outcome construct was allowed to be quadratic with random effects for all growth parameters, and any nonsignificant variances were trimmed.

As Table 21.5 shows, the interaction between LEARN and linear time on INTERP is significant. This indicates that the contemporaneous positive association between the approaches to learning score and interpersonal skill ratings become stronger over time. For example, as indicated in column 3 of Table 21.5, the expected association between these two variables at kindergarten fall (i.e., when

TABLE 21.5 Coefficients and Standard Errors for Multilevel Models Including Time-Varying Predictor by Time Interactions

Predictor	Outcome	Simple effect of predictor	Linear time interaction	Quadratic time interaction
LEARN	INTERP	0.6365***	0.0070***	NS
		(0.0035)	(0.0013)	
LEARN	READ	0.1602***	−0.0293***	0.0029***
		(0.0035)	(0.0030)	(0.0005)
LEARN	MATH	0.1312***	−0.0436***	0.0064***
		(.0029)	(0.0026)	(0.0004)
LEARN	INTERN	−0.2632***	−0.0114***	NS
		(0.0037)	(0.0014)	
EXTERN	INTERP	−0.5120***	−0.0787***	0.0093***
		(0.0052)	(0.0056)	(.0010)
EXTERN	READ	−.0234***	−0.0247***	0.0034***
		(0.0039)	(0.0033)	(.0005)
EXTERN	MATH	−0.0315***	−.0035**	NS
		(.0037)	(.0012)	
EXTERN	INTERN	0.2242***	0.0369***	−0.0040***
		(0.0049)	(0.0054)	(.0010)

Time = 0) is .6365 and can be interpreted in much the same way as an unstandardized regression coefficient: for each one unit increase in LEARN at kindergarten fall, the expected value of interpersonal skill at that time point increases by .6365 points. For each additional year that passes, the contemporaneous association is expected to increase by .0070 points (column 4). So at fifth grade spring, the expected value of interpersonal skills increases by .6750 (i.e., .6365 + .0070 ∗ 5.5) for each one unit increase in LEARN. Similarly, Table 21.5 shows that the negative effects of LEARN on INTERN and the negative effects of EXTERN on MATH become significantly more pronounced over time. The predicted associations between the ADHD predictor variables and the outcome variables at each time point correspond fairly well—but not perfectly—to coefficients produced via OLS regressions conducted on data from each time point separately. Although this model may not precisely reproduce the regression coefficients at each time point, it does allow for a single parameter test of the change in such coefficients across time that is not available from analyses conducted on data from each measurement occasion separately.

Five of the eight analyses presented in Table 21.5 involve a significant interaction between the time-varying ADHD symptoms predictor and the quadratic time function, and for all of these, the interactions involving the linear and quadratic time functions are of opposite sign. This indicates that the initial changes in the magnitude of the ADHD symptoms variable and the outcome will eventually change direction. For example, the coefficients for the effect of LEARN on MATH in columns 3 and 4 of Table 21.5 indicate a positive association at kindergarten fall that initially decreases in magnitude over time. However, because of the positive sign of the quadratic interaction in column 5, this trend reaches a minimum around third grade spring, and the magnitude of the association increases thereafter. The maxima or minima for the other four associations with quadratic time interactions, however, reach their minima or maxima much closer to the end of the grade range included in the data. Care should be taken to ensure that these reversals are a true feature of the data and not just an artifact of the necessarily symmetric time function used in this particular example.

The model specification in equation 21.50 as well as those in Hedeker (2004) and the previous ECLS-K analyses assume that the effects of the time-varying covariates are fixed. That is, the association between the time-varying covariate and the outcome measure is the same for all subjects. Moreover, the extent to which the covariate's association with the outcome changes over time is also assumed to be the same for all subjects. However, with a sufficiently large sample, it may be possible to explore whether, in fact, these effects differ across individuals. This can be accomplished by adding error terms u_{2i} and u_{3i} to the L2 equations predicting the β_{2i} and β_{3i} coefficients, respectively. Then the variance of u_{2i} estimates (τ_{22}) indicates the extent to which individuals vary in the association between their predictor and outcome values when time is at its zero point, and the variance of the u_{3i} estimates (τ_{33}) indicates the extent to which individuals vary in how much this association changes over time.

Mediation Change over Time Model

The mediation change over time (M-COT) model builds upon the idea of creating interactions between predictors and the explicit time variable in a multilevel growth model to examine possible change in both the *a* and *b* paths of a mediation model over time. In contrast to other longitudinal mediation models that will be presented below, the focus of this model is not on establishing the temporal precedence of effects necessary for causal pathways. Rather, the M-COT model is largely descriptive and explores how the various paths in the basic model of Figure 21.1, specifically the *a* path from *X* to *M* and the *b* path from *M* to *Y*, change over the course of a longitudinal study. The $X \rightarrow M$ and $M \rightarrow Y$ relations within the model might be cross sectional, or *X* might be assessed prior to *M*, and *M* might be assessed prior to *Y*. In the former case, the M-COT model would simply describe how the contemporaneous associations between variables

change over time. In the latter case, the M-COT model would describe how the lagged relations among variables change as time progresses.

This discussion will concentrate on the M-COT model for exploring cross sectional mediation in longitudinal data. This model is appropriate for data with repeated measures of the outcome variable Y. The initial variable X and the mediator variable M could also be measured repeatedly or could be time-invariant individual variables. The following model assumes a $1 \rightarrow 1 \rightarrow 1$ model with all three variables measured repeatedly at the same time points. The M-COT model can be specified as a two-level random coefficient (i.e., multilevel) model with time points nested within individuals (Arruda & Krull, 2013). For a simple model that examines linear change over time in the a and b paths, the multiple equation form of the model to estimate a paths would be:

$$\begin{aligned} \text{L1:} \quad & M_{ti} = \beta_{0i} + \beta_{1i} time_{ti} + \beta_{2i} X_{ti} \\ & \quad + \beta_{3i} time_{ti} X_{ti} + e_{ti} \\ \text{L2:} \quad & \beta_{0i} = \gamma_{00} + u_{0i} \qquad \beta_{2i} = \gamma_{20} \\ & \beta_{1i} = \gamma_{10} + u_{1i} \qquad \beta_{3i} = \gamma_{30} \end{aligned} \tag{21.53}$$

and the single-equation form would be

$$\begin{aligned} M_{ti} = {} & \gamma_{00} + \gamma_{10} time_{ti} + \gamma_{20} X_{ti} + \gamma_{30} time_{ti} X_{ti} \\ & + u_{0i} + u_{1i} time_{ti} + \mathrm{e}_{ti} \end{aligned} \tag{21.54}$$

In this particular model, the γ_{20} coefficient estimates the association between X and M when time is at its zero point. The γ_{30} coefficient estimates how much this association changes for each additional unit of time.

The multilevel equation to generate β path estimates would be:

$$\begin{aligned} \text{L1:} \quad & Y_{ti} = \beta_{0i} + \beta_{1i} time_{ti} + \beta_{2i} X_{ti} + \beta_{3i} time_{ti} X_{ti} \\ & \quad + \beta_{4i} M_{ti} + \beta_{5i} time_{ti} M_{ti} + e_{ti} \\ \text{L2:} \quad & \beta_{0i} = \gamma_{00} + u_{0i} \quad \beta_{2i} = \gamma_{20} \quad \beta_{4i} = \gamma_{40} \\ & \beta_{1i} = \gamma_{10} + u_{1i} \quad \beta_{3i} = \gamma_{30} \quad \beta_{5i} = \gamma_{50} \end{aligned} \tag{21.55}$$

in the multiple equation form, and

$$\begin{aligned} \mathrm{Y}_{ti} = {} & \gamma_{00} + \gamma_{10} time_{ti} + \gamma_{20} X_{ti} + \gamma_{30} time_{ti} \mathrm{X}_{ti} \\ & + \gamma_{40} M_{ti} + \gamma_{50} time_{ti} \mathrm{M}_{ti} + u_{0i} + u_{1i} time_{ti} + \mathrm{e}_{ti} \end{aligned} \tag{21.56}$$

in the single equation form. The γ_{40} coefficient in this particular model estimates the association between M and Y when time is at its zero point. The γ_{50} coefficient estimates how much this association changes for each additional unit of time.

Combining the appropriate coefficients from the two models provides an expression for how the mediated effect ab changes over time (Arruda & Krull, 2014a). The additional subscripts M and Y in the following equations indicate whether a given coefficient came from the first multilevel equation (M) regressing M on X or the second multilevel equation (Y) regressing Y on X and M simultaneously. The combined expression is simply the product of the terms relevant to the a effect in equation 21.54 and those relevant to the β effect in equation 21.6:

$$ab_{ti} = (\gamma_{20M} + \gamma_{30M} time_{ti})(\gamma_{40Y} + \gamma_{50Y} time_{ti}) \tag{21.57}$$

Manipulating and rearranging this expression gives

$$\begin{aligned} ab_{ti} = {} & \gamma_{20M}\gamma_{40Y} + (\gamma_{20M}\gamma_{50Y} + \gamma_{30M}\gamma_{40Y}) time_{ti} \\ & + \gamma_{30M}\gamma_{50Y} time_{ti}{}^2 \end{aligned} \tag{21.58}$$

The first term in this expression, $\gamma_{20M}\,\gamma_{40Y}$, estimates the mediated effect (i.e., the product of the a and b paths) when time is at its zero point. The second term, $\gamma_{20M}\,\gamma_{50Y} + \gamma_{30M}\,\gamma_{40Y}$, estimates the instantaneous linear change in the mediated effect when time is at its zero point. The third term, $\gamma_{30M}\gamma_{50Y}$, estimates the quadratic change in the mediated effect over time and indicates how the instantaneous linear change in the mediated effect itself changes over time.

Approximate standard errors for the three terms in equation 21.58 can be obtained via the multivariate delta method (Arruda & Krull, 2014a). Specifically, the standard errors are as follows:

$$SE(\gamma_{20M}\gamma_{40Y}) = sqrt(\gamma_{20M}{}^2\sigma^2{}_{\gamma 40Y} + \gamma_{40Y}{}^2\sigma^2{}_{\gamma 20M}) \tag{21.59}$$

$$\begin{aligned} SE(\gamma_{20M}\gamma_{50Y} + \gamma_{30M}\gamma_{40Y}) = {} & sqrt(\gamma_{20M}{}^2\sigma^2{}_{\gamma 50Y} \\ & + \gamma_{50Y}{}^2\sigma^2{}_{\gamma 20M} + \gamma_{40Y}{}^2\sigma^2{}_{\gamma 30M} + \gamma_{30M}{}^2\sigma^2{}_{\gamma 40Y}) \\ & + (2 * (\gamma_{20M}\gamma_{30M}\sigma_{\gamma 40Y\gamma 50Y}) + (\gamma_{40Y}\gamma_{50Y}\sigma_{\gamma 20M\gamma 30M})) \end{aligned} \tag{21.60}$$

$$SE(\gamma_{30M}\gamma_{50Y}) = sqrt(\gamma_{30M}{}^2\sigma^2{}_{\gamma 50Y} + \gamma_{50Y}{}^2\sigma^2{}_{\gamma 30M}) \tag{21.61}$$

These standard errors can be used to conduct hypothesis tests or develop confidence intervals for each of the terms in equation 21.58. Thus we can explicitly test the trend in *ab* over time.

One useful way to think about this model is to consider the ultimate form of change in the *ab* estimate of the mediated effect that will result from combining different trends in *a* and *b*. As currently specified, three types of trends are possible for the *a* path: the *a* path could be zero (both the γ_{20M} and γ_{30M} coefficients are nonsignificant), a nonzero constant over time (the γ_{20M} coefficient is significant but the γ_{30M} coefficient is nonsignificant), or a linearly increasing function of time (the γ_{30M} coefficient is significant, regardless of the significance of the γ_{20M} coefficient). The same three trends are also possible for the *b* path (i.e., zero—both the γ_{40Y} and the γ_{50Y} coefficients are nonsignificant, constant—the γ_{40Y} coefficient is significant but the γ_{50Y} coefficient is nonsignificant, or linearly changing—the γ_{50Y} coefficient is significant, regardless of the significance of the γ_{40Y} coefficient). Then there are nine possibilities for the multiplicative combination, as shown in Table 21.6. Of course, whenever the nature of either of the two paths is zero, the multiplicative function *ab* will also be zero. Thus five of the nine combinations ultimately produce no mediated effect. Only four combinations produce nonzero values for the *ab* function over time, and these correspond to the four terms in equation 21.58.

With this limited set of possibilities (i.e., allowing only zero, constant, and linear trends for *a* and *b*), the only way that the *ab* product could indicate a constant mediated effect over time is if both *a* and *b* are nonzero constants. There are two ways that *ab* could show a pattern of linear change over time, specifically (1) if *a* is a nonzero constant and *b* changes linearly over time, or (2) if *b* is a nonzero constant and *a* changes linearly over time. The product *ab* will change quadratically over time only if both *a* and *b* change linearly.

By substituting each of the unique time values into the equation, it is possible to calculate the estimated mediated effect *ab* at each time point. Standard errors of these time point-specific estimates can be calculated using the multivariate delta method. Simulation studies (Arruda & Krull, 2013, 2014b) comparing the time point-specific estimates of *ab* from the M-COT model with those generated by fitting a series of OLS regression models to the data from each time point found that both approaches generated unbiased estimates of *ab* at each time point. When the data generating process for *a* and *b* was truly linear over time (i.e., when the analysis model form matched the true model), however, time point-specific estimates produced by the M-COT model had smaller standard errors than those generated using standard regression techniques to estimate mediated effects at each time point separately. Thus the M-COT approach is more highly powered than the regression approach. Across the range of simulated conditions, the standard errors of mediated effects at specific time points were 14-47% smaller in the M-COT model, resulting in 25-89% more power to detect nonzero effects than using standard regression techniques (Arruda & Krull, 2014b).

The M-COT model was used to assess changes in mediated effects over time in the ECLS-K data. Results of separate analyses in which the ADHD symptoms variables predicted interpersonal skill and academic performance mediators and an internalizing behavior outcome are presented in Table 21.7. The negative effect of LEARN on INTERN through INTERP has a negative linear trend and no significant quadratic trend, so this effect become increasingly pronounced over time. Similar patterns hold for the effects on LEARN on INTERN through READ and MATH, but these include a significant positive quadratic component, so the increasingly negative mediated effects level off at later grades for these academic performance mediators. The effects of EXTERN on INTERN through the three mediators all show similar patterns, with positive mediated effects at kindergarten fall and significant linear and quadratic trends. This indicates that the mediated effects become stronger over time, and the rate of increase speeds up at later grades.

TABLE 21.6 Combinations of Significant and Nonsignificant Coefficients That Produce Various Trends of the Mediated Effect over Time

γ_{20M}	γ_{30M}	*a* path trend	γ_{40Y}	γ_{50Y}	*b* path trend	*ab* trend
NS	NS	Zero	NS	NS	Zero	Zero
NS	NS	Zero	Sig	NS	Constant	Zero
NS	NS	Zero	NS or sig	Sig	Linear	Zero
Sig	NS	Constant	NS	NS	Zero	Zero
Sig	NS	Constant	Sig	NS	Constant	Constant
Sig	NS	Constant	NS or sig	Sig	Linear	Linear
NS or sig	Sig	Linear	NS	NS	Zero	Zero
NS or sig	Sig	Linear	Sig	NS	Constant	Linear
NS or sig	Sig	Linear	NS or sig	Sig	Linear	Quadratic

TABLE 21.7 Coefficients, Standard Errors, and Mediated Effect Trends for the M-COT Model

X	*M*	*a* at K fall	*a* linear trend	*b* at K fall	*b* linear trend	Med at K fall	Med linear trend	Med Quad Trend
LEARN	INTERP	0.6365^{***}	0.0070^{***}	-0.1333^{***}	-0.0027	-0.0849^{***}	-0.0027^{*}	$-1.89E^{-05}$
		(0.0035)	(0.0013)	(0.0053)	(0.0020)	(0.0034)	(0.0013)	($1.44E^{-05}$)
LEARN	READ	0.2457^{***}	-0.0213^{***}	-0.0010	-0.0283^{***}	-0.0003	-0.0069^{***}	0.0006^{***}
		(0.0042)	(0.0012)	(0.0036)	(0.0019)	(0.0009)	(0.0005)	($5.25E^{-05}$)
LEARN	MATH	0.1915^{***}	-0.0174^{***}	-0.0125^{**}	-0.0247^{***}	-0.0024^{**}	-0.0045^{***}	0.0004^{***}
		(0.0033)	(0.0010)	(0.0041)	(0.0015)	(0.0008)	(0.0003)	($3.59E^{-05}$)
EXTERN	INTERP	-0.5352^{***}	-0.0297^{***}	-0.1798^{***}	-0.0066^{***}	0.0962^{***}	0.0089^{***}	0.0002^{***}
		(0.0043)	(0.0016)	(0.0046)	(0.0018)	(0.0026)	(0.0011)	($5.45E^{-05}$)
EXTERN	READ	-0.0396^{***}	-0.0058^{***}	-0.0681^{***}	-0.0451^{***}	0.0027^{***}	0.0022^{***}	0.0003^{***}
		(0.0047)	(0.0013)	(0.0036)	(0.0019)	(0.0004)	(0.0003)	($5.97E^{-05}$)
EXTERN	MATH	-0.0315^{***}	-0.0035^{**}	-0.0892^{***}	-0.0329^{***}	0.0081^{***}	0.0014^{***}	0.0001^{**}
		(0.0037)	(0.0012)	(0.0040)	(0.0015)	(0.0004)	(0.0002)	($3.98E^{-05}$)

The M-COT model is unique among techniques designed to assess mediation in longitudinal data because it assesses mediation cross sectionally, but the cross sectional associations are not necessarily stable over time. It is assumed that there is some short-term process that has reached equilibrium at each measurement occasion, but a longer-term process that is not stationary. This model might prove useful in explaining conflicting results in developmental studies that have examined mediation at different ages or across different timespans. The model specified here is a $1 \rightarrow 1 \rightarrow 1$ mediation model, but the general framework could be easily adapted to accommodate individual-level predictor and mediator variables at L2. The model was also limited to linear trends over time for both the *a* and *b* paths. A simulation study is currently underway to examine more complex trends for *a* and *b* as well as ideal methods for determining the form of the *a* and *b* trends in real world data where the true data generating process is not known. The current model allows intercepts and time trends to vary across individuals but assumes that the *a* and *b* paths are fixed. If the data warrant, these assumptions could be removed by incorporating methods similar to those of Bauer et al. (2006). Finally, critiques about assessing mediation cross sectionally could be addressed by estimating lagged relations among variables (e.g., $X1 \rightarrow$ M2 $\rightarrow$ Y3; $X2 \rightarrow$ M3 $\rightarrow$ Y4; $X3 \rightarrow$ M4 $\rightarrow$ Y5) rather than the cross sectional associations as shown here.

Longitudinal Mediation in Autoregressive Panel Models

When *X*, *M*, and *Y* are measured repeatedly over time, autoregressive panel data models can be extended to the three variable case to investigate longitudinal mediation. When the autoregressive mediation model involves two waves of data, fitting the autoregressive models to estimate mediation effect is somewhat limited, in that either the relation between *X* and *M* or the relation between *M* and *Y* is contemporaneous. Gollob and Reichardt (1991) argued that it takes time for a cause to have an effect and thus, at least $k + 2$ waves of data are needed to estimate indirect effects in a model with a chain of *k* mediators. Thus, with a single mediator, data should ideally be collected at least across three waves or time points, so *X* can affect *M* and *M* can affect *Y* in time. Otherwise, some parameter constraints or assumptions are needed, based on theory and prior knowledge (Gollob & Reichardt, 1991), such as assumptions of stationarity and knowledge of test-retest reliability.

Figure 21.13 presents an example of the autoregressive mediation model with two waves of data, where the effect of *X* on *M* and the effect of *M* on *Y* are specified as lag-1 effects in an SEM. The autoregressive models described in this section are based on the observed variables, ignoring the measurement errors. With inclusion of multiple

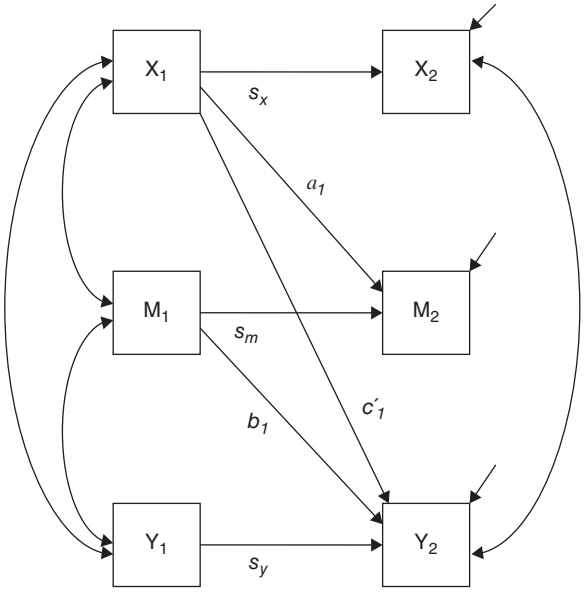


Figure 21.13 An autoregressive model with longitudinal mediation across two waves.

indicators, the repeated measures of X, M, and Y can be modeled as latent variables and the indirect effects can be estimated based on the lagged effects between the latent variables. The model in Figure 21.13 can be described by equations 21.62 to 21.64.

$$X_2 = i_{x2} + s_x X_1 + e_{x2} \tag{21.62}$$

$$M_2 = i_{m2} + a_1 X_1 + s_m M_1 + e_{m2} \tag{21.63}$$

$$Y_2 = i_{y2} + b_1 M_1 + c'_1 X_1 + s_y Y_1 + e_{y2} \tag{21.64}$$

where the variables measured at time 1 are treated as predetermined as follows:

$$X_1 = i_{x1} + e_{x1} \tag{21.65}$$

$$M_1 = i_{m1} + e_{m1} \tag{21.66}$$

$$Y_1 = i_{y1} + e_{y1} \tag{21.67}$$

where i_{x1}, i_{m1}, and i_{y1} are the means of X_1, M_1, and Y_1, respectively, and e_{x1}, e_{m1}, and e_{y1} are deviations from the means. Typically, X_1, M_1, and Y_1 are specified to be correlated. As described previously, i_{m2} and i_{y2} in equations 21.63 and 21.64 are regression intercepts, and e_{m2} and e_{y2} are residuals. The coefficients a_1 and b_1 represent the effect of X_1 on M_2 and the effect of M_1 on Y_2, respectively, while the coefficient c'_1 denotes the direct effect of X_1 on Y_2. The product of the estimates of the coefficients a_1 and b_1, i.e., $a_1 b_1$, is the estimated longitudinal indirect effect. Although the coefficient b_1 represents the relation between M and Y before X affects M, the $a_1 b_1$ estimator would be a reasonable estimate of the longitudinal indirect, because under the stationarity assumption the relation between M_1 and Y_2 (b_1) would be equal to the relation between M_2 and Y_3 that would have been measured after the second wave (Cole & Maxwell, 2003). The standard error of the estimated indirect effect can be obtained using the formulas described earlier based on the relevant path coefficients and their standard errors to conduct the significance test and compute confidence intervals.

Another example of the autoregressive mediation model with two wave data is the model in Figure 21.14 (Mackinnon, 2008). The relations relevant to the mediational pathways can be expressed by the following equations 21.68 and 21.69:

$$M_2 = i_{m2} + a_1 X_1 + a_2 X_2 + s_m M_1 + e_{m2} \tag{21.68}$$

$$Y_2 = i_{y2} + b_1 M_1 + b_2 M_2 + c'_1 X_1 + c'_2 X_2 + s_y Y_1 + e_{y2} \tag{21.69}$$

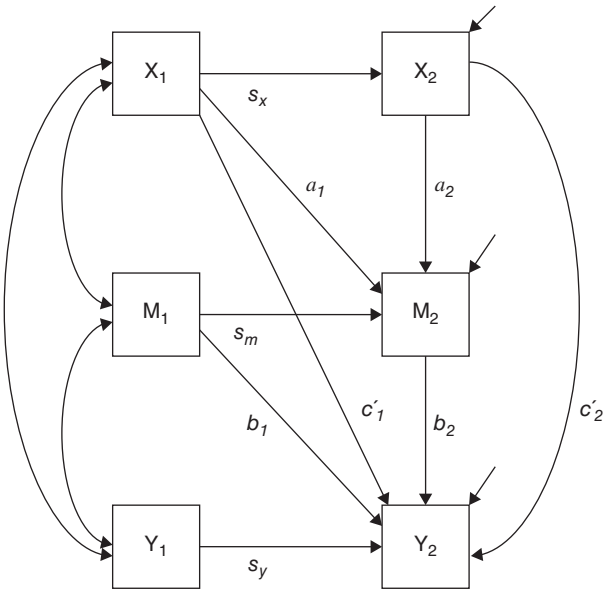


Figure 21.14 An autoregressive mediation model with longitudinal and contemporaneous mediation across two waves.

With the addition of the contemporaneous relations (a_2, b_2, and c'_2) to the previous model in Figure 21.13, the coefficient a_1 in the above model represents the effect of X_1 on M_2 after controlling for the effects of X_2 and M_1, while the coefficient a_2 represents the effect of X_2 on M_2 after controlling for the effects of X_1 and M_1. Similarly, the coefficients b_1 and b_2 indicate the effect of M_1 on Y_2 after controlling for the effects of X_1, X_2, M_2, and Y_1 and the effect of M_2 on Y_2 after controlling for the effects of X_1, X_2, M_1, and Y_1, respectively. There are several ways to estimate mediated effects in the model in Figure 21.14: $a_1 b_1$, $a_2 b_2$, and $a_1 b_2$. As described earlier, the indirect effect $a_1 b_1$ is based on the across-time or lag-1 relations, while the indirect effect $a_2 b_2$ is estimated based on the contemporaneous relations among the variables measured at time 2. The indirect effect $a_1 b_2$ is based on the across-time relation between X_1 and M_2 and the contemporaneous relation between M_2 and Y_2. Cole and Maxwell (2003) recommend the $a_1 b_1$ estimator, because the estimates $a_2 b_2$ and $a_1 b_2$ involve cross sectional relations and are likely to be biased when time is needed for the cause to exert its effect on the outcome (Gollob & Reichardt, 1987).

In the two-wave autoregressive mediation models, information on stability, stationarity, and equilibrium is quite limited, in that we cannot actually test whether these properties hold across time. However, estimating autoregressive effects is beneficial for avoiding omitted variable bias. An autoregressive effect is defined as the effect that a variable has on itself at a later time (Gollob & Reichardt, 1987) and

is denoted by the coefficients s_x, s_m, and s_y in the above models. These coefficients also estimate the stability of a variable across time (Kenny, 1979). When the dependent variable at time t is predicted by the predictors at time $t - 1$, the levels of the dependent variable at time $t - 1$ may represent unmeasured variables that are correlated with the predictors and estimating autoregressive relations can control for such unmeasured variables (Gollob & Reichardt, 1991). Without controlling for those omitted variables or confounders, the causal effects will be biased. Thus, the estimates of a_1 and a_2 in Figures 21.13 and 21.14 will be less biased when estimating s_m and so will b_1 and b_2, when estimating s_y. These less biased coefficients in the mediational pathways will result in the indirect effect that is closer to the true effect size.

When X, M, and Y are measured repeatedly over three or more occasions, the parameterizations in the autoregressive models become more flexible. For example, the effect of M_2 on Y_3 that relied on the stationarity assumption in the two wave data (b_1 in Figures 21.13 and 21.14) can now be estimated. With three or more waves of data, estimating lag effects according to the causal order of X to M to Y is possible, although timing of measurements is a remaining issue, because it is difficult to predict how long it takes for one variable to have its optimal effect on another. Having more waves of data also provides opportunities to examine when stability, stationarity, and equilibrium are reached. It may take a long time for a system of X, M, and Y to achieve stability, stationarity, or equilibrium and sometimes it may be achieved beyond the time span the data cover. While these characteristics can only be assumed in the cross sectional or two wave designs, researchers can check these assumptions with more waves of data by testing equivalence of parameter estimates across waves. Finally, time specific indirect effects can be estimated across multiple waves. The magnitude of effect, either the autoregressive effects or the lagged effects, may vary across time (Cole & Maxwell, 2003). The effect of X_1 on M_2 may be larger or smaller than the effect of X_2 on M_3 and the resulting indirect effects may differ depending on which time points are involved (Cole & Maxwell, 2003; Gollob & Reichardt, 1987, 1991). It might be an interesting research task to detect when the significant indirect effect arises, holds, and desists.

Figure 21.15 shows an example of the autoregressive mediation model with four waves of data, in which the relations among X, M, and Y are specified as lag-1 effects. The model in Figure 21.15 can be described in the following equations:

$$\mathrm{X}_2 = i_{x2} + s_{x1}\mathrm{X}_1 + e_{x2} \tag{21.70}$$

$$\mathrm{X}_3 = i_{x3} + s_{x2}\mathrm{X}_2 + e_{x3} \tag{21.71}$$

$$\mathrm{X}_4 = i_{x4} + s_{x3}\mathrm{X}_3 + e_{x4} \tag{21.72}$$

$$\mathrm{M}_2 = i_{m2} + a_1\mathrm{X}_1 + s_{m1}\mathrm{M}_1 + e_{m2} \tag{21.73}$$

$$\mathrm{M}_3 = i_{m3} + a_2\mathrm{X}_2 + s_{m2}\mathrm{M}_2 + e_{m3} \tag{21.74}$$

$$\mathrm{M}_4 = i_{m4} + a_3\mathrm{X}_3 + s_{m3}\mathrm{M}_3 + e_{m4} \tag{21.75}$$

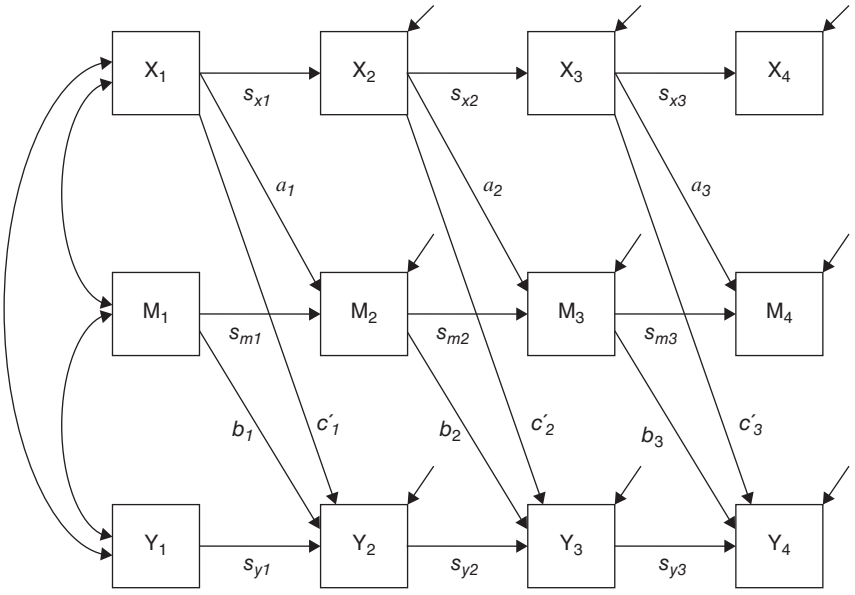


Figure 21.15 An autoregressive mediation model with longitudinal mediation across four waves.

$$\mathrm{Y}_2 = i_{y_2} + b_1\mathrm{M}_1 + c'_1\mathrm{X}_1 + s_{y_1}\mathrm{Y}_1 + e_{y_2} \quad (21.76)$$

$$\mathrm{Y}_3 = i_{y_3} + b_2\mathrm{M}_2 + c'_2\mathrm{X}_2 + s_{y_2}\mathrm{Y}_2 + e_{y_3} \quad (21.77)$$

$$\mathrm{Y}_4 = i_{y_4} + b_3\mathrm{M}_3 + c'_3\mathrm{X}_3 + s_{y_3}\mathrm{Y}_3 + e_{y_4} \quad (21.78)$$

Again, the variables measured at time 1 are predetermined in the same way as in equations 21.65 to 21.67. With more than two waves of data, the effect of X on M and the effect of M on Y are represented by more than one path coefficient. In Figure 21.15 and equations 21.70 to 21.78, the coefficients a_1, a_2, and a_3 all represent the time-specific effect of X on M. Similarly, the coefficients b_1, b_2, and b_3 represent the time-specific effect of M on Y controlling for X, although the coefficients b_2 and b_3 are the effects of M that were affected by X, while b_1 is the effect of M before X exerts its effect on M. The coefficients c'_1, c'_2, and c'_3 reflect the time-specific direct effects of X on Y.

The indirect effects for the model in Figure 21.15 can be estimated in several ways. As we did for the two wave data, one can estimate the indirect effect for each lag such as a_1b_1, a_2b_2, and a_3b_3. Another option is to estimate the indirect effect reflecting the temporal ordering of X, M, and Y, such as a_1b_2 and a_2b_3. The indirect effect and its standard error can be obtained following the formulas described earlier and can be used for significance testing and construction of confidence limits. If the stationarity assumption holds across 4 waves, all the a paths will be equivalent and so will the b paths. In such case, the estimated indirect effects (i.e., a_1b_1, a_2b_2, a_3b_3, a_1b_2, a_2b_3) will be the same. On the other hand, if the path coefficients vary across waves, the indirect effects will be different depending on the time points involved and specific indirect effects can be examined. For example, if four measurement occasions span across different developmental phases, the indirect effect observed at the earlier phase may differ from the indirect effect at the later phase. Differences in the indirect effects across different phases may be an interesting research question. If the autoregressive mediation model is estimated in an SEM framework, the equivalence of path coefficients across time can be tested and the parameters can be constrained accordingly before obtaining the indirect effect.

In addition to the indirect effects listed already that are based on the two longitudinal relations between $X_t \rightarrow M_{t+1}$ and $M_t \rightarrow Y_{t+1}$ (e.g., a_1b_1, a_2b_2, a_3b_3, a_1b_2, a_2b_3), there are other time-specific indirect effects in the model in Figure 21.15, such as $s_{x1}a_2b_3$ and $a_1b_2s_{y3}$. These individual indirect effects are time-specific indirect effects, as they represent causal mechanisms from X to M to Y for specific time intervals. Gollob and Reichardt (1991) argued that these individual time-specific indirect effects may be of less interest to researchers because mediation may evolve gradually over the course of time, rather than occurring at a specific time point. The overall indirect effect may then provide a better estimate for the indirect effect. The *overall indirect effect* is defined as the sum of all the *individual time-specific indirect effects,* i.e., all the indirect effects from X at time 1 to Y at the last time point mediated by M at any time between the first and the last time points (Gollob & Reichardt, 1991). In the model in Figure 21.15, there are three such individual time-specific indirect effects: (1) $X_1 \rightarrow X_2 \rightarrow M_3 \rightarrow Y_4$ ($s_{x1}a_2b_3$), (2) $X_1 \rightarrow M_2 \rightarrow M_3 \rightarrow Y_4$ ($a_1s_{m2}b_3$), and (3) $X_1 \rightarrow M_2 \rightarrow Y_3 \rightarrow Y_4$ ($a_1b_2s_{y3}$). Computing the total indirect effect and its standard error, as well as the total direct effect and its standard error, is available in most SEM software programs, including EQS (Bentler & Wu, 2006), LISREL (Jöreskog & Sörbom, 2006), Proc CALIS in SAS (SAS Institute, 2014), and Mplus (Muthén & Muthén, 2014). It is worth noting that the accuracy of the total indirect effect would rely on the specified mediation model, however. For example, if the mediation model in Figure 21.15 is a true model but the measurement at time 2 is omitted, resulting in the model with data measured at time 1, 3, and 4, the estimated total indirect effect will not equal the true overall indirect effect. See Gollob and Reichardt (1991) for illustration.

We fit an autoregressive panel SEM with longitudinal mediation paths to first, third, and fifth grade ECLS-K data. Though it is possible to fit autoregressive models with unequal lags between time points (Voelkle, Oud, Davidov, & Schmidt, 2012), for the sake of simplicity, only the first, third, and fifth grade measures with constant 2-year lags are used in this example. Table 21.8 presents

TABLE 21.8 Coefficients and Standard Errors for Tests of Longitudinal Mediation in an Autoregressive Panel Model

*X*1	*M*3	*Y*5	*a*	***b***	Med
LEARN	INTERP	INTERN	0.1985***	−0.0063	−0.0013
			(0.0131)	(0.0112)	(0.0022)
LEARN	READ	INTERN	0.0514***	−0.1674***	−0.0086***
			(0.0035)	(0.0204)	(0.0012)
LEARN	MATH	INTERN	0.0511***	−0.1270***	−0.0065***
			(0.0041)	(0.0156)	(0.0010)
EXTERN	INTERP	INTERN	−0.3229***	−0.0959***	0.0310***
			(0.0135)	(0.0091)	(0.0032)
EXTERN	READ	INTERN	−0.0268***	−0.2468***	0.0066***
			(0.0037)	(0.0203)	(0.0011)
EXTERN	MATH	INTERN	−0.0157***	−0.1829***	0.0029***
			(0.0043)	(0.0155)	(0.0008)

coefficients and standard errors for a, b, and the longitudinal mediated effect from ADHD symptoms at first grade through interpersonal skills and academic performance at third grade on internalizing behavior at fifth grade. The results indicate significant longitudinal mediated effects of LEARN on INTERN through both READ and MATH (but not INTERP) and longitudinal effects of EXTERN on INTERN through all three mediators. However, CFI and RMSEA values indicated that these models did not fit the data well.

Another type of autoregressive mediation model is shown in Figure 21.16 with the mediational pathways described in equations 21.79 to 21.84:

$$M_2 = i_{m2} + a_1 X_1 + a_4 X_2 + s_{m1} M_1 + e_{m2} \quad (21.79)$$

$$M_3 = i_{m3} + a_2 X_2 + a_5 X_3 + s_{m2} M_2 + e_{m3} \quad (21.80)$$

$$M_4 = i_{m4} + a_3 X_3 + a_6 X_4 + s_{m3} M_3 + e_{m4} \quad (21.81)$$

$$Y_2 = i_{y2} + b_1 M_1 + b_4 M_2 + c'_1 X_1 + c'_4 X_2 + s_{y1} Y_1 + e_{y2} \quad (21.82)$$

$$Y_3 = i_{y3} + b_2 M_2 + b_5 M_3 + c'_2 X_2 + c'_5 X_3 + s_{y2} Y_2 + e_{y3} \quad (21.83)$$

$$Y_4 = i_{y4} + b_3 M_3 + b_6 M_4 + c'_3 X_3 + c'_6 X_4 + s_{y3} Y_3 + e_{y4} \quad (21.84)$$

In addition to the longitudinal relations between X, M, and Y specified in Figure 21.15, the model in Figure 21.16 includes the contemporaneous relations among X, M, and Y at each wave. These relations are estimated by the coefficients a_4, a_5, and a_6 for the relations between X and M at Time 2, Time 3, and Time 4, respectively, and the coefficients b_4, b_5, and b_6 for the relations between M and Y. The product of path coefficients for the contemporaneous relations, such as a_4b_4, a_5b_5, and a_6b_6, provide the estimates of the contemporaneous indirect effect. When X and M have immediate effects, the longitudinal relations may miss the real effects due to the time lag between measurements. In such cases, the contemporaneous indirect effects may be closer to the true indirect effects (MacKinnon, 2008).

We fit models including both longitudinal and contemporaneous mediation pathways to the ECLS-K first through fifth grade data. The results are summarized in Table 21.9. These models fit the data better than the previous set that included only the longitudinal pathways (all CFIs > .95), but RMSEA values were in the .08–.10 range, which is still larger than desired for optimal fit. Longitudinal mediated paths (X1 → $M3$ → $Y5$) involving the INTERP and READ (but not MATH) mediators were significant or marginally significant and in the expected direction. Several mediated effects involving contemporaneous a or b paths were also significant in models including the INTERP mediator and READ mediators.

Although having more than two waves for the autoregressive models may allow for the flexibility in model specification and the opportunities to obtain more detailed information about the indirect effects, it may introduce

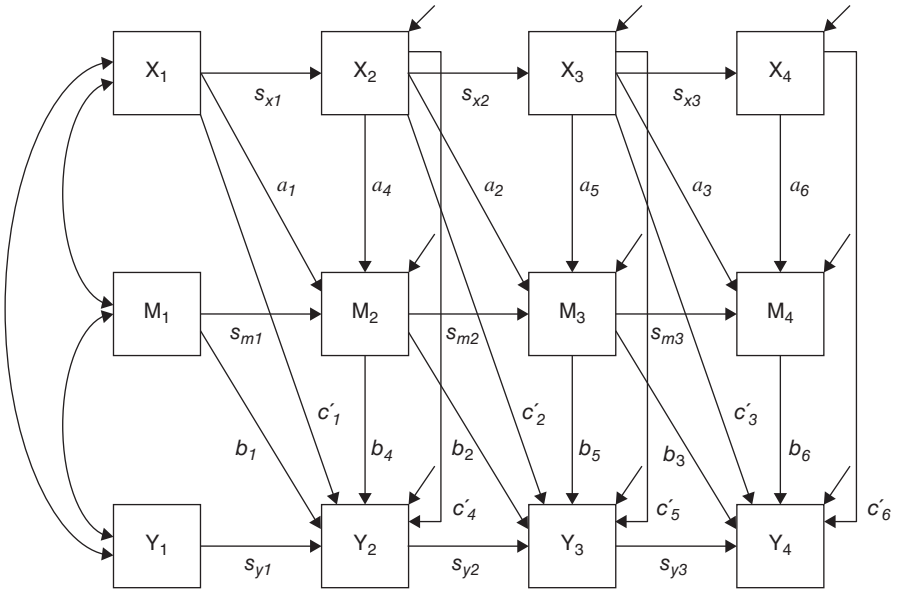


Figure 21.16 An autoregressive mediation model with longitudinal and contemporaneous mediation across four waves.

TABLE 21.9 Coefficients and Standard Errors for Tests of Longitudinal and Contemporaneous Mediation in an Autoregressive Panel Model

	X = LEARN			X = EXTERN		
$X \rightarrow$ INTERP $\rightarrow$ INTERN	a	b	Med	a	b	Med
$X1 \rightarrow M2 \rightarrow Y2$	-0.1535^{***}	-0.1326^{***}	0.0204^{***}	-0.0308^{*}	-0.2076^{***}	0.0064^{*}
	(0.0108)	(0.0118)	(0.0023)	(0.0128)	(0.0109)	(0.0027)
$X1 \rightarrow M2 \rightarrow Y3$	-0.1535^{***}	0.0400^{***}	-0.0061^{**}	-0.0308^{*}	-0.0255^{*}	$0.0008^{\dagger}$
	(0.0108)	(0.0122)	(0.0019)	(0.0128)	(0.0113)	(0.0005)
$X2 \rightarrow M2 \rightarrow Y2$	0.6912^{***}	-0.1326^{***}	-0.0917^{***}	-0.6232^{***}	-0.2076^{***}	0.1294^{***}
	(0.0087)	(0.0118)	(0.0082)	(0.0110)	(0.0109)	(0.0072)
$X2 \rightarrow M2 \rightarrow Y3$	0.6912^{***}	0.0400^{***}	0.0276^{**}	-0.6232^{***}	-0.0255^{*}	0.0159^{*}
	(0.0087)	(0.0122)	(0.0084)	(0.0110)	(0.0113)	(0.0070)
$X2 \rightarrow M3 \rightarrow Y3$	-0.1445^{***}	-0.1435^{***}	0.0207^{***}	-0.0057	-0.2161^{***}	0.0012
	(0.0113)	(0.0124)	(0.0024)	(0.0135)	(0.0110)	(0.0029)
$X3 \rightarrow M3 \rightarrow Y3$	0.6698^{***}	-0.1435^{***}	-0.0961^{***}	-0.5954^{***}	-0.2161^{***}	0.1287^{***}
	(0.0086)	(0.0124)	(0.0084)	(0.0115)	(0.0110)	(0.0070)
$X \rightarrow$ READ $\rightarrow$ INTERN						
$X1 \rightarrow M2 \rightarrow Y2$	0.0232^{***}	-0.0172	-0.0004	-0.0083	-0.1257^{***}	$0.0010^{\dagger}$
	(0.0040)	(0.0273)	(0.0006)	(0.0044)	(0.0274)	(0.0006)
$X1 \rightarrow M2 \rightarrow Y3$	0.0232^{***}	-0.1066^{***}	-0.0025^{*}	-0.0083	-0.1675^{***}	$0.0014^{\dagger}$
	(0.0040)	(0.0340)	(0.0009)	(0.0044)	(0.0350)	(0.0008)
$X2 \rightarrow M2 \rightarrow Y2$	0.0618^{***}	-0.0172	-0.0011	-0.0365^{***}	-0.1257^{***}	-0.0046^{***}
	(0.0038)	(0.0273)	(0.0017)	(0.0045)	(0.0274)	(0.0011)
$X2 \rightarrow M2 \rightarrow Y3$	0.0618^{***}	-0.1066^{***}	-0.0066^{**}	-0.0365^{***}	-0.1675^{***}	0.0061^{***}
	(0.0038)	(0.0340)	(0.0021)	(0.0045)	(0.0350)	(0.0015)
$X2 \rightarrow M3 \rightarrow Y3$	0.0144^{***}	0.0472	0.0007	-0.0022	-0.0428	0.0001
	(0.0031)	(0.0356)	(0.0005)	(0.0035)	(0.0362)	(0.0002)
$X3 \rightarrow M3 \rightarrow Y3$	0.0302^{***}	0.0472	0.0014	-0.0198^{***}	-0.0428	0.0008
	(0.0030)	(0.0356)	(0.0011)	(0.0036)	(0.0362)	(0.0007)
$X \rightarrow$ MATH $\rightarrow$ INTERN						
$X1 \rightarrow M2 \rightarrow Y2$	0.0246^{***}	-0.0373	-0.0009	0.0048	-0.1095^{***}	-0.0005
	(0.0047)	(0.0228)	(0.0006)	(0.0051)	(0.0229)	(0.0006)
$X1 \rightarrow M2 \rightarrow Y3$	0.0246^{***}	-0.0461	-0.0011	0.0048	-0.0781^{**}	-0.0004
	(0.0047)	(0.0289)	(0.0007)	(0.0051)	(0.0297)	(0.0004)
$X2 \rightarrow M2 \rightarrow Y2$	0.0569^{***}	-0.0373	-0.0021	-0.0400^{***}	-0.1095^{***}	0.0044^{***}
	(0.0045)	(0.0228)	(0.0013)	(0.0052)	(0.0229)	(0.0011)
$X2 \rightarrow M2 \rightarrow Y3$	0.0569^{***}	-0.0461	-0.0026	-0.0400^{***}	-0.0781^{**}	0.0031^{*}
	(0.0045)	(0.0289)	(0.0005)	(0.0052)	(0.0297)	(0.0013)
$X2 \rightarrow M3 \rightarrow Y3$	0.0160^{***}	-0.0190	-0.0003	-0.0083	-0.0894^{**}	0.0007
	(0.0039)	(0.0278)	(0.0005)	(0.0044)	(0.0282)	(0.0005)
$X3 \rightarrow M3 \rightarrow Y3$	0.0364^{***}	-0.0190	-0.0007	-0.0222^{***}	-0.0894^{**}	0.0020^{**}
	(0.0038)	(0.0278)	(0.0010)	(0.0045)	(0.0282)	(0.0007)

complexity of parameterization. For example, a variant of the model in Figure 21.16 may include cross-lagged effects among X, M, and Y by adding the direct effects from M_{t-1} to X_t and Y_{t-1} to M_t based on the temporal sequence in which the variables are measured. Specifying these cross-lagged relations may reflect data more reasonably than the models in Figure 21.15 and 21.16, because X, M, and Y are related to each other. However, this model violates the causal sequence of X to M to Y specified by the mediation model because of the reversed paths from M to X and Y to M. As expected, choosing a correct model can be a tricky task with more parameters to be estimated. When the models are estimated in the SEM framework, it would be appropriate to estimate a series of nested models and conduct a series of chi-square difference tests to choose a model that better reflects the data, as well as the theory. In addition, equality of path coefficients, such as paths for the effects of X on M (i.e., a_1, a_2, and a_3) can be also tested by comparing the models with and without the equality constraints.

Latent Growth Curve Mediation Models

The latent growth curve (LGC) modeling (Meredith & Tisak, 1990; Muthén & Curran, 1997; Willett & Sayer, 1994) is another approach that can be used to evaluate longitudinal mediation models when X, M, and Y are repeatedly measured across three or more time points (Cheong, 2011; Cheong, MacKinnon, & Khoo, 2003; Mackinnon, 2008). Unlike the autoregressive mediation models, where the indirect effect is based on the differences among the study participants, the indirect effect in

the LGC mediation model is conceptualized in terms of within-individual changes. In the autoregressive mediation models, the indirect effect is interpreted as the expected effect of X on Y at time t through M at time $t-1$, if there was one unit change in X prior to time $t-1$. Here, the changes in X, M, and Y are assumed equivalent to differences among participants in those variables. In LGC mediation models, individuals' trajectories of X, M, and Y are estimated using the repeated measures across multiple measurement time points and the indirect effect is determined based on the relations between the within-individual changes in X, M, and Y.

The capability of modeling mediation in terms of within-individual changes is important in developmental research, where the core research questions center on development, which is typically defined as within-individual change, and individual differences in development (Nesselroade, 1991). Using difference scores in a two-wave design is the simplest way of measuring individual change; however, difference scores contain minimal information about individual change (Rogosa, 1988; Rogosa, Brandt, & Zimowski, 1982; Willett & Sayer, 1994). The change patterns may follow nonlinear (e.g., quadratic or exponential) patterns or fluctuate in level between the two measurement time points. Growth curve modeling is considered a better method for investigating longitudinal within-individual changes and individual differences in those changes (Rogosa, 1988; Rogosa et al., 1982). Although growth curve models can be formulated both in the multilevel modeling framework (Raudenbush & Bryk, 2002; Goldstein, 1995) and in the SEM framework (Meredith & Tisak, 1990; Muthén & Curran, 1997; Willett & Sayer, 1994), we focus on the SEM approach in this section.

In the LGC approach to mediation, the mediation process is typically modeled using a parallel process model (Muthén & Curran, 1997; Stoolmiller, 1994). The repeated measures of X, M, and Y are modeled as distinctive growth processes and the mediation is specified in the relations among the growth factors. As it is recommended that researchers investigate the longitudinal pattern of change in each variable before examining the longitudinal relations between the variables (Raudenbush, 2001; Rogosa, 1988), the estimation of growth trajectories that best describe the changes in X, M, and Y should precede modeling mediation based on the relations among the latent growth factors. In this approach, the growth trajectories of X, M, and Y are modeled in the measurement model, where the repeated measures relate to the latent growth factors according to the specified time scale. Once the growth trajectory shape is appropriately modeled for each X, M, and Y, the full parallel process model is specified to model the mediation process in the structural model among the latent growth factors.

Figure 21.17 shows an example of the LGC mediation model, where the growth trajectories of X, M, and Y are modeled as linear, based on repeated measures obtained with equal intervals across three waves. According to the specified time scales, the intercept factors (i.e., η_1, η_3, and η_5) represent the level of each variable at time 1, i.e., the initial status. The slope factors (i.e., η_2, η_4, and η_6) represent the change or growth rate of each variable per time unit. The mean and the variance of the intercept factor estimate the average level of initial status of each variable and the individual differences in the initial status. The mean and the variance of the latent slope factor estimate the average growth rate of each variable and the individual differences in the growth rates. Details of model specifications for LGC models can be found elsewhere (e.g., Muthén & Khoo, 1998; Willett & Sayer, 1994). The mediation process is specified in the relations among the latent factors shown in the middle of the model in Figure 21.17. More specifically, the individual differences in the initial status and the growth rates of M and Y are modeled as functions of the individual differences in the initial status and the growth rates of X and M.

The relations among the latent factors in Figure 21.17 are described in the following equations:

$$\eta_{3i} = \pi_{0\mathrm{m}} + d_1\eta_{1i} + e_{3i} \tag{21.85}$$

$$\eta_{4i} = \pi_{1\mathrm{m}} + a\eta_{2i} + d_2\eta_{1i} + e_{4i} \tag{21.86}$$

$$\eta_{5i} = \pi_{0\mathrm{y}} + d_3\eta_{1i} + d_5\eta_{3i} + e_{5i} \tag{21.87}$$

$$\eta_{6i} = \pi_{1\mathrm{y}} + b\eta_{4i} + c'\eta_{2i} + d_4\eta_{1i} + d_6\eta_{3i} + e_{6i} \tag{21.88}$$

where η_{1i}, η_{3i}, η_{5i} are the intercept factors and η_{2i}, η_{4i}, η_{6i} are slope factors for the trajectory of X, M, and Y, respectively, varying across individuals indicated by the subscript i. In the previous equations, $\pi_{0\mathrm{m}}$, $\pi_{0\mathrm{y}}$, $\pi_{1\mathrm{m}}$, and $\pi_{1\mathrm{y}}$ are the regression intercepts of the growth factors and e_{3i}, e_{4i}, e_{5i}, and e_{6i} are residuals of the growth factors. Equations 21.85 and 21.87 describe the intercept factors of M and Y, respectively, as functions of the intercept factors of X and M. These directional paths in Figure 21.17 are based on the causal theory of X to M to Y. If the first measures of X, M, and Y are obtained simultaneously, they can be specified as covariances. Equation 21.86 models the slope factor of M as a function of the intercept and the slope factors of

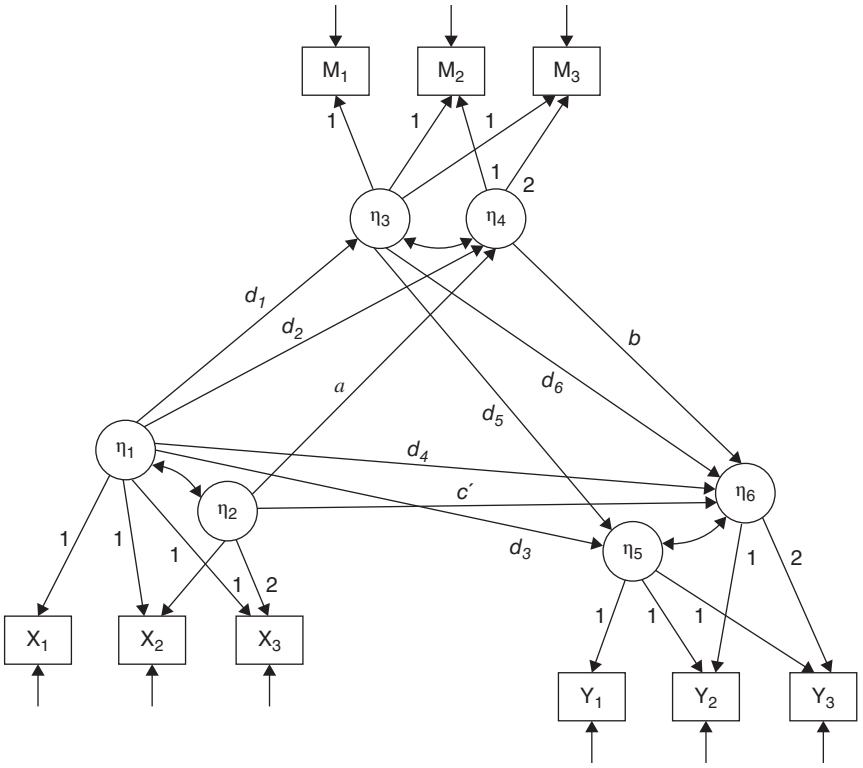


Figure 21.17 A latent growth curve mediation model across three waves.

X, while equation 21.88 specifies the slope factor of *Y* predicted by the intercept and the slope factors of *X* and *M*. Although there are several possible indirect effects that can be of interest in this model, the main focus is on the relations among the slope factors, as the benefit of the LGC approach is the capability of modeling mediation in terms of within-individual changes in *X*, *M*, and *Y*. The coefficient *a* represents the effect of the growth rate of *X* on the growth rate of *M*. The coefficient *b* represents the effect of the growth rate of *M* on the growth rate on *Y*. The product of the two coefficients (i.e., *ab*) provides the estimate of the indirect effect that is based on the within-individual changes, determined by the extent to which the change in *X* affects the change in *M* (*a*) and the extent to which the change in *M* affects the change in *Y* (*b*). The standard error of the indirect effect and its standard error can be obtained in the same way described earlier and used for the significance test of the indirect effect.

We fit a series of simple LGC mediation models to ECLS-K data from all 5 waves. Though preliminary models suggested that growth models did not fit the ADHD symptoms, interpersonal skill, and internalizing behavior data well, we nonetheless proceeded with the mediation models for the sake of illustration, specifying linear trajectories for the *X*, *M*, and *Y* variables. Unsurprisingly, we encountered frequent convergence problems; estimation of the variance of the slope of the mediator trajectory seemed particularly problematic. This variance was estimated as negative in the LEARN → INTERP → INTERN model and was very close to zero in the LEARN → READ → INTERN and EXTERN → MATH → INTERN models. It was necessary to constrain this parameter (and its covariance with the intercept of the mediator trajectory) to zero to achieve convergence in the LEARN → MATH → INTERN and EXTERN → INTERP → INTERN models. The EXTERN → READ → INTERN model did not converge, even with this constraint. Both the CFI and RMSEA indicate poor fit for all models. Tests of the mediated effects involving predictor, mediator, and outcome slopes are reported in Table 21.10. For all models that converged, the estimate of the *a* path relating the slope of the predictor to the slope of the mediator was significant, though the relation between the slopes of LEARN and MATH was counterintuitively negative. Only two *b* path estimates were significant: the slope of READ was negatively related to the slope of INTERN, and the slope of INTERP was counterintuitively positively related to the slope of INTERN. Tests of the *ab* estimates of mediated effects show significant mediation in the LEARN → READ → INTERN and EXTERN → INTERP → INTERN models, but the

TABLE 21.10 Coefficients and Standard Errors in an LGM Mediation Model

X	*M*	*Y*	*a*	*b*	Med
LEARN	INTERP	INTERN	1.0538***	−0.0501	−0.0528
			(0.0229)	(0.0335)	(0.0353)
LEARN	READ	INTERN	0.1491***	−0.1429***	−0.0213***
			(0.0087)	(0.0307)	(0.0047)
LEARN	MATH	INTERN	−0.1135***	−.01165	0.0013
			(0.0143)	(0.1490)	(0.0169)
EXTERN	INTERP	INTERN	−1.0272***	3.7495***	−3.851***
			(0.0242)	(0.0507)	(0.1046)
EXTERN	READ	INTERN	Did not converge.		
EXTERN	MATH	INTERN	−0.1056***	0.0796	−0.0084
			(0.0113)	(0.0409)	(0.0044)

mediated effect in the latter model is in a counterintuitive direction. Though LGC mediation models can be very useful in explicating mediation effects in longitudinal data, this particular technique does not seem appropriate for this particular data, as a growth framework is not well suited to capture change over time in most of the variables. As mentioned earlier, it is required to inspect the pattern of change in the study variables carefully before putting the trajectories together in a comprehensive LGC mediation model.

When the measures of *X*, *M*, and *Y* across waves are concurrent, the relations between the slope factors in the mediation model may be interpreted as correlational because the growth rates are estimated based on the measures across the same time span. However, this is less problematic than purely cross sectional data, because the growth rates are estimated based on the entire time span that the data cover and the effects of the changes in the predictors at earlier waves would be reflected in the changes in the outcome variables at later waves. An alternative method is to estimate growth trajectories in two-stage piecewise growth curve models (Raudenbush & Bryk, 2002; Khoo, 2001), where distinct growth trajectories are specified for early and later phases and the time ordering of the growth trajectories of *X*, *M*, and *Y* can be modeled accordingly. The indirect effects then can be estimated for different phases, such as the indirect effect via the earlier growth of *M* on the earlier growth of *Y*, the indirect effect via the earlier growth of *M* on the later growth of *Y*, and the indirect effect via the later growth of *M* on the later growth of *Y*.

Estimating LGC Mediation as a Multilevel Model

The LGC mediation models discussed in the previous section were estimated using structural equation modeling techniques. The SEM framework is ideally suited for such analyses, as its ability to estimate a number of simultaneous equations allows for the estimation of predictive and outcome effects of the same variable within a single analysis. For example, in a mediational analysis, the mediator serves as an outcome variable when examining the *a* path and as a predictor variable when examining the *b* path. It is relatively simple within this framework to have one arrow pointing to a variable (i.e., that variable is an outcome) and another pointing from that variable (i.e., that variable is a predictor of another variable) in an SEM diagram and to create the appropriate equations to estimate both effects simultaneously.

The mediated effect involving latent slope parameters in Figure 21.17 could potentially also be tested within a multilevel modeling framework. The difficulty here is that latent growth in *X* is a predictor of latent growth in *M*, and that latent growth in *M* is a predictor of latent growth in *Y*. In a single multilevel model, it is simple to allow a variable to predict the growth parameter—just include that predictor in the L2 equation predicting that parameter—but there is no obvious way to expand the model to allow the growth parameter itself to predict another variable. However, after running a multilevel model, a researcher can obtain Empirical Bayesian (EB) estimates of growth parameters for each individual. These estimates combine individual-specific information from a single subject's own data and the normative trajectory for the entire sample to produce stable and reliable estimates of person-specific effects (Candel & Winkens, 2003). The EB estimates of intercepts and slopes of the *X* variable can be entered as L2 predictors of the growth parameters in a multilevel growth analysis of the mediator variable to produce estimates of the d_1, d_2, and *a* parameters in Figure 21.17. Similarly, EB estimates of intercepts and slopes can be obtained when running the growth analysis for the mediator variable, and these (along with the EB estimates of intercepts and slopes from the first analysis) can be entered as L2 predictors of the intercept and slope parameters in the multilevel growth analysis of the outcome variable to produce the

remaining coefficients in Figure 21.17, including the b coefficient that relates the mediator slope to the outcome slope. Conducting multiple multilevel analyses seems a bit less elegant than the single comprehensive SEM, but equivalent estimates are produced, and thus longitudinal mediation can be assessed in either framework.

Another possibility for recasting the LGC mediation model in the multilevel framework involves including all three variables (X, M and Y) in a single multivariate multilevel model (Hox, 2010; Raudenbush & Bryk, 2002). One way of conceptualizing such a model is as a three-level framework, with the variables distinguished at L1, repeated measures at L2, and individual subjects at L3. With three measurement occasions as shown in Figure 21.17, the data set would be organized with nine lines of data per subject, one for each of the X, M and Y variables at each of the three time points. The actual X, M, and Y values would be entered into a single generically named variable, V. Three dummy variables (D_1, D_2, and D_3) would be created to identify which observations were associated with each of the three variables, and these dummies, in conjunction with a variable indicating the time point of measurement, would uniquely identify each of the nine unique values.

The L1 model would consist of the three dummy variables, two of which will be zero for any given observation:

$$\text{L1:}\quad V_{vti} = \pi_{1ti}D_{1ti} + \pi_{2ti}D_{2ti} + \pi_{3ti}D_{3ti} \qquad (21.89)$$

for variable v at time point t for subject i. Notice that there is no intercept and no error term in this equation. An intercept would imply that predictors could affect all observations equally, and we wish to keep X, M and Y distinct in this model. An error term is unnecessary because this level of the model simply sorts the variables by type, which should involve no error.

The L2 models would be:

$$\begin{aligned}\text{L2:}\quad \pi_{1ti} &= \beta_{10i} + \beta_{11i}time_{ti} + r_{1ti}\\ \pi_{2ti} &= \beta_{20i} + \beta_{21i}time_{ti} + r_{2ti}\\ \pi_{3ti} &= \beta_{30i} + \beta_{31i}time_{ti} + r_{3ti}\end{aligned} \qquad (21.90)$$

and at L3:

$$\begin{aligned}\text{L3:}\quad \beta_{10i} &= \gamma_{100} + u_{10i} \quad \beta_{11i} = \gamma_{110} + u_{11i}\\ \beta_{20i} &= \gamma_{200} + u_{20i} \quad \beta_{21i} = \gamma_{210} + u_{21i}\\ \beta_{30i} &= \gamma_{300} + u_{30i} \quad \beta_{11i} = \gamma_{310} + u_{31i}\end{aligned} \qquad (21.91)$$

These equations at each level can be algebraically combined to give the single-equation form of the model:

$$\begin{aligned}V_{vti} = {} & D_{1ti}(\gamma_{100} + \gamma_{110}time_{ti} + r_{1ti} + u_{10i} + u_{11i}time_{ti})\\ & + D_{2ti}(\gamma_{200} + \gamma_{210}time_{ti} + r_{2ti} + u_{20i} + u_{21i}time_{ti})\\ & + D_{3ti}(\gamma_{300} + \gamma_{310}time_{ti} + r_{3ti} + u_{30i} + u_{31i}time_{ti})\end{aligned} \qquad (21.92)$$

This is exactly the same as running three separate multilevel growth models on the X, M, and Y variables, but all three equations are combined into a single analysis. The advantage of the single analysis is that the L3 variance–covariance matrix of the u's will be 6×6 and will contain off-diagonal elements that represent associations between growth in X and growth in M (a correlational version of the a path in Figure 21.17) and between growth in M and growth in Y (a correlational version of the b path in Figure 21.17).

Because there is no error term at L1, this multivariate analysis could alternately be specified as a two-level model. This is actually the more practical specification, as it is quite difficult to suppress the L1 error term or constrain it to zero in most multilevel software packages. In separate equation form, the L1 model would be

$$\begin{aligned}\text{L1:}\quad V_{ti} = {} & \beta_{10i}D_{1ti} + \beta_{11i}D_{1ti}time_{ti} + \beta_{20i}D_{2ti}\\ & + \beta_{21i}D_{2ti}time_{ti} + \beta_{30i}D_{3ti}\\ & + \beta_{31i}D_{3ti}time_{ti} + r_{1ti} + r_{2ti} + r_{3ti}\end{aligned}$$

Again, the specification contains no explicit intercept. The three dummy variables represent intercepts for their specific variables. Moreover, the time variable is entered in interaction with each of the dummy variables. These interactions represent the three slopes associated with the X, M, and Y variables. In addition, this equation includes three distinct error terms. This model could be estimated, for example, in SAS Proc Mixed (SAS Institute, 2014), which has the facility to estimate multiple L1 error terms corresponding to the distinct levels of a L1 variable with the group = option. A single variable distinguishing X, M, and Y with uniquely coded values would need to be created to accomplish this. The L2 specification for this model would be identical to the L3 specification of the three-level multivariate model and thus the single-equation forms of both models are identical.

In the general multivariate multilevel model, associations among slopes are correlational and thus nondirectional. Mediation analyses, however, typically involve

directional hypotheses ($X \rightarrow M$ and $M \rightarrow Y$). Examining such directional effects in the longitudinal mediation model requires the ability to include latent variables on the predictor side of the equation. The HLM program incorporates just such facility into the HLM2, HLM3, MLM, and MLM2 modules (Raudenbush, Bryk, Cheong, Congdon, & du Toit, 2011). The HLM manual provides an example in which a latent intercept is used to predict a latent slope. The method described in the manual could easily be expanded to simultaneously estimate growth parameters for more than one outcome variable and allow the slope of one to predict the slope of another. However, the latent variable modeling window in HLM currently allows each latent variable to be used as either a predictor or an outcome, but not both. So in a multivariate model including multiple measurements of X and M, this program could allow the slope of X to predict the slope of M, producing a coefficient and standard error for the a path in Figure 21.17. However, because M cannot simultaneously serve as both an outcome (on the a path from the X slope) and a predictor (on the b path to the Y slope), it would still be necessary to run a second multivariate multilevel analysis with multiple measurements of X, M, and Y to allow the slopes of X and M to predict the slope of Y to produce the b and c' coefficients in Figure 21.17. So directional coefficients can be produced, and a mediated effect can be computed and tested from these, but incorporating the entire process into a single analysis will need to wait for further development of multilevel software.

Autoregressive Latent Trajectory Mediation Models

Integrating the unique strengths of the autoregressive models and the LGC models, Bollen and his colleagues (Bollen & Curran, 2004; Bollen & Zimmer, 2010) proposed the autoregressive latent trajectory (ALT) model. The ALT model parameterizes autoregressive relations between the repeated measures as in the autoregressive models, while simultaneously estimating the growth trajectories based on the pattern of changes across time. The multivariate application of the ALT model allows for parameterization of the autoregressive relations within each variable, the lagged relations between variables at specific measurement occasions, and the relations between the within-individual changes in those variables. Applying ALT models to investigation of mediation, researchers may be able to capture time-specific indirect effects as well as indirect effects based on the growth trajectories that are estimated across the entire time span the data cover.

In the ALT model, the measures of X, M, and Y at time 1 are treated as predetermined and including this predetermined time 1 measure takes care of the biases resulting from omitted variables prior to the time span the data cover. Individual trajectories in the ALT model are estimated based on the measures from time 2 to time t. The autoregressive relations within each variable are specified between the repeated measures from time 1 to time t. Thus, the measure at time t for $t \geq 2$ is specified as the function of the variable itself measured at time $t - 1$ and the trajectory components (i.e., intercept and slope factors) estimated based on the variable measured from time 2 to time t. For example, X_2 is determined by X_1 and the intercept factor, while X_3 is determined by X_2, the intercept factor, and the slope factor. The intercept factor in the ALT model captures the portion of the measure at time 2 that remains unexplained by the time 1 measure and the slope factor represents the growth rate estimated based on time 2 to time t measures. For an ALT model to be identified, four or more waves of data are needed (Bollen & Curran, 2006). Details about the ALT model specifications can be found elsewhere (Bollen & Curran, 2004; Bollen & Zimmer, 2010).

The model in Figure 21.18 depicts an example of the ALT mediation model, where X, M, and Y are measured across four time points. The lag-1 effects between X, M, and Y at each wave are shown in the middle of the figure relating X_{t-1} to M_t to Y_{t+1}. The mediation relations among the individual trajectories are represented by the dotted lines connecting directionally between the intercept factors and the slope factors of X, M, and Y. The time specific indirect effects are estimated based on the lagged effects between the repeated measures of X, M, and Y (e.g., $X_1 \rightarrow M_2$ and $M_2 \rightarrow Y_3$) as in the autoregressive mediation models and the indirect effect based on the within-individual changes in X, Y, and M is estimated in the relations between the slope factors (i.e., $\eta_2 \rightarrow \eta_4 \rightarrow \eta_6$) as in the LGC mediation models. These two different types of indirect effects are independent of each other, as the autoregressive and the lagged effects are net of individual trajectory components in the ALT models. As expected, model specification can be complicated and it may take several steps to determine the appropriate final model; however, the ALT models can provide unique opportunities to simultaneously estimate and separate out the time-specific and the within-individual change based indirect effects in one comprehensive model. Given the difficulties encountered in estimating the LGC mediation models in the ECLS-K data, we did not explore ALT models for our particular example.

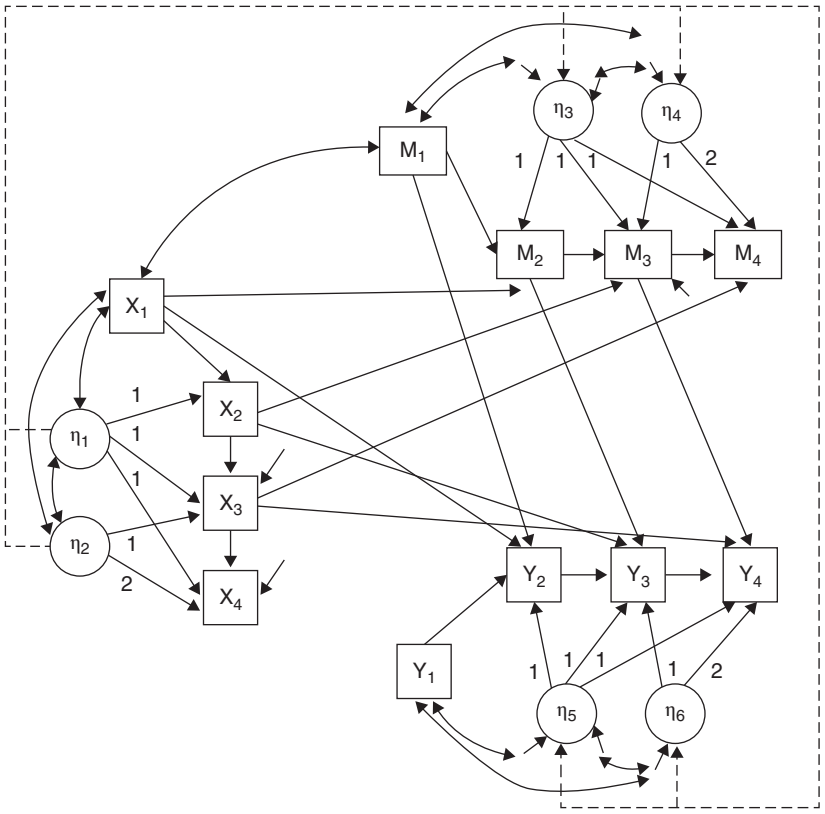


Figure 21.18 An autoregressive latent trajectory mediation model across four waves.

Latent Change Score Models for Moderation and Mediation

Before describing moderation and mediation in latent variable difference score models, a brief word is needed about manifest variable difference score models for longitudinal data. With two waves of data, MacKinnon (2008) suggests forming change scores for *X*, *M*, and *Y*, substituting the difference scores into the equations for the single mediator, and then testing for mediation using one of the cross sectional tests of mediation mentioned earlier. For three or more waves of data, difference scores may be computed and placed into an OLS autoregressive model to test for mediation. Although the steps for fitting the model and testing for mediation are the same as in the autoregressive mediation model, the interpretation of the results is quite different. Instead of a variable at one wave influencing another variable at a later wave, now it is the difference in scores on a variable across two waves influencing the difference in the scores of another variable across two later waves.

With a categorical moderator variable, the simplest method to test for moderation in a LCS model would be to run a multiple group analysis and compare the parameter estimates in the different groups. Depending on the model constraints, this would test whether the proportional or constant change between groups was different or could even test whether one group changed proportionally while another changed constantly or did not change. For observed continuous time-invariant predictors, the latent change score parameters can be regressed onto both predictors and the interaction term. Interactions between latent time-invariant predictors or between a latent time-invariant predictor and a latent change score parameter are possible as well, but require more advanced methods; Marsh, Wen, and Hau (2006) provided an overview of some of these methods.

There are several ways an LCS model can be incorporated into a mediation model. Figure 21.19 shows one example of a LCS longitudinal mediation model where the mediated effect is composed of the slopes of the individual LCS models. That is, the slope of the change in *X* predicts the slope of the change in *M*, which in turn affects slope of the change in *Y*. This is comparable to the parallel process LGC model described by Cheong et al. (2003) because the processes are occurring at the same time. If the processes were measured after one another, the then a serial process LCS model could be specified.

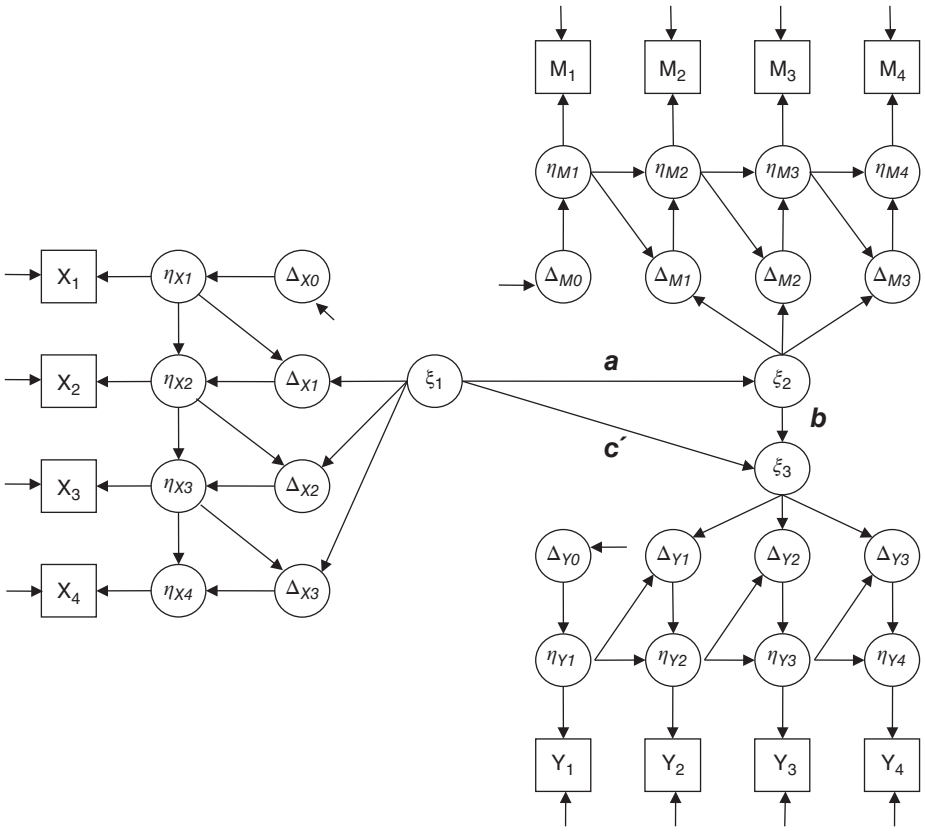


Figure 21.19 Parallel process latent change score mediation model.

MacKinnon (2008) described a different LCS longitudinal mediation model, shown in Figure 21.20. In this model, rather than have the slopes of the change in individual variables predict one another, the error-free true score η variables from one variable predict the latent change scores, Δ, in the other variable. For instance, η_{x1} predicts Δ_{m1} and η_{m2} predicts Δ_{y3}. This model is closer to an autoregressive model, because the variables only affect other variables at later waves. Note that the mean structure has been omitted from Figures 21.19 and 21.20 for ease of presentation. Additionally, an LCS model with *a* paths linking change in the predictor to change in the mediator (i.e., from Δ'_xs to Δ'_ms) and *b* paths linking change in the mediator to change in the outcome (i.e., from Δ'_ms to Δ'_ys) could plausibly be used to test longitudinal mediation.

The process of fitting the LCS mediation models in Figures 21.19 and 21.20 is similar to fitting a LGC mediation model (MacKinnon, 2008). The first step is to fit the individual LCS models for *X*, *M*, and *Y*. This allows the researcher to specify the best fitting LCS model for each variable, such as determining whether a dual change or proportional change model fits better. The second step is to add the mediation paths using the structural model. As in the LGC mediation models, other paths may be appropriate, such as a path from η_{x1} to the Δ_{y2}, which is similar to the lag 1 direct effects from the autoregressive model. The Chi-square goodness of fit test, the RMSEA, and the CFI may be used to assess overall model fit.

Testing for mediation can be accomplished in several ways. First, the joint significance test could be used by examining the significance of each of the individual paths. Second, specific indirect paths can be examined by using a MODEL INDIRECT command in the Mplus program and requesting bootstrap confidence intervals. As with the autoregressive models and LGC, the overall indirect effect can be examined using EQS, SAS PROC CALIS, and LISREL, but only Mplus will test specific indirect effects for significance. For in depth examples of fitting LCS models with LISREL see McArdle (2001) and Ferrer and McArdle (2003).

We fit LCS mediation models to the first, third, and fifth grade ECLS-K data. Table 21.11 presents the results of the parallel process analysis similar to Figure 21.19. All six

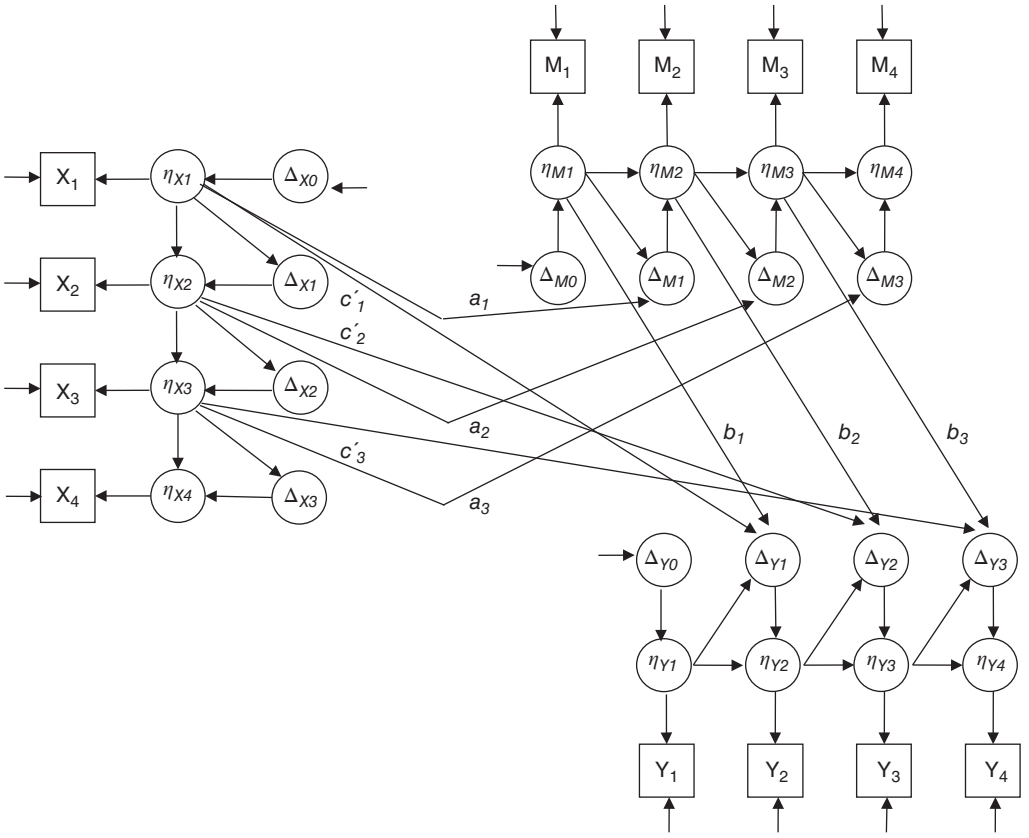


Figure 21.20 Alternate parallel process latent change score mediation model. Source: Adapted from MacKinnon (2008).

TABLE 21.11 Coefficients and Standard Errors in LCS Parallel Process Mediation Models

X	*M*	*Y*	*a*	*b*	Med
LEARN	INTERP	INTERN	1.1302***	−0.6494***	−0.7340***
			(0.0231)	(0.0323)	(0.0394)
LEARN	READ	INTERN	0.2639***	0.6345***	0.1674***
			(0.0245)	(0.0667)	(0.0235)
LEARN	MATH	INTERN	0.2226***	−0.0155	−0.0035
			(0.0130)	(0.0153)	(0.0034)
EXTERN	INTERP	INTERN	1.6164***	−0.0035	−0.0057
			(0.1587)	(0.1162)	(0.1878)
EXTERN	READ	INTERN	−0.2297***	0.2966***	−0.0681***
			(0.0201)	(0.0302)	(0.0091)
EXTERN	MATH	INTERN	−0.1864***	0.1590***	−0.0296***
			(0.0226)	(0.0130)	(0.0043)

models included significant *j* and *k* coefficients for *X*, *M*, and *Y*, indicating both proportional and constant change over time for all constructs. RMSEA and CFI values for all models indicated less than ideal fit, particularly for models with interpersonal skills as the mediator. The tests of mediation in Table 21.11 indicate that the slopes of change in interpersonal skills and reading performance significantly mediated the association between the slope of change in approaches to learning and internalizing behavior. Also, the slopes of change in both academic performance variables mediated the association between the slope of change in externalizing behavior and the slope of change in internalizing behavior. The direction of the mediated effect, however, was counterintuitive for three of the four mediated pathways.

A second series of LCS mediation models was fitted to the first through fifth grade ECLS-K data in which true scores predicted latent change as in Figure 21.20. The results are reported in Table 21.12. Because three time points were included in the analysis, two distinct *a* and two distinct *b* paths were estimated in each model. a_1 and b_1 examine effects of first grade true scores on latent change from first to third grade, and a_2 and b_2 examine effects of third grade true scores on latent change from third to fifth grade. RMSEA and CFI values indicated less than ideal fit in all models, especially for those involving the interpersonal skills mediator. The tests of mediation in Table 21.12 indicate a significant a_1b_1 estimate of mediation

TABLE 21.12 Coefficients and Standard Errors in LCS Mediation Models with Paths from True Scores to Latent Change

X	M	Y	a_1	a_2	b_1	b_2	Med_{a1a2}	Med_{a1b2}	Med_{a2b2}
LEARN	INTERP	INTERN	1.0144***	0.9676***	1.1118***	0.0227	1.1278***	0.0230	0.0220
			(0.0435)	(0.0469)	(0.1107)	(0.0445)	(0.1223)	(0.0452)	(0.0431)
LEARN	READ	INTERN	0.0004	0.0770**	0.0360	0.0813	0.0001	0.0001	0.0063
			(0.0059)	(0.0057)	(0.0317)	(0.0437)	(0.0002)	(0.0005)	(0.0034)
LEARN	MATH	INTERN	−0.0170**	0.0143**	−0.0008	0.0764**	0.0001	−0.0013	.0011
			(0.0066)	(0.0068)	(0.0318)	(0.0310)	(0.0005)	(0.0007)	(.0007)
EXTERN	INTERP	INTERN	−0.9607***	−0.7689***	−0.0449**	0.3928**	0.0431**	−0.3774**	−0.3020**
			(0.0179)	(0.0253)	(0.0200)	(0.1272)	(0.0192)	(0.1224)	(0.0983)
EXTERN	READ	INTERN	−0.0053	−0.0374***	0.0286	0.1117**	−0.0002	−0.0006	−0.0042**
			(0.0061)	(0.0064)	(0.0268)	(0.0348)	(.0002)	(.0007)	(.0015)
EXTERN	MATH	INTERN	0.0057	−0.0137	−0.0033	0.0998***	−0.0001	0.0006	−0.0014
			(0.0070)	(0.0074)	(0.0266)	(0.0266)	(.0002)	(.0007)	(0.0008)

for LEARN → INTERP → INTERN, but the temporally ordered a_1b_2 estimate was nonsignificant. All three possible estimates of mediated effects for EXTERN → INTERP → INTERN were significant. The a_2b_2 estimate in the EXTERN → READ → INTERN model was the only significant mediated effect involving the academic performance variables.

Moderation and Mediation in Exponential Decay Models

Similar to standard multilevel models, moderation in an exponential decay multilevel growth model is accomplished by adding the moderating variable and appropriate interaction term at the correct level. If the moderator is a time-varying predictor, Z_{ti}, and interacts with another time-varying predictor, X_{ti}, that is not time, then both the predictors and the interaction term, $X_{ti}Z_{ti}$, would be added at L1 in equation 21.35.

$$
\begin{aligned}
\text{L1:}\quad & Y_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i})\exp[\beta_{1i}t] \\
& \quad + \beta_{3i}X_{ti} + \beta_{4i}Z_{ti} + \beta_{5i}Z_{ti}X_{ti} + r_{ti} \\
\text{L2:}\quad & \beta_{0i} = \gamma_{00} + u_{0i} \\
& \beta_{1i} = \gamma_{10} + u_{1i} \\
& \beta_{2i} = \gamma_{20} + u_{2i} \\
& \beta_{3i} = \gamma_{30} + u_{3i} \\
& \beta_{4i} = \gamma_{40} + u_{4i} \\
& \beta_{5i} = \gamma_{50} + u_{5i}
\end{aligned}
\tag{21.93}
$$

Because of the nonlinear relation between time and the outcome variable, a linear interaction between time and a time-varying covariate is difficult to conceptualize and will not be discussed for the multilevel or latent variable exponential decay models.

If the moderator is a time-invariant predictor, Z_i, of one of the growth parameters, for example β_0, and interacts with another time-invariant predictor of the same growth parameter, X_i, then both the predictors and the interaction, X_iZ_i, between these predictors would be added to the corresponding L2 equations in equation 21.35.

$$
\begin{aligned}
\text{L1:}\quad & Y_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i})\exp[\beta_{1i}t] + r_{ti} \\
\text{L2:}\quad & \beta_{0i} = \gamma_{00} + \gamma_{01}X_i + \gamma_{02}Z_i + \gamma_{03}X_iZ_i + u_{0i} \\
& \beta_{1i} = \gamma_{10} + u_{1i} \\
& \beta_{2i} = \gamma_{20} + u_{2i}
\end{aligned}
\tag{21.94}
$$

L2 interactions may be of more interest for the exponential decay model as they test whether the effect of a time-invariant predictor on the initial value, rate of change, and asymptote value is dependent on the value of the moderator. For the latent variable exponential decay model with a time-invariant categorical moderator variable, the simplest method to test for moderation would be to run a multiple group analysis and compare the parameter estimates in the different groups. This would test whether the initial value, rate of change, and asymptote value were the same for the different groups. For observed continuous time-invariant predictors, the latent growth parameters can be regressed onto both the predictors and the interaction term. As with the LCS model, interactions between latent time-invariant predictors or between a latent time-invariant predictor and a latent growth parameter are possible for the latent exponential decay model, but require more advanced methods described by Marsh et al. (2006).

If one of the variables is a time-varying predictor, X_{ti}, and the other is a time-invariant predictor, Z_i, then

a cross-level interaction can be estimated by including the time-varying predictor at L1 and including the time-invariant variable as a predictor of the time-varying variable in the L2 equation in equation 21.35.

$$\begin{aligned}
\text{L1:}\quad & Y_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i})\exp[\beta_{1i}t] + \beta_{3i}X_{ti} + r_{ti} \\
\text{L2:}\quad & \beta_{0i} = \gamma_{00} + u_{0i} \\
& \beta_{1i} = \gamma_{10} + u_{1i} \\
& \beta_{2i} = \gamma_{20} + \gamma_{21}Z_i + u_{2i} \\
& \beta_{3i} = \gamma_{30} + \gamma_{31}Z_i + u_{3i}
\end{aligned} \tag{21.95}$$

This also allows for interactions between time-invariant predictors and time if Z_i is added as a predictor of β_1. Note that the time-invariant predictor must also be included as an L2 predictor of β_2 in order for the first-order effect Z_i to be estimated in the model, which is necessary for the correct estimation of the interaction term $X_{ti}Z_i$.

Though not technically an interaction, differential effects based on the relations between the growth parameters can be examined using the off-diagonal elements of the covariance matrix of the random effects, $\boldsymbol{\tau}$. For example, if higher initial values were associated with higher asymptote values, then τ_{02} would be positive and significantly different from zero. The same association can be examined in the latent variable exponential decay using the covariances in the $\boldsymbol{\Phi}$ matrix.

The first step in constructing an exponential decay multilevel mediation model is to rewrite the equations from the linear single mediator model in the form of equation 21.35. When M and Y decay exponentially and X is a continuous variable, X is added at L1 of equation 21.35 and the effect of X on M becomes

$$\begin{aligned}
\text{L1:}\quad & M_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i})\exp[\beta_{1i}t] + a_iX_{ti} + r_{ti} \\
\text{L2:}\quad & \beta_{0i} = \gamma_{00} + u_{0i} \\
& \beta_{1i} = \gamma_{10} + u_{1i} \\
& \beta_{2i} = \gamma_{20} + u_{2i} \\
& a_i = \gamma_{30}
\end{aligned} \tag{21.96}$$

where γ_{00}, γ_{10}, and γ_{20} represent the mean values across subjects for the initial value, rate of change, and asymptote for M partialling out the effect of X, respectively, and γ_{30} is the effect of X partialling out the effect of time (i.e., the natural exponential decay of M). Because M is also continuous, X and M are added at L1 of equation 21.35 and the effect of X and M on Y becomes

$$\begin{aligned}
\text{Level1:}\quad & Y_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i})\exp[\beta_{1i}t] + b_iM_{ti} \\
& \qquad + c'_iX_{it} + r_{ti} \\
\text{Level2:}\quad & \beta_{0i} = \gamma_{00} + u_{0i} \\
& \beta_{1i} = \gamma_{10} + u_{1i} \\
& \beta_{2i} = \gamma_{20} + u_{2i} \\
& b_i = \gamma_{30} \\
& c'_i = \gamma_{40}
\end{aligned} \tag{21.97}$$

where γ_{00}, γ_{10}, and γ_{20} represent the mean values across subjects for the initial value, rate of change, and asymptote for Y partialling out the effects of X and M, respectively, γ_{30} is the effect of M partialling out the effects of time (i.e., the natural exponential decay of Y) and X, and γ_{40} is the effect of X partialling out the effects of time and M. Krull and MacKinnon (2001) call this a $1 \rightarrow 1 \rightarrow 1$ model because all three variables occur at L1.

Here the three mediation parameters, a_i, b_i, and c'_i, have all been treated as fixed effects, meaning that the mediation effects do not vary between individuals. Adding random effects for these three paths would not change the interpretations of the coefficients, but it would indicate that the effect differs among individuals, an indication of the presence of moderation. For example, if an L2 predictor was added that predicted b_i, such as a baseline value of M, this would create a cross-level interaction between that predictor and M, meaning the predictor of b_i is a moderator of the effect of M on Y. For more information on moderation of a mediated effect see Fairchild and MacKinnon (2009); for more information on mediation at different levels see Bauer et al. (2006), Kenny et al. (2003), Krull and MacKinnon (2001), and Preacher et al. (2010).

When X is a time-invariant variable, such as random assignment to a treatment, instead of entering X as an L1 covariate, X must be entered into the multilevel model at L2. The multilevel equations for the model of X on M then change from equation 21.95 to

$$\begin{aligned}
\text{L1:}\quad & M_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i})\exp[\beta_{1i}t] + r_{ti} \\
\text{L2:}\quad & \beta_{0i} = \gamma_{00} + a_0X_i + u_{0i} \\
& \beta_{1i} = \gamma_{10} + a_1X_i + u_{1i} \\
& \beta_{2i} = \gamma_{20} + a_2X_i + u_{2i}
\end{aligned} \tag{21.98}$$

Assuming X is a dichotomous dummy coded variable, then γ_{00}, γ_{10}, and γ_{20}, are estimates of the initial value, rate of change, and asymptote of M for the group coded 0, respectively, and a_0, a_1, and a_2 are the differences between the two groups in X such that $\gamma_{00} + a_0$, $\gamma_{10} + a_1$, and $\gamma_{20} + a_2$ are the initial value, rate of change, and asymptote of M for the group coded 1, respectively.

The effects of M and X on Y change from equation 21.96 to

$$\begin{aligned}
\text{L1:} \quad & Y_{ti} = \beta_{2i} + (\beta_{0i} - \beta_{2i}) \exp[\beta_{1i} t_t] + b_i M_{ti} + r_{ti} \\
\text{L2:} \quad & \beta_{0i} = \gamma_{00} + c_0' X_i + u_{0i} \\
& \beta_{1i} = \gamma_{10} + c_1' X_i + u_{0i} \\
& \beta_{2i} = \gamma_{20} + c_2' X_i + u_{0i} \\
& b_i = \gamma_{30}
\end{aligned} \tag{21.99}$$

Again assuming X is a dichotomous dummy coded variable, γ_{00}, γ_{10}, and γ_{20}, are estimates of the initial value, rate of change, and asymptote of Y for the group coded 0, partialling out the effect of M, respectively, c_0', c_1', and c_2' are the differences between the two groups in X partialling out the effect of M, such that $\gamma_{00} + c_0'$, $\gamma_{10} + c_1'$, and $\gamma_{20} + c_2'$ are the asymptote value, initial value, and rate of change of Y for the group coded 1, respectively, and γ_{30} is the effect of M partialling out the effect of time (i.e., the natural exponential decay of Y) and the effect of X on the exponential decay of Y. Krull and MacKinnon (2001) call this a $2 \rightarrow 1 \rightarrow 1$ model because X is at L2 while M and Y are at L1.

Adding M as a time-varying predictor in at L1 so that the model estimates the relation between M and Y at the same point in time violates the temporal precedence assumption. An alternate method would be to introduce a lag such that M predicts Y at later time points. With a lag of one wave, M_{it} in equation 21.98 would change to $M_{(t-1)i}$, so that each measurement of M predicts the measurement of Y at the next time point. Then b_i would be the average relation across time between M at time t and Y at time $(t + 1)$, partialling out the effect of time. One issue with lags is that M must be measured prior to Y. Also, although the model makes no assumption that the measurements of M or Y are equally spaced, when the relation between M and Y is lagged, if the measurements are not equally spaced, b_i will represent the average effect across different amounts of time. For example, if M and Y were measured at 1, 3, and 12 months, introducing a lag of one wave would mean that $M_{1\text{month}}$ would predict $Y_{3\text{months}}$, a lag of 2 months, while $M_{3\text{months}}$ would predict $Y_{12\text{months}}$, a lag of 9 months, an undesirable situation.

The value of M immediately following an intervention could also be entered as an L2 predictor of the rate of change and asymptote of Y. If X had a significant effect on the initial value of M, which in turn predicted the rate of change and asymptote of Y, then the effect of X on the rate of change and asymptote of Y would be mediated through its initial effect on M. This does not satisfy temporal precedence since the initial value of Y is used to estimate the rate of change and asymptote of Y, but it does make a stronger case for mediation than in equation 21.98.

Using the exponential decay model shown in equations 21.95 and 21.96, when X and M are added to the model at Level 1, mediation can be tested using the joint significance test by testing a and b for significance. The mediated effect $a_i b_i$ can also be tested by dividing it by the standard error

$$\begin{aligned}
Var(\widehat{ab} + \hat{\sigma}_{aj,bj}) = {} & b^2 Var(\hat{a}) + a^2 Var(\hat{b}) + Var(\hat{a}) Var(\hat{b}) \\
& + 2ab Cov(\widehat{ab}) + Cov(\widehat{ab})^2 + Var(\hat{\sigma}_{aj,bj})
\end{aligned} \tag{21.100}$$

that includes the covariance of the a and b parameters (Bauer et al., 2006; see also Kenny et al., 2003; Krull & MacKinnon, 1999, 2001; MacKinnon, 2008). However, this assumes that the distribution of the mediated effect is normal, which is often not the case. For this reason, creating a confidence interval around the mediated effect using the percentile bootstrap may be preferred.

Testing for mediation when X is entered at L2 such as in equations 21.97 and 21.98 is slightly different than for the continuous L1 X variable model in equations 21.95 and 21.96 as there are now three individual effects of X on M, a_0, a_1, and a_2. Each of these parameters represents a different effect of X on M, and because of this, three different mediated effects can be estimated, $a_0 b_i$, $a_1 b_i$, and $a_2 b_i$, all of which may be tested for significance using one of the methods described earlier. As discussed during the presentation of autoregressive models, each of these mediated effects represents a specific indirect effect, any or all of which may be of substantive interest.

Several different exponential decay mediation models are possible when using an LGC approach. The first is a direct extension of the model in equations 21.95 and 21.96. Illustrated in Figure 21.21, this model begins by fitting separate exponential decay LGC models for X, M, and Y. Then the observed M variables are regressed on the observed X variables and the observed Y variables are

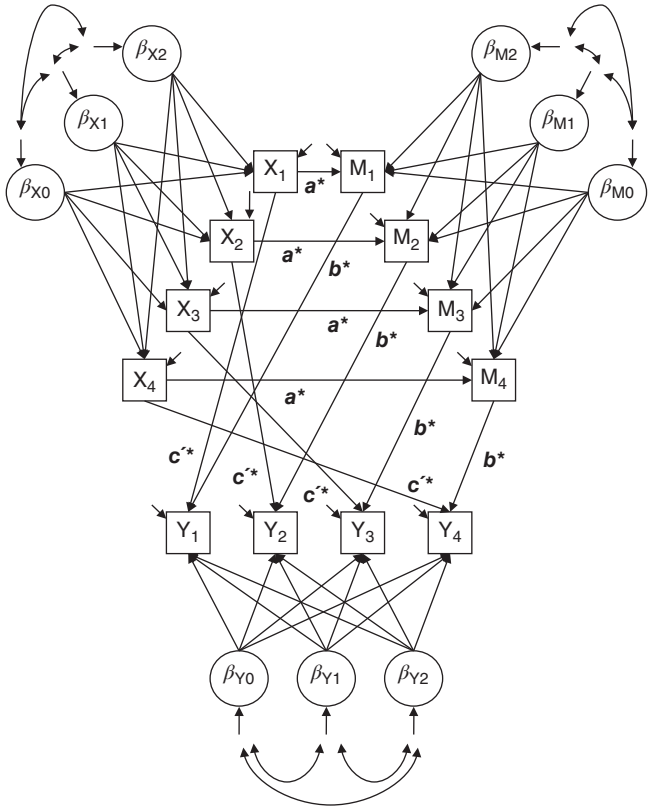


Figure 21.21 An exponential decay latent growth curve model with a continuous X variable and direct paths between observed variables.

regressed on the observed X and M variables, constraining all of the paths between sets of observed variables to be equal. For the dichotomous X variable model in equations 21.97 and 21.98, separate exponential decay LGC models are fit for M and Y, and the observed Y variables are regressed on the observed M variables, again constraining these paths to be equal. The latent growth parameters from M and Y are then regressed onto X.

Another form of exponential decay LGC mediation model is a parallel process model. As was described earlier, an exponential decay LGC model is equivalent to a linear LGC model, only with an exponential decay trajectory across time. Hence, the individual linear LGC models in the parallel process LGC mediation model may be replaced with exponential decay LGC models to form a parallel process LED mediation model, as shown in Figure 21.22.

The parallel process mediation model in Figure 21.22 only contains paths between the rate of change growth parameters. However, depending on the theory behind the model, other paths may be appropriate. For instance, if X were dichotomous and had an effect on the initial value of M, then it may be of interest to include a path between the initial value of M and the rate of change of Y. Or, continuing with the dichotomous X example, if M reached asymptote before Y, it could be of interest to investigate whether the asymptote of M had an effect on the asymptote of Y. Testing for mediation would be conducted similarly to the parallel process LGC by testing the significance of individual paths or by testing specific indirect paths for significance. In addition, various fit indices given by most structural equation modeling software can be used to assess overall model fit.

Testing for mediation in the exponential decay LGC model is similar to testing for mediation in the multilevel exponential decay model in that the meditational parameters are tested for significance or individual indirect effects are directly tested for significance. One advantage of the latent variable framework is the addition of fit indices, which are provided by most SEM software packages and provide valuable information about the overall fit of the model and the presence of a significant mediated effect. Another advantage is the ability to model measurement error and have multiple indicators for a variable. Finally, the latent variable framework allows for the correlation of residuals and growth parameters as well as the ability to relax the equality constraints for the mediation parameters (e.g., the effect of M_1 on Y_1 does not have to equal the effect of M_2 on Y_2).

Because the ECLS-K variables in our example do not include a treatment or intervention, we did not fit illustrative exponential decay models to this data.

Moderated Mediation in SEM

Sometimes a researcher may wish to test a hypothesis about whether longitudinal mediation effects differ depending on the value of a particular variable, i.e., moderated longitudinal mediation. If the longitudinal mediation is modeled using one of the SEM approaches outlined above, an appropriate test of such moderation can be conducted using techniques for incorporating moderation into this framework, the nature of which depend on the type of moderator involved. If the moderator Z represents a categorical variable with a relatively small number of levels, such as clearly defined group membership (e.g., sex, religion, grouping on age), the multiple group approach (i.e., stacked analysis) in SEM (Jöreskog, 1971) can be used for investigating the moderated mediation effects (Little, 2013; MacKinnon, 2008). Since the independent variable and the mediator may display differential effects in the different groups, stacking groups together allows for comparisons of the parameters, with some parameters

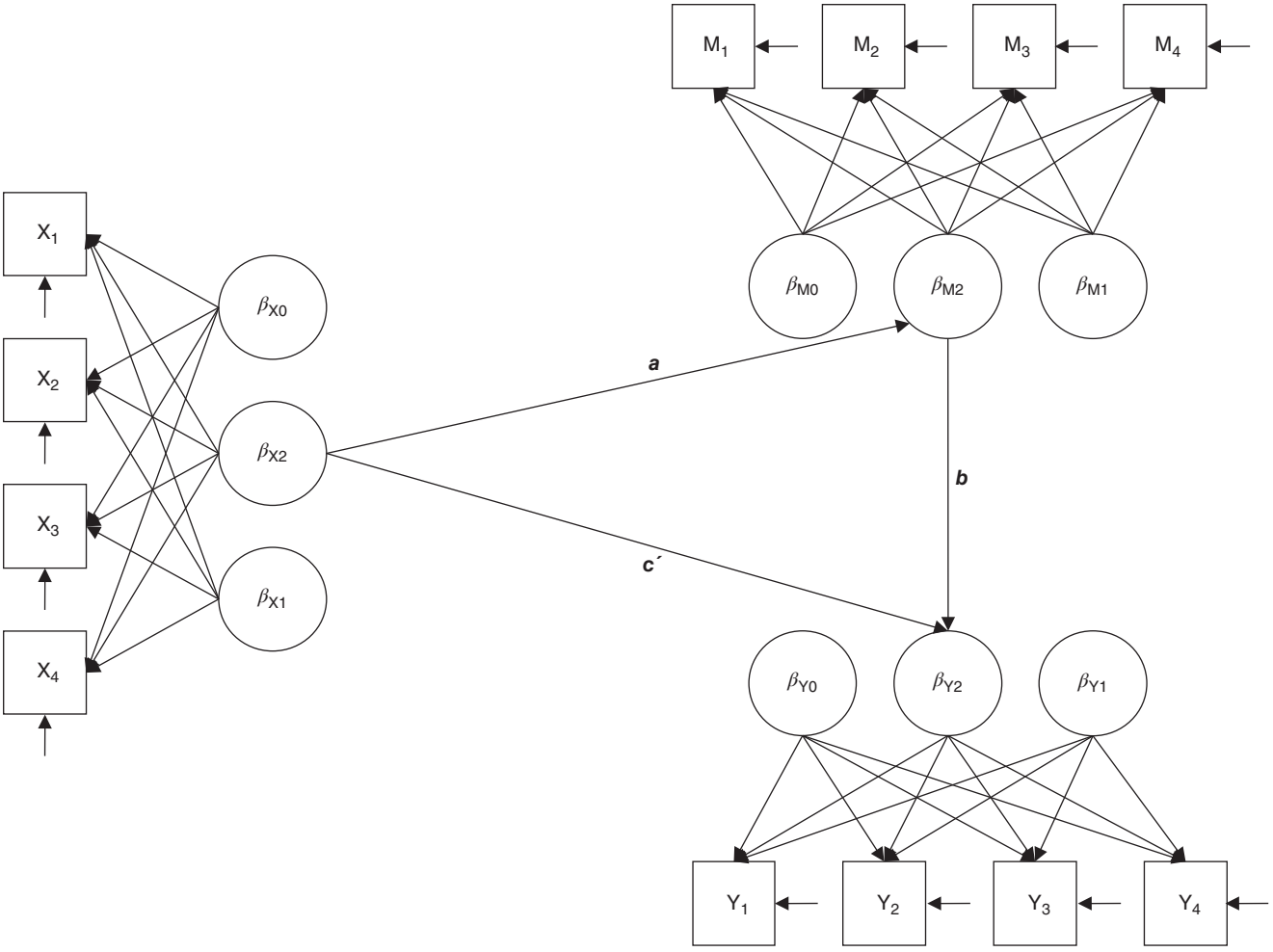


Figure 21.22 A parallel process exponential decay latent latent growth curve model with a dichotomous *X* variable and direct paths between latent growth parameters.

constrained to be equal, while other parameters specified to vary between the stacked groups. The parameters that differ between the groups indicate interactions between the variable providing the group membership and the variables in the model, such as *X* and *M*.

Typically, it is assumed that the same mediation model holds for the stacked groups and the equality of parameter estimates for the relations between *X* and *M* (i.e., *a* paths), *M* and *Y* (i.e., *b* paths), or *X* and *Y* (i.e., *c*′ paths) are examined. Bollen (1989) suggested that researchers start with the model without restricting any parameters across the groups. A satisfactory fit would indicate that the same form of model holds across the stacked groups and provide a rationale for further testing the equivalence of the parameters. The moderated mediation can be investigated from the final model where the equivalent parameters are constrained and nonequivalent parameters are freely estimated. This approach can be applied to structural equation models with observed variables and also models with latent variables for estimating the interaction effect between the latent variable and the categorical group variable (Rigdon, Schumacker, & Wothke, 1998). When *X*, *M*, or *Y* are latent variables, a series of hierarchical tests for invariance should be conducted for the measurement models before examining the equality of the structural relations, to assure that the measures used for the latent variables *X*, *M*, and/or *Y* are equivalent across the groups. If the growth trajectories of *X*, *M*, and *Y* are estimated for the LGC mediation, researchers should inspect if the trajectory shapes are equivalent or different across the stacked groups before testing the moderated mediation. The steps for testing measurement invariance can be found elsewhere (Horm & McArdle, 1992; Jöreskog, 1971; Meredith, 1993).

In a stacked model for moderated mediation, several hypotheses can be tested, including a test of the equality of the relation between *X* and *M* (i.e., *a* paths), a test of the

equality of the relation between M and Y (i.e., b paths), or a test of the equality of the relation between X and Y (i.e., c' paths) across the groups. More importantly, further analysis to test the equality of the indirect effects (i.e., ab estimators) is needed. While the tests of the equality of the relations among X, M, and Y may provide the information on the source of differential indirect effects across the groups, it is possible that the estimated indirect effects obtained in the stacked groups may not statistically differ even when the magnitude of the a paths or the b paths differ across groups. The equality of the path coefficients can be tested by comparing a series of nested models where the relevant paths are constrained to be equal across groups. The equality of the estimated indirect effects can be tested by a t test by dividing the difference in the mediated effects by the pooled standard error of the mediated effect as follows:

$$t_{a_1b_1-a_2b_2} = {}^{(a_1b_1-a_2b_2)}/\sqrt{s^2_{pooled}} \qquad (21.101)$$

In equation 21.100, a_1b_1 and a_2b_2 are the estimated indirect effects from each of the stacked groups and $\sqrt{s^2_{pooled}}$ is the pooled standard error of the mediated effect in each group (MacKinnon, 2008). The equality tests described for the two-group case in this section can be easily extended to cases with more than two groups. The analysis procedures described above can be carried out for each pair of groups to locate the sources of the moderation by group membership.

When the moderator Z is a continuous observed variable interacting with an observed X or M variable, the moderated mediation can be examined using the product term created to reflect the moderation of Z in the relations between X and M (i.e., a path), between M and Y (i.e., b path), or between X and Y (i.e., c' path). Again, the two variables involved in the product term (e.g., X and Z) need to be centered with respect to their means (e.g., $X - \overline{X}$ and $Z - \overline{Z}$) before creating the product term, because a product term based on the uncentered variables will be highly correlated with the first-order effect terms and the high correlations between the predictors may introduce collinearity problems (Aiken & West, 1991; Marquardt, 1980). Centering the variables will minimize or remove the correlations between the product term and the first-order effect terms. The centered variables and their product term are then entered in the model to predict M or Y to test the moderation by Z in the specific relations in the mediation model. With significant interaction effects, further probing of the interaction patterns can be conducted by obtaining simple slopes at different levels of the moderator Z as described earlier in this chapter. Some SEM software programs such as Mplus (Muthén & Muthén, 2014) calculate the indirect effects at different levels of Z and produce the plots of indirect effects corresponding to particular values of Z.

When the moderator is a latent variable interacting with other latent variables in the model, the moderated mediation model can be investigated using the latent interaction variable. Since the use of latent variables in SEM has many advantages, including the capability of controlling for measurement errors that could obscure the interaction effects, various approaches to estimating interactions between latent variables (Arminger & Muthén, 1998; Bollen & Paxton, 1998; Jöreskog, 1998; Kenny & Judd, 1984; Klein & Moosbrugger, 2000; Marsh, Wen, & Hau, 2004; Wall & Amemiya, 2000) have been proposed in the past years. These approaches commonly specify the indicators of the latent interaction variable with the product of the indicators of the two latent variables, but they differ in the ways they specify the nonlinear constraints used for the factor loadings and variance associated with the latent interaction variable. As expected, the modeling process of these approaches may be complicated and unwieldy, and this may be the reason why such methods have not been frequently applied to the substantive research (Rigdon et al., 1998). However, the latent interaction variable approaches have the potential to be applied to estimating moderated mediation, although more research is warranted to investigate the applicability of these approaches for the models with more than one interaction term (Marsh et al., 2004).

To illustrate a basic moderated longitudinal mediation analysis, we fit a multiple groups SEM for boys and girls using the autoregressive mediation model including both lagged and contemporaneous a and b paths (as in Figure 21.16 and Table 21.9) for the LEARN → INTERP → INTERN pathway, which was the best fitting longitudinal mediation model in the first to fifth grade ECLS-K data. Comparisons of the fit of nested models revealed that several parameter estimates differed by gender. Specifically, the autoregressive path from LEARN at third grade to LEARN at fifth grade and both autoregressive paths involving the INTERP mediator were larger for boys than for girls. In addition, boys had larger error variances and disturbance variances than girls for all three variables at all time points. The only mediation effect parameters that differed by gender were the lagged a paths. Boys had stronger associations between first grade LEARN and third grade INTERP ($a_{boys} = -0.1786$, SE = 0.0142, $p < .0001$ vs. $a_{girls} = -0.1272$, SE = 0.0150, $p < .01$) and between third grade LEARN and fifth grade INTERP than girls ($a_{boys} = -0.1686$, SE = 0.0152, $p < .0001$ vs. $a_{girls} =$

–0.1250, SE = 0.0155, $p < .01$). The test of the difference between boys' and girls' *ab* estimates for the temporally ordered $X1 \rightarrow M3 \rightarrow Y5$ mediation effect was significant ($a_1b_1 - a_2b_2 = -0.0019$, SE = 0.0010, $p < .05$), indicating that the effect of approaches to learning on internalizing behavior mediated by interpersonal skill was, in fact, stronger for boys than for girls.

MODERN CAUSAL INFERENCE AND LONGITUDINAL MEDIATION MODELS

There has been a dramatic increase in research on causal inference related to mediation analysis over the last decade (Imai, Keele & Tingley, 2010; MacKinnon, 2008; Pearl, 2012, Valeri & VanderWeele, 2013). In the psychology literature, causal inference has primarily focused on the need for longitudinal rather than cross sectional relations to demonstrate mediation (Cheong et al., 2003; Cole & Maxwell, 2003, Judd & Kenny, 1981; MacKinnon, 2008; Maxwell & Cole, 2007). These articles emphasize the central role of demonstrating temporal precedence such that *X* occurs before and causes later change in *M* and *M* occurs before and causes later change in *Y*, as discussed in a recent article (Maxwell, Cole, & Mitchell, 2011) and commentary on this topic (West, 2011). Temporal precedence refers to the demonstration that for a variable to be a cause of an effect, it must occur before the effect.

The mediation model is a longitudinal model such that *X* causes *M* and *M* causes *Y*, so evidence for this longitudinal causal process is ideally obtained from longitudinal data. Mediation relations between variables measured at one time represent associations between levels of the variables such that a person with high scores on *X* also has higher scores on *M* and *Y*, as an example. In some situations, temporal order between the variables is reasonable such as when *X* represents random assignment to an intervention or measures of *M* are taken before measures of *Y*. Cole and Maxwell (2003) demonstrated algebraically the bias that can result when cross sectional relations are treated as if they are longitudinal relations. The notion that relations between levels of variables at a single time point differ from relations across time applies to any difference across any period of time. For example, the mediated effect for measures that differ one minute in time may differ from measures that differ by one year in time. It is not necessarily the cross sectional versus longitudinal nature of measures that is important. It is the match between when change occurs between variables in the true population mediation model and whether the variables are measured when this mediation relation can be detected (MacKinnon, 2008).

Another important causal inference topic related to temporal precedence is the influence of omitted variable bias on mediation relations (MacKinnon & Pirlott, 2015). There has been an early and sustained focus of attention in the social sciences that accurate estimation of mediation depends on the consideration of other variables that may explain or alter a mediation conclusion from a research study (James, 1980, Judd & Kenny, 1981). In the medical and epidemiology literature, causal inference for mediation has primarily addressed the issue of how confounder variables may bias mediation estimation. Omitted variable bias and confounder bias are considered synonyms in this chapter representing unmeasured variables that influence mediation relations. The potential outcome model provides a way to define causal mediation analysis in the context of confounders or omitted variables. The potential outcome model for causal inference is relevant for many fields and its application has steadily grown. The beginnings of the potential outcome approach for mediation in the social sciences can be traced to an influential paper by Holland (1988). Holland applied the Rubin causal model (Rubin, 1974) to demonstrate the challenges of identifying causal mediation effects even when *X* represents randomization to conditions (see also Sobel, 2008). In epidemiology, Robins and Greenland (1992) described the restrictive assumptions necessary for identification of causal mediation. Pearl (2001) described and developed a solution discussed in Robins and Greenland (1992) to obtain causal mediation effects that serves as the source for a series of important recent papers on causal mediation from the potential outcome perspective (Imai, Keele & Tingley, 2010; Imai, Keele, & Yamamoto, 2010; Lynch, Cary, Gallop, & Ten Have, 2008; Pearl, 2012; Ten Have et al., 2007; Valeri & VanderWeele, 2013; VanderWeele & Vansteelandt, 2009; Vansteelandt, 2009). These new papers provide some practical guidance and software for researchers to use. All of these previous papers have focused primarily on the cross sectional mediation case of measurement of *X*, *M*, and *Y*, and not the longitudinal case.

The purpose of this section is to describe potential outcome model approaches for investigation of mediation effects in the context of longitudinal data. First the potential outcome model for evaluating intervention data is described, followed by applications of this model to the case of mediation. Mediation and moderation relations are described for this potential outcome model. Additional information regarding the potential outcome model for longitudinal data is outlined followed by the estimation

of causal estimators from data. Other approaches to longitudinal mediation from a modern causal inference perspective are briefly described.

Potential Outcomes Model

The potential outcomes approach to estimating causal effects (Holland, 1986, 1988; Pearl, 2009; Rubin, 1974) distinguishes between an individual's observed and counterfactual outcomes. A counterfactual outcome is an outcome for a participant in a research condition in which that participant did not serve (the condition contrary to actual condition they did serve in). For the case of two groups, let variable X be an intervention with level x ($x = 1$ for the intervention, $x = 0$ for the control) and variable Y be the outcome variable. The potential outcomes framework considers all conditions that an individual could serve in, including both intervention and control conditions, even though observed data can only be obtained for one of the two groups. In other words, these models consider all potential outcomes, so if an individual is assigned to the intervention group, the potential outcome $Y(1)$, is the observed outcome Y. The counterfactual outcome for that individual is the value for the outcome variable if that person was assigned to the control group, $Y(0)$. The individual causal effect is equal to $Y(1) - \mathrm{Y}(0)$, even though the value in the control group cannot be observed for a participant in the intervention group. Because it is not possible for the same individual to simultaneously serve in both intervention and control groups, the individual causal effect is not estimable—a problem called the fundamental problem of causal inference. However, assuming that participants are exchangeable, then it is possible to compute a causal effect averaged across individuals in each group and this average causal effect is computed as $\mathrm{E[Y(1) - Y(0)]}$, or the expected average difference between intervention and control groups. This average causal effect is a causal estimator under certain assumptions, primarily that individuals have been randomized to the two conditions.

The potential outcome approach applied to mediation introduces a mediating variable M with level m that mediates the relation between X and Y. The potential outcome approach to mediation demonstrates several complexities in the estimation of mediation. The additional complexity is shown in the additional effects that are now relevant when including the mediator, M, in causal sequence from X to M to Y (Hogan & Lancaster, 2004; Jo, 2008; Pearl, 2001, 2012; Robins & Greenland, 1992; VanderWeele & Vansteelandt, 2009). In addition to the average causal effect now called the total effect (TE) of X on Y, there are five additional causal effects: the controlled direct effect (CDE) which is the direct effect of X on Y at a certain value m of M, a direct effect in the control group called the pure natural direct effect (PNDE), a direct effect in the intervention group called the total natural direct effect (TNDE), the indirect effect in the control group called the pure natural indirect effect (PNIE), and the indirect effect in the intervention group called the total natural indirect effect (TNIE). Use $Y(x, m)$ to denote the potential outcome for an individual under the intervention level x and mediator level m. X takes the value $x = 0$ for the control group, and $x = 1$ for the intervention group. If the actual value of M is m for an individual, then the counterfactual value of M for that individual is denoted as m'.

The *controlled direct effect* of intervention X on outcome Y is the direct effect of the intervention on the outcome at a fixed level m of the mediator M:

$$\mathrm{CDE} = Y(1, m) - Y(0, m) \tag{21.102}$$

The *natural direct effect* of X on Y is different from the CDE in that M is set to the level $M(x)$, the level that would have naturally occurred under one of the conditions of X, so there are now two natural direct effects corresponding to intervention and control groups. In the case of $M(0)$, the PNDE is the effect of intervention X on outcome Y when X did not influence the mediator M (or the participants were assigned the mediator level under the control condition).

$$\mathrm{PNDE} = Y1, M(0) - Y(0, M(0)) \tag{21.103}$$

In the case of $M(1)$, the TNDE is the effect of intervention X on outcome Y when X did not influence the mediator M (or the participants were assigned the mediator level under the intervention condition).

$$\mathrm{TNDE} = Y1, M(1) - Y(0, M(1)) \tag{21.104}$$

Following the same approach, the *pure natural indirect effect* is the effect of the intervention on Y when changing the level m of M in the control condition. In other words, the PNIE is the effect of the intervention on outcome when the level of M is changed when X is set to 0.

$$\mathrm{PNIE} = Y0, M(1) - Y(0, M(0)). \tag{21.105}$$

The *total natural indirect effect* (TNIE) is the effect of the intervention Y when changing the level of M in the

intervention condition. In other words, the TNIE is the effect of intervention on outcome when the level of M is changed when X is set to 1.

$$\text{TNIE} = Y1, M(1) - Y(1, M(0)) \tag{21.106}$$

The total effect is equal to the sum of the pure natural direct effect plus the total natural indirect effect, and the total effect is also equal to the sum of the pure natural indirect effect plus the total direct effect. Because there are different effects for intervention and control groups, an interaction is included in this model insomuch as the direct and indirect effects may differ in the two groups. The direct and indirect effects are different in the two groups if the interaction between X and M in the prediction of Y is nonzero. If the interaction is zero, then the two direct effects are equal and the two indirect effects are equal and these results reduce to the traditional estimator of the direct and indirect effects. Importantly, Pearl (2001) demonstrates that the decomposition of the total effect into the natural direct and indirect effects holds even in models with interactions and nonlinear models such as logistic regression (i.e., the pure natural direct effect plus the Total Natural Indirect Effect equals the total effect).

These estimators described already are estimators of causal effects under certain assumptions. Two assumptions are required for controlled direct effect to be identified (Pearl, 2001; VanderWeele, 2010):

1. No unmeasured confounders for the relation between X and Y.
2. No unmeasured confounders for the relation between M and Y.

Two additional assumptions are required for natural direct and indirect effects to be identified:

3. No unmeasured confounders for the relation between X and M.
4. No measured or unmeasured M to Y confounders are affected by intervention.

Assumptions (1) and (3) refer to the ignorability of intervention (i.e., the intervention independent of potential outcomes for the mediator and outcome) given observed pretreatment confounders. This assumption is satisfied with randomization of X.

Assumption (2) refers to the ignorability of the mediator and the potential outcomes for Y given the observed intervention and pretreatment confounders. This ignorability of the mediator assumption may be difficult to satisfy in actual research because randomization of M is usually not plausible for many studies. In other words, the value of the mediator is not randomly assigned to participants but is self-selected. Even when conditioning on observed confounders for the relation between M and Y, there can still be unobserved confounders that would lead to violation of this assumption. This assumption is strong and is often ignored in studies even though a causal interpretation of a mediated effect is not possible without its existence (VanderWeele & Vansteelandt, 2009; MacKinnon, 2008). Methods described in this article hold that assumption 3 is true and methods to investigate this assumption are only starting to be developed (Coffman & Zhong, 2012). Assumption 4 states that there are not confounders affected by the intervention that confound the M to Y relation. One goal of multiple mediator models is to assess these additional posttreatment confounders in a way that allows for causal estimation. However, approaches to deal with multiple mediators are an active area of current research in causal inference (VanderWeele & VanSteelandt, 2013). And in the application of the potential outcome model for longitudinal mediation, effects of earlier waves on later waves could be postintervention confounders that violate assumption 4.

The assumptions described above have also been described as sequential ignorability assumptions in the research literature on causal mediation (Imai, Keele, & Tingley, 2010; Pearl, 2001; VanderWeele & Vansteelandt, 2009). The linear regression approach as well as potential outcome methods including the natural direct and indirect effects assumes *sequential ignorability*, which consists of the ignorability of the intervention assignment and the ignorability of the mediator. In other words, random assignment of X ensures that c and a represent causal effects (with assumptions 1 and 2) but b and $c\prime$ do not have a causal interpretation without further assumptions (Holland, 1988). For instance, given that the individuals are randomly assigned to conditions of an intervention, there are not confounders of the X to Y and X to M relations. However, because participants are not randomly assigned to values of M, the relation between M and Y, b, is not a causal effect because there can be confounders that account for the effect of M on Y.

Comparison of Traditional and Potential Outcome Mediation Analysis

For the case of continuous M and Y as in most social science studies, there is a direct correspondence between the

estimators of the direct and indirect effects in traditional and the potential outcome methods. If all assumptions are satisfied, traditional and potential outcome methods come to the same conclusion about direct and indirect effects if the interaction of X and M on Y is zero. So the traditional estimators of ab and c' correspond to the natural indirect and natural direct effects causal estimators. When the interaction between X and M is included in the statistical analysis, the natural indirect effects correspond to simple mediation effects in the traditional analysis where the simple mediation effects are at specific values of the mediator. However, typically the interaction of X and M is included in traditional analysis as a way to test the assumption that the causal relation of M to Y does not differ across levels of X. If the interaction is nonsignificant, it is often not included in traditional mediation analysis mainly because the relation of M to Y is often based on prior research. To clarify, if the interaction is included in the statistical model then there are simple mediation effects in traditional analysis corresponding to different b paths in the statistical analysis. These effects would reflect the mediated effect at different values of the mediator. There are corresponding effects in the potential outcome approach that refer to the effects for the control group called the pure effects and the effects for the intervention group called the total effects. For the potential outcome approach, the interaction of X and M is included in the analysis to calculate the different direct and indirect effects in each group.

Moderation in the Potential Outcome Model

A described already, the potential outcome model has different direct and indirect effects in the intervention and control groups. These additional estimators are only present if the interaction of X and M is nonzero. In this potential outcome framework, an interaction of indirect effects corresponds to testing the equality of the natural indirect effect in the two groups. Similarly, moderation effects could be obtained by testing the causal effects across subgroups where each subgroup could have different natural direct and indirect effects. A statistical test of the interaction across subgroups could be accomplished by dividing the difference between natural indirect effects across groups by a multivariate delta method standard error for the difference between the causal effects or by using a resampling method such as the bootstrap to create a confidence interval for the difference between the effects and testing whether zero is in the interval.

Potential Outcome Mediation Model with Longitudinal Data

The causal perspective on longitudinal mediation elucidates a problem in the regression analysis of data with more than one wave. The description of this problem in the causal inference literature is in the context of time-varying interventions or situations in which earlier waves of data predict later treatment and later values of the outcome (Robins & Hernan, 2009). The problems with longitudinal data from a causal inference perspective are illustrated here using a study of the effects of loneliness on depression as described in VanderWeele, Hawkley, Thisted, and Cacioppo (2011). There is evidence that loneliness and depression are separate constructs and that loneliness leads to increased depression (see VanderWeele et al., 2011) but it would be useful to estimate the size of this effect. Say that depression at the fourth time of measurement is the focus of the analysis. Depression at the fourth time point is predicted by the following variables: depression at the first, second, and third times and loneliness at the first, second, and third times. All of these variables are likely to have some effect on Time 4 depression, but there is a problem with estimating all of these effects at once. Depression at Time 2 is on the pathway from loneliness at Time 1 to depression at Time 4, so it may mediate the effect of loneliness on later depression thereby reducing the total effect of loneliness on later depression. In other words, depression at Time 2 could be a mediator of the relation of Time 1 loneliness on depression at Time 4. Because depression at Time 2 is a potential mediator, it would not be ideal to adjust effects if a researcher was interested in estimating the total effect of earlier loneliness on later depression. Depression at Time 2 is also related to both depression and loneliness at Time 3 so it is also a confounder of these variables on Time 4 depression. Because it is a possible confounder, depression at Time 2 must be adjusted to get an accurate estimate of the relation of loneliness and depression at Time 4. So depression at Time 2 should be adjusted for to remove confounding, and it should also not be adjusted for so that it does not block the total effect of loneliness at Time 1 on depression at Time 3. The correct analysis would both adjust and not adjust for Time 2 depression, which is not possible with regression analysis. Robins, Hernan, and Brumback (2000) proposed a solution to this problem by conducting an analysis that removes confounder effects so the true effects can be estimated. In this context, a model called a marginal structural model removes effects of variables that may serve as a confounder to estimate the effects of

loneliness on depression at later waves of measurement. The approach uses a weighting method to render an unconfounded data set.

In terms of an intervention, *X*, and a mediation model with two waves of data for *M,* *M*1 and *M*2, and *Y,* *Y*1 and *Y*2, say we are looking at the effect of the intervention, *X*, on the outcome *Y*2. If control is made for a variable that is a consequence of the intervention, the mediator *M*2, then the relation of *X* to *Y*2 will be biased. By doing this, part of the effect of the intervention is blocked. For the mediation relation, *X* to *M*2 to *Y*2, control for *X* is needed to get the correct relation of *M*2 to *Y*2, that is *X* is a confounder of the *M*2 to *Y*2 relation. It is not possible with a single regression model to obtain an unbiased estimate of *X* on *Y*2 because it must simultaneously control for *M*2 and not control for *M*2. With a structural equation model, it is possible to estimate relevant indirect effects for effects from *X* to *Y*2 when *X* is uncorrelated with *M*1 and *Y*1 because the level of *X* is randomized to groups. The single mediated effect in this model corresponds to *X* to *M*2 to *Y*2. With the marginal structural model and a nonrandomized *X*, the time 1 measures *M*1 and *Y*1 (before the intervention is delivered) are used to weight *M*2 to remove confounding and then a weighted analysis is used to estimate the causal effect of the unconfounded *M*2 variable on *Y*2. A similar approach is illustrated below.

Inverse Probability Weighting with Longitudinal Data

An approach to obtaining effects adjusted for confounders in longitudinal mediation models is to use observed covariates to measure confounder effects and adjust analyses to remove the confounder bias (Robins et al., 2000). The goal of these analyses is to create a pseudo-population of data in which confounder bias has been removed and then conduct analyses on this new confounder-free data set. This new confounder-free data set avoids the problem of simultaneously adjusting for and not adjusting for an intermediate variable that is also a confounder. It accomplishes the goal by separately estimating the confounder effect and other effects. A weighting strategy is used to weight observations based on how much the observations are confounded to create an unconfounded data set. A statistical model is first developed for the prediction of the mediator based on covariate information from baseline measures. These baseline measures can be the measure of *M* and *Y* at baseline as well as other available variables. The weights for each participant are obtained from the residuals of two models (1) a statistical model in which only *X* predicts the mediator, and (2) a statistical model in which *X* and all covariates are used to predict the mediator. A weight is formed by dividing the probability of the residual in the first model by the probability of the residual in the second model. A weighted regression model is used in the analysis to predict *Y* with *M* and *X* (Coffman, 2011; Coffman & Zhong, 2012; Robins, Hernan, & Brumbeck, 2000; Toh & Hernan, 2008). The coefficient relating *M* to *Y* is the *b* coefficient with confounder bias removed and sandwich estimators of standard errors are used to create confidence intervals for the effect. This method assumes that all measures of confounder bias have been measured and included in the analysis to create the weights (thereby leading to accurate estimation of the relation of *M* to *Y*, i.e., by adjustment for potential confounders). If there are repeated measures of *X*, *M*, and *Y*, then weights are obtained for each predicted value of *M* at each time with prior values of *X* included as covariates to adjust for confounder bias. The weights in the final analysis for the prediction of *Y* are the product of the weights obtained for the prediction of each wave of *Y*. Weights that adjust for missing data can also be included.

Clearly, a substantial assumption of this method is that there are no unmeasured confounders, which may be difficult to satisfy in some situations. One important check for the model is to assess the extent to which there is enough overlap in the weights in each group with plots and tables to investigate the balance of the weighting procedure. To date the marginal structural model has not been widely used in psychology but it is more common in epidemiology and the health sciences. It is among the most straightforward of causal inference methods to apply. As described already, VanderWeele et al. (2011) applied the marginal structural model method to investigate the effect of loneliness on depression across five waves.

There are several methods to estimate weights for inverse probability weighting that may be especially useful when the weights vary a great deal from very small to very large (Cole & Hernan, 2008). The weights for each participant represent how much that participant's data are used in the final regression of *X* and *M* on *Y*. If the denominator in the weight equation becomes extremely small, the weights for some persons may be excessively large so that one person may overly influence the results of the analysis. One method to deal with this problem is the use of truncated weights to avoid some subjects having too much or too little influence on the analysis. A relative alternative approach avoids calculation of the weights by creating new data sets where confounder effects are removed using a method called *g-estimation*. G-estimation is not described here but is available in several sources (Loeys et al., 2014; Vansteelandt, 2009).

Causal Estimators for the ECLS-K Data Set

To illustrate the causal estimates, three variables were selected from the ECLS-K data set, an independent variable measure of externalizing behavior at first grade, a mediating measure of reading performance at time third grade, and an outcome measure of internalizing behavior at fifth grade. These measures were used as indicators of a mediating process such that externalizing behavior reduces reading performance, which then increases later depression. Covariates included the kindergarten fall and spring measures of externalizing, reading, and internalizing variables along with gender and age to adjust for potential confounding. The goal of the analysis was to estimate causal effects across waves adjusted for possible confounders where the confounders were measured at the first two waves of measurement. The program described in Valeri and VanderWeele (2013) was used to estimate the natural direct and indirect effects described earlier. To simplify the analysis, only complete cases were used in the analysis for a total of 7,355 individuals. First, the interaction of first grade externalizing behavior and first grade reading performance was not statistically significant in the prediction of Y (.011, SE = .032, $p < .5$) so separate estimates of direct and indirect effects at different levels of first grade externalizing behavior were not computed. In other words, the two different causal estimators of effects were not necessary because the nonsignificant interaction indicated that effects did not differ across levels of the mediator. As a result there is one natural direct effect (NDE = .046, SE = .007, LCL = .033, UCL = .059), one natural indirect effect (NIE = .004, SE = .0008, LCL = .002, UCL = .005) and the sum of these effects equals the total effect (TE = .049, SE = .007, LCL = .036, UCL = .062). All of the effects are statistically significant, which is perhaps not surprising given the large sample size. The metric of the effects is in terms of a one standard deviation change (.540 units) in externalizing at first grade. Dividing the indirect effect by the standard deviation of Y, suggests that a standard deviation change in externalizing led to an indirect effect via reading performance of about .04 standard deviations in internalizing symptoms.

FUTURE DIRECTIONS

There are many potentially fruitful directions for future research regarding longitudinal moderation and mediation. For example, Bayesian analysis of longitudinal data is one particular area where the development of new analytic methods may produce better estimates of hypothesized effects and produce tests that more appropriately reflect substantive theory. Bayesian estimation allows researchers to incorporate prior information into the analysis, which can potentially produce more efficient estimates of moderation and mediation effects (Yuan & MacKinnon, 2009). Moreover, while many traditional estimation techniques rely on large-sample theory, Bayesian estimation is exact, making this approach particularly suitable for data with small samples (Ozechowski, 2014). This aspect of Bayesian analysis is also particularly relevant to mediation effects. Maximum likelihood estimation provides estimates of parameters and standard errors and assumes normal distributions based on large sample theory. However, the *ab* estimate of the mediated effect is known to be nonnormal (MacKinnon et al., 2007), and this problematic violation often necessitates the use of specialized techniques (e.g., bootstrapping or the application of the distribution of the product) for hypothesis testing. However, Bayesian analysis produces the whole (skewed) posterior distribution of the mediated effect and requires no such assumption. Muthén and Asparouhov (2012) presented a Bayesian approach to structural equation modeling, which could potentially include both mediation and moderation. Yuan and MacKinnon (2009) described a Bayesian approach to modeling multilevel mediation, and Ozechowski (2014) demonstrated the use of Empirical Bayes methods for testing mediation in a small sample.

Another possible focus of future research is person-centered moderation and mediation. While more typical variable-centered approaches concentrate on examining relations among variables, person-centered approaches examine patterns of responses for individuals. The person is the unit of analysis (Bauer & Shanahan, 2007), and analytic techniques such as cluster analysis, latent profile analysis, latent class analysis, and latent transition analysis can be used to empirically sort individuals into distinct groups according to their patterns of variable values and associations. For example, individuals might be classified as showing patterns either consistent or inconsistent with mediation. Collins, Graham, and Flaherty (1998) demonstrated such an analysis and show that under particular conditions, variable-centered approaches might detect mediation when there are, in fact, no differences in the proportion of individuals showing patterns consistent with mediation in control and treatment groups. Bauer & Shanahan (2007) illustrate how a person-centered approach can be used to model complex moderated effects.

The recent rapid development of new technology and methods for collecting intensive longitudinal data has resulted in a need for analytic methods suitable for

examining moderation and mediation in such large data sets. For example, current technology allows the collection of intensive ecological momentary assessment (EMA) data, where subjects repeatedly report on key variables while they go about their daily lives. An EMA data set might include hundreds of observations obtained via smartphone from a given individual over the course of a few days or perhaps just one observation per day for several weeks, months, or an entire year. Fitness tracking devices might record several physical activity and condition variables repeatedly for a period of several hours. fMRI data contain activation information about multiple voxels over the time course of an experimental session. Analysis of such rich data sets with traditional techniques can prove cumbersome (Baraldi, Wurpts, MacKinnon, & Lockhard, 2014). As the collection of such data becomes increasingly frequent, there is a clear need for the development of specific methods to explore moderation and mediation in such big data applications. Baraldi et al. (2014) described particular multilevel models that can be used to address relevant questions in EMA data and to explore the effects of personalized, technologically delivered ecological momentary interventions. Lindquist (2012) presented an extension of the mediation model for fMRI data that allows the mediating variable to be a continuous function rather than a single scalar measure and includes causal estimators of effects. The combination of intensive longitudinal data and a person-centered analytic approach may potentially allow the examination of research questions that could not be easily addressed with smaller data sets and traditional variable-centered analytic techniques. For example, groups of subjects who confirm or do not confirm the pattern of mediation at different phases of time during a study (e.g., confirming mediation at early versus later phases) could be identified and their transition patterns over the course of time can be examined to predict their status on an outcome variable.

There is also a clear need for more research on temporal design issues. Every researcher designing a longitudinal study needs to make a number of explicit decisions about the data collection schedule. This audience would benefit from clear recommendations regarding the ideal number as well as the frequency and spacing of observations for various types of research scenarios. Some research in this area has, in fact, been conducted for univariate and bivariate growth models where interest centers on testing the effects of treatment on growth (e.g., Raudenbush & Liu, 2001) or variabilily in growth across individuals and covariation among growth rates (e.g., Rast & Hofer, 2014). However, similar work focusing on the power to detect moderation and mediation effects has yet to be conducted.

In the interest of developing models that more accurately reflect developmental processes, future research should also focus on methods to parsimoniously represent nonlinear effects. Polynomial trends are, by far, the most common functions currently used to represent change over time, and linear models are the more frequently employed of these. However, most growth is probably not strictly linear (Fritz, 2014). Adding higher order polynomial terms may increase the realism of the model, but these additional factors are often difficult to interpret and incorporate into more complex analyses (such as those involving mediation and moderation). Cudeck and Harring (2007) described a number of nonlinear functions and discuss the considerations involved in choosing a particular nonlinear form to represent change. In addition to such nonlinear trends in an outcome variable over time, it is also possible that mediation or moderation effects might themselves be nonlinear. Our models typically assume that the moderated or mediated effect operates identically throughout the range of the predictor variable. However, such an assumption may be overly simplistic, and developing methods and recomendations for testing these effects would be a useful contribution to the literature. Semiparametric and related generalized additive modeling approaches are important directions in this work (Hastie & Tibshirani, 1990; Keele, 2008; Shen, Chou, Pentz, & Berhane, 2014).

Though the methods described in this chapter focus primarily on interinterdividual analysis, it is potentially useful to distinguish between intra- and interindividual processes. Most psychologcal studies are focused on variation between cases (Molenaar, 2004), and explaining intraindividual processes through reference to interindividual findings is a simple generalization of the ecological fallacy (Robinson, 1950). Molenaar (2004) defines the conditions under which the structure of intra- and interindividual variation will be analogous and argues that these will rarely if ever be met in real psychological processes. If a researcher wishes to explore within-person mediation, using a $1 \rightarrow 1 \rightarrow 1$ mulitilevel mediation model (e.g., Krull & MacKinnon, 2001), combined with judicious decisions regarding the centering of the various predictors in the model, can help to disentangle these processes. To date, however, very little work has focused on ideal methods for exploring intraindividual effects and how best to approach such questions in real world data sets.

The combination of mediation and moderation into a single analytic framework is another area that would benefit from future quantitative research and application in substantive data. Currently the most frequent method for exploring whether mediated effects might, in fact, be

moderated by a fourth variable is the rather simplistic approach of creating subgroups based on the moderator and conducting a multiple group SEM. Such an approach is well suited when the moderator is truly categorical, but artificially categorizing a continuous variable so that it can be forced into such an analysis is far from ideal. Dichotomizing a continuous variable via a median split has many undesirable consequences, including loss of power and spurious main effects and interactions (MacCallum, Zhang, Preacher, & Rucker, 2002). The development and application of more sophisticated techniques that avoid such inappropriate treatment of variables will result in much better tests of hypotheses involving mediated moderation and moderated mediation in longitudinal data. These might involve continuous variable approaches to moderation within an SEM or incorporation of person-centered methods that empirically identify subgroups of subjects based on patterns of multiple variables.

Finally, causal inference approaches in general and for mediation in particular are also being rapidly developed. Several books are now appearing that should help disseminate these methods (Hernan & Robins, in press; Morgan & Winship, 2007; Pearl, 2009), though longitudinal mediation modeling has not been widely addressed in these books. There are some recent applications with longitudinal data (Ertefaie & Stephens, 2010; Lindquist, 2012; O'Malley, 2012). Some interesting recent work attempts to combine both dynamic mediation models and modern causal inference (Aalen, Roysland, & Gran 2012; Aalen, Roysland, Gran, Kouyos, & Lange, 2014). Aalen et al. (2012) described two overlapping approaches to causal inference with longitudinal mediation corresponding to interventionist and mechanistic viewpoints. The interventionist approach focuses on estimating causal effects when there is randomization or the evaluation of the status of a system when there is and when there is not an intervention. Typically this approach focuses on the potential outcomes model as described already, although some approaches do not require counterfactuals (Dawid, 2000). The mechanistic approach focuses on how effects come about rather than how an intervention would cause effects. As described by Aalen et al. (2012, p. 832) the "mechanisms in the structure of the world are present whether humans are there to intervene or not, and hence seem to be the more fundamental part." As a compromise between interventionist and mechanistic positions, Aalen et al. (2012, p. 843) stated, "Now our modest suggestion is as follows: if a direct effect cannot reasonably be defined as a controlled or natural direct effect in the counterfactual sense because the required hypothetical manipulation of the mediator is inconceivable, then we can alternatively view these effects as being represented by flow in a dynamic system, so that the direct effect corresponds to the flow not passing through the mediator. The indirect effect can similarly be understood as that passing through the mediator." Aalen et al. went on to describe a stochastic differential equation model for mediation that includes dynamic models for X, M, and Y processes including a dynamic model for how X, M, and Y are related. Aalen et al. apply this idea to mediation in survival analysis data. Other recent work also addresses dynamic change based on functional data analysis and mediation (Lindquist, 2012), though this approach also targets the estimation of natural and controlled effect causal estimators described previously. More application of these methods to actual data is an important future direction of this research in psychology.

Because development is longitudinal by definition, and because so many of the hypotheses developmental psychopathologists seek to test involve multiple variables interacting and affecting each other in sequence, the concepts of longitudinal moderation and mediation are particularly important in this area of research. The various analytic models presented in this chapter test how effects change over time or the mechanisms through which earlier causes produce their later effects. Application of these methods in longitudinal data will help to advance the field and achieve better understanding of the complex processes that unfold over time to produce adaptive and maladaptive behavior.

REFERENCES

Aalen, O. O., Roysland, K., & Gran, J. M. (2012). Causality, mediation and time: A dynamic viewpoint. *Journal of the Royal Statistical Society Series A*, *175*, 831–861.

Aalen, O. O., Roysland, K., Gran, J. M., Kouyos, R., & Lange, T. (2014). Can we believe the DAGs? A comment on the relationship between causal DAGs and mechanisms. *Statistical Methods in Medical Research*. doi: 10.1177/0962280213520436

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Thousand Oaks, CA: Sage Publications.

Arminger, G., & Muthén, B. (1998). A Bayesian approach to nonlinear latent variable models using the Gibbs sampler and the Metropolis-Hastings algorithm. *Psychometrika, 63*(3), 271–300.

Arruda, E. H., & Krull, J. L. (2013). *The use of multilevel random coefficient models for the analysis of mediational change over time*. Paper presented at the Biannual Multilevel Modeling Conference, Utrecht, The Netherlands.

Arruda, E. H., & Krull, J. L. (2014a). *The use of multilevel random coefficient models for the analysis of mediational change over time*. Poster presented at the annual meeting of the American Educational Research Association, Philadelphia, PA.

Arruda, E. H., & Krull, J. L. (2014b). *The use of multilevel random coefficient models for the analysis of mediational change over time*. Poster presented at the Annual Modern Modeling Methods Conference, Storrs, CT.

Baraldi, A. N., Wurpts, I. C., MacKinnon, D. P., & Lockhard, G. (2014). Mechanisms of behavior change from technology-based interventions. In L. Marsch, S. Lord, & J. Dallery (Eds.), *Leveraging technology to transform behavioral healthcare* (pp. 187–199). New York, NY: Oxford University Press.

Barcikowski, R. S. (1981). Statistical power with group mean as the unit of analysis. *Journal of Educational Statistics, 6*, 267–285.

Barnard-Brak, L., & Brak, V. (2011). Pharmacotherapy and academic achievement among children with attention-deficit/hyperactivity disorder. *Journal of Child and Adolescent Psychopharmacology*, *21*, 597–603.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173–1182.

Bauer, D. J., Preacher, K. J., & Gil, K. M. (2006). Conceptualizing and testing random indirect effects and moderated mediation in multilevel models: New procedures and recommendations. *Psychological Methods, 11*, 142–163. doi: 10.1037/1082-989X.11.2.142

Bauer, D. J., & Shanahan, M. J. (2007). Modeling complex interactions: Person-centered and variable-centered approaches. In T. D. Little, J. A. Bovaird, & N. A. Card (Eds.), *Modeling contextual effects in longitudinal studies* (pp. 255–284). Mahwah, NJ: Erlbaum.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238–246.

Bentler, P. M., & Wu, E. J. C. (2006). *EQS for Windows v. 6.1* [computer software]. Encino, CA: Multivariate Software, Inc.

Blozis, S. A. (2004). Structured latent curve models for the study of change in multivariate repeated measures. *Psychological Methods, 9*, 334–353. doi: 10.1037/1082-989X.9.3.334

Blozis, S. A. (2007). On fitting nonlinear latent curve models to multiple variables measured longitudinally. *Structural Equation Modeling, 14*, 179–201. doi: 10.1080/10705510701293445

Blozis, S. A., Conger, K. J., & Harring, J. R. (2007). Nonlinear latent curve models for multivariate longitudinal data. *International Journal of Behavioral development, 31*, 340–346. doi: 10.1177/0165025407077755.

Bolger, N., & Laurenceau, J.-P. (2013). *Intensive longitudinal methods: An introduction to diary and experience sampling research*. New York, NY: Guilford.

Bollen, K. A. (1989). *Structural equations with latent variables*. New York, NY: Wiley.

Bollen, K. A., & Curran, P. J. (2004). Autoregressive latent trajectory (ALT) models: A synthesis of two traditions. *Sociological Methods & Research, 32*, 336–383.

Bollen, K. A., & Curran, P. J. (2006). *Latent curve models: A structural equation perspective*. Hoboken, NJ: Wiley.

Bollen, K. A., & Paxton, P. (1998). Two-stage least squares estimation of interaction effects. In R. E. Schumacker & G. A. Marcoulides (Eds.), *Interaction and nonlinear effects in strucutral equation modeling* (pp. 125–154). Mahwah, NJ: Erlbaum.

Bollen, K. A., & Zimmer, C. (2010). An overview of the autoregressive latent trajectory (ALT) model. In K. van Montfort, J. Oud, & A. Satora (Eds.), *Longitudinal research with latent variables* (pp. 153–176). Heidelberg, Germany: Springer.

Browne, M. W. (1993). Structured latent curve models. In C. M. Cuadras & C. R. Rao (Eds.), *Multivariate analysis: Future directions 2* (pp. 171–198). Amsterdam, The Netherlands: North Holland.

Burstein, L., & Miller, M. D. (1980). Regression-based analysis of multi-level educational data. *New Directions for Methodology of Social and Behavioral Sciences, 6*, 194–211.

Candel. M. J. J. M., & Winkens B. (2003). Performance of Empirical Bayes estimators of level-2 random parameters in multilevel analysis: A Monte Carlo study for longitudinal designs. *Journal of Educational and Behavioral Statistics, 28*, 169–194.

Cheong, J. (2011). Accuracy of estimates and statistical power for testing mediation in latent growth modeling. *Structural Equation Modeling, 18*, 195–211.

Cheong, J., MacKinnon, D. P., & Khoo, S.-T. (2003). Investigation of mediational processes using parallel process latent growth curve modeling. *Structural Equation Modeling, 10*, 238–262. doi: 10.1207/S15328007SEM1002_5

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology, 8*, 598–600.

Coffman, D. L. (2011). Estimating causal effects in mediation analysis using propensity scores. *Structural equation modeling: A multidisciplinary journal*, *18*(3), 357–369.

Coffman, D. L., & Zhong, W. (2012). Assessing mediation using marginal structural models in the presence of confounding and moderation. *Psychological Methods, 17*, 642–664.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.

Cole, D. A., & Maxwell, S. E. (2003). Testing mediational models with longitudinal data: Questions and tips in the use of structural equation modeling. *Journal of Abnormal Psychology*, *112*, 558–577.

Cole, S. R., & Hernan, M. A. (2008). Constructing inverse probability weights for marginal structural models. *American Journal of Epidemiology*, *168*, 656–664.

Collins, L. M., Graham, J. W., & Flaherty, B. P. (1998). An alternative framework for defining mediation. *Multivariate Behavioral Research, 33*, 295–312.

Cudeck, R. (1996). Mixed-effects models in the study of individual differences with repeated measures data. *Multivariate Behavioral Research, 31*, 371–403. doi: 10.1207/s15327906mbr3103_6

Cudeck, R., & du Toit, S. H.C. (2002). A version of quadratic regression with interpretable parameters. *Multivariate Behavioral Research, 37*, 501–519.

Cudeck, R., & Harring, J. R. (2007). The analysis of nonlinear patterns of change with random coefficient models. *Annual Review of Psychology, 58*, 615–637.

Curran, P. J. (20023) Have multilevel models been structural equation models all along? *Multivariate Behavioral Research, 38*, 529–569.

Currie, J., & Stabile, M. (2006). Child mental health in childhood and human capital accumulation: The case of ADHD. *Journal of Health Economics, 25*, 1094–1118.

Currie, J., & Stabile, M. (2009). Mental health in childhood and human capital. In J. Gruber (Ed.), *The problems of disadvantaged youth: An economic perspective* (pp. 115–148). Chicago, IL: University of Chicago Press.

Davidian, M., & Giltinan, D. M. (1995). *Nonlinear models for repeated measurement data*. New York, NY: Chapman and Hall/CRC Press.

Dawid, A. P. (2000). Causal inference without counterfactuals. *Journal of the American Statistical Association*, *95*(450), 407–424.

Dearing, E., & Hamilton, L. C. (2006). Contemporary advances and classic advice for analyzing mediating and moderating variables. *Monographs of the Society for Research in Child Development, 71*, 88–104.

Department of Health and Human Services, Administration for Children and Families. (1996–2001). *Early Head Start research and evaluation study* [computer file]. Available from http://www.icpsr.org/

Drabick, D. A., & Kendall, P. C. (2010). Developmental psychopathology and the diagnosis of mental health problems among youth. *Clinical Psychology: Science and Practice, 17*, 272–208. doi: 10.1111/j.1468-2850.2010.01219.x.

Duncan, O. D. (1969). Some linear models for two-wave, two-variable panel analysis. *Psychological Bulletin, 72*(3), 177–182.

Duncan, T. E., Duncan, S. C., & Strycker, L. A. (2006). *An introduction to latent variable growth curve modeling: Concepts, issues, and applications* (2nd ed.). New York, NY: Lawrence Erlbaum Associates.

Edwards, J. R., & Lambert, L. S. (2007). Methods for integrating moderation and mediation: Ageneral anlytical framework using moderated path analysis. *Psychological Methods, 12*, 1–22.

Efron, B., & Tibishirani, R. J. (1993). *An introduction to the bootstrap*. New York, NY: Chapman & Hall.

Elder, T. E. (2010). The importance of relative standards in ADHD diagnoses: Evidence based on exact birth dates. *Journal of Health Economics, 29*, 641–656. doi: 10.1016/j.jhealeco.2010.06.003

Enders, C. K., & Tofighi, D. (2007). Centering predictor variables in cross-sectional multilevel models: A new look at an old issue. *Psychological Methods, 12*, 121–138.

Ertefaie, A., & Stephens, A. D. (2010). Comparing approaches to causal inference for longitudinal data: Inverse probability of treatment weighting versus propensity scores. *International Journal of Biostatistics, 6*(2), Article 14.

Fairchild, A. J., & MacKinnon, D. P. (2009). A general model for testing mediation and moderation effects. *Prevention Science, 10*, 87–99. doi: 10.1007/s11121-008-0109-6

Ferrer, E., & McArdle, J. J. (2003). Alternative structural models for multivariate longitudinal data analysis. *Structural Equation Modeling, 10*, 493–524.

Fletcher, J., & Wolfe, B. (2008). Child mental health and human capital accumulation: The case of ADHD revisited. *Journal of Health Economics, 27*, 794–800.

Frazier, P. A., Tix, A. P., & Barron, K. E. (2004). Testing moderator and mediator effects in counseling psychology research. *Journal of Counseling Psychology, 51*, 115–134.

Frazier, T. W., Youngstrom, E. A., Glutting, J. J., & Watkins, M. W. (2007). ADHD and achievement meta-analysis of the child, adolescent, and adult literatures and a concomitant study with college students. *Journal of Learning Disabilities*, *40*, 49–65.

Fritz, M. S. (2014). An exponential decay model for mediation. *Prevention Science, 15*, 611–622. doi: 10.1007/211121-01300390-x.

Gaub, M., & Carlson, C. L. (1997). Gender differences in ADHD: A meta-analysis and critical review. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 1036–1045.

Gershon, J., & Gershon, J. (2002). A meta-analytic review of gender differences in ADHD. *Journal of Attention Disorders*, *5*, 143–154.

Gogineni, A., Alsup, R., & Gillespie, D. (1995). Mediation and moderation in social work research. *Social Work Research, 19*, 57–63.

Goldstein, H. (1995). *Multilevel statistical models* (2nd ed.). New York, NY: Wiley.

Gollob, H. F., & Reichardt, C. S. (1987). Taking account of time lags in causal models. *Child Development, 58*(1), 80–92.

Gollob, H. F., & Reichardt, C. S. (1991). Interpreting and estimating indirect effects assuming time lags really matter. In L. M. Collins & J. L. Horn (Eds.), *Best methods for the analysis of change: Recent advances, unanswered questions, future directions* (pp. 243–259). Washington, DC: American Psychological Association.

Gresham, F. M., & Elliott, S. N. (1990). *Social skills rating scale: Elementary scale A ("How often")*. Circle Pines, MN: American Guidance Service.

Hastie, T., & Tibshirani, R. (1990). *Generalized additive models*. London, UK: Chapman and Hall.

Hayes, A. F. (2013). *Introduction to mediation, moderation and conditional process analysis: A regression-based appraoch*. New York, NY: Guilford Press.

Hayes, A. F. (2014). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication Monographs, 76*, 408–420.

Hedeker, D. (2004). An introduction to growth modeling. In D. Kaplan (Ed.), *The SAGE handbook of quantitative methodology for the social sciences* (pp. 215–234).. Thousand Oaks, CA: Sage.

Hernan, M. A., & Robins, J. M. (in press). *Causal inference*. London, UK: Chapman & Hall.

Hoffman, L., & Stawski, R. S. (2009). Personas as contexts: Evaluating between-person and within-person effects in longitudinal analysis. *Research in Human Development, 6*, 97–120.

Hofmann, D. A., & Gavin, M. B. (1998). Centering decisions in hierarchical linear models: Implications for research in organizations. *Journal of Management, 24*, 123–161.

Hogan, J. W., & Lancaster, T. (2004). Instrumental variables and inverse probability weighting for causal inference from longitudinal observational studies. *Statistical Methods in Medical Research*, *13*, 17–48.

Holland, P. W. (1986). Statistics and causal inference (with comments), *Journal of the American Statistical Association*, *81*, 945–970.

Holland, P. W. (1988). Causal inference, path analysis, and recursive structural equation models. *Sociological Methodology, 18,* 449–484.

Holmbeck, G. N. (1997). Toward terminological, conceptual, and statistical clarity in the study of mediators and moderators: Examples from the child-clinical and pediatric psychology literatures. *Journal of Consulting and Clinical Psychology, 5*, 599–610.

Horm, J. L., & McArdle, J. J. (1992). A practical and theoretical guide to measurement invariance in aging research. *Experimental Aging Research, 18*, 117–144.

Hox, J. J. (2010). *Multilevel analysis: Techniques and applications* (2nd ed.). New York, NY: Routledge.

Humphreys, K. L., Katz, S. J., Lee, S. S., Hammen, C., Brennan, P. A., & Najman, J. M. (2013). The association of ADHD and depression: Mediation by peer problems and parent–child difficulties in two complementary samples. *Journal of Abnormal Psychology, 122*, 854–867.

Humphreys, L. G. (1960). Investigations of the smipex. *Psychometrika, 25*, 313–323.

Imai, K., Keele, L., & Tingley, D. (2010). A general approach to causal mediation analysis. *Psychological Methods, 15,* 309–334.

Imai, K., Keele, L., & Yamamoto, T. (2010). Identification, inference and sensitivity analysis for causal mediation effects. *Statistical Science, 25*(1), 51–71. doi:10.1214/10-STS321

James, L. R. (1980). The unmeasured variable problem in path analysis. *Journal of Applied Psychology, 65,* 415–421.

Jo, B. (2008). Causal inference in randomized experiments with mediational processes. *Psychological Methods, 13*(4), 314–336. doi: 10.1037/a0014207

Jöreskog, K. G. (1971). Simultaneous factor analysis in several populations. *Psychometrika, 36*, 409–426.

Jöreskog, K. G. (1979). Statistical estimation of structural models in longitudinal-developmental investigations. In J. R. Nesselroade & P. B. Baltes (Eds.), *Longitudinal research in the study of behavior and development* (pp. 303–352). New York, NY: Academic Press.

Jöreskog, K. G. (1998). Interaction and nonlinear modeling: Issues and approaches. In R. E. Schumacker & G. A. Marcoulides (Eds.), *Interaction and nonlinear effects in structural equation modeling* (pp. 239–250). Mahwah, NJ: Erlbaum.

Jöreskog, K. G., & Sörbom, D. (2006). *LISREL for Windows v. 8.7* [computer software]. Lincolnwood, IL: Scientific Software International, Inc.

Judd, C. M., & Kenny, D. A. (1981). Process analysis estimating mediation in treatment evaluations. *Evaluation Review, 5*(5), 602–619.

Kawabata, Y., Tseng, W. L., & Gau, S. S. F. (2012). Symptoms of attention-deficit/hyperactivity disorder and social and school adjustment: The moderating roles of age and parenting. *Journal of Abnormal Child Psychology*, *40*, 177–188.

Keele, L. (2008). *Semiparametric regression for the social sciences*. New York, NY: Wiley.

Kenny, D. A. (1979). *Correlation and causality*. New York, NY: Wiley.

Kenny, D. A., & Judd, C. M. (1984). Estimating the nonlinear and interactive effects of latent variables. *Psychological Bulletin, 96*, 201–210.

Kenny, D. A., Korchmaros, J. D., & Bolger, N. (2003). Lower level mediation in multilevel models. *Psychological Methods, 8*, 115–128. doi: 10.1037/1082-989X.8.2.115

Khoo, S. T. (2001). Assessing program effects in the presence of treatment-baseline interactions: a latent curve approach. *Psychological Methods, 6*(3), 234–257.

Klein, A., & Moosbrugger, H. (2000). Maximum likelihood estimates of latent interaction effects with the LMS-method. *Psychometrika, 65*(4), 457–474.

Kline, R. B. (2011). *Principles and practice of structural equation modeling* (3rd ed.). New York, NY: Guilford Press.

Krause, M. R., Serlin, R. C., Ward, S. E., Rony, R. Y., Ezenwa, M. O., & Naab, F. (2010). Testing mediation in nursing research: Beyond Baron and Kenny. *Nursing Research, 59*, 288–294. doi: 10.1097/NNR.0b013e3181dd26b3.

Kreft, I. G. G., de Leeuw, J., & Aiken, L. S. (1995). The effect of different forms of centering in hierarchical linear models. *Multivariate Behavioral Research, 30*, 1–21.

Krull, J. L., & MacKinnon, D. P. (1999). Multilevel mediation modeling in group-based intervention studies. *Evaluation Review, 23*, 418–444. doi: 10.1177/0193841X9902300404

Krull, J. L., & MacKinnon, D. P. (2001). Multilevel modeling of individual and group level mediated effects. *Multivariate Behavioral Research, 36*, 249–277. doi: 10.1207/S15327906MBR3602_06

Lahey, B. B., Hartung, C. M., Loney, J., Pelham, W. E., Chronis, A. M., & Lee, S. S. (2007). Are there sex differences in the predictive validity of *DSM–IV* ADHD among younger children? *Journal of Clinical Child and Adolescent Psychology*, *36*, 113–126.

Laird, N. M., & Ware, H. (1982). Random-effects models for longitudinal data. *Biometrics, 38*, 963–974.

Lee, S. S., Lahey, B. B., Owens, E. B., & Hinshaw, S. P. (2008). Few preschool boys and girls with ADHD are well-adjusted during adolescence. *Journal of Abnormal Child Psychology*, *36*, 373–383.

Lindquist, M. A. (2012). Functional causal mediation analysis with an application to brain connectivity. *Journal of the American Statistical Association, 107*, 1297–1309.

Little, T. D. (2013). *Longitudinal structural equation modeling*. New York, NY: Guilford Press.

Loeys, T., Moerkerke, B., De Smet, O., Bysse, A., Steen, J., & Vansteelandt, S. (2014). Flexible mediation analysis in the presence of nonlinear relations: Beyond the mediation formula. *Multivariate Behavioral Research*, *48*(6), 871–894.

Longford, N. T. (1993). *Random coefficient models*. Oxford, UK: Oxford University Press.

Lynch, K. G., Cary, M., Gallop, R., & Ten Have, T. R. (2008). Causal mediation analyses for randomized trials. *Health Services & Outcomes Research Methodology, 8*(2), 57–76.

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods, 7*, 19–40.

MacKinnon, D. P. (2008). *Introduction to statistical mediation analysis*. New York, NY: Lawrence Erlbaum Associates.

MacKinnon, D. P., & Dwyer, J. H. (1993). Estimating mediated effects in prevention studies. *Evaluation Review, 17*, 144–158.

MacKinnon, D. P., Fritz, M. S., Williams, J., & Lockwood, C. M. (2007). Distribution of the product confidence limits for the indirect effect: Program PRODCLIN. *Behavior Research Methods, 39*, 384–389.

MacKinnon, D. P., Krull, J. L., & Lockwood, C. M. (2000). Equivalence of the mediation, confounding and suppression effect. *Prevention Science, 1*, 173–181.

MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G., & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods, 7*, 83–104. doi:10.1037/1082-989x.7.1.83

MacKinnon, D. P., Lockwood C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research, 39*, 99–128.

MacKinnon, D. P., & Pirlott, A. G. (2015). Statistical approaches for enhancing causal interpretation of the *M* to *Y* relation in mediation analysis. *Personality and Social Psychology Review*, *1*, 30–43. doi: 10.1177/1088868314542878

MacKinnon, D. P., & Valente, M. J. (2014). Mediation from multilevel to structural equation modeling. *Annals of Nutrition and Metabolism, 65*, 196–202. doi: 10.1159/000362505

MacKinnon, D. P., Warsi, G., & Dwyer, J. H. (1995). A simulation study of mediated effect measures. *Multivariate Behavioral Research, 30*, 41–62.

Marquardt, D. (1980). You should standarized the predictor variables in your regression models. *Journal of the American Statistical Association, 75*, 74–103.

Marsh, H. W., Wen, Z., & Hau, K.-T. (2004). Structural equation models of latent interactions: Evaluation of alternative estimation strategies and indicator construction. *Psychological Methods, 9*(3), 275–300.

Marsh, H. W., Wen, Z., & Hau, K. T. (2006). Structural equation models of latent interaction and quadratic effects. In G. R. Hancock & R. O. Mueller (Eds.), *Structural equation modeling: A second course* (pp. 225–265). Greenwich, CT: Information Age Publishing.

Martens, M. P., & Haase, R. H. (2006). Advanced applications of structural equation modeling in counseling psychology research. *Counseling Psychologist, 34*, 806–838.

Masten, A. S., & Cicchetti, D. (2010). Editorial: developmental cascades. *Development and Psychopathology, 22*, 491–495. doi:10.1017/S0954579410000222.

Maxwell, S. E., & Cole, D. A. (2007). Bias in cross-sectional analyses of longitudinal mediation. *Psychological Methods, 12,* 23–44.

Maxwell, S. E., Cole, D. A., & Mitchell, M. A. (2011). Bias in cross-sectional analyses of longitudinal mediation: Partial and complete mediation under an autoregressive model. *Multivariate Behavioral Research, 46*(5), 816–841.

Maxwell, S. E., & Delaney, H. D. (1993). Bivariate median splits and spurious statistical significance. *Psychological Bulletin, 113*, 181–190.

McArdle, J. J. (2001). A latent difference score approach to longitudinal dynamic structural analysis. In R. Cudeck, M. du Toit, & D. Sorbom (Eds.), *Structural equation modeling: Present and future* (pp. 341–380). Lincolnwood, IL: Scientific Software International.

McArdle, J. J., & Hamagami, F. (2001). Latent difference score structural models for linear dynamic analyses with incomplete longitudinal data. In L. M. Collins & A. G. Sayer (Eds.), *New methods for the analysis*

of change (pp. 138–175). Washington, DC: American Psychological Association.

McArdle, J. J., & Nesselroade, J. R. (2003). Growth curve analysis in contemporary psychological research. In J. Schinka & W. Velicer (Eds.), *Comprehensive handbook of psychology: Research methods in psychology* (Vol. *2*, p. 447–480). New York, NY: Wiley.

Meeker, Jr., W. Q., Cornwell, L. W., & Aroian, L. A. (1981). The product of two normally distributed random variables. In W. J. Kennedy & R. E. Odeh (Eds.), *Selected tables in mathematical statistics* (Vol. *7*, p. 1–256). Providence, RI: American Mathematical Society.

Meredith, W. (1993). Measurement invariance, factor analysis, and factorial invariance. *Psychometrika, 58*, 525–543.

Meredith, W., & Tisak, J. (1990). Latent curve analysis. *Psychometrika, 55*(1), 107–122.

Molenaar, P. C.M. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement, 2*, 201–218.

Morgan, P. L., Staff, J., Hillemeier, M. M., Farkas, G., & Maczuga, S. (2013). Racial and ethnic disparities in ADHD diagnosis from kindergarten to eighth grade. *Pediatrics*, *132*, 85–93.

Morgan, S. L., & Winship, C. (2007). *Counterfactuals and causal inference: Methods and principles for social research*. Cambridge, UK: Cambridge University Press.

Muller, D., Judd, C. M., & Yzerbyt, V. Y. (2005). When moderation is mediated and mediation is moderated. *Journal of Personality and Social Psychology, 89,* 852–863.

Muthén, B., & Asparouhov, T. (2012). Bayesian SEM: A more flexible representation of substantive theory. *Psychological Methods, 17*, 313–335.

Muthén, B. O., & Curran, P. J. (1997). General longitudinal modeling of individual differences in experimental designs: A latent variable framework for analysis and power estimation. *Psychological Methods, 2*, 371–402.

Muthén, B., & Khoo, S. T. (1998). Longitudinal studies of achievement growth using latent variable modeling. *Learning and Individual Differences, 10*(2), 73–101.

Muthén, B. O., & Muthén, L. (2014). *Mplus v. 7.2: The comprehensive modeling program for applied researchers* [computer software]. Los Angeles, CA: Muthén & Muthén.

Nesselroade, J. R. (1991). Interindividual differences in intraindividual changes. In L. M. Collins & J. L. Horn (Eds.), *Best methods for the analysis of change: Recent advances, unanswered questions, future directions* (pp. 92–105). Washington, DC: American Psychological Association.

Nosek, B. A., Graham, J., Lindner, N. M., Kesebir, S., Hawkins, C. B., Hahn, C., … Tenney, E. R. (2010). Cumulative and career-stage impact of social-personality psychology programs and their members. *Personality and Social Psychology Bulletin, 36*, 1283–1300.

O'Malley, A. J. (2012). Instrumental variable specifications and assumptions for longitudinal analysis of mental health cost offsets. *Health Services and Outcomes Research Methodology*, *12*, 254–272.

Owens, E. B., Hinshaw, S. P., Lee, S. S., & Lahey, B. B. (2009). Few girls with childhood attention-deficit/hyperactivity disorder show positive adjustment during adolescence. *Journal of Clinical Child & Adolescent Psychology*, *38*, 132–143.

Ozechowski, T. J. (2014). Empirical Bayes MCMC estimation for modeling treatment processes, mechanism of change, and clinical outcomes in small samples. *Journal of Consulting and Clinical Psychology, 82*, 854–867.

Pardo, A., & Román, M. (2013). Reflections on the Baron and Kenny model of statistical mediation. *Annales de Psicología, 29*, 614–623.

Pearl, J. (2001). *Direct and indirect effects*. Proceedings of the 17th Conference on Uncertainty in Artificial Intelligence (pp. 411–420). San Francisco, CA: Morgan Kaufmann.

Pearl, J. (2009). *Causality: Models, reasoning, and inference*. Cambridge, UK: Cambridge University Press.

Pearl, J. (2012). The causal mediation formula: A guide to the assessment of pathways and mechanisms. *Prevention Science: The Official Journal of the Society for Prevention Research, 13*(4), 426–436.

Pedhazur, E. J. (1997). *Multiple regression in behavioral research: Explanation and prediction* (3rd ed.). Fort Worth, TX: Harcourt Brace College Publishers.

Pinheiro, J. C., & Bates, D. M. (2000). *Mixed-effects models in S and S-plus*. New York, NY: Springer.

Pituch, K. A., Stapleton, L. M., & Kang, J. Y. (2006). A comparison of single sample and bootstrap methods to assess mediation in cluster randomized trials. *Multivariate Behavioral Research, 41*, 367–400.

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, and Computers, 36*, 717–731.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*, 879–891.

Preacher, K. J., Rucker, D. D., & Hayes, R. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research, 42*, 185–227.

Preacher, K. J., Zyphur, M. J., & Zhang, Z. (2010). A general multilevel SEM framework for assessing multilevel mediation. *Psychological Methods, 15*, 209–233. doi: 10.1037/a0020141

R Development Core Team. (2013). *R: A language and environment for statistical computing (Version 2.6.1)* [Computer software]. Vienna, Austria: R Foundation for Statistical Computing.

Rast, P., & Hofer, S. M. (2014). Longitudinal design considerations to optimize power to detect variances and covariances among rates of change: Simulation results based on actual longitudinal studies. *Psychological Methods, 19*, 133–154.

Raudenbush, S. W. (2001). Comparing personal trajectories and drawing causal inferences from longitudinal data. *Annual Review of Psychology, 52,* 501–525.

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2nd ed.). Thousand Oaks, CA: Sage.

Raudenbush, S. W., Bryk, A. S., Cheong, Y. F., Congdon, R. T., & du Toit, M. (2011). *HLM 7: Hierarchical linear and nonlinear modeling*. Chicago, IL: Scientific Software International.

Raudenbush, S. W., & Liu, X. F. (2001). Effects of study duration, frequency of observation, and sample size on power in studies of group differences in polynomial change. *Psychological Methods, 6*, 387–401.

Reisby, N., Gram, L. F., Bech., P., Nagy, A., Petersen, G. O., Ortmann, J., … Christiansen, J. (1977). Imipramine: Clinical effects and pharmacokinetic variability. *Psychopharmacology, 54*, 263–272.

Rigdon, E. E., Schumacker, R. E., & Wothke, W. (1998). A comparative review of interaction and nonlinear modeling. In R. E. Schumacker & G. A. Marcoulides (Eds.), *Interaction and nonlinear effects in structural equation modeling* (pp. 1–16). Mahwah, NJ: Erlbaum.

Robins, J. M., & Greenland, S. (1992). Identifiability and exchangeability for direct and indirect effects. *Epidemiology, 3*, 143–155.

Robins, J. M., & Hernan, M. A. (2009). Estimation of the causal effects of time-varying exposures. In G. Fitzmaurice, M. Davidian, G. Verbeke, & G. Molenberghs (Eds.), *Longitudinal data analysis* (pp. 553–599). New York, NY: Chapman & Hall.

Robins, J. M., Hernán, M. A., & Brumback, B. (2000). Marginal structural models and causal inference in epidemiology. *Epidemiology, 11*(5), 550–560. Retrieved from http://www.jstor.org/stable/3703997

Robinson, W. S. (1950). Ecological correlations and the behavior of individuals. *American Sociological Review, 15*, 351–357.

Rogosa, D. (1988). Myths about longitudinal research. In K. W. Schaie, R. T. Campbell, W. Meredith, & S. C. Rawlings (Eds.), *Methodological issues in aging research* (pp. 171–209). New York, NY: Springer.

Rogosa, D., Brandt, D., & Zimowski, M. (1982). A growth curve approach to the measurement of change. *Psychological Bulletin, 92*(3), 726–748.

Rose, B. M., Holmbeck, G. N., Coakley, R. M., & Franks, E. A. (2004). Mediator and moderator effects in developmental and behavioral pediatric research. *Journal of Developmental and Behavioral Pediatrics, 25*, 58–67.

Rubin, D. B. (1974). Estimating causal effects of treatments in randomized and nonrandomized studies. *Journal of Educational Psychology, 66,* 688–701. doi: 10.1037/h0037350

Rutter, M. (1999). Resilience concepts and findings: Implications for family therapy. *Journal of Family Therapy, 21*, 119–144.

SAS Inc. (2014). *SAS (Version 9.3)* [Computer software]. Cary, NC: SAS Inc.

Schneider, H., & Eisenberg, D. (2006). Who receives a diagnosis of attention deficit/hyperactivity disorder in the United States elementary school population? *Pediatrics*, *117*(4), e601–e609.

Selig, J. P., Preacher, K. J., & Little, T. D. (2012). Modeling time-dependent association in longitudinal data: A lag as moderator approach. *Multivariate Behavioral Research, 47*, 697–716.

Shen, E., Chou, C. P, Pentz, M. A., & Berhane, K. (2014). Quantile mediation models: A comparison of methods for assessing mediation across the outcome distribution. *Multivariate Behavioral Research*, *49*, 471–485. DOI:10.1080/00273171.2014.904221

Singer, J. D., & Willett, J. B. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. Oxford, UK: Oxford University Press.

Sobel, M. E. (1982). Asymptotic confidence intervals for indirect effects in structural equation models. In S. Leinhardt (Ed.), *Sociological methodology* (pp. 290–312). Washington, DC: American Sociological Association.

Sobel, M. E. (1986) Some new results on indirect effects and their standard errors in covariance structure models. In N. Tuma (Ed.), *Sociological methodology* (pp. 159–186). Washington, DC: American Sociological Association.

Sobel, M. E. (2008). Identification of causal parameters in randomized studies with mediating variables. *Journal of Educational and Behavioral Statistics, 33,* 230–251. doi: 10.3102/1076998607307239.

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research, 25*, 173–180.

Stone, S., Brown, T., & Hinshaw, S. (2010). ADHD-related school compositional effects: An exploration. *Teachers College Record*, *112*(5), 3–4.

Stoolmiller, M. (1994). Antisocial behavior, delinquent peer association, and unsupervised wandering for boys: Growth and change from childhood to early adolescence. *Multivariate Behavioral Research, 29*, 263–288.

Ten Have, T. R., Joffe, M. M., Lynch, K. G., Brown, G. K., Maisto, S. A., & Beck, A. T. (2007). Causal mediation analysis with rank preserving models. *Biometrics, 63,* 926–934. doi: 10.1111/j.1541-0420.2007.00766.x

Tofighi, D., & MacKinnon, D. P. (2011). RMediation: An R package for mediation analysis confidence intervals. *Behavior Research Methods, 43*(3), 692–700. doi: 10.3758/s13428-011-0076-x

Toh, S., & Hernan, M. A. (2008). Causal inference from longitudinal studies with baseline randomization. *International Journal of Biostatistics*, *4*(1), Article 22. doi: 10.2202/1557-4679.1117.

Tourangeau, K., Nord, C., Lê, T., Sorongon, A. G., & Najarian, M. (2009). *Early childhood longitudinal study, kindergarten class of 1998–99 (ECLS-K), combined user's manual for the ECLS-K eighth-grade and K–8 full sample data files and electronic codebooks (NCES 2009–004)*. Washington, DC: National Center for Education Statistics, Institute of Education Sciences, U.S. Department of Education.

U.S. Department of Education, National Center for Education Statistics. (2009). Early childhood longitudinal study, kindergarten class of 1998–99 (ECLS-K) kindergarten through eighth grade full sample public-use data and documentation (DVD). *(NCES 2009-005)*. Washington, DC: Author.

Valeri, L., & VanderWeele, T. J. (2013). Mediation analysis allowing for exposure-mediator interactions and causal interpretation: Theoretical assumptions and implementation with SAS and SPSS macros. *Psychological Methods*, *18*, 137–150.

VanderWeele, T. J. (2010). Bias formulas for sensitivity analysis for direct and indirect effects. *Epidemiology, 21*, 540–551. doi: 10.1097/EDE.0b013e3181df191c

VanderWeele, T. J., Hawkley, L. C., Thisted, R. A., & Cacioppo, J. T. (2011). A marginal structural model analysis for loneliness: Implications for intervention trials and clinical practice. *Journal of Consulting and Clinical Psychology 79*(2), 457–468.

VanderWeele, T. J., & Vansteelandt, S. (2009). Conceptual issues concerning mediation, interventions and composition. *Statistics and Its Interface (Special Issue on Mental Health and Social Behavioral Science), 2*, 457–468.

VanderWeele, T. J., & Vansteelandt, S. (2013). Mediation analysis with multiple mediators. *Epidemiologic Methods, 2*(1), 95–115.

Vansteelandt, S. (2009). Estimating direct effects in cohort and case–control studies. *Epidemiology*, *20*, 851–860.

Voelkle, M. C., Oud, J. H. L., Davidov, E., & Schmidt, P. (2012). An SEM approach to continuous time modeling of panel data: Relating authoritarianism and anomia. *Psychological Methods, 17*, 176–192.

Wall, M. M., & Amemiya, Y. (2000). Estimation for polytomial structural equation models. *Journal of the American Statistical Association, 95*, 929–940.

West, S. G. (2011). Causal inference in cross sectional and longitudinal meditational models: Introduction to the special section. *Multivariate Behavioral Research, 46*, 812–815.

Wiley, D. E., & Wiley, J. A. (1970). The estimation of measurement error in panel data. *American Sociological Review, 35*, 112–117.

Willett, J. B. (1997). Measuring change: What individual growth modeling buys you. In E. Amsel & K. A. Renninger (Eds.), *Change and development: Issues of theory, method, and application* (pp. 213–243). Mahwah, NJ: Lawrence Erlbaum Associates.

Willett, J. B., & Sayer, A. G. (1994). Using covariance structure analysis to detect correlates and predictors of individual change over time. *Psychological Bulletin, 116*, 363–381.

Winship, C., & Morgan, S. L. (1999). The estimation of causal effects from observational data. *Annual Review of Sociology, 25*, 659–707.

Yuan, Y., & MacKinnon, D. P. (2009). Bayesian mediation analysis. *Psychological Methods, 14*, 301–322.

Zhao, X., Lynch, J. G., & Chen, Q. (2010). Reconsidering Baron and Kenny: Myths and truths about mediation analysis. *Journal of Consumer Research, 37*, 197–206.

CHAPTER 22

Latent Growth Modeling and Developmental Psychopathology

JUNGMEEN KIM-SPOON and KEVIN J. GRIMM

CORE THEORETICAL PRINCIPLES OF DEVELOPMENTAL PSYCHOPATHOLOGY AND LATENT GROWTH MODELING APPROACHES 986
The Pathways Framework 987
Resilience 987
Multiple Levels of Analysis 988
Normality and Psychopathology 988
Developmental Analysis 989
BASIC ASSUMPTIONS AND LONGITUDINAL DESCRIPTIVE ANALYSES 989
Primary Goals of Longitudinal Research 989
Planning and Sampling in a Longitudinal Study 989
Key Issues in Latent Growth Modeling 990
THE LATENT GROWTH MODELING 995
Historical Development 995
Latent Growth Curve as a Structural Equation Model 996
Common Latent Growth Models 997
MULTILEVEL MODELING FOR STUDYING DEVELOPMENTAL TRAJECTORIES 1000
Alternative Specification 1001
Specific Growth Models 1001
Direct Expansions of the Latent Growth Modeling Framework 1002
Multiple Group Growth Model 1002
Latent Growth Modeling Studies for Understanding Developmental Pathways in Terms of Continuity–Discontinuity and Equifinality–Multifinality 1003
Latent Growth Modeling Studies for Understanding Contextual Influences 1009
LATENT CHANGE (DIFFERENCE) SCORE MODELING 1015
Univariate Latent Change Score Models 1015
Bivariate Latent Change Score Models 1017
Latent Change Score Modeling Studies for Understanding Risk and Protective Mechanisms as Predictors, Mediators, and Moderators 1018
Latent Change Score Modeling Studies for Understanding Contextual Influences 1021
GROWTH MIXTURE MODELING 1027
Growth Mixture Model 1027
Cautions 1029
Growth Mixture Modeling Studies for Understanding Risk and Protective Mechanisms as Predictors, Mediators, and Moderators 1029
Growth Mixture Modeling Studies for Understanding Contextual Influences 1032
Growth Mixture Modeling for Multiple Levels of Analysis 1034
CONCLUSION AND FUTURE PERSPECTIVE 1036
REFERENCES 1037

CORE THEORETICAL PRINCIPLES OF DEVELOPMENTAL PSYCHOPATHOLOGY AND LATENT GROWTH MODELING APPROACHES

In this chapter, variations of latent growth modeling and their applications in developmental psychopathology are reviewed. In each section, we first discuss developmental methodology and quantitative techniques for studying developmental trajectories in developmental psychopathology, focusing on latent growth modeling. We begin by reviewing core principles of developmental psychopathology, which is conceptualized as an evolving interdisciplinary science that studies the interplay among the biological, psychological, and social-contextual aspects of normal and abnormal development across the lifespan (Cicchetti, 2010, 2015). Developmental psychopathology posits the following important questions: What are the causes of a particular disorder? What are the transformations in behavioral (or phenotypic)

Jungmeen Kim-Spoon was supported by a grant from the National Institute of Drug Abuse (DA036017). Kevin J. Grimm was supported by National Science Foundation Grants (REESE Program Grant-DRL-0815787 and REAL Program Grant-1252463). We thank Samanata Shrestha for her editorial assistance.

manifestation? Where do changes happen in the pathway to psychopathology?

Within the developmental psychopathology perspective, maladaptation is viewed as evolving through the successive adaptations in the person's environment. That is, maladaptation is the complex result of a myriad of risk and protective factors operating over time; and it is not some sort of trait a person has or an expression of a pathogen (Sroufe, 1997). Accordingly, key research questions in developmental psychopathology focus on (1) discovery of factors that place individuals on *pathways* probabilistically leading to later disturbances, and (2) *processes,* which maintain individuals on such pathways once enjoined.

Developmental psychopathology is a perspective that is especially applicable to the investigation of critical transitional turning points in development across the life span. Empirical research in developmental psychopathology involves knowing about the trajectories and pathways of disorders across the life span and asking the following questions: What happened before the onset of the disorder? How does it progress? Do the manifestations of the symptoms continue or stop at some point? As disorders may appear at any point in the life span, it is imperative to examine the course, phases, and sequelae of disorders. The goals of developmental psychopathology are broad and integrative, seeking to understand adaptation and maladaptation across the life course, and to comprehend the complex interplay among multiple levels of functioning including biological, psychological, and social processes in the development.

Next, a few core theoretical principles of developmental psychopathology guide our reviews of empirical studies using latent growth modeling.

The Pathways Framework

The premise of developmental psychopathology is that the same laws that govern normal development also govern pathological development (Cicchetti, 2015). Therefore, pathology is viewed in terms of developmental deviation and a significant deviation in adaptation represents an increased probability of problems in dealing with (negotiating) subsequent developmental issues. A particular adaptational failure at any point in time is best viewed as placing an individual on a pathway (or trajectory) potentially leading to disorder. For example, anxious attachment (maladaptive pattern of attachment) is not viewed as psychopathology per se, but in terms of developmental risk for disturbance. Developmental psychopathology also believes that change is possible at any points on the pathway. Even when a maladaptive pathway is enjoined, return to positive functioning often remains possible. Thus, this perspective emphasizing the concept of pathways is closely related to the concept of resilience.

Developmental psychopathology also emphasizes that the same rules of development do not necessarily apply to all children and families. In this regard, two theoretical concepts are relevant. First, *equifinality* suggests that there are multiple pathways to similar manifest outcomes. For example, a pattern of maladaptation that has many features in common, such as depressive mood, lack of social engagement, and low self-esteem, may be the result of distinctly different developmental pathways, one rooted in alienation, and one rooted in anxiety and helplessness. Second, *multifinality* suggests that a single pathway can lead to multiple outcomes. For example, not all maltreated children develop similar disorders or any disorders. Some maltreated children show depression, others show conduct disorder, and still others show substance use problems. Yet there still are some maltreated children who do not show any critical psychopathology.

Resilience

Developmental psychopathologists are interested in studying pathways to competent adaptation despite exposure to conditions of adversity because the results will help obtain a better understanding of both normal development and psychopathology. Resilience refers to the fact that some children facing adversity nonetheless do well or return to positive functioning following a period of maladaptation (Sroufe, 1997). Resilience is shown in the positive end of the distribution of developmental outcomes among individuals at high risk. These resilient individuals maintain adaptive functioning in spite of serious risk hazards (Rutter, 1990). Developmental psychopathologists see resilience as a dynamic process encompassing positive adaptation within the context of significant adversity.

According to Luthar, Cicchetti, and Becker (2000), two critical conditions are implicit in this notion: (1) exposure to significant threat or severe adversity; and (2) the achievement of positive adaptation despite major assaults on the developmental process. Luthar and colleagues (2000) further differentiated diverse developmental processes determined by protective and vulnerability factors. Protective factors have direct ameliorative effects at both high- and low-risk conditions. Thus, protective effects are exhibited when the main overall effects on the at-risk individuals are promoting their positive functioning. Such main effects of protective factors can be described as

protective-stabilizing processes where individuals with the attribute show stability in competence despite increasing risk, whereas individuals without the attribute decrease in competence. Protective factors may operate to have interactive or moderating effects. First, protective-enhancing processes may be seen when the attribute (moderator) allows individuals to interact with stress in such a way that their competence is augmented with increasing risk. Second, protective-reactive processes may be described when the attribute generally confers advantages but less so when risk is high.

In contrast to protective factors, vulnerability factors are identified when the main overall effects on the at-risk individuals are negative, thus demoting their positive functioning. Those individuals with the attribute are expected to manifest greater maladjustment at both high- and low-risk conditions. Such main effects of vulnerability factors are described as vulnerable-stable processes, where the general disadvantages of individuals with the attribute remain stable despite changing levels of stress. Interactive or moderating effects of vulnerability factors can be shown in vulnerable and reactive processes where the overall disadvantage linked with the attribute is heightened with increasing levels of stress. Overall, these vulnerability and protection processes illustrate that the impact of stress on competence levels depends on protective or vulnerability factors.

Indeed, prior research in developmental psychopathology suggests that many risk factors do not have direct, additive effects but rather indirect, interactive effects. Rutter (1990) demonstrated that risk factors did not lead to an increased rate of psychiatric disorder if they occurred truly in isolation. Rather, the rate was increased if there were two or more concurrent risk variables. For example, family discord has an adverse effect only when it is accompanied by other adversities. Research on the impact of parental divorce suggests that divorce accompanies many stressors that may take a toll on children, including economic hardship (decreased income for the single-mother family) and maternal depression (Hetherington, Bridges, & Insabella, 1998). Researchers in developmental psychopathology should be reminded that one should consider the impact of a particular risk factor in the presence of other independent or related risk factors. This perspective is related to the multiple level approach principle in developmental psychopathology discussed in the following section.

Multiple Levels of Analysis

Most developmental psychopathologists will agree that singular, linear cause of psychopathology will rarely be obtained. More often, adequate prediction of either disturbance or resilience requires considering multiple risks and protective factors and their interplay (Cicchetti & Sroufe, 2000). In addition, within a developmental psychopathology perspective, the development of psychopathology is viewed as unfolding along different developmental pathways among different individuals. Thus, it is expected that relevant causal processes vary among individuals who show the same pattern of disorders (i.e., equifinality), and that there is heterogeneity in the expression of disorders (i.e., multifinality) (Cicchetti, 2010; Cicchetti & Rogosch, 1996). Therefore, in studying developmental pathways and processes of psychopathology, it is important to investigate simultaneously different psychopathological outcomes as well as individuals' functioning in multiple domains of development and across multiple settings.

Developmental psychopathology studies involving multiple levels of analyses have been increasing. For example, recent empirical studies of resilience have incorporated neurobiological and molecular genetic analyses in addition to the traditional measures of psychosocial contributors (Cicchetti & Curtis, 2007). The integrated knowledge gained by employing multiple biological and psychological levels of analyses is particularly beneficial for contributing to the ability to design individualized preventive interventions to facilitate the development of resilient functioning (Cicchetti & Toth, 2006).

Contextual factors play an important role in shaping the processes of maladaptation, psychopathology, and resilience. For example, prior research demonstrates that social-contextual experiences can influence neurobiological structure and functioning (Cicchetti & Tucker, 1994; Grossman et al., 2003). Within a developmental psychopathology perspective, researchers are goaded to move away from the search for single pathogens and to move toward the search for a complex of influences that initiate a developmental pathway that is probabilistically associated with disturbances. Thus, systematic examination of multiple levels of contexts, including community-, institutional-, and societal-level influences, is encouraged in developmental psychopathology research (Cicchetti & Rogosch, 1999).

Normality and Psychopathology

Why do developmental psychopathologists study atypical, high-risk populations? It is because they believe not only that knowledge from the investigation of normal development can inform the study of high-risk conditions and psychopathology but also that the investigation of

risk and pathology can enhance our comprehension of normal development (Cicchetti, 2010, 2015). Developmental psychopathology is studying how individuals function through examinations of extremes in the distribution. For example, as will be illustrated later, studies of maltreatment indicate that children who have experienced the extremes of dysfunction in parenting tend to show a broad range of psychological maladaptation. Such a finding indicates the criticality of adaptive parenting practices in the development of normal populations.

Developmental Analysis

In developmental psychopathology research, it is necessary to examine the relationships between predictors and problem behaviors over time and across ages (Cicchetti, 2015; Cicchetti & Rogosch, 1999). Accordingly, longitudinal research beginning even prior to the onset of disorder is important since such research is necessary for untangling causal mechanisms and processes. Some variables may have stronger links with onset of problems, while others may be more tied to persistence or desistance of the symptoms. Only longitudinal research can identify risk and protective factors associated with different stages in the progression of psychopathology. More importantly, examining longitudinal trajectories (as opposed to a snapshot) is essential for identifying developmental pathways to maladaptation, psychopathology, and resilience, as well as understanding diversity in process and outcomes of maladaptation, psychopathology, and resilience. As such, the core theoretical principles of developmental psychopathology demand empirical evaluation based on longitudinal data.

BASIC ASSUMPTIONS AND LONGITUDINAL DESCRIPTIVE ANALYSES

Primary Goals of Longitudinal Research

In their seminal paper, Baltes and Nesselroade (1979) described the history and rationale for conducting longitudinal research. In their work, they outlined five rationales for collecting longitudinal data, which could be studied only with longitudinal, repeated-measures data: identifying (1) intraindividual change; (2) interindividual variability in intraindividual change; (3) interrelationships among intraindividual changes; (4) determinants of intraindividual change; and (5) interindividual variability in the determinants of intraindividual change. Additionally, Baltes and Nesselroade indicated that the primary reason for collecting longitudinal data was the identification of intraindividual change. Since their seminal paper, the primary approach used to evaluate and model intraindividual change is the latent growth model (McArdle, 1986; Meredith & Tisak, 1984, 1990).

The latent growth model not only allows for the study of intraindividual or within-person change but also allows for the study of interindividual variability in intraindividual change (or between-person differences in within-person change) and determinants of intraindividual change (or causes/mechanisms of within-person change). The latent growth model has grown out of several modeling traditions, but is now dominated by the structural equation modeling (SEM) and multilevel modeling (MLM; also referred to as the mixed-effects and random coefficient model) frameworks. The SEM and MLM frameworks each have benefits and limitations (Ghisletta & Lindenberger, 2004); however, over time, the two frameworks have grown to be more similar, and the relative advantages of each approach have been minimized.

Planning and Sampling in a Longitudinal Study

The study of intraindividual change should be approached with careful planning and design. The design of a longitudinal study requires thoughtful consideration regarding the sampling of individuals, the sampling of measurement occasions, and the sampling of constructs (Cattell, 1952). The sampling of individuals is a first obvious consideration when designing any research study; however, additional considerations need to be made when planning a longitudinal study because the sampling of individuals can impact the time metric (sampling of measurement occasions) as well as the sampling of constructs.

In a longitudinal study, the time metric, the time scale over which change has occurred, can be referenced by the beginning of the study. Example time metrics may be *measurement occasion* number (e.g., 1, 2, 3) and *chronological time* (e.g., 1 month, 12 months, 24 months). Such time metrics are common in psychopathology when studying intraindividual change subsequent to the beginning of an intervention (e.g., baseline, 1 month follow-up, 12-month follow-up). Alternatively, the time metric can be referenced at the individual level. Example time metrics may be chronological time since birth (age), time since an individual event (e.g., birth of child, marriage, death of family member), and time to an individual event (e.g., time to crawling, time until death). When planning of structuring change over an individual time metric, the sampling of individuals requires careful thought.

For example, a study of intraindividual changes in depression during adolescence could be structured as a cohort-based study where 11-year-olds are sampled at the first occasion and then followed for the next five years at regular or irregular intervals. Alternatively, a sample of 11-year-olds, 12-year-olds, and 13-year-olds can be sampled at the first occasion and the followed for the next 3 years at regular or irregular intervals. This type of accelerated longitudinal study (Bell, 1954) requires a different sampling of individuals.

The sampling of individuals also influences the sampling of constructs and the scales intended to measure the construct. Often, researchers will choose scales that are appropriate for measuring interindividual differences within the sample at the first measurement occasion without considering whether the scales are sensitive to expected intraindividual changes or whether the scales will be appropriate for the sample at the conclusion of the study. Measures must be sensitive to intraindividual change over the time period studied; however, most psychological measures are not evaluated for this ability—the majority of scales are psychometrically evaluated with a cross sectional sample. It is important to note that reliable and valid measures for studying interindividual differences are not necessarily reliable and valid for measuring intraindividual change.

A second consideration for the sampling of scales is to look for scales that remain appropriate for the sample at the conclusion of the study. Often, the same measurement scales are utilized over the entire course of the study because researchers want to use the observed changes in the scores as a dependent variable. If this is done, it is important for the measures to be valid and reliable for all individuals at all time points and sensitive to change over the entire range of the time that the scale is utilized. For example, utilizing a child behavior problem scale for 3- to 5-year-olds may not be optimal when studying development from ages 4 to 7 because the sample ages out of the age range for which the scale was developed.

Alternatively, researchers can plan for changes in the measurement of constructs over the course of the study. For some studies, it is unreasonable to have measures that are reliable, valid, and sensitive to intraindividual change over the entire course of the longitudinal study due to the length of the study and the sample characteristics and how the sample characteristics are expected to change over the course of the study. For example, studies of lifespan development may need to utilize different assessments over the course of the study as the sample ages (e.g., McArdle et al., 2009). In these cases, it is essential to build in scale and/or item overlap across time to create scores that are quantitatively comparable. If this is not done, another approach is to use a separate sample to do equating/linking. There are upsides (e.g., do not over burden longitudinal sample) and downsides (e.g., inability to examine invariance within longitudinal sample) of this approach.

Thoughtful consideration for the sampling of measurement occasions is also key to gaining the most information from the longitudinal study. The sampling of measurement occasions includes the number of measurement occasions as well as the timing or spacing of measurement occasions. However, a third consideration is whether or not the number of measurement occasions and the timing of assessments is the same or varies over individuals (McArdle & Woodcock, 1997). For this we simply note that much can be gained by intentionally building flexibility and variability into the measurement schedule of individuals and note that all aspects of the sampling of measurement occasions can be studied through simulation prior to any data collection (see Ferrer & Grimm, 2012). The sampling of measurement scales was discussed above, so here we simply reiterate the importance of utilizing measurement instruments that are sensitive to change over the planned data collection period and that many validated instruments may be unable to track the growth and changes taking place within the individual.

Key Issues in Latent Growth Modeling

Primary issues to the successful application of latent growth models include issues related to the measurement of the outcome of interest and issues related to the time scale over which the developmental process occurs and over which change is statistically modeled (Grimm & Ram, 2011).

Measurement

Latent growth models are typically applied to repeated-measures data obtained from multiple individuals. Among the first questions when approaching such data are how the construct was measured. Specific questions that arise are as follows:

1. What are the measurement properties of the instrument?
2. Are the measurement properties equivalent over time?
3. Was the instrument sensitive to and able to capture within-person change?
4. Are there distributional issues that need to be considered, such as ceiling effects, floor effects, skew, and

whether the variable provides limited information (e.g., binary, ordinal variables)?

5. Most importantly, is the instrument measuring the same construct in the same metric across time?

Ideally, the measurement instrument should have strong measurement properties (reliability and validity) at each measurement occasion. Additionally, the measurement instrument should be sensitive to change over the time scale of the study and at the level of aptitude (ability, trait-level) of the population. For example, utilizing the Woodcock-Johnson Applied Problems subtest may be useful for measuring changes in mathematics-related skills over a 2-year period for a sample of third to fifth grade students. However, the same measure is inappropriate for measuring mathematics-related skills over a 2-month period in a sample of preschool children. The reasons for the difficulty level of the test and the implied level of aptitude of the samples as well as the time span of the study must be thoughtfully considered. Moreover, certain instruments that are ideal for measuring relatively large changes in aptitude are poor at measuring relatively small changes in aptitude.

A check on the distribution of scores from the instrument over time can highlight potential issues when approaching the data from a modeling standpoint. Ideally, scores will be more or less normally distributed at each measurement occasion or point along the time metric (e.g., each grade, each year). Deviations from normal distributions can indicate potential issues as well as potential opportunities. Truncated distributions, such as those with floor or ceiling effects, may require statistical adjustment when modeling. Depending on the nature of truncation, models can be fit to account for the truncation—for example, ceiling or floor effects can be statistically modeled as well as zero-inflated distributions (see Olsen & Schafer, 2001). If these distributional anomalies are not accounted for within the statistical model, incorrect solutions and conclusions can be drawn (Wang, Zhang, McArdle, & Salthouse, 2008). Nonnormal distributions, such as those that arise from count data (e.g., How many cigarettes smoked within the last 2 weeks?) or binary data (e.g., Have you smoked within the past 2 weeks?) should be modeled with the appropriate statistical distribution (e.g., Poisson, logit). Otherwise, model fit and parameter estimates can be biased. Alternatively, a skewed distribution may indicate the need for a mixture distribution (McLachlan, & Peel, 2000), which can arise because the sample is actually comprised from multiple subpopulations. In these situations, a growth mixture model (Muthén & Muthén, 2000; Muthén & Shedden, 1999) or latent class trajectory model (Nagin, 1999) is needed.

One of the most important considerations is that the instrument measures the *same construct* in the *same metric* at each point along the time metric. This enables the scores obtained from the instrument to be compared quantitatively. Thus, intraindividual change can be tracked. The question of whether the *same construct* was measured in the *same metric* is a question of measurement invariance (Meredith, 1964a, 1964b, 1965, 1993). Statistically, this question can be examined through fitting a series of item factor analyses or item response models (see Meredith & Horn, 2001; Widaman & Reise, 1997). However, when a single score is obtained from an instrument, measurement invariance is often assumed without thorough investigation. In these cases, researchers run the risk of drawing incorrect conclusions. It is not appropriate to assume measurement invariance holds simply because the same questions were asked at each measurement occasion; however, this is often done. Additionally, it is incorrect to assume measurement invariance does not hold when different questions were asked at different measurement occasions (see McArdle et al., 2009).

Time Metrics

A second key issue for understanding intraindividual change is the time metric, over which intraindividual change is thought to occur and the time scale for statistically tracking longitudinal change. Within the growth curve model, there is a single dominant time scale used to track intraindividual change. This dominant time scale serves as a proxy for tracking the time over which the underlying change process occurs (Grimm & Ram, 2011). Examples of time scales are chronological age, gynecological or maturational age, time since the beginning of the study, measurement occasions, time until death or other event (e.g., marriage, graduation), and grade in school. To further illustrate this point, we have plotted physical height for a sample of females from the National Longitudinal Survey of Youth–Children and Young Adults (NLSY-CYA; Center for Human Resource Research, 2004) in Figure 22.1 with different time scales. We chose the NLSY-CYA because this study began in 1986 with a sample of children from mothers who participated in the National Longitudinal Survey of Youth in 1979 (NLSY-79). Thus, children were different ages in 1986, and children were also added to the study as the study progressed. Furthermore, we chose physical height because its developmental progression is strongly influenced by chronological and maturational age.

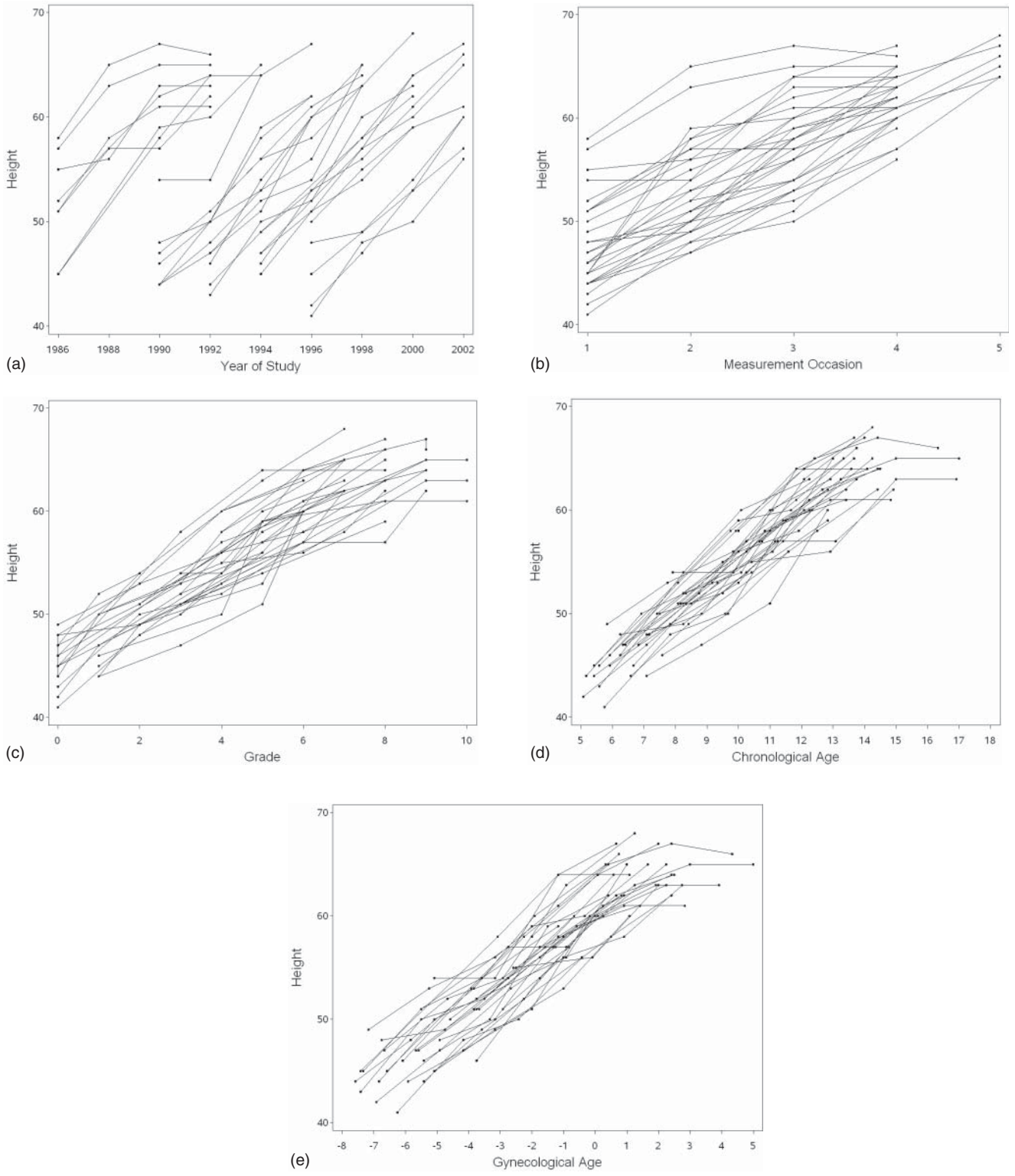


Figure 22.1 Longitudinal plots of individual changes in height based on different time metrics for a sample of female participants of the National Longitudinal Survey of Youth–Children and Young Adults. *Source*: Bureau of Labor Statistics, U.S. Department of Labor, and National Institute for Child Health and Human Development. Children of the NLSY79, 1979–2010. Produced and distributed by the Center for Human Resource Research, The Ohio State University. Columbus, OH: 2012.

Figure 22.1a is a plot of physical height against year in study from 1986 through 2002. This is obviously an inappropriate time scale to model changes in physical height given what we know about the developmental process and given the lack of systematicity in the individual trajectories of the longitudinal plot. At the same time, this plot is helpful because it highlights how different participants began the study in different years. Figure 22.1b is a plot of the same data against measurement occasion. Measurement occasion is a commonly utilized time scale for many longitudinal studies; however, for modeling changes in physical height in the NLSY-CYA, it is also inappropriate because participants entered the study at different ages. Additionally, measurement occasion, as a researcher-defined time scale, is unlikely to be the time scale over which intraindividual change is dependent upon (Grimm & Ram, 2011). On the positive side, this plot highlights how participants have a different number of measurement occasions.

Figure 22.1c is a plot of physical height against year in school or grade. Grade is closely aligned with chronological age and maturational age, but is also inappropriate for studying individual changes in physical height. One issue that is evident in this plot is that children may repeat a grade (or two) and this can be seen as vertical lines within an individual trajectory. That is, the value of the time scale remains the same at two different measurement occasions. Obviously, grade could be measured more finely, such as grade plus the percent of school completed since the beginning of the school year or the amount of time since the child began kindergarten for the first time. Ideally, the chosen time metric will accurately represent the amount of time that has elapsed between assessments. For these data, year in school or grade does not adequately do this.

Figure 22.1d is a plot of physical height against chronological age. Here, we see that children began the study at different ages and begin to see the developmental process that we know—stable increases in height during childhood, rapid increases during the pubertal years, and little to no changes in height during late adolescence/early adulthood. This is the time metric often utilized when modeling changes in physical height (see Grimm et al., 2011). We note that chronological age is a common time-metric when studying intraindividual changes in many types of biological and psychological constructs; however, other scalings of age may be as, or even more, appropriate (Nesselroade & Featherman, 1997). As a final illustration relevant to this point, we have plotted physical height against gynecological age in Figure 22.1e. Gynecological age was defined as time to or time since the individual's age of menarche. In this figure, we see that changes in physical height were somewhat more rapid near age of menarche (gynecological age = 0) compared with changes that occurred after or before menarche. Certain research questions and theories posit alternative scalings of age as the ideal time scale to model intraindividual change and these should be utilized to the extent possible (e.g., terminal decline; Gerstorf et al., 2008).

Ideally, the chosen time metric will adequately represent the amount of time that has passed between assessments as well as represent a dominant reason why the intraindividual changes have occurred as they have. One approach is to consider the time metric, which is able to align participants, such that the shape of developmental changes is most similar. In practice, we recommend examining multiple feasible time metrics before proceeding with analyses. This can be done with a series of plots, as in Figure 22.1, and done conceptually and theoretically. In the majority of situations, we acknowledge that multiple ongoing time metrics are likely to impact intraindividual change (Grimm & Ram, 2011). For example, changes in depression over adolescence may be related to schooling demands and peers, which change with *year in school*, as well as demands at home, which are likely associated with *chronological age*, as well as biological changes taking place within the individual associated with *maturational age*. These time metrics are strongly associated with one another (highly collinear), forcing researchers to choose between them or model them jointly and deal with the presence of collinearity. We refer readers to McArdle and Bell (1954), Ram et al. (2010), and Wohlwill (1973) for discussions regarding the choice of time metric in latent growth curve models.

Modeling Intraindividual Change

Once the time metric is decided upon, the next step is to determine, at the individual level, how the observed trajectories are associated with the chosen time metric. The majority of applications in growth curve modeling focus on simple functional forms of change, such as linear and quadratic change functions. However, the true underlying change process is likely to be more complicated. Here, a balance must be struck between the complexity of the model and the amount of information present in the data.

There are several approaches to choosing an appropriate individual change model. The first, and most common, approach is to begin with simple change functions and progress through a series of viable alternatives chosen based upon an individual plot, such as those presented in Figure 22.1. The simplest change function is a no-change, no-growth, or intercept-only model, where each individual

has an intercept score that is maintained across the observation period. This is often a model researchers would like to reject, but is a common starting point because of its simplicity and because the model yields fit statistics that can be statistically compared with more complex models because the intercept only model is nested under most growth models. Next, a linear model is fit, which is followed by a quadratic model and higher order polynomials if necessary. Finally, a few inherently nonlinear models (e.g., exponential, Gompertz, logistic) are fit that may be able to mimic the observed overall change pattern (see Grimm & Ram, 2009).

A second approach is to consider the important characteristics of the developmental process and fit a model with those properties, and if this model fits the data well then use the model. For example, important aspects (or parameters where individuals are likely to differ) in the developmental process related to changes in physical height may be rates of change in infancy, childhood, and adolescence along with the timing of puberty and a final adult height. Preece and Baines (1978) derived multiple functional forms with many of these important aspects and applied them to longitudinal height data and evaluated model fit (see also Grimm et al., 2011). We do, however, acknowledge that this approach is challenging when considering the range of psychological constructs and the general lack of strong theories describing important changes in such constructs.

A third approach is to model intraindividual change patterns with a series of splines (see Cudeck & Klebe, 2002; Ram & Grimm, 2007)—a series of lines or simple curves (e.g., quadratic) that connect at specific points in time (knot points) that can be chosen by the researcher or estimated from the data. These models are sometimes referred to as *multiphase* models because the various splines can represent phases of development that have beginning and end points. These models are especially useful when the underlying developmental process is thought to be guided by shifts or when the developmental process is interrupted by an external event, such as a death in the family. In this situation, changes prior to the event and after the event can be thought of as having distinct developmental processes.

A fourth approach is to let the data speak. There is a variety of what we would call exploratory approaches for modeling change (see Grimm, Steele, Ram, & Nesselroade, 2013). For example, Meredith and Tisak (1990) fit a latent basis or unstructured growth model, where an ideal structure of change was derived from the data. Functional forms of change that mimicked this derived change pattern were fit to the data and evaluated. Grimm et al. (in press) advocated a completely exploratory approach to growth modeling where a series of models are fit with an increasing number of latent variables. The associations between the time-metric and the observed trajectories are unstructured (similar to the latent basis model). Thus, in this approach the number of latent variables and the individual patterns of change are unknown, which is similar to how exploratory factor analysis models are fit to cross sectional multivariate data. This approach can yield an appropriate number of latent variables as well as potential forms of change that may be suitable for the data. Additional exploratory approaches are based upon principal component analysis (Davison, 2008; Tucker, 1958, 1966), multidimensional scaling (Ding, Davison, & Petersen, 2005), and smoothing spline models (Guo, 2002).

Interindividual Variability in Intraindividual Change

In latent growth curve models, the intraindividual change pattern or the functional form of change is invariant over participants. For example, if a linear model is chosen as the intraindividual change function, then all individuals are expected to follow a linear change pattern. Interindividual variability in intraindividual change, within the latent growth modeling framework, enters in the parameters, which describe the individual change pattern. That is, even though individuals follow the same functional form (e.g., linear), the parameters of the functional form (e.g., intercept and slope) are allowed to vary over individuals. Allowing the parameters of the functional form to vary over individuals provides for a wide variety of potential developmental patterns, especially with a functional form with multiple parameters (e.g., cubic and spline models).

Theoretically, any parameter of the intraindividual change function can vary over individuals (e.g., intercept and slope of linear model are allowed to vary over individuals); however, there are situations where theory or a lack of information from the data leads certain parameters to be invariant over individuals such that interindividual variability is not manifested. One of these situations was evident when studying intraindividual changes in reading ability (Grimm, Ram, & Estabrook, 2010). The chosen intraindividual change model was a Gompertz model. The Gompertz model has four parameters: a lower asymptote, an upper asymptote, the location of the inflection point, and a rate of change from the lower to the upper asymptote. For reading ability, the lower asymptote is the predicted reading ability when time approached negative infinity. Theoretically, it is logical that interindividual differences in reading ability at this point in time were minimal or nonexistent. Thus, this parameter was not allowed to vary over individuals. Ideally, theoretical notions of the

developmental change process should drive and inform the nature and location of interindividual variability in intraindividual change; however, again we recognize the limitations of the data that are brought to the analysis.

Determinants of Intraindividual Change

As mentioned earlier, one of the rationales for collecting longitudinal data proposed by Baltes and Nesselroade (1979) is the identification of determinants of intraindividual change. The latent growth curve model accommodates this rationale by allowing for interindividual difference variables (e.g., gender and intervention type) to predict the parameters of the growth curve, which describe interindividual variability in intraindividual change. Ideally, these associations will indicate the degree to which the interindividual difference variables are associated with the rate of intraindividual change; however, this is not necessarily the case with complex models that have multiple parameters impacting intraindividual change (Grimm, Castro-Schilo, & Davoudzadeh, 2013). Finally, we note that the identification of *causes* of interindividual differences in intraindividual change can only be identified with appropriate controls, such as random assignment.

THE LATENT GROWTH MODELING

Historical Development

Early work in the modeling and understanding of intraindividual change was very descriptive before the rise of fitting mathematical models to individual-level data. In this approach, linear and a variety of complex nonlinear models were considered and the researchers had multiple goals. A first goal was to minimize the size of the residuals. Models with smaller residuals better represented to data and were seen as superior. A second goal was to have a mathematical function with parameters that had meaning in relation to the intraindividual change process under consideration. These, sometimes contrasting, goals are evident in the study of physical height where certain mathematical models were specified to capture key aspects of development, such as the timing of puberty (e.g., Preece & Baines, 1978), whereas other models were developed to simply map onto the observed data with little attention paid to the interpretability of parameters (e.g., reference). This phase along the developmental continuum, is often referred to as *individual growth modeling* (Willett & Sayer, 1994), continued for a number of years with few modifications (e.g., Berkey, 1982), and remains a useful approach to studying intraindividual change. Furthermore, interindividual variability in intraindividual change could be examined by relating parameters of the mathematical function to between-person variables (e.g., Wishart, 1938).

During the *individual growth modeling* phase, alternative approaches to understanding intraindividual change surfaced. For example, Tucker (1958, 1966) fit principal component analysis models to the matrix of sums of squares and cross-products to decompose the matrix into *generalized learning curves* and individual weights. The *generalized learning curves* represent dominant trends within the data and the individual weights represented the extent to which an individual's trajectory was impacted by each *generalized learning curve*. Tucker used the term *generalized learning curves* due to his work with learning data; however, this approach can be applied to a variety of types of data and is commonly referred to as *Tuckerized curves* (see Grimm, Steele, et al., 2013; Meredith & Tisak, 1984).

Rao (1958) described several approaches for understanding intraindividual change and interindividual variability in intraindividual change. First, Rao (1958) discussed calculating an average rate of change—in essence decomposing the repeated measures into an initial state and a rate of change. In this approach, he also discussed transformations of the time scale to make changes more or less constant (linear change). Rao (1958) also discussed the possibility of using factor analytic methods, specified the growth curve model, and utilized principal components analysis of the uncorrected sum of squares and products matrix to estimate a model. This last routine is equivalent to Tucker's (1958) *generalized learning curves* and was developed independently at the same time.

In 1984, Meredith and Tisak presented their paper, *Tuckerizing curves*, at the International Meeting of the Psychometric Society. In the presentation, Meredith and Tisak described the *latent curve model* and how the model can be specified within the SEM framework. They discussed multiple types of latent growth models including the free curve or latent basis model as well as a negative exponential model and this work was published in 1990 (Meredith & Tisak, 1990). During this time, McArdle expanded on Meredith and Tisak's work by exploiting the flexibility of the structural equation modeling framework (McArdle & McDonald, 1984) to specify combinations of latent growth curves and behavior genetic models (McArdle, 1986), multivariate latent growth models (McArdle, 1988), and multiple group latent growth models (McArdle & Hamagami, 1996). Additionally, Browne and du Toit (1991) and Browne (1993) highlighted how the SEM framework could be used to approximate growth models that followed inherently nonlinear mathematical functions.

At a similar time, Laird and Ware (1982) published a paper in biometrics on random-effects models for longitudinal data describing models for growth and for repeated measures. An early article that reached psychologists was written by Bryk and Raudenbush (1987), who published a *Psychological Bulletin* article on the use of hierarchical linear model (i.e., multilevel model, mixed-effects model, and random coefficient model) to assess change. This spawned the use of this framework for studying growth within the psychological and educational disciplines. The work that followed was focused on how the mixed-effects modeling framework could be used to study inherently nonlinear processes. This work appeared in books by Davidian and Giltinan (1995), Littell et al. (1996), and Pinheiro and Bates (2000). Additional work by Singer (1998) and Willett and Sayer (1994) was prominent in the development of latent growth models and spread the use of the multilevel approach to studying change.

By the late 1990s, it was clear that multilevel models for assessing change and structural equation models for assessing change were equivalent in many cases (Curran, 2003; McArdle & Hamagami, 1996), especially for the most commonly fit models (e.g., polynomial models). Papers followed describing the various ways to fit latent growth models (e.g., Ferrer, Hamagami, & McArdle, 2004) and the relative advantages and disadvantages of each approach (Ghisletta & Lindenberger, 2004). At first, the multilevel model had advantages related to individually spaced measurements and the flexibility of fitting inherently nonlinear models, whereas the structural equation modeling approach had advantages related to indices of model fit, flexibility in the residual structure, and the inclusion of measurement models. Several of these relative advantages have been reduced because each framework has increased in flexibility. Furthermore, general modeling programs (e.g., WinBUGS) have further blurred the differences between the multilevel and structural equation modeling approaches for understanding change.

In the late 1990s and early 2000s, there were two additional important expansions of the general latent curve modeling framework. The first development was the introduction of the growth mixture model by Muthén and Shedden (1999). The growth mixture model allowed for an exploration of growth trajectories and for an additional way to incorporate between-person differences in growth. The growth mixture model combined the finite mixture model and the latent growth model and allowed researchers to cluster individuals based upon their individual growth trajectories. Thus, between-person differences in growth were allowed to manifest themselves by different classes and between-person differences within each class. Additionally, we note a similar development appeared in the latent class trajectory model, which was introduced by Nagin (1999). The difference between the growth mixture model and the latent class trajectory model was that between-person differences were allowed for within each latent class in the growth mixture model but not in the latent class trajectory model.

The second development was the introduction of the latent difference or latent change score modeling framework by McArdle and Hamagami (2001; see also McArdle, 2001). The latent change score model allowed for dynamic associations between multiple outcomes while simultaneously modeling growth trajectories. This model placed an emphasis on time-sequential within-person change and led to subsequent developments in differential models (see Boker, Neale, & Rausch, 2004) and latent acceleration models (Hamagami & McArdle, 2007).

More recently there have been developments that combine several of the models discussed above. For example, Grimm and Ram (2009b) proposed the second-order growth mixture model—a combination of the second-order growth model (McArdle, 1988) and the growth mixture model (Muthén & Shedden, 1999). Grimm, Zhang, Hamagami, and Mazzocco (2013) combined the latent difference score model (McArdle & Hamagami, 2001) with the estimation of inherently nonlinear growth models within the SEM framework (Browne & du Toit, 1991), which allowed for an emphasis on within-person change when modeling nonlinear growth trajectories. Grimm (2006) combined the latent difference score model with the finite mixture model, and Grimm, Ram, and Estabrook (2010) presented a combination of the finite mixture model and the estimation of inherently nonlinear growth models within the SEM framework to examine clustering of nonlinear growth trajectories. Finally, Zhang, McArdle, and Nesselroade (2011) presented growth rate models—rotations of growth models where a latent variable representing the rate of change at a given time point is included in the model to highlight the individual rate of change in nonlinear growth models.

Latent Growth Curve as a Structural Equation Model

Within the SEM framework, the latent growth curve is fit as a restricted common factor model. The growth factors (e.g., intercept and linear slope in a linear growth model) of the growth model are latent variables within the SEM framework. In the specification that follows and all model specifications included in this chapter, we use Greek letters for parameters that are fixed or estimated (e.g., factor

loading matrix) and Latin letters for entities that are not estimated nor fixed (e.g., factor scores).

The general latent growth model can be expressed as

$$\mathbf{Y}_i = \Lambda \mathbf{h}_i + \mathbf{u}_i \tag{22.1}$$

where $\mathbf{Y}_i$ is a $T \times 1$ vector of the repeatedly measured observed scores for individual i;, where T represents the number of repeated assessments based on the chosen time metric; Λ is a $T \times R$ factor loading matrix, which serves to define the growth factors of the growth curve (e.g., intercept and slope); R is the number of growth factors; $\mathbf{h}_i$ is an $R \times 1$ vector of latent factor scores (e.g., intercept and slope scores) for individual i; and $\mathbf{u}_i$ is a $T \times 1$ vector of unique scores for individual i.

The latent factor scores can be written as deviations from the sample-level mean written as

$$\mathbf{h}_i = \boldsymbol{\alpha} + \mathbf{d}_i \tag{22.2}$$

where $\boldsymbol{\alpha}$ is an $R \times 1$ vector of latent variable means, and $\mathbf{d}_i$ is an $R \times 1$ vector of mean deviations for individual i. The covariance matrix of mean deviations is $\boldsymbol{\psi}$, an $R \times R$ matrix indicating the magnitude of between-person differences in the latent growth factors as well as the degree of linear association among the latent growth factors.

The unique scores have expected means equal to 0 (i.e., $E(\mathbf{u}_i) = 0$) and covariance matrix $\boldsymbol{\Theta}$ (i.e., $\text{COV}(\mathbf{u}_i) = \boldsymbol{\Theta}$), where is a $T \times T$ matrix. Often, only the diagonal elements of are estimated, which forces the unique scores to be time-specific. However, additional types of structures of can be examined, such as first-order autoregressive and block-diagonal (see Kwok, West, & Green, 2007). Additionally, many researchers force the diagonal elements to be equal across time (i.e., $\boldsymbol{\Theta} = \mathbf{I}\theta$, where is a $T \times T$ identity matrix and is a scalar).

The latent growth model places a set of expectations on the repeated measures and it is important to understand these expectations to evaluate model fit and misfit. Additionally, understanding model expectations can guide the use of model modifications. The model expectations of observed variables based upon the latent growth model is

$$\boldsymbol{\mu} = \Lambda \boldsymbol{\alpha}$$
$$\sum = \boldsymbol{\Lambda} \boldsymbol{\psi} \Lambda' + \boldsymbol{\Theta} \tag{22.3}$$

where is an $T \times 1$ vector of expected means and is a $T \times T$ expected covariance matrix. The expected mean structure for the latent growth model is simply the factor loading matrix multiplied by the mean vector of the latent variables, and the expected covariance structure follows the common factor analysis model of the latent variable covariance matrix pre and post multiplied by the factor loading matrix plus the residual or unique covariance matrix.

A path diagram of a latent growth curve model with two latent variables is given in Figure 22.2. In such diagrams, squares represent observed variables, circles represent latent variables, and the triangle denotes the constant. One-headed arrows represent directional relationships, such as regression paths and two-headed arrows represent nondirectional or symmetric relations, such as covariances. The labels of the paths in the path diagram are derived from the previous specification. With the path diagram in Figure 22.2 and the path tracing rules derived by Wright (1921), model expectations can be calculated, which replicate the matrix algebra given already.

Common Latent Growth Models

To further show how the SEM framework can be used to specify latent growth models, we present a series of specific latent growth models that are commonly considered in psychology and psychopathology. In each description, we present specifications of the required matrices. In all specifications, we assume there are four equally spaced measurement occasions ($T = 4$).

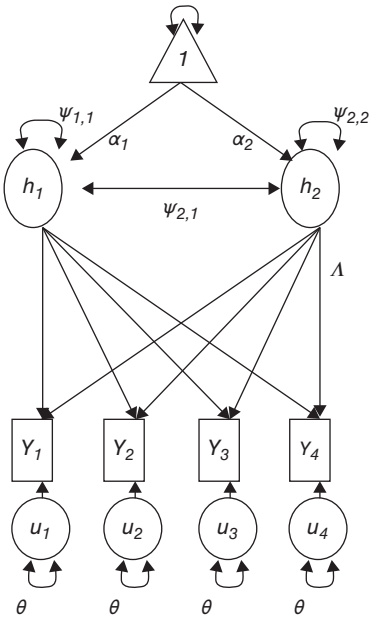


Figure 22.2 A latent growth curve model.

Linear Change

The linear change model is the most commonly fit growth curve in applied research. The linear change model has two latent variables ($R = 2$): an intercept, often centered at the first measurement, and a linear slope representing the individual constant rate of change over the observation period. The intercept and linear slope are allowed to vary over persons and allowed to covary with each other. Conceptually, the linear growth model is similar to fitting a linear regression model to each individual's data, retaining individual parameters for the intercept and slope, and summarizing those estimated parameters to calculate their means to get a sense of the average growth trajectories as well as their variances and covariance to understand between-person differences in the growth parameters and their degree of linear association. The linear growth model is specified as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & \frac{1-k_1}{k_2} \\ 1 & \frac{2-k_1}{k_2} \\ 1 & \frac{3-k_1}{k_2} \\ 1 & \frac{4-k_1}{k_2} \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \tag{22.4}$$

where the repeated measures for individual i is a product of the factor loading matrix multiplied by the factor scores for individual i, plus the unique scores for individual i. In the factor loading matrix, the first column contains fixed values set equal to 1 to define the intercept factor, h_1, and the second column will contain fixed values set equal to change linearly with time to define the linear slope, h_2. The constants are chosen to center the intercept and scale the slope. Often, is set to 1 to center the intercept at the first occasion and is set to 1 to scale the slope in terms of the unit of time used in the selected time metric.

Quadratic Change

The quadratic change model is commonly considered when curvature is evident in the growth pattern and when the linear model does not adequately capture the intraindividual change pattern. The quadratic growth model is specified as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & 0 & 0 \\ 1 & 1 & 1/2 \\ 1 & 2 & 2 \\ 1 & 3 & 9/2 \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \\ h_3 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \tag{22.5}$$

The quadratic model has three latent variables, an intercept, h_1, a linear slope, h_2, and a quadratic slope, h_3. The factor loadings for the intercept are all fixed at 1, the factor loadings for the linear slope change linearly with time, and the factor loadings for the quadratic slope are simply the square of the factor loadings for the linear slope divided by 2 (this is done to scale the quadratic slope in terms of acceleration). We note the same potential to center the intercept and scale the linear and quadratic slope. In this example, the centering and scaling constants, and k_2, are both set equal to 1. With this centering, the intercept represents the predicted value of the outcome at the first occasion; the linear slope represents the individual rate of change at the first occasion; and the quadratic slope represents acceleration, curvature, or how quickly the rate of change is changing, which is constant across time.

The quadratic model is often considered; however, the parameters of the quadratic model can be difficult to map onto theoretical notions of change (Cudeck & du Toit, 2002) and difficult to interpret in isolation because both the linear and quadratic slopes impact the intraindividual rate of change (see Grimm, Castro-Schilo, & Davoudzadeh, 2013). To combat these issues, Cudeck and du Toit (2002) reparameterized the quadratic model to emphasize the time when the maximum (or minimum) value is reached and the maximum (or minimum) value. These parameters are easily interpretable, highlighting interesting aspects of quadratic curve. Unfortunately, Cudeck and du Toit's (2002) approach is rarely considered when fitting the quadratic growth model in applied settings.

Transformations of Time

Polynomial transformations of time, such as in the quadratic model described above, are common in the application of latent growth models but are not the only viable transformations of time. For example, square root or log transformations of time may yield useful results concerning the growth of the construct under study. In essence, the growth process may be linear with respect to a different time scale. If this alternative time scale is utilized, then the process would conform to a linear growth model (see also Rao, 1958). As an example, we present the square root growth model, which can be specified as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 1 & 1 \\ 1 & \sqrt{2} \\ 1 & \sqrt{3} \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \tag{22.6}$$

where the first column of the factor loading matrix defines the intercept and the second column defined the linear slope in square root time. Additionally, if the second column of the factor loading matrix was divided by $\sqrt{3}$, then the slope can be discussed as the amount of change the has occurred from the first to the last measurement occasion; however, the shape of intraindividual change would still follow square root time.

Latent Basis or Unstructured Change

The latent basis or unstructured change model is a very flexible model that borders on exploratory change because the pattern of change is derived from the data. That is, instead of imposing a structure on change, such as linear or quadratic, within the factor loading matrix, the factor loading matrix has elements that are freely estimated. The latent basis model can be written as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 1 & \lambda_{2,2} \\ 1 & \lambda_{3,2} \\ 1 & 1 \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \tag{22.7}$$

The first column of the factor loading matrix is set equal to 1 to define the intercept latent variable. The second column, on the other hand, has two fixed elements and two estimated elements. The first element is set equal to 0, the second and third are estimated from the data, and the last element is set equal to 1. This pattern of loadings define, what most call, a *shape* factor because the estimated factor loadings define the structure or individual change pattern and this pattern of change can vary greatly in shape. The two fixed factor loadings are needed for identification; however, their values and locations within the second column are arbitrary. This configuration centers the intercept at the first measurement occasion and scales the *shape* factor in terms of change from the first to the last occasion. Additionally, the estimated factor loadings represent the proportion of total change that has occurred since the first measurement occasion. Such interpretations are straightforward when change is monotonic; however, the latent basis model can structure change that is not monotonic (e.g., rise and fall). Even though the interpretation of the estimated factor loadings remain, changing the identification constraints to emphasize change to the maximum (or minimum) point can make interpretation more straightforward (see Ram & Grimm, 2007). Finally, we note that the latent basis model has been considered a model that optimizes the rescaling of time due to the estimated factor loadings.

Inherently Nonlinear Change Patterns

An additional collection of latent growth models follows patterns of change that are inherently nonlinear. In such models, a parameter is contained within the function of time—usually in an exponent or raised to an exponent. That is, within the factor loading matrix a nonlinear function is specified where an estimated parameter is contained within that function. Such models have several benefits because of the wide variety of shapes of development that can be modeled and the flexibility for modeling between-person differences in the shape of development (Burchinal & Appelbaum, 1991; Grimm, Ram, & Hamagami, 2011). Two such inherently nonlinear models are the power model and the exponential growth model. The power model can be expressed as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & \beta^0 & -1 \\ 1 & \beta^1 & -1 \\ 1 & \beta^2 & -1 \\ 1 & \beta^3 & -1 \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \tag{22.8}$$

where the factor loading matrix has a series of ones in the first column to define the intercept and the second column defines a power function where an estimated parameter β is raised to the power of time (0, 1, 2, 3) and then one is subtracted from this value. The variety of developmental patterns is determined by the estimated value of β. The exponential model can be written as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & 1 - \exp(-\beta \cdot 0) \\ 1 & 1 - \exp(-\beta \cdot 1) \\ 1 & 1 - \exp(-\beta \cdot 2) \\ 1 & 1 - \exp(-\beta \cdot 3) \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \tag{22.9}$$

where the second column of the factor loading matrix now changes as a function of $1 - \exp(-\beta \cdot t)$, where β is an estimated parameter and t is the value to time based upon the chosen time scale. In the power model and the exponential model, we note that the factor loadings are not fixed values as in the linear, quadratic, or square root models but are also not freely estimated as in the latent basis model. Instead, the values are structured to change according to a specific function.

The utility and flexibility of inherently nonlinear models is shown when allowing the parameters within the factor loading matrix to vary over individuals. This type of model is not directly estimable within the SEM framework; however, this type of model can be approximated and in many

cases the approximation is very good (see Browne, 1993; Browne & du Toit, 1991; Grimm, Ram, & Hamagami, 2011, for details on this approximation).

Multiphase Change

Multiphase change models (see Cudeck & Klebe, 2002) are a type of latent growth model where the repeated measures data are compartmentalized and change is more or less modeled within discrete phases of time. These models also go by the name spline or piecewise growth models. Change within each phase can be modeled with any of the above mentioned growth models; however, relatively simple models, such as a linear or quadratic change model, are often fit within each phase. For example, a bilinear model can be specified as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & 0 & 0 \\ 1 & 1 & 0 \\ 1 & 1 & 1 \\ 1 & 1 & 2 \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \\ h_3 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \tag{22.10}$$

where the first column of the factor loading matrix defines the intercept, which is centered at the first measurement occasion; the second column defines the first linear phase, which represents intraindividual change during the first two measurement occasions; and the third column defines the second linear phase, which represents intraindividual change from the second to fourth measurement occasions. These multiphase models can accommodate a variety of nonlinear change patterns and tend to have parameters that are easily interpretable.

MULTILEVEL MODELING FOR STUDYING DEVELOPMENTAL TRAJECTORIES

The multilevel model for studying developmental trajectories was presented by Laird and Ware (1982) and Bryk and Raudenbush (1987). Growth models can be fit in the multilevel modeling framework as a two-level model because the repeated measurements are nested within individuals. There are several ways to present the multilevel model—we present two common approaches, highlight how the specifications are equivalent, and how the specifications can map onto the structural equation modeling approach for latent growth analyses.

Following Laird and Ware (1982), the multilevel model for change can be specified as

$$\mathbf{Y}_i = \mathbf{X}_i\boldsymbol{\alpha} + \mathbf{Z}_i\mathbf{d}_i + \mathbf{e}_i \tag{22.11}$$

where $\mathbf{Y}_i$ is a $n_i \times 1$ vector of observed scores for individual i, $\mathbf{X}_i$ (chi) is a known $n_i \times R$ design matrix linking $\boldsymbol{\alpha}$, an $R \times 1$ of unknown fixed effects estimates, to $\mathbf{Y}_i$, $\mathbf{Z}_i$ (zeta) is a known $n_i \times K$ design matrix linking $\mathbf{d}_i$, a $K \times 1$ vector of unknown individual effects, to $\mathbf{Y}_i$, and $\mathbf{e}_i$ is an $n_i \times 1$ vector of residuals. The individual effects, $\mathbf{d}_i$, are assumed to be multivariate normal with means equal to 0 and covariance matrix $\boldsymbol{\psi}$ (i.e., $\mathbf{d}_i \sim MVN(0, \boldsymbol{\psi})$). The residuals are assumed to be normally distributed with mean 0 and covariance matrix $\boldsymbol{\Theta}_i$ (i.e., $\mathbf{e}_i \sim N(0, \boldsymbol{\Theta}_i)$). We note that the dimensions of $\boldsymbol{\Theta}_i$ are dependent on i, but the unknown parameters do not depend on i and $\boldsymbol{\Theta}_i = \theta\mathbf{I}$, where $\mathbf{I}$ is an identity matrix, and θ is a common residual variance. This model yields a mean vector of $\mathbf{Y}_i$ equal to $\mathbf{X}_i\boldsymbol{\alpha}$ and covariance matrix $\mathbf{Z}_i\boldsymbol{\psi}\mathbf{Z}_i' + \boldsymbol{\Theta}_i$.

The matrix specification of the multilevel growth model (equation 22.11) follows closely the specification within the SEM framework (equation 22.1). Of note, in the growth model without covariates and traditionally specified, the two design matrices, $\mathbf{X}_i$ and $\mathbf{Z}_i$, are equivalent and are individual functions of time. With these matrices being equivalent, the model more closely mimics the structural equation modeling specification with a separation of the mean vector for the latent variables (i.e., $\boldsymbol{\alpha}$) from the individual deviations (i.e., $\mathbf{d}_i$).

Noting these similarities, we now highlight the main differences. The first difference is the dimension of several matrices. In the multilevel modeling framework, the dimensions of several matrices vary over individuals. The vector of observed scores, the design matrices, and the residual vector have dimensions that vary at the individual level, whereas the comparable matrices within the structural equation modeling framework have dimensions that do not vary over the individual level and have dimensions based on the number of occasions from the chosen time metric.

The next major difference is that the design matrices within the multilevel modeling framework ($\mathbf{X}_i$ and $\mathbf{Z}_i$) vary at the individual level (subscripted by i), whereas the factor loading matrix from the structural equation modeling framework (Λ) does not vary at the individual level. Thus, the multilevel model is able to accommodate individually varying measurement schedules, whereas the structural equation modeling framework is for a fixed measurement schedule that all individuals follow. However, we note two things. First, although the measurement schedule is fixed, all participants do not need to be measured at each of the fixed measurement occasions. Second, recent advances within the SEM framework have allowed for the factor loading matrix to be individually specified

(essentially turning the structural equation model into a multilevel model).

The third major difference between the frameworks is the ways in which the latent variables or random coefficients combine within the model. The structural equation modeling framework is a linear modeling framework, which means that the latent variables can only combine in a linear or additive fashion (i.e., latent variables multiply the factor loading matrix and are then added together to yield predicted values). In the multilevel modeling framework, the random coefficients can also combine in a nonlinear or multiplicative fashion. Therefore, there are models that can be fit within the multilevel, or more specifically the nonlinear multilevel, modeling framework that cannot be fit within the structural equation modeling framework.

Alternative Specification

An alternative way to present the multilevel model is

$$Y_{it} = f(time_{it}, \mathbf{h}_i) + e_{it} \tag{22.12}$$

where Y_{it} is the observed response for the i-th individual at time t, $f(time_{it}, \mathbf{b}_i)$ is a linear or nonlinear function of time with random coefficients, $\mathbf{h}_i' = (h_{i1}, h_{i2}, \ldots, h_{iR})$, that combine linearly or nonlinearly, and e_{it} is the residual for individual i at time t, which is assumed to follow a normal distribution with constant variance (i.e., $e_{it} \sim N(0, \theta)$). The random coefficients are often assumed to follow a multivariate normal distribution, such that $\mathbf{h}_i \sim MVN(\boldsymbol{\alpha}, \boldsymbol{\psi})$, where $\boldsymbol{\alpha}$ is an $R \times 1$ vector of fixed-effects parameters and $\boldsymbol{\psi}$ is a $R \times R$ covariance matrix of random-effects parameters.

Specific Growth Models

Linear Growth

The linear growth model is written as

$$Y_{it} = h_{i1} + h_{i2} \cdot \left(\frac{time_{it} - k_1}{k_2} \right) + e_{it} \tag{22.13}$$

where Y_{it} are the repeated measures for individual i at time t, h_{i1} is the intercept or predicted score when $time_{it} = k_1$ for individual i, k_1 is a constant used to center the intercept, h_{i2} is the linear slope or the predicted amount of change in Y_{it} for a one unit change in $\frac{time_{it}}{k_2}$ for individual i , k_2 is a scaling coefficient used to adjust the metric of $time_{it}$ and the interpretation of h_{i2}, and e_{it} is a time-specific residual score for individual i at $time_{it}$.

The constants, k_1 and k_2, can be set to any value; however, the values for these constants impact interpretation and should be carefully considered. For example, to center the intercept at the zero point of the original time metric, $k_1 = 0$. Thus, the intercept, h_{i1}, would reflect an individual's true level at the attribute when $time_{it} = 0$. This may be reasonable for many time scales, but is inappropriate for other time scales and constructs. For example, when examining individual changes in depression as a function of age during adolescence, it might be reasonable to center the chosen time metric, *age*, around the age when measurements were first taken (e.g., $k_1 = 13$ years). In a similar vein, the choice of k_2 should be carefully considered to scale the time metric in an appropriate way. We note that k_2 is often set equal to 1 to scale the slope, h_{2i}, in terms of the original time metric, $time_{it}$; however, for certain constructs that change more rapidly or more slowly, it is reasonable to choose a different value of k_2.

Quadratic Growth

The quadratic model can be specified as

$$Y_{it} = h_{i1} + h_{i2} \cdot \left(\frac{time_{it} - k_1}{k_2} \right) + h_{i3} \cdot \left(\frac{time_{it} - k_1}{2k_2} \right)^2 + e_{it} \tag{22.14}$$

where h_{i1} is the intercept for individual i, h_{i2} is the linear slope for individual i, h_{i3} is the quadratic slope for individual i, and e_{it} is the time-specific residual score for individual i at $time_{it}$. Similar to the linear growth curve, the intercept remains the predicted score when $time_{it} = k_1$. The linear slope represents the rate of change when $time_{it} = k_1$ and the quadratic slope represents acceleration or curvature.

Inherently Nonlinear Models

One of the benefits of the multilevel modeling framework is the straightforward extension to the *nonlinear* multilevel modeling framework. That is, the multilevel model can be used to fit any type of nonlinear change model. This flexibility is important because development is often highly nonlinear and linearity often serves as an approximation for a truly nonlinear process. In this section, we present two nonlinear models, both of which follow an exponential trajectory, that vary in complexity.

The first exponential model can be written as

$$Y_{it} = h_{i1} + h_{i2} \cdot (1 - \exp(-\beta \cdot time_{it})) + e_{it} \tag{22.15}$$

where h_{i1} is the predicted score when $time_{it} = 0$ for individual i, h_{i2} is the total amount of change from h_{i1} to the asymptotic level for individual i, β is the rate of approach to the asymptotic level, and e_{it} is the time-dependent residual. In this model, β is an estimated (fixed effect) parameter and does not vary across participants, whereas h_{i1} and h_{i2} are random coefficients that vary over participants. This form of the exponential is *additive* because the random coefficients enter the model in a linear/additive fashion because β is an estimated parameter. We note that this model can be fit within the SEM framework (see equation 22.9) as long as nonlinear constraints are available, and the majority of structural equation modeling programs do allow for such constraints.

The second exponential model is written as

$$Y_{it} = h_{i1} + h_{i2} \cdot (1 - \exp(-h_{i3} \cdot time_{it})) + e_{it} \qquad (22.16)$$

where h_{i1} is the predicted score when $time_{it} = 0$ for individual i, h_{i2} is the total amount of change from h_{i1} to the asymptotic level for individual i, h_{i3} is the rate of approach to the asymptotic level for individual i, and e_{it} is the time-dependent residual. Now, the rate of approach varies over individuals. This simple change makes the model multiplicative because h_{i2} is a multiplier of h_{i3} and therefore, these random coefficients enter the model in a nonlinear fashion. Thus, the structural equation modeling framework is unable to estimate this model (see Browne, 1993); however, the multilevel model can estimate the model directly—a big advantage of the multilevel modeling framework.

Direct Expansions of the Latent Growth Modeling Framework

The specifications described are focused on the study of intraindividual change and interindividual variability in intraindividual change. A common next step involves the evaluation of determinants of interindividual variability in intraindividual change. As noted already, this is often accomplished with the inclusion of time-invariant covariates as predictors of the latent growth variables. Moving back to the linear growth model for simplicity, this model can be specified as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 1 & 1 \\ 1 & 2 \\ 1 & 3 \end{pmatrix} \left[\begin{pmatrix} \alpha_1 \\ \alpha_2 \end{pmatrix} + \begin{pmatrix} \gamma_1 \\ \gamma_2 \end{pmatrix} \cdot (X) + \begin{pmatrix} d_1 \\ d_2 \end{pmatrix} \right] + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \qquad (22.17)$$

where α_1 and α_2 is the predicted value of h_1 and h_2 when the covariate X is 0, γ_1 and γ_2 are regression coefficients of the impact of X on h_1 and h_2, and d_1 and d_2 are latent variable disturbances representing unmodeled variability in h_1 and h_2.

Additionally, time-varying covariates are modeled as having an instantaneous impact on the observed score. Such a model can be expressed as

$$\begin{pmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 1 & 1 \\ 1 & 2 \\ 1 & 3 \end{pmatrix} \begin{pmatrix} h_1 \\ h_2 \end{pmatrix} + \begin{pmatrix} \gamma_1 & 0 & 0 & 0 \\ 0 & \gamma_2 & 0 & 0 \\ 0 & 0 & \gamma_3 & 0 \\ 0 & 0 & 0 & \gamma_4 \end{pmatrix} \begin{pmatrix} X_1 \\ X_2 \\ X_3 \\ X_4 \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} \qquad (22.18)$$

where X_1 through X_4 is a time-varying covariate—a single variable that varies across time—and γ_1 through γ_4 are the regression coefficients for the time-varying covariate describing the impact the time-varying covariate has on the outcome variable. Often these effects are constrained to be equal across time (e.g., $\gamma_1 = \gamma_2 = \gamma_3 = \gamma_4$) to mimic the way the model is typically fit within the multilevel modeling framework; however this constraint is not required.

Multiple Group Growth Model

The latent growth model of equation 22.1 can be extended to accommodate multiple observed groups of individuals. In this approach, the data are split based upon the grouping variable and separate growth models are specified for each group. Typically, a series of models are evaluated, which test the invariance of specific collections of parameters, such as the latent variable means, the latent variable covariances, and the structure of change. Extending equation 22.1, we can write the multiple group growth model as

$$\mathbf{Y}_i^{(g)} = \Lambda^{(g)} \mathbf{h}_i^{(g)} + \mathbf{u}_i^{(g)} \qquad (22.19)$$

where $\mathbf{Y}_i^{(g)}$ is a $T \times 1$ vector of the repeatedly measured observed scores for individual i in group g, $\Lambda^{(g)}$ is a $T \times R$ factor loading matrix for group g, $\mathbf{h}_i^{(g)}$ is an $R \times 1$ vector of latent factor scores for individual i in group g, and $\mathbf{u}_i^{(g)}$ is a $T \times 1$ vector of unique scores for individual i in group g.

The individual factor scores are written as deviations from the *group-level* means, expressed as

$$\mathbf{h}_i^{(g)} = \boldsymbol{\alpha}^{(g)} + \mathbf{d}_i^{(g)} \qquad (22.20)$$

where $\boldsymbol{\alpha}^{(g)}$ is an $R \times 1$ vector of latent variable means for group g, and $\mathbf{d}_i^{(g)}$ is an R x 1 vector of mean deviations

for individual i in group g. In the multiple group growth model, the covariance matrix of mean deviations is $\boldsymbol{\psi}^{(g)}$, a group-specific R x R covariance matrix indicates the magnitude of between-person differences in the latent growth factors within each group as well as the degree of linear association among the latent growth factors within each group. Finally, the covariance matrix for unique scores, $\boldsymbol{\Theta}^{(g)}$, is also group specific.

This specification of the multiple group model is equivalent to separately specifying a growth model for each of the $g = 1$ to G groups. In practice, a series of models are fit to evaluate where group differences manifest. There are a variety of approaches to systematically test for group differences, and here we follow the approach advanced by Ram and Grimm (2009). In this approach, the first model is the *invariant model* where all parameter estimates are constrained to be equal over group (i.e., $\Lambda^{(g)} = \Lambda$, $\boldsymbol{\alpha}^{(g)} = \boldsymbol{\alpha}$, $\boldsymbol{\psi}^{(g)} = \boldsymbol{\psi}$, and $\boldsymbol{\Theta}^{(g)} = \boldsymbol{\Theta}$). This model is equivalent to the latent growth model without accounting for any grouping variable. The second model is the *Means Model* where the latent variable means are the only parameter estimates allowed to vary over group (i.e., $\Lambda^{(g)} = \Lambda$, $\boldsymbol{\alpha}^{(g)}$, $\boldsymbol{\psi}^{(g)} = \boldsymbol{\psi}$, and $\boldsymbol{\Theta}^{(g)} = \boldsymbol{\Theta}$). Next, is the *Means+Variances Model* where the latent variable means, the latent variable variances, and the residual variances are allowed to vary over group (i.e., $\Lambda^{(g)} = \Lambda$, $\boldsymbol{\alpha}^{(g)}$, $\boldsymbol{\psi}^{(g)}$ and $\boldsymbol{\Theta}^{(g)}$). The fourth and final model is the *configural invariance model* (i.e., *curves model*), where the factor loading matrix is also allowed to vary over group (i.e., $\Lambda^{(g)}$, $\boldsymbol{\alpha}^{(g)}$, $\boldsymbol{\psi}^{(g)}$ and $\boldsymbol{\Theta}^{(g)}$). In this model, all parameter estimates are separately estimated for each group. Nested chi-square difference tests can be used to compare each model with the more restricted models to determine whether and where the groups differ in estimated parameters.

Latent Growth Modeling Studies for Understanding Developmental Pathways in Terms of Continuity–Discontinuity and Equifinality–Multifinality

Understanding Developmental Pathways Using Latent Growth Modeling

In this section, we illustrate several studies that demonstrate how growth curve models can be used to inform substantive questions in developmental psychopathology research focusing on the core principles of developmental psychopathology. A latent growth modeling study by Kim and Cicchetti (2006) demonstrates how growth curve models can be used to describe differential pathways to psychopathology. This study used latent growth modeling to investigate longitudinal relationships between self-system processes and depressive symptoms among maltreated and nonmaltreated children. One of the main goals was to describe developmental trajectories in self-system processes and depressive symptoms during the elementary school years among maltreated and nonmaltreated children. From a methodological viewpoint, an important advance of this study was to implement a latent growth curve model using SEM to examine interindividual differences in intraindividual variability (McArdle & Bell, 2000) in both initial levels and rates of change in children's self-system processes and depressive symptoms. The participants included 251 children (142 maltreated and 109 nonmaltreated) who attended a summer day camp research program. Children ranged in age from 6 to 11 years ($M = 8.46$, $SD = 1.11$) at Wave 1 and 64% of the children were boys. The four-wave longitudinal data involved 7- to 12-year-olds.

To investigate developmental trajectories of self-system processes and depressive symptoms, we conducted growth curve analyses with a maximum likelihood estimation method, which allows for inclusion of respondents with missing data by using full information maximum likelihood (FIML) estimation (Arbuckle, 1996). In evaluating the overall goodness-of-fit of each model, the root mean square error of approximation (RMSEA; Browne & Cudeck, 1993) index assesses the degree of lack of fit for a model and values less than .05 and .08 are taken to reflect a close fit and a reasonable fit, respectively. The comparative fit index (CFI; Bentler, 1990) varies along a 0–1 continuum in which values greater than .90 and .95 are considered to reflect acceptable and excellent fits to the data, respectively.

We first fit a two-factor growth model over the four time points. The first latent factor was the intercept (or level) representing the initial starting point of the growth function, and the four factor loadings for the latent intercept factor were fixed to one. The second latent factor was the slope of the growth function, representing the rate of change in the growth trajectory over time. The latent intercept and slope factors were freely correlated. To determine the shape of trajectories of study variables for the entire sample, three alternative models were fitted separately for depressive symptoms (measured by the Children's Depression Inventory, CDI; Kovacs, 1985) and self-esteem (measured by the Self Esteem Inventory, SEI; Coopersmith, 1981). The first model was a *no-growth* model that assumed no slope component (i.e., thus assumed no growth or change). The second model was a *linear growth* model that assumed a linear pattern of change over time and fixed values of slope parameters as

[$B(T1) = 0,\ B(T2) = 1,\ B(T3) = 2,\ B(T4) = 3$]. The third model was a *latent basis growth* model that estimated slope parameters from the data to yield an overall group shape. The slope parameter for the first occasion was fixed to zero [$B(T1) = 0$] to allow a separation of the intercept and slope components, and the slope parameter for the last occasion was fixed to one [$B(T4) = 1$] to provide a scale of measurement for the slopes. The slope parameters for the second and the third occasions were freely estimated from the data. Model comparison was facilitated by positing a nested ordering of models in which the parameter estimates for a more restrictive model were a proper subset of those in a more general model.

As can be seen in Table 22.1, the chi-square difference test indicated that a linear growth model provided the best fit to the data for the SEI. For the SEI, significant variance of both intercept ($\sigma^2 = .01,\ SE = .002,\ p < .05$) and slope ($\sigma^2 = .001,\ SE = .000,\ p < .05$) indicated the presence of significant individual differences in initial levels and change in children's self-esteem. Both intercept mean ($M = .68,\ SE = .01,\ p < .05$) and slope mean ($M = .03,\ SE = .004,\ p < .05$) were significantly different from zero, showing that children's self-esteem increased over time. In contrast, a latent basis growth model was the best-fitting model for the CDI. The coefficients of the slope factor loadings represented the percentage of growth relative to the total change occurring over all time points. The pattern of CDI slope factor loadings (i.e., .00, .59, .89, 1.00) reflected a nonlinear change in the CDI scores.

Significant variance existed in both intercept ($\sigma^2 = 38.45,\ SE = 9.42,\ p < .05$) and slope ($\sigma^2 = 25.55,\ SE = 10.93,\ p < .05$), reflecting meaningful individual variability in average level and change in depressive symptoms over time. The intercept mean was significantly different from zero ($M = 9.47,\ SE = .48,\ p < .05$), and the slope mean was negative and significantly different from zero ($M = -3.55,\ SE = .51,\ p < .05$), suggesting that children's depressive symptoms decreased over time. The intercept and the slope factors were negatively correlated ($r = -.74,\ p < .05$), suggesting that children who reported higher initial levels of depressive symptoms tended to show a more rapid decrease in depressive symptoms over time compared to children who reported lower initial levels of depressive symptoms. Therefore, the data indicated a significant decrease in depressive symptoms and linear increases in self-esteem over the four time points. There are no particular theories to delineate particular developmental trajectory patterns of depressive symptoms and self-systems among maltreated and nonmaltreated school-age children. However, this finding was consistent with some previous research that reported decreases in self-reported internalizing problems and increases in perceptions of internal control in similar samples (e.g., Bolger & Patterson, 2001).

In estimating developmental trajectories using latent growth modeling, the main goal is to estimate unobserved or latent trajectory as a function of the set of repeated measures that were observed to describe how the developmental process unfolds within each person—that is, intraindividual change. As shown in this example, the growth curve model can further inform the interindividual changes (the means and variances of the individual trajectory parameters *across* individuals) based on intraindividual trajectories. Within latent growth modeling, each individual is characterized by his or her own unique trajectory that best represents their observed data over multiple time points. The advantage of latent growth modeling is clear especially when we compare latent growth modeling to more traditional models (such as autoregressive regression models) in which a single parameter estimate represents the relation between two repeated measures pooled over all subjects over time (Curran & Willoughby, 2003).

TABLE 22.1 Comparisons of Fitted Growth Curve Models for Self-Esteem and Depressive Symptoms of Maltreated and Nonmaltreated Children[1]

Model Label[2]	χ^2	*df*	*p*(exact)	CFI	RMSEA	*p*(close)	$\Delta\chi^2$	Δdf	*p*(d)
Self-Esteem (SEI)									
1-a. No-growth model	70.70	8	.00	.78	.18	.00			
1-b. Linear growth model	3.52	5	.62	1.00	.00	.85	67.18	3	<.05
1-c. Latent growth model	.76	3	.86	1.00	.00	.94	2.76	2	*ns*
Depressive Symptoms (CDI)									
1-a. No-growth model	73.35	8	.00	.67	.18	.00			
1-b. Linear growth model	12.34	5	.03	.96	.07	.17	61.01	3	<.05
1-c. Latent growth model	5.17	3	.16	1.00	.05	.38	7.17	2	<.05

[1]Sample size is 251 for CDI and SEI data. *p*(exact) = probability of an exact fit to the data; CFI = comparative fit index; RMSEA = root mean square error of approximation; *p*(close) = probability of a close fit to the data.
[2]$\Delta\chi^2$ = difference in likelihood ratio tests; Δdf = difference in *df;* *p*(*d*) = probability of the difference tests.

Studying Turning Points in Developmental Pathways Using Latent Growth Modeling

Hussong and her colleagues (2008) demonstrated a great example of using latent growth modeling for testing turning points that mark the life course of young adults as they moved from risk to resilience or maladjustment. They focused on the concept of snares that defined a turning point away from an expected course of adaptation. Within the context of youth antisocial behavior, the snares referred to the mechanisms responsible for prolonging what might otherwise be a developmentally normative pattern of desisting antisocial behavior. They tested two alternative hypotheses. The snares hypothesis proposed that substance abuse (i.e., the ensnaring factor) would act through a series of proximal influences on crime desistance, consequently making short-term alterations in the trajectory of antisocial behavior. In contrast, the launch hypothesis proposed that substance abuse early in young adulthood may not only identify young men who are on a long-term trajectory of elevated antisocial behavior but also set them on such a trajectory. These two alternative hypotheses were compared to a general deviance hypothesis, in which both substance abuse and antisocial behavior were viewed as multiple indicators of a single latent construct of problem behavior. Specifically, for the snares hypothesis, substance abuse was tested as a time-varying predictor of elevated antisocial behavior against an expected pattern of desistance over time. For the launch hypothesis, substance abuse was tested as a time-invariant predictor of intraindividual differences in the intercepts and slopes that defined trajectories of antisocial behavior over time.

The data were taken from the Dunedin Multidisciplinary Health and Development Study, a longitudinal investigation of a complete birth cohort born in Dunedin, New Zealand (Silva & Stanton, 1996). This study used self-reported substance (alcohol and marijuana) abuse and antisocial behaviors data gathered from men at ages 18, 21, and 26 (N = 461 at age 18 and 438 had complete data). They first estimated an unconditional linear growth curve model to examine whether the individual trajectories of antisocial behaviors varied across men. The unconditional growth curve model had a good fit [$Ø^2(1) = 9.31$, $p = .002$, $CFI = .98$] with a significantly decreasing slope over time ($t = -5.84$, $p < .001$). The variance estimates were both significant for the intercept ($t = 8.96$, $p < .001$) and the slope ($t = 2.69$, $p < .01$), indicating substantial interindividual variability in intraindividual trajectories of antisocial behavior.

Next, they tested the snares hypothesis by fitting a time-varying covariate growth curve model in which the repeated measures of substance abuse were included as time-varying covariates. In this time-varying covariate growth curve model, time-specific influences of alcohol abuse and marijuana abuse on antisocial behavior were estimated above and beyond the influence of each individual's underlying trajectory of antisocial behavior. The time-varying covariate growth curve model fit the data well [$\chi^2(1) = 10.59$, $p = .001$, CFI = .99]. Results indicated that, at age 18 and age 21, men with higher levels of alcohol and marijuana abuse reported significantly elevated levels of antisocial behavior than would be expected based on their individual trajectories alone. At age 26, the effect of alcohol abuse was marginally significant ($p = .07$) and the effect of marijuana was not significant ($p > .10$). This finding was interpreted that substance abuse appeared to ensnare those young men within elevated patterns of antisocial behavior and such an effect became weaker as those men aged through this period of crime desistance.

They further tested the launch hypothesis using a conditional growth curve model in which alcohol abuse and marijuana abuse at age 18 were included as two exogenous predictors of changes in antisocial behaviors. The conditional growth curve model fit the data well [$\chi^2(3) = 11.33$, $p = .01$, $CFI = .99$]. Higher levels of alcohol and marijuana abuse at age 18 predicted higher intercepts of the antisocial behavior trajectories ($t = 9.63$, $p < .001$ for alcohol and $t = 8.81$, $p < .001$ for marijuana). Both alcohol ($t = -2.86$, $p < .001$) and marijuana ($t = -2.86$, $p < .001$) abuse were also negatively related to the slope of the antisocial behavior trajectories. This finding suggested that men with higher substance abuse at age 18 showed steeper decreases in antisocial behavior over time, yet they were significantly elevated in antisocial behavior across all periods of assessment.

Finally, a general deviance hypothesis was tested using a second-order growth curve model in which the two second-order intercept and slope factors for deviance proneness were constructed based on three latent constructs representing deviance proneness at ages 18, 21, and 26. For each first-order latent factor, alcohol abuse, marijuana abuse, and antisocial behavior served as multiple indicators (e.g., the age 18 deviance proneness latent factor was constructed based on alcohol abuse, marijuana abuse, and antisocial behavior at age 18). In examining whether there were significant individual differences in the developmental trajectories of deviance proneness over time, factor loadings within the same construct needed to be constrained to be equal over time. The results revealed

a relatively poor fit, especially based on the chi-square test [$\chi^2(2) = 98.06$, $p < .001$, CFI = .95]. In particular, the source of the poor fit was the equality constraints on the factor loadings within the same construct over time. This finding indicates that the manifestations of the general deviance factor were variable over time. This problem reflects, in part, the challenge of determining continuity in the quality of adaptation across developmental time despite changing behavioral presentations of the developing individual (Sroufe, 1997).

Overall, this study illustrates how the growth curve model can offer a sophisticated approach to study changes over time and examine turning points in intraindividual development. Specifically, the analyses supported both the launch and the snares hypotheses, providing significant insights into the direction of causality in which substance abuse contributed to maintenance of antisocial behavior.

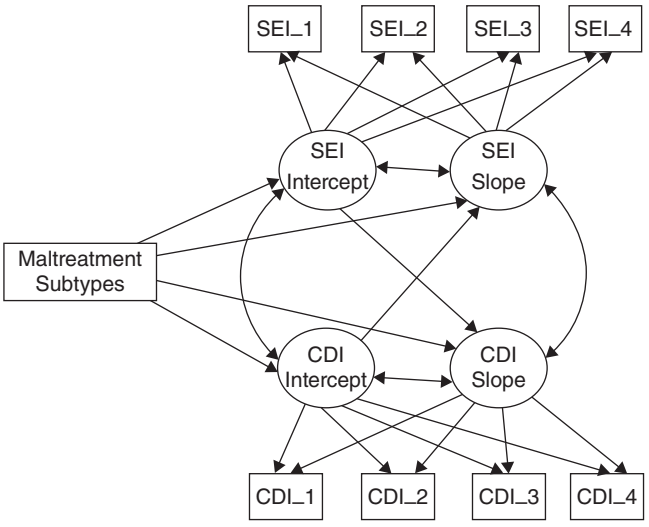


Figure 22.3 Multivariate growth model of self-esteem and depressive symptoms with maltreatment subtypes as predictors.

Understanding Risk and Protective Factors Using Latent Growth Modeling

In the following section, an empirical example is given of a study using latent growth modeling to investigate the effects of risk or protective factors on developmental trajectories leading to maladjustment. In the latent growth modeling study by Kim and Cicchetti (2006) that was reviewed earlier, the impact of different maltreatment subtypes on children's self-esteem and depressive symptoms was examined. As shown in Figure 22.3, both the intercept and slope factors of self-esteem and depressive symptoms were regressed on age (a demographic covariate) and the dichotomous maltreatment variables (presence or absence of emotional maltreatment, physical neglect, physical abuse, and sexual abuse). Different subtypes of maltreatment were assessed using the Maltreatment Classification System (MCS; Barnett, Manly, & Cicchetti, 1993). Of key interest was whether the strength of maltreatment's impact on self-esteem and depressive symptoms, and the relationships between self-esteem growth factors and depression growth factors would vary as a function of gender. Therefore, we used a two-group growth curve model with gender as the grouping variable: boys ($n = 159$) and girls ($n = 92$). For Model 2 in Table 22.2, we first fit a configural invariance model in which all parameters were freely estimated across the two groups, and then imposed equality constraints hierarchically to test the adequacy of the constraints using nested chi-square difference tests (Bollen, 1989).

We tested the equal maltreatment effects model by imposing equality constraints on all regression paths for the effects of four maltreatment subtypes and age on growth factors of self-esteem and depressive symptoms. These equality constraints did not degrade the model fit, suggesting that the effects of maltreatment and age on the self-esteem and depression growth factors were comparable between boys and girls. Examination of parameter estimates in the Equal Maltreatment Effects model indicated that for both boys and girls, physical

TABLE 22.2 Comparisons of Fitted Growth Curve Models for Self-Esteem and Depressive Symptoms of Maltreated and Nonmaltreated Children[1]

Model label[2]	χ^2	*df*	*p*(exact)	CFI	RMSEA	*p*(close)	$\Delta\chi^2$	Δdf	*p*(d)
Two-group multivariate growth analyses of CDI and SEI by gender									
3-a. Configural invariance	112.31	80	.01	.97	.04	.82			
3-b. Equal CDI slope	115.28	82	.01	.97	.04	.82	2.98	2	*ns*
3-c. Equal maltreatment effects	130.20	102	.03	.97	.03	.96	14.92	20	*ns*
3-d. Equal growth relations	156.85	112	.00	.96	.04	.87	26.65	10	<.05

[1]Sample size is 251 for CDI and SEI data. *p*(exact) = probability of an exact fit to the data; CFI = comparative fit index; RMSEA = root mean square error of approximation; *p*(close) = probability of a close fit to the data.
[2]$\Delta\chi^2$ = difference in likelihood ratio tests; Δdf = difference in *df*; *p*(*d*) = probability of the difference tests.
Note: Based in part on "Longitudinal trajectories of self-system processes and depressive symptoms among maltreated and nonmaltreated children" by J. Kim and D. Cicchetti, 2006, *Child Development*, *77*, pp. 624–639.

abuse was related to lower initial levels of self-esteem ($B = -.06$, $SE = .02$, $p < .05$) and higher initial levels of depressive symptoms ($B = 3.63$, $SE = 1.23$, $p < .05$). Physical neglect was associated with higher initial levels of depressive symptoms ($B = 2.62$, $SE = 1.03$, $p < .05$). Regardless of gender, emotional maltreatment was positively related to the initial levels of self-esteem ($B = .05$, $SE = .02$, $p < .05$) and negatively related to change in self-esteem ($B = -.03$, $SE = .01$, $p < .05$). Emotional maltreatment was also significantly predictive of initial levels of depression ($B = -3.12$, $SE = 1.11$, $p < .05$) as well as change in depression ($B = 2.69$, $SE = 1.13$, $p < .05$). Additionally, age was positively related to the initial levels of self-esteem ($B = .04$, $SE = .01$, $p < .05$) and negatively related to the initial levels of depressive symptoms ($B = -1.55$, $SE = .39$, $p < .05$), suggesting that older children reported higher self-esteem and lower depressive symptoms compared to younger children.

Thus, the results of multivariate latent growth modeling analysis with covariates indicated that, regardless of gender, physical abuse was negatively related to initial levels of self-esteem, and physical abuse and physical neglect were positively associated with initial levels of depressive symptoms. The findings regarding the impact of emotion maltreatment suggested complexity. For depressive symptoms, emotional maltreatment was negatively related to initial levels but predicted slower decreases. For self-esteem, emotional maltreatment was positively related to initial levels but predicted slower increases over time. Taken together, these findings illustrate how latent growth modeling can contribute to enhancing our understanding of the developmental processes whereby early risk factors are linked to changes in adaptation and maladjustment in later life.

Understanding Mediating Processes Using Latent Growth Modeling

Using a longitudinal sample of 381 children (who were followed between ages 9 and 11 years), Troop-Gordon and Ladd (2005) studied whether social perceptions (self- and peer beliefs) acted as mediating mechanisms through which peer victimization was related to psychological maladjustment. Data were collected at five time points, including spring fourth grade, fall fifth grade, spring fifth grade, fall sixth grade, and spring sixth grade. Peer victimization was assessed using peer nominations, social self-acceptance was measured using the Harter's Self-Perception Profile for Children (1985), and children's perceptions of the social dispositions of their schoolmates were measured using the Peer Belief Inventory (Rabiner, Keane, & MacKinnon-Lewis, 1993). Children's maladjustment outcomes were measured using self, teacher, and parent reports of internalizing problems and peer, teacher, and parent reports of externalizing problems through various measures including the Child Behavior Checklist (CBCL, Achenbach, 1991) and the Teacher Report Form (TRF; Achenbach, 1991).

The multivariate growth curve model was tested separately for internalizing and externalizing problems. In each model, regression paths from the intercept factor of peer victimization to the intercept factors of both self-acceptance and peer beliefs as well as regression paths from the slope factor of peer victimization to the slope factors of both self-acceptance and peer beliefs were estimated. Within each construct, a regression path from the intercept factor to slope factor was estimated (e.g., the effect of peer victimization intercept on peer victimization slope was estimated). The latent constructs representing developmental changes in internalizing and externalizing problems were constructed based on multiple informants data of residualized scores that were computed by regressing spring sixth grade scores on spring fourth grade scores (e.g., the latent factor for changes in internalizing problems were constructed based on self-, parent-, and teacher-reported residualized scores).

For internalizing problems, the hypothesized model yielded an acceptable fit [$\chi^2(301) = 451.45$, $p < .001$, CFI = .95, RMSEA = .05]. Higher initial levels of peer victimization were predictive of more negative initial levels of self-acceptance ($\beta = -.55$ for boys and –.44 for girls) and peer beliefs ($\beta = -.32$ for boys and –.24 for girls). Moreover, an increasing peer victimization slope was related to declining self-acceptance ($\beta = -.25$ for boys and –.21 for girls). An increasing peer victimization slope was also related to decreasing prosocial presentation of peers for boys ($\beta = -.21$), whereas it was not associated with changes in peer beliefs among girls ($\beta = .04$). In turn, more derogatory initial self-acceptance ($\beta = -.70$ for boys and –.67 for girls) and peer beliefs ($\beta = -.25$ for boys and –.25 for girls) were predictive of increases in internalizing problems. Similarly, more negative growth in self-acceptance ($\beta = -.45$ for boys and –.45 for girls) and peer beliefs ($\beta = -.30$ for boys and –.30 for girls) was predictive of increases in internalizing problems. The indirect effects of initial peer victimization on subsequent change in internalizing problems through initial self-acceptance ($Z = 5.54$, $p < .001$) and through initial peer belief ($Z = 2.06$, $p < .05$) were statistically significant. The indirect effects of changes in peer victimization (i.e., peer victimization slope) through changes in self-acceptance

were significant ($Z = 2.57$, $p < .01$), whereas the indirect effects of changes in peer victimization through changes in peer beliefs were not significant.

Next, for externalizing problems, the final model that provided an acceptable fit [$\chi^2(301) = 467.31$, $p < .001$, CFI = .96, RMSEA = .05] revealed that the paths from intercept and slope factors of self-acceptance were not significantly different from zero. As expected, more derogatory initial peer beliefs ($\beta = -.23$ for boys and –.22 for girls) were predictive of increases in externalizing problems and more negative growth in peer beliefs ($\beta = -.23$ for boys and –.22 for girls) was predictive of increases in externalizing problems. The indirect effects of initial peer victimization on subsequent change in externalizing problems through initial peer belief were significant ($Z = 2.55$, $p < .01$), whereas the indirect effects of changes in peer victimization through changes in peer beliefs were not significant.

The findings of this study suggest that social perceptions construed in middle childhood played as mediating processes through which interpersonal risks, such as peer victimization established in the early elementary school years, are associated with increasingly deteriorating adjustment trajectories. This study illustrates how latent growth modeling can be used to specify developmental processes that are involved in the emergence and maintenance of psychopathology as earlier levels and developing trajectories of risk factors exert negative influences on more proximal psychosocial factors for the psychopathology outcomes.

Understanding Moderating Processes Using Latent Growth Modeling

Latent growth modeling can be used to examine not only additive but also interactive contributions of risk and protective factors to variance in the developmental trajectories of psychopathology outcomes. An *additive effect* model postulates that the risk or protective factor increases or decreases the probability of a maladaptive outcome independently of any probabilistic predictive effect of other risk or protective factors. An *interactive effect* model supposes that the link between the risk or protective factor and probability of maladaptive outcome differs (or is moderated) by the level of another risk or protective factor. For example, using a sample of 1,668 adolescents, Wills and his colleagues examined prediction about relationships of coping dimensions to substance use over the period from 12 to 15 years of age (Wills, Sandy, Yaeger, Cleary, & Shinar, 2001). They used a multivariate growth curve model to investigate whether engagement and disengagement coping variables measured at study outset may predict initial levels and growth in peer and adolescent substance use over time. Behavioral coping (i.e., do something to solve the problem) was used as the index of engagement and anger coping (i.e., get mad at people), helpless coping (i.e., give up on the problem), and hangout coping (i.e., hang out with other kids) were used as indices of disengagement. Peer and adolescent substance use were measured with items that asked about the typical frequency of cigarette, alcohol, and marijuana use. Additionally, life stress was assessed using a 20-item checklist of negative life events (Newcomb & Harlow, 1986).

They estimated latent growth factors (i.e., intercept and slope) of peer and adolescent substance use based on observed data measured in seventh, eighth, and ninth grades. In the latent growth modeling, covariances were estimated between the intercepts for peer and adolescent substance use, between the slopes for peer and adolescent substance use, and between the intercept and the slope factor for both peer and adolescent substance use. A regression path from peer substance use intercept to adolescence substance use slope was estimated, but a nonsignificant regression path from adolescence substance use intercept to peer substance use slope was not included. The final model yielded an acceptable fit [$\chi^2(48) = 132.04$, $p < .001$, RMSEA = .03], and revealed that engagement and disengagement coping operated through somewhat different pathways. Specifically, behavioral coping was related to lower intercepts of adolescent substance use and decreasing slopes of peer substance use over time. Disengagement was associated with higher intercepts for both peer and adolescent substance use and increasing slopes of adolescent substance use over time.

To determine whether effects of coping were contingent on the level of life stress, they performed multiple-group latent growth modeling analysis with two subgroups defined by a median split on life events. Chi-square difference tests indicated that the regression paths from coping to intercepts were significantly greater in the high-stress group [$\Delta\chi^2(3) = 10.07$, $p < .025$] than in the low-stress group. The regression paths from coping to slopes were also significantly greater in the high-stress group [$\Delta\chi^2(2) = 5.50$, $p = .05$]. In fact, the effect of behavioral coping on the slope of peer substance use and the effect of disengagement coping on the slope of adolescent substance use were significant in the high-stress group, but they were not significant in the low-stress group. However, the path from peer substance use intercept to adolescent substance use slope did not differ between the two groups [$\Delta\chi^2(1) = 0.21$, *ns*]. Therefore, the findings highlighted that behavioral coping had greater protective

effects on the development of peer substance use and disengagement coping had greater risk-promoting effects on the development of adolescent substance use at a higher level of life stress compared to a lower level of life stress. Finally, an interaction between risk and protective factors was tested in a multiple group growth curve model using subgroups of high vs. low behavior coping groups. The results revealed the buffering effects of behavioral coping indicated by significantly weaker effects of disengagement coping on the growth factors of substance use in the high behavioral coping group compared to the low behavioral coping group [$\Delta\chi^2(3) = 10.87,\ p < .025$].

This study illustrates testing of moderating effects of risk or protective factors using latent growth modeling. As we presented the conceptual reviews earlier, it appears that behavioral coping operated as a protective-enhancing process because it allowed adolescents to interact with stress in such a way that their behavioral engagement was more beneficial to reduce associating with substance using peers. There also was evidence of the risk factors of disengagement coping operating as the vulnerable and reactive processes where the overall disadvantage linked with the risk factors (i.e., disengagement coping) was heightened with increasing levels of stress. The findings regarding the moderation effect of behavioral coping reducing the potential adverse effects of disengagement types of coping further illuminate that the impact of stress on maladjustment depends on the interaction between protective and risk factors.

With the longitudinal design, the potential moderators of interest can vary over time thus creating the need for quantifying the time-varying moderator variables. Two approaches have been suggested by Flora, Khoo, and Chassin (2006). In the first approach, a potential moderator grouping variable can be estimated as a discrete latent class variable from a univariate growth mixture model (Muthén & Muthén, 2000). For example, suppose that we want to test whether changes in parental warmth over time moderate the links between changes in temperament-based behaviors and maladaptive outcomes. Using growth mixture modeling, we might find three classes for trajectories of parental warmth (stable, increasing, decreasing), and this categorical variable, based on the estimated probabilities, would then serve as a grouping/moderator variable when examining the predictive paths between temperament-based behaviors and the maladaptive outcome of interest. In the second approach, a variable representing each interaction term between the moderator variable and the predictor variable at each time point can be added to the existing growth curve model. Using the same hypothetical study, the interaction term between parental warmth and temperament-based behavior at each time point would be added to the growth curve model in the prediction of the maladaptive outcome. These two approaches to examining time-varying moderation effects may converge or may lead to distinct conclusions about whether and how the moderator in question is operating. However, we think that both approaches are appropriate depending on the specific developmental questions in hand. Time-varying moderation (or interaction) effects are complex, and a variety of statistical models are required to understand how they seem to be operating.

Latent Growth Modeling Studies for Understanding Contextual Influences

Latent Growth Modeling for Cross-Domain Relations in Risk and Psychopathology

Grimm (2007) provided illustrations of studying multivariate longitudinal relations between children's academic achievement and depression using a latent growth modeling framework. The data were drawn from a subsample from the National Longitudinal Survey of Youth (NLSY) (Baker, Keck, Mott, & Quinlan, 1993), a nationally representative study initiated in 1979 by the U.S. Department of Labor to study labor market behavior and experiences. The children of female respondents were assessed biennially from 1986 to 2000. The subsample contained $N = 7,078$ children who were measured at least once between the ages of 8 and 14 ($n = 3,602$ boys) with diverse ethnic backgrounds. Longitudinal data involved four repeated measurements assessed at ages 8, 10, 12, and 14. Academic achievement was measured using the Peabody Picture Vocabulary Test (PPVT)-Revised Form L (Dunn & Dunn, 1981). Raw PPVT-R scores were transformed to age 8 *t*-scores based on the mean and the standard deviation at age 8. Depression was assessed using the Behavior Problems Index (BPI; Zill & Peterson, 1986). The five anxious/depressed scores were summed and used to indicate the child's level of depression and these anxious/depressed scores were transformed into age 8 *t* scores in the same manner as the PPVT-R scores.

First, a bivariate growth curve model was used to examine the following questions regarding longitudinal relations between academic achievement and depression from age 8 to 14: (1) Is the level of academic achievement related to the level of depression? (2) Is the amount of change in academic achievement related to the amount of change in depression? (3) Is the level of academic achievement related to the

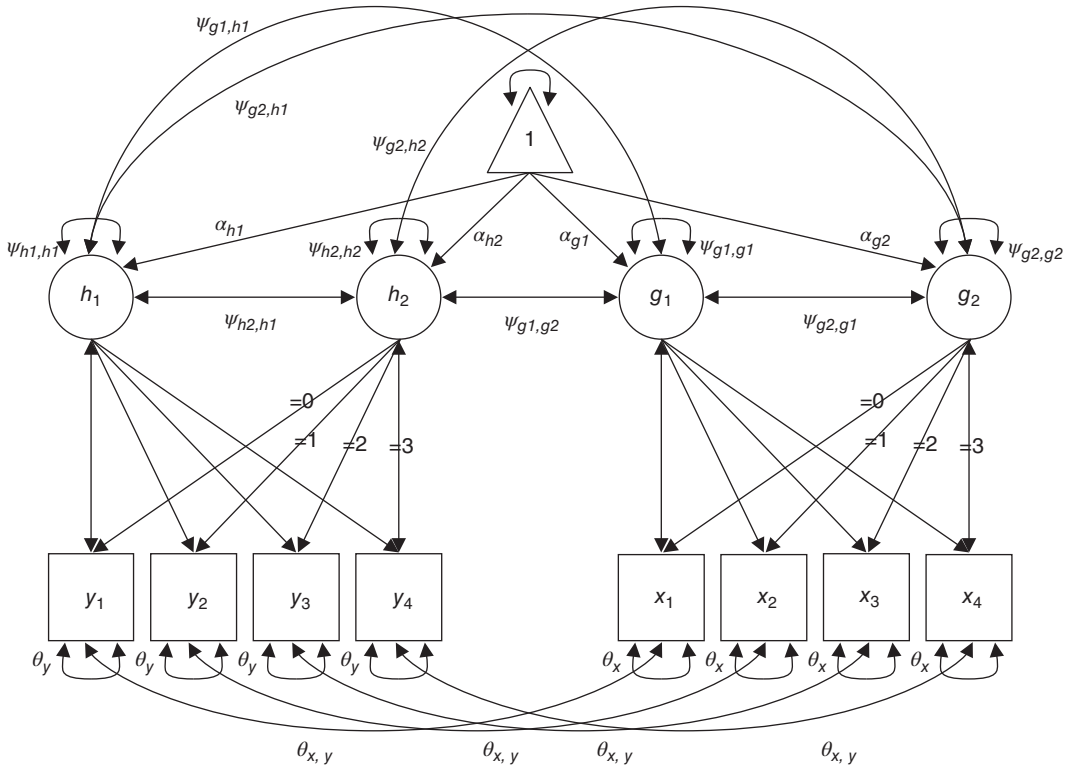


Figure 22.4 A bivariate growth curve model.

amount of change in depression? (4) Is the level of depression related to the amount of change in academic achievement? In the bivariate growth curve model (Figure 22.4), it is assumed that the two constructs are each governed by a separate developmental process and that the two processes are interrelated at the level of the latent growth factors (but not at the level of manifest variables).

Univariate growth curve models were estimated to determine the shape of developmental trajectories of academic achievement and depression. The latent basis model (in which the coefficients were estimated with the exception of two that were fixed for identification) yielded the best fits for both academic achievement and depression, compared to the intercept only and the linear growth models. Depression did not show significant systematic change, whereas academic achievement showed systematic and rapid increases from age 8 to age 14. The bivariate growth curve model fit the data well [$\chi^2(91) = 220$, RMSEA = .01]. In this bivariate growth curve model, there were two significant factor correlations ($p < .05$). First, the correlation between the intercept and the slope of depression was –.18 indicating that students with a lower level of depression at age 8 were likely to have a more positive change in depression from age 8 to 14. Second, the correlation between the intercept of depression and the intercept of academic achievement was –.14, indicating that students with an overall lower level of depression at age 8 were likely to have higher level of academic achievement at age 8.

Grimm (2007) further investigated an alternative growth curve model using a time-varying covariate. In the time-varying covariate growth curve model, the developmental processes of academic achievement were considered primary, whereas the developmental processes of depression were seen as control variables. As shown in Figure 22.5, the latent growth factors of academic achievement were estimated and the repeated measurements of academic achievement were regressed on the repeated measurements of depression. The latent growth factors of academic achievement were allowed to covary with the repeated measurements of depression. The following set of questions was asked: (1) How are the time-varying scores of depression related to the time-varying scores of achievement while controlling for interindividual differences in intraindividual change in academic achievement? (2) How are the time-dependent scores of depression related to the level and slope of the growth curve for academic achievement? (3) How does academic achievement change over time when controlling for depression over time?

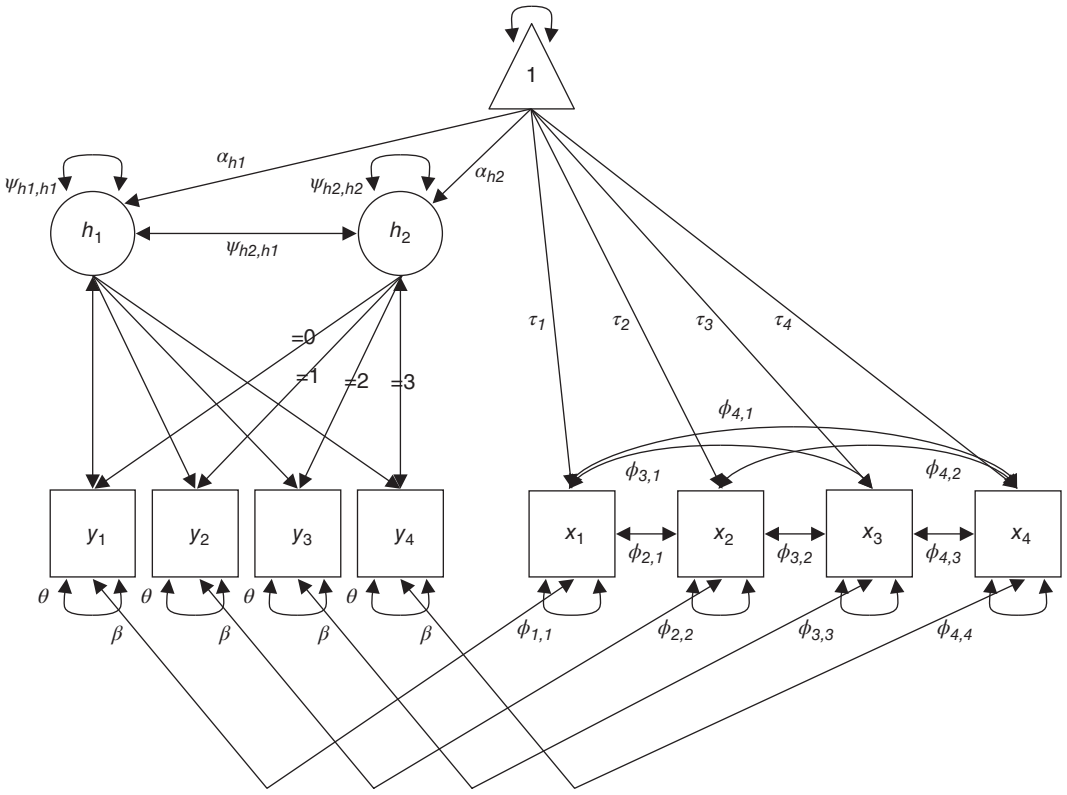


Figure 22.5 A time-varying covariate growth curve model.

The time-varying covariate growth curve model fit the data well [$\chi^2(57) = 109.04$, RMSEA = .01]. The growth factors of academic achievement were negatively correlated with the repeated depression scores ($r = -.02$ to $-.17$). More importantly, however, depression was not significantly related to academic achievement after controlling for changes in academic achievement from age 8 to 14 ($-.01$, $t = .82$, $p = ns$). Similar to the bivariate growth curve model, the average trajectory of academic achievement indicated a positive growth with a decelerating rate of change.

Taken together, the results from the bivariate growth curve model and the time-varying covariate growth curve model appear to offer somewhat different descriptions regarding the developmental relations between academic achievement and depression. The conclusions based on those results provide accurate answers to different developmental questions. These examples of using different types of latent growth modeling for examining longitudinal relations between developmental processes suggest that developmental psychopathologists should use the appropriate quantitative modeling approaches to accurately capture the dynamic nature of development in multiple levels or domains.

Latent Curve Modeling for Multiple Levels of Analysis

Over the past several decades, there has been a growing acknowledgment for the necessity of multidisciplinary and interdisciplinary investigations within the field of developmental psychopathology. Investigations of developmental pathways require researchers to consider biological, psychological, and environmental-contextual processes that result in diverse outcomes of psychopathology (i.e., multifinality) as well as those processes from which varied pathways to resilience might eventuate (i.e., equifinality). Research using multiple levels of analysis suggests that the influence of levels on one another is often bidirectional. That is, biological factors can influence psychological processes to produce psychopathology outcomes and psychosocial experiences can also modify the structure, function, and organization of biological mechanisms including the brain and gene expression (Cicchetti, 2010, 2015).

The following study illustrates how latent growth modeling can be used in the study of developmental psychopathology using multiple levels of analysis. In a recent longitudinal study of African American emerging adults ($N = 399$), Brody and his colleagues investigated

how life stress and genetic status were prospectively related with increases in drug use (Brody et al., 2012). The participants provided three waves of data at ages 17, 19, and 20 years regarding life stress using a checklist of 12 negative life events, peer and romantic partner drug use, vulnerability cognitions (a combination of willingness and intention to use drugs) and their drug use rates (0 = no use and 1 = any use). In addition, a saliva sample from the dopamine receptor D4 (DRD4) gene was genotyped.

It was hypothesized that emerging adults who carry the 7 + R allele would display higher rates of drug use over time when they experienced greater life stress (i.e., first-generation gene × environment or G×E interaction). It was also hypothesized that the first-generation G×E interaction would evince increases in affiliation with drug-using companions (peer and romantic partner drug use) and vulnerability cognitions for drug use, which both serve as two proximal mediators for drug use escalation. Latent growth modeling embedded in a multilevel modeling framework was used.

First, unconditional latent growth modeling analyses were estimated. The results indicated a significant and positive growth of youths' drug use ($\beta = 5.94$, $p < .001$) and companions' drug use ($\beta = 5.47$, $p < .001$) from age 17 to 20 years. There was no significant change in the slope of vulnerability cognitions ($\beta = .19$, $p = .64$). Next, DRD4, life stress, DRD4 × life stress interaction as well as two covariates (intervention status and youth gender) were regressed on the rate of change of drug use, companions' drug use, and vulnerability cognitions. Multilevel logistic regression was used and coefficients were calculated in log odds metric. There were significant interaction effects between DRD4 and life stress on the slopes of youths' drug use ($b = 1.82$, $SE = .92$, $p < .05$), companions' drug use ($b = 2.41$, $SE = .91$, $p < .01$), and vulnerability cognitions ($b = 1.28$, $SE = .58$, $p < .05$).

In the final step, time-varying effects of the two hypothesized mediators (companions' drug use, and vulnerability cognitions) on youths' drug use. Within-person variability in companions' drug use ($\beta = .13$, $p < .01$) and vulnerability cognitions ($\beta = .59$, $p < .001$) significantly predicted the probability of youths' drug use. More specifically, one unit change in companions' drug use over time increased the odds of past month drug use by the youth by 14%, and one unit change in vulnerability cognitions over time increased the odds of youths' drug use by 80%. After the time-varying effects of the two mediators were accounted for in the model, the DRD4 × life stress interaction effects on the slope of youths' drug use was no longer significant.

The findings of this study can be interpreted from the perspective of resilience. That is, the results suggest that genetic status can contribute to resilience by showing that carrying the 6-R allele (as opposed to the 7 + R allele) buffered emerging adults from escalating drug use when they experienced high levels of life stress. Furthermore, tests of the mediated moderation hypothesis revealed two pathways through which the interaction effects between DRD4 and life stress exerted: increases in affiliations with drug-using companions and increases in vulnerability cognitions. As the authors suggested, information regarding such mediating processes is critical for identifying targets for genetically informed prevention programs. This study exemplifies benefits of obtaining a process-level understanding of psychopathology through simultaneous assessment of multiple domains of variables both within and outside of the developing person.

Latent Growth Modeling for Cross-Population Comparisons in Risk and Psychopathology

As mentioned earlier in this chapter, a developmental psychopathology perspective emphasizes the mutual interplay between the study of normality and psychopathology (Cicchetti, 2010). The following investigation illustrates using latent growth modeling for studying interface between normal and abnormal development. Lansford and her colleagues (2006) examined how protective and risk factors alter trajectories of externalizing and internalizing behaviors from kindergarten through Grade 8. The participants included 585 children who were drawn from a multisite community sample and 11.8% of these children had been physically abused during the first 5 years of life. A set of protective and vulnerability factors was selected either because they had been found in previous research to be significantly related to children's externalizing and internalizing behaviors or because of a strong conceptual rationale for their moderating effects of physical abuse on externalizing and internalizing behavior trajectories. Two variables were used as indicators of child characteristics: early social competence and early hostile attribution. Several variables were used as indicators of the factors external to child including early socioeconomic status (SES), adolescent SES, early stress, adolescent stress, neighborhood danger, early proactive parenting, adolescent harsh discipline, unilateral parental decision making (i.e., the degree to which the parent made decisions for the adolescent concerning daily activities), parent-reported parental monitoring, and child-reported parental knowledge (i.e., knowing about various aspects of their children's life including activities and friends). Children's externalizing and internalizing

behaviors were measured based on teachers' reports on the Teacher Report Form of the Child Behavior Checklist (Achenbach, 1991) from kindergarten through Grade 8.

First, conditional latent growth modeling analyses were performed using the nonabused sample where each vulnerability or protective factor was included as a predictor of the latent growth factors of internalizing behavior or externalizing behavior after covarying out ethnicity and gender. These analyses focused on examining the normative main effects of the protective and vulnerability factors on behavior problem trajectories for children who were not physically abused. Three growth factors were specified including an intercept (the level at kindergarten), a linear slope, and a quadratic growth (the square of the slope term).

Next, two-group latent growth modeling analyses were conducted in which the effects of a particular predictor were estimated separately in the abused and nonabused groups. These analyses focused on examining how the protective and vulnerability factors moderate the association between early physical abuse and trajectories of externalizing and internalizing behaviors. Specifically, resilience was operationalized as a form of interaction between physical abuse and the predictor such that abuse had weaker impact at certain values of the predictor. In the two-group latent growth modeling, they imposed equality constraint on the effect of predictor on the developmental trajectory of externalizing or internalizing behavior.

For externalizing behavior, there was a significant interaction between abuse and unilateral parental decision making [$\Delta\chi^2(3) = 9.49,\ p < .03$]. A closer examination of the model-implied trajectories in each group at the high (+1 SD) and the low (−1 SD) levels of unilateral parental decision making (i.e., nonabused low unilateral parental decision making, nonabused high unilateral parental decision making, abused low unilateral parental decision making, and abused high unilateral parental decision making) revealed that abused children with parents low on unilateral parental decision making showed similar levels of externalizing behaviors to those of nonabused children by Grade 8. For internalizing behavior, there were significant interactions between abuse and early hostile attributions [$\Delta\chi^2(3) = 8.96,\ p < .03$], between abuse and early stress [$\Delta\chi^2(3) = 9.39,\ p < .03$], and between abuse and adolescent stress [$\Delta\chi^2(3) = 14.27,\ p < .01$]. A closer examination of the model-implied trajectories of abused and nonabused groups with high vs. low levels of early hostile attributions showed the highest levels of internalizing behaviors among abused children with low early hostile attributions followed by abused children with high early hostile attributions. Thus, this finding suggested that early hostile attributions had beneficial effects on the development of internalizing behaviors among abused children.

The model-implied trajectories in each group at the high and the low levels of early and adolescent stress suggested that family stress was a vulnerability process in the development of internalizing behaviors. Specifically, abused children with elevated early family stress showed the highest levels of internalizing behaviors from kindergarten to Grade 7 when the trajectories between low- and high-stress abused groups crossed. As expected, adolescent stress did not predict internalizing behaviors at kindergarten. Similar to early family stress results, abused children with elevated adolescent family stress showed the highest levels of internalizing behaviors until Grade 7.

The findings for externalizing behaviors, unilateral parental decision making showed a protective-stabilizing effect (Luthar et al., 2000) for abused children. For internalizing behaviors, there was evidence of protective effects of early hostile attributions, but unexpectedly it was higher levels of early hostile attributions that made abused children less vulnerable to developing internalizing behaviors. For the nonabused children, early hostile attributions did not have significant effects on their internalizing behaviors. As the authors suggested, these findings may indicate that making hostile attributions might be an adaptive strategy for abused children to avoid internalizing problems resulting from self-blame in their circumstances. A vulnerability process was suggested in relation to early and adolescent stress, as abused children were significantly more vulnerable to developing internalizing problems when experiencing high levels of family stress. Overall, this study's findings highlight the usefulness of latent growth modeling for examining effects of protective and vulnerability processes on developmental trajectories (rather than developmental outcomes at a single time point) and further illustrate how multiple group latent growth modeling can be used to evaluate trajectories delineating both normal development as well as the development in high-risk conditions.

Implications of Latent Growth Modeling for Prevention and Intervention Science

Studies using latent growth modeling to test the efficacy of prevention and intervention programs are relatively rare. The frequent statistical approach for evaluating prevention and intervention programs has been heavily reliant upon the analysis of covariance (ANCOVA). Yet an ANCOVA approach does not allow researchers to examine the pattern of intraindividual variability over time (including

before and after the introduction of the programs) of the determinants of the changes in intraindividual trajectories. Latent growth modeling has several advantages over an ANCOVA approach (Taylor, Graham, Cumsille, & Hansen, 2000). First, researchers can test the effect of programs (or treatments) on trajectories of change. Second, researchers can estimate the relationship between initial status and rate of change. Third, latent growth modeling allows simultaneous consideration of variables from multiple time points (i.e., multivariate approach) thus increasing statistical power for testing overall program effects that are consistent over time instead of detecting program effects that may be significant only at one time point out of many repeated measurements.

Specifically, the following example illustrates how latent growth modeling can be used to evaluate the efficacy of prevention and intervention programs by investigating effects of programs on the average level of outcomes, rate of change of outcomes, and acceleration or deceleration in the rate of change of outcomes. In their longitudinal study of the efficacy of a randomized drug prevention program, the Adolescent Alcohol Prevention Trial, Taylor and colleagues (2000) compared four groups: (1) an information only group (ICU), which was a control group, and it received lessons about the social and health consequences of using alcohol and other drugs; (2) a resistance training plus information group (RU) that received instruction regarding how to resist pressure to use alcohol and other drugs, in addition to the information lessons; (3) a normative education plus information group (Norm) that received lessons that corrected misperceptions concerning the prevalence and acceptability of alcohol and other drug use among peers and established conservative norms regarding substance use, in addition to the information lessons; and (4) a combined resistance training and normative education plus information group (COMB) that received lessons from each of the other three components. They collected data regarding cigarette and alcohol use among 3,027 adolescents over a 5-year period, beginning in Grade 7 prior to intervention and again in Grades 8, 9, 10, and 11 following the intervention.

In the growth curve model, they constrained the intercept loading to [1 1 1 1 1] and the slope loadings to [−2 –1 0 1 2]. As such, the zero point was perfectly centered over all time points thus the intercept represented the average level over the repeated measurements. In addition to the slope latent factor that represents a linear growth, a quadratic latent factor was introduced to estimate the average degree of curvature, with a positive mean suggesting acceleration and a negative mean suggesting deceleration. Because the Time 1 data were collected prior to introducing the prevention program, two different (but not nested) models were tested with one involving Time 1 measure as a pretest measure and the other involving Time 1 measure as part of the growth.

In the first model where the pretest (Time 1) measure was modeled as a separate covariate, the Norm group showed significantly lower average levels of drug use compared to the ICU group (the reference group). No significant effects were detected regarding the program effect on the slope factor. Furthermore, the Norm group had a positive effect on the quadratic term indicating that the acceleration was slower (or less steep) for the Norm group compared to the ICU group. The pretest covariate (Grade 7) was positively related to the level, whereas it was negatively related to the slope. This means that higher levels of pretest drug use were related to higher average levels of drug use over the time spanning from Grade 8 to Grade 11, yet higher levels of pretest drug use were also related to slower growth rates over the time.

In the second model where the pretest (Time 1) measure was modeled as part of the growth curve, the Norm group, compared to the ICU group, suggested beneficial effects of the prevention program on the slope factor. In addition, similar to the first model, the Norm group had a positive effect on the quadratic term indicating that the acceleration was slower for the Norm group. The intercept factor (Grade 7) was negatively related to the slope factor. Taking the two models together, it seems that the first model worked better for interpreting of program effects on the intercept or level, whereas the second model worked better for interpreting of program effects on the slope.

Overall, the results suggested that the Normative Education program had a beneficial effect on the level of drug use as well as the rate of growth in drug use. Specifically, students in the Norm group, compared to those in the ICU group, showed lower average levels of drug use as well as slower increases in drug use over time. The program effect on the slope factor is particularly useful because excessive rate of growth in drug use during adolescence may indicate atypical development of drug use behaviors (as opposed to typical development) indicating that those adolescents with a steeper trajectory may be at greater risk for subsequent drug use problems and consequently addiction. Furthermore, students in the Norm group maintained a lower, more stable rate of change for alcohol use over the 5 years, particularly the first 2 years. Such slower rates of growth in the early years mean later onset for the adolescents. Given the significant association between early onset of alcohol and drug use and later problem behavior (Hawkins et al.,

1997), it was speculated that interventions that can slow down drug use rates in early adolescence may be effective in reducing problem behaviors in later adolescence or early adulthood (Taylor et al., 2000).

Comparing the two models from a modeling perspective, it seems that including pretest measure as a covariate is useful for interpreting of program effects on the intercept or level, whereas modeling pretest measure as part of a growth curve is useful for interpreting of program effects on the slope. The lesson is that researchers should consider using different latent growth modeling approaches depending on their research questions. The study by Taylor and colleagues clearly demonstrate that latent growth modeling provides a powerful multivariate approach to testing overall program effects on changes in developmental trajectories.

LATENT CHANGE (DIFFERENCE) SCORE MODELING

Latent change score models, previously referred to as latent difference score models (McArdle, 2001; McArdle & Hamagami, 2001), place an emphasis of time-sequential intraindividual change across multiple constructs. These models recognize that intraindividual growth is not constant and may be dependent upon a variety of factors—potentially its prior status as well as the status of another variable at a specific point in time. These ideas and models map on to dynamical systems notions of change. Such models highlight the time-sequential *process* of change recognizing that there are shocks to the development that can and should be modeled.

Univariate Latent Change Score Models

We begin the discussion of latent change score models with commonly considered univariate models. We note that the models presented in this section are limited, but the latent change score framework can accommodate the variety of models discussed in the latent growth modeling section (see Grimm, Zhang, Hamagami, & Mazzocco, 2013).

Latent change score models are built upon *latent change scores* as opposed to latent true scores. The latent change scores are created by fixed structural relations as opposed to calculating observed change scores. To begin the description of such models, we fall back on classical test theory where each observed score is composed of a latent true score and an error or unique score. This can be written as

$$Y_{it} = y_{it} + u_{it} \tag{22.21}$$

where Y_{it} is the observed score for individual i at time t, y_{it} is the true score for individual i at time t, and u_{it} is the unique score for individual i at time t. The unique scores are assumed to be independent, whereas the latent true scores have a fixed autoregressive association, such that the latent true score at time t is set equal to the latent true score at time $t-1$ plus the amount of change that occurred between the two measurement occasions—the latent change score, Δy_{it}, which can be written as

$$y_{it} = y_{it-1} + \Delta y_{it}. \tag{22.22}$$

The latent change score is, in essence, the residual of this regression equation; however, because the regression weight for y_{it-1} is set equal to 1, the residual is the individual difference between the scores – the intraindividual change. This can also be shown by rearranging the terms and solving for the latent change score, such as

$$\Delta y_{it} = y_{it} - y_{it-1} \tag{22.23}$$

Thus, the latent change score is the simple difference between the latent true score at time t and the latent true score at time *t-1*.

Now that the latent change scores are defined, we can write a trajectory equation based upon the latent change scores. The overall trajectory for the latent true scores is written as

$$y_{it} = h_{i1} + \left(\sum_{r=2}^{t} \Delta y_{ir} \right) \tag{22.24}$$

where y_{it} is the latent true score for individual i at time t, h_{i1} is the latent intercept or the value of y_{i1} – the true score at the first measurement occasion, and $\sum_{r=2}^{t} \Delta y_{ir}$ is the summation of the latent change scores from Time 2 through time t. Thus, the trajectory equation for a latent change score model is an initial true level of the attribute, h_{i1}, plus an accumulation of changes that have taken place up to time t $\left(\sum_{r=2}^{t} \Delta y_{ir} \right)$.

The final piece of the latent change score model is a mathematical model for the latent change scores. This mathematical model controls the type of change model (e.g., linear, quadratic). We note that the majority of models that can be fit within the traditional latent growth modeling framework can also be specified within the latent change score modeling framework (see Grimm,

Zhang, et al., 2013). Additionally, some new models have emerged within this framework (as in McArdle, 2001, 2009; McArdle & Hamagami, 2001; McArdle & Nesselroade, 1994).

We begin with reiterating commonly fit latent growth models within the latent change score framework before presenting some models that can only be specified within the latent change score modeling framework. We can start with the linear growth model by writing the following change equation:

$$\Delta y_{it} = h_{i2} \tag{22.25}$$

where h_{i2} is the constant change factor. This model is often referred to as the *constant change model* because the change equation states that the latent change score at time t is simply equal to a latent factor, h_{i2}, that varies over individuals. Thus, changes taking place between consecutive measurement occasions are constant within an individual. Since changes are constant, the overall growth trajectory is linear. This can be seen by writing the predicted true scores for the first four measurement occasions based on the trajectory model for latent change score models (equation 22.24) and this change equation (equation 22.25). The following are equations for the first four measurement occasions (assuming constant spacing between measurement occasions)

$$\begin{aligned} y_{i1} &= h_{i1}, \\ y_{i2} &= h_{i1} + h_{i2}, \\ y_{i3} &= h_{i1} + h_{i2} + h_{i2}, \text{and} \\ y_{i4} &= h_{i1} + h_{i2} + h_{i2} + h_{i2}. \end{aligned} \tag{22.26}$$

Thus, the trajectory is linear over time, $y_{it} = h_{i1} + h_{i2}(t-1)$

The change equation for the quadratic model is

$$\Delta y_{it} = h_{i2} + h_{i3} \cdot (t-1) \tag{22.27}$$

where h_{i2} is the constant change factor, and h_{i3} is the linear change factor. Writing out the trajectory equations for the first four measurements yields

$$\begin{aligned} y_{i1} &= h_{i1} \\ y_{i2} &= h_{i1} + h_{i2} + h_{i3} \\ y_{i3} &= h_{i1} + (h_{i2} + h_{i3}) + (h_{i2} + 2h_{i3}) \\ y_{i4} &= h_{i1} + (h_{i2} + h_{i3}) + (h_{i2} + 2h_{i3}) + (h_{i2} + 3h_{i3}) \end{aligned} \tag{22.28}$$

Rearranging terms of the last equation yields $y_{i4} = h_{i1} + (h_{i2} + h_{i2} + h_{i2}) + (1h_{i3} + 2h_{i3} + 3h_{i3})$ and continuing this progression yields $y_{it} = h_{i1} + h_{i2}(t-1) + h_{i3}\left(\frac{(t-1)^2}{2} + \frac{t}{2}\right)$, which is equivalent to the quadratic model.

As seen in these progressions for the linear and quadratic models, commonly fit latent growth models can be specified within the latent change score modeling framework by specifying an equation for the latent change scores that is the first derivative of the trajectory equation specified within the traditional latent growth modeling framework. Generally, this holds with the exception of models for which the first derivative is undefined for certain values of t (see Grimm, Zhang, et al., 2013).

As we noted, the latent change score framework also opens up opportunities for new models that could not previously be considered within the latent growth modeling framework. These models allow for time-sequential associations between latent true scores and latent change scores as well as time-sequential association between latent change scores over time. For example, changes can be proportional to the latent true score at the previous occasion. Such as model can be written as

$$\Delta y_{it} = \beta \cdot y_{i(t-1)} \tag{22.29}$$

In this *proportional change* model, the coefficient β is the proportional change coefficient or the auto-proportion coefficient and is an estimated parameter. This model yields exponential-like trajectories depending on the magnitude and sign of the initial true score and the proportional change coefficient. For example, with a positive initial true score and a positive proportional change coefficient, the model yields an exponential growth type of change pattern whereby changes accelerate over time.

Another model that is commonly fit combines aspects of the *constant change* model and the *proportional change* model. Thus, the change equation contains both the constant change factor (h_{i2}) and the proportional change () effects. This model is referred to as the *dual change score* model and is written as

$$\Delta y_{it} = h_{i2} + \beta \cdot y_{i(t-1)} \tag{22.30}$$

The combination of the constant change and proportional change within the dual change score model leads to a complex nonlinear growth trajectory that follows an increasing or decreasing, accelerating or decelerating exponential trajectory depending on the magnitude and

sign of the constant change factor, the proportional change coefficient, and the initial value.

The use of latent change scores is a general approach to studying time-sequential change, which has been extended in a variety of ways. For example, McArdle (2001) examined the proportional change model with independent residuals on the latent change scores, which leads to a type of an autoregressive model, and fit latent change score models with common factor scores. Furthermore, Hamagami and McArdle (2007) proposed second-order change models where acceleration as opposed to the rate of change is the key outcome of interest. Grimm et al. (2012) highlighted how prior latent change scores can be included as predictors of current latent change scores and Grimm et al. (2013) showed how such models can be used to fit a variety of nonlinear change models to emphasize the intraindividual rate of change. Thus, it is easy to say that the models presented here are simply the beginning of univariate models that can be considered within the latent change score modeling framework and that the latent change score framework opens the door to a collection of interesting models focused on time-sequential change.

Bivariate Latent Change Score Models

The latent change score framework lends itself to the study of bivariate or multivariate processes because intraindividual change at a given point in time is the outcome of interest and time-sequential effects can be included (e.g., proportional change coefficient). Prior to the introduction of the latent change score framework, multivariable time-sequential associations were often studied with the autoregressive cross-lagged model (Cook & Campbell, 1977; Jöreskog, 1974; Rogosa, 1980). In this model there are autoregressive effects where the prior state of the variable at time $t - 1$ (i.e., $y_{i(t-1)}$) is a predictor of the current state of the variable (i.e., y_{it}) as well as cross-lagged effects where the prior state of a second variable at time $t - 1$ (i.e., $x_{i(t-1)}$) is also a predictor of the current state of the variable (i.e., y_{it}) and vice versa. In terms of understanding intraindividual growth, the autoregressive cross-lagged model only captures relative deviations from a sample-level mean and does not capture systematic intraindividual change.

For this reason, researchers have turned to multivariate growth curves, also referred to as parallel process growth models (i.e., cross-domain growth models; MacCallum, Kim, Malarkey, & Kiecolt-Glaser, 1997; McArdle, 1988). This popular alternative examines whether change in one variable is associated with change in the second variable. Although, this model captures intraindividual changes and associations among intraindividual changes, the model is unable to capture time-sequential associations and all associations involving intraindividual change are between-persons as opposed to within-person changes, which are often of interest.

As noted, the latent change score framework lends itself to the analysis of multiple variables over time. Specifically, a series of change equations are written as opposed to a single univariate change equation. Building on equation 22.30, we can write change equations for and Δx_{it}, based on constant change and proportional change, such as

$$\begin{aligned} \Delta y_{it} &= h_{i2} + \beta_y \cdot y_{i(t-1)} \\ \Delta x_{it} &= g_{i2} + \beta_x \cdot x_{i(t-1)} \end{aligned} \tag{22.31}$$

where h_{i2} and g_{i2} are the constant change factors and β_y and β_x are the proportional change effects for Δy_{it} and Δx_{it}, respectively. From here coupling effects, akin to cross-lagged effects from the autoregressive cross-lagged model, can be added to evaluate how the prior status of x impacts changes in y and how the prior status of y impacts changes in x. Thus, we write

$$\begin{aligned} \Delta y_{it} &= h_{i2} + \beta_y \cdot y_{i(t-1)} + \gamma_y \cdot x_{i(t-1)} \\ \Delta x_{it} &= g_{i2} + \beta_x \cdot x_{i(t-1)} + \gamma_x \cdot y_{i(t-1)} \end{aligned} \tag{22.32}$$

where γ_y and γ_x are the coupling coefficients describing across variable time-sequential associations. We note that the proportional and coupling coefficients are often held to be invariant over time; however, in many cases this constraint is unneeded and relaxing this constraint may theoretically reasonable (see Dogan, Stockdale, Widaman, & Conger, 2010).

Interpretation of bivariate latent change score models is often centered on the *dynamic parameters*—the proportional change and coupling coefficients. If a coupling coefficient is significantly different from zero, it can be said that there is a lead-lag relationship, such that the prior status of one variable provides an indication of the subsequent changes in the second variable. For example, if γ_y is significantly different from zero and γ_x is not, then it can be said that x is a leading indicator of y. We emphasize that this type of association is simply an association, and is not causal in any way. Additionally, it is important to note that this association is observed in the potential absence of other potential leading indicators.

Latent Change Score Modeling Studies for Understanding Risk and Protective Mechanisms as Predictors, Mediators, and Moderators

We used a latent change score modeling approach to study how the effects of negative affect—a risk factor of children's behavioral and emotional problems—may be moderated by attentional regulation in the development of internalizing and externalizing problems (Kim & Deater-Deckard, 2011). Extant literature suggests that a good attention span is critical to healthy social emotional development, especially in the sense that attention control reflects effective cognitive self-regulation of negative emotions such as anger. Using the national sample of children ($N = 1{,}079$) from the Study of Early Child Care and Youth Development (SECCYD), who were assessed four times from 4.5 to 11 years of age, we examined attention span as a moderator of reciprocal links between changes in anger and changes in externalizing and internalizing problems. Composite scores for anger and attention (i.e., poor attention span or distractibility) were computed using indicators from multiple informants including mothers, fathers, and teachers. Every indicator was standardized, averaged, and standardized again to yield composite z scores within each of the four time points for anger and inattention. Externalizing and internalizing problems were reported by mothers using the total raw scores of the Child Behavior Checklist (CBCL; Achenbach, 1991).

The latent change score modeling was used to predict dynamic changes in externalizing/internalizing problems from repeatedly measured anger from age 4.5 to 11. As shown in Figure 22.6, the hypothesized latent change score model included the time-varying predictor of anger to examine the prospective effects of anger on developmental changes in behavioral/emotional problems. Because anger scores were standardized z scores compositing different scales reported by multiple informants, its time series data were constructed as a Markov simplex model based on manifest variables instead of a latent change score model. Furthermore, the hypothesized model estimated bidirectional cross-lagged effects between anger and behavioral/emotional problems as well as the concurrent correlations between the two constructs within each time point. Therefore, this model tested whether anger predicts changes in behavioral/emotional problems or vice versa, after accounting for the autoregressive effects of the prior anger or behavioral/emotional problems.

Two-group structural equation models were used to test the moderating effects of attention in the link between anger and behavioral/emotional problems. The sample was divided into the poor (above median, $n = 540$) versus good attention (below median, $n = 539$) groups based on each individual's grand mean of the inattention composites from 4.5 to eleven years. We first fit a model in which all parameters were freely estimated across the two groups. In subsequent models, equality constraints were added to test numeric invariance between poor and good attention groups with respect to the effects of anger on behavioral/emotional problems (the Equal Anger Effect model) and the effects of behavioral/emotional problems on anger (the Equal Behavior Problem Effect model).

The chi-square difference tests comparing nested model fits revealed that the model in which parameters were separately estimated provided the best fit for externalizing problems. Thus, the results indicated that the poor and good attention groups showed significantly different patterns and magnitude for the effects of anger on externalizing problems as well as for the effects of externalizing problems on anger. Specifically, as shown in Figure 22.6, the poor attention group showed more persistent effects of anger on externalizing problems over time compared to the good attention group. In the poor attention group, anger at 4.5, 7, and 9 years consistently predicted smaller decreases (or in some cases larger increases) in externalizing problems from 4.5 to 7 years ($t = 6.93$, $p < .05$), from 7 to 9 years ($t = 2.64$, $p < .05$), and from 9 to 11 years ($t = 5.12$, $p < .05$), respectively. In terms of the reciprocal effects of externalizing problems on anger, higher initial levels of externalizing problems at 4.5 years predicted increases in anger between 4.5 and 7 years ($t = 4.40$, $p < .05$). Larger increases in externalizing problems between 4.5 and 7 years were related to smaller increases in anger between 7 and 9 years ($t = 3.95$, $p < .05$), whereas larger increases in externalizing problems between 7 and 9 years were related to larger increases in anger between 9 and 11 years ($t = 2.69$, $p < .05$). The model-estimated mean levels of externalizing problems decreased from 12.25 to 7.56 over the four assessments, indicating significant decreases between 4.5 and 7 years and between 9 and 11 years.

For the good attention group, the effects of anger on externalizing problems decreased over time. Anger at 4.5 and 7 years predicted smaller decreases (or in some cases larger increases) in externalizing problems from 4.5 to 7 years ($t = 12.20$, $p < .05$) and from 7 to 9 years ($t = 5.54$, $p < .05$), respectively. However, the prospective effect of anger at 9 years on changes in externalizing problems from 9 to 11 years was not significant ($t = 1.42$, *ns*). Significant reciprocal effects of externalizing problems on anger indicated that higher levels of externalizing

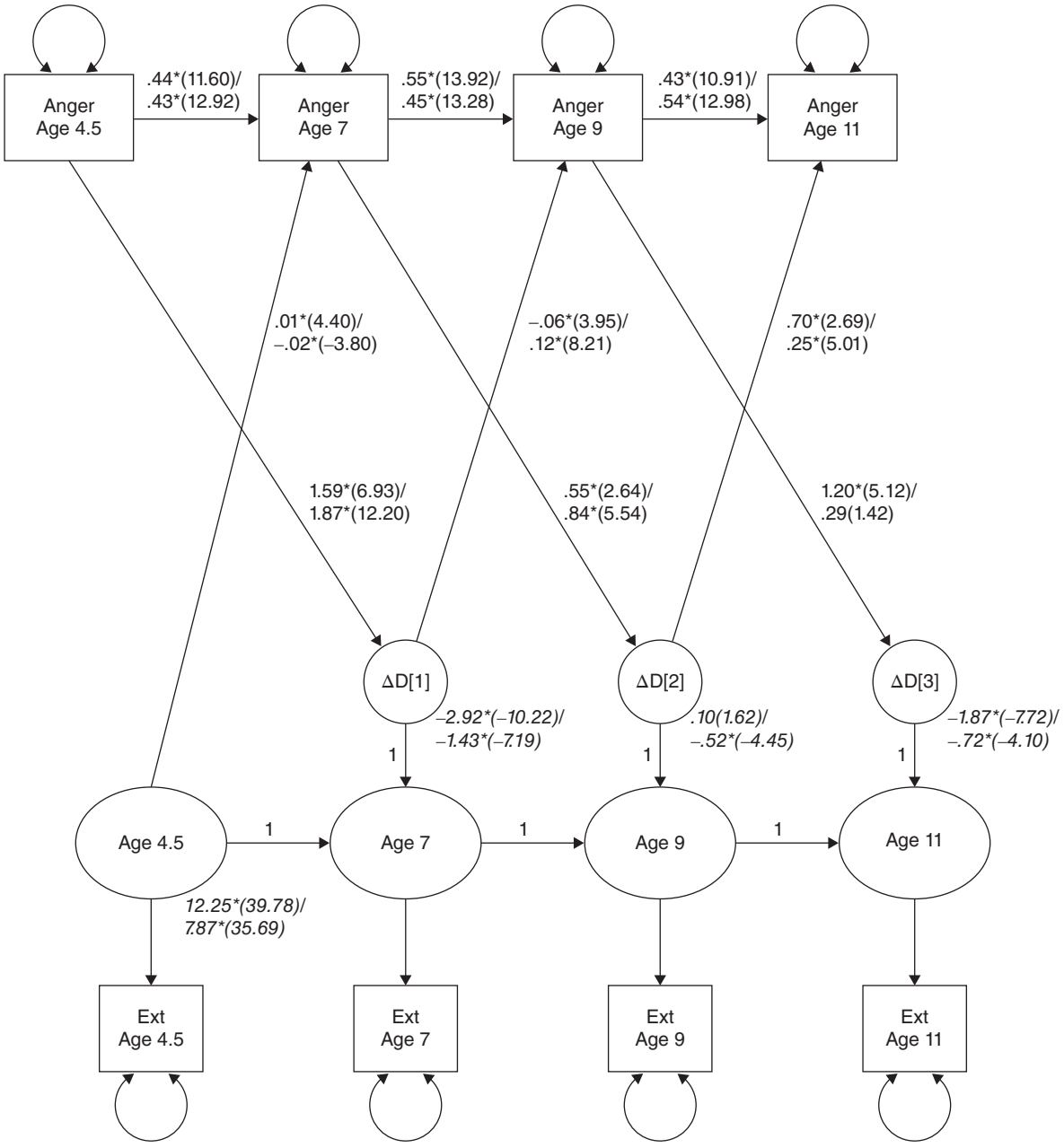


Figure 22.6 Latent change score model of the moderation of inattention between anger and externalizing problems. Values given are unstandardized coefficients with critical ratio (CR) in parentheses. For each path, the coefficients (CR) are listed for poor attention/good attention groups. * $p < .05$. *Source*: Adapted from "Dynamic changes in anger, externalizing and internalizing problems: attention and regulation" by J. Kim and K. Deater-Deckard, 2011, *Journal of Child Psychology and Psychiatry*, *52*, pp. 156–166.

problems at 4.5 years were related to smaller increases in anger between 4.5 and 7 years ($t = –3.80$, $p < .05$). However, increases in externalizing problems between 4.5 and 7 years and between 7 and 9 years predicted increases in anger between 7 and 9 years ($t = 8.21$, $p < .05$) and between 9 and 11 years ($t = 5.01$, $p < .05$), respectively. Children in the good attention group showed significantly lower initial levels of externalizing problems compared with those in the poor attention group, and their estimated means of externalizing problems decreased from 7.87 to 5.20 over the four assessments indicating consistently significant decreases between consecutive time points.

Nested model comparisons indicated that the equal anger effect model provided the best fit for internalizing

problems. Results of the best-fitting model further revealed that the reciprocal effects of internalizing problems on anger were stronger and more persistent in the poor attention group, but the two groups did not differ regarding the effects of anger on internalizing problems. As shown in Figure 22.7, regardless of inattention level, higher anger at 4.5 and 7 years predicted greater increases in internalizing problems from 4.5 to 7 years ($t = 7.12$, $p < .05$) and from 7 to 9 years ($t = 3.93$, $p < .05$), respectively. Differential reciprocal effects between the two attention groups suggested that the patterns of reciprocal effects were more consistent and stronger for the poor attention group. For children in the poor attention group, higher initial levels of internalizing problems at 4.5 years predicted

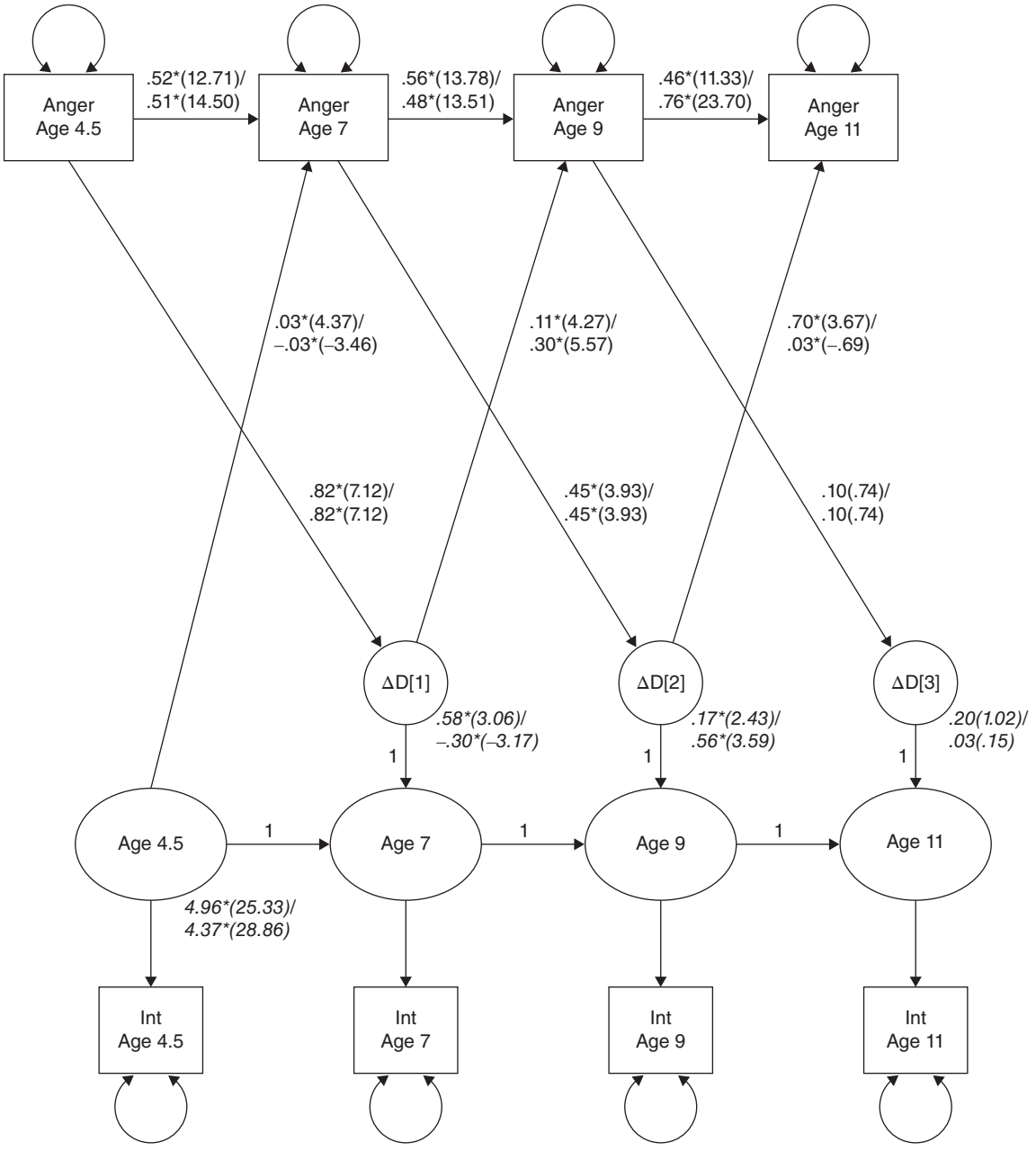


Figure 22.7 Latent change score model of the moderation or inattention between anger and internalizing problems. Values are given unstandardized coefficients with critical ratio (CR) in parentheses. For each path, the coefficients (CR) are listed for poor attention/good attention groups. * $p < .05$. *Source*: Adapted from "Dynamic changes in anger, externalizing and internalizing problems: attention and regulation" by J. Kim and K. Deater-Deckard, 2011, *Journal of Child Psychology and Psychiatry*, *52*, pp. 156–166.

increased anger between 4.5 and 7 years ($t = 4.37,\ p < .05$). Increases in internalizing problems between 4.5 and 7 years and between 7 and 9 years consistently predicted increased anger between 7 and 9 years ($t = 4.27,\ p < .05$) and between 9 and 11 years ($t = 3.67,\ p < .05$), respectively. The model-estimated mean levels of internalizing problems increased from 4.96 to 5.91 over the four repeated assessments, indicating significant increases between 4.5 and 7 years, and between 7 and 9 years.

In contrast, for the good attention group, higher levels of internalizing problems at 4.5 years were associated with smaller increases in anger between 4.5 and 7 years ($t = -3.46,\ p < .05$). Increases in internalizing problems between 4.5 and 7 years predicted increased anger between 7 and 9 years ($t = 5.57,\ p < .05$). Changes in internalizing problems between 7 and 9 years did not predict changes in anger between 9 and 11 years ($t = -.69$, *ns*). Children in the good attention group showed consistently lower levels of internalizing problems compared to those in the poor attention group, with some fluctuations. Their estimated means of internalizing problems showed significant decreases between 4.5 and 7 years (from 4.37 to 4.07), significant increases between 7 and 9 years (from 4.07 to 4.63), and then no significant changes between 9 and 11 years (from 4.63 to 4.66).

To summarize, the findings showed significant moderating effects of attention indicating more persistent effects of anger on externalizing problems in the poor attention group. For both good and poor attention groups, high anger was associated with increases in internalizing problems, but the reciprocal effects of internalizing problems on anger were stronger and more persistent in the poor attention group. Thus, it appears that strong attention regulation may serve to protect children with higher levels of dispositional anger from developing behavioral and emotional problems in middle childhood. This study illustrates how latent change score models can be used to test reciprocal lagged effects between changes in risk factors (e.g., negative affect) and changes in psychopathology and to test moderating (or interactive) effects of protective factors (e.g., attention regulation) in the link between risk factors and psychopathology.

Latent Change Score Modeling Studies for Understanding Contextual Influences

Latent Change Score Modeling for Cross-Domain Relations in Risk and Psychopathology

In the study we reviewed earlier, Grimm (2007) used bivariate latent change score growth models to investigate the developmental relationships between depression and academic achievement in childhood. The latent change score modeling carries all the advantages of the latent growth modeling and offers additional advantages of directly testing time-dependent change. As shown in Figure 22.8, in a latent change score model, the outcome of interest is represented by latent change scores that can be predicted by previous scores. Specifically, four bivariate latent change score growth models were fit to the repeated measurements of achievement and depression. Different restrictions were introduced to determine whether academic achievement or depression were leading or lagging indicators.

The first model was a baseline model where the coupling parameters were fixed to zero. This no-coupling model fit the data well [$\chi^2(99) = 242,\ \text{RMSEA} = .01$]. In the second model, the previous level of depression was predictive of the changes in academic achievement and the model fit was nearly identical to the model with no-coupling effects [$\chi^2(98) = 241,\ \text{RMSEA} = .01;\ \Delta\chi^2 = 1,\ \Delta df = 1$]. However, when the coupling parameter from academic achievement to changes in depression was estimated, the model fit improved significantly [$\chi^2(98) = 233,\ \text{RMSEA} = .01;\ \Delta\chi^2 = 9,\ \Delta df = 1$]. In the final model, bidirectional coupling parameters were estimated and the fit was similar to the third model in which only the coupling parameter from academic achievement to changes in depression was estimated [$\chi^2(97) = 231,\ \text{RMSEA} = .01;\ \Delta\chi^2 = 2,\ \Delta df = 1$].

Thus, the results indicated that academic achievement was the leading indicator in the development of academic achievement and depression in childhood, suggesting that knowing the levels of the prior academic achievement score helped predict the subsequent changes in depression. Specifically, the coupling parameter from academic achievement to changes in depression was significant and negative ($\gamma = -.03,\ p < .05$), indicating that children who had a higher previous level of academic achievement showed larger decreases or smaller increases in depression. The findings further imply that academic achievement may be considered a protective factor against increasing trajectories of depression between ages 8 and 14. As shown in this study, findings from multivariate latent change score models can provide information about which risk or protective factors lead the psychopathological outcomes and which lag in such longitudinal relationships, whereas findings from multivariate LGM reveal only general associations among developmental changes across different variables. One caveat is that, although bivariate latent change score models allow us to examine time-dependent

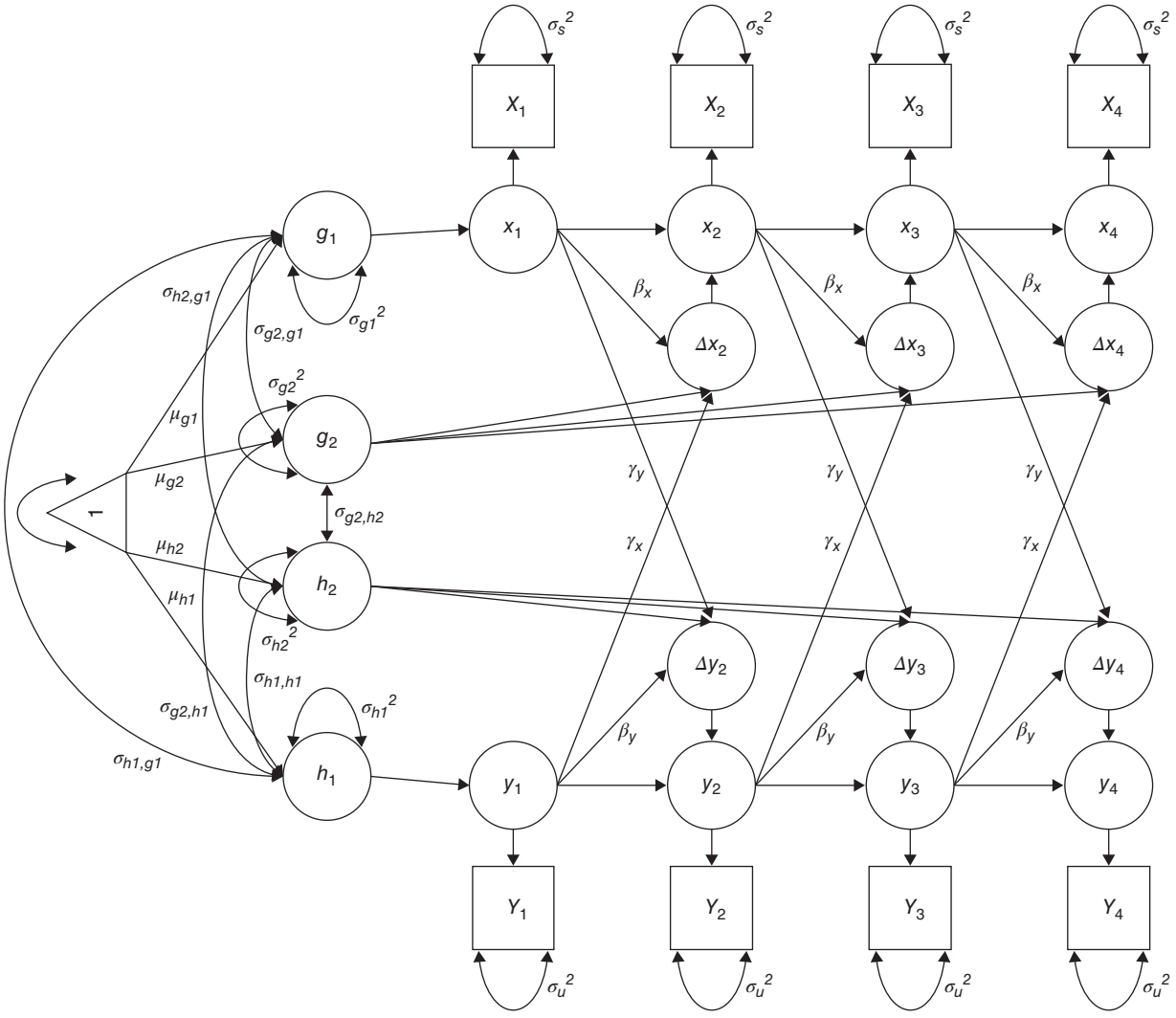


Figure 22.8 A bivariate dual change score model.

relationships (i.e., leading and lagging indicators), they are not causal models.

Latent Change Score Modeling for Multiple Levels of Analysis

Externalizing psychopathology encompasses a broad range of psychiatric disorders, including substance use disorders and conduct disorders, and a characteristic common to different types of externalizing psychopathology is high levels of behavioral disinhibition (Bogg & Finn, 2010). Research also suggests that reduced working memory capacity seems to play a crucial role in disinhibited patterns of behavior associated with externalizing psychopathology (Endres, Rickert, Bogg, Lucas, & Finn, 2011). McArdle and his colleagues used latent change score models to examine dynamic interrelationships between longitudinal changes in memory performance and longitudinal changes in brain structure (McArdle et al., 2004). Participants included 225 healthy adults with no evidence of major clinical disease ($n = 109$ men). These participants were 30–80 years of age at baseline (with an average of 40 individuals per decade), and they were followed up about seven years after the initial assessment ($n = 197$ reevaluated). The brain structure was assessed using the computerized axial tomography (CT) information focusing on the lateral ventricular size (LVS; the mean of the measurements from the left and right hemisphere). Memory was assessed by the Wechsler Memory Scores (WMS; Wechsler, 1945).

They fit four alternative age-based latent change score models to the data by using the observed ages (i.e., developmental trajectories of memory and LVS from age 30 to age 90). The first model was a no-change

model that estimated three parameters including an initial mean, an initial variance, and a constant error variance but no slope parameters ($\chi^2 = 0$, $df = 0$). The second model was the constant change score model in which three additional time-dependent slope parameters were estimated. For LVS, there were small but positive systematic increases in slope means ($\mu = .006$, $p < .05$), slope variances ($\sigma = .004$, $p < .05$), and covariances between intercept and slope ($\rho = -.66$, $p < .05$) over every 5 years of age. This model showed a statistical improvement over the no-change model ($\chi^2 = 114$, $df = 13$). For memory, there were small but negative systematic changes over age ($\mu = -.01$, $p < .05$; $\sigma = .002$, $p < .05$; $\rho = -.28$, *ns*), with a small statistical improvement in fit over the no-change model ($\chi^2 = 13$, $df = 3$).

In the third model, only the proportional changes (β) were estimated while the additive changes (α), slope means, slope variances, and covariances between intercept and slope were fixed to zero. The results showed positive proportional changes for LVS ($\beta = .02$, $p < .05$), and negative proportional changes for memory ($\beta = -.01$, $p < .05$). This proportional change model yielded substantial improvement in fit for LVS ($\chi^2 = 109$, $df = 1$) and minor improvement for memory ($\chi^2 = 5$, $df = 1$). Finally, they fit a dual change model in which the mean of the slopes were allowed to be free and additive changes (α) were fixed to one (for identification purposes). The results indicated positive proportional changes for both LVS ($\beta = .03$, $p < .05$) and memory ($\beta = .05$, $p < .05$) as well significant slope variances for both LVS ($\sigma = .004$, $p < .05$) and memory ($\sigma = .003$, $p < .05$). The linear slope means were significant for memory ($\mu = -.05$, $p < .05$), but not for LVS ($\mu = -.001$, *ns*). Dual change models showed significant improvement over the no-change model ($\chi^2 = 124$, $df =$ 4 for LVS and $\chi^2 = 24$, $df = 4$ for memory) and yielded the best fits compared with the previous models.

Next, four alternative bivariate latent change score models were tested. The first model was a full bivariate latent change score model where all coupling parameters were estimated ($\chi^2 = 0$, $df = 0$). In the second model, the parameter representing the effect of LVS on the latent change scores of memory was fixed to zero and this model yielded a significantly degraded fit compared to the full bivariate latent change score model ($\chi^2 = 20$, $df = 1$). The third model had the parameter representing the effect of memory on the latent change scores of LVS fixed to zero and there was a much smaller loss of fit ($\chi^2 = 3$, $df = 1$). Finally, in the last model, no coupling was allowed and this model showed a notable loss of fit ($\chi^2 = 23$, $df = 2$).

Thus, the results of the best fitting model, no memory to LVS coupling effect model, showed notable and systematic coupling across the LVS and memory variables. A closer examination of the coupling effects indicated a significant negative coupling effect from LVS to changes in memory ($\gamma = -.18$, $p < .05$), but a nonsignificant trivial coupling effect from memory to changes in LVS ($\gamma = 0$, *ns*). Consistent with prior research on neurophysiological influences on neuropsychological and behavioral changes, these findings suggest that an increase in the relative size of the lateral ventricle is a leading indicator of decreases in memory, and the reverse is not apparent.

This study was the first report demonstrating the cross-lagged longitudinal relationship between in vivo brain measurements and cognitive function across adulthood. The latent change score models used in this study highlight the recent advancement in longitudinal modeling techniques that can be utilized as a quantitative test of cross-domain temporal relations within a person over time. It should be noted, however, that as with most longitudinal multivariate data in developmental psychopathology research, the data were not collected in a randomized controlled pre- and posttest design, thus causal influences were confounded and were difficult to isolate.

Latent Change Score Modeling for Cross-Population Comparisons in Risk and Psychopathology

A recent study by Kim-Spoon, Cicchetti, and Rogosch (2013) using a sample of maltreated and nonmaltreated children illustrates how latent change score models can be used to interface between normal and abnormal development. We examined longitudinal contributions of emotion regulation and emotion lability/negativity to internalizing symptomatology in a low-income sample of 171 maltreated and 151 nonmaltreated children. These children attended a week-long day camp program for inner city children and they were annually assessed from age 7 to 10 years. Four subtypes of maltreatment, emotional maltreatment, physical neglect, physical abuse, and sexual abuse, were assessed using the Maltreatment Classification System (MCS; Barnett, Manly, & Cicchetti, 1993). Counselors' ratings on the Emotion Regulation subscale and the Lability/Negativity subscale of the Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1998) were used to capture processes central to emotion regulation and emotion lability/negativity. The emotion regulation subscale consisted of items assessing adaptive regulation, including socially appropriate emotional displays, empathy, equanimity, and emotional understanding. The emotion

lability/negativity subscale was composed of items assessing arousal, reactivity, emotional intensity, expression of negative emotions, and mood lability.

Preliminary latent change score analyses indicated that the means and variances of latent change score factors were not significantly different from zero for emotion regulation and emotion lability/negativity. Therefore, the time series data of predictors (emotion regulation and emotion lability/negativity) were constructed as a Markov simplex model based on manifest variables instead of a latent change score model. The baseline latent change score model for internalizing symptomatology showed significant and negative path estimates for proportional change for internalizing symptomatology, indicating annual decreases in internalizing symptomatology from age 7 to 10 ($b = -1.08$, $SE = .17$, $p < .05$). The model-estimated mean levels of internalizing symptomatology decreased from 6.89 at age 7 to 3.69 at age 10.

We were primarily interested in developmental processes by which emotion regulation and emotion lability/negativity conjointly contributed to change in internalizing symptomatology. We tested time-lagged mediation models estimating whether the prospective prediction of emotion lability/negativity was mediated by emotion regulation or vice versa. These models were tested using two-group structural equation modeling approaches to explore any significant differences between maltreated and nonmaltreated groups. We first tested the Direct Effect models in which the time series data of emotion regulation or emotion lability/negativity predicted the latent change scores of internalizing symptomatology. We compared the configural invariance model in which all parameters were freed to be estimated [$\chi^2(28) = 39.78$, $CFI = .97$, RMSEA = .04 for emotion regulation direct effects and $\chi^2(28) = 50.14$, $CFI = .94$, RMSEA = .05 for lability/negativity direct effects] and the equal direct effect model in which direct effects were fixed to be equal between the two groups [$\chi^2(31) = 40.85$, CFI = .97, RMSEA = .03 for emotion regulation direct effects, and $\chi^2(31) = 51.13$, CFI = .95, RMSEA = .05 for lability/negativity direct effects]. The nonsignificant difference between the configural invariance model and the equal direct effect model indicated that there was no difference in the direct effects of emotion regulation and emotion lability/negativity between maltreated and nonmaltreated children [$\Delta\chi^2 = 1.07$, $\Delta df = 3$, $p = .79$ for emotion regulation and $\Delta\chi^2 = .99$, $\Delta df = 3$, $p = .80$ for lability/negativity]. Specific findings indicated that higher emotion regulation predicted decreases in internalizing symptomatology at all ages ($p < .05$). In contrast, higher emotion lability/negativity predicted increases in internalizing symptomatology from age 7 to 8 and from age 8 to 9 ($p < .05$), but its effect became weaker between ages 9 and 10 ($p = .08$).

Next, we tested two sets of longitudinal mediation models, first, with emotion regulation as a mediating variable in the prospective relations between emotion lability/negativity and change in internalizing symptomatology, and second, with emotion lability/negativity as a mediating variable in the prospective relations between emotion regulation and change in internalizing symptomatology. As shown in Figure 22.9, we examined time-lagged mediation effects (e.g., age 7 emotion lability/negativity → age 8 emotion regulation → change in internalizing symptomatology between ages 8 and 9). Within each set, we performed two-group SEM analyses and compared two nested models to evaluate any differences between maltreated and nonmaltreated children. In the first model, the Configural Invariance Model, all the parameters were freed to be estimated for maltreated and nonmaltreated children. In the second model, equal direct and mediated effect model, both direct and indirect effects were fixed to be equal between maltreated and nonmaltreated groups.

In the first set of models in which emotion regulation was tested as a mediator, the model fit of the equal direct and mediated effect model [$\chi^2(58) = 92.10$, CFI = .96, RMSEA = .04] was not significantly worse than that of the configural invariance model [$\chi^2(31) = 89.22$, CFI = .95, RMSEA = .05], indicating no difference in direct and indirect effects of emotion lability/negativity between maltreated and nonmaltreated groups [$\Delta\chi^2 = 2.88$, $\Delta df = 8$, $p = .94$]. As shown in Figure 22.9, the results of the equal direct and mediated effect model indicated that higher levels of emotion lability/negativity at age 7 and at age 8 predicted lower levels of emotion regulation at age 8 and at age 9, respectively, which in turn predicted a subsequent increase in internalizing symptomatology from age 8 to 9 and from age 9 to 10 respectively. These influences were above and beyond, or controlling for, autoregressive effects and natural change (i.e., decreasing slope) in internalizing symptomatology. However, previously significant direct effects of emotion lability/negativity on internalizing symptomatology became nonsignificant. Sobel's approximate significance tests (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002) for the significance levels of the indirect effects revealed that both of the paths involved in the mediation (emotion lability/negativity → emotion regulation → diff2/3) were significant: $z = 2.21$, $p < .05$ for prediction of diff2; and $z = 2.15$, $p < .05$ for diff3. The significance

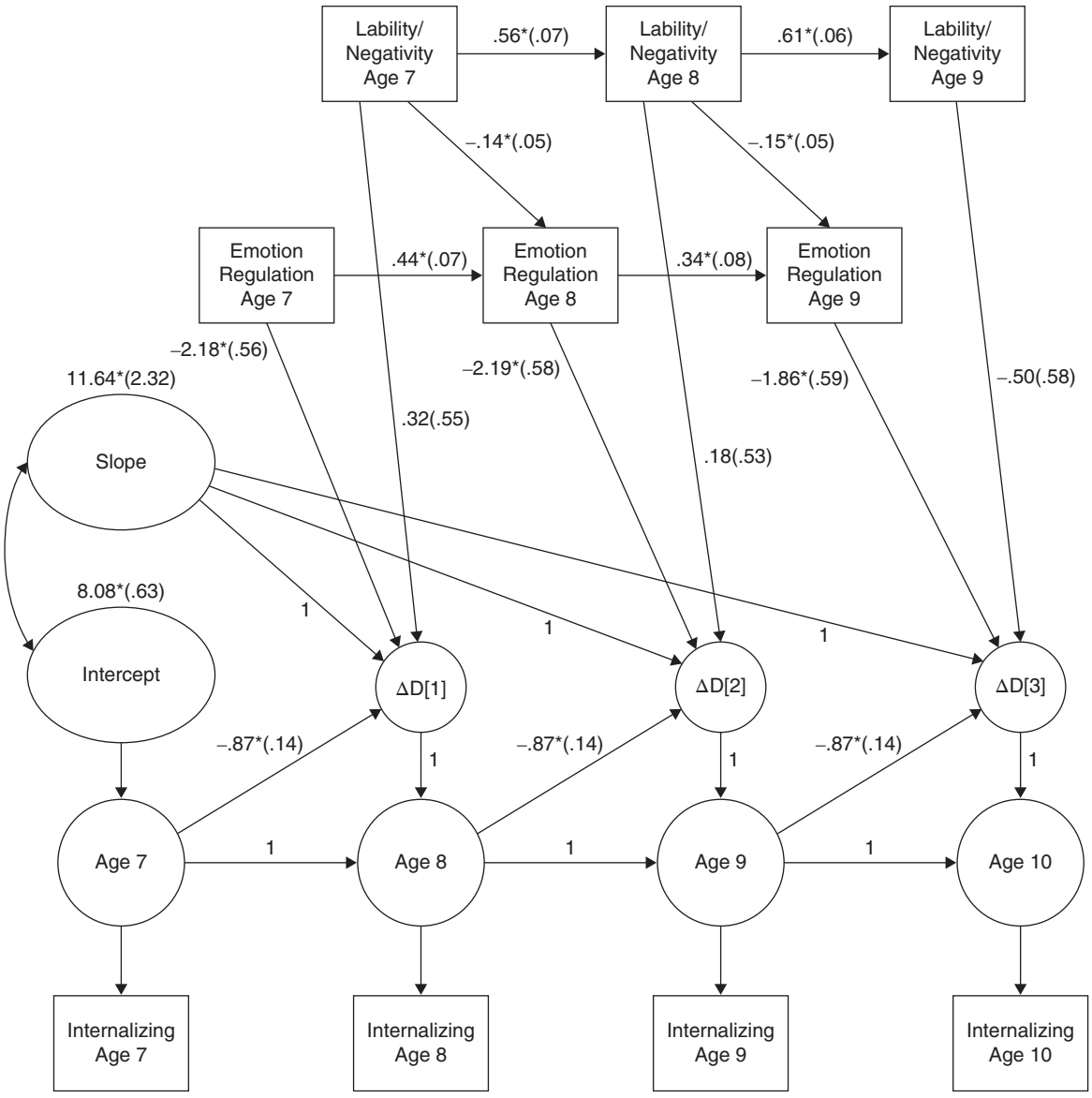


Figure 22.9 Latent change score model of emotion lability/negativity predicting internalizing symptomatology, mediated by emotion regulation. Unstandardized parameter estimates (SE) are presented. *Source*: Adapted from "A longitudinal study of emotion regulation, negative emotionality, and internalizing symptomatology in maltreated and nonmaltreated children" by J. Kim-Spoon, D. Cicchetti, and F. A. Rogosch, 2013, *Child Development*, *84*, pp. 512–527.

test for the equality of the two mediated effects indicated consistent (of similar magnitude) mediated effects of emotion regulation for the link between emotion lability/negativity and internalizing symptomatology across ages ($z = 0.03$, $p = .98$).

In the second set of models in which emotion lability/negativity was tested as a mediator of the link between emotion regulation and change in internalizing symptomatology, the equal direct and mediated effect model [$\chi^2(58) = 105.88$, CFI = .94, RMSEA = .05] was selected as the better-fitting model compared to the Configural Invariance Model [$\chi^2(50) = 101.54$, CFI = .94, RMSEA = .06]. Thus, the results indicated no significant difference in direct and indirect effects of emotion regulation between maltreated and nonmaltreated groups. Specifically, higher levels of emotion regulation directly predicted a subsequent decrease in internalizing symptomatology at all ages ($b = -2.52$, $SE = .56$, $p < .05$ for age 7 emotion regulation → ΔD[1]; $b = -2.62$, $SE = .58$, $p < .05$ for age 8 emotion regulation → ΔD[2]; $b = -2.43$,

$SE = .58$, $p < .05$ for age 9 emotion regulation → ΔD[3]). However, neither the cross-lagged effects of emotion regulation on emotion lability/negativity ($p = .38$˜.50) nor the direct effects of emotion lability/negativity on internalizing symptomatology difference scores ($p = .18$ ˜.49) were significant. Therefore, the results of our mediation models indicated that emotion regulation is a mediating process for the longitudinal association between emotion lability/negativity and internalizing symptomatology, whereas emotion lability/negativity is not a mediating process for the longitudinal association between emotion regulation and internalizing symptomatology.

Overall, the results of testing mediation models using the latent change score modeling suggest that emotion regulation may be a proximal process through which emotion lability/negativity may influence developmental changes in internalizing symptomatology for both maltreated and nonmaltreated children. This study exemplifies how latent change score models can be used to enhance the knowledge base of developmental psychopathology by allowing the examination of cross-lagged associations among multiple levels of processes (such as emotion lability/negativity, emotion regulation) and psychopathological outcomes (such as child internalizing symptomatology) to determine directionality in the prediction of risk and protective factors for change in psychopathology outcomes.

Implications of Latent Change Score Modeling for Prevention and Intervention Science

The following example illustrates how latent change score model can be used to illuminate processes that occur following treatment or intervention. Hawley, Ho, Zuroff, and Blatt (2006) considered how an individual's specific vulnerability as shown in personality and interpersonal context of therapy may influence underlying symptom change during therapeutic processes. Specifically, they examined the longitudinal dynamics between depression and perfectionism (a personality vulnerability factor) as related to the quality of therapeutic alliance during treatment for depression. The latent change score approach was the most suitable choice to understand the multidimensional nature of therapeutic change because it provides a statistical framework to evaluate dynamic longitudinal growth within time series data while considering interrelationships between multivariate change processes.

Participants included 162 patients with major depressive disorder who were identified as treatment completers (those who attended at least 12 treatment sessions over a 15-week period). The perfectionism subscale of the Dysfunctional Attitudes Scale (Weissman & Beck, 1978) was used to assess patients' tendency to view the self in punitive terms (e.g., regarding failure in meeting self-imposed standards). Depression was assessed using the Hamilton Rating Scale for Depression (Hamilton, 1960). Both perfectionism and depression were assessed at intake (i.e., Week 0) and Weeks 4, 8, 12, and 16. To measure the strength of the therapeutic alliance, therapy sessions were rated using an observation-based measure of the Vanderbilt Therapeutic Alliance Scale (Hartley & Strupp, 1983) that distinguishes between patient and therapist contributions to the alliance.

They set out to evaluate the temporal course of growth and change in perfectionism and depression and further determine the longitudinal interrelationship of these variables as related to the therapeutic alliance. They first conducted latent change score univariate analyses of the repeated-measures time series separately for perfectionism and depression to understand the temporal nature of change in therapy. They compared four statistical alternative models to identify a model that best represents longitudinal changes in perfectionism and depression. A dual change score latent change score model was compared to (1) a no change model, (2) a constant change model (estimating changes only in latent slope coefficients), and (3) a proportional change model (estimating changes only that are proportional to the previous latent scores). For both perfectionism and depression, the dual latent change score model was the best fitting model [$\chi^2(7) = 11.19$, CFI = .98, RMSEA = .06 for depression and $\chi^2(8) = 17.87$, CFI = .98, RMSEA = .09 for perfectionism].

Next, the authors conducted bivariate analyses to evaluate dynamic coupling between depression and perfectionism. Based on several theoretical models, they tested four alternative bivariate latent change score models. First, the personality vulnerability model proposed that personality plays an essential role in the etiology, maintenance, and treatment of depression. Second, the scar model proposed that personality change occurs as a consequence of depression. Third, the reciprocal causality model proposed that the relationship between personality and depression is interactive. They also tested a no-coupling model in which no dynamic coupling was allowed between the repeated measures of perfectionism and depression. The no coupling and scar models yielded poor goodness-of-fit indices and thus were not considered further. The personality vulnerability model and the reciprocal causality model provided acceptable model fit estimates [$\chi^2(35) = 56.67$, CFI = .97, RMSEA = .07 for the personality vulnerability model and $\chi^2(31) = 60.33$, CFI = .96, RMSEA = .09 for the reciprocal causality model]. In the reciprocal causality

model, the cross-lag coefficients in which depression leads to personality change were nonsignificant.

Based on model fit indices and parameter significance, the personality vulnerability model was determined to be the best-fitting model. The personality vulnerability model indicated a significant unidirectional coupling from perfectionism to depression change throughout therapy (γ = .10 to .17, $p < .05$). A close examination of the meaning of this significant coupling revealed that patients with higher levels of initial perfectionism would experience less reduction in their depression scores throughout treatment, compared to patients with lower levels of initial perfectionism.

Finally, they examined the role of the therapeutic alliance as related to the bivariate coupled system between depression and perfectionism. The findings supported their hypothesis suggesting that the strength of the therapeutic alliance significantly predicted the rate of change in perfectionism and, in turn, perfectionism predicted the rate of change in depression throughout treatment. In this model, therapeutic alliance was included as a covariate that predicts the rate of change in perfectionism throughout the therapy process. The model yielded a good fit [$\chi^2(64) = 83.01$, CFI = .98, RMSEA = .04] and the effect of therapeutic alliance on latent change scores of perfectionism were significant ($b = -.90$, $p < .05$). Probing the effects of therapeutic alliance suggested that patients with higher therapeutic alliance showed greater decreases in perfectionism throughout treatment compared to patients with lower therapeutic alliance.

Therefore, the findings of this study supported the personality vulnerability model showing significant dynamic coupling between perfectionism and depression in which the level of perfectionism predicted the subsequent rate of change in depression throughout therapy. Furthermore, the results indicated that patient contribution to the therapeutic alliance had a significant indirect effect on depression symptom alleviation, in that a strong alliance was related to improvement in perfectionism vulnerability, which in turn, predicted the rate of change in depression symptoms throughout therapy. These findings demonstrate how latent change score analysis can contribute to obtaining a comprehensive understanding of the therapy process by evaluating indicators that capture the multidimensional nature of the therapeutic process.

GROWTH MIXTURE MODELING

The growth mixture model (Muthén & Shedden, 1999), a combination of the latent growth model and the finite mixture model, continues to be a favorite analytic technique for understanding intraindividual change and interindividual variability in intraindividual change. The growth mixture model adds to the types of interindividual variability in intraindividual change by allowing for multiple classes of individuals. That is, individuals can manifest different change trajectories because they belong to different classes or clusters of individuals and because of variability in change within a given class.

Another perspective on the growth mixture model is that the sample is drawn from multiple populations. That is, the traditional latent growth model assumes that the sample is drawn from a single population characterized by a single set of parameters (i.e., means, variances, and covariances) and if the sample was, in fact, drawn from multiple populations the latent growth model is inappropriate because sets of parameters are needed for each population. In many cases, researchers have a measured variable that may be able to characterize multiple populations. For example, a sample is often composed of males and females, which can be thought of as different populations. If a researcher were interested in testing whether there were differences between males and females in terms of their change trajectories, then the researcher could fit a multiple group growth model and statistically test whether or not the parameter estimates for males and females were invariant. Building upon this example, there may be multiple groups with different sets of parameter estimates, and the groups may not be well defined by a single measured variable. In these situations, it may be reasonable to rely on the observed change trajectories (the data) and, in essence, search for groups of individuals with different sets of parameter estimates.

Growth Mixture Model

The growth mixture model can be conceived as a multiple group growth model in which the grouping variable is latent or unobserved. In a similar vein to the goals of the multiple group growth models—describing differences in longitudinal change for defined subsets of individuals, researchers utilize the growth mixture model to describe differences in longitudinal change for subsamples of individuals who are not well defined in terms of observed or measured variables. In growth mixture modeling the grouping variable is, essentially, unobserved. Thus, the goal is to estimate or create a variable that maximally distinguishes individuals based upon their change trajectories.

The growth mixture model is one approach to estimate a categorical latent variable (latent class variable) in an

attempt to classify individuals into groups based upon their observed change trajectories along with user-defined constraints. Following latent class analysis, this classification is probabilistic because the grouping variable is unobserved, and following the multiple group growth model, the user specifies a series of equality constraints (e.g., on the factor loading matrix) to examine where differences between the latent classes are evident. The growth mixture model can be written as

$$\mathbf{Y}_i^{(c)} = \sum_{c=1}^{C} (\pi_i^{(c)} (\Lambda^{(c)} \mathbf{h}_i^{(c)} + \mathbf{u}_i^{(c)}))$$

$$\text{given } 0 \leq \pi_i^{(c)} \leq 1 \text{ and } \sum_{c=1}^{C} \pi_i^{(c)} = 1. \qquad (22.33)$$

where $\pi_i^{(c)}$ is the individual probability of being a member of class c, which is constrained to range between 0 and 1 and which the all of the class probabilities for individual n sum to 1. The matrices defining the growth model are superscripted by c to indicate the elements of those matrices can vary over latent classes as in the multiple group model. As in the multiple group growth model, we recommend a series of progressive models to evaluate where class differences emerge. Thus, a logical starting point is an invariance model, which is equivalent to the one-class model. In subsequent models, the latent variables means, latent variable covariances and residual variances, and pattern of change can be relaxed. Furthermore, within each of these types of models, multiple models are fit with a different number of classes. This combination of types of models with varying number of classes seeks to determine not only whether class differences emerge, but also where they emerge and how many classes are appropriate.

Determining an appropriate number of latent classes is not a straightforward process and multiple criteria have been recommended. This is because in growth mixture modeling and mixture models generally, there is not a deterministic set of rules to follow (see Ram & Grimm, 2009). The goal here is not necessarily to determine an optimal model for the data based on model fit, but to yield results that will replicate in a new sample. Mixture models specifically search to maximize differences and are therefore extremely data dependent, which can lead to results that are not generalizable. In the model fit comparison approach outlined below, our goal is to yield results that are defensible, viable, and replicable. We also note that statistical programs vary in the amount of fit indices reported. Mplus provides the most amount of fit information and we utilize this available information. To reiterate, the goal is to build a defensible judgment regarding the optimal number of latent classes using a combination of model results, substantive theory, and statistical fit information.

A first consideration when evaluating a given model is to carefully examine the model output to determine whether parameter estimates are all in bounds. Mixture models have a tendency to yield out of bounds parameter estimates, such as negative variances and correlations greater than 1, especially when models are fit with too many classes. Furthermore, the interpretability of the parameters for each latent class is critical for model selection. For example, multiple classes may exhibit nearly identical change trajectories or a class may show a highly unusual change pattern indicating a general lack of replicability. In such cases, these models should be set aside while models with fewer classes are considered carefully. This first consideration is sure to exclude several models.

The remaining models are then compared using comparative fit criteria, such as the Bayesian information criterion (BIC), Akaike information criterion (AIC), and sample size adjusted BIC. All of these fit criteria are calculated from the –2 log-likelihood and a penalty for the number of estimated parameters. The penalty for the AIC is twice the number of parameters and the penalty for the BIC is the natural log of the sample size multiplied by the number of parameters. The sample size adjusted BIC uses ad adjusted sample size in an attempt to reduce the impact of sample size when examining model fit. For all fit criteria, lower values indicate better model fit. Thus, models with lower information criteria more closely represent the data; however, it is important to recognize that these criteria are sensitive to sample size and often favor more parameterized models. The principle that we forward is simply that models that do not fit better than the one-class (latent growth) model can be dismissed and that plotting these criteria against the number of classes (akin to a scree plot) can be useful in making relative comparisons regarding improvement in fit.

The next consideration when comparing models is classification quality, which is based on the confidence with which individuals have been classified in to their respective class. *Entropy*, a statistic that ranges from 0.00 to 1.00 (as implemented in Mplus), is a commonly used global statistic for classification quality. Entropy is based on the conditional probabilities of individuals' class membership (Jedidi, Ramaswamy, & Desarbo, 1993). Higher entropy values indicate a greater confidence in the classifications and that there is adequate separation between the latent classes. Often, a lower bound on entropy is imposed (e.g., 0.80) and models with entropy values below the cut-off

are viewed with skepticism or outright discarded. Overall, we note that models with higher entropy are favored when selecting from models with similar levels of model fit, based on the available information criteria. Additionally, we recommend a thorough examination of the average latent class probabilities, specifically for individuals who are likely to be in the same class. Again, a lower bound is often considered, such as 0.80, which would indicate that for individuals who are likely to be in a given class, there is a 0.80 average probability of being in the class.

The final piece of model fit and model comparison information comes from an array of *likelihood ratio tests* (LRT), which can be used to specifically compare two models that only differ in the number of latent classes. Specifically, these approximate test statistics can be used to compare a given model of interest to a model with one fewer class. The LRTs available in Mplus include the Vuong-Lo-Mendell-Rubin likelihood ratio test (VLMR-LRT), the adjusted Lo-Mendell-Rubin likelihood ratio test (adjusted LRT), and the bootstrap likelihood ratio test. The VLMR-LRT and adjusted LRT apply a corrected likelihood-ratio distribution to compare the model with C classes to the model with $C - 1$ classes (see Lo, Mendell, & Rubin, 2001; Muthén, 2004; Nylund, Asparouhov, & Muthén, 2007; Vuong, 1989). The bootstrap LRT examines the change in likelihood values for the model with C classes and the model with $C - 1$ classes compared to bootstrapped distribution where the model with $C - 1$ classes is the true model. All LRTs provide a p-value for the significance test. A low p-value ($p < .05$) indicates the model with $C - 1$ classes should be rejected in favor of the model with C classes. Again, we note that these LRTs are only to compare models that differ only in the number of classes and cannot be used for comparing models with between-class constraint.

Generally, researchers should pay attention to and report all of the available fit indices, relevant theory, and past research findings when selecting the growth mixture model that provides the most reasonable representation of the data (see also Masyn, 2009). These recommendations provide a practical guide to make sense of all of the available information when deciding on the number of latent classes.

Cautions

Growth mixture models have been used extensively over the past decade and have been able to highlight multiple subpopulations in the development of several constructs, including achievement, behavior problems, and substance use. However, growth mixture models have not been without their limitations and naysayers. Here, we overview some of these potential issues.

First, we note that growth mixture models are exploratory and data driven and with any exploratory model, the true value is replicability. Thus, any single set of results based upon a single data set must be taken cautiously. Second, as highlighted by Bauer and Curran (2003), growth mixture models, and mixture models more generally, can accommodate and represent nonnormality in observed data. That is, if data are nonnormal, then it is likely that a multiple class mixture model will provide a better fit than a single class model. The issue is that nonnormality may indicate a true mixture distribution, but it may simply be caused by measurement anomalies, such as artificial floor and ceiling effects, making inappropriate distributional assumptions (e.g., data come from a Poisson distribution but are treated as being normally distributed), and poor measurement instruments or the misuse of a measurement instrument. Given these possibilities, results from a mixture model can be misinterpreted and there is a real danger in identifying false groups (see Bauer & Curran, 2003, 2004).

Growth Mixture Modeling Studies for Understanding Risk and Protective Mechanisms as Predictors, Mediators, and Moderators

Growth Mixture Modeling Studies for Understanding the Influences of Risk and Protective Mechanisms Using Cross-Population Comparisons

The following study illustrates how growth mixture modeling can be used to examine continuity and discontinuity in developmental trajectories of maladjustment and further investigate risk and protective mechanisms that predict different patterns of developmental trajectories based on cross-population comparisons. Kim, Cicchetti, Rogosch, and Manly (2009) investigated the longitudinal impact of maltreatment parameters on personality processes and maladjustment and prospective relationships between personality trajectory classes and subsequent maladjustment outcomes. The main goal was to study developmental processes contributing to dysfunction and resilience (i.e., multifinality) to better understand pathways to adaptive and maladaptive development of personality and related psychopathology. Therefore, in this study we investigated different psychopathological outcomes (internalizing and externalizing symptomatology) as well as multiple processes eventuating in psychopathological outcomes (ego control and ego resiliency). Given the extreme failure

of the average expectable environment that is associated with child maltreatment, it was expected that maltreated children would manifest maladaptive types of personality processes that include low ego resiliency and tendencies toward ego undercontrol.

Participants included 449 children (249 maltreated and 200 nonmaltreated) who attended a summer day camp research program in a Northeastern city. Children ranged in age from 6 to 9 years ($M = 6.76$, $SD = 1.03$) when they first participated in the camp and they were followed longitudinally between ages 6 and 10. Sixty-three percent of the children were boys, and children were from diverse ethnic backgrounds: 61.2% African American, 23.2% European American, 12.7% Latino, and 2.9% other ethnic groups. The narrative reports of the maltreatment incidents from the Department of Social Services (DSS) records were coded according to the MCS (Barnett et al., 1993). Children's externalizing and internalizing behavior was assessed through completion of the TRF (Achenbach, 1991). Camp counselors' ratings of children's ego resiliency and ego control were obtained using the California Child Q-Set (CCQ; Block & Block, 1980). Ego resiliency was defined as the relative flexibility of impulse control that enables effective regulation of affect and behavior. Ego control was defined as the ability to monitor and modulate impulses, with ego over-control being characterized by excessive inhibition of affect and impulses, whereas ego under-control being characterized by insufficient modulation, including poor frustration tolerance and poor attention seeking (Block & Block, 1980).

Consistent with a person-centered approach to data analysis that emphasizes individual differences in development, we used growth mixture modeling. Three criteria were used to determine the optimal numbers of classes. First, we used a sample-size adjusted Bayesian information criterion (SABIC; Sclove, 1987) that is a parsimony goodness-of-fit index based on the log-likelihood adjusted for the number of parameters. Lower SABIC values reflect a better model fit. Second, we used the adjusted Lo-Mendell-Rubin test (ALMR; Lo, Mendell, & Rubin, 2001) that is a nested model likelihood ratio test and provides a significant test of whether C trajectory classes provided a better fit to the data than did the $C - 1$ class model. Thus, a small probability value (i.e., $p < .05$) indicates that the $C - 1$ class model should be rejected in favor of the model with at least C classes. Finally, we also checked the usefulness of the classes based on the subjective interpretation of the developmental trajectories and the number of children in each class. As shown in Table 22.3, we obtained fit indices for one-, two-, three-, and four-class models of developmental trajectories. In these models, the latent variables means, latent variable covariances and residual variances, and pattern of change were relaxed. In identifying the growth mixture model that best delineates the data, we chose C class model over $C - 1$ model when both the SABIC (i.e. decreased) and the ALMR (i.e., $p < .05$) indicated a better fit for the C class model compared to the $C - 1$ class model.

TABLE 22.3 Fit Indices for Univariate Growth Mixture Modeling of Personality Processes and Behavioral Problems in Maltreated and Nonmaltreated Children[1]

	Maltreated		Nonmaltreated	
Number of classes	SABIC	ALMR	SABIC	ALMR
Ego-resiliency				
1 class	279.68		**127.82**	
2 classes	**261.64**	15.68[2]	107.93	27.13
3 classes	258.58	11.66	103.10	12.75
4 classes	261.87	5.63	88.73	21.86
Ego-control				
1 class	**−330.70**		−279.15	
2 classes	−330.43	8.73	−290.78	19.24[1]
3 classes	−335.67	13.990	**−296.52**	13.62[1]0
4 classes	−338.08	11.280	−294.02	5.740
Externalizing symptomatology				
1 class	**6469.65**		**4732.05**	
2 classes	6469.57	99.060	4704.46	34.49
3 classes	6472.24	6.43	4696.19	016.030
4 classes	6475.33	6.03	4689.63	14.40
Internalizing symptomatology				
1 class	**6383.36**		**4718.05**	
2 classes	6382.25	10.04[1]	4715.69	10.38
3 classes	6381.21	07.78[1]	4712.44	10.75
4 classes	6385.12	05.240	4709.28	011.160

[1] SABIC = Sample-size adjusted BIC; ALMR = Adjusted LMR. Bold face indicates best-fitting models.
[2] $p < .05$.

A two-class model yielded the best fit to the ego resiliency data for maltreated children. As shown in Figure 22.10, the two trajectories started at similar levels, but 59% of children (Class 2, the increasing class) were characterized by a U shape trajectory with decreasing up to age 7 and then continuously increasing afterwards, whereas 41% of children (Class 1, the "declining" class) showed significant decreases in slopes. Turning to ego control, the best-fitting model of the one-class growth mixture model indicated stable and somewhat elevated levels of ego undercontrol for the entire sample of maltreated children, as indicated by the significant intercept mean and the nonsignificant means of the linear and quadratic slopes. Meanwhile, growth mixture modeling analyses revealed three distinct growth trajectories for nonmaltreated children. As shown in Figure 22.11, about half of the nonmaltreated children (53%, Class 2, the low-stable

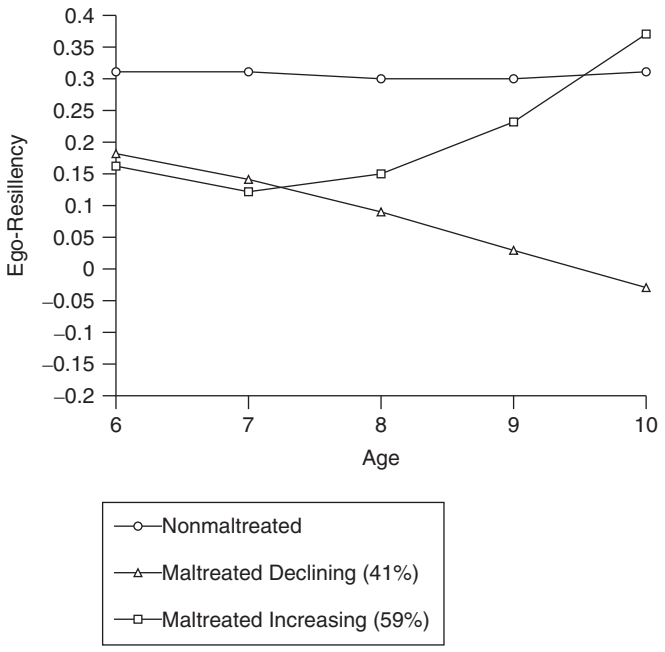


Figure 22.10 Estimated mean growth trajectories of ego-resiliency. *Source*: Note: Adapted from "Child maltreatment and trajectories of personality and behavioral functioning: Implications for the development of personality disorder" by J. Kim-Spoon, D. Cicchetti, F. A. Rogosch, and J. T. Manly, 2009, *Development and Psychopathology*, *21*, pp. 889–912.

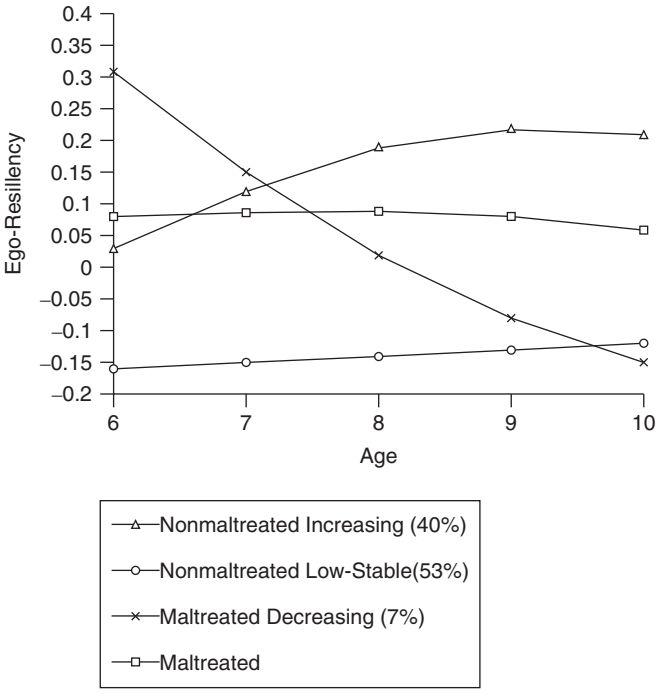


Figure 22.11 Estimated mean growth trajectories of ego-control. *Source*: Adapted from "Child maltreatment and trajectories of personality and behavioral functioning: Implications for the development of personality disorder" by J. Kim-Spoon, D. Cicchetti, F. A. Rogosch, and J. T. Manly, 2009, *Development and Psychopathology*, *21*, pp. 889–912.

class) showed consistently low levels of ego undercontrol (with nonsignificant means for linear and quadratic slopes) and about 40% of children (Class 1, the increasing class) showed a U shaped slope with significant increases. A small number of nonmaltreated children (7%, Class 3, the declining class) showed a higher initial level than the other two classes, and they displayed a decreasing linear trend that was marginally significant ($p = .06$).

After determining the most appropriate number of trajectory classes, we then tested whether latent class differences could be described by maltreatment parameters and gender using a multinomial logistic regression approach for the variables with more than one class and conditional growth curve models for the variables with one class. The main interest was to examine whether different features of the maltreatment experience—such as type, multiplicity (i.e., experiencing multiple subtypes), onset, and chronicity—may have significant independent effects on child outcomes. For these analyses, two models were estimated separately for testing effects of physical or sexual abuse (PA/SA) and for testing the effects of other maltreatment risk factors including multiple subtypes, chronicity, and onset so that we could prevent potential multicollinearity problems. Gender was included in the model as a covariate.

For ego resiliency among maltreated children, the logistic regression coefficient (or logit) predicting membership in the declining class indicated that children with early onset, regardless of the maltreatment subtypes, were more likely to be in the decreasing class with a marginal significance ($b = .69$, $SE = .37$, $p = .07$, *odd ratio* = 2.00). The growth curve model analyses with maltreatment subtypes and gender for ego control produced reasonable fits [$\chi^2\,(15) = 18.69$ and RMSEA = .03 for the model involving the PA/SA predictor, and $\chi^2\,(19) = 23.49$ and RMSEA = .03 for the model involving multiple subtypes, chronicity, and onset]. Experiencing multiple maltreatment subtypes and physical/sexual abuse were related to higher levels of ego undercontrol.

The final step of data analysis validated the distinctiveness of the trajectory classes by testing whether the personality trajectory classes predicted later behavioral adjustment. Our growth mixture modeling results indicated two trajectory classes of ego resiliency among maltreated children and three trajectory classes of ego control among nonmaltreated children. We examined whether those trajectory classes based on personality scores during ages

6–10 years would predict adjustment outcomes observed during ages 10–12 years using ANOVA.

We compared the prospective adjustment outcomes among the five classes that were identified in the current sample, including the declining ego resiliency and the increasing ego resiliency classes among maltreated children, and the increasing ego (under)control, the low-stable ego (under)control, and the declining ego (under)control classes among nonmaltreated children. Significant group differences in externalizing symptomatology were identified by Tukey range tests and linear contrasts, indicating that (1) the maltreated declining ego resiliency class showed the highest levels of externalizing symptomatology, and the nonmaltreated increasing ego (under)control class had significantly higher levels of externalizing symptomatology compared to the maltreated increasing ego resiliency class and the nonmaltreated low-stable ego (under)control class, and (2) the nonmaltreated declining ego (under)control class and the maltreated increasing ego resiliency class showed higher levels of externalizing symptomatology than the nonmaltreated low-stable ego (under)control class, which showed the lowest levels of externalizing symptomatology [Tukey range tests, $F\,(4,\ 333) = 26.64$, $p < .05$; $p < .05$ for all comparisons, except the linear contrast for the nonmaltreated declining ego (under)control class vs. the nonmaltreated low-stable ego (under)control class, $t = -1.71$, $p = .09$]. Turning to internalizing symptomatology, Tukey range tests and linear contrasts revealed that the maltreated declining ego resiliency class and the nonmaltreated declining ego (under)control class showed significantly higher levels of internalizing symptomatology compared to the other three classes [Tuckey range tests, $F\,(4,\ 333) = 10.31$, $p < .05$; and linear contrasts, $t = -2.01$ ˜ $t = -2.24$, $p < .05$].

Thus, our examination of subsequent adjustment outcomes among the five subgroups (consisting of the two ego resiliency classes for maltreated children and the three ego control classes for nonmaltreated children) provided a clearer picture regarding joint contribution of ego resiliency and ego control to internalizing and externalizing symptomatology. Maltreated children in the declining ego resiliency group showed the worst outcomes with distinctively higher levels of both externalizing and internalizing symptomatology. In contrast, maltreated children in the increasing ego resiliency group exhibited better adjustment functioning as indicated by their relatively low levels of externalizing and internalizing symptomatology. Overall, the findings suggest that ego resiliency and ego control—personality processes related to self-regulation—may be important factors in identifying distinct pathways to later personality disorders as well as pathways to resilient functioning.

Traditionally, research on developmental psychopathology has taken predominantly variable-centered approaches to examine how variables differ on average between groups. In contrast, person-centered approaches to developmental psychopathology emphasize the importance of the person as the major unit of analysis and the structure of person profiles across variables. The integrated variable- and person-centered approach provides increased insights about the relationships between risk and protective factors and developmental trajectories of psychopathology. The findings of this study illustrate the potential utility of a growth mixture modeling approach to examining the patterns of developmental trajectories and further highlight that developmental psychopathology researchers should consider that possibility that there are heterogeneous trajectory classes even when class membership is not observed.

Growth Mixture Modeling Studies for Understanding Contextual Influences

Growth Mixture Modeling for Cross-Domain Relations in Risk and Psychopathology

The following longitudinal study illustrates how growth mixture modeling can be used to study cross-domain relations in psychopathology. In a longitudinal study, Reinke, Eddy, Dishion, and Reid (2012) utilized growth mixture modeling to identify patterns of developmental changes of disruptive behaviors and depression and further examine the joint, longitudinal trajectories of symptoms of disruptive behaviors and depression. Participants included 361 adolescents (49% male, 15% non-White) who were recruited from at-risk neighborhoods based on high rates of police contact regarding youth delinquency. Adolescent depressive symptoms and disruptive behavior problems were assessed six times from age 10 to 16 years. Depressive symptoms were assessed using adolescents' reports on the Child Depression Inventory (CDI; Kovacs, 1992) and disruptive behavior problems were assessed using mothers' reports on the Child Behavior Checklist (CBCL; Achenbach, 1991). Distal outcomes during emerging adulthood (age 18 to 23 years) included youth reports of disruptive behavior problems using the Youth Self-Report (YSR: Achenbach, 1991), depressive symptoms using the Brief Symptom Inventory (BSI: Derogatis, 1975), substance use using the Michigan Alcoholism Screening Test (MAST; Selzer, 1971) and the Drug Abuse Screening Test (DAST; Skinner 1982), diagnosis of antisocial personality disorder (ASPD) and major depressive disorder (MDD)

using Diagnostic Interview Schedule (DIS-IV, American Psychological Association, 1994), and police arrests (juvenile and adult arrests) using court data.

Univariate growth mixture modeling analyses were performed to identify distinct subgroups based on patterns of growth in depressive symptoms and disruptive behavior problems. Four indicators were used to determine the best-fitting growth mixture model including the Akaike information criterion (AIC; Akaike, 1987), the Bayesian information criterion (BIC; Schwartz, 1978), and the sample-size adjusted (SABIC; Sclove, 1987), as well as the entropy. For depression, comparing between the four-class and five-class solutions, the addition of a fifth class did not provide unique information (AIC = 10998.25, BIC = 11113.99, SABIC = 11018.82, Entropy = .93 for the four-class solution; and *AIC* = 10957.13, BIC = 11088.30, SABIC = 10980.44, entropy = .92 for the five-class solution). Thus, the four-class model was chosen as the best-fitting model. The four classes included a group showing increases in depression ($n = 18$, 5.1%), a group showing high levels of depression ($n = 18$, 5.2%), a group showing decreases in depression ($n = 31$, 8.9%), and a group showing low levels of depression ($n = 283$, 80.9%). Latent class regression analysis further indicated that adolescents in the increasing and decreasing depression classes were significantly more likely to report depression as young adults (odds ratio = 18.70 and 4.27, respectively) compared to the adolescents in the low depression class. Furthermore, compared to the low depression class, adolescents in the increasing depression class were significantly more likely to report disruptive behavior as young adults (odds ratio = 18.70). Adolescents in the high depression class were significantly more likely to report problems with alcohol (odds ratio = 16.39) and drugs (odds ratio = 4.06).

For disruptive behavior problems, comparing between the three-class and four-class solutions, the addition of a fourth class did not provide unique information (AIC = 10547.22, BIC = 10627.13, SABIC = 10560.52, Entropy = .96 for the three-class solution; and AIC = 10510.69, BIC = 10602.01, SABIC = 10525.89, Entropy = .96 for the four-class solution). Thus, the three-class model was chosen as the best-fitting model. The three classes included a group showing increases in disruptive behavior problems ($n = 19$, 5.9%), a group showing high levels of disruptive behavior problems ($n = 11$, 3.2%), and a group showing consistently low levels of disruptive behavior problems ($n = 302$, 90.9%). Latent class regression analysis further indicated that adolescents in the increasing and high disruptive behavior problems classes were significantly more likely to report disruptive behavior problems as young adults (odds ratio = 2.85 and 3.28, respectively). In addition, adolescents in the increasing disruptive behavior class, when compared to the low disruptive behavior class, were significantly more likely to report depression as young adults (odds ratio = 6.59), to be arrested as juveniles (odds ratio = 7.85), and to report problems with alcohol as young adults (odds ratio = 2.89). Adolescents in the high disruptive behavior class, compared to the low disruptive behavior class, were significantly more likely to report problems with alcohol (odds ratio = 2.19) and drugs (odds ratio = 5.04), and to be arrested (odds ratio = 10.32) as young adults.

Furthermore, parallel process growth mixture modeling was used to integrate the trajectories of disruptive behavior and depressive symptoms into one model and investigate the co-occurrence of disruptive behavior problems and depression. Specifically, parallel process growth mixture models evaluated the joint probability of individuals simultaneously being in a class for each of the two growth classes as well as the conditional probability of membership in each disruptive behavior class conditional on each depression class, and vice versa. Among adolescents in the high depression class, 11% were in the high disruptive behavior class and 22% were in the increasing disruptive behavior class. In contrast, among adolescents in the low depression class, only 5% were in the high disruptive behavior class and 3 % were in the increasing disruptive behavior class. Among adolescents in the high disruptive behavior class, 5% were in the high depression class and 16% were in the decreasing depression class. Among adolescents in the increasing disruptive behavior class, 15% were in the high depression class and 8% were in the decreasing depression class. In comparison, among adolescents in the low disruptive behavior class, 2% were in the high depression class and 5% were in the decreasing depression class. Thus, these findings indicate that adolescents with high depression may be at greater risk for co-occurring disruptive behavior problems and also that adolescents with high or increasing disruptive behavior problems may be at greater risk for co-occurring depression.

Findings from this study highlight the utility of person-centered approaches for the study of developmental courses across different dimensions of psychopathology. Using the growth mixture modeling can be particularly beneficial in providing crucial information regarding comorbidity—that was identified based on developmental trajectories—as a source of heterogeneity. These subtypes may represent distinct subgroups that should be

differentially targeted in terms of preventive intervention efforts.

Growth Mixture Modeling for Multiple Levels of Analysis

The following study illustrates how growth mixture modeling procedure can be utilized to examine the developmental significance of early individual risk factors on subsequent risk for maladjustment outcomes. Using longitudinal data across several domains and multiple developmental time points, Lynne-Landsman, Bradshaw, and Ialongo (2010) evaluated a dynamic cascade model of substance use by examining the indirect effects of early risk factors on substance use during adolescence and adjustment in adulthood. Specifically, they examined the process by which early-onset aggressive behavior problems and academic readiness influence risk for substance use during adolescence and further adjustment problems during young adulthood.

Data were drawn from a sample of 678 urban, primarily African American youth (87% African American and 13% Caucasian; 53% male) followed from first grade through age 21. Early risk factors included parental discipline and parental rejection, oppositional defiant behavior, academic failure, peer rejection, and affiliation with substance-using peers. Parental discipline and parental rejection in first grade were assessed using The Structured Interview of Parent Management Skills and Practices, Parent Version (Capaldi & Patterson, 1989). Oppositional defiant behavior and peer rejection in first and third grades were assessed using the Teacher Observation of Classroom Adaptation—Revised (Werthamer-Larsson, Kellam, & Wheeler, 1991). Academic achievement in first, third, and eighth grades was measured using teachers' reports on the child's overall grade performance. Substance-using friend affiliation in seventh grade was assessed using adolescents' reports on the friends drug use subscale from the Monitoring the Future National Survey (Johnston, O'Malley, & Bachman, 1995). Teachers provided data on adolescent conduct problems via the Classroom Behavior Checklist. Frequency of substance use in the last year (cigarettes, alcohol, and marijuana) was measured using adolescents' reports on the questions from the Monitoring the Future National Survey (Johnston et al., 1995). Adjustment in young adulthood was indicated using five variables measured at age 21 including educational attainment (e.g., graduating high school on time), mental health problems (life-time major depressive disorder and antisocial personality disorder), drug abuse and dependence, risky sexual behaviors (unsafe sexual behaviors and pregnancy), and criminal behavior (court records of involvement in the juvenile and adult criminal justice systems).

They first conducted a series of growth mixture modeling analyses to model developmental trajectories of cigarette, alcohol, and marijuana use in high school (from ninth to twelfth grades). For cigarette, a two-class model provided the best fit to the data suggesting a group of abstainers ($n = 437$, 82%) and a group of adolescents with "ncreasing cigarette use ($n = 96$, 18%). For alcohol use, a three-class model was the best-fitting model suggesting three distinct trajectories of none/low alcohol use ($n = 583$, 83%), "moderate" alcohol use ($n = 88$, 13%), and heavy alcohol use ($n = 28$, 4%). Similarly, for marijuana use, a three-class model was the best-fitting model. The three classes represented distinct groups of abstainers ($n = 437$, 82%), increasing marijuana users ($n = 58$, 11%), and heavy marijuana users ($n = 36$, 7%).

Next, predictors and distal outcomes that were associated with the high school substance use trajectories were examined. For adolescent cigarette use, parental education ($b = .20$, $SE = .08$, $p = .02$) and female gender ($b = .48$, $SE = .24$, $p = .04$) were associated with significant increases in the odds of being in the abstainers group. In terms of the distal outcomes of adjustment in young adulthood, those who belonged to the increasing class, compared to abstainers, were less likely to graduate on time ($\chi^2 = 26$, $p < .001$), and were more likely to report poor mental health ($\chi^2 = 8.66$, $p < .01$), alcohol abuse ($\chi^2 = 7.20$, $p < .01$), marijuana dependence ($\chi^2 = 16.38$, $p < .001$), unsafe sexual activities ($\chi^2 = 26$, $p < .001$), and involvement in criminal justice system ($\chi^2 = 19.61$, $p < .001$).

For adolescent alcohol use, parental education and gender were not significant predictors of trajectory classes. In terms of the distal outcomes of adjustment in young adulthood, the only adult outcome that was differentiated by the trajectory classes was antisocial personality disorder. Those who belonged to the moderate use class, compared to the no/low use class, were more likely to have antisocial personality disorder ($\chi^2 = 6.42$, $p < .01$). Turning to adolescent marijuana use, parental education differentiated the heavy use class from the increasing class ($b = .32$, $SE = .14$, $p = .02$) and the abstaining class ($b = .31$, $SE = .11$, $p = .003$). Female gender ($b = .77$, $SE = .31$, $p = .01$) were associated with significant increases in the odds of being in the abstaining class compared to the increasing class. With respect to the distal outcomes of adjustment in young adulthood, compared to abstainers, those who belonged to the increasing class and heavy use class were less likely to graduate high school

on time ($\chi^2 = 17.87$, $p < .001$), and were more likely to show antisocial personality disorder ($\chi^2 = 15.54$, $p < .01$), alcohol abuse ($\chi^2 = 18.12$, $p < .001$), marijuana abuse and dependence ($\chi^2 = 14.79$, $p < .001$ and $\chi^2 = 21.63$, $p < .001$, respectively), unsafe sexual activities ($\chi^2 = 7.06$, $p < .05$), and involvement in the criminal justice system ($\chi^2 = 16.21$, $p < .001$).

Finally, the developmental cascade model was tested to determine if the effect of each childhood/adolescent predictor on substance use class membership was mediated by the subsequent predictors along the cascade. The indirect effects were calculated by the product of the two pathways: of the pathway from each predictor at time t to a subsequent predictor at time $t + 1$ (α) and the pathway from each predictor to substance use class membership (β). For cigarette use, the indirect effect of oppositional defiant behavior in first grade on high school cigarette use class membership was mediated by peer rejection in third grade ($\alpha\beta = .04$, $SE = .02$, $p = .05$). Both peer rejection and academic achievement in third grade had indirect effects on high school cigarette use class membership via affiliation with substance-using peers ($\alpha\beta = .05$, $SE = .02$, $p = .04$ and $\alpha\beta = .05$, $SE = .03$, $p = .04$, respectively). In turn, substance-use peer affiliation was indirectly associated with high school cigarette use class membership through conduct problems in eighth grade ($\alpha\beta = .11$, $SE = .04$, $p = .002$).

For alcohol use, both peer rejection and academic achievement in third grade had indirect effects on high school alcohol use class membership via affiliation with substance-using peers ($\alpha\beta = .06$, $SE = .03$, $p = .02$ and $\alpha\beta = .06$, $SE = .03$, $p = .02$, respectively). In addition, substance-use peer affiliation was associated with high school cigarette use class membership mediated through conduct problems in eighth grade ($\alpha\beta = .11$, $SE = .04$, $p = .001$). Turning to marijuana use, the indirect effect of oppositional defiant behavior in first grade on high school marijuana use class membership through peer rejection in third grade was marginally significant ($\alpha\beta = .05$, $SE = .03$, $p = .06$). Both peer rejection and academic achievement in third grade had indirect effects on high school marijuana use class membership via affiliation with substance-using peers ($\alpha\beta = .07$, $SE = .03$, $p = .04$ and $\alpha\beta = .07$, $SE = .03$, $p = .03$, respectively). Substance-use peer affiliation was associated with high school marijuana use class membership indirectly through conduct problems in eighth grade ($\alpha\beta = .13$, $SE = .04$, $p = .002$).

Thus, the results of testing a developmental cascade model highlight how growth mixture modeling analyses can be used not only to examine heterogeneity in developmental trajectories of psychopathology but also to identify multiple levels of pathways to psychopathology trajectories. The main finding indicated distinct developmental trajectories of high school cigarette and marijuana use that were explained by the peer rejection pathway (rather than the academic achievement pathway). That is, early childhood peer rejection was related to subsequent affiliation with deviant substance-using peers, which resulted in higher odds of following trajectories of using cigarette and marijuana. This study also underscores testing the validity of the groupings by examining difference in long-term outcomes across trajectory groups. However, future research will be beneficial to replicate results for further validation of the groups.

Implications of Growth Mixture Modeling for Prevention and Intervention Science

Studies using growth mixture modeling to assess prevention and intervention effects are extremely rare despite that Muthén and colleagues (2002) have published a nice example of applying growth mixture modeling for evaluating effects of a randomized preventive intervention program. Basically, using growth mixture modeling allows researchers to examine effects of prevention and intervention programs on subgroups that are characterized by different types of growth trajectories. Muthén and colleagues (2002) examined the effect of a randomized intervention, the Good Behavior Game, aimed at intervening during first and second grades to reduce aggressive behaviors that were assessed through middle school and beyond. Participants included 119 boys in the intervention group and 80 boys in the control group. The main question was to examine if subgroups of children benefit differently from the intervention. In growth mixture modeling, intervention effects were expected to be captured by the average slopes of each class.

First, separate analyses were performed for the control and the intervention group to study the normative development of aggressive behavior in the absence of an intervention shown in the control group compared to the intervention group. Model comparison for different numbers of classes was guided by a BIC. For the control group, the four-class solution was deemed to best describe the data and those four classes included high (8%), medium (38%), low (41%), and late-starters (13%). For the intervention group, the three- and four-class models revealed that the High class showed a decline earlier than the control group, suggesting a beneficial effect of the preventive intervention program especially for boys who were at the greatest risk.

Next, the joint analysis of the control and the intervention group, and both the three- and four-class models were compared. For the three-class model, beneficial effects of the intervention were shown with the lowered aggression trajectory for the high class of the intervention group, compared with the high class of the control group. It should be noted that, although the difference in the means was sizable ($\gamma = -.45$; with γ indicating a change in average growth rate that can be different for different classes), it was not significantly different from zero (95% CI : $-.92$, 0.02). There was no indication of intervention effects for the Medium and Low classes, suggesting that the intervention effects were more effective for children with the highest risk. Similar patterns for the intervention effects on the High group were observed in the 4-class model. Beneficial effects of the intervention were also suggested for the Late-starter group with late-starters who received intervention showing a flatter trajectory compared to late-starters in the control group. However, the likelihood-ratio test of any intervention effect over the four classes was very small ($\chi^2(8) = 1.40$, $p > .05$). The authors attributed the lack of significance to the low power given small class sizes (e.g., 18 boys in the intervention group and 12 boys in the control group) as well as large within-class variation.

This study illustrates how growth mixture modeling can be employed to examine different intervention effects in the different subgroups as well as to investigate when those effects are likely to appear. Furthermore, as Muthén and colleagues (2002) suggested, the results of this study imply future directions that researchers may select different types of interventions for individuals belonging to different trajectory subgroups using longitudinal screening procedures.

CONCLUSION AND FUTURE PERSPECTIVE

The main goal of this chapter was to demonstrate currently existing latent growth modeling approaches, examine implications of these approaches for testing developmental psychopathology theory, and illustrate examples of using these approaches to enhance our understanding of risk, disorder, and adaptation focusing on the developmental psychopathology principles. Investigators interested in developmental psychopathology will continue benefiting from the advancement of latent growth modeling methodology. This is especially because the understanding of individuals with high-risk conditions and psychopathological disorders will require careful examination of interindividual differences in intraindividual changes based multidomain and prospective assessments. Latent growth modeling provides a tool to delineate continuity/discontinuity and turning points in developmental pathways. Furthermore, the recent development of growth mixture modeling allows researchers to model multiplicity of processes and outcomes in the development of adaption, maladaptation, and psychopathology, and thus better appreciate equifinality and multifinality in the developmental pathways.

Growth curve models are the current standard approach to study within-person changes over time and have many benefits over classical approaches to repeated-measures data because of their emphasis on *individual* change and the ability to statistically quantify the magnitude of between-person differences in the magnitude of within-person change. At the same time, advances in the conceptualization of individual change to examine time-dependent effects on change, nonlinear change, and a greater emphasis on the individual have called into question the utility of the growth curve model. We expect such movements to continue, highlight these trends, and discuss potential avenues for the expansion of growth curve models.

With the work of McArdle and Hamagami (2001), the conceptualization of individual change as examined with growth curve models was transformed from a deterministic model (a smooth trend characterized by few parameters plus noise) to a stochastic one where dynamic noise (separate from residual variation or measurement noise) could be estimated and impact the predicted trajectories over time. Additionally, time-lagged associations between multiple on-going processes could be studied, such that change at a given time point was potentially impacted by prior states within and across developmental processes. Theoretically, this makes sense as it is unlikely that a smooth developmental trajectory would not be perturbed by events taking place at the individual level and changes are often dependent on the prior and/or current states and rates of change in multiple constructs. We expect continued movement in the direction of stochastic, rather than deterministic, models.

This movement is coinciding with changes taking place in the ways in which longitudinal data are being collected. Researchers are increadingly relying on cell phones to collect data as participants live their lives. The enormous amount of data streams provided by cell phones and the accessibility of participants to researcher invoked ongoing interventions has led to statistical challenges in the analysis of such data. Such data are often analyzed on an individual-by-individual basis where the aggregation

of results can be difficult because of the idiosyncracy of people's daily lives. However, the data streams can be analyzed and interventions can be timed based on the data streams. Such individually timed interventions can reduce between-person differences in change and status prior to intervention, and the impacts of the intervention can be realized with growth curve type models since the aggregation of information across individuals is more easily justified.

In closing, we identify future research directions for developmental psychopathology research that will benefit from employing latent growth modeling approaches. First, with respect to the emergence of psychopathology, all periods of life are consequential in the sense that the developmental process can take a pernicious turn toward psychopathology at any phase (Cicchetti & Rogosch, 1999). More investigations are needed that elucidates developmental trajectories across the life course (e.g., infancy through senescence). Second, our examination of published illustrations suggests that prior research using latent growth modeling predominantly focused on the psychopathology outcomes of internalizing and externalizing symptomatology. It is recommended that latent growth modeling approaches are applied to broader high-risk conditions and mental disorders that have traditionally been investigated in a nondevelopmental fashion, including Down syndrome, fragile X, autism, schizophrenic disorder, bipolar disorder, and borderline personality disorder. In particular, studying developmental trajectories across different populations will help us learn about what is specific to a given risk condition as well as what characterizes a number of conditions (i.e., similarities vs. differences). Such cross-population studies should engage not only processes linked to pathological outcomes but also pathways to resilience and the effects of intervention and prevention.

Third, using latent growth modeling for future research that involves multiple domains of psychopathology will be particularly fruitful to better understand the concept of comorbidity from the perspective of developmental psychopathology. Such studies will better inform us regarding how we think about comorbidity, how we analyze comorbidity, how much is due to differentiation or truly unique disorders, and if differential pathways exist for comorbid symptoms. Fourth, to better capture the nature of transactional developmental processes, future investigations using latent growth modeling must strive to apply a multilevel developmental psychopathology perspective (Cicchetti, 2010) to attain genetic–neurobiology–psychology trajectories within individuals as well as culture, community, family, and individual trajectories. It has been a decade since Curtis and Cicchetti (2003) emphasized the need for research that applies a multiple-levels-of-analysis perspective to evaluate the efficacy of preventive interventions. As efficacious interventions are expected to change both behavior and biology through alterations in gene expression as well as changes in the brain (Kandel, 1999), deeper understanding for the mechanism of change will be obtained through investigating the change processes in multiple levels of functioning.

Finally, from an analytic point of view, continuous time models (Oud & Jansen, 2000) and the use of differential equation models will become more common, partially brought on by the increase in the number of assessments. Such models more easily account for unequally spaced measurement occasions and allow for the comparison of parameter estimates across studies that are based on different time lags by relating typically utilized discrete time models to their underlying models in continuous time. We expect continued improvements in the estimation of complex nonlinear and growth mixture models as well as approaches to research synthesis in longitudinal studies of change to accumulate information over studies and datasets.

REFERENCES

Achenbach, T. M. (1991). *Integrative guide for the 1991 CBCL/4–18, YSR, and TRF profiles*. Burlington, VT: Department of Psychiatry, University of Vermont.

Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*, 317–332. doi: 10.1007/bf02294359

American Psychological Association. (1994). *Diagnostic and statistical manual of mental disorder* (4th ed.). Washington, DC: American Psychiatric Association.

Arbuckle, J. L. (1996). Full information estimation in the presence of incomplete data. In G. A. Marcoulides & R. E. Schumacker (Eds.), *Advanced structural equation modeling: Issues and techniques* (pp. 243–277). Mahwah, NJ: Erlbaum.

Baker, P. C., Keck, C. K., Mott, F. L., & Quinlan, S. V. (1993). *NLSY child handbook. A guide to the 1986–1990 national longitudinal survey of youth child data* (revised ed.). Columbus, OH: Ohio State University.

Baltes, P. B., & Nesselroade, J. R. (1979). History and rationales for longitudinal research. In J. R. Nesselroade & P. B. Baltes (Eds.), *Longitudinal research in the study of behavior and development* (pp. 1–39). New York, NY: Academic Press.

Barnett, D., Manly, J. T., & Cicchetti, D. (1993). Defining child maltreatment: The interface between policy and research. In D. Cicchetti and S. L. Toth (Eds.), *Child abuse, child development, and social policy* (pp. 7–73). Norwood, NJ: Ablex. doi: 10.1016/j.chiabu.2008.12.002

Bauer, D. J., & Curran, P. J. (2003). Distributional assumptions of growth mixture models: Implications for overextraction of latent trajectory classes. *Psychological Methods*, *8*, 338–363. doi: 10.1037/1082-989X.8.3.338

Bauer, D. J., & Curran, P. J. (2004). The integration of continuous and discrete latent variable models: Potential problems and promising opportunities. *Psychological Methods*, *9*, 3–29. doi: 10.1037/1082-989X.9.1.3

Bell, R. Q. (1954). An experimental test of the accelerated longitudinal approach. *Child Development*, *25*, 281–286. doi: 10.2307/1126058

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, *107*, 238–246. doi: 10.1037/0033-2909.107.2.238

Berkey, C. S. (1982). Bayesian approach for a nonlinear growth model. *Biometrics*, *38*, 953–961. doi: 10.2307/2529875

Block, J., & Block, J. H. (1980). *The California Child Q-Set*. Palo Alto, CA: Consulting Psychologists Press. (Original work published 1969.)

Bogg, T., & Finn, P. R. (2010). A self-regulatory model of behavioral disinhibition in late adolescence: Integrating personality traits, externalizing psychopathology, and cognitive capacity. *Journal of Personality*, *78*, 441–469. doi: 10.1111/j.1467-6494.2010.00622.x

Boker, S. M., Neale, M. C., & Rausch, J. R. (2004) Latent differential equation modeling with multivariate multi-occasion indicators. In K. van Montfort, H. Oud, & A. Satorra (Eds.), *Recent developments on structural equation models: Theory and applications* (pp. 151–174). Amsterdam, The Netherlands: Kluwer. doi: 10.1007/978-1-4020-1958-6_9

Bolger, K. E., & Patterson, C. J. (2001). Pathways from child maltreatment to internalizing problems: Perceptions of control as mediators and moderators. *Development and Psychopathology*, *13*, 913–940.

Bollen, K. A. (1989). *Structural equations with latent variables*. New York, NY: John Wiley & Sons.

Brody, G. H., Chen, Y., Yu, T., Beach, S. R. H., Kogan, S. M., Simons, R. L., … Philibert, R. A. (2012). Life stress, the dopamine receptor gene, and emerging adult drug use trajectories: A longitudinal, multilevel, mediated moderation analysis. *Development and Psychopathology*, *24*, 941–951. doi: 10.1017/S0954579412000466

Browne, M. W. (1993). Structured latent curve models. In C. M. Cuadras & C. R. Rao (Eds.), *Multivariate analysis: Future directions* (Vol. 2, pp. 171–198). Amsterdam, The Netherlands: North-Holland.

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. Bollen & S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Beverly Hills, CA: Sage. doi: 10.1177/0049124192021002005

Browne, M., & du Toit, S. H. C. (1991). Models for learning data. In L. Collins & J. L. Horn (Eds.), *Best methods for the analysis of change* (pp. 47–68). Washington, DC: American Psychological Association.

Bryk, A. S., & Raudenbush, S. W. (1987). Application of hierarchical linear models to assessing change. *Psychological Bulletin*, *101*, 147–158. doi: 10.1037/0033-2909.101.1.147

Burchinal, M., & Appelbaum, M. I. (1991). Estimating Individual Developmental Functions: Methods and Their Assumptions. *Child Development*, *62*(1), 23–43. http://doi.org/10.2307/1130702

Capaldi, D. M., & Patterson, G. R. (1989). *Psychometric properties of fourteen latent constructs from the Oregon Youth Study*. New York, NY: Springer–Verlag. doi: 10.1007/978-1-4612-3562-0

Center for Human Resource Research. (2004). *A guide to the 1979–2002 National Longitudinal Survey of Youth*. Washington, DC: U.S. Department of Labor, Bureau of Labor Statistics.

Cicchetti, D. (2010). Developmental psychopathology. In M. E. Lamb, A. M. Freund, & R. M. Lerner (Eds.), *The handbook of life-span development, Vol 2: Social and emotional development* (pp. 511–589). Hoboken, NJ: John Wiley & Sons.

Cicchetti, D., & Curtis, W. J. (2007). Multilevel perspectives on pathways to resilient functioning. *Development and Psychopathology*, *19*, 627–629. doi: 10.1017/S0954579407000314

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597–600. doi: 10.1017/S0954579400007318

Cicchetti, D., & Rogosch, F. A. (1999). Conceptual and methodological issues in developmental psychopathology research. In P. C. Kendall, J. N. Butcher, & G. N. Holmbeck (Eds.), *Handbook of research methods in clinical psychology* (2nd ed., pp. 433–465). New York, NY: John Wiley & Sons.

Cicchetti, D., & Toth, S. L. (2006). Developmental psychopathology and preventive intervention. In A. Renninger & I. Sigel (Eds.), *Handbook of child psychology* (6th ed., pp. 497–547). New York, NY: Wiley. doi: 10.1002/9780470147658.chpsy0413

Cicchetti, D., & Tucker, D. (1994). Development and self-regulatory structures of the mind. *Development and Psychopathology*, *6*, 533–549. doi: 10.1017/S0954579400004673

Cook, T. D., & Campbell, D. T. (1979). *Quasi-experimentation: Design and analysis issues for field settings*. Boston, MA: Houghton Mifflin. doi: 10.1207/s15327752jpa4601_16

Coopersmith, S. (1981). *The Self Esteem Inventories*. Palo Alto, CA: Consulting Psychologists Press.

Cudeck, R., & du Toit, S. H. C. (2002). A nonlinear form of quadratic regression with interpretable parameters. *Multivariate Behavioral Research*, *37*, 501–519. doi: 10.1207/S15327906MBR3704_04

Cudeck, R. A., & Klebe, K. J. (2002). Multiphase mixed-effects models for repeated measures data. *Psychological Methods*, *7*, 41–63. doi: 10.1037/1082-989X.7.1.41

Curran, P. J. (2003). Have multilevel models been structural equation models all along? *Multivariate Behavioral Research*, *38*, 529–569. doi: 10.1207/s15327906mbr3804_5

Curran, P. J., & Willoughby, M. T. (2003). Implications of latent trajectory models for the study of developmental psychopathology. *Development and Psychopathology*, *15*, 581–612. doi: 10.1017/S0954579403000300

Curtis, W. J., & Cicchetti, D. (2003). Moving research on resilience into the 21st centry: Theoretical and methodological considerations in examining the biological contributors to resilience. *Development and Psychopathology*, *15*, 773–810. 10.1017/S0954579403000373

Davidian, M., & Giltinan, D. M. (1995). *Nonlinear models for repeated measurement data*. New York, NY: Chapman and Hall. doi: 10.1198/1085711032697

Davison, M. L. (2008). Hypothesis generation in latent growth curve modeling using principal components. *Educational Research and Evaluation*, *14*, 321–334. doi: 10.1080/13803610802249498

Derogatis, L. R. (1975). *Brief symptom inventory*. Baltimore, MD: Clinical Psychometric Research.

Ding, C. S., Davison, M. L., & Petersen, A. C. (2005). Multidimensional scaling analysis of growth and change. *Journal of Educational Measurement*, *42*, 171–191. doi: 10.1111/j.1745-3984.2005.00009

Dogan, S. J., Stockdale, G. D., Widaman, K. F., & Conger, R. D. (2010). Developmental relations and patterns of change between alcohol use and number of sexual partners from adolescence through adulthood. *Developmental Psychology*, *46*, 1747–1759. doi: 10.1037/a0019655

Dunn, L. M., & Dunn, L. M. (1981). *Peabody Picture Vocabulary Test – Revised*. Circle Pines, MN: American Guidance Service.

Endres, M. J., Rickert, M. E., Bogg, T., Lucas, J., & Finn, P. R. (2011). Externalizing psychopathology and behavioral disinhibition: Working memory mediates signal discriminability and reinforcement moderates response bias in approach-avoidance learning. *Journal of Abnormal Psychology*, *120*, 336–351. doi: 10.1037/a0022501

Ferrer, E., & Grimm, K. J. (2012). Issues in collecting longitudinal data. In H. Cooper, P. M. Camic, D. L. Long, A. T. Panter, D. Rindskopf, & K. J. Sher (Eds.), *APA Handbook of research methods in psychology: Vol 2. Research designs: Quantitative, qualitative, neuropsychological, and biological* (pp. 275–290). Washington, DC: American Psychological Association. doi: 10.1037/13620-016

Ferrer, E., Hamagami, F., & McArdle, J. J. (2004). Modeling latent growth curves with incomplete data using different types of structural equation modeling and multilevel software. *Structural Equation Modeling*, *11*, 452–483. doi: 10.1207/s15328007sem1103_8

Flora, D. B., Khoo, S. T., & Chassin, L. (2006). Moderating effects of a risk factor: Modeling longitudinal moderated mediation in the development of adolescent heavy drinking. In T. D. Little, J. A. Bovaird, & N. A. Card (Eds.), *Modeling contextual effects in longitudinal studies* (pp. 231–254). Mahwah, NJ: Lawrence Erlbaum.

Gerstorf, D., Ram, N., Estabrook, R., Schupp, J., Wagner, G. G., & Lindenberger, U. (2008). Life satisfaction shows terminal decline in old age: Longitudinal evidence from the German Socio-Economic Panel Study (SOEP). *Developmental Psychology*, *44*, 4, 1148–1159. doi: 10.1037/0012-1649.44.4.1148

Ghisletta, P., & Lindenberger, U. (2004). Static and dynamic longitudinal structural analyses of cognitive changes in old age. *Gerontology*, *50*, 12–16. doi: 10.1159/000074383

Grimm, K. J. (2006). *A longitudinal dynamic analysis of the impacts of reading on mathematical ability in children and adolescents*. Unpublished doctoral dissertation, University of Virginia, Charlottesville, VA.

Grimm, K. J. (2007). Multivariate longitudinal methods for studying developmental relationships between depression and academic achievement. *International Journal of Behavioral Development*, *31*, 328–339. doi: 10.1177/0165025407077754

Grimm, K. J., Castro-Schilo, L., & Davoudzadeh, P. (2013). Modeling intraindividual change in nonlinear growth models with latent change scores. *GeroPsych*, *26*, 153–162. doi: 10.1024/1662-9647/a000093

Grimm, K. J., & Ram, N. (2009a). Nonlinear growth models in Mplus and SAS. *Structural Equation Modeling*, *16*, 676–701. doi: 10.1080/10705510903206055

Grimm, K. J., & Ram, N. (2009b). A second-order growth mixture model for developmental research. *Research in Human Development*, *2–3*, 121–143. doi: 10.1080/15427600902911221

Grimm, K. J., & Ram, N. (2011). Growth curve modeling from an SEM perspective. In B. Laursen, T. Little, & N. Card (Eds.), *Handbook of developmental research methods* (pp. 411–431). New York, NY: Guilford Publications.

Grimm, K. J., Ram, N., & Estabrook, R. (2010). Nonlinear structured growth mixture models in Mplus and OpenMx. *Multivariate Behavioral Research*, *45*, 887–909. doi: 10.1080/00273171.2010.531230

Grimm, K. J., Ram, N., & Hamagami, F. (2011). Nonlinear growth curves in developmental research. *Child Development*, *82*, 1357–1371. doi: 10.1111/j.1467-8624.2011.01630.x

Grimm, K. J., Steele, J. S., Ram, N., & Nesselroade, J. R. (2013). Exploratory latent growth models in the structural equation modeling framework. *Structural Equation Modeling*, *20*, 258–591. doi:10.1080/10705511.2013.824775

Grimm, K. J., Zhang, Z., Hamagami, F., & Mazzocco, M. M. (2013). Modeling nonlinear change via latent change and latent acceleration frameworks: Examining velocity and acceleration of growth trajectories. *Multivariate Behavioral Research*, *48*, 117–143.doi: 10.1080/00273171.2012.755111

Grossman, A. W., Churchill, J. D., McKinney, B. C., Kodish, I. M., Otte, S. L., & Greenough, W. T. (2003). Experience effects on brain development: Possible contributions to psychopathology. *Journal of Child Psychology and Psychiatry*, *44*, 33–63. doi: 10.1111/1469-7610.t01-1-00102

Guo, W. (2002). Functional mixed effects models. *Biometrics*, *58*, 121–128. doi: 10.1111/j.0006-341X.2002.00121.x

Hamagami, F., & McArdle, J. J. (2007). Dynamic extensions of latent difference score models. In S. M. Boker & M. L. Wegner (Eds.), *Quantitative methods in contemporary psychology* (pp. 47–85). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Hamilton, M. A. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, *6*, 56–62. doi: 10.1136/jnnp.23.1.56

Harter, S. (1985). *Manual for the self-perception profile for children*. Denver, CO: University of Denver.

Hartley, D. E., & Strupp, H. H. (1983). The therapeutic alliance: Its relationship to outcome in brief psychotherapy. In J. Masling (Ed.), *Empirical studies in analytic theories* (Vol. 1, pp. 1–37). Hillsdale, NJ: Erlbaum.

Hawley, L. L., Ho, M.-H. R., Zuroff, D. C., & Blatt, S. J. (2006). The relationship of perfectionism, depression, and therapeutic alliance during treatment for depression: Latent difference score analysis. *Journal of Consulting and Clinical Psychology*, *74*, 930–942. doi: 10.1037/0022-006X.74.5.930

Hetherington, E. M., Bridges, M., & Insabella, G. M. (1998). What matters? What does not?: Five perspectives on the association between marital transitions and children's adjustment. *American Psychologist*, *53*, 167–184. doi: 10.1037/0003-066X.53.2.167

Hussong, A. M., Curran, P. J., Moffitt, T. E., & Caspi, A. (2008). Testing turning point using latest growth curve models: Competing models for substance abuse and desistance in young adulthood. In P. Cohen (Ed.), *Applied data analytic techniques for turning points research* (pp. 81–104). New York, NY: Routledge.

Jedidi, K., Ramaswamy, V., & Desarbo, W. S. (1993). A maximum likelihood method for latent class regression involving a censored dependent variable. *Psychometrika*, *358*, 375–394. doi: 10.1007/BF02294647

Johnston, L. D., O'Malley, P., & Bachman, J. G. (1995). *National survey results on drug use from the Monitoring the Future study, 1975–1994: Vol. 1. Secondary school students*. Technical Report No. 95-4026). Washington, DC: NIH.

Jöreskog, K. G. (1974). Analyzing psychological data by structural analysis of covariance matrices. In R. C. Atkinson, D. H. Krantz, R. D. Luce, & P. Suppas (Eds.), *Contemporary developments in mathematical psychology* (pp. 1–56). San Francisco, CA: Freeman.

Kandel, E. R. (1999). Biology and the future of psychoanalysis: A new intellectual framework for psychiatry revisited. *American Journal of Psychiatry*, *156*, 505–524.

Kim, J., & Cicchetti, D. (2006). Longitudinal trajectories of self-system processes and depressive symptoms among maltreated and nonmaltreated children. *Child Development*, *77*, 624–639. doi: 10.1111/j.1467-8624.2006.00894.x

Kim, J., Cicchetti, D., Rogosch, F. A., & Manly, J. T. (2009). Child maltreatment and trajectories of personality and behavioral functioning: Implications for the development of personality disorder. *Development and Psychopathology*, *21*, 889–912. doi: 10.1017/S0954579409000480

Kim, J., & Deater-Deckard, K. (2011). Dynamic changes in anger linking to developmental trajectories of internalizing and externalizing problems: The moderating role of attention. *Journal of Child Psychology and Psychiatry*, *52*, 156–166. doi: 10.1111/j.1469-7610.2010.02301.x

Kim-Spoon, J., Cicchetti, D., & Rogosch, F. A. (2013). A longitudinal study of emotion regulation, negative emotionality, and internalizing symptomatology in maltreated and nonmaltreated children. *Child Development*, *84*, 512–527. doi: 10.1111/j.1467-8624.2012.01857.x

Kovacs, M. (1985). The children's depression inventory. *Psychopharmacology Bulletin*, *21*, 995–998.

Kovacs, M. (1992). *Children's depression inventory manual*. North Tonawanda, NY: Multi-Health Systems.

Kwok, O.-M., West, S. G., & Green, S. B. (2007). The impact of misspecifying the within-subject covariance structure in multiwave longitudinal multilevel models: A Monte Carlo study. *Multivariate Behavioral Research*, *42*, 557–592. doi: 10.1080/00273170701540537

Laird, N. M., & Ware, J. H. (1982). Random-effects models for longitudinal data. *Biometrics*, *38*, 963–974. doi: 10.2307/2529876

Lansford, J. E., Malone, P. S., Stevens, K. I., Dodge, K. A., Bates, J. E., & Pettit, G. S. (2006). Developmental trajectories of externalizing and internalizing behaviors: Factors underlying resilience in physically abused children. *Development and Psychopathology*, *18*, 35–55. doi: 10.1017/S0954579406060032

Littell, R. C., Milliken, G. A., Stroup, W. W., & Wolfinger, R. D. (1996). *SAS system for mixed models*. Cary, NC: SAS Institute Inc.

Lo, Y., Mendell, N. R., & Rubin, D. B. (2001). Testing the number of components in a normal mixture. *Biometrika*, *88*, 757–778. doi: 10.1093/biomet/88.3.767

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, *71*, 543–562. doi: 10.1111/1467-8624.00164

Lynne-Landsman, S. D., Bradshaw, C. P., & Ialongo, N. S. (2010). Testing a developmental cascade model of adolescent substance use trajectories and young adult adjustment. *Development and Psychopathology*, *22*, 933–948. doi: 10.1017/S0954579410000556

MacCallum, R. C., Kim, C., Malarkey, W. B., Kiecolt-Glaser, J. K. (1997). Studying multivariate change using multilevel models and latent curve models. *Multivariate Behavioral. Research*, *32*, 215–253. doi: 10.1207/s15327906mbr3203_1

MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G., & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods*, *7*, 83–104. doi: 10.1037/1082-989X.7.1.83

Masyn, K. (2009). Discrete-time survival factor mixture analysis for low-frequency recurrent event histories. *Research in Human Development*, *6*, 165–194. doi: 10.1080/15427600902911270

McArdle, J. J. (1986). Latent variable growth within behavior genetic models. *Behavior Genetics*, *16*, 163–200. doi: 10.1007/BF01065485

McArdle, J. J. (1988). Dynamic but structural equation modeling of repeated measures data. In J. R. Nesselroade & R. B. Cattell (Eds.), *Handbook of multivariate experimental psychology* (vol. 2, pp. 561–614). New York, NY: Plenum. doi: 10.1007/978-1-4613-0893-5_17

McArdle, J. J. (2001). A latent difference score approach to longitudinal dynamic structural analyses. In R. Cudeck, S. du Toit, & D. Sorbom (Eds.), *Structural equation modeling: Present and future* (p. 342–380). Lincolnwood, IL: Scientific Software International.

McArdle, J. J. (2009). Latent variable modeling of differences and changes with longitudinal data. *Annual Reviews of Psychology*, *60*, 577–605. doi: 10.1146/annurev.psych.60.110707.163612

McArdle, J. J., & Bell, R. Q. (2000). An introduction to latent growth models for developmental data analysis. In T. D. Little, K. U. Schanebel, & J. Baumert (Eds.), *Modeling longitudinal and multiple-group data: Practical issues, applied approaches, and scientific examples* (pp. 69–108). Mahwah, NJ: Erlbaum.

McArdle, J. J., & Epstein, D. (1987). Latent growth curves within developmental structural equation models. *Child Psychology*, *58*, 110–133. doi: 10.2307/1130295

McArdle, J. J., Grimm, K. J., Hamagami, F., Bowles, R. P., & Meredith, W. (2009). Modeling lifespan growth curves of cognition using longitudinal data with multiple samples and changing scales of measurement. *Psychological Methods*, *14*, 126–149. doi: 10.1037/a0015857

McArdle, J. J., & Hamagami, F. (1996). Multilevel models from a multiple group structural equation perspective. In G. Marcoulides & R. Schumacker (Eds.), *Advanced structural equation modeling techniques* (pp. 89–124). Hillsdale, NJ: Erlbaum.

McArdle, J. J., & Hamagami, F. (2001). Latent difference score structural models for linear dynamic analyses with incomplete longitudinal data. In L. Collins & A. Sayer (Eds.), *New methods for the analysis of change* (pp. 139–175). Washington, DC: American Psychological Association. doi: 10.1037/10409-005

McArdle, J. J., Hamagami, F., Jones, K., Jolesz, F., Kikinis, R., Spiro III, A., & Albert, M. S. (2004). Structural modeling of dynamic changes in memory and brain structure using longitudinal data from the normative aging study. *Journal of Gerontology: Psychological Sciences*, *59B*, P294–P304. doi: 10.1093/geronb/59.6.P294

McArdle, J. J., & McDonald, R. P. (1984). Some algebraic properties of the Reticular Action Model for moment structures. *British Journal of Mathematical and Statistical Psychology*, *37*, 234–251.

McArdle J. J., Nesselroade J. R. (1994). Structuring data to study development and change. In S. H. Cohen & H. W. Reese (Eds.), *Life-span developmental psychology: Methodological innovations* (pp. 223–267). Hillsdale, NJ: Erlbaum

McArdle, J. J., & Woodcock, J. R. (1997). Expanding test–retest designs to include developmental time-lag components. *Psychological Methods*, *2*, 403–435. doi: 10.1037/1082-989X.2.4.403

McLachlan, G. J., & Peel, D. (2000). *Finite mixture models*. New York, NY: John Wiley & Sons, Inc. doi: 10.1002/0471721182

Meredith, W. (1964a). Notes on factorial invariance. *Psychometrika*, *29*, 177–185. doi: 10.1007/BF02289699

Meredith, W. (1964b). Rotation to achieve factorial invariance. *Psychometrika*, *36*, 187–206. doi: 10.1007/BF02289700

Meredith, W. (1965). A method for studying differences between groups. *Psychometrika*, *30*, 15–29. doi: 10.1007/BF02289744

Meredith, W. (1993). Measurement invariance, factor analysis and factorial invariance. *Psychometrika*, *58*, 525–543. doi: 10.1007/BF02294825

Meredith, W., & Horn, J. L. (2001). The role of factorial invariance in modeling growth and change. In L. M. Collins & A. G. Sayer (Eds.), *New methods for the analysis of change* (pp. 203–240). Washington, DC: American Psychological Association. doi: 10.1037/10409-007

Meredith, W., & Tisak, J. (1984). *"Tuckerizing" curves*. Paper presented at the Psychometric Society annual meetings, Santa Barbara, CA.

Meredith, W., & Tisak, J. (1990). Latent curve analysis. *Psychometrika*, *55*, 107–122. doi: 10.1007/BF02294746

Muthén, B. (2004). Latent variable analysis. Growth mixture modeling and related techniques for longitudinal data. In D. Kaplan (Ed.), *The Sage handbook of quantitative methodology for the social sciences* (pp. 345–368). Thousand Oaks, CA: Sage Publications. doi: 10.4135/9781412986311.n19

Muthén, B., & Muthén, L. K. (2000). Integrating person-centered and variable-centered analysis: Growth mixture modeling with latent trajectory classes. *Alcoholism: Clinical and Experimental Research*, *24*, 882–891. doi: 10.1111/j.1530-0277.2000.tb02070.x

Muthén, B., & Shedden, K. (1999). Finite mixture modeling with mixture outcomes using the EM algorithm. *Biometrics*, *55*, 463–469. doi: 10.1111/j.0006-341X.1999.00463.x

Nagin, D. S. (1999). Analyzing developmental trajectories: A semiparametric, group-based approach. *Psychological Methods*, *4*, 139–157. doi: 10.1037/1082-989X.4.2.139

Nesselroade, J. R., & Featherman, D. L. (1997). Establishing a reference frame against which to chart age-related change. In M. A. Hardy (Ed.), *Studying aging and social change: Conceptual and methodological issues* (pp. 191–205). Thousand Oaks, CA: Sage.

Newcomb, M. D., & Harlow, L. L. (1986). Life events and substance use among adolescents: Mediating effects of perceived loss of control and meaninglessness in life. *Journal of Personality and Social Psychology*, *51*, 564–577. doi: 10.1037/0022-3514.51.3.564

Nylund, K. L., Asparouhov, T., & Muthén, B. (2007). Deciding on the number of classes in latent class analysis and growth mixture

modeling. A Monte Carlo simulation study. *Structural Equation Modeling*, *14*, 535–569. doi: 10.1080/10705510701575396

Olsen, M. K., & Schafer, J. L. (2001). A two-part random-effects model for semicontinuous longitudinal data. *Journal of the American Statistical Association*, *96*, 730–745. doi: 10.1198/016214501753168389

Oud, J. H. L., & Jansen, R. A. R. G. (2000). Continuous time state space modeling of panel data by means of SEM. *Psychometrika*, *65*, 199–215.

Pinheiro, J. C., & Bates, D. M. (2000). *Mixed effects models in S and S-Plus*. New York, NY: Springer-Verlag. doi: 10.1007/978-1-4419-0318-1

Preece, M. A., & Baines, M. J. (1978). A new family of mathematical models describing the human growth curve. *Annals of Human Biology*, *5*, 1–24. doi: 10.1080/03014467800002601

Rabiner, D. L., Keane, S. P., & MacKinnon-Lewis, C. (1993). Children's beliefs about familiar and unfamiliar peers in relation to their sociometric status. *Developmental Psychology*, *29*, 236–243. doi: 10.1037/0012-1649.29.2.236

Ram, N., Gerstorf, D., Fauth, E., Zarit, S., & Malmberg, B. (2010). Aging, disablement, and dying: Using time-as-process and time-as-resources metrics to chart late-life change. *Research in Human Development*, *7*, 27–44. doi: 10.1080/15427600903578151

Ram, N., & Grimm, K. J. (2009). Growth mixture modeling: A method for identifying differences in longitudinal change among unobserved groups. *International Journal of Behavioral Development*, *33*, 565–576. doi: 10.1177/0165025409343765

Rao, C. R. (1958). Some statistical methods for the comparison of growth curves. *Biometrics*, *14*, 1–17. doi: 10.2307/2527726

Reinke, W. M., Eddy, J. M., Dishion, T. J., & Reid, J. B. (2012). Joint trajectories of symptoms of disruptive behavior problems and depressive symptoms during early adolescence and adjustment problems during emerging adulthood. *Journal of Abnormal Child Psychology*, *40*, 1123–1136. doi: 10.1007/s10802-012-9630-y

Rogosa, D. R. (1980). A critique of cross-lagged correlation. *Psychological Bulletin*, *88*, 245–258. doi: 10.1037/0033-2909.88.2.245

Rutter, M. (1990). Psychosocial resilience and protective mechanisms. In J. Rolf, A. S. Masten, D. Cicchetti, K. H. Nuechterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 181–214). New York, NY: Cambridge University Press. doi: 10.1017/CBO9780511752872.013

Schwartz, G. (1978). Estimating the dimensions of a model. *Annals of Statistics*, *6*, 461–464. doi: 10.1214/aos/1176344136

Sclove, L. S. (1987). Application of model-selection criteria to some problems in multivariate analysis. *Psychometrika*, *53*, 333–343. doi: 10.1007/BF02294360

Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: The quest for a new diagnostic instrument. *American Journal of Psychiatry*, *127*, 1653–1658.

Shields, A., & Cicchetti, D. (1998). Reactive aggression among maltreated children: The contribution of attention and emotion regulation. *Journal of Clinical Child Psychology*, *27*, 381–395. doi: 10.1207/s15374424jccp2704_2

Silva, P. A., & Stanton, W. R. (1996). *From child to adult: The Dunedin Multidisciplinary Health and Development Study*. Auckland, New Zealand: Oxford University Press.

Singer, J. (1998). Using SAS Proc Mixed to fit multilevel models, hierarchical models, and individual growth curves. *Journal of Educational and Behavioral Statistics*, *24*, 323–355. doi: 10.3102/10769986023004323

Skinner, H. A. (1982). The drug abuse screening test. *Addictive Behaviors*, *7*, 363–371. doi: 10.1016/0306-4603(82)90005-3

Sroufe, A. (1997). Psychopathology as an outcome of development. *Development and Psychopathology*, *9*, 251–268. doi: 10.1017/S0954579497002046

Taylor, B. J., Graham, J. W., Cumsille, P., & Hansen, W. B. (2000). Modeling prevention program effects on growth in substance use: Analysis of five years of data from the Adolescent Alcohol Prevention Trial. *Prevention Science*, *1*(4), 183–197.

Troop-Gordon, W., & Ladd, G. W. (2005). Trajectories of peer victimization and perceptions of the self and schoolmates: Precursors to internalizing and externalizing problems. *Child Development*, *76*, 1072–1091. doi: 10.1111/j.1467-8624.2005.00898.x

Tucker, L. R (1958). Determination of parameters of a functional relation by factor analysis. *Psychometrika*, *23*, 19–23. doi: 10.1007/BF02288975

Tucker, L. R. (1966). Learning theory and multivariate experiment: Illustration by determination of generalized learning curves. In R. B. Cattell (Ed.), *Handbook of multivariate experimental psychology* (pp. 476–501). Chicago, IL: Rand McNally.

Vuong, Q. H. (1989). Likelihood ratio tests for model selection and non-nested hypotheses. *Econometrica*, *57*, 307–333. doi: 10.2307/1912557

Wang, L., Zhang, Z., McArdle, J. J., & Salthouse, T. A. (2008). Investigating ceiling effects in longitudinal data analysis. *Multivariate Behavioral Research*, *43*, 476–496. doi: 10.1080/00273170802285941

Wechsler, D. (1945). A standardized memory scale for clinical use. *Journal of Psychology*, *19*, 87–95. doi: 10.1080/00223980.1945.9917223

Weissman, A. N., & Beck, A. T. (1978). *Development and validation of the dysfunctional attitude scale: A preliminary investigation*. Paper presented at the annual meeting of the American Educational Research Association, Toronto, Ontario.

Werthamer-Larsson, L., Kellam, S. G., & Wheeler, L. (1991). Effect of firstgrade classroom environment on child shy behavior, aggressive behavior, and concentration problems. *American Journal of Community Psychology*, *19*, 585–602. doi: 10.1007/BF00937993

Widaman, K. F., & Reise, S. P. (1997). Exploring the measurement invariance of psychological instruments: Applications in the substance use domain. In K. J. Bryant, M. Windle, & S. G. West (Eds.), *The science of prevention: Methodological advances from alcohol and substance abuse research* (pp. 281–324). Washington, DC: American Psychological Association. doi: 10.1037/10222-009

Willett, J. B., & Sayer, A. G. (1994). Using covariance structure analysis to detect correlates and predictors of individual change over time. *Psychological Bulletin*, *116*, 363–381. doi: 10.1037/0033-2909.116.2.363

Wills, T. A., Sandy, J. M., Yaeger, A. M., Cleary, S. D., & Shinar, O. (2001). Coping dimensions, life stress, and adolescent substance use: A latent growth analysis. *Journal of Abnormal Psychology*, *110*, 309–323. doi: 10.1037/0021-843X.110.2.309

Wishart, J. (1938), Growth-rate determinations in nutrition studies with the bacon pig and their analysis. *Biometrika*, *30*, 16–28. doi: 10.2307/2332221

Wohlwill, J. F. (1973). *The study of behavioral development*. New York, NY: Academic Press. doi: 10.1126/science.184.4137.673

Wright, S. S. (1921). Correlation and causation. *Journal of Agricultural Research*, *20*, 557–585.

Zhang, Z., McArdle, J. J., & Nesselroade, J. R. (2012). Growth rate models: Emphasizing growth rate analysis through growth curve modeling. *Journal of Applied Statistics*, *39*, 1241–1262. doi: 10.1080/02664763.2011.644528

Zill, N., & Peterson, J. L. (1986). *Behavior Problems Index*. Washington, DC: Child Trends.

CHAPTER 23

Integrative Data Analysis for Research in Developmental Psychopathology

EUN-YOUNG MUN, YANG JIAO, and MINGE XIE

CHAPTER OVERVIEW 1042
UTILITIES FOR RESEARCH IN DEVELOPMENTAL PSYCHOPATHOLOGY 1047
Etiology 1047
Infrequently or Rarely Observed Behaviors 1049
Screening and Diagnostic Tests 1050
Prevention and Intervention 1051
RESEARCH SYNTHESIS 1051
Inclusion Criteria for Studies 1052
Publication Bias and Selection Bias 1053
Selection of Variables and Harmonization of Groups and Measures 1054
Classical Meta-analysis Approaches 1055
Combining 2 × 2 Tables 1056
Model-Based Approaches 1059
Complex Research Synthesis 1062
DATA EXAMPLES 1067
Pooled Data 1068
Outcome Analysis 1071
Software Programs and Packages 1079
TRANSLATIONAL IMPLICATIONS OF INTEGRATIVE DATA ANALYSIS 1080
FUTURE DIRECTIONS AND LIMITATIONS 1080
CONCLUSIONS 1081
REFERENCES 1082

CHAPTER OVERVIEW

The importance of research synthesis from a body of evidence using more rigorous, systematic, and quantitative approaches has been echoed in the field over years; yet most of the literature reviews are narrative or qualitative in nature. It is possible to conduct two different narrative literature reviews and to have contrasting sets of hypotheses if the reviews were not sufficiently extensive or done selectively. Even in extensive and systematic reviews, determining which studies are more important and relevant than others to report or discuss in detail may very well be within the realm of subjective judgments. Iain Chalmers

The project described was supported by Award Number R01 AA 019511 from the National Institute on Alcohol Abuse and Alcoholism (NIAAA). The third author's work was also supported in part by the National Science Foundation (NSF)-DMS1107012. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIAAA, the National Institutes of Health, or the NSF.

We thank Helene R. White and Anne E. Ray of the Center of Alcohol Studies, Rutgers, The State University of New Jersey; Jimmy de la Torre and Yan Huo of the Graduate School of Education, Rutgers, The State University of New Jersey; and David C. Atkins, David Huh, and Mary Larimer of the Department of Psychiatry and Behavioral Sciences, University of Washington, for their contributions to Project INTEGRATE.

We also thank the following researchers who contributed their data to Project INTEGRATE (in alphabetical order): John S. Baer, Department of Psychology, The University of Washington, and Veterans' Affairs Puget Sound Health Care System; Nancy P. Barnett, Center for Alcohol and Addiction Studies, Brown University; M. Dolores Cimini, University Counseling Center, The University at Albany, State University of New York; William R. Corbin, Department of Psychology, Arizona State University; Kim Fromme, Department of Psychology, The University of Texas, Austin; Joseph W. LaBrie, Department of Psychology, Loyola Marymount University; Mary E. Larimer, Department of Psychiatry and Behavioral Sciences, The University of Washington; Matthew P. Martens, Department of Educational, School, and Counseling Psychology, The University of Missouri; James G. Murphy, Department of Psychology, The University of Memphis; Scott T. Walters, Department of Behavioral and Community Health, The University of North Texas Health Science Center; Helene R. White, Center of Alcohol Studies, The State University of New Jersey; and Mark D. Wood, Department of Psychology, The University of Rhode Island.

[1]Color versions of Figures 23.1, 23.3, 23.8 and 23.10 are available at http://onlinelibrary.wiley.com/book/10.1002/9781119125556

(2003) poignantly pointed out missed opportunities of the past citing that, for example, cases of sudden infant death syndrome (SIDS), for babies sleeping on their fronts (tummy), could have been prevented much earlier, had there been a rigorous, systematic and quantitative review of available evidence. He also cited exemplary cases of the past that saved numerous lives (e.g., low doses of aspirin reducing the risk of cardiovascular morbidity and mortality; see O'Rourke, 2007, for a historical account of this report that appeared in the *Lancet* editorial in 1980). Furthermore, as the number of relevant studies increases, it becomes much more difficult to summarize findings from multiple studies in a narrative review.

Quantitative research synthesis can help us to draw inferences that are not afflicted with chance findings and optimism bias, when conducted routinely and vigorously with the most up-to-date compiled evidence. Quantitative research synthesis is aimed at quantifying the overall effect of interest across studies and providing the uncertainty surrounding the point estimate. The resulting summary estimate can be helpful for deciding the benefits of the effect in question if, for example, a new treatment were brought to market. In the field of preventions and interventions, one can examine the average effect size of existing interventions, and subsequently channel efforts into more effective interventions, thus saving limited resources available in the current, tight funding environment. The Cochrane Collaborative Network (Higgins & Green, 2011) and the Human Genome Epidemiology Network (Ioannidis et al., 2006) represent such efforts to promote systematic reviews and to obtain efficient and reliable findings, respectively, by utilizing large-scale evidence in the medical and epidemiological genetic research fields.

In addition, recent high profile reports and discussions have fueled the need for quantitative research synthesis. These reports pointed out that findings from single studies are often not replicable, and are subject to publication bias and afflicted by low power and high false discovery rates (e.g., Begley & Ellis, 2012; Ioannidis, 2005). Thus, findings from single studies that are conducted independently, regardless of how important they may sound, may be best interpreted with some caution and scrutiny. At the same time, data from individual studies may be utilized more often for a large-scale research synthesis to obtain more robust evidence in the future.

Synthesizing data across studies in a meta-analysis allows one to obtain more credible estimates of effects than are possible from individual studies and to achieve better generalization of how causal relationships may change over variations in study features (Shadish, Cook, & Campbell, 2002). From a more discovery-oriented or exploratory perspective, individual single studies are not well equipped for novel discoveries due to lack of power. By combining data from multiple sources, we can overcome several important, inherent limitations of single studies. In the future, single studies may serve two distinct goals: a preliminary investigation for novel discoveries, and a component data set as part of a bigger, pooled data set, for more robust and generalized inference.

The momentum toward more robust research synthesis via quantitative approaches has been building in recent psychological research. Quantitative research synthesis from available data—either published aggregated data (AD) in terms of effect size estimates or raw individual participant-level data (IPD)—has prominently been discussed as one of the central strategies for building a cumulative knowledge base. The journal *Prevention Science* recently published a special issue focusing on subgroup analysis by pooling data from multiple trials or by utilizing more advanced analytic approaches in prevention and intervention research (Supplee, Kelly, MacKinnon, & Barofsky, 2013). In addition, *Psychological Methods* published a special issue focusing on integrative data analysis (IDA) in 2009 (Curran, 2009), discussing advantages and disadvantages of pooling and combining raw data from multiple studies, compared with either single studies (Curran & Hussong, 2009) or meta-analysis using AD (Cooper & Patell, 2009). Similarly, *Perspectives on Psychological Science* recently published a special section on replicability (Pashler & Wagenmakers, 2012). In this special section, the Open Science Collaboration (2012) and other contributors have called for sharing of data and/or publishing raw data for IPD meta-analysis to achieve better transparency and higher standards for research (e.g., Ioannidis, 2012).

The current chapter discusses quantitative methods for research synthesis. We discuss any quantitative methods aimed at synthesizing information from multiple, independent sources under the big IDA tent. The covered methods encompass meta-analysis using AD for different outcomes, such as odds ratio, mean difference, or other effect size estimates; meta-analysis using IPD; and other approaches to analyzing multiple, independent data sets. This chapter is intended to provide a more inclusive coverage of these various quantitative approaches under the umbrella of IDA (Figure 23.1). Curran and Hussong (2009) defined IDA more specifically as "the statistical analysis of a single data set that consists of two or more separate samples that have been pooled into one" (p. 82) to distinguish IDA from either AD meta-analysis or single studies and to highlight the unique advantages and

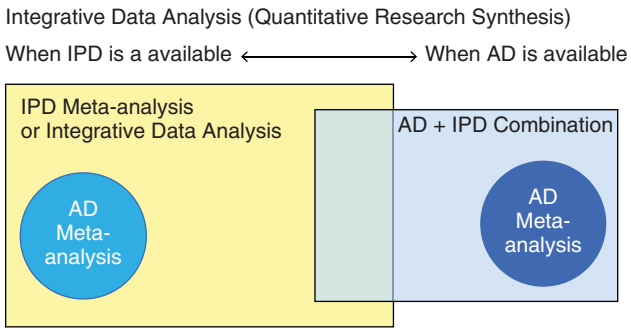


Figure 23.1 Integrative data analysis (IDA) and meta-analysis using individual participant-level data (IPD) and aggregated data (AD). AD meta-analysis can be conducted either as part of IDA when IPD is available (see the circle inside the rectangle on left) or based on reported AD in publications (the circle inside the right rectangle). See footnote 1.

challenges associated with analyzing raw IPD. In contrast, we include AD meta-analysis under the IDA framework because results from AD meta-analysis can be obtained by using raw IPD (Figures 23.1 and 23.2). For example, the overlap in the middle between the two rectangles in Figure 23.1 indicates situations where IPD is available for some of the studies and consequently AD meta-analysis is possible. The circles for AD meta-analysis within the two rectangles are shaded in different colors to reflect some differences in the procedures and possibly different results involved in AD meta-analysis depending on the source of data. The strict distinction between AD and IPD may not be useful for the purpose of research synthesis. Because IPD is not always available and IPD analysis can be quite challenging, analysis based on combinations of AD and partially available IPD may provide an alternative in some situations (see Yamaguchi et al., 2014, for a proposed method that combines these two data structures) while taking advantage of all available data. Figure 23.1 shows the relationships between IDA, IPD meta-analysis, and AD meta-analysis. Note that in the current chapter, we use the term IDA in two different ways. On one hand, we broadly use IDA as a general evidence-based framework for research synthesis that includes all quantitative methods. At the concrete level of analysis, on the other hand, we use IDA interchangeably with IPD meta-analysis and contrast it against AD meta-analysis. Table 23.1 provides a list of several important approaches and summarizes notable characteristics and challenges of each approach.

Despite some differences across various methods, these quantitative approaches under the broad umbrella of IDA share many benefits identified by Curran and Hussong (2009): built-in replication, increased statistical power, increased sample heterogeneity, increased frequencies of low base-rate behaviors, broader psychometric assessment of constructs, extended period of study, and increased efficiency in time and money. Analysis utilizing IPD may provide an especially good framework for facilitating new discoveries, as well as strengthening research practices, by improving the sample analyzed and by adopting advanced, better analytical approaches that may not have been available before. From the perspective of single studies, this approach may also propel desirable changes in the way individual single studies are conducted in the future. For example, in the field of brief motivational interventions to reduce excessive drinking and related harm among college students, Mun and her colleagues (2015) recommended that single studies be designed and implemented to increase overlap in measures and follow-up assessments across trials, to reduce heterogeneity in intervention groups across trials, and to improve transparency and documentation overall.

Figure 23.2 depicts various analytic approaches to research synthesis in a snapshot. First, at the broadest level, we distinguish methods based on whether IPD is available. In the absence of IPD, one has to rely on AD for quantitative synthesis. For the analysis of AD, one can combine AD across studies using fixed-effects or random-effects meta-analysis models (see the Model-Based Approaches section). One can further analyze between-study variation by using meta-regression, which can reveal study-level moderators of the effect under investigation. Meta-analysis using AD tends to be larger in scale in terms of the number of studies and sample size because collecting and analyzing AD is relatively straightforward and also because there is no need to establish commensurate measures. Effect size estimates provide readily available standardized measures in AD meta-analysis.

Meta-analysis has been on the rise in clinical research. It has had an exponential rise in the number of publications as a publication type in PubMed and also as a method covered in an influential journal, *Statistics in Medicine*, between 1990 and 2006 (Sutton & Higgins, 2008). Similarly, between 1997 and 2012, the papers published in a flagship journal in the field of developmental psychopathology, *Development and Psychopathology*, increasingly either reported findings from a meta-analysis or utilized evidence from a meta-analysis reported elsewhere when generating hypotheses or discussing findings (Figure 23.3).

When IPD is available, meta-analysis using IPD can be conducted. One can closely check and correct data, if necessary, and reanalyze data by using more suitable analytical models for robust inference. IPD meta-analysis has long been championed and discussed as a promising method in the field of medical research synthesis (e.g., Steinberg et al.,

TABLE 23.1 Research Synthesis Methods

Approaches	Notable characteristics	Weaknesses or challenges
Nonempirical research synthesis		
Narrative review of single studies	• Can quickly be conducted • Can draw information from a wide range of studies (e.g., animal models, cross sectional studies, experimental studies) • Vote counting, the process of counting the number of significant and nonsignificant studies, is often used	• May be subject to publication bias and reviewer subjectivity • Variations across studies can become too complex as the number of studies increases • Limitations of single studies cannot be overcome • The results from single studies cannot be quantified using a precise metric • Vote counting is not a valid approach
Empirical research synthesis		
Classical meta-analysis approaches	• Relatively easy to conduct • Essentially fixed-effects model	• Groups or conditions to be compared should be similar across studies • Publication bias remains a threat, which can be countered by conducting an extensive search of the published and unpublished literature
Model-based meta-analysis: Fixed-effects model (using either AD or IPD)	• One common true effect for all studies is assumed • Within-study variability, not between-study variability, is assumed • Between-study heterogeneity may be accommodated by grouping similar studies together and stratifying them or by including covariates • Covariates can be included	• Between-study heterogeneity is not part of the model, which may be an unreasonable assumption • Study-level missing data (i.e., did not assess) can cause difficulty estimating a common model across studies • Inference cannot be generalized beyond the sample of studies analyzed (in terms of sample characteristics and study designs/effects)
Model-based meta-analysis: Random-effects model (using either AD or IPD)	• A distribution of true study-specific effects is assumed • Between-study heterogeneity (i.e., different intervention effects and design/sample characteristics) can be taken into account • Population-based inference can be made • Compared to a fixed-effects model, relatively greater weights are assigned to smaller studies • Compared to a fixed-effects model, this will result in wider confidence intervals around pooled effect estimates • Covariates can be included	• Study-level missing data (i.e., did not assess) can cause difficulty estimating a common model across studies • Relatively a large number of studies are needed. With a few studies, estimates may not be reliable • Can be difficult to characterize the population to which inference is made
Complex research synthesis—multivariate meta-analysis, network meta-analysis, IDA*		
Network meta-analysis (using either AD or IPD but much more promising for IPD)	• When IPD analysis is used, it is widely considered the "gold standard"—efficient, unbiased, and powerful • With IPD, both study-level and participant-level covariates can be included • Particularly useful for research synthesis of randomized controlled trials (RCTs) • Multiple intervention comparisons are possible via direct, indirect, and mixed evidence of intervention effects	• Randomization of groups within studies is needed to derive relative intervention effects • Within-study covariance matrix can be hard to obtain in analysis using AD • Without IPD, ecological inference bias may occur when using study-level covariates • Can be computationally intensive with IPD
Multivariate meta-analysis (using either AD or IPD but much more promising for IPD)	• When IPD analysis is used, it is widely considered the "gold standard"—efficient, unbiased, and powerful • With IPD, both study-level and participant-level covariates can be included • With IPD, analytic models are not limited to those used in single studies; many different statistical models are feasible • Correlated parameters for multiple outcomes, time points, or intervention groups can be estimated	• Randomization of groups within studies is needed to derive relative intervention effects • IPD may not be available for clinical trial data due to privacy concerns • With IPD, data cleaning and checking take considerable time and resources • With IPD, common metrics across studies need to be established to minimize missing data and to interpret individual-level findings • Key study design characteristics may vary, causing study-level missing data • Can be computationally intensive with IPD
Other etiological (longitudinal) IDA**	• Take advantage of existing data sets that are expensive to collect (e.g., fMRI, DNA, or longitudinal data) • Create a pooled data set that has more desirable characteristics than possible from single studies • More advanced and appropriate analysis can be conducted • Used for validation and to quantify an average effect size • Have greater power to detect small effects	• Data dimensions can be exceedingly large • Compiled data can be very sparse due to study-level missing data (e.g., candidate genes not available in some studies) • Common metrics across studies need to be established to minimize missing data and to interpret individual-level findings • Can be computationally intensive • Typically a few studies are analyzed together as a single data set

*AD = Aggregated data; IPD = Individual participant-level data (or individual patient-level data in medical research).

**In principle, IDA investigations of etiological and/or long-term longitudinal studies can use the same analytical methods as those listed under the complex synthesis methods. It is listed separately to highlight the challenges of harmonizing and analyzing extremely high dimensional data.

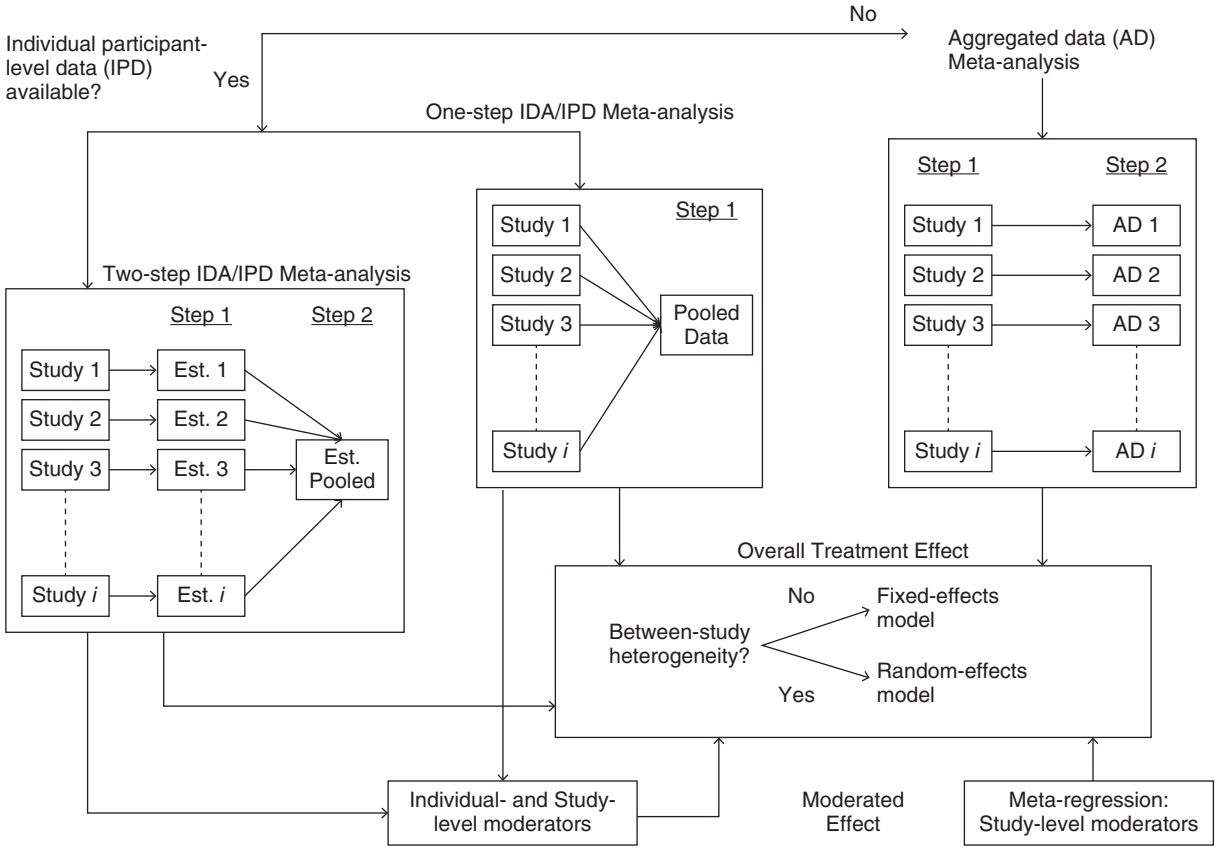


Figure 23.2 Analytical approaches under IDA.

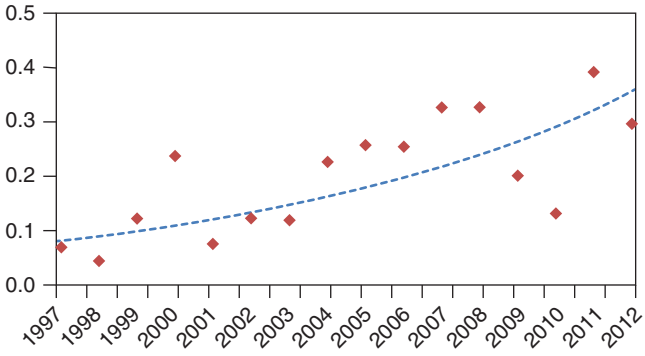


Figure 23.3 Proportion of the articles citing meta-analysis studies published in *Development and Psychopathology* from 1997 to 2012. See footnote 1.

1997; Stewart, 1995) but is a newly emerging approach for psychological research. IPD meta-analysis studies in psychological research have been rare primarily because of the challenge to establish measurement invariance across studies for valid inference. This can be a formidable challenge particularly for psychological measures (Hussong, Curran, & Bauer, 2013). Establishing measurement invariance can be challenging enough even when the same measure is used across key target groups (e.g., different developmental periods or different countries; Steenkamp & Baumgartner, 1998). It can be quite another challenge when different items or measures are used across studies and any overlap in items and measures is tenuous. Once measurement invariance across groups or studies is established, individuals can be placed on common metrics across different groups or studies.

Integrated analysis can then be conducted under various advanced analytical model frameworks (Figure 23.2). IPD meta-analysis can be very attractive in the sense that it can achieve two important goals: (1) strengthening our inference on prespecified (or confirmatory) hypotheses; and (2) giving an opportunity for serendipity, namely exploring new insights from IPD. In relation to the second goal, subgroups or moderated relationships can be examined via analyzing IPD (Borenstein & Higgins, 2013; Brown et al., 2013; Sutton & Higgins, 2008). Moderated effects require large samples to detect when they truly exist. The availability of both individual-level and study-level covariates makes it feasible to formally test these moderated relationships.

Within the IDA framework, one-step analysis is probably more typical, but places a greater burden on IDA researchers to resolve any discrepancies between studies and to deal with missing data as pooled IPD is analyzed in a single analytical model. Alternatively, two-step integrative analysis can proceed as follows: IDA researchers, after harmonizing data across studies (see the Selection of Variables and Harmonization of Groups and Measures section), develop a common analytical model and conduct separate analysis for each study included in the pooled data set. Estimates resulting from separate analyses from the first step can then be pooled across studies in the second integrative step (see the Outcome Analysis section for examples). Relative to the one-step approach, this may more flexibly address design differences across studies when analyzing the combined data. Alternatively, the two-step IDA can be conducted by original investigators under the same analytic strategy instead of sharing original data sets with IDA researchers (Brown et al., 2013). Brown and colleagues called this a *parallel analysis* strategy. Choosing an appropriate method for IPD meta-analysis may depend on several factors, including how easily data can be shared among researchers; how similar and dissimilar studies are in relation to research questions; the number of studies and, more generally, the dimension of the combined data; and how resources and research credits can be shared between research teams. IDA or IPD meta-analysis is a relatively new approach, but its applications are expected to increase in the coming years.

The current chapter will present methods of research synthesis for the field of developmental psychopathology, present classical and emerging meta-analysis approaches, provide data examples, and discuss future directions.

UTILITIES FOR RESEARCH IN DEVELOPMENTAL PSYCHOPATHOLOGY

In a review of the past achievements of developmental psychopathology and its future directions, Cicchetti and Toth (2009) stated that developmental psychopathology as a discipline has long sought to transcend many existing dualisms to better understand the developmental processes involved in maladaptive, as well as competent, trajectories across the life course. In addition to breaking down the schism between normative and nonnormative development, developmental psychopathology as a field has advocated a systems perspective (Ford & Lerner, 1992) and a holistic approach to development (Magnusson, 2000) to better understand complex developmental processes. The dynamic systems perspective and its nonlinear analytic tools, when combined with better known concepts and tools for linear statistical modeling approaches, may help to break the existing knowledge barriers in the literature.

Researchers in the field of developmental psychopathology have been early adopters of advanced analytical methods and led debates about their utilities (e.g., Bergman & Magnusson, 1997; Sterba & Bauer, 2010). Furthermore, researchers have championed the importance of maintaining feedforward and feedback loops between etiological discovery-oriented research and prevention and intervention research, while emphasizing that cultural vistas may affect the developmental processes of those living in them (Cicchetti & Toth, 2009). In essence, developmental psychopathology is a discipline that synthesizes data either directly or indirectly in single studies or multiple studies across multiple systems, across scales, and across time. By doing so, developmental psychopathology transcends the typical boundaries of disciplines in search of new insights and ideas.

We discuss ways in which methods for research synthesis can help shed new light for the field of developmental psychopathology. Some of the application examples in the literature are discussed to highlight the benefits of IDA, as well as the areas for improvement in the future.

Etiology

One of the critical challenges for research in developmental psychopathology is to understand developmental continuity and discontinuity of an underlying problem despite changing or different norms across age, gender, race and ethnicity, and other contexts. Focusing on age-related patterns, specific behaviors can repeatedly be assessed using the same set of questions across age. However, the same behavior observed at different ages may signal different levels or forms of risk. Likewise, different behaviors observed at different ages may indicate the same underlying trait. There are many excellent examples across different research fields. In the field of alcohol research, for example, there is a clear age-related trend in the prevalence of alcohol use prior to, during, and after college years (Substance Abuse and Mental Health Services Administration, 2012), and its associated risks and consequences vary across these distinctive developmental phases (e.g., White, Lee, Mun, & Loeber, 2012). Similarly, the legal drinking age differs across countries (Kuntsche et al., 2013), which affects the meaning of early onset drinking and its associations with other risky behaviors across nations.

The basic understanding of the underlying continuity and discontinuity across developmental phases is needed for screening individuals for intervention and for a better understanding of the etiology of alcohol problems. This issue of the continuity and discontinuity across time has been widely noted as an important research goal in developmental psychopathology (Sroufe & Rutter, 1984). In reality, however, this goal is difficult to achieve in individual studies. The complexity related to age is exacerbated when we consider race and ethnicity, as well as other situational and contextual influences. Single studies are typically limited in terms of demographic, contextual, and other situational influences. When data from multiple studies are combined, however, one can examine age-related continuity and discontinuity across different contexts and evaluate its generalizability.

In one of the early research studies that utilized meta-analysis in developmental psychopathology, Weiss and Garber (2003) examined developmental differences in the manifestation of depressive symptoms by meta-analyzing core, as well as associated, symptoms from 20 studies that varied in age, gender, and source of samples (clinical, diagnosed, normative). A total of 29 symptoms were examined in relation to age by using Cohen's effect size estimate *d* (Cohen, 1988). They found that for the vast majority of symptoms, significant study-to-study (i.e., between-study) heterogeneity in estimates existed, indicating that age-related patterns in symptoms substantially differed across studies. Of the 10 symptoms that were not significantly heterogeneous, the following five symptoms—anhedonia, hopelessness, hypersomnia, weight gain, and social withdrawal—showed higher levels among older individuals, which supported the notion that the specific manifestations of depressive symptoms may vary with development. Due in part to the high percentage of study-level heterogeneity, however, Weiss and Garber concluded the evidence inconclusive to call depression developmentally isomorphic. However, the implicit assumption involved in the meta-analysis under which this question was examined (i.e., a common true effect across studies under fixed-effects models) may have been unnecessarily too strict.

Recent meta-analytic studies tend to focus on explaining between-study variation in effect sizes (between-study heterogeneity) rather than assuming that effect sizes are the same across all studies. In general, it is more plausible to assume that study-specific effect sizes exist across studies, due to observed and unobserved differences in study contexts, designs, outcome measures, interventions, and subjects (Borenstein, Hedges, Higgins, & Rothstein, 2009). When the results from various studies differ, one can use this as an opportunity to investigate factors that may account for the observed heterogeneity across studies by allowing study-level covariates to be included in meta-regression (see the Model-Based Approaches section). When IPD is available, the search for explanations can be done at both the individual- and study-level.

Some studies may combine data from just a few studies. AD meta-analysis starts with an extensive search of all eligible and available evidence. However, three-quarters of meta-analyses indexed in the *Cochrane Database of Systematic Reviews* include five or fewer studies (Davey, Turner, Clarke, & Higgins, 2011). Nonetheless, evidence from two studies is better than evidence from one study, if all else is equal. By pooling original data from multiple sources, even if limited in number, one can extend the period under observation and test hypotheses in a more heterogeneous, diverse sample, which can strengthen our confidence in the derived conclusions. These benefits may be particularly salient for IDA studies that combine data from long-term, longitudinal studies because these studies are virtually impossible to replicate and also because they tend to feature homogeneous samples.

For example, Hussong et al. (2007) obtained original IPD from two longitudinal studies of children of alcoholics (COAs) and combined them to examine externalizing behavior problems in COAs. The two longitudinal studies combined and harmonized were the Michigan Longitudinal Study (MLS; Zucker et al., 2000), a community-based study of alcoholic parents and their young biological children and comparison families within the same neighborhoods, and the Adolescent/Adult Family Development Project (AFDP; Chassin et al., 1991) that followed adolescent COAs and their matched controls as well as their parents. Most cohorts for the MLS were originally between the ages of 3 and 5 and followed up every 3 years thereafter, with yearly assessments between the ages of 11 and 17. The AFDP project interviewed the adolescents when they were between the ages of 11 and 15. They were followed up twice when they were between the ages of 12 and 16 and again between the ages of 13 and 17. The MLS had observations covering mostly from ages 3 to 14, whereas the AFDP had observations spanning from ages 10 to 17. When these two samples were combined, the overlap that existed for ages 10 to 14 provided an important chain in measurement models and the basis to test subsequent, substantive hypotheses. With the two distinctive but similar samples combined, an observational period was stretched from ages 2 to 17 for the entire combined sample. This pooled sample had some of the core characteristics (i.e., COAs and non-COAs) in common, but differed in other aspects (e.g., race/ethnicity, and geographical and socioeconomic backgrounds).

With the combined data described above, Hussong and her colleagues (2007) examined whether (1) the number of alcoholic parents in families; (2) comorbid parental diagnoses (i.e., depression and antisocial personality disorder); and (3) subtypes of parental alcoholism (i.e., controls, depressed alcoholic type, and antisocial alcoholic type) were linked to the development of externalizing behavior problems. They found that children in multi-alcoholic families were at greater risk for externalizing symptoms that emerged by mid-adolescence. Children in comorbid alcoholic families had a stable early risk for greater externalizing symptoms, compared to those in noncormobid alcoholic families. These observations were internally replicated—meaning that the effect was observed in two samples—within the same analytic model. Thus, in addition to lengthening an observational window and increasing sample heterogeneity, IDA can be used to provide opportunities for built-in, internal replications and for better-powered studies compared with single individual studies (Curran & Hussong, 2009; Hussong et al., 2013).

Infrequently or Rarely Observed Behaviors

Compiling and analyzing data from multiple, similarly designed studies can be particularly beneficial for research in developmental psychopathology when rare behaviors are of particular interest. In typical single studies, a low base rate behavior or atypical behavior is often ignored—neither researched nor reported. However, when isolated or infrequent behaviors of interest are aggregated across multiple studies, this combined information can provide a valuable insight for developmental psychopathology. For clinical trials, it is now required to report adverse effects as one of the basic data for the ClinicalTrials.gov site (Tse, Williams, & Zarin, 2009). Adverse effects can then be compiled across studies and used to assess the overall safety risk of clinical interventions, as well as the efficacy.

In the field of pharmaceutical clinical trials, there has been a high profile case in recent years, demonstrating the importance of infrequently observed behaviors and its treatment in analysis. It involves Avandia (rosiglitazone), marketed by GlaxoSmithKline (GSK), which was intended to improve blood sugar levels in diabetics. Nissen and Wolski (2007) compiled adverse event data from 42 studies and concluded that the drug significantly increased the risk for myocardial infarction (heart attacks). According to Finkelstein and Levin (2012), GSK's stock price dropped 7.8% on the day of the publication of the study by Nissen and Wolski. Thousands of lawsuits were filed against GSK, and in 2010 alone GSK had charges totaling $6.76 billion against earnings to deal with the Avandia cases (Finkelstein & Levin, 2012). Several other subsequent meta-analysis publications ensued that used different approaches (e.g., Liu, Liu, & Xie 2014; Tian et al., 2009) from the one taken by Nissen and Wolski. One of the most contentious issues surrounding this controversy involved the treatment of zero adverse observations in studies. Because the rate of adverse outcomes was low (i.e., approximately 0.5%), it was possible not to see any adverse outcomes in some of the small studies. In typical two-arm (i.e., treatment and control) clinical trials, if adverse outcomes are not observed in both arms, it is difficult to quantify the risk, because the risk is mathematically undefined using well-known risk measures, such as odds ratio. Nissen and Wolski chose to exclude data altogether from any studies with zero adverse observations and used Peto's method of combining odds ratios (Yusuf, Peto, Lewis, Collins, & Sleight, 1985; see the Peto's Odds Ratio section for Peto's method) only from studies with nonzero observations. Subsequent investigations tackled zero adverse observations in original studies by adding a constant, by imputing data, or by using alternative methods by which risk differences could be defined.

We can learn from this Avandia controversy that zero events can be important to report in original studies, and that they can and should be investigated by compiling data from multiple studies. Even if no reasonable statistical approach can be taken, zero events out of 100,000 cases certainly carry a different meaning than zero events out of 1,000 cases. Especially in the field of developmental psychopathology, many behaviors of interest that occur rarely have not been studied extensively. Although rare, these behaviors are nonetheless important to better understand because of their potentially huge impact. For example, using data from a longitudinal project on the development of delinquency and antisocial behavior among boys, Lee and White (2012) reported that those who experienced childhood maltreatment were more likely than those who did not experience it (defined as an officially substantiated record by age 13) to die before the ages 27–32 and ages 34–38, respectively, for two different birth cohorts. More specifically, 7.4% of 202 maltreated men died, whereas 2.5% of 711 nonmaltreated men died. Overall, 35 deaths were observed during the 913 person-year observation period, yielding an average mortality rate of 3,834 deaths per 100,000 person-years (7,426 for maltreated vs. 2,813 for nonmaltreated men). Many individuals followed-up by this project were exposed to violence in childhood and adolescence, and died from, or committed, homicide (see also Loeber et al., 2005). Childhood maltreatment, for this high-risk sample, further increased their risk of early death.

As the example described above shows, rare behavioral data have the potential to provide an important new insight in the field, yet many such behaviors have been understudied. From the analyst's perspective, it also makes sense to combine data from individual studies because low base-rate, binary behaviors (such as death or maltreatment) and their associated binomial confidence intervals can be quite erratic (Brown, Cai, & DasGupta, 2001). When data from multiple studies are combined, the number of cases increases, although average prevalence rate remains the same, which improves the stability of model estimation and reduces the influence of extreme observations, thus achieving better precision.

Even when behaviors of interest may not be as rare as what was previously discussed, many clinical outcomes of interest have generally low base rates. Behaviors with prevalence rates of 10 to 20% are huge numbers at the population level, but can still be difficult to study in single studies. The median and mean total sample sizes for articles published in four major psychological journals (*Journal of Abnormal Psychology, Journal of Applied Psychology, Journal of Experimental Psychology: Human Perception and Performance,* and *Developmental Psychology*) in 2006 were 40 and 196, respectively (Marszalhk, Barber, Kohlhart, & Holmes, 2011). At this level of sample size, a behavior with the prevalence of 5% or less, despite highly prevalent at the population level, would be practically impossible to examine in single studies. Furthermore, some of the advanced analytic approaches for nonnormally distributed data, including Poisson or zero-inflated hurdle mixed models, tend to be more complex and challenging and require many parameters to be estimated (Atkins, Baldwin, Zheng, Gallop, & Neighbors, 2013). Thus, pooling data from multiple, similar studies and analyzing them together for research synthesis can be particularly attractive for many clinical behaviors of interest.

Screening and Diagnostic Tests

It is important to develop screening and diagnostic tests and to derive cut-off scores that are sensitive for detecting true cases (i.e., sensitivity) yet correctly differentiate those cases that, in truth, do not meet the criteria (i.e., specificity). Only a small proportion of individuals typically meet any clinical criteria by definition. Thus, one can achieve better sensitivity and specificity estimates of a screening tool by combining multiple data sets. Both estimates—sensitivity and specificity—are important to simultaneously consider when making informed decisions under which a particular screening tool should be used. This is because these estimates are typically considered in the context of the cost of administering diagnostic or screening tests, and the consequences of missing potential cases and of falsely diagnosing noncases. In addition, these two estimates are *negatively related within studies* because lowering a threshold may enhance sensitivity but increase the likelihood that noncases are falsely identified as meeting the cutoff. As a result, these estimates are also related across studies. Thus, one estimate (either sensitivity or specificity) needs to be considered in the context of the other in the same analysis when pooling data from multiple sources (Reitsma et al., 2005). Failing to do so can lead to misleading or inaccurate conclusions.

One of the recent advances made in the meta-analysis methodological literature involves analysis of multiple, related outcome variables (see Jackson, White, & Thompson, 2010; see the Complex Research Synthesis section). The capacity to examine multiple related outcomes in a meta-analysis framework is an important advance when examining comparative advantages of various tools for diagnostic accuracy. In the field of medical research, Reitsma et al. (2005) reanalyzed data previously reported in a univariate meta-analysis that examined a single combined measure of sensitivity and specificity—the log of the diagnostic odds ratio (DOR; Scheidler, Hricak, Yu, Subak, & Segal, 1997)—using a bivariate meta-analysis. Their reanalysis provided contextualized, richer conclusions than the previous univariate meta-analysis of DOR (Scheidler et al., 1997). More specifically, Scheidler et al. concluded that three screening tools—lymphangiography (LAG), computed tomography (CT), and magnetic resonance imaging (MRI)—performed similarly when diagnosing lymph node metastases in women with cervical cancer. They further suggested that given the invasive nature of the LAG procedure, CT and MRI may be preferable over LAG. When both sensitivity and specificity were jointly estimated as two related outcomes in a bivariate meta-analysis, however, CT and MRI were found to be comparable in terms of sensitivity but LAG was significantly more sensitive than CT (Reitsma et al., 2005). In addition, LAG was lower in specificity compared to either CT or MRI. Thus, even though the three screening tools could not be distinguished in terms of their overall accuracy based on a single combined measure of sensitivity and specificity in the previous univariate meta-analysis (Scheidler et al., 1997), this new bivariate meta-analysis approach provided useful information about the relative accuracy of three different screening tools. Based on the new results, one may suggest that for high-risk individuals, the invasive nature of the LAG procedure be weighed less, in favor

of its better sensitivity, when making a decision about which screening tool to use. In contrast, for non-high-risk individuals, the lower specificity of the LAG procedure, which could lead to unnecessary anxiety, combined with its invasive nature, suggest that other screening procedures may be more preferable.

As illustrated in the example of cervical cancer screening tools, there is a value in knowing the unique estimates of both sensitivity and specificity from a clinical perspective when deciding which tools to use for whom. Given the purpose of screening measures, pooling data from independent studies can provide a *needed scale and diversity* in terms of both sample and clinical tool when examining relative advantages and disadvantages. In a way, it can emulate a multisite planned study without the enormous resource required. Similarly for etiological research, pooled data from multiple sources can provide a better sense of how strict or lenient one should be when deriving cutoff scores for positively meeting the diagnostic criteria, as the recommendation based on the finding from a single homogeneous sample may not be ideal for general populations. Screening individuals and reaching accurate diagnoses are the critical initial steps for formulating strategies for prevention and intervention. Therefore, research synthesis through innovative methods holds important promise for the field of developmental psychopathology.

Prevention and Intervention

In the era of evidence-based, health decision-making, central questions become those of *what works* (on the whole), *what works for whom* (subgroups or treatment modifiers), and *how it works* (mechanisms of behavior change). Although individual studies have long attempted to address these questions, there are good reasons to believe that findings from single studies alone may not be sufficient in guiding these evidence-based treatment decisions. First, there is a growing concern about the dismal reproducibility (replicability) in the biomedical and psychological research fields (Begley & Ellis, 2012; see also Ioannidis, 2005; Nosek, Spies, & Motyl, 2012), prompting further high-profile publications illustrating ironies about the *p*-values (Nuzzo, 2014) and calling for better standards for clinical trials (Begley & Ellis, 2012). Second, the effect sizes reported in single, typically small studies may be overestimated (Cumming & Maillardet, 2006; Kraemer, Mintz, Noda, Tinklenberg, & Yesavage, 2006). The magnitude of any intervention effect is important to correctly estimate to determine its cost effectiveness and to develop strategies for dissemination. However, effect size estimates vary widely across studies that differ in designs, measures, and participants; thus, research on the true magnitude of the intervention effect may be necessary via large, controlled multisite studies or meta-analysis studies.

The danger of overly relying on evidence from individual studies can be seen in the case of the efficacy of antidepressants in recent literature. Turner, Matthews, Linardatos, Tell, and Rosenthal (2008) meta-analyzed the data submitted to the Food and Drug Administration (FDA) between 1987 and 2004 (i.e., 74 individual trials for a total of 12 drugs and 12,564 patients), and compared their own findings with the published findings from the same trials. Their reanalyses indicated that effect sizes had been substantially overestimated in published studies (i.e., publication bias; see the Publication Bias and Selection Bias section). On average, Turner and colleagues found a 32% difference in effect size estimates between the FDA data and the published data. Thus, supporting evidence for intervention efficacy in single studies may be best viewed cautiously. Subsequent analysis found that this positive outcome bias was associated with deviations from study protocol, such as switching from an intent-to-treat analysis to a per-protocol analysis (Moreno et al., 2009). Similarly, a recent reanalysis of randomized clinical trial data reported that 35% of the reanalyzed studies required a different interpretation from that of the original article, including changed direction, magnitude, and statistical significance of findings (Ebrahim et al., 2014).

There is another source of bias in our perception of evidence for intervention efficacy. Researchers investigating interventions are more likely to cite previous studies with positive outcomes than equally valid studies with disappointing outcomes; this is referred to as the *optimisim bias* (also referred to as the *citation bias*; Chalmers & Matthews, 2006). This bias, along with the publication bias, can have harmful consequences, such as spending limited resources on less promising trials instead of spending time and resources to develop better interventions.

In the next section, we provide a review of quantitative methodologies that are aimed at synthesizing information from multiple studies. We focus on methods for AD meta-analysis, IPD meta-analysis, and IDA.

RESEARCH SYNTHESIS

The first known use of a meta-analytic method was by Karl Pearson in 1904. Pearson examined the association between inoculation and mortality from typhoid among soldiers who had volunteered for inoculation against

typhoid for their deployment in various places across the British Empire (O'Rourke, 2007). It was not until 1976 that the term *meta-analysis* was introduced by Gene Glass as "the statistical analysis of a large collection of analysis results from individual studies for the purpose of integrating the findings" and the method took off (O'Rourke, 2007; p. 580). Meta-analysis has caught on in many substantive fields, especially in medicine. For the past two decades, the Cochrane Collaboration (2013, http://www.cochrane.org/about-us) has been a major influence in the field of medical research, conducting over 5,000 systematic reviews that can affect evidence-based health decisions. Recent methodological advances in the field of meta-analysis include methods utilizing IPD and complex meta-analysis methods, such as multivariate meta-analysis and meta-regression. Below we review some of the considerations needed for conducting meta-analysis. We then review classical and model-based approaches to meta-analysis as well as complex research synthesis methods involving IPD or multivariate meta-analysis.

Inclusion Criteria for Studies

One of the first decisions in research synthesis involves which studies should be screened and combined. In meta-analysis, inclusion and exclusion criteria for selecting studies need to be carefully specified to protect against selection bias. There are two stages of screening in a typical meta-analysis. The first stage determines eligibility criteria that are based on the goal of the meta-analysis to ensure that only relevant studies are included. The second stage involves more careful screening and coding of relevant information (Berman & Parker, 2002).

As in any empirical research, research synthesis starts with certain goals that it is intended to meet. Is the study intended to derive average effect sizes of the relationships between emotion regulation strategies and symptoms of psychopathology in observational studies? Or is it to validate findings from a new intervention to general populations? Depending on the answers to these questions, studies to be included or excluded can be decided. For the former objective, it may be reasonable to include both cross sectional and longitudinal studies that are observational or etiological in nature, as well as experimental studies. For the latter question of the efficacy of a new intervention, however, it would be reasonable to eliminate any single studies that are not randomized controlled trials.

As an illustration, there are several meta-analysis or systematic review studies in the field of brief alcohol interventions for college students (Carey, Scott-Sheldon, Carey, & DeMartini, 2007; Carey, Scott-Sheldon, Elliott, Garey, & Carey, 2012; Cronce & Larimer, 2011; Larimer & Cronce, 2007). The review by Cronce and Larimer (2011), which was not quantitative but systematic, was intended to provide a quick overview of recent findings in the field of alcohol interventions for college students. Cronce and Larimer used the following inclusion criteria: studies should (1) report alcohol-focused behavioral outcomes from individual-focused preventive interventions for college students; (2) be published or conducted between 2007 and 2010; and (3) utilize a randomized controlled trial (RCT) or quasi-experimental design (i.e., one active condition and one control or wait-listed, and randomization). Similarly, Carey et al. (2007) included any published studies in their meta-analysis if studies (1) examined any educational, behavioral, or psychological alcohol intervention; (2) sampled college or university students; (3) used an RCT design; (4) assessed drinking behaviors as outcome measures; and (5) provided sufficient information to calculate between-group effect size estimates. These inclusion criteria set the boundaries of the generalizability of subsequent inference. Based on these inclusion criteria, a list of search terms may be developed to cast a wide net during the search phase. A flowchart (e.g., the *P*referred *R*eporting *I*tems for *S*ystematic reviews and *M*eta-*A*nalysis [PRISMA]; Moher, Liberati, Tetzlaff, Altman, & The PRISMA group, 2009) is often provided to show the systematic search and selection process of eligible studies, which looks like a CONSORT flowchart (Schulz, Altman, & Moher, 2010) for an RCT.

The American Psychological Association (APA) Publications and Communications Board Working Group (2008) developed the meta-analysis reporting standards (MARS), which are different from the journal article reporting standards (JARS) for typical empirical articles. The MARS includes specific recommendations for all sections of a meta-analysis. Specifically, for describing search strategies, the MARS recommends that the following information be provided:

- Reference and citation databases searched
- Registries searched and keywords used
- Time period in which studies needed to be conducted
- Other efforts to retrieve all available studies, including listserv queries and personal contacts made
- Method of addressing reports in languages other than English
- Process for determining study eligibility, including aspects of studies examined, number and qualification of judges, and their agreement
- Treatment of unpublished studies

And for coding procedures:

- The number and qualification of coders
- Interrater agreement and how disagreements were resolved
- Assessment of study quality
- How missing data were handled

In the case of IDA investigations of etiological and longitudinal studies, the number of similar studies may be fairly limited. Some of the studies considered for IDA investigations may be ongoing and complex, and data may not be available publicly. In such situations, the availability of the original investigators throughout all phases of IDA may become one of the important considerations. Etiological longitudinal studies focusing on developmental pathways of children of alcoholic parents (e.g., Hussong, Huang, Curran, Chassin, & Zucker, 2010) or twin or genome-wide association studies (GWAS; Schumann et al., 2011) that are geared toward understanding genetic susceptibility in the regulation of alcohol consumption may be such examples.

When original studies are under way at the same time as IDA investigations, an additional set of considerations at the outset of IDA investigations may be especially helpful. Hussong et al. (2013) recommended the following steps when building the supportive team science environment needed for IDA research: (1) decide how the responsibility and resources for data preparation will be divided up between the original study teams and the IDA research team; (2) make clear what the IDA study aims to answer that is different from the aims of original research projects; (3) coordinate research teams across manuscripts within the IDA study and the original study teams; (4) reflect the contributions of researchers in a balanced way when deciding publication credit; and (5) communicate about study differences and reconcile any discrepancies between IDA study findings and original study publications.

An alternative model of collaborations between the original and IDA investigators may be a prospective one. In the field of genetic epidemiology, there is a movement toward establishing collaborative networks of consortia prospectively from the onset of studies with the possibility in mind that their data may be utilized as an input data set for a larger investigation down the road (Ioannidis et al., 2006). In prospective collaborations, some of the critical design elements, such as phenotypes, exposures, end points, and analyses, can be coordinated in advance to be sufficiently similar or the same across studies within consortia (i.e., *networks of single studies*; see also Figure 23.4). This type of prospective collaborations among researchers can lessen the burden of labor-intensive data unpacking (Hussong et al., 2013) needed for typical IDA investigations in the future.

Publication Bias and Selection Bias

When planning a meta-analysis, one needs to consider the potential threat of publication bias. *Publication bias* or *file drawer problem* refers to the selective publication of studies with statistically significant outcomes (e.g., beneficial

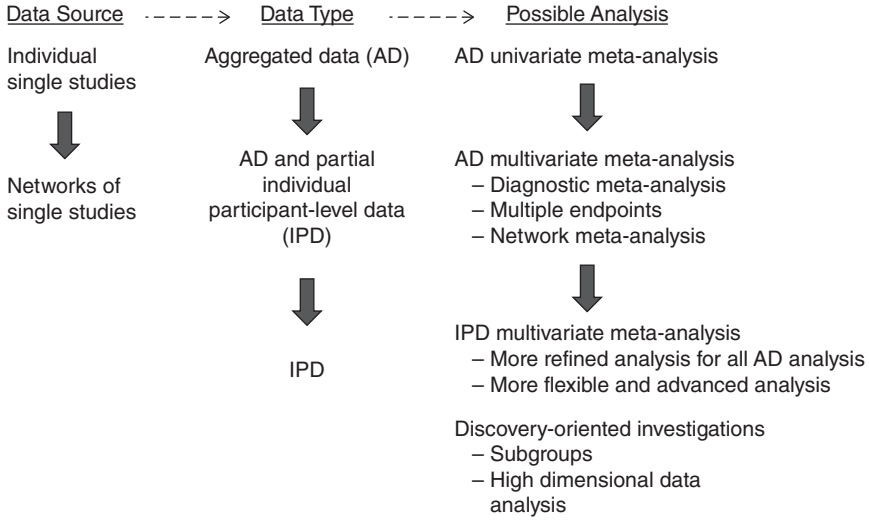


Figure 23.4 Current and future directions of research synthesis. Black arrows indicate the emerging directions and dotted arrows indicate the flow of research synthesis from data to analysis. Note that IPD approaches are computationally intensive and may not always be feasible.

intervention effects). The inclusion of unpublished data in meta-analysis may be helpful for avoiding or lessening publication bias. However, some authors have used the inclusion of only published studies to ensure some minimal research standards. As an alternative, individual studies can be scored on a number of items that indicate quality. Summed *quality scores* can then be used when deciding a minimum threshold score for studies to meet to be included or as study weights in a meta-analysis. However, it should be cautioned that quality scores can be calculated in many different ways, which can result in different conclusions (Herbison, Hay-Smith, & Gillespie, 2006), despite sharing common frameworks (Chalmers et al., 1981; Shadish et al., 2002) for assessing the validity of studies.

Publication bias can happen if oversampling of studies with significant findings occurs because they are more easily identifiable or accessible or because they are from colleagues in the same discipline (i.e., citation bias, availability/cost bias, familiarity bias; Borenstein et al., 2009). *Selection bias* can occur when studies are included or omitted for systematic reasons. For example, authors may include certain types of studies in meta-analysis while excluding or missing others selectively, which can result in a biased sample of studies. A recent review of meta-analysis studies published in the American Psychological Association and the Association for Psychological Science journals from 2004 to 2009 indicates that meta-analysis studies that include unpublished studies are just as likely to show evidence of publication bias as those that do not, due to selection bias (Ferguson & Brannick, 2012).

There are several ways to examine publication bias. A *funnel plot* is a graphical approach to examining publication bias. It displays the estimated effect sizes on the x axis and sample size or related measure (e.g., standard error) on the y axis for a sample of studies. It is based on the assumption that smaller studies will exhibit more variable effect sizes due to their lack of precision, and thus the scatter plot should look like a funnel. Figure 23.5 shows examples of a funnel plot. An asymmetrical funnel plot (bottom figure) suggests the presence of publication bias. Several other procedures also exist, such as the trim-and-fill method (Duval & Tweedie, 2000), Begg and Mazumdar's rank correlation test (Begg & Mazumdar, 1994), and Egger's regression asymmetry test (Egger, Smith, Schneider, & Minder, 1997). Nonetheless, the presence of this publication bias is not always examined or adjusted in a meta-analysis. Ferguson and Brannick (2012) found that 30% of the recent meta-analysis studies did not report checking publication bias in their sample of studies.

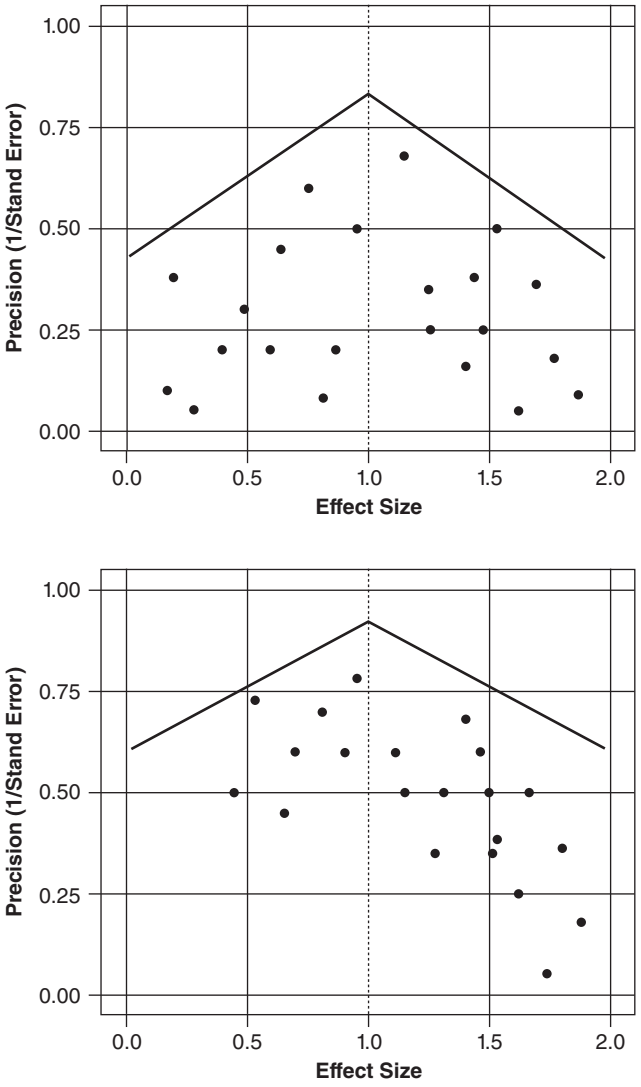


Figure 23.5 Symmetrical (top) and asymmetrical (bottom) funnel plots. The bottom figure shows evidence of publication bias.

Selection of Variables and Harmonization of Groups and Measures

Next, what are the outcomes of interest? For this question, outcome variables need to be operationally defined. In medical research, often an outcome is the occurrence of a certain binary event, such as morbidity (yes–no) and mortality (yes–no) as end points of a disease, and therefore can clearly be defined using a binary indicator. In other situations, a success may be operationally defined based on meeting cutoff scores on some indicator variables. For example, alcohol consumption can be quantified as the alcohol intake per day in grams of pure alcohol (e.g., Roerecke & Rehm, 2012) to examine the relationship

between alcohol use and ischemic heart disease (IHD). The average amount of alcohol consumed per day can then be categorized as less than 12 grams (one standard drink) or more than 24 grams (more than two standard drinks).

For psychological traits, however, it is more difficult to place individuals on common metrics (Curran & Hussong, 2009; Huo et al., 2014). Furthermore, the definition of a critical independent variable (e.g., intervention conditions) can be substantially different at the operational level across studies; thus, it must be made equivalent across studies before any synthesis can be conducted. In the IDA study of brief motivational interventions (BMIs) for college students (Mun et al., 2015), for example, we found that some of the intervention and control groups sharing the same labels were not equivalent across studies in terms of their intervention content, personalization, and other procedures. In the case of control groups, they differed, ranging from an assessment-only control group to a treatment-as-usual control group. In some studies, a treatment-as-usual group was closer to an educational group than to an assessment-only control group. We addressed this problem by carefully coding all intervention content materials and their delivery characteristics, which was carried out by two independent expert raters (Ray et al., 2014). Similarly, the operational definition of alcoholic parents can be slightly different across the three longitudinal studies of children of alcoholics included in the Cross Study Project (Hussong et al., 2013). Some of the between-study differences may be accounted for by adding covariates (e.g., COA and COA by study) in subsequent analytic models. When possible, however, it may be best to harmonize groups and derive equivalent measures across studies, prior to analysis, to reduce between-study heterogeneity. More important, any reported findings may be biased without the commensurate measures and groups established across component studies.

As for the selection of variables for outcome analysis, harmonization of measures is typically not required for meta-analysis using AD since the unit of analysis is already standardized. However, this is a serious challenge for IPD meta-analysis or IDA. The need for ensuring commensurate measures across studies is widely discussed as a notable barrier for IDA or meta-analysis using IPD, especially in psychological research (Cooper & Patall, 2009; Curran & Hussong, 2009). As indicated in the literature, the resources needed to establish commensurate measures for individuals within and across studies are quite extensive (Hussong et al., 2013; Mun et al., 2015, for detail). The task of establishing measurement equivalence starts from poring over codebooks and data to see if they are consistent across time within studies as well as across studies. If items are different in terms of the referent time frame, stem, or body of questions, or if response options are different across studies, they should be treated differently until these differences are resolved through harmonization processes. Some of the examples can be seen in Hussong et al. (2013) and Mun et al. (2015).

Once the demanding data preparatory work is completed, one can proceed to analyze measurement models specifically developed to meet the unique needs of each IDA study. In the field of psychological IDA research, item-level data have been analyzed via utilizing item response theory (IRT) based models or factor analysis (FA) based models. These models include a unidimensional, two-parameter logistic (2-PL) IRT analysis (Curran et al., 2008; Hussong et al., 2007), a multi-unidimensional 2-PL IRT analysis for multiple groups (Huo et al., 2014), a generalized partial credit model (Mun et al., 2015) for polytomous items, and longitudinal invariant Rasch test analysis (LIRT; McArdle, Grimm, Hamagami, Bowles, & Meredith, 2009). For other applications, moderated nonlinear factor analysis (MNLFA; Bauer & Hussong, 2009; Curran et al., 2014) has been utilized. Each analytical approach, although the goal of the task remains the same, has originated to address the unique demands of different IDA studies.

The entire process of establishing common metrics across studies, starting from checking data to estimating latent trait scores, can quickly become quite complex as the number of items in studies increases, as the number of studies increases, and as the number of observations or the observed duration increases. Liu, Liu, and Xie (2015) considered a new methodology to synthesize information from independent studies with heterogeneous designs, which may help to lessen the constraint that common design metrics exist across studies. But more research is still needed. Some of the challenging issues that arise from more complex situations include missing data, high dimensionality, and differential item functioning (DIF) (see Mun et al., 2015, for detailed discussion). Other measurement models, adaptations, and computing algorithms are expected to accommodate these challenges in the field of IDA research in the future.

Classical Meta-analysis Approaches

The earliest meta-analysis approaches combine *p*-values, which is particularly suitable for situations under which the only available data in original studies are *p*-values or *z* statistics. There are also a number of classical approaches

for synthesizing discrete and, in particular, binary data from independent studies. In this section, we review these approaches.

p-Value Combination

Assume that the p-values from original studies test the same null hypothesis. We can use Fisher's method (Fisher, 1948), which can be traced back to the 1930s, to combine the p-values and provide an overall inference. Consider the case with k studies. Fisher's method combines the p-values into a X^2 test statistic using the formula

$$X_{2k}^2 = -2\sum_{i=1}^{k} \ln(p_i) \sim \chi_{2k}^2$$

where p_i is the p-value of study i. Under the null hypothesis, the test statistic X^2 has a chi-squared distribution with $2k$ degrees of freedom. We often use this combined X^2 test statistic to obtain an overall p-value for combined inference. Another closely related approach is Stouffer's Z (Stouffer, Suchman, DeVinney, Star, & Williams, 1949). This approach converts p-values to Z scores first and then combines the resulting Z scores. More specifically, we first let

$$Z_i \sim \Phi^{-1}(1 - p_i)$$

where Φ is the standard normal cumulative distribution function. Then, the combined Z score

$$Z \sim \frac{\sum_{i=1}^{k} Z_i}{\sqrt{k}}$$

follows a standard normal distribution under the null hypothesis. Other p-value combination methods include Tippett's (Min) method, Max method, and Sum method, etc. (Marden, 1991).

Weights can also be applied in p-value combination methods to account for different sample sizes across studies and to improve the efficiency of the combination methods. For instance, in Stouffer's Z score method, if the Z score for study i is weighted by w_i, then the combined weighted Z score is

$$Z \sim \frac{\sum_{i=1}^{k} w_i Z_i}{\sqrt{\sum_{i=1}^{k} w_i^2}}.$$

Along with the unweighted Stouffer's Z score, the weighted Z score follows a standard normal distribution under the null hypothesis. Note that Stouffer's Z score method, as well as other p-value combination methods, can be used also for combining results from multiple independent tests within a single study (see Donovan, Wood, Frayjo, Black, & Surette, 2012, for an example of how this method can be used in applied research).

Combining 2 × 2 Tables

Mantel-Haenszel Method

Many outcomes of interest are binary, discrete event data. There can be many important, naturally binary outcomes, such as mortality or meeting the diagnostic criteria for disorders. Whether an adverse event occurred following a pharmaceutical trial is also one example. Even with continuous numerical data, dichotomizing may be preferable in some situations (Shentu & Xie, 2010) when there are errors in measurement or when obtained data can be naturally fluctuating due to their sensitivity to internal and external stimulation (e.g., blood pressure). Furthermore, dichotomized outcomes can be more straightforward to communicate to various stakeholders (Mun, Bates, & Vaschillo, 2010).

To explain some of the methods for binary data, Table 23.2 shows model setting for 2 × 2 tables for k independent studies, where X_i and Y_i follow a binomial distribution with fixed numbers n_i and m_i of binary observations, respectively. We denote $X_1, X_2, \ldots, X_k$ and $Y_1, Y_2, \ldots, Y_k$ as responses for k studies following binomial distributions with expectations expressed as p_1 and p_2, respectively; t_i stands for the total number of events for study i. For discrete binary data, odds ratio or log odds ratio is often used as an effect size estimate. Some studies also consider other measures, such as risk ratio, risk difference, etc. The goal of a meta-analysis for 2 × 2 tables is to synthesize the overall effect across k independent studies.

The Mantel-Haenszel (MH) method (Mantel & Haenszel, 1959) estimates common parameters from 2 × 2 tables under a fixed-effects model, which assumes a common odds

TABLE 23.2 Model Setting of 2 × 2 Tables for *k* Independent Studies

	Event	Nonevent	Total
Treatment	X_i	$n_i - X_i$	n_i
Control	Y_i	$m_i - Y_i$	m_i
Total	t_i	$N_i - t_i$	N_i

ratio across studies (see Table 23.2 for the model setting used). The MH method proceeds:

- Estimate odds ratio and its variance for each individual study:

$$\hat{\pi}_i = \frac{R_i}{S_i} = \frac{\frac{X_i(m_i - Y_i)}{N_i}}{\frac{Y_i(n_i - X_i)}{N_i}} \text{ and}$$

$$\widehat{Var}(\hat{\pi}_i) = \hat{\pi}_i^2 \left(\frac{P_i}{R_i} + \frac{Q_i}{S_i} \right)$$

$$= \hat{\pi}_i^2 \left(\frac{1}{X_i} + \frac{1}{n_i - X_i} + \frac{1}{Y_i} + \frac{1}{m_i - Y_i} \right) \text{ where}$$

$$R_i = \frac{X_i(m_i - Y_i)}{N_i}, \quad S_i = \frac{Y_i(n_i - X_i)}{N_i},$$

$$P_i = \frac{X_i + m_i - Y_i}{N_i}, \text{ and}$$

$$Q_i = \frac{Y_i + n_i - X_i}{N_i}$$

- Estimate the combined odds ratio from k studies:

$$\hat{\pi}_{MH} = \frac{\sum_{i=1}^{k} R_i}{\sum_{i=1}^{k} S_i} = \frac{\sum_{i=1}^{k} \frac{X_i(m_i - Y_i)}{N_i}}{\sum_{i=1}^{k} \frac{Y_i(n_i - X_i)}{N_i}}$$

and

$$\widehat{Var}(\hat{\pi}_{MH}) = \hat{\pi}_{MH}^2 \left[\frac{\sum_{i=1}^{k} P_i R_i}{2\left(\sum_{i=1}^{k} R_i \right)^2} + \frac{\sum_{i=1}^{k} \left(P_i S_i + Q_i R_i \right)}{2\left(\sum_{i=1}^{k} R_i \right)\left(\sum_{i=1}^{k} S_i \right)} + \frac{\sum_{i=1}^{k} Q_i S_i}{2\left(\sum_{i=1}^{k} S_i \right)^2} \right]$$

The MH method provides consistent and asymptotically efficient estimates when study sizes are large or when a large number of small studies are combined (Breslow, 1981). Standard error of estimate can be obtained by using the final previous equation (Robins, Breslow, & Greenland, 1986).

Peto's Odds Ratio

Peto's odds ratio method (Yusuf et al., 1985) is also commonly used for 2 × 2 table data in meta-analysis. Similar to the MH method, it yields a weighted log odds ratio estimate under the fixed-effects model framework (see the Model-Based Approaches section). However, this method is different from the MH method in the sense that, instead of using observed odds ratios, an approximated log odds ratio is calculated for each study and then combined. Hence, it is sometimes known as the Peto's one-step method or the "O–E" method since log odds ratio is estimated by using the observed number of events (O) and the expected number of events (E).

Peto's odds ratio method can be computed as follows:

- Estimate log odds ratio and its variance for study i:

$$\hat{\theta}_i = \frac{O_i - E_i}{V_i}$$

$$\widehat{Var}(\hat{\theta}_i) = \frac{1}{V_i}$$

where $O_i = \mathrm{X_i}$, $E_i = \frac{t_i n_i}{N_i}$, $V_i = \frac{t_i m_i n_i (N_i - t_i)}{N_i^2 (N_i - 1)}$

- Estimate the combined log odds ratio from all k studies:

$$\hat{\theta}_{Peto} = \frac{\sum_{i=1}^{k} O_i - \sum_{i=1}^{k} E_i}{\sum_{i=1}^{k} V_i}$$

and

$$\widehat{Var}(\hat{\theta}_{Peto}) = \frac{1}{\sum_{i=1}^{k} V_i}$$

For both the MH and Peto's methods, the confidence interval for the estimated log odds ratio can then be obtained by using the previous formula along with z critical value. The Peto's method, as well as the MH method, relies on the combined estimators approximating to a standard normal (z) distribution.

Both the MH and Peto's methods rely on large sample theory to justify the validity of the approaches. There is also an exact meta-analysis method for combining 2 × 2 tables based on Fisher's exact test and conditional likelihood inference (see, e.g., Gart, 1970). For the parameter of risk difference, Tian et al. (2009) proposed a simple

procedure to combine confidence intervals of risk difference for 2 × 2 tables without assigning any arbitrary numbers to empty cells in meta-analysis, and reanalyzed the controversial data reported in Nissen and Wolski (2007). For more recent discussions of exact meta-analysis of discrete data, see Liu et al. (2014).

Data Example

To illustrate combining discrete binary data across similar studies, we utilize the intervention data reported in Wilk, Jensen, and Havighurst (1997). Wilk and colleagues examined the effectiveness of brief alcohol interventions targeted for adult heavy drinkers. They searched for brief interventions that were (1) motivational in nature, (2) as short as 10–15 minutes, and (3) aimed to reduce drinking and related harm to treat nondependent heavy or problem drinkers. Two databases, MEDLINE and PsychLIT, were searched with the following, more specific inclusion criteria: (1) focus on alcohol abuse/dependence or heavy drinking; (2) focus on intervention and outcome; (3) human subjects between the ages of 19 and 65; (4) publication in English; and (4) a randomized control trial that includes a control group. The resulting sample consisted of 3,948 heavy drinkers across 12 clinical trials. The brief interventions common to all trials were short, and all sessions included feedback, education, and advice. The intervention outcome was measured by using a binary indicator of moderated alcohol use at 6–12 months post intervention. Of the 12 trials, eight reported outcome data. Table 23.3 shows 2 × 2 tables, observed odds ratios, and their associated confidence intervals (last column) for all eight studies as well as overall estimates from the Peto's one-step method and the MH method (the bottom two rows).

For the purpose of illustration, here we use the 2 × 2 table for the second study (Study 2 in Table 23.3). Moderation in use indicates that participants reduced their heavy drinking, whereas nonmoderation indicates no change in drinking at 6–12 months post intervention. Typically, odds ratio, as a measure of association, is highly skewed and asymmetrical. Thus, log odds ratio and its standard errors are often computed instead for inference. The log odds ratio for Study 2 can be calculated using

$$\text{Log OR}_2 = \log\left(\frac{14/66}{4/70}\right) = \log\left(\frac{14\times 70}{4\times 66}\right) = 1.31.$$

Next, we show how to compute the corresponding 95% confidence interval. First, the standard error of the log odds ratio 1.13 is

$$se_2 = \sqrt{\frac{1}{14}+\frac{1}{66}+\frac{1}{4}+\frac{1}{70}} = 0.59.$$

Assuming the log odds ratio is approximately normally distributed, its 95% confidence interval is

$$1.31 \pm 1.96 \times 0.59 = [0.15, 2.47].$$

TABLE 23.3 Pooling Data from 2 × 2 Tables Using the Peto's Odds Ratio and the Mantel-Haenszel Method

Article	Group	Moderation	Nonmoderation	Observed odds ratio	95% CI
			Individual study estimate		
1	Intervention	201	247	2.25	[1.70, 2.97]
	Control	122	337		
2	Intervention	14	66	3.71	[1.16, 11.85[a]]
	Control	4	70		
3	Intervention	9	24	1.09	[0.38, 3.11]
	Control	10	29		
4	Intervention	391	367	1.80	[1.40, 2.33]
	Control	134	227		
5	Intervention	9	50	1.74	[0.44, 6.95]
	Control	3	29		
6	Intervention	22	27	3.26	[1.25, 8.49]
	Control	8	32		
7	Intervention	34	35	2.14	[1.05, 4.34]
	Control	20	44		
8	Intervention	34	102	1.23	[0.60, 2.54]
	Control	13	48		
			Pooled estimate		
Peto's odds ratio				1.95	[1.66, 2.30]
Mantel-Haenszel odds ratio				1.98	[1.67, 2.34]

Source: Wilk et al. (1997).
[a] = the upper limit of this *CI* is different from the text due to rounding.

This log odds ratio can then be converted back to odds ratio as follows:

$$\mathrm{OR}_2 = \exp(\mathrm{Log\ OR}_2) = \exp(1.31) = 3.71.$$

Similarly, the 95% confidence interval for the odds ratio can be obtained by exponentiation of lower and upper limits of the log odds ratio:

$$[\exp(0.15),\ \ \exp(2.47)] = [1.16, 11.82].$$

The MH estimate can be obtained by combining these odds ratios across studies via the combining equation shown previously. The MH odds ratio estimate was 1.98 with its 95% confidence interval ranging from 1.67 to 2.34.

Whereas the MH method combines observed odds ratios only, the Peto's method uses both observed and expected odds ratios (see the Peto's Odds Ratio section). Peto's log odds ratio for Study 2 can be calculated as

$$\mathrm{Log\ OR}_2^{\mathrm{P}} = \frac{14 - (14+4)\times(14+66)/(14+66+4+70)}{\mathrm{V}_2} = 1.16$$

and its associated standard error

$$\mathrm{V}_2 = \frac{(14+4)\times(4+70)\times(14+66)\times(66+70)}{(14+66+4+70)^2\times(14+66+4+70-1)} = 4$$

with

$$se_{\mathrm{Log\ OR}_2^{\mathrm{P}}} = \sqrt{\frac{1}{\mathrm{V}_2}} = 0.5.$$

If we convert the Peto's log odds ratio to odds ratio, it becomes 3.19, which is different from the previous observed odds ratio 3.71 obtained using the MH method.

The combined overall odds ratio across all eight studies using the Peto's method was 1.95 with its 95% confidence interval ranging from 1.66 to 2.30. The combined overall estimate is also different from that obtained by using the MH method. However, both estimates suggest we reject the null hypothesis. Based on the size of the overall odds ratio, our interpretation is that those who participated in the brief interventions were almost twice as likely to moderate their drinking compared to controls. The results from both methods are expected to be similar given that the rate (37%) of the outcome (i.e., moderation in drinking in the combined sample) was not too severely low, and also because the two randomized groups were similar in size. In other extreme data situations, the two methods can result in different conclusions. It has been shown that for meta-analysis of rare events (e.g., 1% or less), the Peto's method is the least biased and most powerful method, according to a simulation study that evaluated several methods, including the MH method (Bradburn, Deeks, Berlin, & Russell Localio, 2007). Bradburn et al. also observed that the Peto's method can yield biased results when sample sizes substantially differ between groups (e.g., treatment and control) in 2 × 2 tables, and that it yields worse results than the MH method (conducted without any corrections for zero cells) when the rate of event is not that extreme (5% or 10% as opposed to 1% or less).

Model-Based Approaches

There are at least two major sources of variation to consider when combining statistics (e.g., mean difference) across studies. First, *sampling variability* may vary across studies. Studies with larger samples have more information and hence better precision, which can be indicated in taller and narrower distributions. In contrast, studies with smaller samples have shorter and wider distributions and less precision surrounding their means (Figure 23.6). Second, the statistics of interest can be assumed to have a common value (Figure 23.6) or to have come from an underlying distribution of study-specific values (Figure 23.7). The first source of variation refers to *within-study variability* and the latter addresses *between-study variability*.

Fixed-Effects Model

A fixed-effects model assumes that each study summary statistic, Y_i, is a realization from a population of study

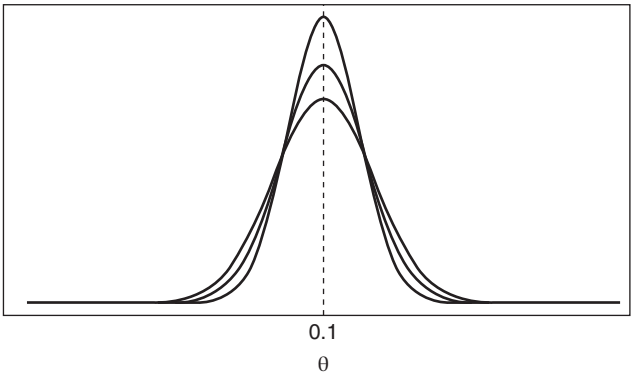


Figure 23.6 A fixed-effects model. It assumes a common underlying mean θ and different variances s_i^2 due to sampling variability for study i. This figure was drawn based on Figure 3 from Normand (1999).

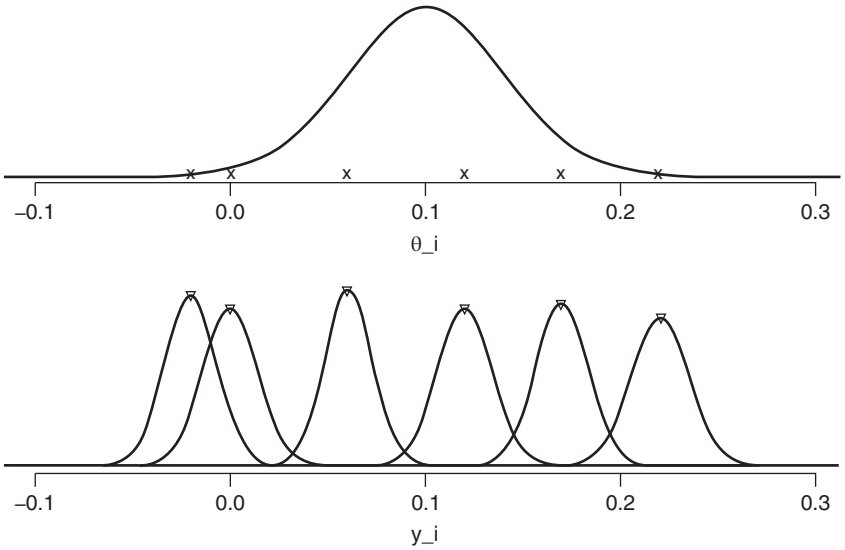


Figure 23.7 A random-effects model. It assumes a superpopulation with a mean θ and a variance τ^2 (top figure) from which distributions with different means θ_i and variances s_i^2 (bottom figure) are drawn. Figure 23.7 was drawn based on Figure 4 from Normand (1999).

estimates with a common true value θ. Let θ be the parameter of interest (e.g., mean intervention effect size) and there are $i = 1,\ 2, ..., k$ independent studies. Then, θ value quantifies the average effect size across k studies, which is assumed to be the same across studies (see Figure 23.6 for a graphic illustration) so that there is no subscript for θ. Variance of summary statistic, Y_i for study i is assumed to be known, which is denoted as s_i^2. This variance estimate can differ across studies and is indicated by subscript i. Within-study variability s_i^2 can be seen as tall and skinny (better precision) vs. short and wide (less precision) distributions in Figure 23.6.

A fixed-effects model usually takes the inverse variances (s_i^2) of the estimates as weights when pooling data. Between-study variability surrounding θ is assumed to be essentially zero or nothing more than what is expected from random errors. This can be more formally expressed as

$$Y_i \sim N(\theta,\ s_i^2) \text{ for } i = 1, 2, \ldots, k.$$

To see if this assumption holds, the test of homogeneity across studies is conducted by using the Q statistic (Cochran, 1950)

$$Q = \sum_i W_i (Y_i - \overline{Y}_w)^2$$

where W_i is the inverse of the sampling variance for study i and $\overline{Y}_W = \sum_i W_i Y_i / \sum_i W_i$ is the weighted estimator of the effect of interest (e.g., estimated mean intervention effect). Under the null hypothesis, Q follows a chi-squared distribution with $k - 1$ degrees of freedom. This test quantifies the extent of heterogeneity by examining the proportion of the total variation in point estimates that can be attributed to heterogeneity across studies. If the Q test is rejected, we can conclude that there exists significant variation across studies due to many reasons, including differences in study designs, procedures, measures, and samples. Note that this Q test suffers from low power (Sutton & Higgins, 2008), and I^2 test (Higgins & Thompson, 2002) may be an alternative. More important, it may be unrealistic and too strict to assume that there is one true common effect size across all studies and that no variability exists surrounding the true effect size (Jackson et al., 2010; Thompson & Pocock, 1991).

Random-Effects Model

A random-effects model assumes that each study summary statistic, Y_i, comes from a study-specific distribution, and that the expectations of these study-specific distributions follow a common distribution. Hence, in addition to within-study variability s_i^2, there is study-to-study variation surrounding the underlying common effect θ, which can be expressed as variance τ^2. The top figure in Figure 23.7 shows the underlying superpopulation with a mean θ and a variance τ^2, from which study-specific true effect size estimates are drawn.

These relationships can be expressed more formally as follows:

$$Y_i \,|\, \theta_i,\ s_i^2 \sim N(\theta_i,\ s_i^2) \text{ and } \theta_i \,|\, \theta,\ \tau^2 \sim N(\theta,\ \tau^2).$$

Hence, the distribution of each study summary measure, Y_i, after averaging over the study-specific effects, is normally distributed with mean θ and variance $s_i^2 + \tau^2$. The distribution of θ_i can be helpful for identifying subgroups of intervention procedures that are more effective. When between-study variability $\tau^2 = 0$, random-effects models reduce to fixed-effects models. Given that between-study variability τ^2 is typically greater than zero, fixed-effects models will almost always have narrower confidence intervals than random-effects models. Likewise, the larger the τ^2, the wider the confidence intervals for the effect of interest. This is because the underlying between-study variation of the effect is incorporated into weights in a random-effects model (DerSimonian & Laird, 1986).

Estimators of Variance

Most commonly used methods for estimating variance are the maximum likelihood estimator (MLE), restricted maximum likelihood (REML), DerSimonian and Laird (DL; DerSimonian & Laird, 1986), method of moments, and Bayesian (see Normand, 1999, for a review of estimators, including equations and detailed discussion). To obtain θ estimates in a model-based model, the MLE and Bayesian methods are used. The REML, DL, and method of moments are used to estimate between-study variance in a random-effects model. Note that both fixed-effects and random-effects models are typically based on the assumption of a normal distribution. Sensitivity to this distributional assumption or to any individual study may need to be examined (Normand, 1999). For example, leaving one study out of analysis in a series of analyses and examining the stability of the estimates across these analyses may be performed as part of sensitivity analysis. However, there may be limited options when a few studies are analyzed.

Fixed-Effects Versus Random-Effects Model

Synthesizing data from sufficiently similar studies that have some variation in measures, samples, and designs has an important benefit in that it provides new understanding about the boundaries of the effect being examined. When the evidence of a significant difference exists across studies based on the Q test of homogeneity, it is often used as the basis for choosing a random-effects model over a fixed-effects model. However, the significant Q test can also be used to imply that at least one study distinctively differs from others in the effect size estimates examined. In a meta-analysis of intervention studies, this may indicate the presence of an important subgroup in which the efficacy of intervention substantially differs from the rest. Thus, pooling these heterogeneous intervention studies into one model may not make sense when estimating the overall intervention effect size. For example, if there are outlier studies with strongly negative intervention effects, it may not be informative to include them with the rest of the studies that have clear positive intervention effects, only to yield no overall effect. Rather, it may be more useful to pool data only from *sufficiently similar* studies. Alternatively, the magnitude of heterogeneity across studies can be reduced by including informative covariates in the model. Covariates can be study-level variables for an AD meta-analysis and individual- and study-level variables for an IPD meta-analysis.

Otherwise, when heterogeneous studies are combined in a fixed-effects meta-analysis model, studies with larger samples can dominate the combined estimate in a meta-analysis, essentially throwing away studies with small samples (Al khalaf, Thalib, & Doi, 2011). Similarly, pooling data from heterogeneous studies in a random-effects model may yield findings that may shift an inverse variance weighted meta-analysis back toward an unweighted mean estimate (Al khalaf et al., 2011).

The decision of which model to use may depend on other practical considerations. When there is a sufficiently large number of similar studies with normally distributed outcome data (i.e., effect size estimates or IPD outcome variable), a random-effects model can be used, in which studies are viewed as a random sample drawn from a superpopulation of studies and, consequently, any resulting inference can be generalized back to this broad population. If there are a limited number of studies, however, one may need to be cautious about using a random-effects model, since in this case the between-study variability τ^2 may not be estimated accurately and the quality of the overall meta-analysis estimator may be affected. In such a situation, a fixed-effects model can be used not to draw broad inference but to obtain specific summary results about the pooled studies (see also Borenstein, Hedges, Higgins, & Rothstein, 2010).

Last, when the number of individual studies is finite, Claggett, Xie, and Tian (2014) proposed an alternative, resampling-based *nonparametric meta-analysis* approach that is more flexible than both random- and fixed-effects models. Note that, in the fixed-effects and random-effects models, we often assume that the underlying study-level parameters are either exactly the same across individual studies and that they are realizations of a random sample from a population (often normal), respectively. In the approach by Claggett et al., one needs to assume only that the study-level parameters are unknown fixed

parameters. This approach draws inferences about, for example, the quantiles of the set of parameters using study-specific summary statistics. This nonparametric approach proposed by Claggett et al. is more flexible than the fixed-effects model method, because it allows the underlying study-level parameters to be different across different studies. It is also more flexible than the random-effects model method because it does not need to limit the underlying study-level parameters to be from a normal distribution or any other assumed known distribution, and also because it accommodates any population of the underlying study-level parameters.

Unifying Approach to Synthesize Data from Multiple Studies

Xie, Singh, and Strawderman (2011) proposed a broad, unifying approach to combining data from multiple studies. This approach can encompass all of the procedures reviewed previously, including those from model-based approaches (i.e., fixed-effects and random-effects models using MLE or Bayesian estimator) under one inclusive theoretical umbrella. This approach by Xie et al. is more comprehensive than combining point estimates (e.g., means) or *p*-values like Stouffer's Z or confidence interval estimates from multiple studies. When combining data, it uses the so-called confidence distribution (CD). CD is a sample-dependent distribution function that can represent confidence intervals of all levels for a parameter of interest. It uses a distribution function, instead of a point (point estimator) or an interval (confidence interval), to estimate a parameter of interest. For example, in the simple normal example with sample $x_i \sim N(\mu, 1)$, for $i = 1, \ldots, n$, the CD approach invokes the distribution function $N(\overline{x},\ 1/n)$ to estimate, instead of estimating just a point estimate, sample mean $\overline{x}$ and 95% confidence interval $(\overline{x} - 1.96/\sqrt{n},\ \overline{x} + 1.96/\sqrt{n})$, for the parameter of interest, μ. Analogous to a Bayesian posterior distribution that contains a wealth of information for Bayesian inference, CD contains rich information for constructing frequentist inference (Xie & Singh, 2013). Unlike a few point estimators or confidence intervals, CD contains information about not only important point estimators—mean, median, and mode—but also confidence intervals of all levels and *p*-values for various tests. Depending on the set up, the CD approach to combining data can be used to make either *approximate* (i.e., justified based on large sample theory) or *exact inference* for the parameters of interest. The CD combining approach has also been generalized for *robust meta-analysis* that is resistant to errors in data, misspecified models, and outlying or bad studies (Xie et al., 2011). An R package *gmeta* has been developed to combine data using conventional as well as robust meta-analysis approaches (Yang & Xie, 2010).

Complex Research Synthesis

Complex research synthesis, although somewhat arbitrary to define, involves models that (1) incorporate evidence on multiple related parameters (e.g., multiple intervention comparisons or multiple end points—two or more related outcome variables), (2) specifically model data from different study designs (e.g., experimental studies and observational studies), or (3) involve raw IPD (Sutton & Higgins, 2008). In other words, complex research synthesis is a collection of emerging analytical approaches that are aimed at taking full advantage of existing data to respond to research questions not easily addressed in a single individual study or in a typical AD meta-analysis.

Multivariate Meta-analysis

Multivariate meta-analysis generally refers to the joint synthesis of multiple related parameters across studies, which can be found in terms of multiple intervention conditions, covariates, or outcomes, and repeatedly observed single measures. These variables are correlated within studies by their nature and are referred to as the *within-study correlation*. In contrast, the *between-study correlation* indicates how the true estimates are related among studies. A multivariate model takes into account within-study correlations, which allows one to *borrow strength* from other related data and obtain more desirable estimates. Riley (2009) demonstrated in a simulation study that ignoring within-study correlations results in increases in the standard errors of overall effect estimates (precision) and mean-square error (between-study variance), and results in bias when nonignorable missing data exist. The diagnostic application example we discussed in the Screening and Diagnostic Tests section (i.e., Reitsma et al., 2005) utilized a bivariate meta-analysis for the joint estimation of two related estimates (i.e., sensitivity and specificity). The utilities of multivariate meta-analysis, however, extend much beyond diagnostic meta-analysis. Multivariate meta-analysis methods can be utilized for synthesizing treatment effects on multiple related outcomes (also called *multiple end points*), multiple interventions, or exposure groups (also called *network meta-analysis*; see Jackson, Riley, & White, 2011, for a review).

More formally and generally, multivariate meta-analysis can be shown as below. When multiple outcomes are

synthesized using the framework of a random-effects model, we typically assume the following multivariate model:

$$\mathrm{Y}_i|\theta_i, \mathrm{S}_i \sim N(\theta_i, S_i)$$
$$\theta_i|\theta, \sum \sim N\left(\theta, \sum\right)$$

where Y_i denote the $p \times 1$ vector of outcomes of interest for study i; θ_i is the true underlying effect for study i; and S_i is the within-study covariance matrix, which is assumed to be known. N denotes the multivariate normal distribution with dimension p. We also assume that θ_i varies from study to study and follows a multivariate normal distribution with overall mean θ and between-study covariance matrix Σ. The estimate $\hat{\theta}$ can be expressed in terms of $\hat{\Sigma}$:

$$\hat{\theta} = \left(\sum_{i=1}^{n}\left(S_i + \hat{\Sigma}\right)^{-1}\right)^{-1}\left(\sum_{i=1}^{n}\left(S_i + \hat{\Sigma}\right)^{-1}\mathrm{Y}_i\right)$$

where $\hat{\Sigma}$ can be obtained by using the MLE, REML, method of moments, or Bayesian method. The estimated variance is

$$\mathrm{Var}(\hat{\theta}) = \left(\sum_{i=1}^{n}\left(S_i + \hat{\Sigma}\right)^{-1}\right)^{-1}.$$

If some studies have missing data for the vector of interest, one way of incorporating these studies with missing data in multivariate analysis may be to replace the missing data with estimates with a negligible weight and information. This can be achieved by setting very large within-study variances to the missing data points and by constraining within-study correlations with other end points to be zeros (Jackson et al., 2010). Thus, multivariate meta-analysis methods achieve better efficiency in using all available information than univariate methods.

The idea that multivariate meta-analysis should take into account the correlated nature of data when there are multiple end points is not new, although its utilization has been rare in applied research. Jackson and colleagues (2011) summarized the following five advantages of multivariate meta-analysis over univariate methods: First, we can obtain estimates for all effects in a single modeling framework. A single integrated multivariate meta-analysis is more elegant than multiple univariate ones. Conceptually, it is analogous to conducting a multivariate analysis of variance (MANOVA) as opposed to conducting a series of analysis of variance (ANOVA) when there is a need to analyze multiple related outcomes. Second, we can utilize the relationships among the multiple effects examined when making inferences. For example, in quadratic growth curve models, linear and quadratic growth parameters are often negatively associated and this information can be interpreted and used in a multivariate analysis. Similarly, sensitivity and specificity estimates are negatively associated and this information can be used when jointly estimating the region of significance.

Third, we can obtain parameter estimates with better statistical properties. When we utilize the correlations among related end points in a multivariate analysis, each end point can borrow strength from the other related end points, resulting in smaller standard errors for the overall estimates compared to those from separate univariate models. Moreover, it also results in smaller mean square error of the between-study variance. This gain in estimation is directly related to the within-study correlation. As the within-study correlation increases, we generally obtain better estimates. Fourth, we can make potentially different clinical conclusions, compared to those from multiple univariate meta-analyses. By providing all results in a single multivariate meta-analysis, it is easier to compare the results than it is from different analyses that make different assumptions. Fifth, the multivariate methods have the potential to reduce bias due to partial reporting of data. These advantages apply to both AD and IPD analysis. With IPD, however, the implications of the multivariate meta-analysis methods significantly change. Its promise is very appealing and clear. At the very least, within-study correlations can be directly estimated using IPD. More important, data can be newly and directly analyzed using IPD with advanced analytical models.

Multivariate meta-analysis is particularly well suited for use in prevention and intervention research. Many behavioral outcome measures are highly correlated and typically not fully reported in publications. When multiple related outcome data exist in studies and when they are examined one by one (and separately at each follow-up) or selectively, it raises the possibility of chance findings, especially in studies with small samples (Tversky & Kahneman, 1971). This research practice can lead to misleading conclusions with regard to whether the intervention is efficacious overall. In some situations, investigators may selectively report some outcomes and omit others when publishing, which is a questionable research practice that should be avoided (Bakker, van Dijk, & Wicherts, 2012). Given that the average behavioral intervention effect is generally small, the likely conclusion one can make under

this questionable research practice is that an intervention has an effect for some of the outcome variables but not for others.

When it comes to univariate meta-analysis using AD, multiple related outcomes are analyzed separately one by one, which can result in different samples for different analysis because not all studies report or have those outcome measures. As a consequence, each meta-analysis includes only those studies that have the measure being analyzed. Any different conclusions across different outcome measures from univariate meta-analysis methods can be attributed to different studies or samples being included, as well as to different intervention benefits for different outcomes. For a relatively understudied outcome variable, it is also possible to have just a few available studies, which seriously limits our ability to draw any firm conclusions in a typical univariate meta-analysis. Multivariate meta-analysis is a promising new approach, especially when the method is used with IPD. Below, we discuss how the multivariate meta-analysis methods can be used for comparing multiple intervention groups.

Network Meta-analysis

Network meta-analysis refers to synthesis of a network of trials connected by evidence (Jansen et al., 2011). When a multivariate meta-analysis is performed for comparing multiple intervention groups, it can be viewed as a network meta-analysis (Hoaglin et al., 2011; Jansen et al., 2011). This approach enables us to draw conclusions about relative intervention effects of different intervention approaches that have not been compared head-to-head in a trial using a network of direct and indirect evidence (Jansen et al., 2011). It takes advantage of a greater share of available evidence than a traditional meta-analysis of RCTs, which typically features comparing just two arms (i.e., intervention and control). Furthermore, when a new intervention surfaces, it is rarely compared against another, effective intervention approach. As a result, there is a dearth of evidence suggesting relative benefits when attempting to make evidence-based health decisions. Via network meta-analysis, the existing interventions can be contrasted with new ones through connected evidence, even when they were never directly compared.

To know the relative strengths of all available interventions, it may be best to include them in a large-scale study and compare them pairwise to derive estimates of relative intervention effects. However, with limited resources, this is practically impossible to carry out. In the absence of direct head-to-head comparisons of competing interventions, network meta-analysis utilizes networks of direct and indirect comparisons to derive relative intervention effect size estimates between competing interventions. Figure 23.8 illustrates the concepts of direct and indirect evidence. The solid lines between ovals indicate that direct head-to-head intervention comparisons are possible. The dotted lines indicate indirect comparisons are possible. As long as indirect pairwise comparisons can be anchored, indirect comparisons of multiple intervention conditions are possible. For example, head-to-head pairwise comparisons of intervention conditions A and B and A and C are anchored on the common condition A. With A serving as the anchor, relative effects of intervention conditions B versus C can be obtained indirectly (the figure on left). Intervention conditions B and C are connected indirectly through the condition A. This is the essence of an indirect comparison through using an anchor intervention in a network of evidence. The figure on the right shows that both direct and indirect comparisons are possible between intervention conditions B and C. Many more intervention conditions can be compared even though they are not directly compared in any trials. For example, intervention conditions B and D, B and E, C and D, C and E, and D and E are possible due to the anchor intervention A. As more intervention conditions are linked to the existing network, more interventions can be directly and indirectly compared.

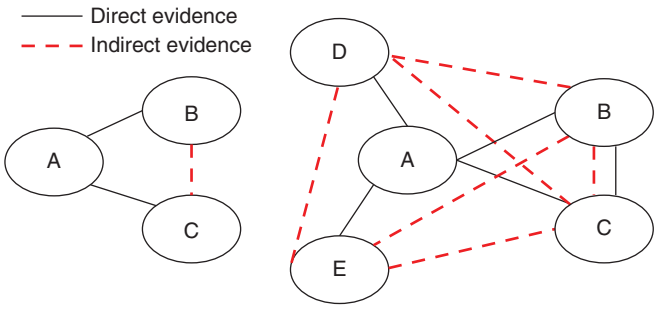


Figure 23.8 Network meta-analysis. The figure on the left shows an example of an indirect comparison between interventions B and C. The figure on the right shows mixed networks of evidence (both direct and indirect) for interventions B and C. Interventions B and C are connected via both solid and dotted lines. See footnote 1.

It is important to note that there are necessary assumptions for network meta-analysis to be valid—similarity and consistency (Jansen et al., 2011). The assumption of similarity necessitates that trials included in relative comparisons should be *sufficiently similar* in terms of participant characteristics, protocols, and measures and that any between-study variation in these characteristics should not systematically modify the intervention effect across studies. The assumption of *consistency* dictates that

when both direct and indirect comparisons are possible in a network of connected evidence (Figure 23.8, on the right side), the evidence from direct and indirect comparisons should be consistent. Jansen and colleagues (2011) recommended that network meta-analysis be used when the evidence base consists of more than two RCTs connecting more than two interventions.

IPD Meta-analysis and Integrative Data Analysis

As discussed thus far, IPD meta-analysis or IDA can be conducted to address whether the effect of interest exists across the board and how large the effect is at the population level. When utilized in the context of multivariate meta-analysis for multiple related parameters, IDA or IPD meta-analysis can be a particularly compelling approach because the advantages of multivariate methods can be fully exploited.

At the same time, some cautionary comments about IPD are needed. When IPD is analyzed in a large study, one can achieve much needed precision about point estimates and standard errors. In a small IDA project with a few studies, however, it is difficult to obtain such precision, especially when considerable between-study variability exists. For this reason, there should be a sufficiently large number of studies available for IPD meta-analysis and consequently greater resource, compared to AD meta-analysis (Mun et al., 2015; Steinberg et al., 1997). For any population-based inference, some researchers have suggested that 10–20 studies may be needed for its population representation and validity of parameter estimates (Hussong et al., 2013). In relation to this population based inference, it is also important to communicate how the set of studies included in IDA relates to the body of existing studies in the literature.

In this section, we focus on combining IPD for comparing multiple intervention groups across studies. Whenever there are multiple competing intervention conditions in some of the studies, data are typically combined and analyzed only from the studies that have the target intervention groups, and this analysis is repeated for all intervention pairs. However, the downside of this approach is that the studies being included in each set of analysis can be different, and any effect size estimate cannot be directly compared because uncertainty surrounding the estimates can be due to the different samples analyzed. Furthermore, it is unreasonable to assume that these interventions or multiple outcomes are independent within studies. Thus, comparing intervention conditions separately is an inefficient use of data, and can also lead to misleading and/or biased estimates.

In a multivariate meta-analysis, we can include all conditions across studies as long as they can be linked together by utilizing the correlational structure among these conditions. In the current chapter, we illustrate a new two-stage multivariate meta-analysis for IPD. We use the methodology proposed by Liu et al. (2015) but extend this method for IPD. This analysis proceeds in two stages. At the first stage, the analyst formulates a model to run for each data set based on a substantive conceptual model. This model can be flexibly designed by the analyst based on the research goals and data characteristics of the study. It can be a latent curve model with polynomials, piecewise model, or mixed-effects model. In addition, an intervention effect can be variously defined and derived. It can be derived from a model that includes baseline outcome levels. Alternatively, one can specify an intervention effect on the rate of growth of an outcome behavior. In addition, different distributions of outcome measures can also be accommodated by selecting a model of the appropriate form.

At the second stage, the vectors of estimated coefficients of the selected model across studies are analyzed to obtain the vector of overall estimates and its covariance matrix. For the second step, different estimators for the between-study covariance matrix, such as the MLE or method of moments, can be utilized. If estimates for some of the covariates are not available from the first stage analysis of individual data sets because the covariates were not assessed originally or because estimates were not identifiable (e.g., all participants were men), the coefficients for these covariates for some specific studies would be selectively omitted when estimates are combined at the second stage. For example, if there is a total of five coefficients in the formulated model, three may exist in one study but four in another. It is possible that none of the studies has all five coefficients. The design differences across studies are accommodated by applying appropriate mapping matrices (see Liu et al., 2015 for technical details). This multivariate meta-analysis approach is well suited for IPD because IPD can be analyzed separately for each study using an advanced or complex model and, consequently, resulting coefficients and their within-study covariance matrices can be directly obtained. For a multivariate meta-analysis with AD to be feasible, within-study covariance matrices should be reported in publications in the first place or made available subsequently. However, this IPD approach may not be feasible in some situations. Although there are no restrictions as to the number of coefficients that can be analyzed and combined, it can quickly become challenging as the number of coefficients or studies increases and as sparseness goes up.

To present the method more formally, we first assume that the model to be analyzed at the first stage is

$$y_{ijt} = \beta_i^T x_{ijt} + u_{ij} + \varepsilon_{ijt}$$

where y_{ijt} is the outcome score for participant j at time t in study i, x_{ijt} and β_i are the corresponding design and coefficient vectors, respectively. u_{ij} is the random intercept for participant j in study i, and ε_{ijt} is the residual error term. Each β_i is a subset of the vector β, and its estimate $\widehat{\beta}_i$ and the covariance matrix of $\widehat{\beta}_i$ denoted as $\widehat{\Sigma}_i$, are obtained separately for each study (first stage) and subsequently combined across studies (second stage).

At the second combining stage, we assume a hierarchical model with the form

$$\beta_i|\theta_i, S_i \sim N_{P_i}(\theta_i, S_i);$$
$$\theta_i|\theta, \Sigma \sim N_P(A_i\theta, A_i\Sigma A_i^T)$$

where β_i and θ_i are the observed and true parameter vectors for study i, respectively. S_i is the covariance matrix of θ_i, which is typically assumed to be known. θ and Σ are the population parameters and the corresponding covariance matrix Σ can be estimated by many estimation methods, such as the REML. A_i is the mapping matrix for study i that indicates missing data and maps θ to θ_i. The particular estimation approach utilizes the CD method (see the Unifying Approach to Synthesize Data from Multiple Studies section) and explicitly incorporates the overall structure of missing data into the model through a mapping matrix A_i. If there are no missing data points (i.e., covariates or coefficients), A_i becomes an identity matrix. The CD estimator consequently becomes equivalent to other estimation methods (see Jackson et al., 2011), which is extremely unlikely for any IPD analyses in behavioral and clinical research.

When applying the CD method, the combined estimates of θ and its covariance, denoted as $\widehat{\theta}_E^{(c)}$ and $S_{c,E}$, respectively, have the following forms:

$$\widehat{\theta}_E^{(c)} = \left(\sum_{i=1}^{n} A_i^T \left(S_i + A_i\widehat{\Sigma}_i A_i^T\right)^{-1} A_i\right)^{-1} \times \left(\sum_{i=1}^{n} A_i^T \left(S_i + A_i\widehat{\Sigma}_i A_i^T\right)^{-1} A_i A_i^T \beta_i\right)$$

and

$$S_{c,E} = Var(\widehat{\theta}_E^{(c)}) = \left(\sum_{i=1}^{n} A_i^T \left(S_i + A_i\widehat{\Sigma}_i A_i^T\right)^{-1} A_i\right)^{-1}.$$

These equations are used when combining estimates of θ and its covariance. This multivariate meta-analysis approach for comparing multiple intervention groups using IPD can be seen as a network meta-analysis application when multiple interventions are compared across studies. At the same time, this multivariate approach using IPD is more general and flexible than typical network meta-analysis methods using AD in the sense that the effects combined in the former reflect input from all available data. Furthermore, any data and model can be combined in principle. Thus, this new two-stage approach using IPD is not restricted to what has been reported by primary studies.

It is also important to note that IDA is not limited to a large number of studies. It can be conducted to pool just two or three non-RCT studies into one. When two or more non-RCT studies are pooled into one, the resulting pooled data can still enjoy many advantages of IDA. The pooled data set would be more heterogeneous in terms of samples and study characteristics, be larger in sample size, and have more follow-ups for a longer duration. Overall, combined data from two studies may be better than data from a single study, if the measurement equivalence can be established across the studies being pooled. IDA or IPD meta-analysis, regardless of the nature of its component studies, is highly innovative. Not only does it strengthen causal inference based on existing data, but more importantly it allows testing of a new set of hypotheses and mechanisms of change that may not be feasible to examine in single studies. One possibility for new analyses is to detect moderated effects and explore subgroups, which can be explored in the pooled data of RCTs or non-RCTs.

Subgroups, Moderated Effects, and New Discoveries

IPD meta-analysis or IDA can be useful for understanding subgroups because individual-level, as well as study-level, covariates can be explicitly studied in connection with different intervention effects. Subgroups in this context extend beyond subgroups of individuals to include any meaningful differences that may affect intervention outcomes, such as any differences in intervention protocols and follow-up periods. These investigations are of critical interest in intervention and prevention research, as they suggest clues for mechanisms of behavior change at the individual level. Through the use of IDA, one can conduct new analyses well suited to reveal subgroups for a large pooled sample. IPD analysis can overcome issues of *low power, publication bias,* and *ecological fallacy*.

The power advantage of IPD analysis over single studies or AD analysis is generally well known. Investigations

aimed at revealing subgroups require a considerably large sample in original single studies. In comparison with AD analysis, the power to detect these effects by using the summary-level data will often be prohibitively low, and IPD analysis is necessary to detect effects of covariates at the individual level (Sutton & Higgins, 2008). However, with careful considerations, an AD meta-analysis may gain back the power even under heterogeneous designs (Liu et al., 2015).

As for publication bias, single studies lack sufficient power to detect moderated effects. Thus, statistically nonsignificant moderated effects may go unreported in many publications. Furthermore, the analytic approaches chosen for subgroup analysis in single studies vary widely, which makes it even more difficult to combine effect sizes across individual studies in AD meta-analysis. More important, there is an issue of interpretation. This applies to the relative advantage of IPD over AD. When study-level covariates are used to explain any differences in the effects of interest, this can lead to invalid statistical conclusions.

Meta-regression (via using AD; Figure 23.2) requires careful interpretation, as it is more prone to ecological bias than analysis using IPD. *Ecological fallacy* or *ecological bias* occurs when conclusions are made about the mechanism at the lower-level data (e.g., individual participants) based on the higher-level data (e.g., study-level information). For example, in the field of college alcohol intervention research, some of the studies comprise all men or all women. If gender is included as a study-level moderator of the treatment effect in a meta-regression analysis, which is then confounded with study membership, the resulting conclusion may be subject to ecological bias. Thus, IPD meta-analysis is better suited than single studies and AD meta-analysis when examining subgroups (Brown et al., 2013; Simmonds & Higgins, 2007).

Of course, IPD analysis has other advantages, such as the capabilities to check the quality of data and reanalyze data using more appropriate approaches (Curran & Hussong, 2009; Sutton, Kendrick, & Coupland, 2008). For these reasons, IPD meta-analysis is widely considered as the "gold standard" in the statistical literature (Lin & Zeng, 2010; Sutton & Higgins, 2008). At the same time, it has been echoed by many that such analyses require much more time and efforts to carry out (Cooper & Patall, 2009; Mun et al., 2015; Steinberg et al., 1997; Sutton et al., 2008).

The advantages of IDA need to be understood in the context of challenges. IDA requires the availability of data and subsequent contacts with original investigators who have conducted relevant studies. Other challenges include the need for harmonizing groups and measures across studies. These challenges go up when the number of studies increases because the number of variations to be harmonized across studies goes up along with the number of studies (see Mun et al., 2015, for some examples). In addition, as the number of studies goes up, overall percentage of missing data at the study level (i.e., did not assess or could not be harmonized) may become prohibitively huge when individual data sets are combined for a single-step integrated analysis. When one of the goals of IDA is to fully utilize available data in analysis, missing data can cause great difficulties for IDA studies throughout all stages of analysis.

Table 23.1 provides an overview of various methods discussed in this chapter. Figures 23.1, 23.2, and 23.4 graphically summarize the relationships among the source of data, type of data being pooled, analytic methods, and approaches to the univariate and multivariate methods. Figure 23.4 shows emerging directions in the field of research synthesis. Namely, future innovations may be obtained by prospectively coordinating and designing single studies as part of research networks, then channeling IPD from these sources for innovative multivariate analysis, and maximally utilizing all available data for more robust inference and new discoveries. Next, we illustrate two data examples from an IDA research project that pooled data from multiple alcohol intervention studies for college students.

DATA EXAMPLES

In the field of college alcohol intervention research, brief motivational interventions (BMIs) have received empirical support for reducing excessive alcohol use and related problems among college students at least on a short-term basis (Carey et al., 2007; Cronce & Larimer, 2011). However, there are several outstanding questions, including inconsistent intervention effects within and between studies, as well as the small intervention effect sizes reported in the literature. Furthermore, the mechanisms of behavior change remain sketchy. Project INTEGRATE (Mun et al., 2015) was a response to address these outstanding questions. More specific goals were to examine (1) whether BMIs are efficacious for bringing about changes in theory-based behavior targets, such as normative perceptions about peer alcohol use and the use of protective behavioral strategies while drinking; (2) whether positive changes in behavior targets predict greater reductions in alcohol use and negative consequences; (3) whether subsets of interventions are more promising; and (4) whether subgroups of individuals exist for whom different interventions are more efficacious.

Pooled Data

To compile data from BMIs, we contacted individual investigators in spring 2009 who had published data on BMIs. All but one agreed to share their data. In the case of the single exception, there was an ongoing project within the research team that had similar research goals. There was also a snowballing recruitment of other investigators. The unpublished data by these investigators were also compiled. Unlike a typical meta-analysis project, we did not systematically search databases or launch an exhaustive search of all eligible studies. Thus, the sample of studies we have is a convenience sample that is nonetheless broadly representative of the existing intervention studies conducted between 1990 and 2009 (published between 1998 and 2010). The combined data set was diverse in terms of original investigators, college campuses from which participants were recruited, demographic characteristics, and intervention study designs (Table 23.4; see also Mun et al., 2015).

Sample

Data were pooled from 24 independent studies (Studies 1–7, Studies 8a, 8b, and 8c, and Studies 9–22; see Table 23.4), resulting in a combined data set consisting of 12,630 participants (42% men; 58% first-year or incoming students) who were assessed two or more times from baseline up to 12 months. The majority of the sample is White (74%), with 12% Asian, 7% Hispanic, 2% Black, and 5% belonging to other or mixed ethnic groups. Approximately 15% are college students mandated to complete a university program (e.g., a BMI or educational program) as a result of alcohol-related infractions; 27% are members (or pledged to be a member) of fraternities and sororities; and 13% are varsity athletes or members of club sports. The majority of the individual studies included in Project INTEGRATE have been previously described in the published literature (see Mun et al., 2015, for more information).

Intervention Groups and Procedures

All studies included one or more BMI conditions, with the majority (21 studies) including either a control condition or other comparison condition (i.e., alcohol education). Across studies, some groups with the same intervention label were different; and several other groups with different intervention labels were actually similar when examined at the operational level. To determine which groups are equivalent to and different from others, we developed a quite detailed coding procedure to go over all published and unpublished intervention materials and quantitatively code their content and characteristics. Two content experts coded independently and any discrepancies were discussed in detail, at first between them and, if necessary, with the rest of the research team (see Ray et al., 2014 for details). We consequently relabeled intervention groups based on their intervention characteristics and content.

Newly labeled five intervention groups for Project INTEGRATE were Motivational Interview plus Personalized Feedback (MI + PF), stand-alone Personalized Feedback (PF), Group Motivational Interview (GMI), Alcohol Education (AE), and Control. There were also several unique conditions that did not fit these categories, including an MI + PF condition combined with an alcohol expectancy challenge, an MI without PF, and an MI + PF condition combined with a parent-based intervention. Participant recruitment and selection also varied across studies, ranging from volunteer students recruited with flyers to students who were required to complete an alcohol program because they violated university rules about alcohol.

In a preplanned, large multisite RCT, intervention conditions or groups can be perfectly balanced as in a factorial design, which is practically not feasible for IDA studies. For Project INTEGRATE, five intervention conditions sparsely existed across 24 studies. Table 23.5 shows the unbalanced nature of the intervention conditions in terms of their number and type for a subset of studies that were utilized in the current chapter. Studies 9 and 13/14 had all four groups (GMI excluded), but all other studies had two or three groups. Some of the studies did not have a control group (Studies 1 and 3), and only three studies had an Alcohol Education group. If these groups were to be analyzed simultaneously in one-step IDA using typical analytic approaches, it is likely that missing conditions would create estimation difficulties in analysis.

Measures

We now describe how measures were harmonized and how latent traits were estimated across studies for two of the measures used in the outcome analysis. See also Huo et al. (2014) for technical detail and Mun et al. (2015) for an accessible overview of the measurement approach taken for Project INTEGRATE.

Measures: Protective Behavioral Strategies

Protective behavioral strategies refer to specific cognitive behavioral strategies that can be employed to reduce risky drinking and limit harm from drinking (Martens et al., 2004). We identified five major questionnaires used to assess protective behavioral strategies: the 10-item Protective Behavioral Strategies (PBS) measure taken from the

TABLE 23.4 Project INTEGRATE: Study Designs (Adapted from Mun et al., 2015)*

Study	Representative reference	Intervention	First year (%)	Men (%)	White (%)	*N*	*N* at follow-up				
							1 mo.	2 mo.	3–4 mo.	6 mo.	9-12 mo.
Mandated college students											
1	White, Mun, Pugh, and Morgan (2007)	MI + PF, PF	62	60	73	348	--	--	319	--	219
2	White, Mun, and Morgan (2008)	PF, Control	63	71	69	230	--	199	--	106[1]	--
3	Barnett, Murphy, Colby, and Monti (2007)	MI + PF, AE	67	49	66	225	--	--	206	--	211
4	Cimini et al. (2009)	GMI, AE,[a] AE[a]	49	62	80	682	--	--	--	471	430
5	LaBrie, Lamb, Pedersen, and Quinlan (2006)	GMI	71	60	74	167	158	148	139	125	--
6	LaBrie, Thompson, Huchting, Lac, and Buckley (2007)	GMI	49	0	58	115	110	110	110	--	--
7.1	Fromme and Corbin (2004)	GMI,[b] Control	58	76	75	124	106	--	--	61[2]	--
Volunteer college students											
7.2	Fromme and Corbin (2004)	GMI[b], Control	38	59	59	452	332	--	--	221	--
8a	Larimer et al. (2007)	PF, Control	40	35	86	1,486	--	--	--	--	1,122
8b	Larimer et al. (2007)	PF, Control	37	41	64	2,155	--	--	--	--	1,618
8c	Larimer et al. (2007)	PF, Control	22	34	83	600	--	--	--	--	304
Volunteer first-year or incoming college students											
10.2	Baer, Kivlahan, Blume, McKnight, and Marlatt (2001)	Control	100	41	78	87	--	--	--	--	81
11	Walters, Vader, and Harris (2007)	PF, Control	100	59	64	383	--	272	288	--	--
15	LaBrie, Huchting, et al. (2008)	GMI, Control	100	0	56	263	261	260	258	--	--
16	LaBrie et al. (2009)	GMI, Control	100	0	57	287	282	277	268	250	--
17	LaBrie, Pedersen, Lamb, and Quinlan (2007)	GMI	100	100	65	120	110	105	90	56	--
22	Wood et al. (2010)	MI + PF, MI + PF + PBI,[c] Control	100	43	87	758	--	--	--	--	687
Volunteer heavy drinking college students											
9	Lee, Kaysen, Neighbor, Kilmer, and Larimer (2009)	MI + PF, PF, AE, GMI, GMI,[a] Control	100	38	71	604	--	--	504	485	--
10.1	Baer et al. (2001)	MI + PF, Control	100	46	84	348	--	--	--	--	322
12	Wood et al. (2007)	MI + PF, AE,[a,c] MI + PF + AE,[a,c] Control	4	47	91	335	276	--	257	258	--
13	Murphy, Benson, and Vuchinich (2004)	MI + PF, PF	13	32	94	54	--	--	--	51	--
14	Murphy et al. (2001)	MI + PF, AE, Control	41	46	94	84	--	--	79	--	79
21	Walters, Vader, Harris, Field, and Jouriles (2009); Walters, Vader, Harris, and Jouriles (2009)	MI + PF, PF, MI without PF[c], Control	41	35	84	288	--	--	261	252	251
Intercollegiate student athletes or fraternity, sorority, and service organization members											
18	Martens, Kilmer, Beck, and Zamboanga (2010)	PF, PF,[a] Control	32	26	85	329	289	--	--	259	--
19	LaBrie, Hummer, Neighbors, and Pedersen (2008)	GMI,[a] Control[a]	19	31	67	1,178	966	922	--	--	--
20	Larimer et al. (2001)	MI + PF, Control	78	52	84	928	--	--	--	--	631

*N = 12,630. Studies are numbered arbitrarily. MI + PF = Motivational Interview plus Personalized Feedback; PF = Stand-alone Personalized Feedback; GMI = Group Motivational Interview; AE = Alcohol Education; PBI = Parent-based Intervention.

[a] = These groups are quite unique despite having the same label as others without the superscript;

[b] = GMI in Study 7 provided personalized feedback, whereas other GMIs did not;

[c] = These groups represent unique intervention conditions not found in any other studies included in Project INTEGRATE.

[1]The control group (n = 119) for Study 2 received feedback at 2 months post baseline and thus their follow-up data at 6 months post baseline were excluded;

[2]Mandated students who were in the control group (n = 24) in Study 7.1 received GMI at 1 month post baseline and their follow-up data at 6 month post baseline were excluded. "--" indicates that follow-up was not administered. Follow-up sample sizes were based on selective alcohol use measures and could differ from those reported in published articles.

TABLE 23.5 Project INTEGRATE: Intervention Groups Across Studies After Removing Ineligible Studies for the Multivariate Meta-analysis Data Example

	1	2	3	8a	8b	8c	9	10	11	12	13/14	18	20	21	22
MI + PF*	X	--	X	--	--	--	X	X	--	X	X	--	X	X	X
PF	X	X	--	X	X	X	X	--	X	--	X	X	--	X	--
Alcohol Education	--	--	X	--	--	--	X	--	--	--	X	--	--	--	--
Control	--	X	--	X	X	X	X	X	X	X	X	X	X	X	X

*MI + PF = Motivational Interview plus Personalized Feedback; PF = Stand-alone Personalized Feedback. "--" indicates the intervention groups are missing by design.

National College Health Assessment survey (American College Health Association, 2001); the 15-item Protective Behavioral Strategies Scale (PBSS; Martens et al., 2005); the 37-item Self Control Strategies Questionnaire (SCSQ; Werch & Gorman, 1988); a seven-item Drinking Restraint Strategies (DRS) scale used in Wood, Capone, Laforge, Erickson, and Brand (2007); and a nine-item Drinking Strategies (DS) scale reported in Wood et al. (2010). The PBS was assessed by Studies 1, 2, 8a, 8b, 8c, and 9; the PBSS was assessed in Studies 4, 16, 18, and 21; the SCSQ by Study 3; the DRS by Studies 12 and 22; and the DS by Study 22. In these studies, items were assessed in Likert-type scales. Note that many items were similar in wording. Given that slightly differently worded items can be responded differently by responders, we initially treated them as different items. In addition, we limited item-level data only for those who reported recent drinking (i.e., past 1 to 3 months) due to the nature of the construct.

Of the compiled pool of items, items indicating abstinence from drinking (two items; *cut back on drinking* and *choose not to drink alcohol*) or manner of drinking (one item; *drink shots of liquor*) were dropped, as they do not conceptually fit the definition of protective behavioral strategies. Study 3 included an additional 16 items related to protective behavioral strategies, which were dropped from the pool because they were assessed only in Study 3 and could not be linked to any other items across studies. Study 7 assessed protective, as well as risky, behaviors (a total of 17 items) as a set of behaviors that one can engage during drinking, such as "leaving drinks unattended" or "drank lower alcohol content beverage." Participants were asked to provide the total number of times that they participated in such activities in the past month (possible values ranging from 0 to 99). Their response patterns were very different from those of other studies measured on a Likert-type scale and difficult to harmonize reasonably. We thus decided not to include these items in the pool.

A total of 58 protective behavioral strategy items assessed by 13 studies (Studies 1–4, 8a–8c, 9, 12, 16, 18, 21, and 22) were then analyzed using item response theory (IRT) analysis. Different questionnaires had different stem questions leading to specific items. Some of the major variations were as follows:

- The following set of questions asks about your drinking behavior. Please indicate how often you did the following . . . " (PBS)
- Please indicate the degree to which you engage in the following behaviors when using alcohol or "partying" . . . (PBSS)
- How often did you use each of these strategies to deliberately limit your drinking? (SCSQ)
- Do you deliberately try to . . . (DRS)
- How often do you . . . (DS)

We assumed that these stem questions did not make a difference. Of the remaining 58 items, 20 were combined into five individual items because they were very similarly worded. Examples of similarly worded questions were "avoid drinking games," "avoid or limit your participation in drinking games to drink less," and "avoid playing 'drinking games.'" Collapsing such similarly worded items may not be ideal, as participants can respond differently to slightly differently worded items or different leading (stem) questions. However, to be able to link items across studies, we made an assumption that these similarly worded items were essentially the same in eliciting responses. Many pooled items were similar, as they had been adopted or modified from other existing questionnaires included in the pool of items. If we were to take the strictest route and treat all items as different, one consequence would be to analyze only those few studies that administered exactly the same questionnaire for exactly the same time frame using exactly the same response options. For example, the PBS was administered in six studies and the PBSS was administered in four studies. If these data sets were analyzed separately, it would be difficult to draw any direct comparisons between the two sets of analyses.

In general, there is a trade-off between item coverage across studies and quality of information when

harmonizing measures. The coverage of items will be greater when we accept slightly imperfect items as commensurate across studies. However, the coverage will suffer if slight variations are not tolerated across studies. This decisional balance may have to be determined individually depending on the research questions, available data, and subsequent analytic methods (to see examples in which harmonization may not be reasonable, see Mun et al., 2015).

Our subsequent analysis did not involve comparing intervention cases and control cases across studies (called *breaking randomization* or *naïve comparison*). We compared intervention cases against their own controls within studies, before their relative differences were pooled across studies. Thus, we made an assumption that this item linkage we established would not yield biased results. In situations where naïve comparisons are made, however, one cannot assume that even the same item has the same item function across studies. In other words, when participants in one study are combined with participants in another study without the nested data structure taken into consideration, item functions may differ across studies, which may result in biased inference. In such situations, DIF tests across studies and appropriate follow-up actions may be needed to demonstrate measurement equivalence across studies.

Response options were harmonized and recoded to indicate 0 = never; 1 = rarely, seldom, occasionally, or sometimes; 2 = often or usually; and 3 = always. Response options rarely, seldom, occasionally, and sometimes were originally assessed as different categories but were combined into one because endorsement rates were relatively low for these categories. Also, for protective behavioral strategies, answers in either extreme (i.e., never or always) and adjacent steps from the extremes are more important than any difference that exists in the middle of the scale (e.g., rarely vs. seldom or occasionally vs. sometimes).

A total of 43 items (38 unique and five collapsed items) were then analyzed, specifying a single, underlying dimension of protective behavioral strategies. Because we deemed it important to distinguish *often* from *always*, we used a generalized partial credit model (GPCM; Muraki, 1992) to assign partial credit for polytomous items (for more detailed explanations of the GPCM parameters, see Mun et al., 2015). We developed Markov chain Monte Carlo (MCMC) algorithms to fit several IRT models, including the GPCM model, within a hierarchical Bayesian framework. Huo et al. (2014) provided the theoretical and technical details of the IRT models and MCMC algorithms, which were written in Ox (Doornik, 2009).

Measures: Alcohol-Related Problems–Neglecting Responsibilities

Alcohol-related problems is one of the critical outcome measures and our measurement approach for this construct is described in greater detail elsewhere (see Huo et al., 2014; Mun et al., 2015). Briefly, individual items and items from well-known questionnaires for alcohol-related problems were identified from each study and compiled in a separate data set. These items mostly came from the Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989), the Young Adult Alcohol Problems Screening Test (YAAPST; Hurlbut & Sher, 1992), the Brief Young Adult Alcohol Consequences Questionnaire (BYAACQ; Kahler, Strong, & Read, 2005), the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuente, & Grant, 1993), the Positive and Negative Consequences Experienced questionnaire (PNCE; D'Amico & Fromme, 1997), and the Alcohol Dependence Scale (ADS; Skinner & Allen, 1982; Skinner & Horn, 1984). For each item, responses were dichotomized to indicate 1 = Yes; 0 = No, because this response format was the common denominator across studies. When someone did not drink during the time frame referenced, the score was recoded as zero.

We utilized both a unidimensional IRT model and a four-dimension IRT model for alcohol-related problems for Project INTEGRATE. In the former, a single overall, severity latent trait is assumed to give rise to item responses. In the latter, four distinct but related latent trait dimensions give rise to item responses within their dimensions, which were Neglecting responsibilities, Interpersonal difficulties, Dependence-like symptoms, and Acute heavy-drinking, respectively. Estimated correlations among the four dimensions exceeded 0.8, which indicates that these dimensions are highly correlated but still distinct. Both IRT models showed acceptable fit and parameter estimates were similar between the two models. In the current chapter, we focus on a single dimension of alcohol-related problems obtained from the four-dimensional IRT model–Neglecting responsibilities (NR) as an example in the Outcome Analysis section. Some of the example items for this dimension were "Got into trouble at work or school because of drinking" and "Missed a day (or part of a day) of school or work."

Outcome Analysis

For outcome analysis, we provide data examples for model-based meta-analysis and multivariate meta-analysis (network meta-analysis) using data for latent trait scores for protective behavioral strategies (henceforth referred

to as PBS) and neglecting responsibilities (henceforth referred to as NR), respectively. For the first data example, we utilized both fixed-effects and random-effects models to examine whether college students who were assigned to a brief alcohol intervention utilized protective behavioral strategies more often their counterpart controls at the first, immediate follow-up. Protective behavioral strategies are increasingly seen as an important behavioral target of alcohol intervention efforts for college students. For the second example, we demonstrated how multiple intervention arms could be combined across studies and analyzed in a single combined, multivariate analysis using the CD approach.

Model-Based Meta-analysis

For model-based meta-analysis, we used PBS data. Of the 13 studies (Studies 1–4, 8a–8c, 9, 12, 16, 18, 21, and 22) that assessed PBS, we excluded three studies due to the lack of an equivalent control group (Studies 1, 3, and 4). Studies 9 and 21 had multiple intervention arms and we combined them into one intervention group (i.e., MI + PF, PF, and Alcohol Education were combined for Study 9; MI + PF and PF were combined for Study 21). Thus, we had a two-arm design (one intervention and one control) for a total of 10 studies, and examined their PBS data at their first follow-up. The first follow-up assessment occurred within 1–3 months for Studies 2, 9, 12, 16, 18, and 21; 9 months for Study 22; and 12 months for Studies 8a, 8b, and 8c.

Results indicated that there was no significant between-study heterogeneity, Q (df = 9) = 12.03, p = 0.21. The statistical conclusion from either a fixed-effects or a random-effects model was the same. That is, there were no significant differences between the intervention and control groups in terms of their tendency to utilize PBS at the first follow-up. The overall mean difference in PBS latent traits between the two groups was 0.05 under the fixed-effect model. The 95% confidence interval [–0.002 to 0.102] included 0. The random-effects model estimated the overall mean difference to be 0.05 and had the slightly larger 95% confidence interval [–0.008 to 0.104] to account for between-study variability, although in the present case there was no statistically significant, between-study variability in the mean differences in PBS scores. Figure 23.9 shows the results from the fixed-effects model in a forest plot. At the individual study level, only Study 8a showed a statistically significant, favorable outcome for the intervention group, compared to control.

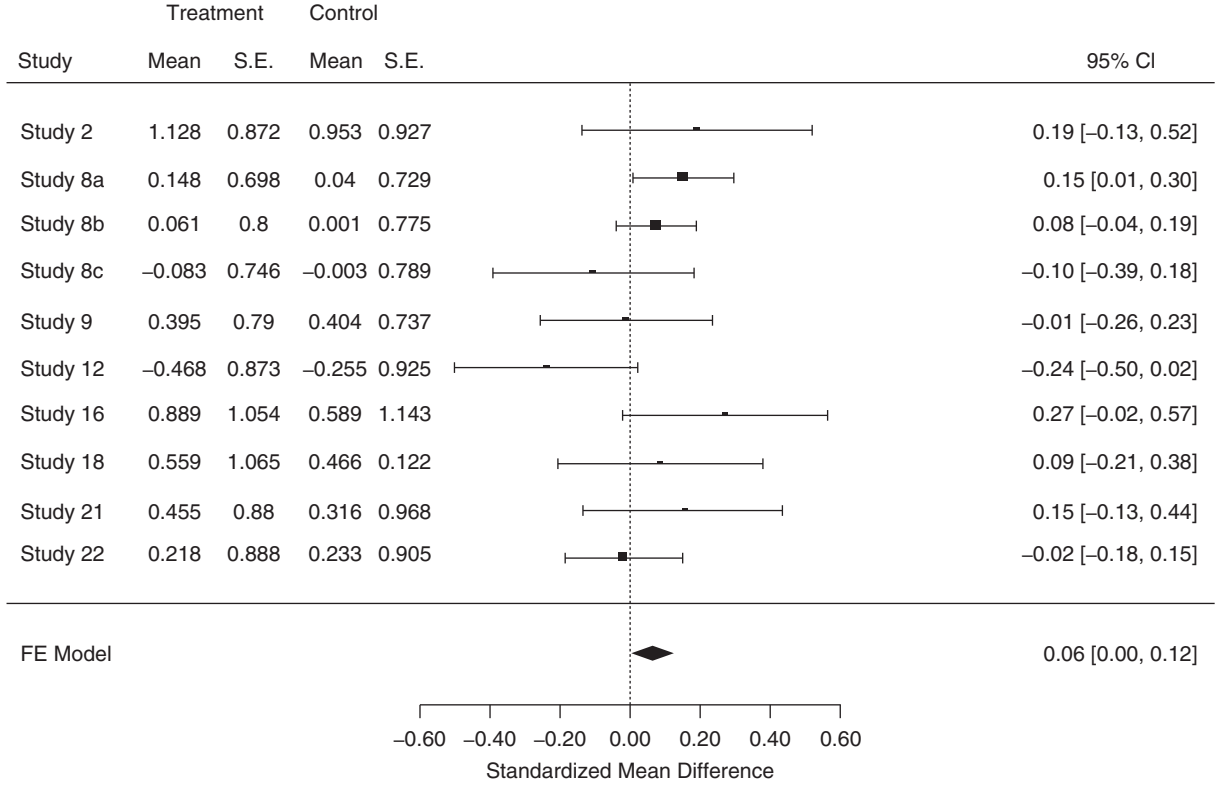


Figure 23.9 Protective behavioral strategies at the first follow-up under the fixed-effects model for 10 studies.

Although there was no statistically significant, between-study variability in estimates, the nonsignificant Q statistic should not literally be interpreted as evidence to indicate that the effect sizes are consistent across studies, since such nonsignificance can be due to lack of power. With a small number of studies or large within-study variances in individual studies, even substantial between-study dispersion might yield a nonsignificant Q statistic (Borenstein et al., 2009). In the forest plot shown in Figure 23.9, we had narrow confidence intervals (small within-study variances) in large studies, such as Studies 8a and 8b, but in many other studies with small samples (e.g., Studies 16 and 18), the within-study variances and associated confidence intervals were large. In addition to the highly discrepant sample sizes (Table 23.4), studies varied in terms of their follow-up periods and sample characteristics, and also the interventions implemented. For example, the eligibility criteria for participation in Study 12 (Wood et al., 2007) were geared toward enrolling high-risk heavy drinkers: (1) 14 or more drinks per week for men and 10 per week for women; (2) at least one episode of heavy drinking in the past 30 days; and (3) endorsement of at least two alcohol-related consequences in the past year. Although Wood et al. excluded and referred the students who reported more than 40 drinks per week or exhibited moderate to severe dependence to the university counseling center, this sample placed on the extreme end of risk for excessive and problematic drinking, relative to other study samples (see Huh et al., 2015, for alcohol use data across studies). Furthermore, this sample was estimated to have the lowest levels of PBS at baseline across all 10 studies (Figure 23.10). For these reasons, when we excluded data from Study 12, the results from the remaining nine studies indicated a favorable outcome for the intervention group at follow-up; overall mean difference = 0.08, with 95% confidence interval ranging from 0.01 to 0.15 (Figure 23.11).

The point about this subsequent analysis without Study 12 is not to draw a conclusion that the intervention was efficacious for increasing the utilization of PBS, which was not the case for the pooled sample of 10 studies, but to suggest that the intervention's role in the utilization of PBS may differ depending on the levels of alcohol use and their PBS utilization at baseline. To test this hypothesis, however, a further investigation is required that incorporates individual-level covariates in the model. Without examining the individual level data, drawing any conclusion based on study-level data (like the one that we tentatively discussed here) may be subject to ecological fallacy, and lead to inaccurate inference about how changes occur following an intervention.

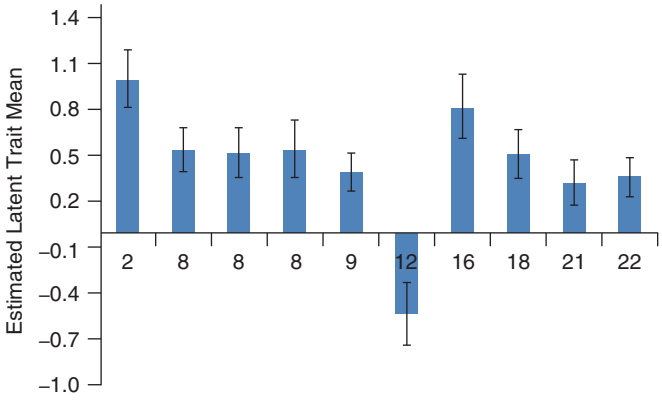


Figure 23.10 PBS latent trait means across studies estimated from the GPCM analysis. The second, third, and fourth bars indicate data from Studies 8a, 8b, and 8c, respectively. The error bars indicate two times standard errors in each direction. Participants in Studies 2 and 16 were estimated to utilize PBS more often than students in other studies. Study 12 was an outlying study—Participants in Study 12 were least likely to utilize PBS. See footnote 1.

Multivariate Meta-analysis

For multivariate meta-analysis, we used the new two-stage approach for IPD described previously. We had the following inclusion criteria for studies: at least two intervention arms existed and participants were randomly assigned to these groups (Studies 5, 6, and 17 excluded); and the intervention utilized should not be unique (Studies 4, 7, and 19 excluded). Studies 15 and 16 were further excluded because they were exclusively for first-year female students, which represented a missing data problem (due to no variability) for two covariates (first-year student and gender) and posed a difficulty at the second stage of combining estimates. In addition, the latter two studies were small in sample size, were conducted on the same college campus by the same investigators, and utilized the same intervention (i.e., GMI). Due to the elimination of other GMI studies, these two studies became unique in the pooled data set. Of the studies that met the inclusion criteria, we further removed some conditions (e.g., alcohol expectancy challenge, parent-based intervention, or MI without PF) because they were deemed sufficiently different from other intervention conditions and/or were unique to specific studies and could not be linked to other studies. Studies 13 and 14 were combined into one study for the purpose of analysis. Study 13 included an MI + PF intervention and a stand-alone PF intervention, and Study 14 included an MI + PF intervention and an assessment-only control group. In-person MIs combined with PF in both studies were, by and large, identical (i.e., high risk samples, same PF design,

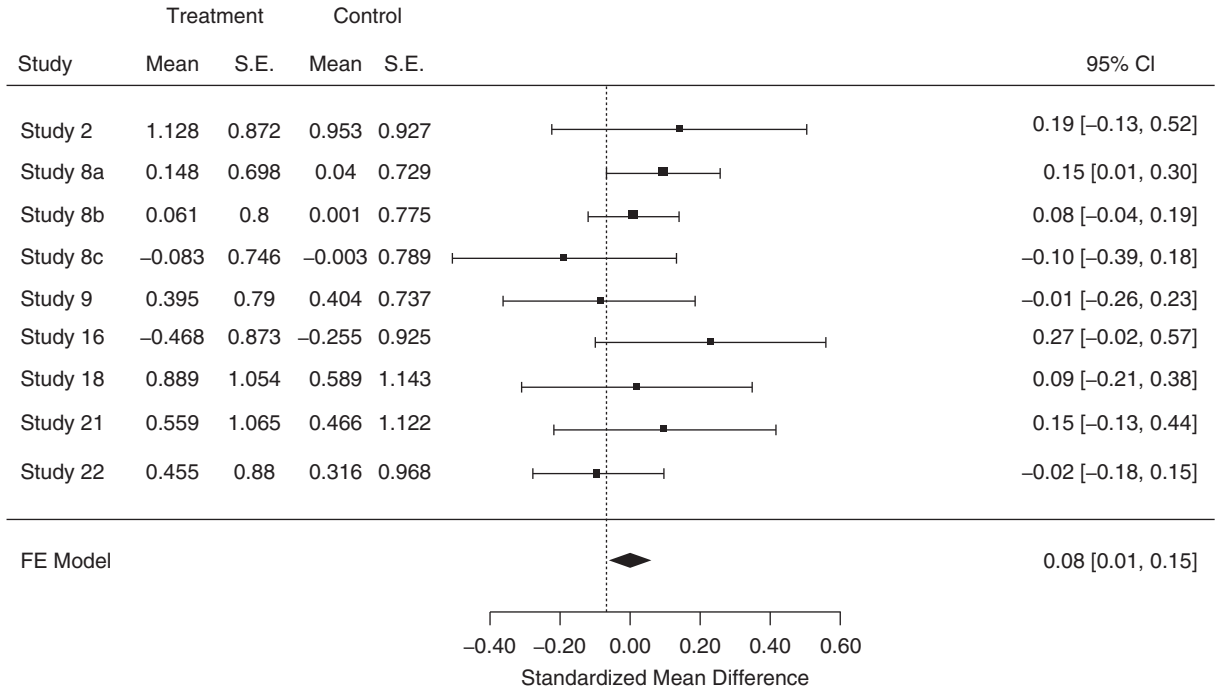


Figure 23.11 PBS at the first follow-up under the fixed-effects model for nine studies (Study 12 removed).

led by the same investigators, and on the same campus), and there were no baseline differences across these two groups. In addition, these two studies had very small samples. Thus, we collapsed these two studies into one combined study (i.e., Study 13/14), allowing an MI + PF group and a PF to be contrasted with a control condition.

Table 23.5 shows that only a subset of studies provides information for direct, head-to-head comparisons of intervention groups. For example, for a head-to-head, direct comparison between MI + PF and PF, data from Studies 1, 9, 13/14, and 21 can be analyzed. For a comparison between MI + PF and Alcohol Education, data from Studies 3, 9, and 13/14 can be utilized. For a comparison between MI + PF and control, Studies 9, 10, 12, 13/14, 20, 21, and 22 can be analyzed. However, for these potential subset analyses, eligible studies would be different, and the number of studies to be analyzed would be much smaller than the entire sample of eligible studies. This would be an inefficient use of available data and could also create inconsistent findings due to different data sets being analyzed for different comparisons. Furthermore, this subsetting strategy does not accommodate the fact that some of the intervention conditions are from the same studies and are hence related. Multivariate meta-analysis overcomes these data characteristics by taking advantage of all available information from all studies.

We first fit a random intercept growth model using NR latent trait scores as an outcome variable separately for each study. The regression model included a total of 15 covariate terms (Table 23.6). Alcohol outcomes following an intervention among college students typically show a pattern of an immediate decline followed by a rebound (e.g., Mun, White, & Morgan, 2009). Thus, we modeled the outcome over time by using a quadratic growth model (month post intervention as a time variable). We included three covariates to control for their differences in alcohol-related problems—being male, White, and first-year student. In addition, we included PBS scores at baseline as a covariate because it was related to dropout at follow-ups in some of the studies. To test intervention effects, we added interaction terms between each of the growth parameters (i.e., linear and quadratic growth slopes) and each of the three intervention groups (i.e., MI + PF, PF, and Alcohol Education). Naturally, three variables to contrast the three intervention groups against control were also included in the model. Note that the actual model for each study was different due to missing covariates at the study level. For example, if a study did not assess PBS, the model had a total of 14 covariates for that study (e.g., Study 13/14 in Table 23.6). Similarly, for studies that had just one follow-up, any coefficients involving a quadratic growth parameter could not be estimated. The

TABLE 23.6 Patterns of Covariates by Study for IPD Multivariate Meta-analysis*

Study	1	2	3	8a	8b	8c	9	10	11	12	13/14	18	20	21	22
Man vs. woman	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
White vs. non-White	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
First-year vs. other	X	X	X	X	X	X	--	--	--	X	X	X	X	X	--
PBS at baseline	X	X	X	X	X	X	X	--	--	X	--	X	--	X	X
Control**	--	X	--	X	X	X	X	X	X	X	X	X	X	X	X
Alcohol Education	--	--	X	--	--	--	X	--	--	--	X	--	--	--	--
PF	X	X	--	X	X	X	X	--	X	--	X	X	--	X	--
MI + PF	X	--	X	--	--	--	X	X	--	X	X	--	X	X	X
Linear slope (LS)	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
Quadratic slope (LS)	X	--	X	--	--	--	X	--	X	X	X	X	--	X	--
LS * Control**	--	X	--	X	X	X	X	X	X	X	X	X	X	X	X
LS * Alcohol Education	--	--	X	--	--	--	X	--	--	--	X	--	--	--	--
LS * PF	X	X	--	X	X	X	X	--	X	--	X	X	--	X	--
LS * MI + PF	X	--	X	--	--	--	X	X	--	X	X	--	X	X	X
QS * Control**	--	--	--	--	--	--	X	--	X	X	X	X	--	X	--
QS * Alcohol Education	--	--	X	--	--	--	X	--	--	--	X	--	--	--	--
QS * PF	X	--	--	--	--	--	X	--	X	--	X	X	--	X	--
QS * MI + PF	X	--	X	--	--	--	X	--	--	X	X	--	--	X	--

*"--" indicates missing by design. These include variables not assessed by study (PBS); the entire sample consisted of first-year students only (Studies 9, 10, and 11); particular intervention groups were not employed; and only one follow-up assessment available during the 12-month period, and thus their quadratic slope parameter in change could not be estimated.

**= in actual models, this group becomes a referent group for the three other intervention groups, and is not counted in the number of covariates included in the model.

overall pattern of covariates analyzed for each study is shown in Table 23.6.

The growth model was analyzed separately for each of the 15 studies (13/14 was analyzed as one study) in the first stage of the analysis using IPD. In the second stage, a vector of the estimates of regression coefficients and its covariance matrix were retrieved for each study and subsequently combined with the estimates from other studies using the formulas and the REML estimation approach shown in the IPD Meta-analysis and Integrative Data Analysis section.

The multivariate meta-analysis method utilized for this data example combines multiple estimates from multiple studies simultaneously and borrows information, when information is missing, from other studies by incorporating their correlations. This approach differs from existing multivariate meta-analysis approaches utilizing AD. This approach also differs from those combining single or bivariate estimates from multiple studies in the sense that what is integrated across studies is the entire model specified at the first stage of the multivariate meta-analysis. This IPD multivariate meta-analysis approach utilizes more data than any other existing approaches to meta-analysis. The combined model from the second stage can be interpreted and utilized for subsequent investigations.

Table 23.7 shows the IPD multivariate meta-analysis results from the combined model. With the effects of other covariates adjusted, Alcohol Education had higher levels of NR problems compared to control, reflecting their baseline difference (Figures 23.12–23.14) that was maintained throughout the follow-up period. In-person MI combined with PF (MI + PF) or PF did not differ from control. For the efficacy of these interventions, we focused on the interaction terms between growth parameters and intervention conditions. There was a statistically significant interaction effect on NR problems between the linear slope

TABLE 23.7 Parameter Estimates from IPD Multivariate Meta-analysis*

	Estimate	*SE*	*p*
Intercept	–0.907	0.150	0.000
Man (1; 0 = Woman)	0.008	0.047	0.865
White (1; 0 = Non-White)	0.045	0.034	0.182
First-year (1; 0 = Other)	0.055	0.030	0.070
PBS at baseline	–0.240	0.032	0.000
Linear slope (LS)	–0.034	0.012	0.007
Quadratic slope (QS)	0.002	0.002	0.185
Alcohol Education (vs. Control)	0.216	0.081	0.007
PF (vs. Control)	–0.011	0.031	0.733
MI + PF (vs. Control)	0.035	0.044	0.427
LS × Alcohol Education	0.010	0.026	0.684
LS × PF	0.007	0.004	0.088
LS × MI + PF	**–0.030**	**0.010**	**0.002**
QS × Alcohol Education	0.000	0.002	0.905
QS × PF	–0.001	0.001	0.552
QS × MI + PF	**0.001**	**0.001**	**0.349**

**SE* = Standard Error. PBS = Protective Behavioral Strategies. MI + PF = Motivational Interview plus Personalized Feedback. PF = Stand-alone Personalized Feedback. The efficacy of the MI + PF can be seen in Figure 23.14.

of growth and MI + PF. The interaction term between the quadratic growth parameter and MI + PF was not statistically significant. These two terms are highlighted in bold in Table 23.7 because it is impossible to evaluate any growth trends or growth-related effects in a compartmentalized, piecewise manner. In a typical growth curve model, for those whose initial levels are high, their subsequent growth trends tend to be subdued. It would be misleading to focus on one growth parameter without considering the other as the first-order polynomial term (linear) is dominated by the second-order term (quadratic) in growth models.

Thus, to demonstrate this intervention effect as a whole, we estimated the NR trait levels at two important follow-up periods—6 months and 12 months post intervention—by plugging the meta-analyzed regression coefficients into the model and calculating the estimated scores for the intervention groups. For some studies, this resulted in extrapolated estimates and for others, these were actual estimates based on observed follow-up data (Figures 23.12–23.14). Note that confidence intervals (and error bars) in Figures 23.12–23.14 were calculated by using different formulas depending on the focus of the comparisons (within-group vs. between-group). In estimation, we used the following values for covariates: male = 1, first-year = 1, White = 1, and PBS = average value. Negative NR scores were due to anchoring (of a group in IRT analysis) and shorter bars indicate more severe problems for the present data in Figure 23.12.

Since first-year students tended to have more severe problems and those with greater PBS had lower levels of problems, depending on the covariates of interest, these bar graphs could be adjusted a little lower (or longer; i.e., less serious problems) for non-first-year students and

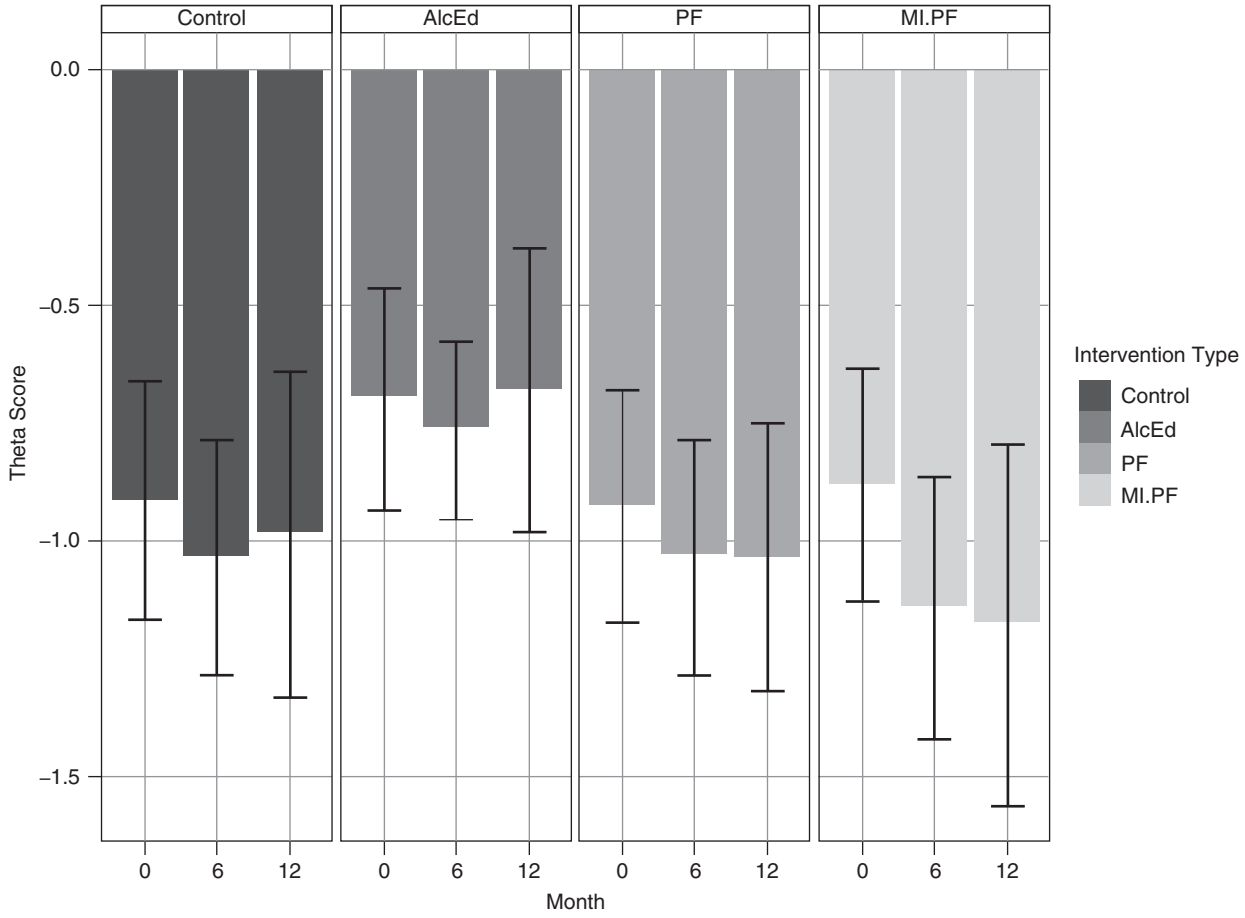


Figure 23.12 Model-based estimates of NR latent trait scores at baseline, 6 months and 12 months post intervention by group. NR = Neglecting responsibilities due to drinking; AlcEd = Alcohol Education; PF = Stand-alone Personalized Feedback; MI.PF = Motivational Interview plus Personalized Feedback (MI + PF); y-axis indicates NR scores. Higher scores (i.e., shorter bars in this figure) indicate greater severity. Error bars indicate 95% confidence intervals. Relatively speaking, there was a trend toward improving for those in the PF and MI + PF conditions.

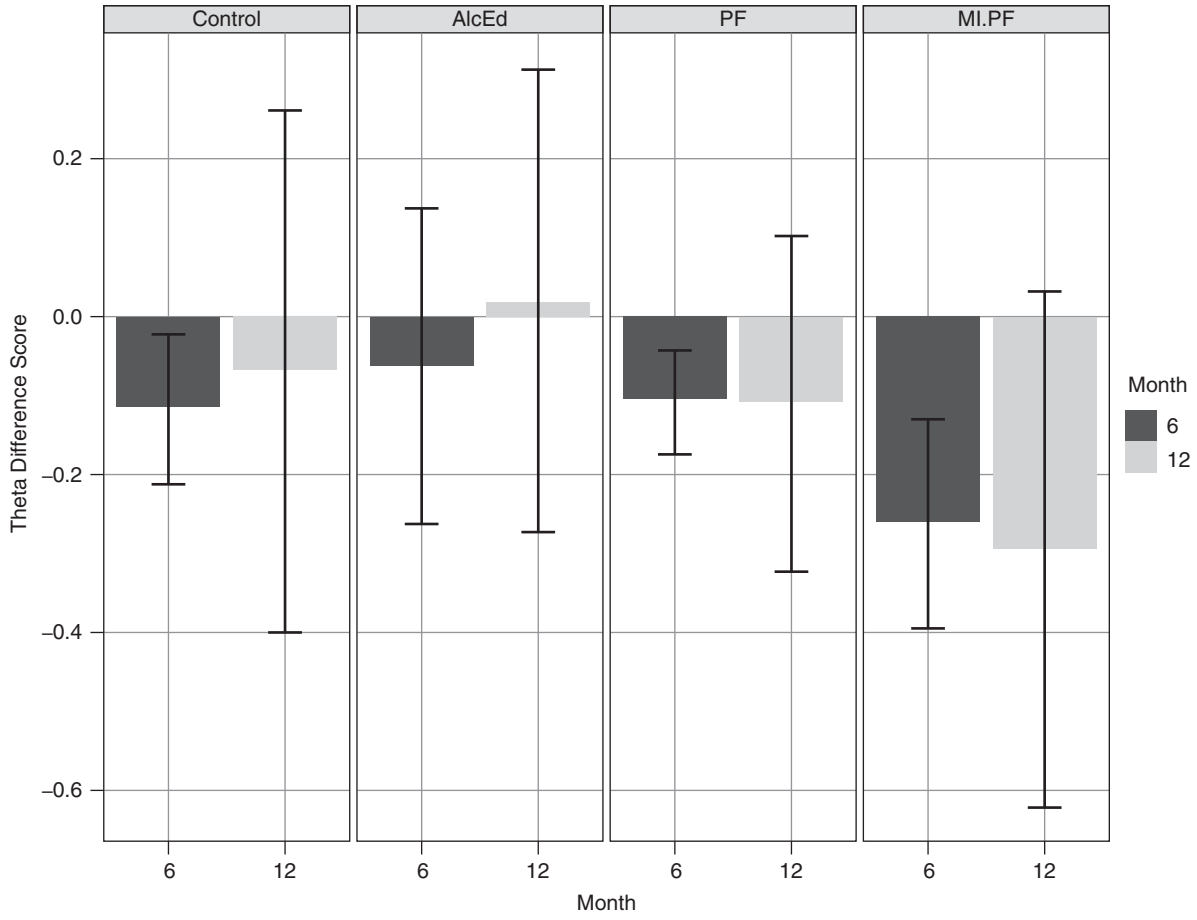


Figure 23.13 Model-based estimates of change at 6 months and 12 months post intervention by group. NR = Neglecting responsibilities due to drinking; AlcEd = Alcohol Education; PF = Stand-alone Personalized Feedback; MI.PF = Motivational Interview plus Personalized Feedback (MI + PF); y-axis indicates NR change scores. Error bars indicate 95% confidence intervals for these change scores. Confidence intervals that do not include zero indicate a statistically significant reduction at a given time point within groups. All groups with the exception of Alcohol Education showed a statistically significant reduction at 6 months. The significant reduction disappeared at 12 months for all three groups.

for those with higher levels of PBS at baseline. Relatively speaking, there was a trend toward improvement for PF and MI + PF. Figure 23.13 shows the statistically significant reductions from baseline at 6-month follow-up but not at 12-month follow-up for control, PF, and MI + PF. No evidence of change was found for Alcohol Education. Higher difference scores (positive scores on y axis) indicate higher levels of NR trait scores at follow-ups relative to their baseline levels (i.e., NR score went up; in other words, greater problems), whereas lower difference scores (negative scores on y axis) indicate reductions in NR scores at 6-month and 12-month follow-ups, compared with baseline. Given that control also significantly reduced NR at 6-month follow-up (zero not included in 95% confidence interval), the significant reductions shown by the PF and MI + PF groups could not be attributed to the interventions received. Thus, we then specifically compared the changes shown by three intervention groups against the change by control. Figure 23.14 shows the results. Compared to control, MI + PF showed significantly lower levels of NR at both 6-month and 12-month follow-ups. Those in the MI + PF condition were significantly better than those who received no intervention at both 6-month and 12-month follow-ups (zero not included in the 95% confidence intervals; Figure 23.14).

A few other things may be worth mentioning. Confidence intervals for 12-month outcome data were greater than those for 6-month data, which reflects greater uncertainty at 12-month follow-up, especially for PF, due to relative lack of data. Second, Alcohol Education exhibited greater problems at all three time points, reflecting their

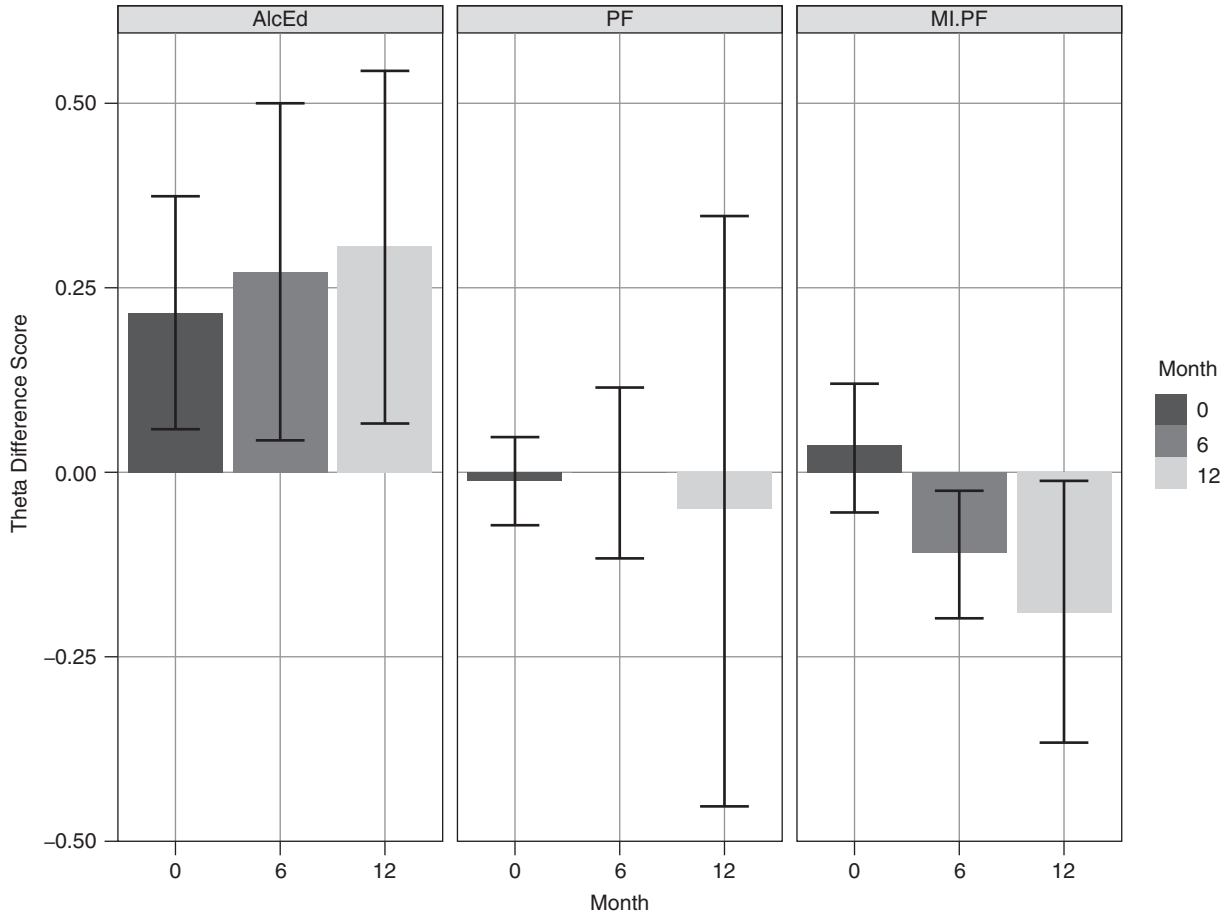


Figure 23.14 Multivariate meta-analysis of multiple intervention comparisons, showing a graphic demonstration of the significant MI + PF intervention effect compared with control. NR = Neglecting responsibilities due to drinking; AlcEd = Alcohol Education; PF = Stand-alone Personalized Feedback; MI.PF = Motivational Interview plus Personalized Feedback (MI + PF). Error bars indicate 95% confidence intervals. Confidence intervals that do not include zero indicate that the intervention condition at a given time point differed significantly in the estimated NR trait scores, compared with control. MI + PF, compared with control, showed significantly lower levels of NR trait scores at both 6 months and 12 months post intervention.

baseline differences. Combined with the data shown in Figure 23.13, we can say that Alcohol Education neither made it worse nor better for participants. The participants assigned to Alcohol Education more or less maintained their NR levels. This analysis was repeated for another dimension of alcohol-related problems and the results were largely similar (not reported). Thus, we can conclude that first, an in-person MI intervention with PF may make a difference in ameliorating alcohol-related problems among college students, at least up to 12 months; second, a stand-alone PF intervention may not be helpful for reducing alcohol-related problems, compared to control.

This finding is similar to the one reported in Huh et al. (2015) that utilized different analytic methodologies for a slightly different set of studies from Project INTEGRATE. In Huh et al., we used Bayesian multilevel overdispersed Poisson hurdle models in one-step IDA analysis to estimate intervention effects on drinks per week and peak drinking, and Gaussian models for alcohol problems in one-step IDA analysis. To overcome the unbalanced design, we utilized a three-level multilevel model, in which study-by-intervention condition was used as the highest, third level (repeated measures as the first level and individuals as the second level). To derive the overall intervention efficacy estimate (overall efficacy), as well as estimates for each of the intervention groups, we utilized posterior distributions from Bayesian analysis. The results indicated that there was no evidence of the overall intervention (BMI vs. control) effect on any of the outcomes examined but there was a small intervention effect by MI + PF on alcohol-related problems (Huh et al., 2015).

The substantive conclusion made in the current chapter is significant because the field of college alcohol interventions has embraced web-based normative feedback interventions, based on the previous findings from single studies that suggested that stand-alone PF interventions are just as efficacious as in-person MIs with PF at least for short term (e.g., see Walters & Neighbors, 2005 for a review). The current finding suggests that to reduce alcohol-related harm, an in-person MI may be needed. Although web-based interventions are easier to reach those who are in need and are attractive for their scalability, the findings from the IDA investigations reported in this chapter and in Huh et al. (2015) cautiously suggest a need to delve further into the mechanisms behind the in-person MI interventions with PF and to develop new interventions that may be more efficacious.

Note that the analytical examples shown in this chapter, as well as the one in Huh et al. (2015), are computationally demanding although the nature of the challenges is different. For the current IPD multivariate meta-analysis, the dimension of covariates was an important factor to consider. We added just four demographic covariates to the model, but the analysis required 15 × 15 covariance matrices (for 15 covariates) to be pooled across 15 separate analyses (for 15 studies with Studies 13 and 14 combined). The estimation of the between-study covariance matrix was quite challenging, especially with the embedded missing data in these matrices. Despite these challenges noted, the data examples shown in this section demonstrate new possibilities for the field of meta-analysis and IDA. Instead of focusing on qualitative or subjective interpretations of the relevant literature or counting the number of studies with a statistically significant intervention effect, IDA can be utilized using more advanced and flexible methodologies developed in recent years. It can overcome inherent limitations of single studies and AD meta-analysis. The value of fully utilizing existing data has been important and will most likely remain important in the coming years.

Software Programs and Packages

Univariate meta-analysis can be performed using commercially available software packages like Comprehensive Meta-analysis (CMA; Borenstein, Hedges, Higgins, & Rothstein, 2005) and MetAnalysis (Leandro, 2005) and other free DOS-based programs. Bax, Yu, Ikeda, and Moons (2007) reviewed their various features and different levels of usability in great detail. Table 23.8 lists available meta-analysis packages developed for R (R Core Team, 2014) and user-defined commands developed for Stata (StataCorp, 2013). For multivariate methods, *metafor* (Viechtbauer, 2010) can perform multivariate meta-analysis under fixed- and random-effects models, and the latter can be fitted through MLE or REML. In contrast, *mvtmeta* package (Chen, 2012) performs multivariate meta-analysis using the method of moments estimator for between-study covariance matrix when fitting random-effects model; *mvmeta* package (Gasparrini, Armstrong, & Kenward, 2012) uses the same method of moments estimation but allows missing data and handles meta-regression; and *metaSEM* package (Cheung, 2014) offers functions to perform fixed-effects and random-effects multivariate meta-analysis under the framework of structural equation modeling. All of these packages can handle both AD and IPD.

TABLE 23.8 Software Programs for Meta-analysis*.

	Univariate Meta-analysis	Multivariate Meta-analysis
R (packages)	epiR meta metafor rmeta gmeta	mvmeta mvtmeta metaSEM
Stata (User written commands)	metan metacum metareg	mvmeta mvmeta_make

*More information, including manuals and references, can be found at http://cran.r-project.org/web/views/MetaAnalysis.html and http://www.stata.com/support/faqs/statistics/meta-analysis.

For the multivariate meta-analysis illustrated in the present chapter, we developed our own functions to use in the R programming environment. The existing packages for multivariate meta-analysis discussed above could not be applied due to the complex data structure that we had. As indicated previously in the Multivariate Meta-analysis section, the dimension of the data estimated was high (15 × 15 covariance matrix) and this complexity was compounded with missing data. Thus, the second author wrote custom functions that were suited for the data. Essentially, the idea was to obtain the likelihood function while incorporating the missing data structure, which could not be accomplished using the existing packages. More specifically, we estimated the 15 × 15 between-study covariance matrix using REML by using the R package *optimx*. We also utilized different methods available in the *optimx* package (Nash & Varadhan, 2011) to confirm the final estimates reported in this chapter. We then calculated meta-analyzed individual coefficient estimates (reported in Table 23.7) by plugging the REML estimates into the last

two equations shown in the IPD Meta-analysis and Integrative Data Analysis section. Figures 23.12–23.14 were drawn using the combined individual estimates reported in Table 23.7.

TRANSLATIONAL IMPLICATIONS OF INTEGRATIVE DATA ANALYSIS

IDA is an emerging methodological approach that is well suited for translational research. Translational research, although variously understood by different people, is generally known as the bench-to-bedside translation of knowledge from basic sciences to produce new clinical or treatment options for patients (Woolf, 2008). Through this translation, a new clinical treatment can be brought to scale to treat hard-to-treat problems and to narrow health disparities. For the successful translation of knowledge for evidence-based, bedside clinical practice, however, the integrity and validity of evidence is critical: Evidence-based practice is only as good and valid as the evidence that it is based on.

IDA, especially meta-analysis using IPD, has many promising utilities over single studies and over traditional meta-analysis utilizing summary data from published reports. More broadly, with regard to translational research, IDA may have at least three unique advantages. First, IDA studies using IPD can scrutinize existing findings in the literature and generate more robust findings and conclusions than possible based on single studies or traditional meta-analysis. Second, via IDA, one can examine mechanisms of behavior change with data that are better suited to address them. Third, through the advanced, state-of-the-art multivariate models, one can overcome compartmentalized pieces of evidence at various levels of analysis to create a more comprehensive understanding based on integrated quantitative evidence.

In terms of scrutinizing existing findings and strengthening our statistical inference, meta-analysis is increasingly seen as an important tool to shore up our confidence in evidence. In the present chapter, we suggest that traditional meta-analysis using summary statistics may be viewed as part of IDA using IPD, a broader research synthesis approach involving original raw participant-level data. Both of these approaches are expected to be relied on in the coming years; yet more advances and applications may be in store for IDA using IPD. There have been public calls to raise research standards for single studies and, in response, several stakeholders, such as editors of major journals and the National Institutes of Health (NIH), have proposed a number of changes in research practice (Collins & Tabak, 2014). According to Collins and Tabak, one of the initiatives considered by NIH is to develop a searchable data index for locating and accessing primary data. Thus, IDA using IPD may be called upon to provide a self-correcting function of science, especially for preclinical research, at more rapid pace than before.

Even for clinical research, the examples of antidepressant medications (Turner et al., 2008) and reanalysis of RCTs (Ebrahim et al., 2014) reviewed earlier in the current chapter illustrate the dangers of overly relying on data from published single studies. Therefore, from this perspective, it is promising that researchers are calling for a need to build participatory, prospective collaborative projects to tackle hallmark clinical questions such as what works (on the whole), what works for whom (subgroups or treatment modifiers), and how it works (mechanisms of behavior change) through innovative analysis of pooled data from multiple RCTs (e.g., Collaborative Data Synthesis for Adolescent Depression Trials [CDSADT]; NIMH Collaborative Data Synthesis for Adolescent Depression Trials Study Team, 2013).

In addition, it is very attractive that, via IDA, one can transcend compartmentalized pieces of evidence at various levels of analysis. In its place, one can generate a new understanding that is based on integrated quantitative evidence, which has been largely unobtainable from single studies or review studies that exclusively rely on summary data from single studies. Using IDA, similar studies can be linked to provide comparative evidence of various treatment approaches; generate evidence for treatment efficacy from multiple related outcomes; and provide clues as to the duration of treatment efficacy. Instead of subjectively interpreting and evaluating multiple coefficients obtained from multiple analyses in a piecemeal fashion, we can better grasp, for example, which treatment may be best of all available approaches in the field through the use of advanced methods well suited for IDA, as demonstrated in the data example provided in this chapter.

FUTURE DIRECTIONS AND LIMITATIONS

The research environment for IDA is ripe in many disciplines, including that of psychology. There are several thrusts behind this momentum. First, there is a great movement away from the current research practices that heavily depend on null hypothesis significance testing (NHST), toward new statistical practices that emphasize estimation of effect sizes and confidence intervals, and meta-analysis

(Cumming, 2014). The journal *Psychological Science*, a flagship journal for the Association for Psychological Science, announced new changes in publication standards and practices to be implemented starting January 2014 (Eich, 2014). To shore up the foundations of scientific inquiries, the journal's editors have proposed five broad changes: (1) remove word limits for the methods and results sections; (2) challenge researchers to clarify the what, why, and how questions of the research; (3) adopt the four-item disclosure statement aimed at making visible what goes on behind publications (i.e., undisclosed flexibility in data collection and analysis; Simmons, Nelson, & Simonsohn, 2011); (4) promote open practices by creating incentives to share data and materials when publishing and to declare full research protocols in advance; and (5) emphasize effect sizes, confidence intervals, and meta-analysis. When these changes are fully implemented, metadata repository systems that are searchable and easily accessible by others will create opportunities to quickly validate existing findings.

Second, from the perspective of exploratory, discovery-oriented investigations, data sharing can yield unexpected, serendipitous exploratory findings, which may lead to a prespecified, confirmatory study. Indeed, there are many national survey datasets that can be downloaded for public use. Moreover, in recent years, NIH has launched large, multisite projects expecting that they can serve as seed projects for other investigations. Big initiatives, such as the Human Connectome Project (HCP) and the National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA), take advantage of improved computing capability, easier access to the web for storing and searching information, and convenient use of biological indices to better understand a complex phenomenon across different time scales and across different domains.

A third important thrust is the development of innovative methods for research synthesis. As this chapter demonstrates, there has been an explosion of more flexible, efficient, and powerful research methods to accommodate various needs, and these developments will likely continue. With the improvement in computing capability, we can run computationally demanding models, such as Bayesian models that were unthinkable not long ago. Elucidating the complex developmental processes that we see in developmental psychopathology requires a transdisciplinary systems prospective that cuts across scale and time, which has also been the guiding vision at NIH to promote scientific breakthroughs (Mabry, Olster, Morgan, & Abrams, 2008). IDA is a promising new approach in the era of big data to build a knowledge base that is more robust and cumulative and to move us forward toward more integrated and innovative research.

Despite our enthusiasm for IDA, some cautionary caveats are in order. Controlled, multisite, large studies do not always yield the same results as those from meta-analysis studies. In addition, different meta-analysis approaches—IPD or AD and different models—can lead to different estimates or conclusions (e.g., Haines & Hill, 2011). Recall, in the case of Avandia illustrated previously, the FDA restricted marketing of the drug based on findings from several meta-analysis studies showing greater heart attack risk while simultaneously offering a disclaimer stating that large-scale RCTs did not show a statistically significant risk elevation (Finkelstein & Levin, 2012). Thus, this case demonstrates that there is a need to evaluate all available evidence as a whole, and to be cautious about overly relying on one approach.

In addition, the time and resources required for IDA are considerable and one needs to carefully weigh the benefits of IDA relative to the resources needed. Data processing for IDA can be as intensive as collecting original data (Hussong et al., 2013) because of the need to examine and scrutinize all aspects of the pooled data prior to harmonizing and conducting further analysis to establish measurement invariance across important groups. Missing data resulted from combining data from multiple studies also pose a huge challenge to overcome for studies utilizing IPD. However, as the overall research environment is moving toward preregistration of a trial, reporting of all outcome variables, and better disclosure of methods and results, the environment may become more favorable for IDA studies in the future. Even for nonexperimental studies, similar, more open research practices are likely to follow, which may make it easier to conduct IDA studies.

CONCLUSIONS

Systematic research synthesis, particularly IDA involving original IPD from multiple sources, provides exciting new capabilities. IDA is well suited to address unmet needs in the field of developmental psychopathology and critical challenges in the current research environment. At the same time, there is a need to further develop research methods intended for research synthesis and to bridge our knowledge gaps in implementing these methods for IDA. Despite its many challenges, IDA is expected to be utilized more often in the future as a means to strengthen our research practice and lead to new discoveries.

REFERENCES

Al khalaf, M. M., Thalib, L., & Doi, S. A. R. (2011). Combining heterogenous studies using the random-effects model is a mistake and leads to inconclusive meta-analyses. *Journal of Clinical Epidemiology, 64*(2), 119–123. doi: 10.1016/j.jclinepi.2010.01.009

American College Health Association (2001). *National College Health Assessment ACHA- NCHA reliability and validity analyses*. Baltimore, MD: American College Health Association.

American Psychological Association Publications and Communications Board Working Group on Journal Article Reporting Standards (2008). Reporting standards for research in psychology: Why do we need them? What might they be? *American Psychologist, 63*(9), 839–851. doi: 10.1037/0003-066X.63.9.839

Atkins, D. C., Baldwin, S. A., Zheng, C., Gallop, R. J., & Neighbors, C. (2013). A tutorial on count regression and zero-altered count models for longitudinal substance use data. *Psychology of Addictive Behaviors, 27*(1), 166–177. doi: 10.1037/a0029508

Baer, J. S., Kivlahan, D. R., Blume, A. W., McKnight, P., & Marlatt, G. A. (2001). Brief intervention for heavy-drinking college students: 4-year follow-up and natural history. *American Journal of Public Health, 91*(8), 1310–1318.

Bakker, M., van Dijk, A., & Wicherts, J. M. (2012). The rules of the game called psychological science. *Perspectives on Psychological Science, 7*(6), 543–554. doi: 10.1177/1745691612459060

Barnett, N. P., Murphy, J. G., Colby, S. M., & Monti, P. M. (2007). Efficacy of counselor vs. computer-delivered intervention with mandated college students. *Addictive Behaviors, 32*(11), 2529–2548. doi: 10.1016/j.addbeh.2007.06.017

Bauer, D. J., & Hussong, A. M. (2009). Psychometric approaches for developing commensurate measures across independent studies: Traditional and new models. *Psychological Methods, 14*(2), 101–125. doi: 10.1037/a0015583

Bax, L., Yu, L.-M., Ikeda, N., & Moons, K. (2007). A systematic comparison of software dedicated to meta-analysis of causal studies. *BMC Medical Research Methodology, 7*(1), 40. doi: 10.1186/1471-2288-7-40

Begg, C. B., & Mazumdar, M. (1994). Operating characteristics of a rank correlation test for publication bias. *Biometrics, 50*(4), 1088–1101. doi: 10.2307/2533446

Begley, C. G., & Ellis, L. M. (2012). Drug development: Raise standards for preclinical cancer research. *Nature, 483*(7391), 531–533. doi: 10.1038/483531a

Bergman, L. R., & Magnusson, D. (1997). A person-oriented approach in research on developmental psychopathology. *Development and Psychopathology, 9*(2), 291–319. doi: 10.1017/S095457949700206X

Berman, N., & Parker, R. (2002). Meta-analysis: Neither quick nor easy. *BMC Medical Research Methodology, 2*(1), 10. doi: 10.1186/1471-2288-2-10

Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2005). *Comprehensive Meta-Analysis (Version 2)* [Computer software]. Engelwood, NJ: Biostat.

Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2009). *Introduction to meta-analysis*. West Sussex, UK: Wiley.

Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2010). A basic introduction to fixed-effect and random-effects models for meta-analysis. *Research Synthesis Methods, 1*(2), 97–111. doi: 10.1002/jrsm.12

Borenstein, M., & Higgins, J. P. T. (2013). Meta-analysis and subgroups. *Prevention Science, 14*(2), 134–143. doi: 10.1007/s11121-013-0377-7

Bradburn, M. J., Deeks, J. J., Berlin, J. A., & Russell Localio, A. (2007). Much ado about nothing: A comparison of the performance of meta-analytical methods with rare events. *Statistics in Medicine, 26*(1), 53–77. doi: 10.1002/sim.2528

Breslow, N. (1981). Odds ratio estimators when the data are sparse. *Biometrika, 68*(1), 73–84. doi: 10.2307/2335807

Brown, C. H., Sloboda, Z., Faggiano, F., Teasdale, B., Keller, F., Burkhart, G., . . . Perrino, T. (2013). Methods for synthesizing findings on moderation effects across multiple randomized trials. *Prevention Science, 14*(2), 144–156. doi: 10.1007/s11121-011-0207-8

Brown, L. D., Cai, T. T., & DasGupta, A. (2001). Interval estimation for a binomial proportion. *Statistical Science, 16*(2), 101–117. doi: 10.2307/2676784

Carey, K. B., Scott-Sheldon, L. A., Carey, M. P., & DeMartini, K. S. (2007). Individual-level interventions to reduce college student drinking: A meta-analytic review. *Addictive Behaviors, 32*(11), 2469–2494. doi: 10.1016/j.addbeh.2007.05.004

Carey, K. B., Scott-Sheldon, L. A., Elliott, J. C., Garey, L., & Carey, M. P. (2012). Face-to-face versus computer-delivered alcohol interventions for college drinkers: A meta-analytic review, 1998 to 2010. *Clinical Psychology Review, 32*(8), 690–703. doi: 10.1016/j.cpr.2012.08.001

Chalmers, I. (2003). Trying to do more good than harm in policy and practice: The role of rigorous, transparent, up-to-date evaluations. *ANNALS of the American Academy of Political and Social Science, 589*(1), 22–40. doi: 10.1177/0002716203254762

Chalmers, I., & Matthews, R. (2006). What are the implications of optimism bias in clinical research? *The Lancet, 367*(9509), 449–450. doi: 10.1016/S0140-6736(06)68153-1

Chalmers, T. C., Smith Jr., H., Blackburn, B., Silverman, B., Schroeder, B., Reitman, D., & Ambroz, A. (1981). A method for assessing the quality of a randomized control trial. *Controlled Clinical Trials, 2*(1), 31–49. doi: 10.1016/0197-2456(81)90056-8

Chassin, L., Rogosch, F., & Barrera, M. (1991). Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology, 100*, 449–463. doi: 10.1037/0021-843X.100.4.449

Chen, H. (2012). *mvtmeta: Multivariate meta-analysis* (R package version 1.0). Available from http://cran.r-project.org/web/packages/mvtmeta/mvtmeta.pdf

Cheung, M. W.-L. (2014). Fixed- and random-effects meta-analytic structural equation modeling: Examples and analyses in R. *Behavior Research Methods, 46*, 29–40. doi:10.3758/s13428-013-0361-y

Cicchetti, D., & Toth, S. L. (2009). The past achievements and future promises of developmental psychopathology: The coming of age of a discipline. *Journal of Child Psychology and Psychiatry, 50*(1–2), 16–25. doi: 10.1111/j.1469-7610.2008.01979.x

Cimini, M. D., Martens, M. P., Larimer, M. E., Kilmer, J. R., Neighbors, C., & Monserrat, J. M. (2009). Assessing the effectiveness of peer-facilitated interventions addressing high-risk drinking among judicially mandated college students. *Journal of Studies on Alcohol and Drugs, Supplement*, *16*, 57–66.

Claggett, B., Xie, M., & Tian, L. (2014). Meta analysis with fixed, unknown, study-specific parameters. *Journal of the American Statistical Association*, *109*, 1667–1671. doi: 10.1080/01621459.2014.957288

Cochran, W. G. (1950). The comparison of percentages in matched samples. *Biometrika, 37*(3–4), 256–266. doi:10.1093/biomet/37.3-4.256

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum.

Collins, F. S., & Tabak, L. A. (2014). NIH plans to enhance reproducibility. *Nature, 505*, 612–213.

Cooper, H., & Patall, E. A. (2009). The relative benefits of meta-analysis conducted with individual participant data versus aggregated data. *Psychological Methods, 14*(2), 165–176. doi: 10.1037/a0015565

Cronce, J. M., & Larimer, M. E. (2011). Individual-focused approaches to the prevention of college student drinking. *Alcohol Research & Health, 34*(2), 210–221.

Cumming, G. (2014). The new statistics: Why and how. *Psychological Science, 25*(1), 7–29. doi: 10.1177/0956797613504966

Cumming, G., & Maillardet, R. (2006). Confidence intervals and replication: Where will the next mean fall? *Psychological Methods, 11*(3), 217–227. doi: 10.1037/1082-989X.11.3.217

Curran, P. J. (2009). The seemingly quixotic pursuit of a cumulative psychological science: Introduction to the special issue. *Psychological Methods, 14*(2), 77–80. doi: 10.1037/a0015972

Curran, P. J., & Hussong, A. M. (2009). Integrative data analysis: The simultaneous analysis of multiple data sets. *Psychological Methods, 14*(2), 81–100. doi: 10.1037/a0015914

Curran, P. J., Hussong, A. M., Cai, L., Huang, W., Chassin, L., Sher, K. J., & Zucker, R. A. (2008). Pooling data from multiple longitudinal studies: the role of item response theory in integrative data analysis. *Developmental Psychology, 44*(2), 365–380. doi: 10.1037/0012-1649.44.2.365

Curran, P. J., McGinley, J. S., Bauer, D. J., Hussong, A. M., Burns, A., Chassin, L., . . . Zucker, R. (2014). A moderated nonlinear factor model for the development of commensurate measures in integrative data analysis. *Multivariate Behavioral Research, 49*(3), 214–231. doi: 10.1080/00273171.2014.889594

D'Amico, E. J., & Fromme, K. (1997). Health risk behaviors of adolescent and young adult siblings. *Health Psychology, 16*(5), 426–432. doi: 10.1037/0278-6133.16.5.426

Davey, J., Turner, R. M., Clarke, M. J., & Higgins, J. P. (2011). Characteristics of meta-analyses and their component studies in the Cochrane Database of Systematic Reviews: A cross-sectional, descriptive analysis. *BMC Medical Research Methodology, 11*(1), 160. doi: 10.1186/1471-2288-11-160.

Doornik, J. A. (2009). *Object-oriented matrix programming using Ox (Version 3.1)* [Computer software]. London, UK: Timberlake Consultants.

DerSimonian, R., & Laird, N. (1986). Meta-analysis in clinical trials. *Controlled Clinical Trials, 7*(3), 177–188. doi: 10.1016/0197-2456(86)90046-2

Donovan, E., Wood, M., Frayjo, K., Black, R. A., & Surette, D. A. (2012). A randomized, controlled trial to test the efficacy of an online, parent-based intervention for reducing the risks associated with college-student alcohol use. *Addictive Behaviors, 37*(1), 25–35. doi: 10.1016/j.addbeh.2011.09.007

Duval, S., & Tweedie, R. (2000). Trim and fill: A simple funnel-plot–based method of testing and adjusting for publication bias in meta-analysis. *Biometrics, 56*(2), 455–463. doi: 10.1111/j.0006-341X.2000.00455.x

Ebrahim, S., Sohani, Z. N., Montoya, L., Agarwal, A., Thorlund, K., Mills, E. J., & Ioannidis, J. P. A. (2014). Reanalyses of randomized clinical trial data. *Journal of American Medical Association, 312*(10), 1024–1032. doi: 10.1001/jama.2014.9646

Egger, M., Smith, G. D., Schneider, M., & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *British Medical Journal, 315*(7109), 629–634. doi: 10.1136/bmj.315.7109.629

Eich, E. (2014). Business not as usual. *Psychological Science, 25*(1), 3–6. doi: 10.1177/0956797613512465

Ferguson, C. J., & Brannick, M. T. (2012). Publication bias in psychological science: Prevalence, methods for identifying and controlling, and implications for the use of meta-analyses. *Psychological Methods, 17*(1), 120–128. doi: 10.1037/a0024445

Finkelstein, M. O., & Levin, B. (2012). Meta-analysis of "sparse" data: Perspectives from the Avandia cases. *Jurimetrics, 52*(2), 123–153.

Fisher, R. A. (1948). Questions and answers #14. *The American Statistician, 2*(5), 30–31. doi: 10.2307/2681650

Ford, D. H., & Lerner, R. M. (1992). *Developmental systems theory*. Newbury Park, CA: SAGE.

Fromme, K., & Corbin, W. (2004). Prevention of heavy drinking and associated negative consequences among mandated and voluntary college students. *Journal of Consulting and Clinical Psychology, 72*(6), 1038–1049. doi: 10.1037/0022-006X.72.6.1038

Gart, J. J. (1970). Point and interval estimation of the common odds ratio in the combination of 2 × 2 tables with fixed marginals. *Biometrika, 57*(3), 471–475. doi: 10.1093/biomet/57.3.471

Gasparrini, A., Armstrong, B., & Kenward, M. G. (2012). Multivariate meta-analysis for non-linear and other multi-parameter associations. *Statistics in Medicine, 31*(29), 3821–3839. doi: 10.1002/sim.5471

Haines, T. P., & Hill, A.-M. (2011). Inconsistent results in meta-analyses for the prevention of falls are found between study-level data and patient-level data. *Journal of Clinical Epidemiology, 64*(2), 154–162. doi: 10.1016/j.jclinepi.2010.04.024

Herbison, P., Hay-Smith, J., & Gillespie, W. J. (2006). Adjustment of meta-analyses on the basis of quality scores should be abandoned. *Journal of Clinical Epidemiology, 59*(12), 1249.e1241-1249.e1211. doi: 10.1016/j.jclinepi.2006.03.008

Higgins, J. P. T., & Green, S. (Eds.). (2011). *Cochrane handbook for systematic reviews of interventions (ver 5.1.0)*. Available from www.cochrane-handbook.org: The Cochrane Collaboration.

Higgins, J. P. T., & Thompson, S. G. (2002). Quantifying heterogeneity in a meta-analysis. *Statistics in Medicine, 21*(11), 1539–1558. doi: 10.1002/sim.1186

Hoaglin, D. C., Hawkins, N., Jansen, J. P., Scott, D. A., Itzler, R., Cappelleri, J. C., . . . Barrett, A. (2011). Conducting indirect-treatment-comparison and network-meta-analysis studies: Report of the ISPOR Task Force on indirect treatment comparisons good research practices: Part 2. *Value in Health, 14*(4), 429–437. doi: 10.1016/j.jval.2011.01.011

Huh, D., Mun, E.-Y., Larimer, M. E., White, H. R., Ray, A. E., Rhew, I., . . . Atkins, D. C. (2015). Brief motivational interventions for college student drinking may not be as powerful as we think: An individual participant-level data meta-analysis. *Alcoholism: Clinical and Experimental Research, 39*(5), 919–931. doi: 10.1111/acer.12714

Huo, Y., de la Torre, J., Mun, E.-Y., Kim, S.-Y., Ray, A. E., Jiao, Y., & White, H. R. (2014). A hierarchical multi-unidimensional IRT approach for analyzing sparse, multi-group data for integrative data analysis. *Psychometrika. Advance online publication.* doi: 10.1007/s11336-014-9420-2

Hurlbut, S. C., & Sher, K. J. (1992). Assessing alcohol problems in college students. *Journal of American College Health, 41*(2), 49–58. doi: 10.1080/07448481.1992.10392818

Hussong, A. M., Curran, P. J., & Bauer, D. J. (2013). Integrative data analysis in clinical psychology research. *Annual Review of Clinical Psychology, 9*(1), 61–89. doi: 10.1146/annurev-clinpsy-050212-185522

Hussong, A. M., Huang, W., Curran, P. J., Chassin, L., & Zucker, R. A. (2010). Parent alcoholism impacts the severity and timing of children's externalizing symptoms. *Journal of Abnormal Child Psychology, 38*(3), 367–380. doi: 10.1007/s10802-009-9374-5

Hussong, A. M., Wirth, R. J., Edwards, M. C., Curran, P. J., Chassin, L. A., & Zucker, R. A. (2007). Externalizing symptoms among children of alcoholic parents: Entry points for an antisocial pathway to alcoholism. *Journal of Abnormal Psychology, 116*(3), 529–542. doi: 10.1037/0021-843X.116.3.529

Ioannidis, J. P. A. (2005). Why most published research findings are false. *PLoS Med, 2*(8), e124. doi: 10.1371/journal.pmed.0020124

Ioannidis, J. P. A. (2012). Why science is not necessarily self-correcting. *Perspectives on Psychological Science, 7*(6), 645–654. doi: 10.1177/1745691612464056

Ioannidis, J. P. A., Gwinn, M., Little, J., Higgins, J. P. T., Bernstein, J. L., Boffetta, P., . . . , & The Human Genome Epidemiology Network and the Network of Investigator Networks (2006). A road map for efficient and reliable human genome epidemiology. *Nature Genetics, 38*(1), 3–5. doi: 10.1038/ng0106-3

Jackson, D., Riley, R., & White, I. R. (2011). Multivariate meta-analysis: Potential and promise. *Statistics in Medicine, 30*(20), 2481–2498. doi: 10.1002/sim.4172

Jackson, D., White, I. R., & Thompson, S. G. (2010). Extending DerSimonian and Laird's methodology to perform multivariate random effects meta-analyses. *Statistics in Medicine, 29*(12), 1282–1297. doi: 10.1002/sim.3602

Jansen, J. P., Fleurence, R., Devine, B., Itzler, R., Barrett, A., Hawkins, N., . . . Cappelleri, J. C. (2011). Interpreting indirect treatment comparisons and network meta-analysis for health-care decision making: Report of the ISPOR Task Force on indirect treatment comparisons good research practices: Part 1. *Value in Health, 14*(4), 417–428. doi: 10.1016/j.jval.2011.04.002

Kahler, C. W., Strong, D. R., & Read, J. P. (2005). Toward efficient and comprehensive measurement of the alcohol problems continuum in college students: The Brief Young Adult Alcohol Consequences Questionnaire. *Alcoholism: Clinical and Experimental Research, 29*(7), 1180–1189. doi: 10.1097/01.alc.0000171940.95813.a5

Kraemer, H., Mintz, J., Noda, A., Tinklenberg, J., & Yesavage, J. A. (2006). Caution regarding the use of pilot studies to guide power calculations for study proposals. *Archives of General Psychiatry, 63*(5), 484–489. doi: 10.1001/archpsyc.63.5.484

Kuntsche, E., Rossow, I., Simons-Morton, B., Bogt, T. T., Kokkevi, A., & Godeau, E. (2013). Not early drinking but early drunkenness is a risk factor for problem behaviors among adolescents from 38 European and North American countries. *Alcoholism: Clinical and Experimental Research, 37*(2), 308–314. doi: 10.1111/j.1530-0277.2012.01895.x

LaBrie, J. W., Huchting, K. K., Lac, A., Tawalbeh, S., Thompson, A. D., & Larimer, M. E. (2009). Preventing risky drinking in first-year college women: Further validation of a female-specific motivational-enhancement group intervention. *Journal of Studies on Alcohol and Drugs, Suppl*(16), 77–85.

LaBrie, J. W., Huchting, K., Tawalbeh, S., Pedersen, E. R., Thompson, A. D., Shelesky, K., . . . Neighbors, C. (2008). A randomized motivational enhancement prevention group reduces drinking and alcohol consequences in first-year college women. *Psychology of Addictive Behaviors, 22*(1), 149–155. doi: 10.1037/0893-164X.22.1.149

LaBrie, J. W., Hummer, J. F., Neighbors, C., & Pedersen, E. R. (2008). Live interactive group-specific normative feedback reduces misperceptions and drinking in college students: A randomized cluster trial. *Psychology of Addictive Behaviors, 22*(1), 141–148. doi: 10.1037/0893-164X.22.1.141

LaBrie, J. W., Lamb, T. F., Pedersen, E. R., & Quinlan, T. (2006). A group motivational interviewing intervention reduces drinking and alcohol-related consequences in adjudicated college students. *Journal of College Student Development, 47*(3), 267–280.

LaBrie, J. W., Pedersen, E. R., Lamb, T. F., & Quinlan, T. (2007). A campus-based motivational enhancement group intervention reduces problematic drinking in freshmen male college students. *Addictive Behaviors, 32*(5), 889–901. doi: 10.1016/j.addbeh.2006.06.030

LaBrie, J. W., Thompson, A., Huchting, K., Lac, A., & Buckley, K. (2007). A group motivational interviewing intervention reduces drinking and alcohol-related negative consequences in adjudicated college women. *Addictive Behaviors, 32*(11), 2549–2562. doi: 10.1016/j.addbeh.2007.05.014

Larimer, M. E., & Cronce, J. M. (2007). Identification, prevention, and treatment revisited: Individual-focused college drinking prevention strategies 1999–2006. *Addictive Behaviors, 32*(11), 2439–2468. doi: 10.1016/j.addbeh.2007.05.006

Larimer, M. E., Lee, C. M., Kilmer, J. R., Fabiano, P. M., Stark, C. B., Geisner, I. M., . . . Neighbors, C. (2007). Personalized mailed feedback for college drinking prevention: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 75*(2), 285–293. doi: 10.1037/0022-006X.75.2.285

Larimer, M. E., Turner, A. P., Anderson, B. K., Fader, J. S., Kilmer, J. R., Palmer, R. S., & Cronce, J. M. (2001). Evaluating a brief alcohol intervention with fraternities. *Journal of Studies on Alcohol, 62*(3), 370–380.

Leandro, G. (2005). *Meta-analysis in medical research*. Malden, MA: BMJ Books.

Lee, C., & White, H. R. (2012). Effects of childhood maltreatment on violent injuries and premature death during young adulthood among urban high-risk men. *Archives of Pediatrics & Adolescent Medicine, 166*(9), 814–820. doi: 10.1001/archpediatrics.2012.244

Lee, C. M., Kaysen, D. L., Neighbor, C., Kilmer, J. R., & Larimer, M. E. (2009). *Feasibility, acceptability, and efficacy of brief interventions for college drinking: Comparison of group, individual, and web-based alcohol prevention formats.* Unpublished manuscript, Department of Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA.

Lin, D. Y., & Zeng, D. (2010). On the relative efficiency of using summary statistics versus individual-level data in meta-analysis. *Biometrika, 97*(2), 321–332. doi: 10.1093/biomet/asq006

Liu, D., Liu, R. Y., & Xie, M. (2014). Exact meta-analysis approach for discrete data and its application to 2 × 2 tables with rare events. *Journal of the American Statistical Association*, *109*, 1450–1465. doi: 10.1080/01621459.2014.946318

Liu, D., Liu, R. Y., & Xie, M. (2015). Multivariate meta-analysis of heterogeneous studies using only summary statistics: Efficiency and robustness. *Journal of the American Statistical Association, 110*, 326–340. doi: 10.1080/01621459.2014.899235

Loeber, R., Pardini, D., Homish, D. L., Wei, E. H., Crawford, A. M., Farrington, D. P., . . . Rosenfeld, R. (2005). The prediction of violence and homicide in young men. *Journal of Consulting and Clinical Psychology, 73*(6), 1074–1088. doi: 10.1037/0022-006X.73.6.1074

Mabry, P. L., Olster, D. H., Morgan, G. D., & Abrams, D. B. (2008). Interdisciplinarity and systems science to improve population health: A view from the NIH Office of Behavioral and Social Sciences Research. *American Journal of Preventive Medicine, 35*(2, Supplement), S211–S224. doi: 10.1016/j.amepre.2008.05.018

Magnusson, D. (2000). The individual as the organizing principle in psychological inquiry: A holistic approach. In L. R. Bergman, R. B. Cairns, L.-G. Nilsson, & L. Nystedt (Eds.), *Developmental science and the holistic approach* (pp. 33–48). Mahwah, NJ: Erlbaum.

Mantel, N., & Haenszel, W. (1959). Statistical aspects of the analysis of data from retrospective studies of disease. *Journal of the National Cancer Institute, 22*, 719–748.

Marden, J. I. (1991). Sensitive and sturdy p-values. *The Annals of Statistics, 19*(2), 918–934. doi: 10.2307/2242091

Marszalhk, J. M., Barber, C., Kohlhart, J., & Holmes, C. B. (2011). Sample size in psychological research over the past 30 years. *Perceptual & Motor Skills, 112*(2), 331–348. doi: 10.2466/03.11.pms.112.2.331-348

Martens, M. P., Ferrier, A. G., Sheehy, M. J., Corbett, K., Anderson, D. A., & Simmons, A. (2005). Development of the Protective Behavioral Strategies Survey. *Journal of studies on alcohol, 66*(5), 698–705.

Martens, M. P., Kilmer, J. R., Beck, N. C., & Zamboanga, B. L. (2010). The efficacy of a targeted personalized drinking feedback intervention among intercollegiate athletes: A randomized controlled trial. *Psychology of Addictive Behaviors, 24*(4), 660–669. doi: 10.1037/a0020299

Martens, M. P., Taylor, K. K., Damann, K. M., Page, J. C., Mowry, E. S., & Cimini, M. D. (2004). Protective behavioral strategies when drinking alcohol and their relationship to negative alcohol-related consequences in college students. *Psychology of Addictive Behaviors, 18*(4), 390–393. doi: 10.1037/0893-164X.18.4.390

McArdle, J. J., Grimm, K. J., Hamagami, F., Bowles, R. P., & Meredith, W. (2009). Modeling life-span growth curves of cognition using longitudinal data with multiple samples and changing scales of measurement. *Psychological Methods, 14*(2), 126–149. doi: 10.1037/a0015857

Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., & The PRISMA Group (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *PLoS Med, 6*(7), e1000097. doi: 10.1371/journal.pmed.1000097

Moreno, S. G., Sutton, A. J., Turner, E. H., Abrams, K. R., Cooper, N. J., Palmer, T. M., & Ades, A. E. (2009). Novel methods to deal with publication biases: Secondary analysis of antidepressant trials in the FDA trial registry database and related journal publications. *BMJ, 339*, b2981. doi: 10.1136/bmj.b2981

Mun, E.-Y., Bates, M. E., & Vaschillo, E. (2010). Closing the gap between person-oriented theory and methods. *Development and Psychopathology, 22*(2), 261–271. doi: 10.1017/S0954579410000039

Mun, E.-Y., de la Torre, J., Atkins, D. C., White, H. R., Ray, A. E., Kim, S.-Y., . . . , & The Project INTEGRATE Team (2015). Project INTEGRATE: An integrative study of brief alcohol intervention trials for college students. *Psychology of Addictive Behaviors, 29*(1), 34–48. doi: 10.1037/adb0000047

Mun, E.-Y., White, H. R., & Morgan, T. J. (2009). Individual and situational factors that influence the efficacy of personalized feedback substance use interventions for mandated college students. *Journal of Consulting and Clinical Psychology, 77*(1), 88–102. doi: 10.1037/a0014679

Muraki, E. (1992). A generalized partial credit model: Application of an EM algorithm. *Applied Psychological Measurement, 16*(2), 159–176. doi: 10.1177/014662169201600206

Murphy, J. G., Benson, T. A., & Vuchinich, R. E. (2004). A comparison of personalized feedback for college student drinkers delivered with and without a motivational interview. *Journal of Studies on Alcohol, 65*(2), 200–203.

Murphy, J. G., Duchnick, J. J., Vuchinich, R. E., Davison, J. W., Karg, R. S., Olson, A. M., . . . Coffey, T. T. (2001). Relative efficacy of a brief motivational intervention for college student drinkers. *Psychology of Addictive Behaviors, 15*(4), 373–379. doi: 10.1037/0893-164X.15.4.373

Nash, J. C., & Varadhan, R. (2011). Unifying optimization algorithms to aid software system users: Optimx for R. *Journal of Statistical Software, 43*(9), 1–14.

NIMH Collaborative Data Synthesis for Adolescent Depression Trials Study Team (2013). Advancing science through collaborative data sharing and synthesis. *Perspectives on Psychological Science, 8*(4), 433–444. doi: 10.1177/1745691613491579

Nissen, S. E., & Wolski, K. (2007). Effect of rosiglitazone on the risk of myocardial infarction and death from cardiovascular causes. *New England Journal of Medicine, 356*(24), 2457–2471. doi: 10.1056/NEJMoa072761

Normand, S.-L. T. (1999). Meta-analysis: formulating, evaluating, combining, and reporting. *Statistics in Medicine, 18*(3), 321–359. doi: 10.1002/(sici)1097-0258(19990215)18:3<321::aid-sim28>3.0.co;2-p

Nosek, B. A., Spies, J. R., & Motyl, M. (2012). Scientific utopia: II. Restructuring incentives and practices to promote truth over publishability. *Perspectives on Psychological Science, 7*(6), 615–631. doi: 10.1177/1745691612459058

Nuzzo, R. (2014). Scientific method: Statistical errors. *Nature, 506*, 150–152. doi: 10.1038/506150a

Open Science Collaboration. (2012). An open, large-scale, collaborative effort to estimate the reproducibility of psychological science. *Perspectives on Psychological Science, 7*(6), 657–660. doi: 10.1177/1745691612462588

O'Rourke, K. (2007). An historical perspective on meta-analysis: Dealing quantitatively with varying study results. *Journal of the Royal Society of Medicine, 100*(12), 579–582. doi: 10.1258/jrsm.100.12.579

Pashler, H., & Wagenmakers, E. J. (2012). Editors' introduction to the special section on replicability in psychological science: A crisis of confidence? *Perspectives on Psychological Science, 7*(6), 528–530. doi: 10.1177/1745691612465253

R Core Team. (2014). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing.

Ray, A. E., Kim, S.-Y., White, H. R., Larimer, M. E., Mun, E. Y., Clarke, N., . . . , & The Project INTEGRATE Team. (2014). When less is more and more is less in brief motivational interventions: Characteristics of intervention content and their associations with drinking outcomes. *Psychology of Addictive Behaviors, 28*, 1026–1040. doi: 10.1037/a0036593

Reitsma, J. B., Glas, A. S., Rutjes, A. W. S., Scholten, R. J. P. M., Bossuyt, P. M., & Zwinderman, A. H. (2005). Bivariate analysis of sensitivity and specificity produces informative summary measures in diagnostic reviews. *Journal of Clinical Epidemiology, 58*(10), 982–990. doi: 10.1016/j.jclinepi.2005.02.022

Riley, R. (2009). Multivariate meta-analysis: The effect of ignoring within-study correlation. *Journal of the Royal Statistical Society: Series A, 172*(4), 789–811. doi: 10.1111/j.1467-985X.2008.00593.x

Robins, J., Breslow, N., & Greenland, S. (1986). Estimators of the Mantel-Haenszel variance consistent in both sparse data and large-strata limiting models. *Biometrics, 42*(2), 311–323. doi: 10.2307/2531052

Roerecke, M., & Rehm, J. (2012). The cardioprotective association of average alcohol consumption and ischaemic heart disease: A systematic review and meta-analysis. *Addiction, 107*(7), 1246–1260. doi: 10.1111/j.1360-0443.2012.03780.x

Saunders, J. B., Aasland, O. G., Babor, T. F., De La Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on early detection of persons with harmful alcohol consumption-II. *Addiction, 88*(6), 791–804. doi: 10.1111/j.1360-0443.1993.tb02093.x

Scheidler, J., Hricak, H., Yu, K. K., Subak, L., & Segal, M. R. (1997). Radiological evaluation of lymph node metastases in patients with cervical cancer: A meta-analysis. *Journal of the American Medical Association, 278*(13), 1096–1101. doi: 10.1001/jama.1997.03550130070040

Schulz, K. F., Altman, D. G., & Moher, D. (2010). CONSORT 2010 statement: Updated guidelines for reporting parallel group randomised trials. *PLoS Med, 7*(3), e1000251. doi: 10.1371/journal.pmed.1000251

Schumann, G., Coin, L. J., Lourdusamy, A., Charoen, P., Berger, K. H., Stacey, D., . . . Elliott, P. (2011). Genome-wide association and genetic functional studies identify autism susceptibility candidate 2 gene (AUTS2) in the regulation of alcohol consumption. *Proceedings of the National Academy of Sciences, 108*(17), 7119–7124. doi: 10.1073/pnas.1017288108

Shadish, W. R., Cook, T. D., & Campbell, D. T. (2002). *Experimental and quasi-experimental designs for generalized causal inference*. New York, NY: Houghton Mifflin.

Shentu, Y., & Xie, M. (2010). A note on dichotomization of continuous response variable in the presence of contamination and model misspecification. *Statistics in Medicine, 29*(21), 2200–2214. doi: 10.1002/sim.3966

Simmonds, M. C., & Higgins, J. P. T. (2007). Covariate heterogeneity in meta-analysis: Criteria for deciding between meta-regression and individual patient data. *Statistics in Medicine, 26*(15), 2982–2999. doi: 10.1002/sim.2768

Simmons, J. P., Nelson, L. D., & Simonsohn, U. (2011). False-positive psychology: Undisclosed flexibility in data collection and analysis allows presenting anything as significant. *Psychological Science, 22*, 1359–1366. doi: 10.1177/0956797611417632

Skinner, H. A., & Allen, B., A. (1982). Alcohol dependence syndrome: Measurement and validation. *Journal of Abnormal Psychology, 91*(3), 199–209.

Skinner, H. A., & Horn, J. L. (1984). *Alcohol Dependence Scale: Users guide*. Toronto, Canada: Addiction Research Foundation.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development, 55*(1), 17–29. doi: 10.2307/1129832

StataCorp. (2013). *Stata statistical software (version 13)* [Computer software]. College Station, TX: StataCorp LP.

Steenkamp, J.-B. E. M., & Baumgartner, H. (1998). Assessing measurement invariance in cross-national consumer research. *Journal of Consumer Research, 25*, 78–107. doi: 10.1086/209528

Steinberg, K. K., Smith, S. J., Stroup, D. F., Olkin, I., Lee, N. C., Williamson, G. D., & Thacker, S. B. (1997). Comparison of effect estimates from a meta-analysis of summary data from published studies and from a meta-analysis using individual patient data for ovarian cancer studies. *American Journal Epidemiology, 145*(10), 917–925.

Sterba, S. K., & Bauer, D. J. (2010). Matching method with theory in person-oriented developmental psychopathology research. *Development and Psychopathology, 22*(2), 239–254. doi: 10.1017/S0954579410000015

Stewart, L. A. (1995). Practical methodology of meta-analyses (overviews) using updated individual patient data. *Statistics in Medicine, 14*(19), 2057–2079. doi: 10.1002/sim.4780141902

Stouffer, S. A., Suchman, E. A., DeVinney, L. C., Star, S. A., & Williams, Jr., R. M. (1949). *The American soldier: vol. 1. Adjustment during army life*. Princeton, NJ: Princeton University Press.

Substance Abuse and Mental Health Services Administration (SAMHSA) (2012). *National Survey on Drug Use and Health (NSDUH)*. Retrieved from http://www.samhsa.gov/data/NSDUH/2012SummNatFindDetTables/DetTabs/NSDUH-DetTabsSect5peTabs1to56-2012.htm#Tab5.31b

Supplee, L. H., Kelly, B. C., MacKinnon, D. P., & Barofsky, M. Y. (2013). Introduction to the special issue: Subgroup analysis in prevention and intervention research. *Prevention Science, 14*(2), 107–110. doi: 10.1007/s11121-012-0335-9

Sutton, A. J., & Higgins, J. P. T. (2008). Recent developments in meta-analysis. *Statistics in Medicine, 27*(5), 625–650. doi: 10.1002/sim.2934

Sutton, A. J., Kendrick, D., & Coupland, C. A. (2008). Meta-analysis of individual- and aggregate-level data. *Statistics in Medicine, 27*(5), 651–669. doi: 10.1002/sim.2916

Thompson, S. G., & Pocock, S. J. (1991). Can meta-analyses be trusted? *The Lancet, 338*(8775), 1127–1130.

Tian, L., Cai, T., Pfeffer, M. A., Piankov, N., Cremieux, P.-Y., & Wei, L. J. (2009). Exact and efficient inference procedure for meta-analysis and its application to the analysis of independent 2×2 tables with all available data but without artificial continuity correction. *Biostatistics, 10*(2), 275–281. doi: 10.1093/biostatistics/kxn034

Tse, T., Williams, R. J., & Zarin, D. A. (2009). Reporting "basic results" in ClinicalTrials.gov. *Chest, 136*(1), 295–303. doi: 10.1378/chest.08-3022

Turner, E. H., Matthews, A. M., Linardatos, E., Tell, R. A., & Rosenthal, R. (2008). Selective publication of antidepressant trials and its influence on apparent efficacy. *New England Journal of Medicine, 358*(3), 252–260. doi: 10.1056/NEJMsa065779

Tversky, A., & Kahneman, D. (1971). Belief in the law of small numbers. *Psychological Bulletin, 76*(2), 105–110. doi: 10.1037/h0031322

Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software*, *36*, 1–48.

Walters, S. T., & Neighbors, C. (2005). Feedback interventions for college alcohol misuse: What, why and for whom? *Addictive Behaviors, 30*(6), 1168–1182. doi: 10.1016/j.addbeh.2004.12.005

Walters, S. T., Vader, A. M., & Harris, T. R. (2007). A controlled trial of web-based feedback for heavy drinking college students. *Prevention Science, 8*(1), 83–88. doi: 10.1007/s11121-006-0059-9

Walters, S. T., Vader, A. M., Harris, T. R., & Jouriles, E. N. (2009). Reactivity to alcohol assessment measures: An experimental test. *Addiction, 104*(8), 1305–1310. doi: 10.1111/j.1360-0443.2009.02632.x

Walters, S. T., Vader, A. M., Harris, T. R., Field, C. A., & Jouriles, E. N. (2009). Dismantling motivational interviewing and feedback for college drinkers: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 77*(1), 64–73. doi: 2009-00563-015 [pii]10.1037/a0014472

Weiss, B., & Garber, J. (2003). Developmental differences in the phenomenology of depression. *Development and Psychopathology, 15*(2), 403–430. doi: 10.1017/S0954579403000221

Werch, C. E., & Gorman, D. R. (1988). Relationship between self-control and alcohol consumption patterns and problems of college students. *Journal of Studies on Alcohol, 49*(1), 30–37.

White, H. R., & Labouvie, E. W. (1989). Towards the assessment of adolescent problem drinking. *Journal of Studies on Alcohol, 50*(1), 30–37.

White, H. R., Lee, C., Mun, E.-Y., & Loeber, R. (2012). Developmental patterns of alcohol use in relation to the persistence and desistance of serious violent offending among African American and Caucasian young men. *Criminology, 50*(2), 391–426. doi: 10.1111/j.1745-9125.2011.00263.x

White, H. R., Mun, E.-Y., & Morgan, T. J. (2008). Do brief personalized feedback interventions work for mandated students or is it just getting caught that works? *Psychology of Addictive Behaviors, 22*(1), 107–116. doi:10.1037/0893-164x.22.1.107

White, H. R., Mun, E.-Y., Pugh, L., & Morgan, T. J. (2007). Long-term effects of brief substance use interventions for mandated college students: Sleeper effects of an in-person personal feedback intervention. *Alcoholism: Clinical and Experimental Research, 31*(8), 1380–1391. doi: 10.1111/j.1530-0277.2007.00435.x

Wilk, A. I., Jensen, N. M., & Havighurst, T. C. (1997). Meta-analysis of randomized control trials addressing brief interventions in heavy alcohol drinkers. *Journal of General Internal Medicine, 12*(5), 274–283. doi: 10.1046/j.1525-1497.1997.012005274.x

Wood, M. D., Capone, C., Laforge, R., Erickson, D. J., & Brand, N. H. (2007). Brief motivational intervention and alcohol expectancy challenge with heavy drinking college students: A randomized factorial study. *Addictive Behaviors, 32*(11), 2509–2528. doi: 10.1016/j.addbeh.2007.06.018

Wood, M. D., Fairlie, A. M., Fernandez, A. C., Borsari, B., Capone, C., Laforge, R., & Carmona-Barros, R. (2010). Brief motivational and parent interventions for college students: A randomized factorial study. *Journal of Consulting and Clinical Psychology, 78*(3), 349–361. doi: 10.1037/a0019166

Woolf, S. H. (2008). The meaning of translational research and why it matters. *Journal of the American Medical Association, 299*(2), 211–213. doi: 10.1001/jama.2007.26

Xie, M., & Singh, K. (2013). Confidence distribution, the frequentist distribution estimator of a parameter: A review. *International Statistical Review, 81*(1), 3–39. doi: 10.1111/insr.12000

Xie, M., Singh, K., & Strawderman, W. E. (2011). Confidence distributions and a unifying framework for meta-analysis. *Journal of the American Statistical Association, 106*(493), 320–333. doi: 10.1198/jasa.2011.tm09803

Yamaguchi, Y., Sakamoto, W., Goto, M., Staessen, J. A., Wang, J., Gueyffier, F., & Riley, R. D. (2014). Meta-analysis of a continuous outcome combining individual patient data and aggregate data: a method based on simulated individual patient data. *Research Synthesis Methods, n/a-n/a.* doi: 10.1002/jrsm.1119

Yang, G., & Xie, M. (2010). *gmeta: An R package unified meta-analysis methods through combining confidence distributions*. Piscataway, NJ: Department of Statistics, Rutgers, The State University of New Jersey.

Yusuf, S., Peto, R., Lewis, J., Collins, R., & Sleight, P. (1985). Beta blockade during and after myocardial infarction: An overview of the randomized trials. *Progress in Cardiovascular Diseases, 27*, 335–371.

Zucker, R. A., Fitzgerald, H. E., Refior, S. K., Puttler, L. I. Pallas, D. M., & Ellis, D. A. (2000). The clinical and social ecology of childhood for children of alcoholics: Description of a study and implications for a differentiated social policy. In H. E. Fitzgerald, B. M. Lester, & B. S. Zuckerman (Eds.), *Children of addiction: Research, health and policy issues* (pp. 174–222). New York, NY: Garland Press.

Author Index

Aalen, O. O., 980
Aasland, O. G., 1071
Abad-Franch, F., 174
Abaied, J. L., 297
Abbott, D.A., 493
Abela, J. R., 289, 300
Abela, J. Z., 556
Abelson, R. P., 332
Aber, J. L., 296, 529, 536, 679, 719
Aber, M. S., 851
Aberg, K. A., 121
Abidin, R. R., 36
Ablard, K. E., 342
Ablow, J. C., 35, 37, 241, 250, 297
Abma, J. C., 632
Abraham, F. D., 727, 736
Abraham, R. H., 727, 736
Abramowitz, M., 884, 885
Abrams, D. B., 1081
Abrams, K. Y., 340
Abramson, L. Y., 288, 488, 494, 537
Abrohms, I. F., 446
Abuhamdeh, S., 400, 404
Abwender, D. A., 637
Academy of Medical Sciences, 131
Achee, J., 745
Achenbach, T. M., 7, 10, 12, 17, 24, 25, 27, 47, 51, 52, 53, 54, 55, 59, 60, 62, 63, 64, 65, 66, 72, 73, 76, 77, 78, 79, 83, 84, 85, 86, 87, 100, 109, 110, 353, 856, 1007, 1013, 1018, 1030, 1032
Acitelli, L. K., 491
Ackard, D. M., 643
Ackerman, B. P., 228, 232, 268, 269
Ackerman, N. W., 581
Ackermann, C., 291
Adam, E. K., 362, 367, 368
Adam, K. S., 362
Adams, D., 275
Adams, E., 284, 698
Adams, J., 847
Adams, N., 847
Adams, R. E., 554
Adamson, G., 785
Adamson, L. B., 291, 452, 455
Adelman, C. B., 562, 565
Ader, R., 165, 175
Adolphs, R., 283
Adrian, M., 284, 293, 298
Aelterman, N., 426
Afrank, J., 622
Aggen, S., 858
Agresti, A., 822, 823, 825
Ahmad, I., 405
Ahnert, L., 347, 348, 356
Aho, K., 700
Aiken, J. R., 704
Aiken, L. S., 923, 926, 927, 928, 937, 972
Aikins, J. W., 538, 552, 556, 560, 648
Ainley, M., 517
Ainsworth, M. D. S., 163, 169, 201, 206, 326, 328, 329, 330, 332, 336, 337, 367, 396, 679
Ajzen, I., 563
Akaike, H., 1033
Akee, R., 143
Akerman, A., 233, 234
Akkerhuis, G. W., 109
Aksan, N., 35, 246, 698, 700
Alanen, E., 568
Alansky, J. A., 337, 341
Albersheim, L., 169, 296, 347
Albert, D., 558
Albrecht, E. C., 746
Albus, K. E., 346
Aldao, A., 268, 294
Alder, N., 691
Alexander, J. F., 584
Alexander, K. L., 147
Alibali, M., 676
Alicke, M., 481
Alik, J., 798
Alisch, R. S., 168
Al khalaf, M. M., 1061
Allan, S., 245
Allan, W., 532
Allen, D.N., 8
Allen, M., 510
Allen, B. A., 1071
Allen, J. P., 168, 169, 246, 296, 349, 541, 564, 589
Allen, K., 589
Allen, L., 108
Allen, N. B., 278, 294, 297, 301, 558, 559, 726, 852
Allen, V. L., 113
Alley, A., 109
Allgood, S. F., 798
Allison, E. R., 761
Allison, K., 284
Allison, P. D., 731, 760, 775, 777
Allison, S. T., 560
Alloy, L. B., 288, 494, 537
Allport, G. W., 508, 812, 813
Allwood, M., 218, 219
Almas, A. N., 298
Almeida, A., 503
Almeida, D. M., 224, 588
Almeida, N. D., 166
Alpert, A., 236, 287, 616
Als, H., 291, 452, 455
Alsup, R., 923
Althaus, M., 109
Altman, D. G., 1052
Alvidrez, J., 8
Amabile, T. M., 399
Amadó, A., 284
Amato, P. R., 199, 232
Ambady, N., 281
Ambrosini, P. J., 53, 110
Amemiya, Y., 972
American College Health Association, 1070
American Psychiatric Association, 36, 51, 53, 54, 59, 60, 61, 62, 78, 98, 99, 102, 527, 593, 635, 799
American Psychological Association, 1033
American Psychological Association Publications Communications Board Working Group on Journal Article Reporting Standards, 1052
Ames, D. R., 458
Amiel-Tison, C., 159
Amir, N., 223
Ammaniti, M., 333
Amsterdam, B. K., 457, 458, 461
Anders, S., 170
Andersen, S. L., 171
Anderson, S.C., 401
Anderson, A. L., 271
Anderson, A. V., 501
Anderson, C. A., 699
Anderson, C. M., 169
Anderson, E. P., 330
Anderson, K. E., 131
Anderson, L. P., 590
Anderson, R., 277
Anderson, T. W., 767
Anderson-Fye, E. P., 498
Andersson, H., 800, 801, 802, 803, 804, 809, 810, 850
Andersson-Norinder, J., 275
Andreas, A., 34
Andreas, D., 341
Andreas, J., 701
Andrews, B., 696
Andrews, D. W., 557, 563, 728
Andrews, G., 636, 640
Andrews, J. A., 640, 642
Andrews, J. D. W., 368
Andrews, M. H., 175
Andrews, R., 8
Angeleri, R., 240
Angoff, W. H., 103
Angold, A., 1, 2, 4, 30, 31, 34, 36, 97, 98, 99, 101, 102, 103, 104, 105, 110, 111, 112, 114, 116, 117, 118, 121, 142, 143, 303, 635, 638, 644
Angrist, J. D., 144
Angstadt, M., 364

Ani, C., 170
Anisman, H., 643
Annis, N. M., 643
Ansara, D. L., 808
Ansorge, M. S., 170
Anthony, C., 239, 240
Anthony, E. J., 485
Antonishak, J., 702
Apetroaia, A., 297
Appelbaum, M. I., 999
Appelhans, B. M., 164
Appiah, A. K., 410
Appleyard, K., 349
Apter, A., 497
Arabie, P., 828
Arbuckle, J. L., 767, 774, 1003
Arend, R., 168, 359
Arendt, R., 147
Arman, A. C., 445
Armer, M., 530
Arminger, G., 822, 972
Armistead, L. P., 604, 605
Armstrong, B., 1079
Armstrong, L. M., 277
Arnett, J. J., 603
Arnocky, S., 707
Arnold, L. E., 536
Arnold, M., 266, 267
Arntz, A., 666, 699
Aroian, L. A., 930
Aronson, E., 213
Arranz, E., 358
Arriaga, X. B., 649
Arrieta, C., 707
Arruda, E. H., 951, 952
Arseneault, L., 133
Arsenio, W. F., 284, 541, 698
Artamendi, J., 358
Arterberry, M. E., 448
Asch, S. E., 565
Aseltine, R. H., Jr., 229, 785, 786
Asgary, V., 395
Ash, P., 595
Ashbaker, M., 696
Ashenden, B., 50
Asher, S. R., 247, 527, 528, 551
Ashwood, P., 165
Asparouhov, T., 773, 775, 778, 779, 782, 978, 1029
Assor, A., 391, 392, 393, 395, 401, 402, 403, 404, 405, 407, 408, 409, 410, 412, 413, 416, 418
Astington, J. W., 359
Ataca, B., 603
Atella, L. D., 272
Atkins, D. C., 1050
Atkinson, L., 242, 245, 338, 339, 340, 349, 351, 352, 353
Atladóttir, H. O., 143, 177
Attermann, J., 145
Attkisson, C., 79
Atzaba-Poria, N., 552
Atzil, S., 363, 364
Au, A., 495
Aude, J. C., 828
Auerbach, R. P., 621
Augustine., 459
Aunola, K., 393, 394, 553
Austin, A., 666, 706
Avenevoli, S., 635
Averill, J. R., 514
Aviezer, O., 353
Avinun, R., 346
Avitsur, R., 164
Avros, R., 829
Axen, I., 798
Ayaz, H., 388
Ayduk, O., 698
Ayoub, C., 277, 479, 488, 678, 679, 680
Azar, B., 782
Azem, T., 297, 746
Azis-Clauson, C., 140

Baak, K., 274
Băban, A., 289
Babinski, D., 282, 643
Babor, T. F., 1071
Bach, D. R., 302
Bachman, J. G., 915, 1034
Bachorowski, J. A., 268
Backman, L., 160
Badalamenti, A. F., 737
Badzinski, D. M., 284
Baer, J. S., 1069
Bagner, D. M., 158
Bagwell, C., 485, 551, 553
Bahadur, M. A., 615
Bahrick, L., 458
Baicker-McKee, C., 104
Bailey, J. B., 698, 702
Baillargeon, R., 453
Baines, M. J., 994, 995
Bakeman, R., 726, 729, 732
Baker, B. L., 276
Baker, E. L., 798, 807
Baker, J. H., 130
Baker, L., 98, 419
Baker, P. C., 1009
Bakermans-Kranenburg, M. J., 38, 167, 173, 231, 247, 271, 291, 295, 297, 331, 333, 334, 335, 338, 339, 340, 341, 343, 346, 347, 348, 349, 351, 352, 353, 354, 356, 360, 362, 365, 366, 367, 368
Baker-Ward, L., 278
Bakker, M., 1063
Bal, S., 398
Balbuena, L., 294
Baldwin, A. L., 393
Baldwin, C. P., 113
Baldwin, L. M., 606
Baldwin, S. A., 1050
Bale, T. L., 103, 174
Balla, D. A., 36
Ballard, M., 240
Ballén, C., 707
Balsam, K. F., 647
Baltes, P. B., 61, 62, 160, 908, 989, 995
Balthazor, M., 538
Balto, K., 615
Balwin, A., 113
Bamford, C., 284
Bandalos, D. L., 767
Bandura, A., 49, 400, 478, 504, 516, 543, 562
Banerjee, R., 286
Banfield, J. D., 822
Banjanin, S., 175
Bank, L., 622, 640, 649, 652
Banks, M. S., 448
Banks, R. B., 848
Banks, S. J., 364
Banner, N. F., 705
Banny, A. M., 547
Baraldi, A. N., 770, 979
Barbas, H., 689
Barber, B. K., 232, 233, 391, 405, 589, 590, 617
Barber, C., 1050
Barbieri, M. A., 159
Barcikowski, R. S., 935
Bardenstein, K. K., 484
Bargh, J. A., 268, 292
Bar-Haim, Y., 38, 172, 241
Barker, D. J., 116, 159, 174, 176
Barker, E. D., 131, 132, 160, 537, 547, 548, 551, 698, 699
Barker, T. V., 231
Barkley, R. A., 59, 60, 643
Barlow, A., 28
Barnard-Brak, L., 924
Barnes, J., 131
Barnett, D., 275, 406, 599, 679, 1006, 1023, 1030
Barnett, L., 369
Barnett, N. P., 1069
Barnett, W. S., 180, 184
Barnum, K. R., 737
Barofsky, M. Y., 1043
Baron, E., 502
Baron, R. M., 132, 139, 780, 922, 923, 929, 949
Baron-Cohen, S., 284, 457
Barone, L., 359
Barone, S., Jr., 158
Baroni, E., 275
Barr, C. S., 167, 173
Barr, G., 450
Barr, R. G., 274
Barrera, M., 1048
Barrett, E. P., 650
Barrett, J., 157
Barrett, K. C., 265, 267
Barrett, L. F., 500
Bárrig, J. P. S., 294
Barrios, A., 274
Barrios, L., 641
Barry, C., 565
Barry, C. T., 667, 698
Barry, R. A., 167, 343
Barsalou, L. W, 445
Barsyte-Lovejoy, D., 173
Barthel, D., 283
Barthel, M., 356
Bartholomew, K., 345, 362
Bartko, J. J., 12
Barton, M. L., 833
Barton-Arwood, S., 687
Bascoe, S. M., 208, 211, 216, 217, 218, 223, 230, 237, 238, 239, 241, 251
Bass, J. D., 418
Basseches, M., 689
Bassett, H. H., 284, 293
Bateman, A., 369
Bates, B., 346
Bates, D. M., 940, 996
Bates, E., 454, 461
Bates, J. E., 117, 219, 276, 290, 294, 295, 351, 535, 536, 543, 545, 547, 698
Bates, M. E., 822, 917, 918, 1056
Bateson, G., 582, 583
Bath, K. E., 857
Bathula, D., 839
Batson, C. D., 223
Battle, J., 494
Bauer, D. J., 641, 799, 804, 805, 806, 809, 818, 822, 847, 849, 938, 939, 953, 968, 969, 978, 1029, 1046, 1047, 1049, 1053, 1055, 1065, 1081

Bauer, J. J., 511, 512
Bauer, P. J., 278, 280, 301, 455
Bauermeister, J. A., 646
Baum, A., 548
Baumann, A., 622
Baumeister, R. F., 221, 411, 492, 494, 495, 499, 549
Baumert, J. E., 761
Baumgarten-Katz, I., 361
Baumgartner, H., 1046
Baumgartner, S. E., 545
Baumrind, D., 396
Baune, B. T., 165
Bautista, L. E., 145
Bavelier, D., 676
Bax, L., 1079
Bayer, J. K., 414
Bayer, P., 557
Bayley, N., 50
Bazinet, A., 294
Beach, S. R. H., 593, 596, 597, 621
Beale, E. M. L., 767
Bean, R. A., 590
Beard, J. L., 170, 667
Beardsall, L., 277
Bearman, S., 643
Beaton, A. E., 785
Beatty, M., 353
Beauchaine, T. P., 220, 294, 667
Beaver, K. M., 134, 173, 555
Bebbington, P., 97, 502
Bechara, A., 388
Bechtel, W., 168
Beck, A. T., 69, 417, 494, 495, 537, 1026
Beck, J. E., 282, 293, 294, 359, 613
Beck, J. G., 636, 637
Beck, K., 233
Beck, N. C., 1069
Becker, A. E., 498
Becker, B., 247, 248, 987, 1013
Becker-Blease, K., 600, 604
Beckmann, D., 230
Beebe, B., 168, 291, 295
Beeghly, M., 284, 288, 291, 475, 678, 680, 746
Beekman, C., 862
Beeney, J. E., 367
Beer, J. S., 458
Beery, S. H., 637
Begg, C. B., 1054
Begley, C. G., 1043, 1051
Beglinger, L. J., 786
Behar, E., 268
Beidel, D. C., 232, 636
Beijers, R., 172
Beijersbergen, M. D., 335, 346, 347, 348, 349
Beiswenger, K. L., 395
Beitchman, J. H., 55
Beldavs, Z.G., 622
Belden, A. C., 1, 2, 3, 4, 36
Bell, A. M., 245, 246
Bell, B., 360
Bell, C., 595
Bell, D., 218, 219
Bell, K., 168
Bell, R. Q., 61, 131, 789, 990, 993, 1003
Bell, S. M., 330, 336
Bell-Dolan, D. J., 534, 537, 542
Bellinger, D., 94, 113
Bellmore, A. D., 499, 538, 547
Bellugi, U., 284
Belsky, J., xi, 97, 113, 131, 135, 167, 173, 208, 226, 227, 231, 240, 247, 248, 271, 274, 291, 295, 296, 297, 327, 330, 331, 337, 339, 341, 345, 347, 350, 351, 352, 353, 360, 366, 399, 587, 621
Belz, A. M., 862
Bem, D. J., 636
Bem, S., 476, 544
Bemporad, J. R., 593
Benas, J. S., 298
Bendikas, E. A., 589
Benet-Martinez, V., 509
Benikos, N., 687
Benita, M., 409
Benjamin, L. S., 416, 421, 609
Benjet, C., 547
Benner, A. D., 296
Bennett, P. H., 138, 591
Bennett,C., 368
Benoit, D., 340, 349, 351, 352, 353, 833
Ben-Sasson, A., 10
Ben-Shlomo, Y., 116
Benson, H., 511
Benson, K., 101
Benson, M. J., 224
Benson, R. M., 55
Benson, T. A., 1069
Bentler, P. M., 761, 762, 767, 775, 780, 822, 941, 956, 1003
Bento, T., 746
Berardi, N., 159, 174
Berenbaum, H., 268
Berenson, K., 698
Berg, D., 494
Bergeman, C., 852
Berger, J., 563, 564
Berger, S. L., 120
Bergeron, L., 78, 110
Bergin, A. E., 799
Bergman, K., 160, 164, 175
Bergman, L. R., 541, 706, 707, 718, 724, 800, 801, 802, 803, 804, 805, 806, 808, 809, 810, 813, 822, 827, 829, 830, 837, 838, 850, 857, 866, 868, 877, 884, 895, 908, 1047
Bergman, T., 451, 455
Bergman, Y., 168
Bergner, R. M., 500
Bergstrom, R. L., 497
Berhane, K., 979
Berk, L. E., 278
Berkey, C. S., 995
Berking, M., 300
Berkson, J., 60, 100
Berlin, J. A., 1059
Berlin, L. J., 295, 349, 360
Berman, M. G., 221
Berman, N., 1052
Bernard, K., 183, 298, 338, 355, 366
Bernardinell, L., 130
Bernbach, E., 339
Berndorff, D., 517
Berndt, T. J., 551, 561, 565
Bernier, A., 339, 352, 358, 362, 368, 369, 387, 388, 396, 397, 428
Bernieri, E., 392, 402, 408
Bernstein, A., 84
Berntson, G. G., 406
Bernzweig, J., 292
Berry, J. W., 509
Bersheid, E, 719
Bertenthal, B., 457, 475, 724
Bertin, E., 274
Berzenski, S. R., 287
Berzonsky, M. D., 412
Berzuini, C., 130
Beunckens, C., 775
Bevilacqua, L., 270, 271
Beyers, W., 389, 390, 421, 428
Bianco, L., 170
Bias, L., 915
Bibace, R., 707
Bibring, E., 494
Bick, J., 540
Bidell, T. R., 471, 474, 476, 478, 479, 480, 482, 508, 666, 673, 676, 678, 679, 680, 684
Beidel, D. C., 232
Biederman, J., 59, 60, 78, 667
Bielby, R. M., 147
Bienenstock, J., 170
Bierman, K. L., 218, 223, 304, 535, 636, 689
Biesanz, J. C., 296, 494, 781
Bifulco, A., 131
Bigelow, A. E., 288, 452
Biggs, B. K., 547
Bigsby, E., 670
Bilbe, G., 167
Bilbo, S. D., 165, 177
Bilenberg, N., 28
Binns, H. J., 2
Birch, H.G., 131
Birditt, K. S., 199
Birkhoff, G., 846
Birmaher, B., 30, 31, 32, 34, 165
Bishop, B., 87
Bishop, D. S., 606
Bishop, Y. M. M., 868
Biuckians, A., 644
Björkqvist, K., 537, 544
Bjorn, J., 84
Blacher, J., 276
Black, J. E., 103, 157, 162, 273
Black, M. M., 606
Black, R. A., 1056
Blackburn, R., 667
Blackwell, K., 677
Blackwell, M., 770, 771
Blair, C., 222, 251, 698
Blair, C. B., 280, 292, 293
Blair, R. J. R., 285
Blais, J. G., 632, 650, 858
Blais, M. R., 404
Blake, D. D., 361
Blanchard, C. M., 413
Blandon, A. Y., 246, 294, 300
Blaney, S., 498
Blankson, A. N., 276
Blanton, H., 563
Blass, E., 450
Blass, T., 554
Blatt, L., 455
Blatt, S. J., 417, 494, 496, 1026
Blatz, W. E., 201, 206, 329
Blau, G. M., 701
Blehar, M. C., 201, 206, 328, 329, 332, 336, 367, 396
Bleiberg, E., 423, 484, 485, 487
Bleker, O. P., 159
Blender, J. A., 161
Bleske, A., 648
Block, J., 675, 676, 695, 799, 801, 812, 814, 815, 816, 817, 818, 837, 838, 1030
Block, J. H., 675, 676, 695, 1030
Block, N., 444
Blokland, K., 340

Bloom, P., 277, 453
Blos, P., 387, 388, 389, 396, 398
Blossfeld, H., 731
Blozis, S. A., 940, 945, 946
Blume, A.W., 1069
Blumstein, A., 638
Bluthe, R. M., 363
Blyth, D. A., 101, 479
Boardman, J. D., 565
Bober, S., 398
Boccia, M. L., 457
Bock, H. H., 827
Boden, M. T., 294
Bodner, T. E., 774, 777, 783, 784
Boer, F., 700
Boergers, J., 229
Bogart, K. R., 281
Bogat, G. A., 224, 798, 802, 804, 805, 806, 807, 809, 818, 825, 826, 831, 833, 839, 867, 868, 869, 870, 872, 887, 893, 913, 915
Bogg, T., 1022
Boggs, S. R., 751
Bohanek, J. G., 599
Bohlin, G., 351
Bohon, C., 654
Boivin, M., 532, 537, 546, 547, 551, 553, 565, 698, 699
Boker, S. M., 996
Bokhorst, C. L., 341, 342
Boksa, P., 272
Boldt, L. B., 421
Bolger, K. E., 171, 1004
Bolger, N., 935, 938, 968, 969
Bollen, K. A., 819, 851, 941, 963, 971, 972, 1006
Bolt, E. N., 296
Bolten, M., 290
Bomyea, J., 223
Bonanno, G. A., 500, 666, 752
Bond, M. H., 603
Bonem, L., 643
Bongers, I. L., 71, 72, 668
Bonica, C., 636
Bontempo, D., 856
Booij, O., 740
Boomsma, D. I., 75, 84, 134, 826
Boone, L., 417, 419
Booth, A., 232
Booth-LaForce, C., 224, 338, 348, 349, 532
Bor, W., 3
Borelli, J. L., 170, 362, 552, 556
Borenstein, M., 1046, 1048, 1054, 1061, 1073, 1079
Borge, A. H., 537, 551
Borgerhoff Mulder, M., 221
Boris, N. W., 162, 225
Borke, H., 284
Borkenau, P., 813, 848, 849, 858, 859
Bornstein, M. H., 157, 249, 602, 698
Borsboom, D., 666, 667
Borst, S., 495
Bos, H. M. W., 646
Bos, P. A., 363
Boscardin, C. K., 798, 807
Bosch, J. D., 536
Boscolo, L., 583
Bosson-Heenan, J., 2, 3, 36
Bost, K. K., 335, 341
Bostow, D. E., 702
Boszormenyi-Nagy, I., 583, 585, 589
Bot, S. M., 559, 565
Bouchard, G., 395
Boucher, C., 340, 404
Boucher, H., 510
Bouchey, H. A., 486, 508, 647
Boulton, M. J., 545, 547
Boundy, C. A. P., 108
Boutwell, B. B., 134
Bowden, C., 84
Bowen, G. L., 584, 604
Bowen, M., 582
Bower, J., 539
Bowes, L., 547
Bowker, A. H., 891
Bowker, J. C., 530, 531, 532, 533
Bowlby, J., 160, 163, 168, 169, 178, 201, 205, 206, 224, 274, 326, 327, 328, 330, 332, 333, 346, 349, 350, 369, 396, 397, 589
Bowles, R. P., 990, 991, 1055
Boyce, W. T., 173, 231, 239, 247, 271, 291, 295, 297, 366, 722
Boyd-Franklin, N., 590, 606
Boykin, A. S. M., 535
Boylan, K., 667
Boyle, M., 667
Boyle, C. L., 251
Boyle, M. H., 607, 621
Boynton, G. M., 445
Boysen, G. A., 705
Braafladt, N., 293
Bracha, Z., 28
Brackett, M. A., 269
Bradburn, M. J., 1059
Bradbury, T. N., 635
Bradley, M., 268
Bradley, R. H., 232
Bradley, S. J., 388
Bradshaw, C. P., 542, 1034
Braet, C., 398
Braithwaite, V. A., 614
Brak, V., 924
Brakel, A. L., 666, 699
Brand, N. H., 1069, 1070, 1073
Brandt, D., 67, 959
Brand, S. R., 666
Brandtstädter, J., 918
Branje, S. J., 235, 726, 746
Branje, S. T., 552, 554
Brannick, M. T., 1054
Braungart, J. M., 291, 295
Braungart-Rieker, J. M., 291, 295
Braunwald, K. G., 296
Brazelton, T. B., 291, 452, 455
Brechwald, W. A., 542, 559, 564
Breen, W. E., 637, 652
Breinlinger, K., 453
Brekelmans, M., 746
Brendgen, M., 133, 222, 223, 541, 543, 544, 546, 547, 552, 554, 555, 564, 565, 566, 698, 699
Brendle, J. R., 637
Brener, N. D., 641
Brennan, K. A., 345
Brennan, P. A., 164, 532, 651
Brennan, R. L., 891
Brenner, J., 530
Brenning, K., 398, 408, 410
Brent, D. A., 30, 31
Brent, H. P., 158
Breslow, N. E., 111, 112, 1057
Bresnahan, M., 138
Bresnick, S., 486, 508
Bretherton, I., 168, 201, 277, 284, 326, 332, 333, 334, 346, 349, 358, 385, 397, 475, 679
Breton, J. J., 49, 78
Breuer, J., 98, 464, 677, 678
Brewer, M. B., 213
Briand-Malenfant, R., 487
Bricker, D., 19, 20, 21
Bridgemohan, C., 19
Bridges, L., 348
Bridges, L. J., 292, 395, 397, 401, 409
Bridges, M., 988
Bridgett, D. J., 6
Briere, J., 488
Briggs-Gowan, M. J., 2, 3, 4, 6, 7, 8, 10, 12, 14, 15, 17, 18, 19, 20, 21, 24, 25, 28, 29, 34, 35, 36, 37, 38, 285, 667
Brighi, A., 503
Briskman, J. A., 169, 181, 182
Britner, P. A., 169, 173, 327
Britt, H. R., 643
Brittain, H. L., 546
Brockman, C., 298
Brockmeyer, S., 362
Broderick, P. C., 300
Brodersen, L., 355
Brody, G. H., 6, 113, 296, 604, 605, 609, 621, 1011, 1012
Broeren, S., 29, 666
Broesch, T., 457
Broidy, L. M., 109
Bromet, E. J., 131, 892
Brondino, M. J., 118
Bronfenbrenner, U., 171, 718, 809
Bronfman, E., 291, 340
Brook, A., 443
Brook, J. S., 420, 422
Brooks, J., 330
Brooks, N. H., 590
Brooks-Gunn, J., 240, 475, 605
Brooks-Russell, A., 649
Brophy-Herb, H. E., 276
Brose, A., 707, 847
Brown, G., 8
Brown, A., 286
Brown, A. B., 408
Brown, A. L., 675
Brown, A. S., 118, 121, 157, 170, 177
Brown, B. B., 557
Brown, C. A., 620
Brown, C. H., 767, 1046, 1047, 1067
Brown, D., 858
Brown, D.W., 166
Brown, E., 199
Brown, G. L., 337
Brown, G. W., 112, 131
Brown, J., 277
Brown, J. D., 645
Brown, K. W., 397
Brown, L., 495
Brown, L. D., 1050
Brown, M. B., 767
Brown, N. L., 541
Brown, R. A., 115
Brown, R. L., 761
Brown, R. M., 223
Brown, R. W., 167
Brown, S. L., 223
Brown, T., 278, 924
Browne, J. V., 283
Browne, M. W., 780, 940, 945, 946, 995, 996, 1000, 1002, 1003
Brownell, C. A., 277
Bruce, A. E., 239
Bruce, J., 178, 183, 286

Bruch, H., 386, 418, 419
Bruer, J. T., 158, 183
Brugman, C., 704
Brumariu, L. E., 169
Brumback, B., 130, 131, 141, 976, 977
Brumbaugh, C. C., 327
Bruner, J. S., 451, 456, 459, 688
Brunet, A., 176, 177
Brunstein, P., 406
Brusseau, R. R., 442
Brussoni, M. J., 345
Bruyn, E. H., 646
Bryant, F. B., 296
Bryk, A., 849, 850
Bryk, A. S., 67, 935, 939, 940, 959, 961, 962, 963, 996, 1000
Buchanan, C. M., 229, 615
Buchanan, N. R., 530
Buchel, C., 363
Buchheim, A., 363
Buchs, C., 401
Buckhalt, J. A., 203, 208, 211, 213, 216, 220, 234
Buckholdt, K. E., 297
Buckley, K., 1069
Buckley, M. E., 269
Buddin, B., 476, 477, 673
Buehler, C., 210, 216, 224, 228, 233, 239, 240, 589, 615
Buffardi, L. E., 487
Bufferd, S. J., 34
Buhrmester, D., 527, 553
Buist, K. L., 615
Buitelaar, J. K., 172, 176, 272
Buka, S. L., 10, 634
Bukowski, W. M., 235, 485, 533, 534, 537, 541, 551, 553, 554, 565
Bullard, L., 563
Bullmore, E., 289
Bullock, B. M., 563, 666, 673, 689, 725, 726, 736, 737, 746
Bulteel, K., 302
Buman, M. P., 645
Bumpass, L., 199
Bunge, S. A., 458
Bunting, B. P., 785
Burchinal, M. R., 180, 296, 999
Bureau, J.-F., 168, 589
Burge, D., 643
Burgess, K. B., 356, 531, 532, 537, 695
Burghy, C. A., 286
Burk, W. J., 554, 555, 561, 562, 564, 565
Burkhouse, K. L., 297
Burks, V., 537
Burlingame, G. M., 737
Burman, B., 227, 620
Burmeister, M., 120, 344
Burnette, M. L., 420, 598
Burns, A., 555
Burns, B. J., 110, 118
Burrow-Sánchez, J., 654
Burstein, L., 935
Burt, N. M., 6
Burt, S. A., 227, 345, 687, 699, 805
Burton, C., 538
Burton, L. M., 604
Burton, P. R., 145
Burwell, R. A., 289
Busch-Rossnagel, N., 803
Bush, K. R., 246
Bush, N. F., 589, 618, 620
Bushman, B. J., 484, 487, 699
Buskirk, A. A., 235, 528
Busnel, M., 271
Buss, C., 164, 176
Buss, D. M., 221
Buss, K. A., 281, 292, 295, 301, 302, 354, 355, 862
Butera, F., 401
Butler, E. A., 291
Butner, J., 268, 645
Butterfield, P., 284
Butterworth, M. R., 589
Butz, M. R., 737
Butzin-Dozier, Z., 183
Buzy, W. W., 641
Byrne, D. G., 554, 745
Byrne, M. L., 294
Byrne, M. W., 362
Bywater, T., 181

Cacioppo, J. T., 103, 406, 976, 977
Caetano, R., 287
Cahan, S., 146
Cai, G., 54
Cai, J., 489
Cai, L., 780
Cai, T., 565
Cai, T. T., 1050, 1055
Cairns, B. D., 108, 116
Cairns, E., 232
Cairns, R. B., 103, 108, 116, 330, 666, 800
Calder, A. J., 283
Caldera, Y. M., 223, 225, 235, 237
Calhoun, C. D., 549, 554
Calkins, S. D., 290, 292, 294, 295, 300, 341, 409, 531, 721, 738
Callaghan, T., 453, 457, 461
Calussi, P., 502
Calverley, R., 678
Cambron, M. J., 491
Cameron, J., 400, 401
Cameron, J. L., 158
Cameron, L., 144
Camodeca, M., 503
Campbell, C. G., 699, 706, 847, 866, 877, 884, 895
Campbell, D., 101
Campbell, D. T., 130, 131, 132, 140, 146, 1017, 1043, 1054
Campbell, F. A., 180
Campbell, G., 500, 501
Campbell, J. M., 4, 17
Campbell, L. E. G., 225, 226
Campbell, R., 283, 296
Campbell, S., 649
Campbell, S. B., 208, 248, 347, 447, 580, 600, 718, 725, 752
Campbell, W., 98
Campbell, W. K., 487
Campione-Barr, N., 387
Campos, J. J., 265, 267, 268, 284, 289, 461, 500, 512
Camras, L. A., 268, 274, 284, 289, 500, 512
Canadian Broadcasting Corporation, 677
Candel, M., 565
Candel, M. J. J. M., 961
Candelori, C., 333
Canino, G., 98, 635
Canning, R. D., 541
Cannon, M., 6
Cannon, T., xi
Cantor, N., 58
Cantwell, D. P., 98
Capaldi, D. M., 541, 562,638, 639, 640, 641, 642, 644, 645, 646, 648, 649, 650, 651, 652, 654, 728, 1034
Caplan, M. Z., 530
Capone, C., 1069, 1070, 1073
Capps, L., 275, 463
Carapetis, J. R., 8
Carbone, M. A., 167
Carchon, I., 456
Card, N. A., 544, 699, 707, 866
Cardaciotto, L-A., 752
Cardemil, E. V., 287
Cardinal, G., 417
Carey, K. B., 1052, 1067
Carey, M. P., 634, 1052, 1067
Carey, S., 453
Carlo, G., 798
Carlson, K. S., 484, 590
Carlson, B. M., 441
Carlson, C. I., 5, 34
Carlson, C. L., 925
Carlson, E., 178
Carlson, E. A., 241, 348, 350, 351, 354, 359, 360, 388, 424, 428
Carlson, G. A., 34
Carlson, M. J., 604, 605, 606
Carlson, V., 296
Carlson, W., 556
Carlson-Sabelli, L., 726
Carmen Romano, M., 729
Carmody, D. P., 286
Carnes, M., 166
Carnochan, P., 284
Carnochan, P. G., 673, 679
Caroll, J. D., 828
Caron, C., 59, 60
Carrere, S., 726, 735
Carroll, K. A., 174
Carroll, R. J., 785
Carter, A. S., 2, 3, 6, 7, 8, 10, 12, 14, 15, 16, 17, 18, 19, 20, 21, 24, 25, 28, 29, 34, 35, 36, 285
Carter, B., 591, 601
Carter, W. H., Jr., 667, 767
Cartwright, K. L., 298
Caruso, D., 513
Carvajal, F., 275
Carver, B., 408
Carver, C. S., 516
Carver, K., 631, 636, 671
Cary, M., 973
Casas, J. P., 145
Casat, C., 78
Cascio, W. F., 401
Case, R., 474, 475, 476, 478, 480, 482, 675, 722
Caserta, M. T., 165, 166
Casey, B. J., 24, 38, 293
Cash, T. F., 643
Caspar, F., 751
Caspi, A., 74, 97, 101, 103, 113, 115, 117, 120, 130, 134, 135, 144, 226, 227, 344, 420, 555, 636, 640, 648, 798, 1005
Cassano, M., 284, 293
Cassibba, R., 335
Cassidy, J., 49, 201, 218, 291, 295, 331, 332, 333, 334, 335, 340, 341, 347, 349, 350, 351, 352, 357, 358, 359, 360, 363, 397
Cassirer, E., 814
Castellanos, X., 686, 687
Castiglioni, M., 487
Castro-Schilo, L., 995, 998
Catalano, R. F., 641

Cattell, R. B., 815, 832, 854, 858, 989
Cauce, A. M., 811
Cauffman, E., 558, 564, 642
Caughy, M. O. B., 296
Cavanagh, M., 294
Cavendish, W. M., 494
Cecchin, G., 583
Cecchini, M., 275
Cedar, H., 168
Cella, D., 6, 7, 37, 667
Center for Human Resource Research, 991, 992
Cepeda, N., 677
Cerasoli, C. P., 404
Cerezo, M. A., 746
Cervone, D., 838
Ceschi, G., 281
Chaffin, M., 162, 367
Chakrabarti, S., 84
Chakraborty, E., 846
Challis, J. R., 174
Chalmers, I., 1042
Chalmers, T. C., 1051, 1054
Chamberlain, L. L., 737
Chamberlain, P., 622, 638, 725, 752
Chambers, C. T., 280
Chambers, R. A., 388
Chambers, S. P., 130
Chambers, W. J., 53, 98
Champagne, F. A., 168, 672
Champoux, M., 103
Chan, A., 551, 552
Chan, E., 19
Chance, S., 242
Chandler, A. L., 224, 588
Chandler, M. J., 507
Chandra, A., 632
Chao, R. K., 602
Chaplin, T. M., 282, 350
Chapman, M., 461
Chard-Wiershem, D., 140
Charman, T., 358
Charnov, E. L., 350
Chartier, K. G., 698
Chase-Lansdale, P. L., 6, 240, 347, 605
Chasiotis, A., 117
Chassin, L., 645, 1009, 1048, 1053, 1055
Chaudhary, N., 810
Chazan, R., 351
Cheah, C. S. L., 219, 546, 552, 556, 602
Cheavens, J. S., 516
Chein, J., 558
Chen, B., 386, 390, 428
Chen, E., 166, 539, 540, 806
Chen, F. S., 453
Chen, G., 302
Chen, H., 420, 422, 532, 645, 1079
Chen, L., 696
Chen, L.-S., 54
Chen, Q., 689, 922, 929
Chen, S. H., 833
Chen, T., 767
Chen, X., 532
Chen, Y.-W., 78
Cheng, H., 28, 487
Cheon, S. H., 426
Cheong, J., 958, 964, 973
Cheong, Y. F., 963
Chess, S., 106, 107, 131, 158
Cheung, C. S. S., 393
Cheung, M., 761
Cheung, M.W. L., 761, 1079
Chew, S. H., 344
Chi, L., 235
Chiang, K. H., 335
Chicz-Demet, A., 177
Childs, R., 785
Chiodo, L. M., 147
Chirkov, V., 390, 391, 399, 402, 405, 427, 428
Chisholm, G., 274
Chisholm, J. S., 205
Chisholm, K., 178
Chiu, C., 489, 509
Chiu, W. T., 644
Chmielewski, M., 79
Chodorow, N., 163
Choi, I., 510, 511
Choi, S. W., 29
Choi-Kain, L. W., 368
Chorney, D. B., 636
Chou, C. P., 979
Choukas-Bradley, S. C., 542, 567
Chow, J., 170
Chow, S. M., 268
Christakis, N. A., 555, 559
Christensen, A., 588, 616
Christie, C., 563
Christopher, J., 537
Chrousos, G. P., 220, 242
Chrysikou, E. G., 169
Chua, S. N., 396
Chugani, D. C., 179
Chugani, H. T., 179
Chung, Y. B., 647
Chung-Hall, J., 649
Church, R. B., 676
Churchill, S. L., 6
Ciampi, A., 176, 177
Ciarrochi, J., 288
Cibelli, C. D., 351, 354
Cicchetti, D., xi, 12, 13, 15, 36, 38, 47, 99, 102, 105, 130, 161, 163, 164, 168, 169, 171, 200, 202, 204, 205, 206, 207, 208, 209, 210, 211, 213, 214, 215, 216, 217, 219, 220, 221, 223, 224, 225, 226, 227, 230, 231, 232, 233, 234, 240, 241, 243, 246, 247, 248, 249, 250, 251, 265, 268, 269, 271, 275, 276, 286, 294, 296, 297, 298, 300, 325, 332, 349, 351, 366, 368, 386, 387, 390, 406, 420, 424, 426, 488, 495, 529, 536, 538, 547, 580, 597, 598, 599, 601, 604, 613, 621, 622, 665, 666, 673, 678, 679, 680, 717, 718, 719, 721, 724, 738, 751, 752, 804, 808, 847, 862, 917, 923, 924, 986, 987, 988, 989, 1003, 1006, 1011, 1012, 1013, 1023, 1025, 1029, 1030, 1031, 1037, 1047
Ciesla, J. A., 242
Cillessen, A. H. N., 499, 534, 535, 538, 542, 543, 552, 554,559, 562, 635
Cimini, M. D., 1069
Cisler, J. M., 294
Claes, L., 417
Claggett, B., 1061, 1062
Clair, S., 745
Clancy, M., 6
Clara, I., 417
Clark, A. G., 538
Clark, D. M., 223
Clark, J., 362
Clark, K. E., 698
Clark, L. A., 513, 858
Clark, M. S., 96
Clark, R., 5, 6, 7, 36
Clark, S., 640, 650
Clark, S. E., 358
Clark, S. L., 296
Clark-Carter, D., 358
Clarke, A. D. B., 160, 183
Clarke, A. M., 160, 183
Clarke, C., 6, 35, 531
Clarke, D., 596, 597
Clarke, M. J., 1048
Clasen, L. S., 271
Cleary, S. D., 1008
Cleland, C. M., 650, 673, 677, 678
Clements, M., 616
Clifton, R. K., 453
Cloitre, M., 361, 367
Clore, G. L., 632
Coakley, R. M., 923
Coan, J. A., 291, 364, 553, 642, 732, 735
Coates, B., 330
Coates, E. E., 35
Coates, J. K., 84
Coats, E. J., 280
Coatsworth, 251
Coatsworth, J. D., 632
Cobb, R., 633, 634
Cobbs, G., 472
Cochran, S. D., 646
Cochran, W. G., 130, 1060
Cochrane Collaboration, 1043, 1048, 1052
Codani, J. J., 828
Coe, C. L., 165, 166, 170, 174, 176
Coffield, A., 608
Coffman, D. L., 975, 977
Cohan, C. L., 635
Cohen, D. J., xi, 76, 130, 219, 265, 325, 717, 718, 719, 751, 752
Cohen, G. L., 560, 562, 564, 565
Cohen, J., 105, 115, 296, 545, 638, 829, 891, 926, 1048
Cohen, L. H., 292
Cohen, M., 295
Cohen, N., 146, 165
Cohen, P., 116, 420, 422, 926
Cohen, R., 678, 679
Cohen, S., 164, 166, 540, 553
Cohen-Woods, S., 165
Cohn, J. F., 208, 286, 291, 296, 347, 359
Cohn, M. A., 512, 513
Coid, J.W., 138
Coie, J. D., 247, 350, 421, 422, 534, 535, 537, 541, 543, 645
Coifman, K., 666, 752
Colapinto, J., 598
Colby, S. M., 1069
Cole, D. A., 934, 954, 955, 973
Cole, H. E., 169, 296
Cole, J., 283
Cole, P. M., 268, 273, 274, 276, 277, 278, 279, 280, 281, 282, 288, 289, 292, 293, 298, 300, 301, 302, 303, 304, 350, 673, 677
Cole, S. R., 977
Cole, S. W., 166, 529, 538, 539, 540, 550
Coleman, C. C., 553
Coleman, P., 748, 810
Coley, R. L., 6
Colle, L., 358
Collier, H., 642, 732
Collins, F. S., 846, 1049, 1080
Collins, L. M., 767, 773, 776, 782, 978
Collins, N. L., 222, 223, 237, 345
Collins, P. Y., 116
Collins, R., 1057

Collins, W. A., 274, 350, 351, 354, 359, 360, 632, 647, 719, 738
Collishaw, S., 85
Colpin, H. 393
Colten, M. E., 229
Colwell, M. J., 223, 235, 237
Combs, D. J. Y., 500, 501
Comer, J. S., 34, 297
Compas, B. E., 6, 232, 285, 293
Compian, L., 633, 634, 642
Compton, S.N., 101, 116, 117
Compton, S. N., 142
Congdon, R. T., 963
Conger, K. J., 232, 940, 946
Conger, R. D., 113, 232, 234, 236, 237, 241, 608, 1017
Connell, A. M., 292, 297, 564, 746
Connell, J. P., 338, 387, 395, 403, 404, 409, 421
Conners, C. K., 25, 28, 54, 55, 65, 82
Connolly, J., 632, 635, 636, 639, 643, 647, 650
Connor, D. F., 698
Connor-Smith, J. K., 293
Conrad, J., 541
Conroy, M. A., 691
Consolacion, T. B., 646
Conway, C. C., 532, 562, 565
Cook, C. 687
Cook, C. A., 3
Cook, C. R., 223
Cook, E. T., 304
Cook, T., 101
Cook, T. D., 131, 132, 140, 146, 149, 1017, 1043, 1054
Cookell, S. J., 498
Cookus, B. A., 110
Cooley, C. H., 459, 477, 482, 490, 491
Cooley, M. R., 232
Cooper, C., 164
Cooper, H., 402, 1043, 1055, 1067
Cooper, J. E., 614
Cooper, P. J., 297, 340
Cooper, R. S., 101
Coopersmith, S., 1003
Copeland, A. P., 608
Copeland, M., 293
Copeland, N. L., 233, 234
Copeland, W. E., 114, 115, 143, 539, 644
Copenhaver, M. D., 876
Coplan, J. D., 174
Coplan, R. J., 530, 531, 532, 533
Coppola, G., 335
Corbin, T., 23
Corbin, W., 1069
Corboz-Warnery, A., 600
Cordaro, A., 596, 597
Cork, L. C., 158A
Corley, R., 167
Corneal, S. E., 810, 826, 848, 849, 857, 858
Cornelison, A., 581
Cornelius, S. W., 61
Cornfield, J., 132
Cornwell, L. W., 930
Correia, I., 503
Cortes, R. C., 304
Cortese, S., 687, 688
Cortina, K. S., 276, 279
Corwyn, R. F., 232
Cosmides, L., 242
Costa, F. M., 640
Costa, P. T., 798
Costa, R., 291
Costantini, A., 335
Costello, A. J., 53, 59, 78, 98
Costello, E. J., 98, 99, 100, 101, 103, 110, 112, 114, 116, 117, 118, 121, 142, 143, 635, 638, 644
Costigan, K. A., 175, 272
Couchman, C. E., 390, 398
Couchoud, E. A., 530
Coupland, C. A., 1067
Cowan, C. P., 35, 37, 250, 251, 581, 586, 587, 589, 598, 601, 607, 610, 620
Cowan, N., 706
Cowan, P. A., 35, 37, 250, 251, 581, 586, 587, 589, 598, 601, 607, 610, 620
Cowart, M., 654
Cowen, E. L., 527
Cox, B. J., 417
Cox, D. I., 494
Cox, J. E., 645
Cox, M. J., 167, 213, 217, 224, 225, 227, 349, 580, 587, 589, 590, 601, 620, 696, 719, 729, 730, 734
Cox, S. J., 297
Coy, K. C., 292
Coyle, K. K., 634
Coyne, J. C., 229, 552, 556
CPPRG, 181
Craig, K. D., 280
Craig, T. J., 892
Craig, W. M., 247, 546, 632, 639, 650, 748
Craighead, W. E., 286
Cramer, A. O. J., 622, 666, 667
Crandell, L., 297
Crandell, W., 165
Crane, D. R., 590
Crapanzano, A., 699
Crawford, T. N., 345
Cremeens, P. R., 223
Crescioni, A.W., 499
Crespi, E. J., 170
Creswell, C., 297
Creswell, J. D., 397
Cribbie, R., 632
Crick, N. R., xi, 223, 535, 536, 537, 544, 547
Criss, M. M., 613
Crittenden, P. M., 243, 295, 304, 589, 678, 679, 680
Crnic, K. A., 292, 587
Crockenberg, S. B., 291, 341
Crockenberg, S. C., 203, 204, 206, 213, 230, 249, 294, 302
Crockett, E. E., 295
Crockett, L., 35, 118
Crockett, M. J., 280
Croft, C., 341, 358
Croiset, G., 404
Cromer, C. C., 527
Cronbach, L. J., 12, 815
Cronce, J. M., 1052, 1067
Crone, E. A., 99, 285, 287, 288, 558, 559
Crosby, L., 638, 639, 640, 642, 643, 645, 648
Cross, S. E., 488
Cross, W., 118
Croughan, J., 53
Crowder, C., 162
Crowe, P. A., 632
Crowell, J., 169, 296
Crowell, J. A., 347, 349, 350, 362
Crowell, S. E., 296
Crowley, D. J., 176
Csikszentmihalyi, M., 400, 404, 506, 512
Cudeck, R. A., 940, 947, 979, 994, 998, 1000, 1003
Cue, K. L., 369
Cui, M., 236, 237, 241
Cuijpers, P., 535
Cullen, K. J., 108
Culver, C., 274
Cumberland, A., 281, 285, 297
Cumming, G., 1051, 1081
Cumming, S. P., 402
Cummings, A. J., 283
Cummings, E. M., 164, 199, 200, 201, 202, 203, 204, 206, 207, 208, 209, 210, 211, 212, 213, 214, 215, 216, 217, 218, 219, 220, 222, 223, 224, 225, 226, 227, 228, 229, 230, 232, 233, 234, 235, 236, 237, 238, 239, 240, 241, 242, 243, 247, 248, 249, 250, 251, 284, 287, 332, 580, 587, 590, 599, 600, 609, 612, 613, 614, 615, 616, 718, 725, 752, 808
Cummings, J. D., 108
Cummings, J. L., 281
Cummings, J. S., 203, 204, 210, 213, 216, 230, 240, 250, 287, 616
Cummings, M. E., 447, 612
Cumsille, P. E., 760, 784, 788, 789, 1014
Cunningham, W. A., 301, 789
Curran, M., 228, 589, 617, 619
Curran, P. J., 851, 944, 958, 959, 963, 996, 1004, 1005, 1029, 1043, 1044, 1046, 1049, 1053, 1055, 1065, 1081
Curran, T., 392
Curren, R. R., 407
Currie, J., 924
Curtis, W. J., 38, 220, 221, 248, 988, 1037
Cuthbert, B. N., 38, 594
Cyders, M. A., 268
Cyr, C., 225, 340, 354, 367
Cytryn, L., 98, 110
Czyz, E., 298

Dabholkar, A. S., 166, 441
Daddis, C., 387
Dadds, M. R., 278, 301
Dagne, G. A., 280, 294, 298, 302, 732
Dahl, A., 267, 268, 285, 287, 288, 293
Dahl, R. E., 99, 157, 160, 558, 559, 700
Dai, S. Y., 441
Dakin, J., 232
Dale, R., 734
Daley, D., 181
Daley, M., 600
Daley, S. E., 635, 643, 652
Dallaire, D. H., 337, 604
Daly, D. L., 857
Damasio, A. R., 388, 445, 448, 689
Damasio, H., 388
D'Amato, F. R., 179
Damian, L. E., 289
D'Amico, E. J., 1071
Damon, W., 461, 474, 475, 477
Danchin, É., 672
Dandreaux, D. M., 699
Danner, E, W., 400
Dannlowski, U., 364
Dantzer, B., 169
Dantzer, R., 540
Danzig, A. P., 34
D'Aprano, A. L., 8
Darcy, K., 636
Dare, C., 102
Darling, N., 632, 635
Darwin, C., 265, 458
Das, P., 300

DasGupta, A., 1050
Datta, A., 345
D'Augelli, A. R., 646
Daunizeau, J., 302
Dauwalder, J. P., 611, 612, 726, 733
Davagnino, J., 2
Davey, A., 761, 778
Davey, J., 1048
Davey Smith, G., 116, 145
Davidian, M., 940, 996
Davidov, E., 956
Davidov, M., 276, 698
Davidson, A. J., 291, 304
Davidson, C. E., 351
Davidson, M. L., 775
Davidson, R. J., 267, 281, 553
Davidson, W. S., 224, 807, 868, 869, 887, 893, 913, 915
Davies, C., 199, 244
Davies, F., 362
Davies, L., 199, 615
Davies, M., 59, 60, 103, 654
Davies, P. T., 164, 199, 200, 201, 202, 203, 204, 205, 206, 207, 208, 209, 210, 211, 212, 213, 214, 215, 216, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 228, 229, 230, 231, 232, 233, 234, 235, 236, 237, 238, 239, 240, 241, 242, 243, 244, 245, 246, 247, 248, 249, 250, 251, 287, 297, 298, 447, 580, 587, 590, 597, 599, 600, 612, 613, 614, 615, 616, 620, 621, 718, 725, 752, 808
Davila, J., 632, 633, 634, 635, 636, 637, 644, 651, 652, 654
Davis, B. T., 236, 287, 294, 298, 301, 535, 616, 620
Davis, E. L., 284
Davis, E. P., 160, 176, 272, 290, 294
Davis, K. S., 641
Davis, M., 363
Davis, R., 416
Davis, S. S., 700
Davis-Kean, P., 646
Davison, M. L., 994
Davoudzadeh, P., 849, 995, 998
Daw, C. S., 734
Dawber, T. R., 96
Dawid, A. P., 130, 132, 980
Dawson, B. S., 632
Dawson, D. A., 133, 144
Dawson, G., 359, 426, 598, 721, 738
Dawson, T., 675
Day, J., 581
Day, K. L., 278, 292
Day, N. E., 111, 112
De, I., 292
Deal, L. W., 645
Dean, W., 168
Deane, F. P., 288
Deane, K. E., 329, 333
Dearing, 501
Dearing, E., 923
Dearing, K. F., 696
Deater-Deckard, K., 131, 223, 1018, 1019, 1020
DeBernardo, 499
de Beurs, E., 16
DeBoard-Lucas, R. L., 287
De Brito, S. A., 297
DeCarlo, C. A., 236, 237, 612
DeCasper, A. J., 271, 283, 443, 456
De Charms, R., 387
Deci, E. L., 6, 385, 386, 387, 388, 389, 390, 391, 392, 393, 394, 395, 398, 399, 400, 401, 402, 403, 404, 405, 406, 407, 408, 409, 410, 411, 412, 414, 415, 416, 417, 418, 420, 421, 424, 425, 426, 427, 505, 516
DeCourcey, W., 394, 395, 401
Deegan, P. E., 847
Deeks, J. J., 1059
Deering, C. G., 595
DeFife, J. A., 678, 679
Defrance, E., 494
DeFries, J. C., 103, 167, 341, 344
DeGarmo, D. S., 622, 725
Degirmencioglu, S. M., 565
Degnan, K., 531
Degnan, K. A., 292, 300
DeGroot, A. D., 149
de Haan, A. D., 394
de Haan, M., 283
de Hullu, E., 699, 700
De Jong, A., 296
de Jong, P. J., 699, 700
DeJonghe, E., 807, 868, 869, 887, 913, 915
DeKlyen, M., 3, 105, 349, 350, 351, 352, 353, 495
Deković, M., 394, 417, 615
De La Fuente, J. R., 1071
Delaney, H. D., 926
Delaney, R., 250
DeLay, D., 552
de Leeuw, J., 937
Delespaul, 666
Del Giudice, M., 219, 239, 240, 242, 243, 358, 805
DeLisi, A. V. 555
Delliquadri, E., 296, 351
DeLongis, A., 494
Delonis, M. S., 291
De Los Reyes, A., 6, 7, 37, 47, 53, 538, 544, 667
Del Vecchio, T., 275
Del Vecchio, W. F., 208
Demaray, M. K., 499, 502
DeMartini, K. S., 1052, 1067
DeMille, C. B., 511
Demirtas, H., 775
Demler, O., 644
Dempsey, A. G., 700
Dempster, A. P., 767
Demyttenaere, K., 116
De Naeghel, J., 404
Denham, S. A., 266, 269, 273, 280, 283, 284, 290, 293, 530
Denis, J. L., 184
Denissen, H., 484, 487
Denmark, F., 502
Dennett, D. C., 440, 454
Dennis, M. S., 502
Dennis, T. A., 268, 284, 289, 292, 302
Denson, T. F., 499
Dent, A. L., 402
Denton, W. H., 593, 595
Denver, R. J., 170
Department of Health Human Services, Administration for Children Families, 947
Depue, R. A., 221, 222, 223, 386
Derefinko, K., 249
Derhovanessian, E., 166
de Rivera, J., 696
Derogatis, L. R., 1032
DeRosier, M. E., 561, 648
De Rosnay, M., 358
Derryberry, D., 276, 290
DerSimonian, R., 1061
Desarbo, W. S., 1028
Descartes, R., 440
De Schipper, J. C., 298
DeShon, R. P., 918
Desmond, C., 604
De Sousa, L., 495
DeSouza, A. T., 532
De Stavola, B. L., 145
de Villers-Sidani, E., 689
DeVinney, L. C., 874, 1056
Devlin, M. J., 498
DeVoe, S. E., 387, 390, 396
de Vries, H., 565, 698
de Waal, F. B. M., 457
DeWall, C. N., 295, 499
de Weerth, C., 172, 554, 730
Dewey, J., 797
De Wolff, M. S., 168, 291, 335, 337, 338, 339, 352
De Zeeuw, P., 687
de Zegher, F., 164
Diamantopoulou, S., 50, 73
Diamond, A., 222
Diamond, L. M., 268, 589
Diaz Lazcoz, Y., 828
Dickson, J.W., 632, 634
Dickstein, S., 10, 18, 21, 23, 600
Di Domenico, S. I., 388, 428
Diego, M., 272
Dielman, T. E., 55
Diener, E., 493
Diener, M., 493
Diener, M. L., 295
Dietrich, J., 646
Dietz, S., 494, 768
Diez-Martin, J., 726
Digdon, N., 99
Dijkstra, J., 561, 565
DiLillo, D., 199
DiMarco, I., 268
Dinan, T., 170
Ding, C. S., 994
Dings, J., 785
Dion, S. C., 413
Diorio, J., 160
DiPietro, J. A., 175, 272
Dirks, M. A., 6, 7, 37, 667
Disabilities, C. o. C. W, 15
Dishion, T. J., 9, 184, 541, 542, 555, 557, 562, 563, 564, 622, 640, 648, 649, 652, 666, 722, 724, 725, 726, 727, 728, 732, 746, 748, 1032
Dittmar, H., 411, 497
Di Vito, C., 275
Dix, T., 281
Dixon, A. K., 215, 245
Dixon, J. A., 730, 768
Dixon, R. A., 718
D'Jamila, G., 503
Dobbin, M. M., 272
Dochy, F., 426
Dodge, K., 117
Dodge, K. A., 218, 219, 223, 251, 350, 534, 535, 536, 537, 543, 545, 547, 559, 698
Dodge, Y., 918
Dogan, S. J., 1017
Dohrenwend, B. P., 108
Doi, S. A. R., 1061
Dolan, C. V., 822, 826
Dolan, R. J., 302
Dolev, S., 339

Doll, R., 112
Dollard, J., 543
Dollberg, D., 292
Dombeck, J. J., 497
Domenech Rodríguez, M., 622
Dominguez, A., 234
Domitrovich, C. E., 304
Donaldson, S. I., 760
Donnellan, M. B., 227, 232, 236, 237, 345, 482, 798, 816
Donnelly, J. P., 667
D'Onofrio, B. M., 134, 135, 346
Donovan, E., 1056
Donovan, J. E., 640
Doob, L. W., 543
Doornik, J. A., 1071
Dorman, M. F., 158
Dornbusch, S. M., 229
Doron, G., 296
Doss, B. D., 643
Dossche, D., 362
Dougherty, L. R., 34
Douglas, L., 717, 721, 722, 724, 726, 734, 746, 749, 751
Doumen, S., 393, 398
Doupe, A. J., 158
Down, J., 106
Down, L., 51
Downey, G., 229, 636, 698
Downs Shattuck, T., 233
Dowsett, C., 777, 789
Doyle, A. B., 53, 78, 222, 223, 398
Dozier, M., 183, 296, 298, 335, 338, 346, 355, 356, 360, 361, 362, 363, 366, 368, 369
Dozois, D. J. A., 223
Drabick, D. A., 925
Drake, K., 360
Drake, R., 847
Dressler, W. W, 5
Driessens, C., 118
Driscoll, A. K., 646
Driver, J. L., 608
Driver, R. E., 401
Drotar, D., 593
Drouin, M., 650
Drummond, J., 277
Drury, S. S., 167, 173, 366
Dube, S. R., 166
Dubois-Comtois, K., 225
Duke, M., 599
Dukewich, T. L., 204, 206, 215
Duku, E., 546
Dulcan, M. K., 53, 55, 59, 78, 98, 110
Duman, R. S., 158
Dumaret, A. C., 136, 182, 669
Dumas, J. E., 278, 611, 612, 718, 726, 733
Dumas, T. M., 649
Dumenci, L., 47, 53, 54, 78, 83, 84, 85, 240
Dumitrescu, A., 181
Duncan, D., 271, 294
Duncan, G. J., 148, 149
Duncan, N., 390, 408
Duncan, O. D., 942
Duncan, S. C., 943
Duncan, T. E., 943
Dunfield, K. A., 301
Dunger, D. B., 164
Dunkel, C., 412
Dunkel-Schetter, C., 177, 296
Dunkley, D. M., 497
Dunn, J., 199, 236, 237, 246, 277, 283, 483, 615
Dunn, L. M., 1009
Dunphy, D., 636
Dunsmore, J. C., 269, 273, 290
Dunton, G. F., 288, 294
DuPaul, G. J., 82, 251
Durbin, C. E., 269
Duriez, B., 387, 395, 403, 404, 405, 406, 411, 412, 418, 421
Durlak, J. A., 184
Durkheim, E., 801
Du Rocher Schudlich, T. D., 204, 206, 211, 215, 216, 226, 227, 230
Durston, S., 687
du Toit, M., 947, 963
du Toit, S. H. C., 995, 996, 998, 1000
Duval, S., 1054
Duyme, M., 136, 182, 669
Duyvesteyn, M. G., 367
Dweck, C. S., 58, 453, 505
Dworkin, G., 388
Dwyer, J. H., 923, 929
Dwyer, K. M., 695
Dyck, M. J., 283
Dykas,M. J., 357, 358, 359, 363
Dziegielewski, S, .F., 643

Eakin, L., 643
Eames, C., 181
Earls, F., 7, 94
Early, M., 218, 219
Easterbrooks, A., 590
Easterbrooks, M. A., 224, 347, 351, 354, 396
Eaton, D. K., 641
Eaton, M. M., 394
Eaton, W. O., 167
Eaton, W. W., 54
Eaves, L. J., 119, 120, 131, 135, 136
Ebrahim, S., 145, 1051, 1080
Ebstein, R. P., 344
Eccles, J. S., 269, 505
Eckenrode, J., 181
Eckhardt, C. I., 654
Eddy, J. M., 563, 622, 654, 725, 728, 1032
Eddy, K. T., 364
Edelbrock, C., 10, 12, 53, 59, 78, 353
Edelbrock, C. S., 98, 109, 856
Edelman, G. M., 448
Edelsohn, G., 35, 118
Edelson, L. R., 275
Edelstein, W., 675
Edens, J. F., 699
Edmonds, D., 2
Education of the Handicapped Act, 55
Edwards, A., 223, 286
Edwards, C. P., 276
Edwards, J. R., 931
Edwards, P., 748
Edwards, S. L., 20, 28, 32, 34
Effros, R. B., 166
Efron, B., 781, 930
Egan, S. K., 546
Egeland, B. R., 160, 169, 237, , 241, 286, 295, 296, 346, 347, 348, 350, 351, 354, 359, 360, 362, 367, 368, 590, 700, 718
Egger, H. L., 1, 2, 4, 30, 31, 33, 34, 36, 303, 635
Egger, M., 1054
Eggum, N. D., 219, 293, 552
Ehrenreich-May, J., 654
Ehrensaft, M. K., 640, 650
Ehrlich, K. B. 358
Eich, C., 541
Eich, E., 1081
Ein-Dor, T., 296
Einfeld, S. L., 10
Eisen, M. 474
Eisenberg, D., 924
Eisenberg, L., 9, 103
Eisenberger, N. I., 223, 241, 277, 280, 281, 285, 292, 293, 294, 295, 297, 414, 530, 541, 549, 550, 553, 554, 698
Eisenberger, R., 400, 401
Eisenhart, C., 898
Eisenhower, A. S., 276
Ekas, N. V., 291
Elam, K. K., 137
Elbert, T., 751
Elder, G. H., 113, 116, 160, 161, 232, 565, 636
Elder, T. E., 925
Elenkov, I. J., 174
Eley, T. C., 167, 699
Elison, J., 499, 500, 501
El-Khouri, B. M., 806, 808, 827, 868
Elkind, D., 398, 410, 485
Ellingson, K. D., 18
Elliot, A. J., 407, 408, 418
Elliott, D. S., 639
Elliott, J. C., 1052
Elliott, R. K., 751
Elliott, S. N., 29, 924.
Ellis, B. J., 173, 219, 231, 239, 242, 243, 247, 271, 291, 295, 297, 366, 651, 805
Ellis, L. M., 1043, 1051
Ellis, R., 5
Ellis, S., 527
Ellis, W. E., 552, 565, 649
Ellison, N., 650
Elman, J. L., 744
Elmore-Staton, L., 203, 208, 211, 213, 216, 234
El-Sheikh, M., 165, 203, 208, 211, 212, 213, 216, 220, 226, 230, 234, 237, 240, 248, 251, 805
Eltinge, J. L., 785
Emde, R. N., 35, 224, 284, 587
Emerson, E., 10
Emery, R. E., 229, 236, 237, 555, 565
Enders, C. K., 494, 760, 761, 762, 763, 764, 766, 767, 768, 769, 770, 774, 775, 776, 777, 778, 780, 781, 782, 783, 784, 937
Endicott, J., 53
Endres, M. J., 1022
Engels, R. C. M. E., 635
Engels, R. E., 559, 564, 565, 738
Engfer, A., 587
English, K., 296
Enlow, M. B., 170
Ennett, S. T., 641, 649
Enns, M. W., 417
Ensminger, M. E., 113
Entringer, S., 164, 176
Entwisle, D. R., 147
Enzle, M. E., 401
Epel, E., 164, 539
Epkins, C. C., 700
Epskamp, S., 667
Epstein, D., 687, 849, 850, 851
Epstein, J. N., 28
Epstein, N. B., 606
Epstein, S., 474, 478, 508
Erath, S. A., 220, 235, 528, 636
Erber, S., 615
Erdelyi, M. H., 677
Erdley, C., 298
Erel, O., 227, 620

Erhardt, D., 536
Erickson, D. J., 1069, 1070, 1073
Erickson, M. F., 164, 286, 296, 351, 367, 718
Erikson, E. H., 169, 411, 591
Erikson, K. L., 450
Erkal, N., 144
Erkanli, A., 103, 112, 114, 121, 143, 638
Ernst, M., 110, 558
Eron, L., 704
Eron, L. D., 67
Ertefaie, A., 980
Escovar, L., 416
Esparza, J., 138
Esser, G., 173
Essex, M. J., 35, 168, 239
Estabrook, R., 994, 996
Estell, D. B. 704
Esteller, M., 168
Estes, W. K., 668
Estes, D., 347, 350
Ethier, N., 706
Ettekal, I., 546, 548
Etzion-Carasso, A., 339
Euser, E. M., 340, 354, 367
Evain-Brion, D., 174
Evans, C. A., 291, 700
Evans, D. E., 697
Evans, E., 340
Evans, G. W., 6, 164, 219, 296
Evans, L. M., 175
Evans, S. E., 199
Evans, W. N., 144
Everitt, B. S., 801, 828, 866
Ewell, K. K., 637
Eyberg, S., 20
Eyberg, S. M., 751
Eye, J., 798
Eyre, H. L., 501, 696
Eysenck, H. J., 815, 867

Fabes, R. A., 223, 241, 273, 277, 281, 285, 292
Fabiano, 687
Facchini, A., 729
Fadiga, L., 447
Fadil, C., 458
Fagan, J. F., 172, 606, 640, 645
Fagnani, C., 345
Fagot, B. I., 351, 353, 645
Fair, D. A., 839
Fairchild, A. J., 132, 781, 931, 932, 968
Faircloth, W. B., 250
Fales, J., 636, 639, 647, 648, 652
Falk, C. F., 780, 781
Falk, K. B., 688
Falke, S. I., 232
Fallot, R. D., 360, 363, 368, 369
Famularo, R., 677, 678
Fan, Y., 286
Fang, J. Q., 391
Fanti, K. A., 699
Faraone, S. V., 59, 60, 78
Farber, E. A., 347, 348
Farkas, G., 10, 924
Farkas, K., 147
Farkas, M. S., 392
Farmer, E.M. 704
Farmer, T. W. 704
Farr, R. H., 604
Farrell, A. D., 704
Farrell, D., 207, 215, 218, 238, 240, 247, 250
Farrie, D., 606
Farrington, D. P., 101, 105, 640, 641
Farris, A. M., 224
Farrow, S., 636, 637
Farrugia, C., 283
Farvolden, P., 245, 247
Fauth, E., 993
Favez, N., 613, 614
Fearon, R. M. P., 330
Featherman, D. L., 993
Feder, L., 839
Federenko, I. S., 272
Fedio, G., 643
Fedorowicz, A. E., 620
Feeney, B. C., 223
Feeney, J. A., 362
Feerick, M. M., 232
Feffer, M., 677
Fehr, E., 222
Fein, G. G., 530
Feinberg, M. E., 237, 250
Feingold, A., 638, 641
Feiring, C., 347, 348, 351, 639, 647, 650, 673, 677, 678
Felbab, A. J., 233
Feldbau-Kohn, S., 640
Feldman, G., 752
Feldman, R., 222, 330, 363, 364
Feldman, R. S., 280, 290, 291, 292, 295
Feldman, S. S., 541
Feldner, M. T., 294
Feldon, J., 272
Felice, M. E., 593
Felitti, V. J., 157
Fendrich, M., 333
Feng, X., 293
Fennig, S., 497, 892
Fenton, T., 678
Fenton, W. S., 95
Ferdinand, R. F., 53, 109
Ferenz-Gillies, R., 169, 296
Ferguson, C. J., 1054
Ferguson, T. J., 696
Ferguson-Smith, A. C., 672
Fergusson, D., 115
Fergusson, D. M., 131, 537, 640
Fernald, A., 283
Fernyhough, C., 339, 358
Ferrell-Smith, F., 499
Ferrer, E., 268, 706, 707, 849, 944, 945, 965, 990, 996
Ferrero, J., 633, 634
Festinger, L., 562
Fetrow, R. A., 638, 654
Fetter, M. D., 557
Feusner, C., 347, 348
Fidalgo, A. M., 826
Field, C. A., 1069
Field, T., 272
Fielder, R. L., 634
Fields, S., 601
Fienberg, S., 767
Fienberg, S. E., 868
Fiese, B. H., 607
Fifer, W. P., 271, 283, 443, 456
Figueiredo, B., 291
Figueredo, A. J., 777
Filene, J. H., 251
Finch, A. E., 87
Fincham, F. D., 213, 230, 241, 249, 286, 287, 615, 620, 633, 634
Fine, S. E., 285, 350
Finkbeiner, C., 761
Finkel, E. J., 499, 671
Finkel, D., 72
Finkelhor, D., 38, 494
Finkelstein, J. W., 820, 821, 824, 827, 879, 899
Finkelstein, M. O., 1049, 1081
Finn, P. R., 1022
Finney, C. E. A., 734
Fireman, G., 284
First, M. B., 593, 594, 596, 597, 903
Fiscella, K., 425
Fisch, R., 586, 591
Fischbacher, U., 222
Fischer, D. B., 174
Fischer, K. F., 471, 474, 475, 476, 477, 478, 479, 480, 482, 488, 508
Fischer, K. W., 284, 471, 474, 475, 476, 478, 479, 480, 482, 508, 611, 666, 669, 671, 673, 674, 675, 676, 678, 679, 680, 681, 684, 701, 707, 708
Fischer, M., 643
Fischer-Kern, M., 362
Fishbein, M., 563
Fisher, A. J., 707
Fisher, K., 457
Fisher, M., 689
Fisher, P., 53, 55, 78
Fisher, P. A., 183, 286, 287, 298, 622
Fisher, P. W., 34, 98, 110
Fisher, R., 361
Fisher, R. A., 82, 83, 1056
Fishman, C. H., 737
Fishman, H.C., 613
Fishman, M. B., 300
Fisk, N. M., 175
Fittoria, M. G., 297
Fitzgerald, E., 450
Fitzgerald, K. A., 297
Fitzmaurice, G. M., 10
Fivaz-Depeursinge, E., 35, 600, 614
Fivush, R., 278, 454, 455, 475, 506, 599
Fizke, E., 283
Flaherty, B. P., 978
Flaherty, E.G., 164
Flanagan, K. S., 636
Flanzer, J. P., 640
Flaubert, G., 460
Flavell, J. H., 285, 399, 478, 676
Fleck, S., 581
Fleeson, J., 588, 589
Flege, J. E., 158
Fleischhauer, E. A., 230, 297
Fleiss, J. L., 337
Fleming, A. S., 157, 169
Fleming, C. B., 641
Fleming, W. S., 296
Fletcher, C., 696
Fletcher, J., 924
Fletcher, K., 643
Flett, G. L., 417, 494
Flink, I. J., 28
Flisher, A. J., 116, 118
Flora, D. B., 1009
Flores, E., 233, 234, 649
Florin, I., 637
Florsheim, P., 605, 606, 609, 645, 654
Flynn, M., 238, 239, 288, 293
Foà, C. 704
Fodor, J. A., 448
Fogassi, L., 447
Fogel, A., 275, 291, 689, 696, 707, 720, 722, 724, 726, 727, 740, 803
Foley, D. L., 100, 120
Fombonne, E., 84

Fonagy, P., 288, 345, 358, 362, 368, 369
Fondacaro, K. M., 285
Foote, J., 425
Foran, H. M., 597
Forbes, E. E., 700
Forbes, L., 340
Ford, D. H., 717, 718, 722, 846, 1047
Ford, J. D., 3, 7, 34, 221, 285
Ford, M. B., 223
Ford, M. T., 404
Ford, R., 111, 119
Ford, T., 23, 502
Forehand, R., 532, 604, 605
Forgatch, M. S., 619, 622, 725, 752
Forgays, D., 230, 249
Forman, E. M., 202, 207, 217, 228, 229, 243, 249, 808
Forsyth, J. P., 294
Forsythe, P., 170
Forthofer, M. S., 633
Fortier, I., 667
Fortuna, K., 345
Fortunato, C. K., 302
Fosco, G. M., 215, 287, 587, 615, 616
Foshee, V. A., 641, 649, 654
Foster, E. M., 130, 144, 149
Foster, J. D., 487
Foster, S. L., 537, 542, 640, 642
Foti, R. J., 798
Foulkes, D., 677
Fournier, M. A., 388, 428
Fowler, J. H., 555, 559
Fowler, S., 289
Fox, N. A., 161, 162, 172, 178, 179, 182, 231, 241, 279, 281, 286, 328, 341, 354, 356, 359, 531, 699, 700, 721, 738
Frabutt, J. M., 235
Fradley, E., 339
Fraga, M. F., 133
Fraleigh, L., 7, 34, 285
Fraley, C., 774, 777
Fraley, R. C., 237, 296, 327, 331, 342, 345, 347, 348, 352
Fram, M. S., 798
Frame, C. L., 532
Frances, A., 704
Franceschi, C., 165, 166
Francis, B., 274
Francis, D. D., 160
Francis, D. J., 798, 807
Franck, K. L., 210, 216, 239
Frankel, C. B., 268, 289
Frankel, K. A., 351
Frankenhuis, W. E., 242, 453
Franks, E. A., 923
Franz, C. E., 345
Frayjo, K., 1056
Frazier, P. A., 923, 925
Fréchette, M., 105
Freckleton, R. P., 760
Frederickson, B. L., 512, 513
Fredrickson, B. L., 174
Freedman, D. X., 95
Freels, S. A., 775
Freeman, D. H., 641
Freeman, W., 720
Fremmer-Bombik, E., 333, 347, 348
French, D. C., 541, 728
French, R. d. S., 58
Frendo, J. L., 174
Fresco, D. M., 268
Freud, A., 102, 107, 108, 408, 673, 677
Freud, S., 49, 54, 98, 107, 326, 444, 445, 464, 494, 496, 512, 673, 677, 678
Freund, G. C., 540
Frewen, P. A., 223
Frick, P. J., 71, 227, 268, 638, 698, 699
Friday, J. C., 7
Friederici, A. D., 283
Friedlmeier, W., 293
Friedman, R., 509
Friedman, M., 388, 425
Friedman, N., 594
Friedrich, W. N., 367
Fries, 738
Friese, M., 499
Frigerio, A., 343, 355
Frijda, N. H., 266, 500, 671
Frijns, T., 746
Friston, K. J., 302
Fritz, G., 495
Fritz, J., 277
Fritz, M. S., 132, 923, 930, 940, 978, 979
Fritz, P. A., 641
Frodi, A., 348, 397, 401
Fromme, K., 560, 1069, 1071
Fromm-Reichmann, F., 581
Frosch, C. A., 295
Frost, R. O., 403, 417
Frueh, B. C., 304
Fruzzetti, A. E., 268
Fryer, J. H., 50
Fu, A. S., 391, 405
Fu, H., 138
Fu, Y., 221
Fuchs, S., 2, 34
Fuertes, M., 291
Fuetsch, M., 636
Fuhriman, A., 737
Fuhrman, T., 184
Fulford, D., 268, 516
Fuligni, A. J., 539, 553, 554
Fulker, D. W., 131, 167, 172
Fung, W. L. A., 138
Furman, W. D., 222, 345, 527, 551, 552, 553, 632, 633, 635, 643, 647, 648
Furth, M. S., 280
Fusar-Poli, P., 363
Futh, A., 168

Gabel, S., 84
Gable, S. L., 407
Gadomski, A., 19
Gadow, K. D., 78, 82, 83
Gaensbauer, T. J., 338
Gaffrey, M. S., 3
Gagne, J. R., 35
Gagné, M., 420
Gail, M. H., 785
Galbally, M., 223
Gale, A., 719
Galen, B., 537, 544
Galewaler, S. T., 595
Galia, S. 667
Galiani, S., 144
Gallagher, K., 5, 6, 7, 36
Gallagher, S., 464
Gallese, V., 445, 447
Galliher, R. V., 635, 646
Gallop, R., 654, 973
Gallop, R. J., 1050
Gallucci, W. T., 173
Gallup, G., 457, 458
Galván, A., 293, 558, 562
Galvao, T. F., 651
Galynker, I. I., 363
Gamble, W., 169, 296
Gamer, M., 363
Gangadharan, L., 144
Ganiban, J., 275, 406
Ganzel, B. L., 219, 220, 221, 548
Gao, H. H., 458
Garbarino, J. 501
Garber, J., 242, 279, 293, 537, 538, 593, 651, 1048
Garcia Coll, C., 35, 232, 233, 234, 531
Garcia-O'Hearn, H., 206, 207
Garcia-Perez, R. O. S. A., 297
Garcia-Preto, N., 602
Gard, D. E., 269
Gard, M. G., 269
Gardiner, J. C., 831, 833
Gardner, B. C., 746, 751
Gardner, C. O., 149
Gardner, K., 732
Gardner, L. M., 20
Gardner, M., 557, 558
Gardner, T. W., 564
Gardner, W. P., 350
Garey, L., 1052
Gariépy, J., 103, 217, 696
Garland, A. F., 78
Garmezy, N., xi, 47, 101, 718
Garner, A. S., 170
Garner, P. W., 281, 285, 292, 296
Garnier Villarreal, M., 777, 786, 789
Garrett-Peters, P. T., 281
Garstein, M. A., 231
Gart, J. J., 1057
Gartstein, M. A., 304
Garvey, A. P., , 707, 803, 805
Garvey, M. A., 165
Garwood, M. M., 291, 295
Gary, L. C., 18
Gasparrini, A., 1079
Gass, K., 246
Gasser, M., 744
Gates, K. M., 861, 862
Gatward, R., 111, 119
Gatz, M., 72
Gatzke-Kopp, L. M., 294, 302
Gau, S. S. F., 925
Gaub, M., 925
Gaudet, N., 78
Gautier, M., 51
Gauvain, M., 292
Gauvin, L., 541
Gavin, A. R., 646
Gavin, L. E., 606
Gavin, M. B., 937
Gaylord, N. K., 199, 212, 240
Gazelle, H., 532, 689
Gazzaniga, M., 445, 448
Ge, X., 113, 131, 298
Gearity, J., 78
Gedo, J. E., 163
Gee, C., 615
Geisbrecht, T., 424
Geller, J., 498, 590
Gelman, A., 771, 773
Gelman, R., 443, 453
Gemmill, A. W., 15
Gendreau, P. L., 544
Gentile, L., 296
Gentner, D., 447
Genz, A., 831

George, C. J., 296, 333, 334, 336, 359
George, M. R. W., 208, 210, 216, 218, 230, 237
Georgiades, K., 607
Georgiou, S., 699
Gerard, J. M, 224, 233, 239, 615
Gerber, A., 369
Gerber, E., 176
Gergely, G., 288, 368, 455, 456
Gergen, K., 508
Germeijs, V., 393
Gerrard, M., 563
Gershman, E. S., 530
Gershoff, E. T., 420
Gershon, J., 925
Gerstorf, D., 160, 861, 993
Gesi Blanchard, A., 9
Gest, S. D., 304, 632
Getz, S., 293
Geva, R., 290, 291
Gewirtz, J. L., 330
Ghaderi, A., 497
Ghera, M. M., 272, 531
Ghisletta, P., 989, 996
Giaconia, R. M., 115
Gibb, B. E., 297, 298, 556
Gibbons, F. X., 296, 563
Gibbs, J. T., 590
Gibson, J. J., 138, 464
Gibson, K., 789
Giedd, J. N., 165, 271, 558
Giguère, B., 621
Gil, K. M., 938, 939, 953, 968, 969
Gilbert, P., 215, 222, 223, 242, 243, 244, 245, 247, 500
Giletta, M., 552, 559, 560, 564, 567
Gilissen, R., 356
Gill, K. L., 292, 295, 647
Gillen, M. M., 643
Gillespie, D., 923
Gillespie, W. J., 1054
Gilliom, L. A., 203
Gilliom, M., 282, 293, 294, 359
Gilman, R., 499
Gilmore, J. H., 166, 172
Gilmore, R., 722
Gilreath, T. D., 761, 774, 777, 782
Giltinan, D. M., 940, 996
Gimeno, D. D., 540
Ginges, J., 501
Gingras, I., 391
Gingras, J. L., 271, 274
Gingrich, J. A., 170
Gini, M., 353
Ginsburg, S. 672
Giordano, J., 602, 667
Giordano, P. C., 632, 648
Giovannelli, J., 296, 351
Girard, A., 546, 547
Girgus, J. S., 494, 633
Girotto, J., 148
Giuliani, A., 729
Given-Wilson, Z., 486
Gjerde, P. F., 484, 586, 587
Glaser, R., 165
Glass, G., 1052
Glazerman, S., 148
Gleason, M. M., 2, 21, 23
Gleason, T. C., 761
Glickman, A. R., 636
Glover, J. A., 646
Glover, V., 159, 160, 164, 174, 175, 176, 271, 272
Gluck, J. P., 158
Gluckman, P. D., 159, 164
Glueck, E., 141
Glueck, S., 141
Glutting, J. J., 925
Glymour, M. M., 145
Glynn, L. M., 160, 176, 177, 271, 290, 294
Godoy, L., 10, 14, 18, 28
Godwin, J., 251
Goeke-Morey, M. C., 201, 202, 203, 204, 206, 208, 210, 211, 212, 213, 214, 215, 216, 219, 224, 226, 228, 230, 232, 240, 242, 613, 616
Goethals, G. R., 560
Goetz, J. L., 223
Gogin, K., 425
Gogineni, A., 923
Gogtay, N., 666
Goins, L. B., 643
Golan, O., 284
Golani, M., 829
Gold, J., 284, 698
Gold, M. S., 767, 775
Gold, P. W., 220, 242
Goldani, M. Z., 159
Goldberg, A., 632, 636, 647
Goldberg, S., 172, 206, 224, 340, 350, 351
Goldberg, W. A., 347
Golding, J., 139, 176
Goldin-Meadow, S., 676
Goldman, D., 270, 271
Goldsmith, H. H., 35, 281, 290, 292, 301, 302, 337, 341
Goldstein, A. L., 298
Goldstein, H., 849, 850, 959
Goldstone, R. L., 58
Goldwyn, R., 334, 358, 360, 368
Gollob, H. F., 934, 953, 954, 955, 956
Gollwitzer, M., 503
Golmaryami, F. N., 667, 698
Gómez, C. A., 634
Gomez, J. J., 268
Gomulak-Cavicchio, B.M., 248
Gonçalves, M. M., 746
Gonnella, C., 296
Gonzales, J. E., 687, 849
Gonzalez, J. M., 785
Gonzalez, M. G., 428
Good, B., 9
Goode, T., 707
Goodie, A. S., 487
Goodman, S. H., 666
Goodman, A., 19, 23, 116
Goodman, C. C., 605
Goodman, G. S., 169
Goodman, K. L., 297, 304
Goodman, L. A., 868, 876, 893
Goodman, R., 19, 21, 23, 85, 111, 116, 119
Goodman, S. H., 226, 227, 666
Goodman-Brown, T., 494
Goodnow, J., 555
Goodnow, J. J., 385
Goodrich, T. J., 601
Goodson, P., 761
Goodwin, P., 632
Goodyer, I., 164
Goossens, F. A., 346
Goossens, L., 395, 403, 405, 411, 412, 418, 426, 532
Gordis, E. B., 232, 246, 587, 615, 619
Gordon, I., 222
Gordon, K. H., 497
Gordon, R. A., 140
Gore, J. S., 488
Gore, S., 229
Gorham, D. R., 890
Gorman, D. R., 1070
Gorman, J. M., 177
Gorsuch, R. L., 855
Gortmaker, S., 634
Goshen, I., 165
Goshin, L., 362
Gosselin, J. T., 478
Goth-Owens, T., 667
Gotlib, I. H., 99, 226, 227, 268, 269, 294, 302, 547, 633, 634
Gotowiec, A., 351
Gottesman, I., 270
Gottfredson, M. R., 811
Gottlieb, E., 409
Gottlieb, G., 103, 120, 161, 219, 271, 441, 448, 666, 669, 672, 696, 705, 707, 718, 846
Gottman, J. M., 235, 281, 551, 587, 608, 615, 619, 642, 726, 729, 732, 735, 736, 751, 752
Gottschall, A. C., 776, 782
Gould, M. S., 108, 118
Gould, S. J., 459
Gould, T. D., 270
Gouze, K. R., 2, 296
Gove, F. L., 168, 359
Gove, P., 51
Gowen, L. K., 633, 634, 642
Gozzo, Y., 166
Grabe, M., 503
Grace, D., 808
Grady, C., 271
Gräff, J., 272
Graff-Dolezal, J., 637
Graham, A. M., 241, 286, 287, 298
Graham, D. L., 170
Graham, J. W., 760, 761, 763, 765, 767, 774, 775, 776, 777, 778, 780, 782, 784, 788, 789, 978, 1014
Graham, M. A., 541
Graham, P. J., 17
Graham, S., 247, 546, 547
Graham, T., 294
Graham-Bermann, S. A., 298
Graling, K., 284
Gramzow, R., 696
Gran, J. M., 980
Granader, Y., 284
Granger, C. W. J., 854
Granger, D. A., 117, 165, 295, 549
Granic, I., 563, 564, 611, 622, 666, 706, 707, 719, 720, 722, 724, 725, 726, 727, 728, 729, 730, 734, 738, 746, 747, 748, 749, 750, 751, 752
Granier-Deferre, C., 271
Granot, D., 218, 219
Granott, N., 688, 689
Granrud, C. E., 448
Grant, B. F., 133, 144
Grant, D., 636, 637
Grant, K. A., 164
Grant, K. E., 163
Grant, M., 1071
Grant, P. J., 165
Grantham-McGregor, S.M., 170
Grassia, M., 298
Grattan, K. E., 271, 274
Gratz, K. L., 244, 268, 297, 298
Graven, S. N., 283
Grawe, K., 737
Gray, K. M., 32

Gray, R., 145
Gray, S. A., 4, 6
Graziano, P., 249
Graziano, W. G., 281
Greco, L. A., 636
Green, C. E., 287
Green, C. S., 676
Green, J., 168, 334, 358
Green, J. A., 274
Green, J. G., 286
Green, M. F., 269
Green, P., 852
Green, R. L., 148
Green, S., 1043
Green, S. B., 997
Greenberg, J., 494
Greenberg, L. S., 305, 751
Greenberg, M. T., 105, 304, 332, 333, 349, 350, 351, 352, 353, 495, 536
Greenberg, T., 118
Greene, C. J., 304
Greene, D., 400, 560
Greene, N., 176
Greenfield, P.M., 650
Greenland, S., 130, 131, 810, 811, 973, 974, 1057
Greenley, R. N., 118
Greeno, C., 268
Greenough, W. T., 103, 157, 162, 273
Greenspan, S. I., 406, 463
Greenstein, D., 271
Greenwald, A. G., 478, 484, 489
Greenwood, R., 139
Gregson, R. A. M., 745
Greig, A., 358
Greig, M. M., 280
Grello, C. M., 632, 634
Gresham, F. M. 29, 687, 924
Grieder, D., 847
Grienenberger, J., 339, 340
Griesler, P. C., 542
Griffin, D. W., 345
Griffin, S., 475, 476
Griffith, W., 554
Griffiths, W., 108
Grilo, C. M., 497
Grimes, K., 59
Grimm, K. J., 294, 300, 651, 849, 990, 991, 993, 994, 995, 996, 998, 999, 1000, 1003, 1009, 1010, 1015, 1016, 1017, 1021, 1028, 1055
Grinshpoon, A., 361
Grizzle, J. E., 785
Groark, C., 328
Grob, G. N., 106
Groh, A. M., 168, 335, 345, 348, 349, 351, 352, 353, 360
Grolnick, W. S., 292, 348, 385, 388, 390, 391, 392, 393, 394, 395, 396, 397, 398, 399, 400, 401, 402, 404, 405, 406, 407, 409, 415, 420, 424, 425, 427
Groothuis-Oudshoorn, K., 770, 771, 772, 775
Gross, J. J., 268, 290, 409
Grossman, A. W., 988
Grossman, R. B., 275
Grossmann, K. E., 283, 295, 296, 333, 347, 348, 353, 354, 355
Grota, L. J., 165
Grotjohn, D. K., 280
Grotpeter, J. K., 537, 544
Group, C. P. P. R., 117
Group for the Advancement of Psychiatry, 55
Group for the Advancement of Psychiatry Committee on the Family (GAP), 593, 595
Grouzet, F. M. E., 385, 417, 856
Grover, R. L., 636, 639, 647, 648, 652
Grow, V. M., 425
Groza, V., 328
Gruber, S. A., 364
Gruenewald, T., 164
Grunbaum, A., 108
Grusec, J. E., 206, 224, 385
Grych, J. H., 213, 215, 224, 227, 230, 240, 241, 249, 286, 287, 587, 615, 616
Guan, G., 174
Guastello, S. J., 726, 737, 744, 751
Gudjonsson, G. H., 696
Gudmundson, J. A., 276, 295
Guerra, N. G., 223, 545
Guerra-Silveira, F., 174
Guffanti, G., 168
Guichard, A.C., 223
Guilford, J. P., 514
Gullotta, T. P., 701
Gumley, A., 361
Gunderson, B. H., 635
Gunderson, J. G., 268, 368
Gunnar, M. R., xi, 103, 157, 160, 161, 162, 164, 171, 172, 173, 178, 179, 182, 183, 219, 286, 295, 296, 341, 354, 355, 356, 363, 553, 622, 808
Gunnell, D., 116
Gunter, P. L. 691
Gunthorpe, D., 808
Güntürkün, O., 457
Guo, G., 555, 565
Guo, W., 994
Guralnick, M. J., 726
Gurland, S. T., 394, 395, 401
Guskin, K. A., 4
Guthrie, D., 178
Guthrie, I. K., 285
Gutiérrez-Peña, E., 819, 867, 868, 869
Guttman, H. A., 592
Guttmann-Steinmetz, S., 350
Gutzwiller-Helfenfinger, E., 503
Guyer, A. E., 2, 3, 36, 219, 288, 294, 301, 546
Guzé, S., 59
Gwinn, M., 121

Ha, T., 635
Haan, N., 677
Haas, B., 8
Haas, E., 563, 728
Haase, R. H., 943
Haberstick, B. C., 565
Habibi, M., 106, 107
Hadas, A., 497
Haden, C. A., 475, 506
Haenszel, W., 1056
Hafen, C. A., 552, 559, 565
Hagekull, B., 351
Hagen, K. A., 622
Haggerty, M. E., 108
Haggis, T., 707
Hahlweg, K., 250
Hahm, A., 499
Haines, T. P., 1081
Hains, S. M., 283
Haith, M., 450, 451, 453, 455
Haitovsky, Y., 761
Hajal, N. J., 273, 281, 297, 298, 301, 302
Haken, H., 720, 721, 733
Hakvoort, E, M., 646
Halberstadt, A. G., 269, 273, 280, 290
Hale, W. W., 494, 726
Hales, C. N., 159
Haley, D. W., 6
Haley, J., 582, 583, 584, 613
Halgunseth, L. C., 234
Hall, G. C. N., 638
Hall, J. A., 565
Hall, S. E., 268, 273, 279, 281, 292, 301, 302
Halligan, S. L., 164, 294
Halliwell, E., 497
Halperin, J. M., 3
Halpern, C. T., 167, 634, 666, 705
Haltigan, J. D., 237, 268, 331, 352
Haltiwanger, J., 489
Halusic, M., 392
Ham, M., 294, 298
Hamagami, F., 761, 851, 944, 945, 990, 991, 993, 994, 995, 996, 999, 1000, 1015, 1016, 1017, 1036, 1055
Hamaker, E. L., 852
Hamamura, T., 488, 510
Hamburg, M. A., 846
Hamby, S. L., 38
Hamel, M., 390
Hamilton, C. E., 288, 296, 302, 333, 346, 347, 348
Hamilton, L. C., 923
Hamilton, M. A., 1026
Hamilton, N., 565
Hamlat, E. J., 288
Hamlin, J. K., 453
Hammen, C. L., 494, 532, 635, 643, 651, 652
Hamming, R. W., 734
Hammond, M., 365
Han, S., 637, 652
Handley, E., 645
Hane, A. A., 354
Hanges, P. J., 704
Hanish, L. D., 223, 277, 292, 545
Hankin, B. L., 289, 300, 488, 556, 635
Hanley, J. R., 118
Hanna, G. L., 84
Hanrahan, M., 362
Hansen, D., 176
Hansen, N. B., 86
Hansen, W. B. 1014
Hanson, M. A., 159, 164
Harari, D., 360, 361
Hardaway, C. R., 604
Harden, K. P., 135, 555, 565, 633, 634
Hardway, C., 706
Hardy, S. A., 861
Hare, A. L., 246
Harel, O., 761, 769, 775, 777, 785, 786
Hare-Mustin, R. T., 601
Harger, J., 248
Hariri, A. R., 271
Harkness, K. L., 239
Harlan, E. T., 281
Harlow, H. F., 158, 178, 326, 327, 399
Harlow, L. L., 1008
Harman, H., 855
Harmatz, M.G., 417
Harnish, J. D., 543, 698
Harold, G. T., 137, 199, 201, 202, 203, 204, 206, 208, 210, 211, 212, 213, 214, 215, 216, 224, 226, 228, 230, 287, 613, 615, 621
Harper, B. 698
Harper, C. I., 233, 234
Harper, L. V., 131, 271

Harper, M. S., 632, 634, 635, 652
Harring, J. R., 940, 946, 979
Harrington, H., 115, 116, 226, 227
Harris, M. J. 417
Harris, P., 459
Harris, P. L., 284, 288, 289, 358
Harris, S., 645
Harris, T. O., 112, 131
Harris, T. R., 1069
Harrison, A., 294
Harrison, H., 553
Harrison, H. M., 632, 635, 636, 639, 647
Harrison, K., 498
Harrison, P. L., 36
Harrist, A.W., 532
Hart, B., 87, 277
Hart, D., 474, 475, 477
Hartelman, P., 726, 745
Harter, K., 213, 587, 620
Harter, S., xii, 284, 399, 471, 472, 473, 474, 475, 476, 477, 478, 479, 480, 481, 482, 483, 484, 485, 486, 487, 488, 489, 490, 491, 492, 493, 494, 495, 496, 497, 499, 500, 501, 502, 505, 506, 507, 508, 509, 511, 513, 517, 557, 632, 673, 1007
Hartigan, J. A., 827, 832, 833
Hartley, D. E., 1026
Hartley, H. O., 767
Hartling, L. M., 500, 501
Hartman, E., 699
Hartmann, H., 385
Hartshorne, H., 812
Hartup, W. W., 330, 535, 543, 551, 552, 567
Hartz, A., 101
Harvey, A., 269
Haselager, G. T., 534
Haslett, N., 30, 31, 32, 33, 34
Haslett, T., 745
Hasselman, F., 729, 730, 734
Hastie, T., 979
Hastings, P. D., 292, 531, 667, 695
Hata, T., 441
Hatchett, S. J., 234
Hatemi, J. A., 854
Hatzenbuehler, M. L., 282, 294, 300, 546
Hau, K.-T., 964, 967, 972
Hauf, A. M. C., 115
Haugen, P. T., 632
Hauser, S. T., 589
Havighurst, T. C., 1058
Haviland, A., 141
Haviland-Jones, J. M., 500
Havránek, T., 868, 870, 896, 905
Hawes, D. J., 301
Hawker, D. J., 545, 547
Hawkins, J. D., 621, 640
Hawkley, L. C., 976, 977
Hawley, L. L., 1026
Hawley, P., 400, 404, 421, 426, 428
Hawley, P. H., 245, 516, 786
Hay, D. F., 102, 104, 111, 112, 530
Hayden, E. P., 269
Hayden, L. C., 5
Haydon, A. A., 634
Haydon, K. C., 274, 331, 345, 347, 349, 352
Hayen, R., 3
Hayes, A. F., 922, 923, 930, 931
Hayes, A. M., 751, 752
Hayes, D. S., 530
Hayes, M. E., 148
Hayes, R. F., 931
Hayling, C. C., 687
Hayne, H., 455
Haynes, O. M., 274
Haynie, D. L., 639, 648
Haynos, A. F., 268
Hay-Smith, J., 1054
Hayward, C., 633, 634, 642
Hazen, N., 228, 340, 589, 617, 618, 619
Hazler, R. J., 546
Hazzard, A., 278
He, M., 267, 268
Healy, M., 767
Heath, R. A., 726, 736
Heaven, P. C. L., 288
Heavin, S., 654
Hebb, D. O., 442
Heberle, A. E., 7, 10
Hechtman, L., 536, 667
Heck, E. J., 737
Heckler, D. R., 700
Hedegaard, M., 176
Hedeker, D., 949, 950
Hedges, L. V., 1048, 1054, 1061, 1073, 1079
Hegley, D., 274
Heider, K., 679
Heiervang, E., 116
Heiges, K. L., 615
Heijmans, B. T., 142
Heilbron, N., 541
Heilmann, W.-R., 870
Heim, C., 164, 171, 286, 363
Heiman, R. J., 390
Heimberg, R. G., 87
Hein, T. C., 281
Heindel, S., 219, 240
Heine, S. J., 488, 510
Heinrich, C., 515
Heinrichs, M., 222
Heinrichs, N., 214
Hejdenberg, J., 696
Hekler, E. B., 268
Helbig, A. L., 304
Helfinstein, S. M., 231
Hellhammer, D. H., 173
Helm, J. L., 706, 707
Helmers, K., 677
Helms, S.W., 542
Helwig, J.R., 251
Helzer, J. E., 53, 61
Hemmings, S., 497
Hemphill, S. A., 414
Hen, R., 170
Hencke, R. W., 677, 678
Henderson, C. R., 850
Henderson, C. R., Jr., 181
Henderson, H. A., 531
Hendler, T., 363, 364
Henggeler, S. W., 118
Hennighausen, K., 169
Henrich, C. C., 516, 786
Henrich, J., 457
Henrichs, J., 10
Henriksen, T. B., 28, 176, 177
Henry, C., 175
Henry, D. B., 704
Henry, N. W., 833
Hensch, T. K., 167
Hensley, M. L., 15
Hensley, R., 537
Henson, B. S., 23
Henson, J. M., 642
Hentges, R., 208, 213, 246
Hepach, R., 284
Hepper, P. G., 441
Herba, C., 285
Herbert, J., 158, 164
Herbert, K. R., 636, 639, 647, 648, 652
Herbison, P., 1054
Herbst, J. H., 798
Herbstman, J. B., 168
Herjanic, B., 98
Herman, J. L., 423
Herman, D. B., 798
Herman, J., 667
Hernán, M. Á., 101, 130, 131, 141, 976, 977, 980
Hernandez, L., 274
Hernández-Díaz, S., 101
Hernandez-Reif, M., 272
Heron, J., 137, 176
Herrenkohl, T. I., 621, 640, 641
Herrera, C., 295
Herrick, A. L., 647
Hershenberg, R., 633
Hertenstein, M. J., 265
Herts, K. L., 300
Hertsgaard, L., 164, 172, 296
Hertzig, M. E., 275
Hervis, O., 609
Heslenfeld, D. J., 295
Hespos, S. J., 464
Hess, L. E., 705
Hesse, E., 295, 330, 331, 334, 340, 347, 348, 360, 362, 368, 680
Hetherington, E. M., 130, 604, 620, 988
Hetrick, S., 116
Hewitt, J. K., 565
Hewitt, L. E., 52
Hewitt, P. L., 417, 494
Heyman, R. E., 593, 596, 597, 607, 611, 640
Heyns, B., 147
Hickok, 447
Hicks, A. M., 268
Hicks, C. W., 642
Hidi, S., 517
Higgins, E., 538
Higgins, E. T., 478, 479, 494
Higgins, J. P. T., 1043, 1044, 1046, 1048, 1054, 1060, 1061, 1062, 1067, 1073, 1079
Higgitt, A. C., 368, 369
Hilburn-Cobb, C., 205, 245, 247
Hilgard, E. R., 677
Hill, A. B., 130
Hill, A. L., 292, 300
Hill, A. M., 1081
Hill, A. P., 392
Hill, E. M., 423
Hill, J., 284, 771
Hill, J. E., 555, 565
Hill, S. D., 457
Hillary, F. G., 861
Hillemeier, M. M., 924
Hillis, S., 852
Hill-Soderlund, A. L., 295, 354
Hilt, L. M., 546
Hinde, R. A., 587, 717, 718, 719
Hindin, M. J., 808
Hines, M., 177
Hingorani, A. D., 145
Hinnant, J. B., 220, 805
Hinshaw, S. P., 536, 718, 751, 924, 925
Hinton, T. S., 285
Hinze, A., 282
Hipwell, A., 282
Hiripi, E., 642

Hirsch, S. I., 581
Hirschfield, P., 148
Hirschman, C., 643
Hirshberg, L., 833
Hitchens, K., 228, 589, 617, 619
Hjalmarsson, R., 557
Hmel, B. A., 385
Ho, A. Y., 562
Ho, M., 647, 648
Ho, M. H. R., 621, 1026
Ho, M. W., 103
Ho, S., 364
Hoaglin, D. C., 1064
Hobsbaum, A., 178
Hobson, R. P., 297, 457
Hocking, R. R., 767
Hockman, C. H., 175
Hodges, A. E., 654
Hodges, E. E., 546, 551, 553
Hodges, J., 172
Hodges, K., 98, 110
Hodgins, H. S., 390, 408, 409
Hoek, H. W., 118
Hoeksma, J. B., 291, 740
Hoekzema, E., 687
Hofer, C., 698
Hofer, M. A., 328
Hofer, S., 856
Hofer, S. M., 760, 761, 784, 979
Hoffman, J. M., 923, 929, 930, 1024
Hoffman, L., 760, 761, 937
Hoffman, L. T., 148
Hoffman, M. L., 421, 455
Hoffner, C., 284
Hofmann, D. A., 937
Hofmann, S. G., 214, 223, 300
Hogan, J. W., 974
Hoglund, W. L., 546
Högnäs, R. S., 604
Holder, J., 362
Holfeld, B., 503
Holland, A. S., 335, 345, 362
Holland, B. S., 876
Holland, C. R., 172
Holland, P. W., 130, 868, 973, 974, 975
Hollenstein, L., 706, 707
Hollenstein, T., 611, 720, 721, 722, 724, 725, 726, 734, 738, 746, 748, 749, 750
Hollis, M., 767
Hollister, L. E., 52
Hollister, R., 148
Holm, S., 876, 877
Holman, J., 614
Holmbeck, G. N., 118, 235, 923
Holmes, A., 271
Holmes, B., 590
Holmes, B. M., 295
Holmes, C. B., 1050
Holmes, R. M., 635
Holmes-Brown, M., 283
Holodynski, M., 275, 293
Holt, A. R., 199, 212, 240
Holt, K., 392, 402
Holt, R., 530
Holt, V. L., 645
Holter, M., 847
Holton, A., 748
Holyoak, K. J., 447
Homberg, J. R., 271
Homel, R., 555
Homish, D. L., 104
Hompes, T., 168
Honaker, J., 770, 771, 775, 776
Honda, H., 143
Honeycutt, H., 705, 846
Hong, M., 699, 700
Hong, Y., 509
Honzik, M. P., 108
Hood, K. E., 103
Hoogendijk, T. G., 700
Hooper, S., 296
Hoorens, V., 458
Hooven, C., 281
Hoover, J. H., 546
Hooyman, A., 401
Hopkins, B., 274, 296
Hopkins, J., 2
Hopmeyer, A., 542
Hops, H., 236, 287, 535, 616, 620, 640, 642, 644
Hopwood, C. J., 706, 804
Horan, W. P., 269
Horm, J. L., 971
Horn, G., 166
Horn, J. L., 991, 1071
Horney, J. D., 809
Horney, K., 508
Hornung, K., 171, 286
Horowitz, H. A., 717, 718
Horowitz, L. M., 362
Horowitz, S., 36
Horvath, S., 486
Horwitz, S. M., 2, 3, 18, 19, 36, 118
Horwood, L. J., 131, 640
Hoshino, T., 822
Hostinar, C. E., 553
Hotchkiss, L., 401
Hou, Y., 510
Houbre, B., 499
Hough, R. L., 78
House, P., 560
Houts, R. M., 589, 651
Howard, W. J., 761, 776
Howe, D., 358
Howe, G. R., 112
Howe, G. W., 132
Howe, M. L., 169, 171, 666
Howe, N., 277, 615
Howell, C. T., 7, 10, 12, 47, 53, 54, 55, 65, 66, 72, 78, 100, 353
Howell, D. C., 866
Howell, K. H., 298
Howerton, C. L., 174
Howes, C., 296, 333, 347
Howes, P. W., 580, 604, 613
Hox, J. J., 939, 962
Hoza, B., 533, 536, 553
Hricak, H., 1050
Hsiao, E. Y., 170
Hsieh, C.-A., 706, 707, 800, 802, 805, 833, 837, 838, 866, 868, 877, 884, 895, 908
Hsu, H., 707, 727, 740, 803
Hsueh, J., 704
Huang, E., 176
Huang, L. N., 590
Huang, W., 1053, 1055
Huang, Z. J., 167
Hubbard, E. M., 445
Hubbard, J. A., 275, 288, 543, 696, 698
Hubel, D. H., 158
Hubley, P., 456
Huchting, K., 1069
Hudak, C., 654
Hudes, E. S., 634
Hudson, A., 281, 297
Hudson, J. A., 506
Hudson, J. I., 642
Hudson, J. L., 699
Hudziak, J. J., 75, 83
Huerta, S., 223
Huesmann, L. R., 67
Hughes, B. L., 458
Hughes, J., 689
Hughes-Scalise, A., 297, 746
Huh, D., 1073, 1078, 1079
Huizenga, H. M., 826
Huizink, A. C., 176, 272
Hulette, A. C., 673
Hulshoff Pol, H. E., 158
Humayun, S., 169
Humbad, M. N., 227
Hummer, J. F., 1069
Humphrey, L. L., 419
Humphreys, K. L., 925, 942
Humphries, S. E., 145
Hunnius, S., 746
Hunsley, J., 3
Huntington, N., 19
Huo, Y., 1055, 1068, 1071
Huppert, T. J., 281
Hurlburt, M. S., 78
Hurlbut, S. C., 1071
Hurst, J. R., 296
Huss, M., 333
Husserl, E., 464
Hussong, A. M., 552, 1005, 1043, 1044, 1046, 1048, 1049, 1053, 1055, 1065, 1081
Huston, L., 700
Hutchings, J., 181
Hutchins, B. C. ,704
Huth-Bocks, A. C., 833, 868, 869, 887
Hutsebaut, D., 404
Huttenlocher, P. R., 166, 441
Huxley, J., 672
Hyde, J. S., 297, 488
Hyde, T., 737
Hyman, C., 535, 537
Hymel, S., 532, 537

Iacobucci, D., 784
Iacono, W. G., 131, 144, 145, 146, 227, 805
Ialongo, N. S., 35, 118, 542, 1034
Ibanez, L., 164
IBM Corp., 771
Igartua, K. J., 647
Iglesias, J., 275
Ikeda, N., 1079
Ilg, U., 291
Imai, K., 973, 975
Imbens, G. W., 144
Immordino-Yang, M. H., 689
Impett, E. A., 642, 643
Inagaki, T. K., 280, 541
In-Albon, T., 751
Individuals with Disabilities Education Act, 55
Indurkhya, A., 10, 826, 827, 828, 832
Ingold-Smith, M., 453
Ingram, M., 10, 14, 28, 32, 34
Inhelder, B., 724
Insabella, G. M., 988
Insel, T. R., 24, 38, 95, 163, 174, 594
Ioannidis, J. P. A., 121, 1043, 1051, 1053
Ipsa, J. M., 234
Ipsen, J., 130
Irace, K., 604
Ireland, W. W., 106

Irons, C., 222, 223
Irons, D. E., 145, 146
Irwin, M. R., 538, 539, 540, 541
Isabella, R. A., 274, 291, 350
Isely, P. J., 707
Isen, A. M., 513
Isenhower, R. W., 730
Ismond, D. R., 55
Israel, A. C., 61, 666
Israel, S., 344
Issakidis, C., 636
Ito, A., 174
Itzhaky, L., 497
Ivanova, M. Y., 47, 53, 54, 76, 78, 79, 86, 109, 116, 666
Ivry, R., 445, 448
Iwanski, A., 293
Iyengar, S. S., 387, 390, 396
Izard, C. E. 228, 232266, 268, 269, 274, 288, 304, 350, 496, 732, 752
Izuma, K., 401

Jablensky, A., 106
Jablonka, E., 672, 707
Jacka, F. N., 170
Jackson, B., 166
Jackson, D., 1050, 1060, 1062, 1063, 1066
Jackson, D. D., 582, 588
Jackson, E. F., 589
Jackson, J., 278
Jackson, J. S., 234
Jackson, M. A., 500, 501
Jacob, K., 394, 395, 401, 422
Jacob, T., 136, 213
Jacobs, A., 885
Jacobs, M. R., 421
Jacobs, N., 698
Jacobsen, T., 333
Jacobson, J. L., 147
Jacobson, K., 453
Jacobson, K. C., 119
Jacobson, S. W., 147
Jacobsson, C., 275
Jacobvitz, D., 340, 589, 590, 617, 618, 619, 620
Jacobvitz, D. B., 228, 237
Jacoris, S., 179
Jacques, S., 281
Jaffee, S. R., 115, 116, 133, 135, 226, 227, 420
Jager, J., 646
Jahoda, M., 385
Jahromi, L. B., 293
James, L. R., 973
James, W., 439, 445, 446, 451, 471, 474, 477, 479, 483, 490, 491, 492, 503, 504, 506, 507, 508, 517, 518
Jamila, M. R., 181
Jamshidian, M., 767
Jang, H., 392, 401, 402, 405, 426
Jang, K. L., 345
Janiszewski, S., 98
Jansen, B. R. J., 740
Jansen, J., 172
Jansen, J. P., 1064, 1065
Jansen, L., 690, 691
Jansen, R. A. R. G., 1037
Janssens, J. M., 368, 369
Jardri, R., 271, 283
Jaskir, J., 351
Jedidi, K., 1028
Jellinek, M., 594
Jencks, C., 149
Jenike, M. A., 416
Jenkins, J., 206, 224, 235, 236, 237, 246
Jenkins, J. M., 607, 621
Jenkins, L., 280
Jenkins, L. N., 499, 502
Jenkins, P., 19
Jenkins, R. L., 52
Jennen-Steinmetz, C., 173
Jennings, E., 286
Jensen, A. L., 53, 55, 78
Jensen, N. M., 1058
Jensen, P. S., 244
Jensen-Campbell, L. A., 548
Jessor, R., 640
Jewell, J. D., 589
Jewell, L., 294, 298
Ji, L., 696
Jia, F., 781, 784, 786, 790
Jianduan, Z., 28
Jiang, X., 534
Jing, H., 493
Jo, B., 974
Joanisse, M. F, 223
Jobe-Shields, L., 297
Jodl, K. M., 349
Joestl, S., 362
Joffe, J. M., 175
Joffe, N. G., 485
Johann, M., 343
Johansson, B., 761
Johansson, M., 168
John, O. P., 409, 798
John, R. S., 206, 207, 224, 287, 615
John, Y. J., 689
Johnson, C. A., 280
Johnson, D. R., 232
Johnson, E., 589, 618
Johnson, J. C., 245, 246
Johnson, J. G., 420, 422
Johnson, J. S., 158
Johnson, M. C., 292, 295, 409
Johnson, M. H., 450, 456, 726, 744
Johnson, R. W., 540
Johnson, S. C., 453
Johnson, S. L., 268
Johnson, V. K., 228, 250
Johnson, W., 342
Johnston, J. R., 209, 218, 225, 238, 251, 615
Johnston, L. D., 915, 1034
Johnstone, S. J., 687
Joiner, T. R., 552
Jokela, M., 145
Jollos, V., 103
Jones, C. J., 858
Jones, D., 117
Jones, D. J., 604, 605, 606
Jones, I., 176
Jones, K., 3, 181, 800, 801, 802, 803
Jones, M. C., 54
Jones, P. B., 138
Jones, R. M., 293
Jones, S. M., 10, 14, 15, 25, 28, 296, 666, 696, 786
Joormann, J., 268, 294
Jordan, C., 288
Jöreskog, K. G., 778, 819, 853, 856, 941, 942, 956, 970, 971, 972, 1017
Jorgensen, T. D., 760, 783, 784, 786, 787
Joseph, M. A., 182
Joshi, N., 274
Jouriles, E. N., 224, 287, 620, 1069
Joussemet, M., 386, 420, 421, 426
Joy, L. A., 148
Joyner, K., 631, 633, 636
Juarez, L., 643
Judd, C. M., 923, 931, 972, 973
Juffer, F., 328, 335, 346, 347, 348, 349, 352, 356, 365, 366, 367, 368
Jung, C. G., 508
Jung, T., 70
Jurist, E. L., 368
Jurkovic, G. J., 589, 590, 603
Juster, R. P., 164, 219, 354, 355
Juvonen, J., 247, 534, 546, 558, 562

Kabbaj, M., 175
Kachadourian, L., 633, 634
Kaczynski, K. J., 234, 587, 601, 616
Kaddour, A., 800, 801, 802, 803
Kagan, J., 35, 50, 159, 341, 531, 667, 695, 699, 705, 706, 717
Kağıtçıbaşı, C., 590, 602, 603
Kahler, C. W., 115, 1071
Kahn, R. E., 268
Kahneman, D., 1063
Kaiser, A. P., 2
Kalas, R., 53, 59, 78, 98
Kalijonen, A., 568
Kalin, N. H., 281
Kalodner, C. R., 118
Kaltiala-Heino, R., 642
Kam, C. M., 767, 773, 776, 782
Kam, K., 488
Kamboukos, D., 601
Kameguchi, K., 589
Kaminski, J. W., 251
Kaminski, P. L., 497
Kammradt, G., 441
Kammrath, L. K., 458
Kamphaus, R. W., 20, 21, 25, 52
Kamphuis, J. H., 545
Kan, M. L., 250
Kanagawa, C., 488
Kanat-Maymon, M., 393
Kanaya, T., 387, 389
Kandel, D. B., 103, 140, 554, 559, 648
Kandel, E. R., 1037
Kane, R., 87
Kang, J. Y., 938
Kang, N., 698
Kang, S., 493
Kanner, L., 50, 107, 108, 463
Kant, I., 443
Kantrowitz, J., L., 368
Kaplan, A., 403, 505
Kaplan, B., 785
Kaplan, D., 766
Kaplan, G., 604
Kaplan, N., 331, 332, 333, 334, 335, 347, 348
Kaplan, R. F., 698
Kaplan, S., 221
Kaprio, J., 144
Karabekiroglu, K., 2
Karavasilis, L., 398
Karbon, M., 292
Karg, K., 120, 344
Karlamangla, A., 164
Karmiloff-Smith, A., 721
Kärnä, A., 568
Karremans, J. C., 295
Kärtner, J., 275
Kasari, C., 275
Kasen, S., 420, 422
Kashdan, T. B., 288, 517, 637, 652, 752
Kashy, D. A., 610

Kasius, M. C., 53, 109
Kaslow, F., 593, 595, 596, 597
Kaslow, N. J., 224, 494
Kasser, T., 411, 413, 421, 422
Kasser, V. G., 390, 395
Katan, M. B., 145, 146
Kataoka, S. H., 18
Katsura, H., 510
Kattar, C., 233
Katz, I., 402
Katz, J., 552
Katz, L. F., 235, 281, 294, 297, 298, 301, 305, 587, 600, 610, 613, 615, 617, 618, 619
Katz, S. J., 532, 651
Kauffman, S. A., 720
Kaufman, J. S., 30, 31, 32, 101
Kaufman, N. K., 184
Kavanagh, K. A., 184, 351, 622, 752
Kawa, S. 667
Kawabata, Y., 925
Kawaguchi, M. C., 635
Kawamura, K. Y., 417
Kaysen, D. L., 1069
Kazak, A. E., 232
Kazdin, A. E., 53, 87, 751
Keane, J., 283
Keane, S. P., 292, 294, 300, 1007
Keating, D. P., 717
Keavney, B., 145, 146
Keck, C. K., 1009
Keck, G. C., 162
Keel, P. K., 805
Keele, L., 973, 975, 979
Keeler, G., 101, 114, 116, 117, 142, 143
Keeley, K., 175
Keenan, K., 2, 3, 4, 32, 33, 34, 36, 59, 60, 78, 104, 268, 282, 296, 351, 808
Keenan-Miller, D., 651
Kehoe, P., 450
Keijsers, L., 555
Keiley, M. K., 220, 226, 537
Keilp, J. G., 495
Keiras, J., 292
Kelemen, D. A., 284, 302
Kellam, S., 35, 113, 117, 118, 304, 1034
Kelleher, I., 6
Keller, G., 293
Keller, H., 117, 275, 602
Keller, P. S., 165, 211, 216, 220, 226
Kelley, K. W., 540
Kelley, M. L., 20
Kellman, P. J., 448, 453
Kelly, B. C., 634, 1043
Kelly, G. A., 478
Kelly, K., 165, 339, 340
Kelly, R. J., 212, 230, 237, 251
Kelly, S., 296
Kelso, J. A. S., 666, 671, 702, 720, 800, 804, 806, 810
Keltner, D., 223
Kemeny, M. E., 539
Kemp, C. J., 746
Kemp, F., 702
Kemp, P. L., 55
Kempes, M., 698
Kenardy, J., 673
Kendall, P. C., 87, 268, 283, 285, 297, 304, 925
Kendell, R. E., 97
Kendler, K. S., 119, 120, 130, 133, 134, 149, 272, 344, 667
Kendrick, D., 1067
Kendrick, T., 118
Kennedy, A. E., 530
Kennedy, E., 537
Kennedy, S., 199, 425
Kennedy, S. J., 20, 28, 32, 34
Kennedy, T., 583
Kennell, J. H., 162
Kenny, D. A., 132, 139, 610, 780, 852, 922, 923, 929, 938, 949, 955, 968, 969, 972, 973
Kenny, E. D., 199, 212, 240
Kenward, M. G., 767, 1079
Kephart, B., 541
Kerig, P. K., 228, 251, 582, 584, 586, 587, 588, 589, 590, 593, 598, 599, 600, 603, 604, 607, 609, 610, 614, 615, 616, 617, 619, 620
Kerker, B. D., 118
Kern, L., 687
Kernberg, O. F., 482, 484
Kernis, M. H., 403
Kerns, K. A., 334
Kerr, D. C. R., 638, 641
Kerr, M. E., 552, 554, 555, 559, 565, 582
Kerr, M. H., 233
Kervella, C., 456
Kessen, W., 454
Kessler, M. D., 98
Kessler, R. C., 116, 163, 173, 633, 635, 642, 644, 645
Kestenbaum, R., 341
Keyes, C., 512
Kharlamov, N. A., 707
Khashan, A. S., 176
Kholodkov, T., 642
Khoo, S. T., 958, 959, 961, 964, 973, 1009
Khoury, M. J., 121
Kiang, L., 345, 491, 497, 501, 539
Kidwell, S. L., 359
Kiecolt-Glaser, J. K., 1017
Kieffer, B. L., 179
Kiel, E. J., 297
Kieras, J. E., 281
Kiesler, D. J., 799, 816
Kiesner, J., 537
Kilbourne, J., 491
Kilgore, K., 748
Killeen, L. A., 268
Kilmer, J. R., 1069
Kim, A., 402, 405
Kim, C., 1017
Kim, D., 272, 803
Kim, E. J., 402
Kim, E. Y., 644
Kim, H. K., 638, 640, 641, 642, 644, 645, 648, 650, 651, 652, 654
Kim, J., 294, 298, 488, 536, 538, 673, 698, 798, 1003, 1006, 1018, 1019, 1020, 1029
Kim, K. H., 761, 775
Kim, M., 488
Kim, P., 164, 296
Kim, S., 276, 291, 353, 421
Kim, T. E., 223
Kim, Y., 390, 399, 427, 497
Kimball, M. M., 148
Kim-Cohen, J., 3, 103, 116
Kimonis, E. R., 699
Kim-Spoon, J., 297, 300, 1023, 1025, 1031
Kinal, M. P., 482
King, G., 770, 771, 775, 776
King, K., 404
King, M. C., 142
King, S., 176, 177
King, T. M., 19
Kingston, N., 785
Kinney, D. K., 170, 176
Kinney, S., 775
Kinnunen, A. K., 167
Kinscherff, R., 678
Kinzler, K. D., 453
Kipnis, J., 165
Kirby, D., 634
Kirp, D. L., 183, 185
Kirschbaum, C., 173
Kirsh, S. J., 218, 357, 358
Kisilevsky, B. S., 271, 283
Kistler, D. J., 286
Kistner, J., 538
Kitayama, S., 390, 488, 602
Kitzgerald, K. A., 230
Kitzman, H., 181
Kitzmann, K. M., 199, 212, 240, 588, 613
Kiuru, N., 552
Kivimäki, M., 145
Kivlahan, D. R., 1069
Kivlighan, K. T., 175
Klagsbrun, M., 333
Klara, M., 502
Klaric, S. H., 53, 59, 78, 98
Klaus, M. H., 162
Klebe, K. J., 994, 1000
Klein, A., 972
Klein, A. M., 2, 34
Klein, D. C., 500
Klein, D. J., 633
Klein, D. N., 34, 269
Klein, P. S., 292
Klein, S. B., 242
Kleinbaum, D. G., 94, 95, 96
Kleinman, A., 9
Kleinman, M., 118
Klepp, K. I., 642
Kliewer, W., 223
Klimes-Dougan, B., 284, 293, 294
Klimstra, T. A., 494
Klin, 15
Kline, J., 98, 110
Kline, R. B., 819, 941, 942
Klinger, D. A., 494
Klinnert, M. D., 284
Kloos, H., 730, 751
Klostermann, S., 297, 746
Klump, K. L., 345, 805
Knaack, A., 294
Knack, J. M., 548
Knafo, A., 276, 344, 346, 404
Knauer, V., 501
Knee, C. R., 408
Knibbe, R. A., 559, 565
Knickmeyer, R. C., 166
Knight, E., 633
Knoble, N. B., 638, 640, 641, 650, 654
Knopik, V. S., 135, 297, 341, 344
Knudsen, E. I., 157, 166
Kobak, R., 335, 351, 352, 369, 397
Kobak, R. R., 169, 296
Kobiella, A., 283
Kobor, M. S., 166
Koch, G. G., 13
Koch, P. B., 646
Kochanska, G., 167, 246, 276, 281, 291, 292, 294, 343, 350, 353, 421, 698, 700
Kochel, K. P., 546, 552
Kochenderfer, B. J., 553
Kochenderfer-Ladd, B., 547, 552
Koenig, J. I., 167

Koestner, R., 386, 390, 391, 392, 396, 399, 400, 402, 408, 420, 426
Koga, S. F., 178
Kogan, N., 6, 28
Koglin, U., 300
Kogos, J., 228, 232
Kohlberg, L., 638
Kohlhart, J., 1050
Kohman, R. A., 177
Kohut, H., 162
Koivisto, A. M., 642
Kok, R., 295
Kokinov, B., 447
Kolko, D., 84
Kolmogorov, A., 848
Konarski, R., 632, 643, 647
Konig, A., 503
Konner, M., 204
Konrath, S., 487
Koolhaas, J. M., 219
Koops, W., 536
Koot, H. M., 71, 72, 85, 535, 538, 668, 726
Kopp, C. B., 280, 291, 292
Korchmaros, J. D., 938, 968, 969
Koren-Karie, N., 339, 359
Koretz, D., 117, 304, 635
Korfmacher, J., 367, 368
Korte, S. M., 219
Korteland, C., 300
Kosfeld, M., 222
Koslowska, K., 620
Koss, K. J., 220, 287
Kosterman, R., 621, 640
Koudijs, E., 16
Kouhara, S., 510
Kouros, C. D., 203, 208, 211, 213, 216, 218, 220, 227, 234, 235
Kouyos, R., 980
Kouzarides, T., 120
Kovacs, M., 82, 110, 293, 359, 494, 495, 1003, 1032
Kowalik, Z. J., 751
Kowalski, C., 61
Kowalski, R. M., 499, 501
Kozlowska, K., 243, 304
Kraemer, H., 1051
Kraemer, H. C., 74
Kraepelin, E., 51
Kramer, P. R., 174
Kramer, S. F., 174
Kranzler, A., 654
Krapp, A., 411
Krappmann, L., 552
Krause, M. R., 922
Krauth, J., 803, 819, 830, 867, 870, 872, 876, 902
Krauthausen, I., 441
Kreager, D. A., 639
Kreft, I. G. G., 937
Kreppner, J., 328
Kreppner, J. M., 161, 162, 163, 172, 173, 174, 178, 182
Kretchmar, M. D., 228, 237
Kretschmann, H. J., 441
Kreuter, F., 776
Kreutzer, T., 160, 237, 351
Krieger, N., 116, 800, 801, 802, 803
Kring, A. M., 268, 269, 475
Kringelbach, M. L., 364
Krishnakumar, A., 233, 239, 240, 606
Kristensen, S., 28
Kroesbergen, E. H., 691
Kroger, J., 410
Krogstad, J. M., 286
Krohn, M. D., 140
Kroonenberg, P. M., 331, 337, 346
Krueger, R., 117
Krueger, R. F., 24, 38, 78, 640, 648
Kruesi, M. J., 333
Kruger, A. C., 457
Krukowski, R. A., 47, 54, 78
Krull, J. L., 929, 937, 938, 951, 952, 968, 969, 979
Kuang, L., 798
Kubzansky, L. D., 166
Küchenhoff, H., 874
Kuczaj, S. A., 284
Kuczkowski, R., 398
Kuehnle, K., 209, 218, 238, 251
Kuelzow, N., 302
Kuersten, R., 617
Kuersten-Hogan, R., 586
Kuh, D. L., 116
Kuhl, J., 388, 389
Kuhl, P. K., 158
Kuhn, T. S., 797, 798, 799, 801, 802, 812
Kumpf, K., 751
Kumsta, R., 132, 139, 328
Kunnen, S. E., 666, 703, 704, 706, 810
Kunter, M., 402
Kuntsche, E., 1047
Kuo, Z. Y., 719
Kupecky, R., 162
Kuperminc, G. P., 168, 349, 590, 603
Kupersmidt, J. B., 534, 535, 541, 645, 648
Kupfer, M. D. J., 597
Kuppens, P., 278, 294, 298, 301, 302, 852
Kupper, L. L., 94, 95, 96
Kuppuswamy, P. S., 281
Kurian, J. R., 179
Kurjak, A., 274
Kurowski, C. O., 409
Kurths, J., 729
Kusche, C. A., 304
Kusurkar, R. A., 404
Kuttler, A., 632, 647
Kvalem, I. L., 642
Kwan, V., 489
Kwang, T., 498, 643
Kwok, O., 856
Kwok, O.-M., 997

Laake, L. M., 6
Labonte, B., 168
Labouvie, E. W., 1071
LaBrie, J. W., 1069
Lac, A., 1069
Lacey, D., 500
Lacourse, E., 3, 131, 132, 699
Ladd, C. O., 174
Ladd, G. W., 532, 534, 537, 546, 547, 548, 552, 553, 555, 698, 1007
Ladouceur, C. D., 99, 297
Laforge, R., 1069, 1070, 1073
LaFreniere, P. J., 278, 718
Lagattuta, K. H., 284
Lagerspetz, K. M., 537, 544
La Greca, A. M., 235, 535, 537, 541, 553, 557, 632, 635, 636, 637, 639, 643, 644, 647, 649, 650, 654
La Guardia, J. G., 385, 388, 390, 398, 399, 407, 415, 420, 424, 427
Lahat, A., 699, 700
Lahey, B. B., 60, 134, 925
Lai, B. S., 636, 644, 650
Lai, C., 275
Laible, D. J., 358, 360, 798
Laird, N. M., 67, 146, 767, 850, 935, 996, 1000, 1061
Laird, R. D., 535
Lakatos, K., 342
Lake, M., 240
Lalande, K., 752
Lalonde, R. N., 621
Lam, L. L., 168
Lamarche, V., 552
Lamb, M. E., 347, 350, 356, 396, 397, 601, 603, 619
Lamb, M. J., 707
Lamb, T. F., 1069
Lambermon, M. W. E., 353
Lambert, L. S., 931
Lambert, M. J., 86
Lamborn, S. D., 233, 682
Lamere, T. G., 646
Lamey, A. V., 717, 722, 724, 725, 726, 734, 746, 747, 748, 749, 750, 751
Lamiell, J. T., 812, 813, 814, 838
Lamm, C., 293, 746
Lampe, C., 636, 650
Lamping, D. L., 23
Lampman, C., 184
Lamy, D., 38
Lancaster, T., 974
Lance, C. E., 856
Land, K. C., 104
Landau, S., 536, 828
Landgraff, C., 650
Landini, A., 243, 304
Landis, J. R., 13, 55
Landoll, R., 635, 636, 644, 649, 650
Landry, R., 395
Lane, K. L., 687, 688
Lane, R. D., 288
Lanfranchi, J., 499
Lang, J., 632
Lang, K. M., 760, 779, 780, 781, 783, 784
Lang, S., 341
Lang, T. J., 174
Lange, G., 210, 216, 239
Lange, T., 980
Langeheine, R., 833
Langer, E., 511
Langhinrichsen-Rohling, J., 639, 640
Langley, K., 137
Langrock, A. M., 203, 204, 206, 213, 230, 293
Langs, R. J., 737
Lansford, J. E., 219, 536, 547, 562, 666, 1012
Lanza, M. L., 745
Laplante, D. P., 176, 177
Lapouse, R., 108
Lapsley, A.-M., 349, 351, 352, 353, 354, 360
Lapsley, D. K., 485, 487
Laranjo, J., 339, 358
Larbi, A., 166
Larimer, M. E., 1052, 1067, 1069
Larkin, R. W., 502
Larochette, A. C., 280
Larose, S., 368
Larrieu, J. A., 225
Larsen, H., 235, 564, 565, 566
Larsen, J. K., 282, 300
Larsen, J. T., 284
Larsen-Rife, D., 234
Larson, B., 498
Larson, J. H., 232

Larson, M., 172
Larson, R. W., 293, 294, 298, 632
Lashley, S., 608
Lask, B., 498
Latendresse, S. J., 565
Latzman, R. D., 297
Lau, S., 495
Laub, J. H., 141, 648, 798, 803, 809, 811
Laucht, M., 173
Laudenslager, M. L., 175
Laughlin, L., 5
Laurenceau, J. P., 183, 288, 356, 366, 752, 935
Laurent, H. K., 69, 297, 641, 642, 652
Lauretti, A., 586, 617
Laurin, K., 349, 351, 352, 353
Lauritzen, S., 762
Laursen, B., 247, 533, 551, 552, 553, 559, 565, 632, 633, 866
Lauterbach, E. C., 281
Lautsch, E., 876, 902
Lavelli, M., 275, 727
Lavigne, J. V., 2, 3, 36, 296
Lavoie, F., 544, 698, 699
Lawlor, D. A., 145
Layte, R., 135
Lazarsfeld, P. F., 833
Lazarus, R. S., 266, 267, 496
Le, H. N., 268
Lê, T., 925
Leadbeater, B. J., 546, 645
Leaf, P. J., 19, 110, 118
Leahy, K. L., 224
Leak, S., 276
Leandro, G., 1079
Leary, A., 600, 610, 613, 617
Leary, M. R., 221, 411, 471, 472, 481, 487, 499, 501, 511, 530, 549
Lebailly, S. A., 2
LeBailly, S. A., 296
Le Blanc, M., 105
Lecanuet, J., 271
Lecanuet, J. P., 441, 451
Leckman, J. F., 222
Lecky, P., 508
Lecours, S., 487
Ledermann, T., 231
LeDoux, J. E., 265, 268
Lee, A., 297
Lee, C. M., 395, 1047, 1049, 1069
Lee, J. M., 634
Lee, K., 406, 721
Lee, K. H., 280
Lee, L. C., 493
Lee, N. R., 271
Lee, S., 423
Lee, S. S., 565, 925
Lee, S. Y., 761
Lee, T., 780
Lee, W., 388
Lee, Y., 645
Lee-Flynn, S., 494
Leerkes, E. M., 230, 276, 292, 294, 295, 297, 301, 302, 304, 338, 352, 359
Leese, M., 828
Leeson, P., 288
Lefkowitz, E. S., 642
Legate, N., 387
Lehmacher, W., 874, 877
Lehman, D. R., 488
Lehrer, J. A., 634
Leichtman, M. D., 489
Leidner, B., 501
Lejeune, J., 51
Lejuez, C., 340
Lekes, N., 388, 391
Lekka, S. K., 230
LeMare, L., 532
Lemay, P., 611, 612, 726, 733
Lemerise, E. A., 541
Lemieux, A., 166
Le Moal, M., 175
Lench, H. C., 284
Lengua, L. J., 294
Lenhart, A., 649, 650
Lennon, M. C., 798
Lenox, K., 535
Lens, W., 392, 400, 402, 404
Lenzenweger, M. F., 386
Lenzi, D., 363
Leon, D. A., 116
Leon, K., 225, 340
Lepage, J. P., 447
Lepper, M. R., 400
Lereya, T. S., 547
Lerner, H. G., 479, 486
Lerner, J. V., 803, 812
Lerner, R. M., 705, 717, 718, 722, 812, 846, 1047
Lesaffre, E., 767
Lesch, K. P., 271
Leslie, A. M., 453
Leslie, D. L., 118
Less, M. T. Y., 495
Less, W., 510
Lester, L. S., 895, 901, 906, 908
Leung, P.W., 28
Leuthe, E., 645
Leve, C. S., 645
Leve, L. D., 199, 226, 227, 298
Levendosky, A. A., 224, 345, 798, 805, 807, 833, 868, 869, 872, 887, 893, 913, 915
Levenson, R. W, 407
Leventhal, B. L., 4, 32, 698
Leventhal, J. M., 19
Leverich, G. S., 184
Levesque, C., 413
Levin, A. V., 158
Levin, B., 1049, 1081
Levin, D. A., 57
Levin, H., 391, 405
Levine, L. J., 284
Levine, M. P., 498
Levine, S., 183, 356, 366
Levitt, M., 458
Levy, A., 726
Levy, D., 148, 339
Levy, K. N., 367
Lewczyk, C. M., 55, 78
Lewin, K., 717, 799, 812, 814, 815, 838
Lewin, L. M., 535
Lewin-Bizan, S., 812
Lewine, R. R. J., 50
Lewinsohn, P. M., 115, 158, 633, 634, 635, 642, 644
Lewis, A. J., 223
Lewis, E., 183, 356, 366
Lewis, G., 137
Lewis, H. B., 500
Lewis, J., 1049, 1057
Lewis, M., 102, 330, 347, 348, 351, 458, 461, 475, 500, 808
Lewis, M. D., 276, 280, 286, 293, 302, 666, 671, 682, 717, 719, 720, 721, 722, 724, 725, 726, 734, 738, 746, 748, 749, 750, 751, 752
Lewis, M. H., 158, 172
Lewis, T., 608
Lewis, T. L., 158
Lewis-Morrarty, E., 366
Lewkowicz, D. J., 446
Lewthwaite, R., 401
Leyland, A. H., 131
Li, F., 541, 562, 563, 728
Li, J., 176, 671, 673, 876
Li, R., 292
Liang, K. Y., 116
Liao, P. J., 226
Liberati, A., 1052
Lichtenberg, J.W., 737
Lichtenstein, P., 699
Lichtwarck-Aschoff, A., 707, 721, 726, 729, 730, 734, 746, 751
Lickenbrock, D. M., 291, 296
Lickliter, R., 666, 672, 696, 705, 846
Licznerski, P., 158
Liddle, C., 161
Liddle, H. A., 251
Lidz, T., 581
Lieberman, A. F., 600
Lieberman, M. D., 280, 541, 550, 553, 554
Liebowitz, M., 636
Lienert, G. A., 803, 819, 830, 867, 868, 869, 870, 871, 872, 874, 887, 890, 891, 892, 896, 905, 908
Lilienfeld, A. M., 102
Lilienfeld, D. E., 102
Lilienfeld, S. O., 61, 424, 667
Lilly, M., 298
Lin, S. P., 177
Linardatos, E., 1051
Linares, O., 645
Lincoln, K., 274
Lindahl, K. M., 228, 234, 582, 587, 601, 603, 604, 605, 606, 608, 609, 613, 615, 616, 619
Linden, D. E. J., 672
Lindenberger, U., 160, 847, 989, 996
Lindhiem, O., 338
Lindhorst, T., 646
Lindhout, I. E., 700
Lindquist. M. A., 979, 980
Lindsay, L. L., 229, 230, 620
Lindsey, E. W., 223, 225, 235, 237
Linehan, M. M., 268, 294, 423
Ling, R., 649
Linting, M., 335
Linville, P. W., 479
Lionetti, F., 359
Liotti, G., 242, 245, 296, 360
Lipman, E., 607
Lippa, Y., 58
Lippitt, R., 815
Lipscomb, S. T., 298
Lipsey, M. W., 118
Lira, A., 170
Lisa, L., 298
Litovsky, R., 453
Littell, R. C., 851, 996
Little, K. C., 635
Little, M., 645
Little, R. J. A., 760, 761, 765, 766, 767, 775, 782, 784
Little, T. D., 10, 14, 15, 25, 28, 245, 516, 544, 552, 568, 699, 707, 760, 761, 765, 774, 776, 777, 780, 781, 783, 784, 785, 786, 789, 790, 866, 946, 947, 948, 970
Liu, C. H., 291
Liu, D., 1049, 1055, 1058, 1065, 1067

Liu, J., 28
Liu, R. Y., 1049, 1055, 1058, 1065, 1067
Liu, S., 851
Liu, W., 276, 509
Liu, X. F., 979
Livesley, W., 345
Lizotte, A. J., 140
Lleras, C., 65
Lo, C., 510
Lo, Y., 1029, 1030
LoBue, V., 214, 216
Lochman, J., 645
Lochman, J. E., 535, 537, 543, 698
Locke, R. L., 281
Locker, A., 339
Lockhard, G., 979
Lockwood, C. M., 781, 923, 929, 930, 978, 1024
Lodder, G. A., 552, 559
Loeb, K., 498
Loeber, R., 100, 101, 104, 105, 108, 421, 1047, 1049
Loehlin, J., 103
Loevinger, J., 385, 388, 800, 838
Loew, B., 250
Loeys, T., 977
Loftus, J., 634
Logan, C., 667
Logue, S., 558
Lohman, B. J., 641, 649
Lohr, M., 646
Loken, E., 866
Lombroso, C. T., 446
Long, A., 108
Long, J. D., 775
Longford, N. T., 761, 935
Longmore, M. A., 632, 648
Longo, M. R., 447
Lonky, E., 400
Loper, A. B., 604
Lopes, F., 614
Lopez, A. D., 116, 119
Lopez, E., 601
Lopez, N., 635, 636, 637, 647
Lopez, S. J., 474, 512, 516, 517
Lorber, M. F., 295, 302
Lorberbaum, J. P., 363
Lord, C., 35, 275
Lorenz, F. O., 241
Lorenz, K., 158, 326, 364
Lou, E., 621
Lou, H. C., 176
Lougheed, J., 726
Lourenco, S. F., 447
Lovden, M., 160
Lovejoy, D. A., 173
Loveland, K. A., 275
Low, J. A., 271
Low, S. M., 499, 501, 618, 647, 648
Lowe, P., 162
Lowell, D. I., 28
Lozano, R., 116, 119
Lozoff, B., 296
Lubach, G. R., 170, 176
Luby, J. L., 1, 2, 3, 4, 36, 268
Lucas, C. P., 53, 55, 78, 98, 109, 110
Lucas, J., 1022
Lucey, P., 35
Luchetta, T., 500
Luckner, 689
Ludlow, A., 588, 593, 598
Lüdtke, O., 402
Ludwig, J., 147, 148, 149
Luebbe, A. M., 218, 219
Luecken, L. J., 164
Luepnitz, D. A., 601
Luijk, M. P., 295, 340, 343, 355
Luke, N., 286
Lukon, J. L., 282, 293, 294, 359
Lukowski, A. F., 278
Lumey, L. H., 177
Lumley, M. N., 239
Luna, B., 281
Luna, I., 604
Lundh, L. G., 798
Lundy, B. L., 339
Lunkenheimer, E. S., 276, 279, 746, 749
Luo, L., 165, 166
Luo, Q., 565
Luo, Z., 778
Lupien, S. J., 164, 354, 355, 363
Lusk, K., 281
Lussier, P., 640
Luster, T., 8, 233, 234
Luthar, S. S., 247, 248, 987, 1013
Lutkepohl, H., 853
Luyckx, K., 410, 411, 412, 418, 494, 532
Luyten, P., 403, 417, 418
Lykken, D. T., 718
Lynam, D. R., 117
Lynch, J. G., 922, 929
Lynch, J. H., 386, 389, 390, 398, 425, 428
Lynch, K. G., 808, 973
Lynch, M. F., 232, 399, 529, 601
Lynch, S. K., 135
Lynn, S. J., 424
Lynne-Landsman, S. D., 542, 1034
Lynskey, M. T., 131, 134
Lyons-Ruth, K., 105, 169, 291, 295, 340, 351, 352, 354, 589
Lyra, M. C. D. P., 810
Lytton, H., 131
Lyubomisky, S., 268

Maas, C. D., 641
Mabry, P. L., 1081
MacArthur Working Group on Attachment, 333, 341
MacBeth, A., 361
MacBeth, T. M., 345
MacCallum, R. C., 878, 918, 980, 1017
Maccari, S., 160, 175
Maccoby, E. E., 229, 330, 391, 405, 421
MacDonald, G., 221, 530, 549
MacDonald, K., 157, 221
MacDonald, K. L., 247
Macfarlane, J.W., 108
MacFie, J., 589
Macho, M., 637
MacKay, I. R., 158
Mackenna, J., 294
MacKenzie, M. J., 207, 236
Mackey, E. R., 636, 637, 647
Mackie, J. L., 130
MacKinnon, D. P., 132, 760, 781, 782, 784, 923, 929, 930, 931, 932, 933, 934, 937, 938, 939, 954, 957, 958, 964, 965, 966, 968, 969, 970, 972, 973, 975, 978, 979, 1024, 1043
MacKinnon-Lewis, C., 235, 1007
Mackintosh, M. A., 304
Mackner, L., 165
MacMahon, B., 130
MacNeill, 247
Macomber, J., 453
Maczuga, S., 924
Maddux, J. E., 478
Madge, N., 147
Madigan, S., 340, 349, 351, 352, 353, 365
Madsen, K. M., 143
Madsen, S. D., 632
Maeda, K., 493
Maehr, M. L., 505
Maes, H. H., 133, 135, 136
Maestripieri, D., 174
Maffei, L., 159, 174
Magai, C., 752
Magdol, L., 227, 640
Mageau, G. A., 352, 386, 387, 396, 397, 420, 426
Magid, L., 650
Magidson, J., 822
Magnuson, J. S., 718, 724
Magnuson, K. A., 148, 149
Magnusson, D., 96, 97, 113, 144, 529, 639, 800, 801, 803, 804, 805, 806, 808, 809, 812, 813, 827, 857, 866, 868, 875, 877, 884, 895, 1047
Mahatmya, D., 296
Mahieu, S., 362
Mahler, M., 427
Mahoney, A., 608
Mahoney, J. L., 117
Mahoney, M. J., 751
Maier, K. S., 833
Maier, M. A., 296
Maier, U., 291
Maillardet, R., 1051
Main, M., 295, 296, 330, 331, 332, 333, 334, 335, 345, 347, 348, 357, 360, 368, 680
Mainhard, M. T., 746
Mair, P., 823, 825, 867, 868, 869, 870, 874, 877, 878, 881, 892, 895, 898, 906, 915, 918
Maiuro, R. D., 638
Malarkey, W. B., 1017
Malassine, A., 174
Malatesta, C. Z., 274
Malatesta-Magai, C., 276
Malcarne, V. L., 285
Males, M., 85
Maletic, V., 540
Malheim, J. E., 497
Malik, N. M., 228, 234, 587, 601, 603, 608, 609, 613, 615, 616, 620
Maliken, A. C., 297, 305
Malmberg, B., 993
Malone, J. C., 678, 679
Malone, P. S., 219, 536, 645
Malson, H., 498
Maly, V., 870
Mancini, J. A., 604
Mancini, T., 704
Mandara, J., 801
Mandler, J. M., 443, 453
Manera, V., 240
Mangelsdorf, S. C., 291, 295, 337, 341, 354, 355
Mangun, G., 448
Manly, J. T., 368, 599, 1006, 1023, 1029, 1030, 1031
Mann, B. J., 203
Mann, E. A., 180, 181
Manning, L. G., 208, 217, 220, 225, 226, 227, 234, 241, 248, 249, 251
Manning, W. D., 632, 648
Mansell, W., 269, 361
Mansour, S., 386

Mantel, N., 1056
Manzi, C., 589
Mao, L., 493
Mapstone, M., 185
Marakovitz, S., 16
Marceau, K., 651
Marcelli, D., 456
March, J., 82
Marcia, J. E., 410
Marcoen, A., 362
Marcovitch, S., 292
Marcynyszyn, L. A., 296
Marden, J. I., 1056
Margett, T. E., 666
Margolin, G., 206, 207, 213, 224, 232, 246, 287, 587, 588, 607, 615, 616, 619
Marian, V., 456
Marín, B. V., 234, 634
Marin, C. L., 508
Marino, S., 503
Markey, C. N., 643
Markey, P. M., 643
Markie-Dadds, C., 367
Markiewicz, D., 222, 223, 398
Markland, D., 404, 426
Markman, E. M., 58
Markman, H., 616
Markman, H. J., 250
Markon, K. E., 79
Marks, D. J., 3
Marks, I. M., 245
Marks, M. J., 327, 352
Markus, H., 563
Markus, H. R., 390, 391, 405, 474, 478, 481, 488, 602
Markus, M. H., 700
Marlatt, G. A., 1069
Marlier, L., 443, 450
Marmot, M. G., 138
Marold, D. B., 472, 494, 495, 506, 507
Marolla, F., 116, 142
Marquardt, D., 972
Marsch, S., 691
Marsh, H. W., 964, 967, 972
Marsh, P., 541
Marshal, M. P., 647
Marshal, N. M., 541
Marshall, M., 8
Marshall, I. H., 809
Marshall, P. J., 179, 231, 241, 354, 356, 531
Marshall, W. A., 112
Marsland, K., 516
Marszalhk, J. M., 1050
Martel, M. M., 294, 667
Martens, M. P., 943, 1068, 1069, 1070
Marti, C. N., 654
Martier, S. S., 147
Martin, C., 222
Martin, J., 358
Martin, J. A., 604
Martin, L. J., 158
Martin, M. J., 201, 203, 204, 206, 207, 208, 209, 210, 211, 213, 215, 216, 217, 218, 219, 220, 224, 225, 226, 230, 231, 234, 243, 244, 245, 246, 247, 248, 250, 251, 298
Martin, N. C., 537
Martin, S. E., 268, 289
Martinez, G. M., 632
Martinez, P., 232
Martinez-Torteya, C., 6, 291, 667, 798, 805, 872, 893
Martini, D. R., 35
Martins, C., 359
Martins, E. C., 359
Marumo, G., 441
Marvin, R. S., 169, 172, 173, 327, 333, 341
Marwan, N., 729
Mascolo, M. F., 611, 669, 671, 676, 679, 680, 688, 689, 696, 706, 707, 708
Mash, E. J., 3
Masheb, R. M., 497
Mashour, G. A., 442
Mask, L., 413
Maslin, C. A., 351
Maslow, A., 508
Mason, J., 274
Mason, W. A., 58, 621, 640
Masse, L. C., 638
Masse, L. N., 184
Masten, A. S., xi, 209, 217, 231, 247, 598, 632, 705, 923
Masten, C. L., 550, 554
Master, A., 58, 505
Masters, J. C., 330
Masterson, J. F., 423
Masyn, K., 1029
Matejka, M., 280
Matheson, K., 643
Mathiesen, K. S., 3
Mathieson, L. C., 537
Matias, C., 168
Matsumiya, Y., 446
Matsumoto, K., 401
Matsumoto, M., 401
Mattaini, M., 502
Matthews, A. M., 1051
Matthews, L. S., 232
Matthews, R., 1051
Matthews, S. G., 174, 175
Matthys, W., 698
Maudsley, H., 107
Maugeais, R., 271
Maughan, A., 243
Maughan, B., 85, 141, 142, 182, 799
Maurer, D., 158, 445, 446
Maxwell, K. A., 565
Maxwell, S. E., 926, 934, 954, 955, 973
May, M. A., 812
Mayer, J. D., 513
Mayer, K. U., 161
Mayer, L. S., 118
Mayes, L. C., 271
Mayeux, L., 542, 562
Maynard, R. A., 130
Mayr, E., 104
Mays, C. W., 112
Mays, S. A., 704
Mays, V. M., 646
Mayseless, O., 218, 219
Mazanov, J., 745
Mazmanian, S. K., 170
Mazumdar, M., 1054
Mazursky-Horowitz, H., 305
Mazzocco, M. M., 996, 1015, 1016, 1017
McAdams, D. P., 507, 815
McAdoo, H. P., 234
McAllister, W., 798
McArdle, J. J., 72, 761, 789, 819, 849, 850, 851, 858, 944, 945, 965, 971, 989, 990, 991, 993, 995, 996, 1003, 1015, 1016, 1017, 1022, 1036, 1055
McArthur, L., 654
McAssey, M., 706, 707
McAuliffe, M. D., 696, 698
McBride, K., 536
McBride, S. W., 170
McCadney, A., 3
McCain, A. P., 20
McCall, B. P., 147
McCall, R., 328
McCall, R. B., 161, 162, 173, 178, 182
McCandless, M. A., 551
McCarley, K., 501
McCarthy, K., 285
McCarthy, K. J., 7, 34
McCartney, K., 103
McCarton, C. M., 182
McClellan, J., 3
McClellan, J. M., 56, 142
McClelland, D. C., 399, 812
McClelland, J., L., 726, 743, 744
McClintock, M. K., 407
McClure-Tone, E. B., 294, 301
McConaughy, S. H., 7, 47, 53, 60, 65, 66, 72, 78, 100
McConnell, E. K., 761
McConnell, E. M., 552
McConnell, M. C., 617
McCord, J., 557
McCormick, M. C., 10
McCown, W. G., 737
McCoy, D. C., 296
McCoy, K., 642, 732
McCoy, K. P., 208, 210, 212, 213, 214, 215, 216, 218, 230, 235, 237, 247
McCrae, R. R., 208, 230, 493, 494, 798
McCranie, E. W., 418
McCrory, C., 135
McCrory, E. J., 286, 297
McCubbin, H. I., 598
McCubbin, M. A., 598
McCue Horwitz, S., 2
McCullagh, P., 823
McDermott, J. M., 286
McDermott Sales, J., 278
McDonald, R. P., 995
McDonald, A. L., 595
McDonald, C., 10, 14, 28
McDonald, R., 287
McDougall, P., 546
McDowell, D. J., 280, 281
McEachern, A. D., 563
McElhaney, J. E., 166
McElhaney, K. B., 246, 349, 541
McElwain, N. L., 224, 295, 338, 358, 589
McEwen, B. S., 164, 219, 285, 354, 355, 363
McFadden, S. H., 752
McFadyen-Ketchum, S. A., 545
McFarland, F. C., 541, 564
McFarland, L., 618
McFarland, M. B., 497
McFie, B. S., 108
McGeary, J., 298
McGill, B., 632
McGillicuddy DeLisi, A. V., 285
McGoldrick, M., 591, 601, 602
McGorry, P., 116
McGowan, P. O., 168
McGrath, K., 610
McGraw, K., 748
McGregor, H. A., 425
McGroder, S., 148
McGue, M., 103, 131, 136, 137, 144, 145, 146, 805
McGuffin, P., 344
McGuffog, C., 351

McGuire, S., 130
McHale, J. L., 295
McHale, J. P., 35, 237, 586, 600, 604, 610, 615, 617, 618, 620
McIlvane, J. M., 199
McIlwain, D., 486
McIntire, M. L., 284
McIntosh, D. N., 275
McKenzie, D., 138
McKenzie, L., 394, 406
McKim, V. R., 131
McKinley, M., 530
McKinney, W. T., Jr., 178
McKnew, D., 98, 110
McKnight, K. M., 777
McKnight, P. E., 777, 1069
McLachlan, G. J., 991
McLanahan, S. S., 144, 605, 606
McLaughlin, B., 9
McLaughlin, K. A., 179, 282, 286, 294, 300, 546
McLoyd, V. C., 233, 234
McMahon, E. M., 298
McMahon, R. J., 648, 698, 699
McMakin, D. L., 700
McManis, M., 706
McMenamy, J. M., 409
McNemar, Q., 891
Mead, G. H., 491
Mead, H. K., 220, 294
Meade, T. W., 145
Meagher, S., 416
Meaney, M. J., 160, 167, 168, 271, 285, 540
Measelle, J. R., 35, 37, 241, 250, 297
Medin, D. L., 58
Medina, A. M., 213
Meehl, P. E., 97, 98, 100, 718, 803, 872
Meek, S. E., 293
Meeker, W. Q., Jr., 930
Meeus, W. H. J., 235, 417, 494, 552, 554, 559, 565, 726, 746
Mehta, D., 168
Mehta, T., 509
Meier, E. A., 174
Meinlschmidt, G., 176
Meins, E., 339, 340, 358
Meiser, T., 825
Meissner, W. W., 423
Melby, J. N., 608
Melfsen, S., 637
Melhuish, E., 131
Mellenbergh, G. J., 869, 877, 900
Melton, G. B., 118
Meltzer, H., 23, 111, 119, 502
Meltzoff, A. N., 284, 446, 447, 455
Mendell, N. R., 1029, 1030
Mendelsohn, J., 15
Mendle, J., 633, 634
Menesini, E., 502, 503
Meng, X., 144
Meng, X. L., 779, 780
Mennin, D. S., 268, 282, 294, 300
Mercer, J., 162
Mercer, S. H., 561
Merchant, M., 293
Merchant, S., 18
Merckelbach, H., 424
Mercken, L., 565
Meredith, W., 851, 856, 857, 958, 959, 971, 989, 990, 991, 994, 995, 1055
Meredith, W. H., 493
Merikangas, K. R., 633, 635, 644
Merleau-Ponty, M., 464
Mermelstein, R., 635
Merrell, K. W., 25, 28, 695
Merrick, S., 169, 296, 347
Merrilees, C. E., 227, 232, 250
Mervis, C. B., 58
Merz, E. C., 173
Mesman, J., 356
Messick, D. M., 560
Messinger, D. S., 268, 275, 727
Messner, M., 286
Metalsky, G. I., 494, 537
Meunier, J. C., 621
Meunier, L. N., 281
Meusel, L. A., 746
Meyer, F., 553
Meyer, G. J., 54, 78
Meyer, I. H., 646
Meyer, J. M., 136
Meyer, U., 272
Meyer-Ortmanns, H., 734
Meyers, J., 294, 858
Meyers, R. H., 767
Mezulis, A. H., 297, 488
Mezzich, J., 58
Mian, N. D., 10, 14, 35
Michel, M. K., 268, 282
Michou, A., 392, 400
Mick, E., 667
Miclea, M., 3
Micucci, J., 603
Miettinen, O. S., 100
Miga, E. M., 169, 296
Miklikowska, M., 387
Miklowitz, D. J., xi, 621, 644
Mikulincer, M., 223, 276, 296, 362, 396, 397, 505
Milan, S., 645
Miles, C. J., 615
Milgrom, J., 15
Milich, R., 536
Mill, J., 133
Miller, A. H., 164, 540
Miller, A. M., 176
Miller, C. J., 79, 516
Miller, D. L., 147
Miller, D. N., 502
Miller, G. E., 164, 166, 539, 540, 806
Miller, J. L., 707
Miller, M. D., 935
Miller, N. E., 543
Miller, P., 778, 779
Miller, P. A., 241
Miller, P. H., 399
Miller, S., 500, 605
Miller, S. A., 399
Miller, W. E., 595
Miller, W. R., 425
Miller-Johnson, S., 180, 535, 541, 645
Milligan, K., 242, 245
Milliken, G. A., 851, 996
Millon, C., 416
Millon, T., 416
Mills, B., 296
Mills, N. T., 165
Millsap, R. E., 856
Mills-Koonce, W. R., 217, 621, 696
Milne, B. J., 115
Mims, V., 400
Min, K., 493
Mincic, M., 284
Minde, K., 362
Mindell, J., 251
Minder, C., 1054
Mineka, S., 103, 214, 216
Minelli, C., 145
Minnes, S., 147
Mintz, J., 1051
Minuchin, P., 598, 601
Minuchin, S., 228, 419, 583, 584, 589, 598, 608, 613, 615, 618, 737
Miquelon, P., 417
Mischel, F., 677
Mischel, W., 677
Mislevy, R. J., 785
Misra, T. A., 639, 640
Mistry, R. S., 296
Mitchell, E. A., 271, 274
Mitchell, J. E., 643
Mitchell, M. A., 973
Mitchell, P. M., 250
Mitchell, R. W., 457
Mitchell, S., 98, 108
Mitrani, V. B., 609
Mitteness, L. S., 857
Moe, V., 148
Moerk, K.C., 269
Moffitt, T. E., 72, 97, 101, 113, 115, 116, 117, 130, 135, 144, 226, 227, 420, 555, 640, 648, 698, 699, 718, 798, 1005
Moher, D., 1052
Mohlman, J., 268
Moilanen, K. L., 293
Moise-Titus, J., 67
Molenaar, P. C. M., 668, 706, 707, 722, 726, 736, 740, 745, 798, 801, 809, 810, 817, 819, 826, 833, 846, 847, 848, 849, 851, 854, 857, 858, 861, 862, 866, 877, 884, 895, 979
Molenberghs, G., 767, 775
Moles, A., 179
Molina, B. G., 536
Molina, W., 274
Moller, A. C., 405
Monahan, K. C., 564, 634
Mondloch, C., 446
Monea, E., 184
Moneta, G., 293
Money, K. M., 170
Monje, M. L., 177
Monk, C., 175
Monk, M. A., 108
Monroe, S. M., 239, 635
Monshouwer, H. J., 536
Monsour, A., 479, 481
Montagu, A., 451, 459
Montague, M., 494, 768
Montemayor, R., 474
Monti, J. D., 297
Monti, P. M., 1069
Montoro, R., 647
Moodie, E. E. M., 846
Moon, I. S., 426
Mooney, R., 158
Moons, K., 1079
Moor, B., 550
Moore, C., 168
Moore, D. R., 605, 606, 645
Moore, E.W. G., 760, 783, 784
Moore, G., 208, 347
Moore, G. A., 208274, 284, 287, 296, 301, 746
Moore, G. H., 898
Moore, J., 634
Moore, K. J., 622
Moore, L. A., 536

Moore, M. J., 451, 455
Moore, M. K., 446, 447, 455
Moore, S. A., 297
Moore, S. R., 633, 634
Moore, S. V., 366
Moosbrugger, H., 972
Morabia, A., 131
Moran, E. K., 268
Moran, G. S., 340, 368
Moravec, B. C., 110
Moreau, M., 287
Morelen, D., 297
Moreno, S. G., 1051
Morey, L. C., 804
Morf, C. C., 486
Morgan, C. P., 174
Morgan, G. D., 1069, 1074, 1081
Morgan, P. L., 10, 924, 980
Morgan, R., 453
Morgan, R. F., 148
Morgan, S. B., 534
Morgenstern, H., 94, 95, 96
Mori, S., 510
Morland, L. A., 304
Morris, A. S., 279, 281, 285, 288, 292, 294, 295, 301, 302, 387, 389
Morris, H., 293
Morris, J., 363
Morris, M. C., 219, 220, 221, 242
Morris, M. W., 509
Morris, P. A., 548, 809
Morris, T. L., 636
Morrison, K., 643
Morrone-Strupinsky, J. V., 222, 223
Morrow, M. T., 696, 698
Morse, R., 148
Morsella, E., 268
Mortazavi, S., 494
Mortimore, P., 141
Morton, J., 450, 456, 726, 744
Morton, S., 116
Mościcki, E., 117, 304
Moscovitch, D. A., 214
Moscovitch, M., 271
Moses, L. K., 296
Mosher, W. D., 632
Moskal, J., 221
Moskowitz, D. S., 386
Moss, E., 168, 225, 366
Moss, H. A., 50
Moss, L., 458
Mosteller, F., 146
Mostow, A. J., 350
Mott, F. L., 1009
Motyl, M., 1051
Motzoi, C., 553
Moulson, M. C., 286
Mouratidis, A., 392, 400, 402, 418, 419
Mouton-Simien, P., 20
Mowrer, O. H., 543
Moyer, V. A., 172
Moynihan, J. A., 165
Mrug, S., 536
Mueller, E., 530
Mufson, L., 654
Muhtae, L., 414
Muir, D.W., 271
Mulder, E. J., 176, 272
Mulhall, P. K., 785
Mullen, K., 644
Muller, D., 931
Muller, N., 636
Muller, U., 385
Mun, E.-Y., 819, 822, 823, 824, 825, 826, 827, 828, 832, 866, 867, 868, 869, 870, 872, 877, 881, 882, 894, 895, 898, 901, 906, 909, 915, 917, 918, 1044, 1047, 1055, 1056, 1065, 1067, 1068, 1069, 1071, 1074
Munakata, Y., 677, 744
Mundt, J. C., 895, 901, 906, 908
Munfakh, J. L., 118
Munholland, K. A., 346, 349
Muñoz, L. C., 699
Muñoz, R. F., 268
Munro, M. J., 158
Muraki, E., 1071
Muraven, M., 420
Murayama, K., 388, 401
Murdock, T. B., 534, 558
Muris, P., 29, 666, 699
Murphy, A., 368, 369
Murphy, C. M., 654
Murphy, J. G., 1069
Murphy, L. M., 561
Murphy, M. L., 539
Murray, C. J. L., 116, 119
Murray, J. D., 726, 735, 736, 751
Murray, K. T., 281, 292, 297
Murray, L., 164, 340, 359, 456
Murray, R., 131, 135
Murray-Close, D., 536, 548
Murry, V. M., 113, 603, 604
Mustillo, S. A., 104, 113, 114, 634, 643
Muthén, B. O., 116, 766, 768, 771, 773, 775, 778, 779, 782, 783, 798, 807, 819, 822, 833, 849, 906, 956, 958, 959, 972, 978, 991, 996, 1009, 1027, 1029, 1035, 1036
Muthén, L., 956, 972
Muthén, L. K., 116, 768, 771, 778, 779, 783, 906, 991, 1009
Muthén, L. M., 807, 819, 822
Myers, D., 148
Myers, J., 502
Myers, L., 3
Myers, M. M., 175
Myers, R. L., 219, 240
Myers, S. S., 281, 285
Myin-Germeys, I., 666

Nachmias, M., 164, 295, 296, 354, 355
Nadam, R., 292
Nadel, J., 456
Nagel, E., 102
Nagel, L., 274
Nagin, D. S., 3, 70, 71, 104, 131, 132, 141, 699, 740, 808, 833, 849, 991, 996
Najarian, M., 925
Najman, J. M., 3, 532
Nakagawa, S., 760
Nakamura, E. F., 635
Nakamura. J., 506
Nandi, A., 667
Nangle, D. W., 636, 639, 647, 648, 652
Naragon, K., 513
Narrow, W. E., 55
Narusyte, J., 135, 346
Nash, J. C., 1079
Nathan, P. J., 364
National Advisory Mental Health Council, 918
National Campaign to Prevent Teen Unplanned Pregnancy, 650
National Institute for Child Health and Human Development, 992
National Institute of Justice, 839
National Institute of Mental Health, 581, 582, 594, 595, 839
National Research Council, 130
National Scientific Council on the Developing Child, 184
Nauta, M. H., 699, 700
Navarro, J., 707
Navsaria, N., 3
Neal, C., 176
Neale, M., 120
Neale, M. C., 996
Neblett, E. W., 567
Nechamkin, Y., 361
Neeb, A., 584
Needham, B. L., 646
Needleman, H. L., 94, 113
Neemann, J., 632
Neff, C., 337
Neff, K. D., 512
Neger, E. N., 18
Negrao, C., 500
Negru, O., 289
Neiderhiser, J. M., 298, 341, 344, 604
Neiderman, M., 498
Neighbors, C., 497, 1050, 1069, 1079
Neilands, T. B., 761
Neisser, U., 456
Neitzel, C., 6, 417
Nelder, J. A., 823
Nelis, D., 269
Nelson, J., 687, 700
Nelson, C. A., III, 171, 173, 178, 179, 182, 283, 286, 301, 328, 721, 738
Nelson, E. E., 294
Nelson, J. A., 292
Nelson, K., 454, 455, 476, 506
Nelson, L. D., 1081
Nelson, L. J., 475, 478, 506
Nelson, M. M., 751
Nelson, S., 184
Nelson, S. E., 563, 666, 726, 736, 737, 746
Nemeroff, C. B., 164, 171, 286
Nepple, T. K., 208
Nesse, R. M., 245
Nesselroade, J. R., 61, 62, 803, 817, 819, 826, 848, 849, 852, 854, 857, 858, 861, 884, 885, 908, 944, 959, 989, 993, 994, 995, 996, 1016, 1017
Neufeld, K., 54
Neufeld, R. W. J., 223
Neufeld, S. J., 291
Neuman, C. J., 457
Neumark-Sztainer, D., 643
Neville, H., 446
Newcomb, A. F., 534, 551
Newcomb, M. D., 639, 1008
Newell, K. M., 726, 736, 819, 826, 846
Newhouse, P. A., 54, 72
Newman, 84
Newman, A. E., 173
Newman, D.L., 115
Newman, J. P., 707
Newport, D. J., 164, 171
Newport, E. L., 158
Newtson, D., 726, 732, 743
Neyrinck, B., 404
Nezlek, J.B., 510
Ng, Y., 386
NICHD Early Child Care Research Network, 183, 331, 348
Nicholl, A. R., 294

Nicholls, E., 486
Nichols, E. B., 604
Nichols, K. E., 531
Nichols, S. R., 29, 277
Nicklin, J. M., 404
Nicolson, N. A., 164
Nida, S., 502
Niedenthal, P.M., 689
Nieder, T., 637
Niemiec, C. P., 386, 387, 392, 405, 407, 409, 555
Nievar, M. A., 233, 234
Nifadkar, S. S., 493
Nigg, J. T., 667, 686, 687, 839
Nijenhuis, E., 424
Nikolas, M. A., 687, 839
NIMH Collaborative Data Synthesis for Adolescent Depression Trials Study Team, 1080
Nisbett, R. E., 400, 510, 511
Nishina, A., 502, 547
Nissen, S. E., 1049, 1058
Nistico, L., 345
Nitsch, D., 145
Nitz, K., 705
Noam, G. G., 59, 495
Nocentini, A., 502, 503
Noda, A., 1051
Nolen-Hoeksema, S., 268, 269, 282, 294, 300, 494, 633
Noll, J. G., 288, 500
Nollen, N., 425
Noller, P., 362
Noom, M. J., 417
Noonan, R. K., 641
Nord, C., 925
Nordling, J. K., 421
Nordness, P. D., 687
Norenzayan, A., 510, 511
Normand, S.-L. T., 1061
Northrup, J. C., 590
Northstone, K., 139
Norton, A., 727, 736
Norwood, W. D., 620
Nosek, B. A., 922, 1051
Notarius, C. I., 287, 752
Notaro, P. C., 291, 295
Novak, M. F., 272
Novara, C., 417
Nowak, A., 726
Nozadi, S. S., 277
Nugent, B. M., 672
Numan, M., 223
Nunnally, J. C., 12
Nurious, P., 481
Nurmi, J., 552, 553
Nurmi, J. E., 393, 394
Nuzzo, R., 1051
Nye, E. C., 361
Nylund, K., 547
Nylund, K. L., 1029

Oakes, J. M., 810
Oakland, T., 36
Obel, C., 134, 176
Oberklaid, F., 532, 699
Oberlander, T. F., 168, 170
Oberman, L.M., 689
Ober-Reynolds, S., 293
O'Bleness, J. J., 421
O'Boyle, C. G., 291
O'Brien, L., 558
O'Brien, M., 276, 294, 300, 615
Obsuth, I., 169
O'Callaghan, M. J., 3
O'Connell, P, 748
O'Connor, B. P., 390
O'Connor, E. E., 678
O'Connor, J. C., 540
O'Connor, T. G., 131, 158, 160, 163, 164, 165, 168, 169, 172, 173, 174, 175, 176, 178, 181, 182, 236, 237, 272, 341, 365, 367, 621, 622
Odak, M., 737
Oddi, K. B., 6
Odgers, C. L., 115, 135
O'Donnell, E. H., 287
O'Donnell, K. J., 160, 164, 174, 175, 176, 177, 272
O'Donovan, A., 539
Oetting, W. S., 145, 146
Offord, D. R., 113
Ogawa, J., 367, 368
Ogden, T., 622
Ogle, C. M., 169
Oh, W., 533
O'Hara, A., 726, 734, 746, 749, 751, 752
O'Hearn, H.G., 287
Öhman, A., 214, 216, 268
O'Kearney, R., 278
O'Keefe, M., 641
Olabarrieta, F., 358
Oland, A. A., 293
Olatunji, B. O., 294
Olchowski, A. E., 761, 774, 777, 782, 784, 788
Olds, D. L., 181, 184
O'Leary, D. D., 165, 166
O'Leary, K. D., 638, 641, 650
O'Leary, S. G., 199, 275
Olejnik, S., 876
Olesen, N. W., 615
Oligny, P., 544
Olino, T. M., 269
Oliver, C., 275, 276
Oliver, P. H., 213, 587, 619
Oliver, R., 546
Oliveri, M. E., 24, 38
Ollendick, T., 489, 493
Olrick, J. T., 173
Olsen, B., 284
Olsen, J., 176
Olsen, J. A., 405
Olsen, M. K., 770, 775, 991
Olson, D. H., 228, 589, 590, 613, 617
Olson, K., 776
Olson, L. S., 147
Olson, W. C., 108
Olster, D. H., 1081
Olthof, T., 484, 487
Olweus, D., 117, 502, 544
O'Malley, A. J., 980
O'Malley, P., 1034
O'Malley, P. M., 915
O'Malley, S., 425
O'Neill, S., 3
Ong, A. D., 706
Ong, K., 164
Onishi, K. H., 453
Oosterman, M., 298, 354
Open Science Collaboration, 1043
Oppenheim, B., 98, 108
Oppenheim, D., 35, 334, 339, 358, 359
Oppenheim, R. W., 441
Oppenheimer, J. E., 297
Orbuch, T. L., 199
Orchard, T., 767, 775, 777
Ormel, J., 74
Ormrod, R., 494
Orobio de Castro, B., 536
O'Rorke, D. J., 402
O'Rourke, K., 1043, 1052
Ortiz, J., 356
Osanai, Y., 9
Osborne, L. N., 241, 620
Osbourne, C., 745
Osgood, C. E., 673
Osgood, D. W., 809
Oshri, A., 547
Osorio, A., 359
Ostendorf, F., 813, 848, 849, 858
Oster, H., 274
Osterlow, J., 637
Ostrov, J. M., 698
Ostvik-White, E., 409
Othmer, E., 416
Othmer, S. C., 416
Otsubo, Y., 510
Ott, M. Q., 646
Ottaviani, E., 165
Otto, H., 602
Otto, Y., 2, 34
Ou, A. Y., 493
Oud, J. H. L., 956, 1037
Ouellet-Morin, I., 549
Oumar, F., 340
Ouston, J., 141
Out, D., 340
Overall, J. E., 52, 890
Overbeek, G., 564, 635
Overholder, J., 495
Overton, W. F., 717, 718
Owen, D., 175
Owen, J., 634
Owen, L. D., 638, 640, 641, 642, 648, 649, 651, 652
Owen, M. T., 224, 225, 296, 347, 349
Owens, E. B., 295, 925
Owens, J. A., 251
Owens, P. L., 118
Oxford, M. L., 645, 646
Oyama, S., 448
Oyer, M., 402
Ozechowski, T. J., 978
Ozer, D. J., 798
Ozer, E. L., 649

Pacifici, C., 250
Padgett, D. A., 164
Padgett, G., 699
Pagani-Kurtz, L., 638
Painter, R. C., 159
Pajer, K., 165
Palazzoli, M. S., 583
Paley, B., 213, 224, 227, 580, 587, 590, 601, 620, 719
Palkovitz, R., 606
Palmer, T. D., 177
Panak, W. F., 537, 538
Panfile, T. M., 360, 798
Panskepp, J., 221, 223, 268, 363
Pantoja, A. P. F., 727
Paolicelli, R. C., 165
Papa, A., 752
Papadakis, A. A., 636, 639, 647, 648, 652
Papini, D., 412
Papp, L. M., 204, 206, 215, 219, 226, 242
Papp, P., 591, 601

Parade, S. H., 276, 295
Pardo, A., 922
Paredes, W., 450
Park, S. Y., 418
Parke, R. D., 218, 232, 233, 280, 281, 357, 534, 555, 586
Parker, E. H., 280
Parker, J., 798
Parker, J. D., 28
Parker, J. G., 235, 527, 528, 551
Parker, K. J., 539, 540
Parker, R., 1052
Parker, S. T., 457
Parkhurst, J. T., 542
Parnas, J., 465
Parner, E. T., 177
Parra, G. R., 297
Parris, L., 294, 502
Parritz, R. H., 295, 354, 355
Parrott, W. G., 407, 501
Parsons, B. V., 584
Parsons, E., 291, 340
Parziale, J., 688, 689
Pasalich, D. S., 279, 301
Pasch, L. A., 234, 649
Pashler, H., 1043
Passos-Ferreira, C., 456
Patall, E. A., 402, 1043, 1055, 1067
Patel, M., 726
Patel, V., 116
Patenaude, R. L., 620
Patrick, H., 425
Patrick, M., 297
Pattee, L., 534
Patterson, C. J., 171, 535, 604, 1004
Patterson, C. P., 648
Patterson, G. R., 237, 542, 563, 585, 595, 611, 619, 622, 640, 648, 666, 722, 724, 725, 726, 728, 730, 732, 746, 748, 752, 1034
Patterson, P. H., 170
Paulhus, D. L., 508, 510
Paulicin, B., 28
Pauli-Pott, U., 230
Pautsch, J., 1, 2, 3, 4, 36
Pawelec, G., 166
Paxton, P., 972
Paxton, S. J., 498, 642
Pearce, M. J., 666
Pearl, J., 101, 113, 130, 973, 974, 975, 980
Pears, K. C., 183, 638, 645, 650, 651
Pearson, K., 1051
Pearson, M., 560
Pearson, M. R., 642
Pechtel, P., 288
Pedersen, E. R., 1069
Pedersen, F. A., 397, 399
Pedersen, N., 761
Pedersen, N. L., 72
Pedersen, S., 537, 551, 639
Pederson, D. R., 338, 340
Pederson, W. C., 499
Pedhazur, E. J., 927
Pee, D., 785
Peebles, C. D., 3
Peel, D., 991
Peer, J., 667
Peets, K., 543
Pelham, W. E., 536
Pellegrini, D. S., 287
Pelletier, J. E., 386, 395, 413
Peloso, E., 183, 356, 366
Pelphrey, K. A., 293
Peltonen, T., 537, 544
Pemberton, C. K., 277
Peng, K., 510, 511
Pennings, H. J., 746
Pentz, M. A., 979
Penza-Clyve, S., 284
Peper, J. S., 99, 293
Pepler, D., 726, 729, 730, 734, 746, 748, 749, 751, 752
Pepler, D. J., 247, 546, 632, 639, 650
Perez, M., 643
Perez, S. M., 292
Perfect, M., 281
Pergamin, L., 38
Perlman, S. B., 281, 287, 293
Permezel, M., 223
Perren, S., 503, 546, 548
Perrin, E. C., 18, 21, 23
Perrine, M. W., 802, 895, 901, 906, 908
Perris, E. E., 453
Perry, D. G., 546
Perry, I. J., 292
Pescosolido, B. A., 110
Pessoa, L., 268
Peter, J., 545
Petermann, F., 300
Peters, E., 554
Peters, T., 283
Petersen, A. C., 994
Peterson, B. S., 166
Peterson, C., 494
Peterson, D., 541
Peterson, G. W., 246
Peterson, J. L., 1009
Peterson, P., 148
Peto, J., 112, 113
Peto, R., 112, 1049, 1057
Petrican, R., 271
Petrill, S. A., 223
Pettit, G. S., 117, 219, 535, 536, 543, 545, 547, 698
Pettit, J. W., 158, 491
Peugh, J. L., 288, 762, 783, 784
Pfander, A., 387
Pfeffer, C. R., 495
Pfeifer, J. H., 286, 287, 298, 558, 559
Phan, K., 364
Phares, V., 601
Philibert, R. A., 167, 343
Philippe, F. L., 391
Phillips, D., 38, 502
Phillips, D. I., 159
Phillips, M., 285
Phillips, S., 499, 501
Phillips, V., 604
Piaget, J., 49, 266, 385, 447, 457, 475, 478, 479, 482, 506, 527, 553, 675, 676, 707
Pianta, R. C., 362, 689, 718
Piasecki, T. M., 78
Picardi, A., 345
Piccinin, A. M., 760
Pickett, K. E., 116, 134
Pickles, A., 85, 97, 105, 113, 131, 132, 135, 178
Piehler, T. F., 563
Pierik, F. H., 177
Pietromonaco, P. R., 296
Pijlman, F. T., 356
Pike, A., 130, 552
Pike, K.M., 498
Pilgrim, C., 565
Pillay, S. S., 364
Pillemer, D. B., 489
Pillow, D., 536
Pina, A. A., 30, 31, 32
Pincus, A. L., 385
Pincus, D., 20, 726, 737
Pine, D. S., 10, 35, 231, 294, 301
Pinheiro, J. C., 940, 996
Pinker, S., 448
Pinquart, M., 347, 348
Pipp, S. L., 673, 675, 678, 681
Pirlott, A. G., 973
Pisani, A. R., 118
Pisani, L., 340
Pittman, L. D., 605
Pituch, K. A., 938
Pitzer, M., 173
Pizzagalli, D. A., 288
Pizzorusso, T., 159, 174
Plant, R., 401, 419, 425
Plaut, S.M., 175
Plomin, R., 103, 130, 131, 167, 341, 344, 345, 604
Plotnik, J., 457
Plotsky, P. M., 160, 174
Ploubidis, G. B., 23
Pluess, M., 231, 247, 248
Plunkett, K., 744
Plutchik, R., 266
Pocock, S. J., 1060
Podolski, C.-L., 67
Polcari, A., 169
Pollack, W., 502
Pollak, S. D., 171, 179, 218, 286, 298, 301
Pollastri, A., 271, 287
Pols, R. G., 118
Pomaki, G., 494
Pomerantz, E. M., 390, 392, 393, 394
Pomery, E. A., 563
Ponizovsky, A. M., 361
Pons, F., 284, 289
Pons-Salvador, G., 746
Pope, H. G., Jr., 642
Popham, W. J., 785
Popoola, O., 280
Porges, S. W., 219, 249
Pornprasertmanit, S., 778, 779
Port, R. F., 721
Portella, A. K., 159
Porter, C. L., 291, 700
Porter, M. R., 541, 564
Poskiparta, E., 568
Poslawsky, I. E., 365
Posner, M. I., 221, 230, 231, 292
Post, R. M., 184
Potenza, M. N., 388
Pott, M., 353
Potworowski, G., 721, 746
Pouget, A., 676
Poulin, F., 551, 552, 555, 639, 699
Poulin, R., 292
Poulin-Dubois, D., 443
Poulton, R., 115
Powell, J. L., 598
Powell, L. J., 453
Power, C., 116
Power, T. G., 281, 292
Powers, B. P., 291
Pozen, D., 557
Prata, G., 583
Preacher, K. J., 878, 918, 923, 930, 931, 937, 938, 939, 946, 947, 948, 953, 968, 969, 980
Prechtl, H. F. R., 449
Preciado, P., 554

Prediger, D. J., 891
Preece, M. A., 820, 821, 824, 879, 899, 994, 995
Preen, E. C., 698
Prentice, D. A., 560
Prentky, R. A., 50
Prescott, C. A., 119, 133, 134, 272
Presnell, K., 643
Preston, S., 223
Price, C. E., 395
Price, J., 245, 247
Price, J. M., 537
Price, R. K., 104
Prichard, J. C., 106, 107
Prigatano, G. P., 283
Prigogine, I., 720
Prinstein, M. J., 219, 535, 537, 538, 541, 542, 543, 544, 546, 552, 556, 557, 559, 560, 562, 564, 565, 567, 647, 648, 649
Prinz, R. J., 232
Prinzie, P., 394, 545, 615
Prior, H., 457
Prior, M., 532, 699
The PRISMA Group, 1052
Pritchard, M., 643
Prom, E. C., 136
Propper, C., 167, 217, 696
Pruchno, R., 885
Przybylski, A. K., 387, 388, 425
Ptasznik, A., 847
Pugh, L., 1069
Pugh, R. H., 856
Pugh, T. F., 130
Puig-Antich, J., 35, 53
Pulfrey, C., 401
Pulkkinen, L., 144
Pungello, E. P., 180
Purcell, K., 650
Pursell, G., 247
Puterman, E., 494
Putnam, F. W., 3, 288, 303, 488, 500, 506, 673, 677, 678
Putnam, S. P., 231, 304, 587
Pyszcznski, T., 494
Python, M., 614
Pyzalski, J., 502

Qi, C. H., 2
Qiao, Z., 174
Qualter, P., 699
Quamma, J. P., 304
Quas, J. A., 169, 284
Quatman, T., 633, 634
Quay, H. C., 52, 54, 55, 62, 65
Queller, S., 58
Quevedo, K. M., 219, 356, 808
Quinkert, A. W., 222
Quinlan, P. T., 740
Quinlan, S. V., 1009
Quinlan, T., 1069
Quinn, D., 294
Quinton, D., 161

Rabian, B., 110
Rabiner, D. L., 535, 1007
Raby, K. L., 295, 343
Rademacher, J., 278
Radke-Yarrow, M., 206, 284, 287, 461
Radzioch, A. M., 268, 279
Raeff, C., 707
Raffaelli, M., 294, 298
Raftery, A. E., 71, 822
Raghavan, C., 268
Raghunathan, T. E., 761, 775, 785
Raiche, G., 858
Raikes, H., 358, 359
Raine, A., 356, 420
Raison, C. L., 540
Raj, T, 271
Raja, R., 532
Rajendran, K., 3
Rakic, P., 441
Rakison, D. H., 443
Rakoczy, H., 283
Ram, N., 302, 642, 651, 706, 707, 803, 810, 848, 849, 858, 861, 990, 991, 993, 994, 995, 996, 999, 1000, 1003, 1017, 1028
Ramachandran, V. S., 445
Ramaswamy, V., 1028
Rambaran, A. J., 565
Rameson, L. T., 541
Ramey, C. T., 174, 180, 184
Ramey, S. L., 184
Ramirez, M. M., 174
Ramisetty-Mikler, S., 287
Ramos-Marcuse, F., 359
Ramsay, D. S., 172, 302, 808
Ramsay, M., 652
Ramscar, M., 169
Ramseyer, F., 752
Rancourt, D., 541, 542, 559, 562, 565
Rand, K. L., 516
Rand, W. M., 829
Randall, A. K., 291
Rank, B., 162
Rank, O., 162
Rantanen, P., 642
Rao, C. R., 851, 995, 998
Rao, U., 30, 31, 32
Rapee, R. M., 10, 14, 20, 28, 32, 34, 637, 652, 699
Rapoport, J. L., 55
Rapp, C., 847
Rasbash, J., 236, 237
Rasi, J. A., 202, 207, 217, 229, 249
Rasmussen, J. L., 617, 618
Rast, P., 979
Ratcliff, K. S., 53
Ratner, H. H., 457
Rauch, S. L., 363
Raudenbush, S. W., 7, 67, 849, 850, 935, 939, 940, 959, 961, 962, 963, 979, 996, 1000
Rausch, J. R., 288, 996
Raval, P. H., 589
Raval, V. V., 589
Raver, C. C., 222, 251, 269, 281, 296, 687
Raviv, T., 293
Ravussin, E., 138
Ray, A. E., 1055, 1068
Ray, J. V., 268
Raya, P., 673
Rayner, R., 54
Raynor, S. R., 287, 587, 615, 616
Raznahan, A., 271
Read, J. P., 1071
Read, S. J., 345
Rebok, G. W., 118
Recchia, H. E., 277
Redfern, S., 358
Redshaw, J., 139
Reeb-Sutherland, B. C., 231
Reed, A., 171, 286
Reef, J., 50, 73, 74
Reese, H.W., 908
Reeve, J., 388, 392, 401, 402, 405, 426
Reeve, R., 675
Reeves, C. W., 294
Regalia, C., 589
Regan, C., 833
Regier, D. A., 597
Rehm, L. P., 494
Rehm, J., 1054
Reich, W., 110, 111
Reichardt, C. S., 934, 953, 954, 955, 956
Reichmann-Decker, A., 275
Reid, R., 687
Reid, J. B., 622, 654, 725, 732, 748, 1032
Reid, J. M., 365
Reid, V. M., 283
Reijntjes, A., 284, 294, 499, 545
Reik, W., 168
Reilly, J. S., 284
Reimer, R. A., 563
Reinherz, H. Z., 115
Reinke, W. M., 1032
Reis, H. T., 407, 719
Reisby, N., 949
Reise, S. P., 856, 991
Reiss, D., 130, 132, 227, 580, 588, 591, 592, 593, 596, 597, 601, 604
Reissland, N., 274
Reiter, J. P., 775
Reitsma, J. B., 1050, 1062
Rende, R., 167
Rendell, M., 740
Rennels, J. L., 283
Renwick, S. D., 667
Repacholi, B. M., 284, 358
Repetti, R. L., 219, 281, 301
Reppucci, N., 702
Rescorla, L. A., 17, 24, 25, 27, 53, 54, 62, 72, 73, 76, 77, 78, 79, 83, 84, 85, 86, 87, 109
Réserbat-Plantey, D., 456
Resnick, G., 353
Restemeier, R., 117
Rettew, D. C., 53, 78
Reusing, S. P., 272
Reyes, E., 274
Reyes, H. L. M., 641
Reynolds, A. J., 180, 181
Reynolds, B., 219
Reynolds, B. M., 281
Reynolds, C. A., 72
Reynolds, C. R., 20, 21, 25, 52
Reznick, J. S., 35, 531
Rheaume, J., 417
Rhee, U., 493
Rhemtulla, M., 761, 774, 775, 776, 777, 784, 786, 789, 790
Rhoades, G. K., 250, 654
Rhoades, K. A., 212, 216, 217, 240
Rhodes, J., 806
Rhodes, J. S., 177
Rhodewalt, F., 485
Rhule-Louie, D. M., 648
Ribeiro, A. P., 746
Rice, 499
Rice, B. J., 118
Rice, D., 158
Rice, F., 137, 176, 272
Rice, L. N., 751
Richards, M. H., 293, 294, 298, 632
Richman, N., 17
Richter, D., 232, 250
Richter, L. M., 604
Richters, J. E., 98, 109, 220, 331, 717, 718, 719, 752, 917

Rickert, M. E., 1022
Ricks, D., 638
Rid, J. B., 752
Ridgeway, D., 201, 277, 284, 334, 358
Riefer, M., 847
Riem, M. M., 363, 364
Ries, S., 515
Rifkin, A., 340
Rifkin-Graboi, A., 170
Rigatuso, J., 172, 355
Rigby, S., 404
Rigdon, E. E., 971, 972
Riggs, S. A., 361, 362, 589, 618
Riksen-Walraven, J., 333, 338, 339
Riksen-Walraven, J. A., 554
Riksen-Walraven, J. M., 172
Riley, R., 1062, 1063, 1066
Rinaldi, C. M., 277, 615
Rinaldi, J., 181, 365
Rincon, C., 636
Ringuette, E. L., 583
Rinpoche, T., 511
Riopel, M., 858
Riper, H., 300
Risch, N., 74
Risi, S., 538
Risko, W., 19
Risler, J. L., 828
Risley, T. R., 277
Rittenhouse, J., 183
Rivera-Hudson, N., 667, 698
Rivers, S. E., 269
Rivier, C. L., 219
Rivkees, S. A., 283
Rivkin, I., 409
Rizzo, C. J., 635
Rizzolatti, G., 447
Robaey, P., 667
Robb, M. D., 342
Robbins, M. S., 584, 609
Robbins, R. J., 401
Roben, C. K., 277
Roberts, B. W., 208
Roberts, C., 87
Roberts, J. E., 296
Roberts, L. E., 498, 499, 751
Roberts, R. E., 78, 79, 642, 644
Robertson, D. L., 180, 181
Robertson, J., 326, 600
Robins, D. L., 19
Robins, J., 1057
Robins, J. M., 97, 98, 99, 101, 104, 113, 116, 130, 131, 141, 145, 973, 974, 976, 977, 980
Robins, L. N., 50, 53
Robins, R. W., 487, 500, 798, 816
Robinson, C., 402, 633, 634
Robinson, D., 678
Robinson, G. K., 849, 850
Robinson, L. R., 281, 285
Robinson, M., 176
Robinson, N. S., 490, 557
Robinson, W. S., 801, 802, 979
Robles, T. F., 219
Robles de Medina, P. G., 272
Robles-Pina, R., 494
Rochat, P., 440, 443, 444, 448, 449, 450, 451, 452, 453, 455, 456, 457, 458, 459, 460, 461, 464, 475, 476, 506
Rodin, G. C., 404, 425
Rodkin, P., 116, 245
Rodrigues-Doolabh, L., 334
Rodriquez, A., 50
Roe, D., 497
Roebroeck, A., 854
Roede, M. J., 61
Roeder, K., 808
Roerecke, M., 1054
Roesch, L., 635
Roeser, R. W., 269, 505
Roeyers, H., 686
Roff, M., 527
Rogers, C., 388
Rogers, C. R., 508, 817
Rogers, M. J., 235
Rogers, S. J., 232
Rogoff, B., 527
Rogosa, D., 67, 959, 1017
Rogosch, F. A., 163, 168, 171, 202, 206, 214, 217, 219, 246, 251, 271, 286, 297, 300, 351, 366, 368, 495, 536, 547, 613, 673, 924, 988, 989, 1023, 1025, 1029, 1031, 1037, 1048
Rogowska, J., 364
Rohde, P., 115, 633, 635, 654
Rohleder, N., 539
Rohling, M. L., 639, 640
Rohner, R. P., 617
Rohwer, G., 731
Roisman, G. I., 237, 276, 331, 335, 342, 345, 347, 349, 351, 352, 353, 354, 360, 362
Röll, J., 300
Rollnick, S., 425, 426
Román, M., 922
Romano, L. J., 698
Romero-Canyas, R., 698
Romney, D. M., 131
Ron, T., 409
Ronai, Z., 343
Roodenrys, S. J., 687
Rosca, P., 361
Rosch, E., 58
Rose, A. J., 542, 543, 556
Rose, B. M., 923
Rose, R. J., 144
Rose, S. P., 673, 675, 676, 678
Roseboom, T. J., 159
Roseby, V., 209, 218, 238, 251
Rose-Krasnor, L., 532
Rosen, C. S., 304
Rosen, K., 203
Rosen, K. S., 353
Rosenbaum, J. E., 131, 141, 148, 296
Rosenbaum, P. R., 131
Rosenberg, M., 474, 475, 479, 491
Rosenblatt, A., 79
Rosenbluth, D., 326
Rosenheck, R. A., 118
Rosenthal, D. R., 541
Rosenthal, M. K., 330
Rosenthal, R., 1051
Rosenthal, S., 347, 348
Rosman, B., 419
Rosman, H., 420
Ross, E. J., 170, 276
Ross, G., 10, 688
Ross, H., 483
Ross, L., 560
Rosseel, Y., 404, 778, 779
Rossi, A. S., 116
Rossi, M. A., 144
Rost, J., 833
Rostosky, S. S., 635
Roswell, H. C., 288
Rotenberg, E. J., 673
Roth, G., 391, 392, 395, 401, 405, 407, 408, 409, 410, 418
Roth, J. A., 638
Rothbart, M. K., 230, 231, 276, 281, 290, 291, 292, 294, 295, 304, 697
Rothbaum, F., 203, 353, 505
Rothenfluh, T., 751
Roth-Hanania, R., 276, 698
Rothman, K. J., 101, 102, 130, 131
Rothstein, H. R., 1048, 1054, 1061, 1073, 1079
Rottenberg, J., 752
Rouhani, P., 675
Rousseau, Jean-Jacques, 459
Rousson, V., 918
Rovee-Collier, C., 455
Rovine, M. J., 761, 810, 826, 848, 849, 851, 852, 857, 858, 861, 870, 876, 893
Rowa, K., 590
Rowden, L., 532
Roy, K., 606
Røysamb, E., 642
Roysland, K., 980
Royston, P., 774, 782
Rubenstein, J. C., 397, 399
Rubenstein, J. L., 166
Rubin, D. B., 130, 144, 146, 760, 761, 763, 764, 765, 766, 767, 768, 769, 770, 772, 773, 774, 775, 777, 779, 780, 782, 783, 784, 973, 974, 1029, 1030
Rubin, K. H., 235, 356, 528, 530, 531, 532, 533, 695
Rubin, R. M., 696
Ruchkin, V., 666
Rucker, D. D., 878, 918, 931, 980
Rudolph, S. G., 494
Rudolph, K. D., 238, 239, 288, 293, 297, 538, 546, 549, 556, 633
Rudy, D., 234
Rueda, M. R., 292
Rueda, R., 231
Ruela, A., 560
Ruffman, T., 277
Ruggiero, G., 487
Ruhland, R., 726, 745
Rumelhart, D. E., 726, 743
Ruocco, A. C., 388
Rusiecki, J. A., 168
Rusk, N., 505
Russell, C. S., 228, 589, 613
Russell, D., 760
Russell, J. A., 284, 358
Russell, S. T., 646
Russell Localio, A., 1059
Rutter, M., xi, 47, 49, 59, 60, 84, 129, 130, 131, 132, 133, 134, 135, 136, 139, 140, 141, 142, 143, 147, 149, 160, 161, 163, 173, 174, 178, 208, 209, 229, 240, 325, 328, 353, 496, 587, 598, 599, 648, 666, 718, 924, 987, 988, 1048
Rutter, M. L., 96, 98, 101, 102, 104, 105, 110, 112, 113, 120
Ruuska, J., 642
Ryan, A., 485
Ryan, K. D., 726, 735
Ryan, N., 30, 32
Ryan, R. M., 6, 223, 385, 386, 387, 388, 389, 390, 391, 392, 393, 394, 395, 397, 398, 399, 400, 401, 402, 403, 404, 405, 406, 407, 408, 409, 410, 411, 412, 413, 414, 415, 416, 417, 418, 419, 420, 421, 422, 423, 424, 425, 426, 427, 428, 505, 516
Ryckoff, I. M., 581

Rydell, A.-M., 351
Ryle, G., 518

Saarni, C., 266, 269, 273, 281, 283, 290, 302, 500, 512, 513, 682
Saavedra, L. M., 30, 31, 32
Sabatini, M. J., 363
Sabelli, H. C., 726
Saboonchi, F., 798
Sabourin, S., 404
Sabroe, S., 176
Sackett, G. P., 158, 174
Sadek, S., 223
Sadhu, J., 594
Sadhwani, I., 494
Sadler, P., 706
Saenger, G., 116, 142
Saffran, J., 451
Sagi, A., 339, 353, 455
Sagi-Schwartz, A., 329, 358, 359
Saint Augustine, 459
Saint Onge, J. M., 565
Saks, E., 465
Sakura, S., 493
Salekin, R. T., 548
Salgado, J., 746
Salmela-Aro, K., 552
Salmivalli, C., 543
Salovey, P., 269, 513
Salpekar, N., 296
Salsman, N. L., 294
Salthouse, T. A., 786, 991
Salvaterra, F., 335
Salvator, A., 147
Sameroff, A., 666, 718
Sameroff, A. E., 271, 303
Sameroff, A. J., 113, 207, 236, 269, 395, 413, 421, 422, 529, 588
Sampson, K., 633, 634, 648
Sampson, R. J., 7, 131, 141, 798, 803, 809, 811
Samuels, J., 798
Samuelson, L. K., 743
Sanchez, D. T., 498, 643
Sanchez, M. M., 174
Sanchez-Mut, J. V., 168
Sanders, M. R., 251, 367
Sandfort, T. G. M., 646
Sandler, J., 485
Sandman, C. A., 160, 176, 177, 271, 290, 294
Sandstrom, M. J., 562
Sandy, J. M., 1008
Sanford, M., 607
Sanson, A., 3, 532, 699
Sanson, A. V., 414
Santa-Barbara, J., 606
Santo, J., 485, 554
Santos, J. C., 495
Santucci, A. K., 295
Sarac, T., 590, 603
Saraiva, C., 495
Sarbin, T. R., 474, 478
Sarfati, D., 278
Saridjan, N., 355
Sarkar, P., 160, 164, 175
Sarkar, S., 166
Sartre, J.-P., 460
SAS Inc., 941, 962
SAS Institute, 771
Sasssaroli, 487
Sassville, T. M., 418
Satir, V., 582
Sauck, C. C., 395
Saudino, J. K., 290, 294
Saudino, K. J., 167
Sauer, B., 441
Saunders, J. B., 1071
Savalei, V., 761, 762, 774, 775, 777, 778, 780, 781, 784, 786
Savard, A., 386
Savelsbergh, G. J.P., 745
Savin-Williams, R. C., 646
Savla, J., 761, 778
Sawalani, G. M., 544
Sawhill, I., 184
Sayer, A. G., 958, 959, 995, 996
Sayers, S. L., 610
Sbarra, D. A., 706, 707
Scabini, E., 589
Scaramella, L. V., 298
Scarpa, A., 698
Scarr, S., 103, 604
Scerbo, A. S., 84
Schaal, B., 441, 443, 450, 451
Schabenberger, O., 851
Schacht, R., 221
Schade, R., 175
Schaefer, E. S., 391
Schaefer, H. S., 291, 553
Schaefer, S., 160
Schafer, J. L., 760, 761, 763, 765, 767, 768, 769, 770, 773, 774, 775, 776, 777, 782, 820, 991
Schafer, R., 388
Schaffner, E., 176
Schaller, M., 241
Schank, R. C., 332
Scharf, M., 632, 647
Schargrodsky, E., 144
Schatz, J. N., 250, 251
Schauerhuber, M., 892
Schechter, D. S., 7
Scheeringa, M. S., 3, 30, 31, 32, 33, 34
Scheff, T. J., 501, 696
Scheffer, D., 117
Scheidler, J., 1050
Scheier, C., 726, 737, 744
Scheier, M. F., 516, 671
Schell, T., 58
Schendel, D. E., 177
Schenker, N., 760
Schensul, J., 702, 703, 704
Scherer, D. G., 118
Scherer, K. R., 281, 679
Schermerhorn, A. C., 203, 210, 213, 216, 217, 225, 230, 232, 236, 237, 240, 250, 600, 609, 611, 612, 616
Schieche, M., 291, 355
Schiepek, G., 704, 731, 751
Schlotz, W., 139
Schmidt, H. D., 808
Schmidt, L. A., 531, 551, 553
Schmidt, M. H., 173
Schmidt, P., 956
Schmiedek, F., 160, 847
Schmittmann, V. D., 667
Schmitz, B., 802
Schmitz, N., 176, 177
Schnabel, K. U., 761
Schnall, M., 422
Schneider, B. H., 349
Schneider, H., 924
Schneider, M., 1054
Schneider, M. L., 175, 176
Schneider, S., 751
Schneiderman, R. L., 3
Schneider-Rosen, K., 99, 168, 296, 718, 804,
Schniering, C. A., 699
Schneiger, A., 184
Schnurr, M. P., 641, 649
Schoemann, A. M., 778, 779, 789
Schoenmaker, C., 335
Schoeny, M. E., 704
Schoff, K., 228, 232
Scholte, R. J., 552, 559, 564
Schonberg, M. A., 282, 293, 294, 359, 613
Schoner, G., 726, 744
Schonwald, A., 19
Schooler, D., 642, 643
Schöpflin, U., 161
Schore, A. N., 672
Schore J. R., 672
Schore, A. N., 364
Schouten, E., 666, 699
Schrater, P., 676
Schrepferman, L. P., 563
Schroeck, F. E., 726, 732
Schudlich, T. D. D. R., 297
Schuengel, C., 298, 340, 354, 368, 369
Schulenberg, J., 915
Schultheiss, O. C., 174
Schultz, D., 304
Schultz, P. P., 407
Schulz, K. F., 1052
Schulz, L. A., 338
Schulz, L. O., 138
Schulz, M. S., 251, 598, 603
Schumacher, J. A., 640
Schumacker, R. E., 971, 972
Schumann, G., 1053
Schuster, C., 892
Schutte, A. R., 666, 706
Schwab, M. E., 593
Schwab-Stone, M. E., 19, 53, 55, 59, 78, 98, 110, 666
Schwannauer, M., 361
Schwarcz, P., 554
Schwartz, A., 622
Schwartz, A. J., 410,402
Schwartz, C. E., 531
Schwartz, D., 542, 545, 546, 547, 846
Schwartz, G., 1033
Schwartz, G. E., 288
Schwartz, J., 410, 699
Schwartz, M., 165
Schwartz, M. E., 302
Schwartz, O. S., 280, 294
Schwartz, S., 130, 131, 646
Schwartz, S. J., 231, 251, 494
Schwartzman, A., 485
Schwartz-Mette, R., 268
Schwarz, A., 457
Schwarz, B., 287
Schweinberger, M., 561
Schweizer, S., 268, 294
Sclove, L. S., 1030, 1033
Scolton, K. L., 218, 357
Scott, J. G., 8, 165
Scott, S., 168, 169, 173, 181, 182, 365
Scott-Sheldon, L. A., 1052, 1067
Seal, K., 392
Searles, J. S., 895, 901, 906, 908
Sears, M. S., 281
Sears, R. R., 391, 405, 543
Seaton, E., 234
Secher, N. J., 176
Seckl, J. R., 167
Sedikides, C., 481, 489

Seeley, J. R., 115, 158, 633, 634, 635, 642, 644
Seeman, T. E., 164, 170
Segal, M. R., 1050
Segal, Z. V., 635, 751
Seid, M., 615
Seidman, E., 702
Seifer, R., 7, 10, 18, 113
Seiffge-Krenke, I., 632, 637
Seifritz, E., 363
Seisenberger, S., 168
Selig, J. P., 946, 947, 948
Seligman, M. E. P., 494, 512
Selikoff, I. J., 112
Selman, R. L., 476, 477, 479, 482, 483
Selvin, H. C., 801
Selwyn, C., 639, 640
Selzer, M. L., 1032
Sen, S., 120, 344
Senders, S. J., 450
Seok, J., 174
Sergeant, J., 516
Serketich, W. J., 278, 718
Serpell, L., 498
Serrat, E., 284
Seth, A. K., 854
Settle, J. E., 555
Shachar-Shapira, L., 652
Shackman, A. J., 286, 298, 301
Shackman, J. E., 286, 298, 301
Shadish, W. R., 101, 130, 131, 132, 140, 146, 149, 1043, 1054
Shaffer, A., 297, 590, 600, 604, 605, 609
Shaffer, D., 49, 53, 55, 78, 98, 109, 110, 118
Shaffer, L., 632, 647
Shafran, R., 269
Shahar, G., 497, 789
Shamir, H., 233, 234
Shamir-Essakow, G., 32, 34
Shanahan, S., 696
Shanahan, L., 114, 644
Shanahan, M. J., 978
Shannis, D., 164
Shannon, C. E., 736
Shannon, E. S., 448
Shannon, K. E., 220
Shantz, C. U., 285
Shapiro, D., 385, 387, 388
Shapiro, J. R., 275
Shapiro, S. L., 511
Sharif, V., 499
Sharma, A., 145, 158
Sharp, C., xi
Shattuck, A., 38
Shatz, J. N., 250, 251
Shaver, P. R., 49, 223, 276, 284, 296, 362, 396, 397, 493, 505, 673, 679
Shaw, C. D., 727, 736
Shaw, D. S., 3, 282, 293, 294, 295, 296, 298, 351, 359, 613
Shaw, H., 654
Shedden, K., 120, 344, 849, 991, 996, 1027
Sheeber, L. B., 236, 278, 287, 294, 298, 301, 987, 988, 1006
Sheehan, K. M., 785
Sheese, B. E., 292
Sheets, V., 923, 929, 930, 1024
Sheikli, H., 501
Sheinman, L., 402
Sheldon, K. M., 390, 421, 428
Sheldon-Keller, A. E., 362
Sheldrick, R. C., 18, 21, 23, 87
Shelton, K. H., 204, 206, 212, 213, 214, 215, 226, 230, 287
Shemesh-Iron, M., 223
Shen, E., 979
Shen, L., 670
Shenk, C. E., 288
Shentu, Y., 918, 1056
Shepard, B., 274, 276
Shepherd, M., 98, 108
Sher, E., 339
Sher, K. J., 1055, 1071
Sherdell, L., 269
Sheridan, J. F., 164, 179, 407
Sherling, G., 244
Sherman, L. J., 340, 359
Sherrill, J. T., 110
Sherrington, C. S., 449
Sheshko, D. M., 300
Shevock, A. E., 760
Shiekhattar, R., 120
Shields, A. M., 223, 276, 279, 420, 746, 749, 1023
Shifflett, K., 250
Shiffman, S., 268
Shiffrin, N. D., 294, 301
Shiffrin, R. M., 58
Shilatifard, A., 120
Shin, H., 488
Shin, T., 775
Shinar, O., 1008
Shindledecker, R., 84
Shiner, R. L., 290
Shipman, K. L., 279, 281, 284, 286, 297
Shirk, S. R., 289
Shirtcliff, E. A., 219, 239, 242, 243, 805
Shmueli-Goetz, Y., 345
Shochet, I. M., 283
Shoemaker, D. M., 785
Shonkoff, J. P., 38, 170
Short, K. R., 453
Short, S. J., 177
Shortt, J. W., 294, 298, 301, 638, 639, 640, 641, 642, 648, 649, 652, 654
Showalter, D., 15
Showers, C., 498
Shrier, L. A., 634
Shrout, P. E., 337
Shugart, M., 286
Shulman, S., 632, 647, 652
Shutter, J., 274
Shuttleworth, F. K., 812
Si, X., 1, 2, 3, 4, 36
Sidani, S., 777
Sidera, F., 284
Sideridis, G., 402
Siebelink, B. M., 16
Siebenbruner, J., 632
Siedman, H., 112
Siegel, A. W., 494
Siegel, R., 635, 636, 643
Siegler, R. S., 477, 689
Sierens, E., 391, 426
Siffert, A., 287
Sigman, M., 275, 463
Sih, A., 245, 246
Sikora, D. M., 542
Silberg, J. L., 119, 120, 135, 136
Silk, J. S., 281, 285, 288, 293, 294, 295, 297, 301, 302, 387, 389, 423, 700
Silva, M. T., 640, 648
Silva, P. A., 97, 113, 115, 117, 135, 1005
Silveira, P. P., 159
Silver, R. B., 10, 18
Silverberg, S., 387, 389, 390
Silverman, A. B., 115
Silverman, W. K., 30, 31, 32, 110
Silverstein, L. B., 601
Silverstein, M., 605
Silverstein, O., 601
Silvia, P. J., 517
Sim, L., 298
Simard, L. R., 667
Simmons, H., 101, 111, 119
Simmons, J. G., 294
Simmons, J. P., 1081
Simmons, R. G., 479
Simmons, R. J., 351
Simmons, T., 5
Simner, J., 445
Simner, M. L., 455
Simon, H., 175
Simon, V. A., 552, 556, 560, 647, 648, 649, 650, 673, 677, 678
Simonoff, J. S., 739
Simons, J. S., 587, 601, 613, 616
Simons, L. G., 203
Simons, R. F., 269
Simons, R. L., 203
Simonsohn, U., 1081
Simon-Thomas, E., 223
Simonton, D. K., 514, 515
Simpson, A., 236, 237
Simpson, J. A., 274, 327, 350
Simpson, N., 364
Sinclair, K. O., 810, 848, 849, 858
Singer, A., 417
Singer, E., 703
Singer, J., 996
Singer, J. D., 939, 949
Singer, L. T., 147
Singer, M. T., 581
Singh, K., 1062
Singsaas, K., 642
Sinha, P., 286
Sinharay, S., 760
Sirois, S., 166
Sirotkin, Y. S., 293
Sisk, C. L., 805
Sitarenios, G., 28
Sjogreen, L., 275
Sjoquist, E. S., 798
Skaggs, N., 555
Skinner, B. F., 266, 400
Skinner, E. A., 293
Skinner, H. A., 606, 1032, 1071
Skomorovsky, A., 643
Skrondal, A., 798
Skuban, E. M., 2, 3, 36, 293
Slade, A., 339, 340, 362
Slade, E., 19
Slade, T., 636
Slatcher, R. B., 301
Slavich, G. M., 529, 538, 539, 540, 550
Slay, P. M., 251
Sleight, P., 1049, 1057
Slep, A. M. S., 199, 640, 650
Slipp, S., 585
Sloman, L., 242, 245, 247
Slopen, N., 286
Slough, N. M., 333
Sluckin, A., 158
Sluckin, W., 158
Smallish, L., 643
Smart, D., 532, 699

Smeeth, L., 145
Smetana, J. G., 387
Smith, A., 141, 558, 650
Smith, A. J., 497
Smith, C. A., 140
Smith, C. L., 278, 292, 295
Smith, D., 501
Smith, D. A., 892
Smith, D. J., 84
Smith, E. E., 58
Smith, G. D., 137, 1054
Smith, G. T., 268
Smith, H. A., 647
Smith, K., 532
Smith, K. D., 281, 282, 301, 304
Smith, K. W., 701
Smith, L., 448, 499, 501
Smith, L. B., 666, 702, 718, 719, 720, 721, 722, 723, 724, 726, 730, 738, 743, 744, 846
Smith, M., 288, 483
Smith, M. A., 235
Smith, N. J., 23
Smith, P., 291
Smith, P. B., 338
Smith, P. K., 545
Smith, R. E., 402
Smith, R. H., 500, 501
Smith, R. S., 610
Smith, S., 442, 493
Smith, S. M., 861
Smith, V., 334, 358
Smith-Donald, R., 281
Smith-Simon, K. E., 292
Smith Slep, A. M., 302, 593, 596, 597
Smits, P. A., 184
Smoll, F. L., 402
Smolla, N., 110
Smoller, J.W, 271, 695
Smyke, A. T., 178, 181
Smyrnios, K. X., 745
Snarey, J., 638
Snidman, N., 35, 531, 699, 705, 706
Snijders, T. A., 560, 561
Snijders, T. B., 552, 554, 560, 561
Snow, J., 96, 117
Snow, M. E., 275
Snyder, A. L., 691
Snyder, C. R., 474, 512, 516, 517
Snyder, J., 280, 294, 298, 302, 622, 724, 726, 731, 732, 734, 738, 746, 748, 749
Snyder, J. J., 546, 547, 563
Snyder, J. R., 229, 230
Soares, I., 359
Soares, J. J., 268
Sobel, M. E., 780, 929, 973
Sobral, D. T., 404
Soenens, B., 386, 387, 389, 390, 391, 392, 394, 395, 398, 403, 405, 406, 408, 410, 411, 412, 417, 418, 419, 421, 426, 428, 555
Sokal, E. M., 165
Sokol, B. W., 385
Sokol, R. J., 147
Solberg, M. E., 544
Solhan, M., 268
Solky-Butzel, J., 390, 399, 427
Solomon, J., 296, 330, 333, 334, 336
Solovitz, B. L., 78
Somerville, L. H., 293, 558
Sommerfeld, P., 707
Song, X. Y., 761
Sonntag, H., 636
Sontag, L. W., 175
Sonuga-Barke, E. J. S., 132, 139, 328, 686
Sörbom, D., 819, 853, 856, 956
Sorongon, A. G., 925
Sorrentino, R.M., 510
Soto, T. W., 10
Soussignan, R., 443, 450
Southam-Gerow, M. A., 268, 283, 285, 304
Spahr, A. J., 158
Spangler, G., 279, 291, 295, 296, 343, 347, 348, 354, 355
Spark, G., 583, 585, 589
Sparrevohn, R. M., 637, 652
Sparrow, S. S., 12, 13, 16, 36
Spaulding, W., 667
Spears, F. M., 296
Spector, F., 445, 446
Speirs, H. J., 167
Spelke, E. S., 453
Speltz, M. L., 3, 350
Speltz, M. T., 105
Spemann, H., 719
Spence, S. H., 3, 10, 14, 20, 28, 34, 537
Spencer, J. P., 666, 706, 743, 744
Spencer, T. J., 667, 706, 707
Spencer-Rodgers, J., 510
Speranza, A. M., 333
Spergel, I. A., 140
Sperling, J. B., 281
Sperling, R. A., 10
Sperry, L., 416, 423
Spicer, D. M., 158
Spieker, S. J., 331, 352
Spiel, C., 876, 891, 893
Spies, J. R., 1051
Spiess, H., 112
Spinath, F. M., 345
Spinrad, T. L., 223, 277, 281, 285, 295, 297
Spirato, A., 495
Spittle, A. J., 28
Spitz, R. A., 178
Spitzer, R. L., 53, 59, 60
Spitznagel, E., 1, 2, 3, 4, 36
Spivey, M. J., 734
Sporns, O., 289
Sportel, B., 699, 700
Spoth, R., 621
Spracklen, K. M., 541, 562, 563, 728
Sprenkle, D. H., 228, 589, 613
Spriggs, D. R., 15
Squires, J., 19, 20, 21
Sravish, A. V., 746
Srikameswaran, S., 498
Sroufe, L. A., xi, xii, 35, 104, 105, 112, 160, 161, 168, 169, 203, 208, 209, 237, 240, 241, 242, 274, 275, 291, 296, 325, 330, 346, 347, 348, 349, 350, 351, 353, 354, 359, 360, 367, 397, 424, 455, 529, 588, 589, 590, 597, 599, 600, 609, 618, 666, 700, 718, 724, 987, 988, 1006, 1048
Stabile, M., 924
Stadler, J., 84
Staelin, R., 761
Staff, J., 924
Stahl, E. A., 271
Stalets, M. M., 1, 3, 4
Stalker, C. A., 362
Stall, R. D., 647
Stam, H. J., 812, 813, 814
Standring, T., 515
Stange, J. P., 288
Stanger, C., 62, 63, 64, 65, 66, 72, 280
Stanley, C., 334, 358
Stansbury, K., 6
Stanton, W. R., 1005
Stanwood, G. D., 170
Staples, A. M., 268
Stapleton, L. M., 938
Star, S. A., 874, 1056
Stark, J. L., 164
Stark, K., 278, 304
Stark, K. D., 589
Stark, T. H., 565
Starr, L. R., 634, 635, 644, 651, 652
State, T. 687
Statman, D., 500
Staton, L., 220
Stattin, H., 96, 97, 113, 117, 144, 529, 552, 554, 555, 559, 565, 639
Staudinger, U. M., 160
Stawski, R. S., 937
Stayton, D. J., 336
Stazi, M. A., 345
St Clair, D., 142
Steele, H., 168, 341, 348, 358, 362, 368, 369
Steele, J. S., 268, 706, 994, 995, 1017
Steele, K., 424
Steele, M., 168, 348, 358, 362, 368, 369
Steenbeek, H. H., 666, 671, 684, 687, 689, 690, 691, 693, 694, 704, 726, 741, 742, 743
Steenkamp, J.-B. E. M., 1046
Steer, R. A., 69
Stefan, C. A., 3
Steffgen, G., 503
Stegge, H., 484, 487
Steglich, C. G., 552, 554, 560, 561
Stegun, I. A., 884, 885
Steiger, J. H., 941
Stein, A., 364, 365
Stein, A. D., 177
Stein, P., 822
Stein, Z. A., 116, 142, 675
Steinberg, K. K., 1044, 1065, 1067
Steinberg, L., 281, 285, 288, 289, 294, 298, 301, 302, 387, 389, 390, 391, 422, 557, 558, 564, 642
Steinberg, S. J., 632, 633, 634, 642, 652
Steiner, J. E., 450
Steinfield, C., 650
Steinglass, P., 591
Steinhauer, P. D., 606
Steinley, D., 829, 837
Stemerdink, N., 687
Stengers, I., 720
Stephen, D. G., 730
Stephens, A. D., 980
Stephenson, J. C., 246
Stephenson,W., 815, 816, 817
Stepp, S. D., 300
Sterba, S. K., 34, 799, 804, 805, 806, 809, 818, 822, 847, 849, 1047
Stern, E., 689
Stern, D., 444, 482
Stern, D. N., 388, 396
Stern, H. S., 760
Stern, L., 98, 110
Stern, W., 799, 812, 813, 814, 838
Sternberg, C., 461
Stettler, N. M., 297
Stevens, E. A., 559, 565
Stevens, K. I., 202, 207, 217, 229, 249
Stevens, S., 132

Stevenson, M. B., 397
Stevenson Hinde, J., 231
Stevenson-Hinde, J., 354
Stevens, W. L., 898
Stewart, E., 118
Stewart, L. A., 1046
Stewart, S. H., 298
Stewart, S. L., 531
Stey, P. C., 485, 487
Stice, E., 643, 654
Stifter, C. A., 288, 291, 295, 698
Stigler, J. W., 493
Stiles, W. B., 746
Stiller, J. D., 398
Stillman, P. E., 301
Stillman, S., 138
Stipek, D., 276, 392
Stirling, E., 497
St-Laurent, D., 225
Stockdale, G. D., 1017
Stocker, C., 490, 557
Stocker, C. M., 235
Stoeber, J., 289
Stolk, M. N., 368
Stolow, D., 300
Stoltz, E., 293
Stolz, H. E., 405, 590, 617
Stone, C., 688
Stone, G., 239, 240
Stone, K., 59
Stone, L. B., 297, 556
Stone, S., 924
Stoneman, Z., 6, 609
Stoolmiller, M., 136, 137, 298, 622, 638, 639, 640, 646, 654, 724, 726, 731, 732, 734, 738, 746, 749, 752, 959
Storch, E. A., 700
Stouffer, S. A., 874, 1056
Stouthamer-Loeber, M., 100, 101, 108, 421, 798
Stovall, K., 346
Stovall-McClough, K., 361, 367
Strachan, E., 667
Strait, L. B., 135
Stranger, B. E., 271
Strathearn, L., 363
Stratton, J., 785, 786
Straube, E., 890, 891, 892
Straus, M. A., 246
Strauss, C. C., 532
Strauss, J., 418, 419
Strauss, J. L., 751, 752
Strawderman, W. E., 1062
Strayhorn, J. M., 35
Striano, T., 275, 283, 452, 453, 455, 456, 457, 458, 459, 461
Stright, A. D., 6
Strober, M., 419
Strong, D. R., 1071
Stroud, C. B., 633, 634, 635, 651, 652
Stroup, W. W., 851, 996
Strupp, H. H., 799, 1026
Strycker, L. A., 943
Stucky, B. D., 544
Stukas, A. A., 486
Stump, K. N., 761
Stupica, B., 340, 359
Sturaro, C., 535
Sturge-Apple, M. L., 164, 168, 201, 204, 205, 206, 207, 208, 209, 211, 213, 214, 215, 216, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 229, 230, 231, 234, 237, 238, 239, 240, 241, 242, 243, 244, 245, 246, 247, 249, 250, 251, 297, 621, 808
Sturman, E. D., 494
Stutz, M., 231, 287
Stutzman, S. V., 233
Styers, M. K., 278
Su, X., 203
Su, Y., 771
Subak, L., 1050
Subrahmanyam, K., 650
Subramanian, S. V., 800, 801, 802, 803
Substance Abuse Mental Health Services Administration (SAMHSA), 1047
Sucato, G., 647
Suchman, E. A., 874, 1056
Suci, G. J., 673
Sudo, N., 170
Sue, S., 493
Suess, G., 333, 347, 353
Suess, P. E., 249
Suh, E., 510
Sullivan, H. S., 553, 673, 679
Sullivan, J., 3
Sullivan, M. W., 276, 280, 286, 294, 302
Sullivan, R. M., 164, 553
Sullivan, T. N., 223
Suls, J., 852
Sumter, S. R., 545
Sundar, S. S., 497
Sundby, J., 642
Sunderani, S., 707
Sung, M., 121
Suomi, S. J., 99, 158, 178, 350
Suomi, S. S., 178
Suor, J.H., 621
Supattathum, S., 876
Supplee, L. H., 293, 1043
Sur, M., 166
Surette, D. A., 1056
Susser, E. S., 118, 130, 131, 142, 157, 170, 177
Susser, M., 94, 95, 116, 802
Sutherland, K. S. 691
Sutton, A. J., 1044, 1046, 1060, 1062, 1067
Sutton, D. B., 172
Suveg, C., 279, 297, 304
Suzuki, K., 165
Svetlova, M., 277
Swain, J. E., 223, 364
Swann, W. B., Jr., 492
Swanson, C. C., 726, 735, 736, 751
Swanson, D. P., 428
Swanson, J. A., 588, 589, 590, 617, 619, 620
Swanson, J. M., 164
Swanson, K. R., 726, 736, 751
Swarr, A., 632
Swearingen, L., 295
Swed, F. S., 898
Swedo, S. E., 165
Sweeney, L., 32, 34
Sweeting, H., 296
Swenson, L., 218, 219
Swisher, L., 286
Syme, S. L., 138
Symons, D. K., 358
Szabó, M., 289
Szajnberg, N. M., 275
Szapocznik, J., 609
Szatmari, P., 667
Szeminska, A., 676
Szyf, M., 168, 540
Szymanski, D. M., 647
Tabak, L. A., 1080
Tabares, A. A., 608
Tabibnia, G., 280
Tackett, J. L., xi, 24, 38
Tafarodi, R., 510
Tager-Flusberg, H., 275
Tal, K., 416
Talati, A., 121
Talge, N. M., 176
Talwar, V., 280, 406
Tan, P. Z., 276, 292, 293, 294
Tanaka, K., 399, 404
Tanaka, M., 170
Tananberg-Karant, M., 892
Tandon, M., 1, 3, 4
Tang, W., 175
Tangney, J. P., 500, 501, 512, 696
Tankersley, L., 225, 235
Tannenbaum, P., 673
Tanner, J. L., 603
Tanner, J. M., 112
Tanner, M. A., 770
Tannock, R., 687
Tarabulsy, G. M., 366
Taradash, A., 639
Tardif, C., 349
Target, M., 345, 362, 368
Tarquinio, C., 499
Tarullo, A. R., 178, 286
Task Force on Statistical Inference, 782, 784
Tasman, A., 903
Tassinary, L. G., 103
Tau, G. Z., 166
Taumoepeau, M., 277
Tavecchio, L., 346
Tavory, I., 672
Taylor, A., 420
Taylor, A. B., 778
Taylor, B. J., 784, 788, 789
Taylor, C., 440, 463
Taylor, C. S., 812
Taylor, C. T., 223
Taylor, J., 605
Taylor, J. R., 388
Taylor, K. A., 223
Taylor, R. D., 234
Taylor, S. E., 170, 550
Taylor, V. H., 170
Taylor, Z. E., 234
Tchanturia, K., 294
Tchetgen, E. J. T., 145
Teague, G. B., 360, 363, 368, 369
Team, E-Risk, Study, 130
Tegethoff, M., 176
Teicher, M. H., 169
Telch, M. J., 545
Tell, R. A., 1051
Tellegen, A., 247, 858
Telzer, E. H., 539, 553, 554
Temes, C. M., 367
Temple, B. W., 641
Temple, J. A., 180, 181
Temple, J. R., 650
Ten Cate, T. J., 404
Tenenbaum, L., 294, 502
Ten Have, T. R., 973
Tereno, S., 359
Terr, L. C., 673, 677, 678
Terracciano, S. M., 366
Terranova, A.M., 699
Terry, A. V., Jr., 175
Terry, D., 581

Terry, R., 535, 537
Tesher, H. B., 164
Teşileanu, T., 734
Tesman, J., 274, 276
Tesser, A., 745
Teten, A. L., 638
Teti, D. M., 342
Teti, L. O. D., 268, 278, 280, 282, 298, 300
Tetzlaff, J., 1052
Thaler, N. S., 8
Thalib, L., 1061
Thapar, A., 137, 176
Thelen, E., 448, 666, 696, 702, 718, 719, 720, 721, 722, 723, 724, 725, 726, 730, 738, 743, 744, 846
Théoret, H., 447
Theran, S. A., 833
Theriault, J., 643
Thibodeaux, S., 732
Thiel, M., 729
Thij, H., 775
Thisted, R. A., 976, 977
Thistlethwaite, D. L., 146
Thoemmes, F., 130
Thoennes, N., 638
Thom, R., 744
Thomaes, S., 484, 487
Thomas, A., 131, 184
Thomas, J. M., 4, 7
Thomas, K. M., 706
Thomas, R., 293
Thomason, M. E., 271, 302
Thomas-Peter, B., 696
Thomassin, K., 297
Thompson, A., 1069
Thompson, J. R., 145
Thompson, K. M., 643
Thompson, L. A., 172
Thompson, M., 666, 701
Thompson, M. P., 201, 224
Thompson, N. J., 798
Thompson, R. A., 284, 290, 301, 347, 350, 358, 359, 475, 482, 516
Thompson, R. J., 547
Thompson, S. G., 784, 1050, 1060, 1063
Thompson-Schill, S. L., 169
Thomsen, L., 453
Thomson, H., 87
Thornberry, T. P., 140
Thornburg, K. L., 164
Thornton, L. C., 268, 699
Thorpe, K., 139
Thrash, T. M., 408
Thurner, S., 177
Thurstone, L. L., 857
Tian, L., 1049, 1057, 1061, 1062
Tibishirani, R. J., 781, 930, 979
Tice, D. M., 221
Tick, N. T., 85
Tickle-Degnen, L., 281
Tiemeier, H., 74, 177
Tilton-Weaver, L. C., 555
Timberlake, D. S., 565
Ting, A. H., 164
Tingley, D., 973, 975
Tinklenberg, J., 1051
Tisak, J., 851, 958, 959, 989, 994, 995
Tix, A. P., 923
Tizard, B., 172
Tjaden, P., 638
Tluczek, A., 5, 6, 7, 36
To, Y. M., 284
Tobi, E. W., 142
Tobin, K., 140, 145
Tobin, M. D., 145
Tobin, R. M., 281
Tobin, V. J., 404, 426
Tochluk, S., 635
Toda, H., 177
Todd, R., 458
Todd, R. D., 24, 38
Tofail, F., 182
Tofighi, D., 930, 937
Toh, S., 977
Tolan, P. H., 4, 32, 105, 698
Tollenaar, M. S., 172
Tolley-Schell, S., 218
Tolson, T. F., 5
Tolstoy, L., 801
Tomasello, M., 284, 400, 428, 452, 455, 456, 457, 459, 460, 461
Tomasi, D., 687
Tomasian, J. C., 280
Tomaszycki, M., 174
Tomkiewicz, S., 136, 182, 669
Tomkins, S. S., 752
Tomlinson, M., 340
Tononi, G., 448
Tooby, J., 242
Toomela, A., 803, 812
Torchetti, L., 486, 487
Törestad, B., 801, 803, 804, 805
Torres, W. J., 500
Toth, S. L., xi, 38, 105, 168, 169, 171, 200, 204, 209, 217, 226, 232, 241, 286, 351, 366, 368, 386, 387, 424, 488, 599, 613, 666, 673, 678, 680, 847, 862, 917, 988, 1047
Tottenham, N., 179, 298, 363
Tourangeau, K., 925
Towe-Goodman, N. R., 808, 839
Tracy, E. R., 734
Tracy, J. L., 487, 500
Træen, B., 642
Tragesser, S. L., 268
Tran, S., 274
Trancik, A., 294
Tranel, D., 388
Trapolini, T., 358
Trauner, D. A., 275
Trause, M. A., 162
Trautwein, U., 402
Treasure, J., 294
Treboux, D., 169, 296, 347, 349, 362
Treffers, P. D. A., 16, 109
Tremblay, R. E., 104, 108, 131, 132, 544, 564, 622, 638, 698, 699, 740
Trenado, R. M., 746
Trentacosta, C. J., 285, 293, 294, 301, 304, 350
Trevarthan, W. R., 451, 459
Trevarthen, C., 455, 456
Trickett, P. K., 500, 677, 704
Trivedi, S. S., 589
Trofimova, I., 494
Tronick, E. Z., 5, 6, 7, 288, 291, 452, 455, 746
Troop-Gordon, W., 547, 549, 1007
Trost, K., 809, 857
Trout, A., 687
Trouton, A., 345
Trower, P., 244
True, M. M., 340
Trull, T. J., 268, 302
Trumbull, D., 501
Truong, N., 646
Trzesniewski, K. H., 482
Tsai, J. L., 335
Tsai, L., 272
Tsai, Y., 402
Tschacher, W., 737, 752
Tschann, J. M., 234, 649
Tse, T., 1049
Tseng, V., 702
Tseng, W. L., 925
Tsuang, M. T., 59, 60, 78
Tsui, A. S., 493
Tucker, D., 988
Tucker, L. R., 851, 994, 995
Tucker-Drob, E., 786
Tuckey, M., 339
Tufis, P. A., 10
Tull, M. T., 297
Turkewitz, G., 446
Turkheimer, E., 555, 565
Turnbull, A. V., 219
Turner, E. H., 1048, 1051
Turner, H. A., 38, 494
Turner, K. M., 367
Turner, R. J., 113
Turner, S. M., 232, 636
Turner, S. P., 131
Turner, T. M., 541
Turpin, R., 51
Tversky, A., 1063
Tweedie, R., 1054
Twenge, J. M., 422, 487
Twombly, E., 19, 20, 21
Tyrrell, C. L., 360, 361, 363, 368, 369
Tyson, R., 726, 736, 751

Uchida, N., 203
Uchino, B., 296
Uckert, K., 558
Udry, J. R., 631, 633, 636
Ueno, K., 646
Uher, R., 344
Uhrlass, D. J., 297, 298, 556
Ujie, T., 203
Ulbricht, J. A., 621
Ulrich, B. D., 718, 722, 723, 725, 726, 738
Underwood, M. K., 537, 541, 544, 645
Ungerer, J. A., 32, 34
Uniform Crime Reports, 638, 641
U.S. Census Bureau, 233
U.S. Department of Education, National Center for Education Statistics, 924
U.S. Department of Health Human Services, Administration on children, youth families, 285
U.S. Department of Labor, Bureau of Labor Statistics, 992, 1009
Upton, K. J., 164
Urban, J., 359
Urberg, K. A., 565
Urdinguio, R. G., 168
Uriburu, D., 233
Ursache, A., 251, 698
Usher, B. A., 279, 281
Üstün, T. B., 116
Utendale, W. T., 667

Vader, A. M., 1069
Vaillancourt, T., 546, 667, 707
Vaillant, G. E., 677
Vaish, A., 284
Valencia, M. E., 138
Valente, E., 536
Valente, M. J., 939

Valente, T. W., 565
Valentino, K., 390, 673
Valeri, L., 973, 978
Valiente, C., 293
Valkenburg, P. M., 545
Valla, J.-P., 78, 110
Vallacher, R. R., 726
Valle, A. K., 642
Valle, L. A., 251
Vallerand, R. J., 390, 404, 410, 417
Vallotton, C., 277
Valsiner, J., 810
van, 38
Van Abbema, D. L., 278
Van Assche, J., 428
van Bakel, H. J., 356
van Beijsterveldt, T. C. E. M., 75
van Belle, J., 687
van Buuren, S., 760, 770, 771, 772, 775, 776
van de Bunt, G. G., 552, 560
Vandell, D. L., 530
Vandenberg, R. J., 856
Van den Bergh, B. R., 160
van den Boom, D., 291, 341, 740
Van den Broeck, A., 416
van de Pol, F., 833
Van der Aalsvoort, G., 689
Van Derbur Atler, M., 673
van der Ende, J. R. A., 50, 53, 71, 72, 73, 74, 85, 109, 668
Vandereycken, W., 417, 419
van der Giessen, D., 726, 746
Van der Hart, O., 424
van der Horst, F. C., 326
Van der Kaap-Deeder, J., 417, 425
van der Kamp, J., 745
van der Kloet, D., 424
van der Maas, H., 666, 722, 726, 740, 745, 822
Van Der Meer, E., 221
van der Meere, J., 686, 687
Vanderminden, J., 38
van der Valk, I., 235
van der Veer, R., 326, 356
Van der Voort, A., 335
VanderWeele, T. J., 973, 974, 975, 976, 977, 978
van der Werff, S. J. A., 286
Vanderwert, R. E., 179
van der Zwaluw, C. S., 565, 605
van Dijk, A., 1063
van Dijk, M., 722, 723, 724, 726, 730, 738, 739, 740, 746
van Dijk, S., 687, 694
Van Dijken, S., 326
Van Dillen, L. F., 295
van Dulmen, M. H., 173
van Eijk, L. T., 174
van Eldik, T., 109
van Engeland, H., 698
van Geert, P., 666, 671, 684, 685, 687, 688, 689, 690, 696, 704, 706, 707, 718, 719, 722, 723, 724, 726, 730, 738, 739, 740, 741, 742, 743, 745, 746, 751
van Geert, P. L., 703, 704
van Geert, P. P., 684, 691, 693, 694
van Gelder, T., 721
Vanhalst, J., 532
van Harmelen, A. L., 286
van Honk, J., 363
Van Hulle, C. A., 35
Van IJzendoorn, H. W., 535
van IJzendoorn, M. H., xi, 167, 168, 173, 223, 231, 247, 271, 291, 295, 296, 297, 328, 329, 331, 333, 334, 335, 336, 337, 338, 339, 340, 341, 342, 343, 345, 346, 347, 348, 349, 350, 351, 352, 353, 354, 356, 360, 362, 365, 366, 367, 368, 369
Van Keer, H., 404
Van Landingham, M. J., 138
Van Lange, P. A., 295
van Lier, P. A., 535, 538, 546, 565
Van Lieshout, C. F., 535
van Lil, K., 515
Van Luit, J. E., 691
Van Meir, C., 174
van Meurs, I., 50, 73, 74
Van Oort, F., 73
van Orden, G. C., 730, 751
van Os, J., 118, 666
Van Petegem, S., 389, 390, 395, 406, 408, 410, 421, 428
van Rijn, H., 740
van Rijn, P. W., 826
Van Ryzin, M. J., 348
Van Someren, M., 740
Vansteelandt, S., 973, 974, 975, 977
Vansteenkiste, M., 386, 388, 389, 390, 391, 392, 395, 398, 400, 402, 403, 404, 405, 406, 407, 408, 410, 411, 412, 414, 417, 418, 419, 421, 425, 426, 427, 428, 555
van't Hoff, M. A., 61
van Velzer, C., 446
van Widenfelt, 16
van Workum, N., 552, 559
Van Zalk, M. W., 552, 554, 561
Van Zeijl, J., 365
Varadhan, R., 1079
Vargha, A., 707
Varjas, K., 294, 502
Vartanian, L., 497
Vaschillo, E., 822, 917, 918, 1056
Vasen, A., 280
Vasilenko, S. A., 642
Vassend, O., 798
Vaughan, J., 698
Vaughn, B. E., 165, 331, 335, 341, 342, 347, 542, 543
Vazquez, D. M., 179
Vedeler, D., 805
Veenstra, R., 561
Veerman, J. W., 109, 536
Velders, F. P., 355
Velting, D., 118
Verbeke, G., 775
Vereijken, C. M., 333, 338, 339
Verhage, M. L., 336
Verhulst, F. C., 50, 53, 62, 63, 64, 65, 71, 72, 73, 74, 85, 109, 668
Verissimo, M., 335
Vermulst, A. A., 564
Vermunt, J. K., 822, 906
Vernberg, E. M., 637
Vernon-Feagans, L. L., 268, 296
Veronese, G., 487
Verrips, G. H., 177
Verschueren, K., 362, 393, 398
Verstuyf, J., 417, 419
Vervoort, E., 398
Vetere, A., 719
Vézina, J., 649
Viding, E., 297, 345, 699
Viechtbauer, W., 1079
Vignoles, V. L., 410, 589
Vilhena-Churchill, N., 298
Vine, V., 268
Vink, J. M., 134
Vinogradov, S., 689
Virk, J., 176
Visher, C. A., 638
Visser, G. H., 272
Visser, J. C., 28
Visser, M., 703, 704
Vitaro, F., 133, 537, 541, 543, 544, 546, 547, 551, 552, 553, 564, 565, 638, 698, 699
Vitenberg, E., 361
Voegtline, K., 698
Voelkle, M. C., 847, 956
Voeten, M., 568
Vogel, D., 240
Vohs, K. D., 221
Volkmann, J. R., 637, 652
Volkmar, F., 15
Volkovich, Z., 829
Volkow, N. D., 687
Volling, B. L., 246, 295, 296, 358
von Bertalanffy, L., 583, 586, 591, 666, 718, 735
Vondra, J. I., 295, 351, 678, 679
von Eye, A., 224, 291, 668, 706, 800, 802, 803, 804, 805, 806, 807, 808, 812, 818, 819, 820, 821, 822, 823, 824, 825, 826, 827, 828, 829, 830, 831, 832, 833, 837, 838, 866, 867, 868, 869, 870, 871, 872, 874, 876, 877, 878, 879, 881, 882, 883, 884, 885, 886, 887, 890, 891, 892, 893, 894, 895, 898, 899, 901, 902, 906, 908, 909, 910, 913, 915, 918
von Eye, M., 891, 892, 901
von Gontard, A., 106, 107
von Hippel, P. T., 774, 775
Vonhold, S. E., 225, 226, 227, 234
von Klitzing, K., 2, 34
von Soest, T., 642
von Weber, S., 876, 902
Vorria, P., 178
Vos, C. M. P., 404
Voss, H. L., 639
Vostanis, P., 502
Votruba-Drzal, E., 6
Vuchinich, R. E., 1069
Vuga, M., 359
Vujeva, H. M., 633, 635
Vuong, Q. H., 1029
Vygotsky, L. S., 6, 278, 527, 553

Wacholder, S., 785
Waddington, C. H., 161
Wadhwa, P. D., 164, 177
Wadsworth, M. E., 73, 232, 293
Waffarn, F., 290, 294
Wagenmakers, E. J., 1043
Wagmiller, R. L., 7
Wagner, B. M., 580, 588, 591, 592, 601
Wagner, E., 461
Wagner, P., 696
Waheed, W., 8
Wakschlag, L. S., 3, 4, 6, 7, 10, 24, 29, 32, 34, 35, 37, 134, 240, 605, 667, 698
Wald, A., 898
Walden, K., 815
Waldinger, R. J., 633
Waldman, I. D., 61
Waldorp, L. J., 666, 667
Waldron, H., 584
Walker, H. M., 695
Walker, J., 501
Walker, J. L., 59, 60, 78

Walker, L. E., 641
Wall, M. M., 972
Wall, S., 201, 206, 328, 329, 332, 336, 367, 396
Wallace, C. S., 157, 162
Wallace, H. M., 486
Wallbott, H. G., 679
Waller, E. M., 556
Waller, N. G., 345, 379
Wallerstein, J. S., 162
Wallis, W. A., 898
Wallort, S., 730, 751
Walls, T. A., 820, 852
Walsh, B., 296
Walsh, F., 597, 598, 599
Walsh, V., 447
Walter, A., 275
Walter, J., 168
Walters, E. E., 633, 644
Walters, M. G., 601, 615
Walters, S. T., 1069, 1079
Wamboldt, F. S., 593, 596, 597
Wamboldt, M. Z., 593
Wampler, K. S., 746, 751
Wampler, R., 232
Wang, L.,673
Wang, J., 746, 749
Wang, L., 163, 174, 493, 510, 532, 673, 781, 852, 991
Wang, M., 29, 290, 294
Wang, Q., 489
Wang, Y., 174, 414, 489, 493
Wang, Z., 174
Wångby, M., 541, 798
Wanner, B., 541, 552
Warburton, W., 486
Ward, M. J., 342, 590, 617
Ware, H., 67, 850, 935
Ware, J. H., 996, 1000
Wargo, J. B., 535
Wargo Aikins, J., 648, 649
Warneken, F., 400
Warren, H. K., 288
Warren, M., 620
Warren, S. L., 35, 700
Warrens, M. J., 829
Warsi, G., 929
Wartner, U. G., 333, 347
Waschbusch, D. A., 301, 536, 667
Washburn, I. J., 638
Wasserman, M. S., 115
Wasserstein, S. B., 235, 553
Waters, E., 169, 201, 206, 284, 296, 328, 329, 330, 331, 332, 333, 335, 336, 346, 347, 348, 349, 354, 367, 396, 397, 542, 543, 679
Waters, H. S., 334, 335
Waters, P., 487, 491
Waters, S. F., 284
Watkins, D., 494
Watkins, E. R., 269
Watkins, M. W., 925
Watson, A., 748
Watson, C. M., 633, 634
Watson, D., 513, 858
Watson, J. B., 54
Watson, J. S., 288, 455, 456, 457
Watson, M. W., 676, 701
Watt, N. F., 50
Watzlawick, P., 582, 586, 591
Waugh, C. E., 269
Way, B. M., 170, 550
Wayland, K. K., 535
Wayment, H. A., 511, 512
Weakland, J. H., 582, 586, 591
Weaver, I. C. 672
Weaver, I. C., 168, 350, 354, 672
Weaver, I. G., 540
Weaver, W., 736
Webber, C. L., Jr., 729, 730
Webster, J. M., 501
Webster-Stratton, C., 181, 365
Wechsler, D., 1022
Weeks, M., 532
Wehby, J. H., 687, 688
Wehmeyer, M. L., 516
Wehner, E. A., 632
Wei, X., 696
Weigard, A., 558
Weinberg, C. R., 131
Weinberg, S., 10
Weinfield, N. S., 169, 296, 346, 347, 348, 350
Weinraub, M., 330, 337
Weinstein, N., 387, 388, 400, 403, 408, 409, 412, 415, 425
Weinstein, S. R., 59
Weir, J. M., 699
Weiss, B., 293, 1048
Weiss, L. A., 393, 394, 406
Weiss, P., xi
Weiss, R. B., 184
Weiss, R. L., 607, 611
Weiss, S. R., 166
Weisshaar, D., 590, 603
Weissman, A. N., 1026
Weissman, M. M., 121
Weisz, J. R., 55, 78, 87, 109, 654
Weitzman, C., 2
Wekerle, C., 298
Welch, E., 497
Weller, J. E., 643
Wellman, H. M., 276, 330, 443, 453
Wells, K. B., 18
Welsh, D. P., 228, 632, 634, 635, 647, 652
Welsh, J. D., 279, 281
Wen, Z., 964, 967, 972
Wenar, C., 588, 593, 598
Wentzel, K. R., 565
Wenzel, A., 637
Werch, C. E., 1070
Werner, E. A., 272
Werner, E. E., 610
Werner, H., 385
Werner, N. E., 537
Werry, J. S., 56
Werthamer-Larsson, L., 35, 118, 1034
Wertheim, E. H., 498
Wessler, R., 800
West, M., 362
West, P., 296
West, S. G., 130, 778, 782, 923, 926, 927, 928, 929, 930, 972, 973, 997, 1024
Westen, D., 423, 424, 488, 678, 679
Westerlund, A., 286
Westers, P., 404
Westmacott, M., 767
Weston, D. R., 347
Westphal, M., 752
West-Stroming, D., 707, 803
Wethington, E., 224, 548, 588
Weusten, J., 687
Whalen, P., 363
Whaley, G. J., 348
Whaley, R. B., 622
Wheatley, K., 145
Wheeler, K., 172
Wheeler, L., 1034
Wheelwright, S., 284
Whiffin, V. E., 418
Whipple, N., 352, 387, 388, 396, 397, 428
Whisman, M. A., 597, 621
Whitaker, D. J., 654
White, C. R., 230, 250, 297
White, D. R., 541
White, H. R., 650, 1047, 1049, 1069, 1071, 1074
White, I. R., 774, 782, 784, 1050, 1060, 1062, 1063, 1066
White, K. M., 608
White, R., 399, 504, 505
White, R. K., 815
White, S. F., 699
Whitesell, N. R., 284, 472, 475, 486, 487, 491, 494, 495, 499, 501, 508
Whitson, S. A., 248
Wicherts, J. M., 1063
Wichstrøm, L., 2, 667
Wickens, T. D., 736, 737
Wickman, E. K., 108
Wickrama, K. A. S., 70
Widaman, K. F., 234, 789, 809, 810, 856, 991, 1017
Widen, S. C., 284
Widman, L., 632
Widom, C. S., 650
Wieder, S., 463
Wiedermann, W., 866, 918
Wiersema, J. R., 686
Wiesel, T. N., 158
Wiki, T., 605
Wilbarger, J. L., 275
Wilde, E.T., 148
Wilder, R. L., 174
Wilens, T. E., 78
Wiley, D. E., 942
Wiley, J. A., 942
Wilk, A. I., 1058
Wilkinson, L., 782, 784
Wilkinson, R. G., 116, 134
Willard, N. E., 650
Willems, P., 565
Willett, J. B., 678, 740, 923, 939, 949, 958, 959, 995, 996
Willheim, E., 7
Williams, G. C., 404, 425, 428
Williams, G. M., 3
Williams, J., 781, 923, 930, 978
Williams, J. C. P., 106
Williams, K., 285, 502
Williams, K. D., 550
Williams, K. R., 223
Williams, L. E., 292
Williams, P. G., 118
Williams, R. J., 1049
Williams, R. M., Jr., 874, 1056
Williams, S., 632, 635
Williams, V., 632
Willoughby, M., 121, 167
Willoughby, M. T., 1004
Wills, T. A., 553, 1008
Wilson, A., 483
Wilson, A. C., 294
Wilson, B., 726, 731, 732
Wilson, C. A., 175
Wilson, D. B., 118, 839
Wilson, I. M., 401
Wilson, J., 233, 234, 638
Wilson, K. S., 530

Wilson, L. C., 698
Wilson, M. N., 5, 234
Wilson, P., 367
Wilson, S., 293
Wilson, T. D., 213
Wimer, C., 141, 809
Wimmers, R. H., 745
Windle, M., 231, 240
Wing, J. K., 97
Wing, R., 268
Wingert, F., 441
Wingfield, J. C., 164, 219
Winkens, B., 961
Winkielman, P., 275, 689
Winn, D. C., 645
Winn, D. M., 605
Winn, L. L., 6
Winnicott, D. W., 163, 385, 397, 415
Winship, C., 980
Winsler, A., 278
Winstok, Z., 499
Winter, C. E., 666, 726, 736, 737, 746
Winter, M. A., 172, 176, 207, 208, 215, 217, 218, 219, 220, 221, 222, 225, 228, 229, 237, 238, 240, 247, 249, 250, 251, 296, 587, 590, 599, 614
Wippman, J., 397
Wirsing, M., 829, 832
Wirth, M. M., 174
Wirtz, C. M., 300
Wirtz, D., 489
Wisco, B. E., 268
Wise, S., 291, 452, 455
Wish, E., 104
Wishart, D., 832
Wishart, J., 995
Wismer Fries, A. B., 179
Wissow, L. S., 19
Witherington, D., 265, 500, 512, 666, 669, 696, 706, 719, 804
Wittchen, H. U., 636
Wittig, B. A., 329, 332
Wohlwill, J. F., 790, 993
Woitach, M. J., 204, 209, 214, 217, 220, 222, 223, 251
Wojslawowicz, J. C., 235, 528, 532
Wolf, C., 672
Wolf, D. P., 483
Wolf, S. A., 165
Wolfe, B., 924
Wolfe, D. A., 654
Wolff, P. H., 446, 455
Wolfinger, R. D., 851, 996
Wolfowitz, J., 898
Wolke, D., 547
Wolkind, S. N., 178
Wolman, B. B., 84
Wolraich, M. L., 593
Wolski, K., 1049, 1058
Woltering, S., 746
Wonderlich, S. A., 643
Wong, C. C. Y., 134
Wong, C. F., 295, 298, 301
Wong, M. S., 359
Wong, N., 396
Wong, W. H., 770
Wood, D., 688
Wood, A. M., 774, 782, 784
Wood, G. A., 632
Wood, J., 301
Wood, M. D., 1056, 1069, 1070, 1073
Wood, P., 858
Woodbury, M. A., 767, 775, 777
Woodcock, J. R., 990
Woodcock, R. W., 789
Woodham, A., 8
Woodhouse, S. S., 340, 358
Woodin, E. M., 618
Woodman, T., 497
Woodruff-Burton, H., 498
Woods, E. R., 645
Woodward, A. L., 453
Woodward, L., 115
Woodward, L. J., 537, 640
Woodward, S., 706
Woody, E., 706
Woolf, S. H., 1080
Woolgar, M., 169
Woolston, J., 118
Wootton, B., 131
Workman, J., 532
World Health Organization, 36, 51, 118, 594
Wörmann, V., 275
Worthman, C. M., 103, 112
Worthman, C. W., 99
Wothke, W., 761, 971, 972
Wouters, S., 393
Wray, N. R., 165
Wright, J., 555
Wright, K., 696
Wright, P., 556
Wright, R. J., 166
Wright, S., 852
Wright, S. S., 997
Wrightman, J., 394, 406
Wu, E. J. C., 956
Wu, W., 778, 781, 784, 786, 790
Wu, Y. T., 28
Wubbels, T., 746
Wulf, G., 401
Wundt, W., 454, 673
Wunsch-Hitzig, R., 108
Wurf, E., 563
Wurpts, I. C., 979
Wust, S., 173
Wuyts, D., 395, 405
Wylie, R. C., 471
Wymbs, B., 643
Wynn, J. K., 269
Wynn, S. R., 402
Wynne, L. C., 581, 586, 588, 591, 592, 595, 599

Xie, H., 116
Xie, M., 918, 1049, 1055, 1056, 1058, 1061, 1062, 1065, 1067
Xin, Z., 235
Xing, G., 184

Yacko, H., 409
Yaeger, A. M., 1008
Yajima, M., 771
Yamaguchi, S., 510
Yamaguchi, Y., 1044
Yamamoto, M., 726, 731, 732
Yamamoto, T., 973
Yamauchi, H., 399, 404
Yan, F., 274
Yang, G., 1062
Yang, T. T., 363
Yap, M. B., 294, 297
Yarrow, L. J., 397, 399
Yasui, M., 9
Yasunaga, S., 510
Yates, T. M., 287, 424, 666
Yehuda, R., 177
Yesavage, J. A., 1051
Yingling, L. C., 595
Yirmiya, N., 275
Yirmiya, R., 165
Yoerger, K., 542, 638, 640
Yoneda, A., 652
Yoo, J. E., 767, 776
Yoo, S. H., 513
Yoshikawa, H., 180, 704
Young, A. W., 283, 296
Young, J. F., 654
Young, L. D., 178
Young, L. J., 174
Young, S., 696
Youngblade, L., 235
Younger, A. J., 532
Young-Masten, I., 108
Youngstrom, E., 228, 232
Youngstrom, E. A., 350, 925
Yourman, J., 108
Yu, G., 235
Yu, J. W., 10
Yu, K. K., 1050
Yu, L.-M., 1079
Yuan, K.-H., 767, 775, 780, 782
Yuan, Y., 939, 978
Yucel, R. M., 775
Yuh, J., 132
Yun-Dai, 688
Yurgelun-Todd, D. A., 364
Yusuf, S., 1049, 1057
Yuwiler, A., 84
Yzerbyt, V. Y., 931

Zabrack, M. L., 148
Zachar, P., 57, 667
Zahavi, D., 457, 464
Zahn-Waxler, C., 206, 276, 277, 278, 279, 280, 281, 282, 284, 287, 298, 300, 301, 304, 461, 698
Zaitsoff, S. L., 498
Zajonc, R. B., 496
Zak, P. J., 222
Zale, E., 220
Zalewski, M., 294
Zaman, W., 599
Zamboanga, B. L., 1069
Zanarini, M. C., 423
Zarbatany, L., 552, 565
Zarin, D. A., 1049
Zarit, S., 993
Zax, M., 395, 413, 421, 422
Zbilut, J. P., 729, 730
Zeanah, C. H., 3, 21, 23, 161, 162, 178, 179, 181, 182, 225, 286, 298, 328, 367, 833
Zeger, S. L., 116
Zegers, M. A., 368, 369
Zeisel, S.A., 296
Zelazo, P. D., 454, 458
Zeldman, A., 425
Zelenko, M., 354
Zeman, J., 279, 281, 284, 293, 294, 298
Zero to Three, 594
Zeskind, P. S., 275
Zettergren, P., 541
Zhang, L., 18
Zhang, Q., 104
Zhang, S., 878, 918, 980
Zhang, W., 696
Zhang, Y., 292

Zhang, Z., 781, 937, 939, 968, 991, 996, 1015, 1016, 1017
Zhao, J., 174
Zhao, X., 922, 929
Zheng, C., 1050
Zheng, F. M., 493
Zhong, S., 344
Zhong, W., 975, 977
Zhou, E. S., 806
Zhou, M., 170
Zhou, Q., 414
Ziaie, H., 291
Zickuhr, K., 650
Ziegenhain, U., 333
Ziegler, T. E., 179
Zijl, R. H., 172
Zikopoulos, B., 689
Zill, N., 1009
Zimet, D. M., 213
Zimmer, C., 963
Zimmer-Gembeck, M. J., 293, 632
Zimmerman, D. W, 878
Zimmerman, S., 722, 724, 725, 726, 746, 750
Zimmermann, B., 296
Zimmermann, P., 279, 293, 296, 343, 347, 348
Zimmermann, R. R., 327
Zimowski, M., 67, 959
Zinsser, K., 293
Zir, Y., 351, 352, 397
Zisook, S., 115
Zohsel, K., 177
Zucker, R. A., 1048, 1053, 1055
Zuckerman, M., 231, 408
Zuena, A. R., 174
Zukin, S. R., 450
Zupan, B. A., 718
Zuroff, D. C., 386, 425, 1026
Zurowski, B., 363
Zwierzynska, K., 547
Zyphur, M. J., 937, 939, 968

Subject Index

Page numbers followed by f and t refer to figures and tables, respectively.

AAI. *See* Adult Attachment Interview
AAP. *See* Adult Attachment Projective
ABAS-II. *See* Adaptive Behavior Assessment System
Abnormal behavior, normal behavior (contrast), 108
Abnormality, viewpoint, 105
Abstractions, 674
Abuse, impact, 678–679
Abusive relationships, 679
Accelerated longitudinal design, cohort-sequential data set conversion, 789t
Acculturation, 508–509
Achenbach System of Empirically Based Assessment (ASEBA), 65, 109
  age-appropriate instruments, completion, 73
  forms, developmental trajectory analysis, 71
  instruments, 72
    age-appropriate versions, 73
    mean Total Problems scores, comparisons, 76
    usage, 74
    versions, 75
  measure, 353
  problem items, syndromes (presence), 86
  syndromes
    CFA support, 76
    derivation, 75–76
Acting structures, order/variability/disorder (self-organization), 704–707
Action
  adaptive/maladaptive forms, self-organization, 671–672
  readiness, 287
    patterns, 297–298
  structure, developmental changes. *See* Skilled action.
  systems, 449
  tendencies, 670–671
Active isolation, 530–531
Active splitting, dissociation (relationship), 677–678
Actor-partner interdependence model (APIM), extension, 610–611
AD. *See* Aggregated data
Adaptive Behavior Assessment System (ABAS-II), 36
Adaptive behavior, structure (epigenesis), 672–673
Adaptive calibration models, 805
Adaptive conflict, 705–706
Adaptive patterns, development pathways, 696–701. *See also* Children
Adaptive socioemotional adjustment, dynamics, 696
ADCLUS, 828
ADD Health data, analyses, 633
Additive effect model, 1008
ADHD. *See* Attention-deficit/hyperactivity disorder
ADIS. *See* Anxiety Disorders Interview Schedule
Adjusted Lo-Mendell-Rubin (ALMR) test, 1030
Adolescent/Adult Family Development Project (AFDP), 1048
Adolescent Alcohol Prevention Trial, 1014
Adolescents (adolescence), 556–566. *See also* Early adolescence; Late adolescence; Middle adolescence
  aggressive impulses
    Ward clustering, dendrogram, 829f
aggressive impulses, development
  log-linear model, observed/expected frequencies, 825t
  model, parameters, 825t
alcohol use, 1034–1035
antisocial peers/peer victimization, involvement, 649
anxiety symptoms/disorders, 635–637
attachment/autonomy, 398–399
attention-deficit/hyperactivity disorder (ADHD), 643–644
authority conflict (prediction), regression results (impact), 729f
cognitive development, Piaget theory (limitations), 478
comorbidity, 644–645
contradictions, normalization, 486
cortical functioning, efficiency, 293
depression, 633–635
depressive symptoms, 282, 633–635
developmental levels, questions, 481
eating disorders, 642–643
egocentrism, 485
emotion, understanding, 284–285
externalizing behaviors, development, 638
externalizing problems/disorders, 637–642
friendships, number/quality, 647–648
internalizing problems/disorders, 633–637
intimate partner violence (IPV), 640–641
mental health (promotion), I-Self (role), 503
methodological issues, 652–653
  measurement issues, 652–653
  mediating/moderating variables, 653
  psychological outcomes/comorbidity issues, 653
  public health implications/translational efforts, 653
  sample issues/study populations, 652
  study designs, 652
mixed-sex friendship groups, 639
negative rule-break bout slope, time-series (example), 728f
Oregon Youth Study (OYS)/Oregon-Couples Study, 638–639
parenthood, psychopathology (association), 645–646
peer context, 647–650
peer influence, 557–566
peers/partners, online communication, 649–650
physical aggression against peers, development
  log-linear model, observed/expected frequencies, 825t
  model, parameters, 825t
psychiatric disorders, identification
  interviewer-based interviews, usage, 110
  interviews, usage, 110
  respondent-based interviews, usage, 110–111
psychopathology, measurement, 108–110
relationships, behavior/experiences, 634–635, 636–637
romantic involvement, 633–634, 636, 639–640
romantic relationships, 631–633
  peer selection/socialization processes, 648–649
same-sex relationships, 646–647

Adolescents (adolescence), *(Continued)*
self-perception, inaccuracies, 485
theory development, issues, 651
translational implications, 653–655
Adolescent Symptom Inventory-4 (ASI-4), 82
Adoption
impact, 139–140. *See also* Environment.
studies, 136–137
usage, 147–148
Adrenaline level, cross-classification (first-order CFA), 875t
Adrenocorticotropic hormone (ACTH), 548
response, 174
Adult attachment. *See* Intervention
assessments, 334–335
associations, 360
mechanisms, 363–364
psychopathology, relationship, 360–363
representations, 369
study, 399
usage, 367–368
Adult Attachment Interview (AAI), 331
classification, distribution, 361
development, 362
results, 334
usage, 338, 343, 345
Adult Attachment Projective (AAP), 363
Adult Behavior Checklist (ABCL), 72
Adultification, 590
Adult-onset MDD, 115
Adults
ASEBA forms, 72
classification, 334
multi-informant data, 54
Adult Self-Report (ASR), 72
Affectional bonding, 592
Affective dissociation, development, 673–686
Affective disturbance, cross-classification (first-order CFA), 875t
Affective mediators. *See* Attachment
Affective, term (usage), 107
Affectivity, 453. *See also* Positive affectivity
Affiliative system, 222–223
Age-appropriate ASEBA instruments, 73
Age at first exposure, study, 112
Age effects, sequential designs (usage), 790f
Agency. *See* Self-agency
defining, 602
sense, 504
Agents, isolation, 680–683
Ages and Stages Questionnaire: Social Emotional (ASQ-SE), 19, 20
Agglomerative clustering, divisive clustering (contrast), 829–830
Agglomerative series, 828–829
Aggregated data (AD), 1043, 1066
IPD, contrast, 1044
usage, 1044f
Aggregates, 706
Aggregates problem, 667–669
change, study, 668–669
ergodicity, problem, 668–669
Aggregative fallacy, 802
Aggression, 543–544. *See also* Reactive aggression
development, 700
pathways, 698–699
forms, 544–545
relational forms, 544
Aggression-Inhibitory Response, 879
Aggressive behavior
longitudinal analyses, acceleration, 62–65
predictive pathways, 66f
six-year predictive pathways, 65, 67
Aggressive impulses (AI), 879
development
log-linear model, observed/expected frequencies, 825t
model, parameters, 825t
path model, 821f
two-group model, 821f, 827f
Ward clustering, dendrogram, 829f
Aggressive syndromes, 63
taxonomic properties, 67
Aggressive youth, striking, 649
AI. *See* Aggressive impulses
Ainsworth Stranger Situation, 453
Akaike information criteria (AIC), 1028, 1033
Alcohol consumption, independence model, 904
Alcohol Dependence Scale (ADS), 1071
Alcohol-related problems, responsibilities (neglect), 1071
Alcohol use, 640–641
Alcohol Use Disorders Identification Test (AUDIT), 1071
Aldohyde dehydrogenase (ALDH) enzyme activity, reduction, 145–146
Allocation bias, possibility, 130–131
Allostasis, concept, 219
Allostatic load models, impact, 219–220
ALMR. *See* Adjusted Lo-Mendell-Rubin
Alpha, protection, 876
Holland-Copenhaver procedure, 884
ALSPAC. *See* Avon Longitudinal Study of Parents and Children
ALT. *See* Autoregressive latent trajectory
Altitude line graph, usage, 739
American Professional Society on the Abuse of Children (APSAC) Task Force, attachment therapies report, 367
Amnesia, veil, 454
A-modal representations, 446–447
Amygdala, role (fMRI studies), 363
Analysis, multiple levels, 1011
Analysis of covariance (ANCOVA), 69, 1013–1014
Analysis of variance (ANOVA), 69, 750, 798, 826
measures, repetition, 807–808
series, 1063
usage, 1032
Anger, 640
frustration (relation), cusp catastrophe model (impact), 745f
moderation/inattention, latent change score model, 1020f
problems, 1018
sadness, combination, 496
Animal studies, importance, 161
Anorexia, 418–419
SDT, application, 419
Anscombe's z test, 886
Antecedents, role. *See* Parental need support/thwarting
Anterior insula (AI), activation, 550
Antisocial behavior (ASB), 640
depression, association, 136
Antisocial disorders, 361–362
Antisocial peers/peer victimization, involvement, 649
Antisocial personality, activation, 422
Antisocial personality disorder (APD), 55, 420–422
Antisocial personality disorder (ASPD), 1032
Antisocial youth
positive rule-break bout slope example, 728f
striking, 649
Anxiety
density plots, 834f, 835f
dichotomized variable, cross-classification (space-segment clusters), 836t
prenatal stress/anxiety, 175–177
symptoms/disorders, 635–637
variables, 888
AnxietyD, cross-classification, 836t
Anxiety Disorders Interview Schedule (ADIS), 30, 32, 110
Anxious attachment, 105
Anxious-solitary youths, 532–533
APD. *See* Antisocial personality disorder
APIM. *See* Actor-partner interdependence model
Appraisal, 287. *See also* Nonconscious appraisals
patterns, 297–298

Approach, term (usage), 799–800
AQS. *See* Attachment Q-Set
Arginine vasopressin (AVP), 548
Arithmetic learning. *See* Developing students; Emotional-behavioral difficulties
Arithmetic lessons, scaffolding dynamics, 689–691
ART. *See* Assisted reproduction technology
ASA. *See* Attachment Script Assessment
ASB. *See* Antisocial behavior
Ascertainment bias, 99–100
  problem, 100
ASD. *See* Autism spectrum disorder
ASEBA. *See* Achenbach System of Empirically Based Assessment
ASI-4. *See* Adolescent Symptom Inventory-4
ASQ-SE. *See* Ages and Stages Questionnaire: Social Emotional
ASR. *See* Adult Self-Report
Assessment. *See* Psychopathology
  advancement, 77–79, 79t
  data, methodological adequacy (determination), 56
  goals/purposes, 2
  taxonomy/diagnosis, relations, 77f
  variability, reduction, 13
Assessment (young children)
  approach, selection, 11–17
  checklist measures, coverage (breadth/depth), 26t–27t
  classification, validity, 14–15
  considerations, 4–9
  diagnostic approaches, 29–30
  Diagnostic Infant and Preschool Assessment (DIPA), 32
  diagnostic interviews, 30–32
    interrater reliability, 33–34
    psychometric properties, 32–33
    test-retest reliability, 32–33
    types, 31t
    validity, 34
  findings, sociocultural factors (impact), 8t
  multidomain checklists, 25, 27–28
  normatization, 15–16
  observational assessment, 35
  Preschool Age Psychiatric Assessment (PAPA), usage, 30–32
  reliability, 12–13
    interpretation rules, 13t
    statistics, 13t
    types, 12t
  response formats, 17
  screening methods, 18–19
    characteristics, 19–20
  screening tools, 20, 23
  tools, 17–36
    selection, 11–17
    types, 11–12
  validity
    cultural validity/norms, 16
    longitudinal developmental context, 14
    measurement ability, 13–14
Assisted reproduction technology (ART), 129, 137
Association, noncausal alternative explanations, 130–132
Attachment, 396–413, 592. *See also* Adolescents; Infants
  affective mediators, 357–359
  assessments. *See* Adults; Children.
  attachment-related information processing, amygdala role (fMRI studies), 363
  behavioral assessments, 332–333
  behavior, conceptualization, 332
  Cassidy-Marvin assessment, 332–333
  cognitive mediators, 357–359
  cues, 336
  developmental psychopathology, relationship, 325
  disorganized attachment
    determinants, 340
    reduction, video-feedback intervention (usage), 365–366
  emotional reactivity, 359–360
  emotion recognition/understanding, relationship, 358–359
  environment, 340–346
  fit, 368–369
  formation, 328
  gene-environment correlation, 346
  genes, impact, 340–346
  genetic/environmental determinants, 344–345
  holding therapies, 367
  individual differences, 328–331
    determinants, 335–346
    measurement, 332–335
  insecurity
    change, difficulty, 365
    environmental/genetic causes, questions, 341
  intergenerational transmission, 335–336
  internal working models, 331–332
  intervention
    effectiveness, meta-analysis, 365
    relationship, 364–369
  longitudinal studies, 359
  mediators, search, 354–360
  organized patterns, 331
  parental behavior, 336–340
  physiological mediators, 354–357
  psychopathology, relationship. *See* Adults.
  representational assessments, 333–334
  revisionist perspective, 348–349
  security. *See* Children.
    autonomy, relationship, 396–399
    continuity/change, 346–349
    environmental/genetic causes, questions, 341
    heritability, 344
    stability, 347
  self-regulation, 359–360
  social-cognitive mediators, 357–359
  social information processing, 357–358
  stability
    perspectives, 348–349
    study, 348–349
  status, discontinuity, 296
  system, 204–205
  temperament, 340–346
  theory (Bowby)
    historical overview, 326–328
    integration, 505
    predictions, 342
    usage, 224
  theory of mind, 358–359
  therapies report (APSAC), 367
  trauma therapies, 367
Attachment-focused interventions, positive effects, 356
Attachment Q-Set (AQS), 329–330, 333, 338
  administration, 347–348
  modified version, usage, 342, 369
  usage, 356
Attachment-relevant strategy, breakdown/disorganization, 330
Attachment Script Assessment (ASA), 334–335
Attention
  difficulties, self-sustaining learning trajectories, 686–695
  problems, context sensitivity, 686–687
  systems, 450
Attentional problems, neurobiological contributions, 687
Attention-deficit/hyperactivity disorder (ADHD), 3, 49, 643–644, 924
  diagnostic category, service specialization, 81
  DSM category, criterial symptom, 54–55
  focus, 28
  mediation/moderation effects, 924
  peer relations, 536
  psychological functioning, 671–672
  Rating Scale-IV, 82
  symptoms
    measurement, 947

Attention-deficit/hyperactivity disorder (ADHD), *(Continued)*
mediated effects, 932–933
variable, 950
Attention-emotional problems (teaching/learning), growth (modeling), 694f
Attractors, 611, 720
description, 723–724
index, 728–729
presence, 720f
strength. *See* Deviant talk attractor.
Attributes, healthy differentiation, 511
Attrition, 763–764
Atypical Behaviors Index (ITSEA), 12
Atypical developmental patterns, 886
Atypical trajectories, 877
Audience, witness function, 500
Augustine. *See* Saint Augustine
Authenticity
jeopardy, 481
promotion, 517
Autism
presence, 10
trademark, 463
Autism spectrum disorder (ASD), 12, 84
diagnosis rate, 143
problems, 17
Autobiographical memory, 475, 506
Autocorrelation patterns, 909
Autocorrelograms, usage, 802
Autoeroticism, 445
Autonomic movements, closed-loop feedback systems, 449
Autonomous motivation, facilitation, 425
Autonomous regulation, 387–396
Autonomy, 385. *See also* Adolescents; Infants
antecedent. *See* Emotional integration.
attachment security, relationship, 396–399
control. *See* Parental autonomy support/control.
definition, 385
disturbances, 385, 413–426
antecedents, 427
graphic overview, 414f
growth/psychopathology, relationship, 385–387
impairments, 413
independence, contrast, 389–390
need satisfaction, promotion, 404–405
role, 386
SDT definition/understanding, 388
spontaneous expression. *See* Human autonomy.
support, 390–391, 397, 425–426. *See also* Developmental processes; Parental autonomy support/control.
importance, 398–399
provision, 392
translational implications, 425–426
Autonomy-supportive conditions, 388
Autonomy-supportive socialization, benefits, 402
Autoregression, 905
first-order cross-legged vector autoregression model, 854f
multilevel autoregressive model, 852
vector autoregression model, 853–854
Autoregressive latent trajectory (ALT)
mediation models, 963
example, 964f
model, 963
Autoregressive (AR) mediation model
longitudinal/contemporaneous mediation, 954t, 957t
longitudinal mediation, 955t
Autoregressive (AR) models, 849–850. *See* Interindividual longitudinal analysis
cross-lagged paths, 943f
longitudinal mediation, 953f
Autoregressive (AR) panel models, contemporaneous mediation test (coefficients/standard errors), 958t
Autoregressive (AR)panel models, longitudinal mediation, 953–958
coefficients, 956t
standard errors, 956t
tests, coefficients/standard errors, 958t
Autotelie, 813
Auxiliary variables, usage, 776–777
Avandia cases, 1049
Averaged developmental trajectories, 908f
Averages, problem, 667–669
change, study, 668–669
ergodicity, problem, 668–669
Avon Longitudinal Study of Parents and Children (ALSPAC), 137–139, 176
Awareness, 504
development. *See* Self-awareness; Social awareness.

Barometric self, 491
BASC. *See* Behavior Assessment System for Children
BASC-2 Behavioral Emotional Screen System (BASC-2 BESS), 20
BASC-2 Preschool measurement, 25, 27
Base measures, selection, 832
Base model, 868, 870, 877. *See also* Configural frequency analysis
defining, 891
selection, 876
Batterer intervention programs (BIPs), 839
Bayesian analysis, 1078
Bayesian estimation methods, 781–782
Bayesian Information Criterion (BIC), 71, 1028, 1033
guidance, 1035
Bayesian MCMC, 770
BDNF. *See* Brain-derived neurotrophic factor
Beck Depression Inventory (BDI), 69
Beer consumption
first-order CFA, 904t
lag cross-classification, 902t
mood, cross-lagged relationship, 906f
numbers cross-classification
first-order CFA, 896t
prediction CFA, 897t
Behavioral disorders, natural kind designation (question), 57
Behavioral genetic methods, usage, 342
Behavioral inhibition, bias, 695
Behavioral interaction components analysis (schematic diagram), event history analysis (usage), 731f
Behaviorally presented self-esteem, 489–490
Behavioral maladaptation, patterns, 666
Behavioral problems
assessment, dimensional tools (usage), 23–24
checklist/questionnaire measures, 21t–22t, 25t
dimensional checklists, 24–29
taxonomies, construction, 57–58
univariate growth mixture modeling, fit indices, 1030t
Behavioral regulations, internalization/integration, 403
Behavioral system, activation, 397
Behavior Assessment System for Children (BASC), 52
Behavior Problems Index (BPI), 1009
Behaviors
autonomous regulation, 387–389
identified regulation, 403
internally controlling regulation, 413
observation, 1049–1050
pattern, organization, 243–244
perceptions, contrast, 616
BEIP. *See* Bucharest Early Intervention Project
Belongingness, absence, 537
Berkeley Puppet Interview, 37
Berkson's bias, 60
Best-fitting model, results, 1020
Best linear unbiased predictions (BLUPs), 850
Best practices, 701–704
Better-than-average effect, observation, 486
Better-than-strategy, usage, 483

Between-cohort predictive correlations (Dutch children), 63f
Between-family risk synergy, 69
Between groups validity, 14
Between-study correlation, 1062
Between-study variability, 1059
BIC. *See* Bayesian Information Criterion
Bicultural adaptation, 508–509
Bidirectional comorbidity, unidirectional comorbidity (contrast), 59–60
Binomial test, 874
Biological processes, impact, 621
Biopolar disorder, 110
Biopsychological model, assumptions, 621
Biopsychological profiles, example, 807f
BIPs. *See* Batterer intervention programs
Birth
  experiential awareness, 443–447
  experiential vicariousness, 447
  newborn, feeling (description), 444–447
BITSEA. *See* Brief Infant Toddler Social Emotional Assessment
Bivariate analyses, usage, 1026
Bivariate dual change score model, 1022f
Bivariate growth curve model, 1010f
Bivariate latent change score models, 1017
BMIs. *See* Brief motivational interventions
Body dissatisfaction, 498
Bonferroni adjustment, 876
Bonferroni-protected α, 874, 889
Bootstrap confidence intervals, 931t
Bootstrapped EM, 770–771
Borderline personality disorders (BPD), 422
  identity, stability (absence), 423
  severe need thwarting, 422–425
Borkenau data
  screen plots, comparison, 859f
  set, 858
Boundaries. *See* Family theory
  dissolution, 618–619
    family systems perspective, 618–619
Bowen family system theory, 582
BPD. *See* Borderline personality disorders
BPI. *See* Behavior Problems Index
Brain-based constraints, 689
Brain-derived neurotrophic factor (BDNF), 167
  polymorphisms, 177
  SNP, genotyping, 366
Bravado trait, 812–813
Breaking randomization, 1071
Brief Infant Toddler Social Emotional Assessment (BITSEA), 19, 20
Brief motivational interventions (BMIs), 1067–1068, 1078
Brief Psychiatric Rating Scale, 890
Brief Symptom Inventory (BSI), 1032
Brief Young Adult Alcohol Consequences Questionnaire (BYAACQ), 1071
Briggs, 700
Broadening function, 512–513
Bucharest Early Intervention Project (BEIP), 178
Build function, 513
Built-in motivational systems, 450
Built-in sameness detection system, 450–452
Bulimia, 418–419
Bullying, 543–544. *See also* Cyberbullying
  assessment, 544
  defining, 543–544
  effects, 499
  impact, 473
  victims, 503

CA. *See* Chronological age
California Child Q-Set (CCQ), 1030
California Q-Set, 817
Callous-unemotional (CU) traits, 279
Candidate genes, research, 271
Cannot Classify classification, 362
CAPA. *See* Child and Adolescent Psychiatric Assessment
Capitalizing on chance, 876
Caregiver report
  instruments, 2
  tools, types, 11t
Caregiving
  assessment process, 5–6
  contexts (young children), 6–9
  impact, 296–297
  neglect/deprivation, 178–179
  system, 223
Catastrophe model. *See* Cusp catastrophe model
Catastrophe modeling, 744–746
Categorical judgments, 267
Categorical time series data, analysis, 737–738
Categorical variables, cross-classification, 868
Causal inference models, 973–978
Causality, locus (internal perception), 387–388
Cause, meaning, 129–130
CBCL. *See* Child Behavior Checklist
CBT. *See* Cognitive-behavioral therapy
CD. *See* Conduct disorder; Confidence distribution
CDI. *See* Children's Depression Inventory
Cells, 868
Centering around the grand mean (CGM), 937
Centering within contexts (CWC), 937
Centers for Disease Control and Prevention (CDC), importance, 118–119
Centrality of mutual recognition, 463–464
Central nervous system (CNS), functioning (documentation), 289
Central tendency measures, 728
CET. *See* Cognitive evaluation theory
CFA. *See* Configural frequency analysis
CFI, 778–779
  values, 966
Change
  development, relationship, 104
  latent change score modeling, usage, 1022–1023
  processes, impact, 591–592
  study, 668–669
Change over time model, 923
Chaos. *See* Parental structure/chaos
  con-fusion, 444
  quantitative measures, 726
Charcot, 464
Checklist measures, coverage (breadth/depth), 26t–27t
Chicago Child-Parent Center (CPC), intervention services, 180–181
Child and Adolescent Psychiatric Assessment (CAPA), 110
Child Assessment Schedule, 110
Child Behavior Checklist (CBCL), 109, 1007, 1032
  Aggressive and Delinquent syndrome scores, usage, 62
  Internalizing and Externalizing scores, 69, 70
  ratings, 85
  raw scores, 1018
Child Behavior Checklist for 1.5–5 (CBCL/1.5– 5), 24, 27–29
  scores, oversampling, 33
Child Depression Inventory, 1032
Child-driven family processes, 609
Child-driven triangulation, 616–617
Childhood sexual abuse (CSA), 650–651
Children. *See* Young children
  abuse, 678–679
  adjustment, 616
  attachment
    assessments, 332–334
    security, 349–354
  attention difficulties
    context sensitivity, 686–687
    motivation/performance relations (state space model), 671f
    neurobiological approaches, strengths/limitations, 687
    self-sustaining learning trajectories, 686–695

Children. *See* Young children *(Continued)*
teaching/learning, dynamics, 687–689
attention-emotional problems (teaching/learning), growth (modeling), 694f
caregiver interaction, 297
characteristics, development, 281
childhood. *See* Early to middle childhood; Middle-to-late childhood; Very early childhood.
developmental levels (questions), 481
self-enhancement. *See* Early to middle childhood; Middle to late childhood.
children of twins design, 135
community factors, 233
competence, SDS (impact), 222f
contribution. *See* Family.
demobilizing pattern, development, 242
development, 175–177
developmental challenges, 241t
early institutional care, impact, 178–179
emotional-behavioral difficulties
development socioemotional adjustment, 695–701
neurobiological approaches, strengths/limitations, 687
self-sustaining learning trajectories, 686–695
teaching/learning, dynamics, 687–689
emotional insecurity, 227
interparental sources, 214–216
emotional security, investigations, 233
functioning, level (sustaining), 688
gender, family processes (variations), 620
impairment, examples, 36
information, establishment, 5
insecurity, sequelae, 217
interaction, dynamics. *See* Group home.
interparental conflict adjustment, emotional security mediation, 209–223
maladjustment, 613
maltreatment. *See* Maltreated children.
affective dissociation, development, 673–686
mean aggressive/delinquent behavior syndrome scores. *See* Dutch children.
mental health (promotion), I-Self (role), 503
multi-informant data, 53–54
needs, caregiver attention, 276
nonverbal response, indication, 9
observational methods, 35
one-child policy (China), 144–145
parent-child relationship properties, 224–226
peer acceptance/rejection, stability, 534
peer status. *See* School-age children.
performance, difficulty (optimal range), 687
problems, experience, 14–15
psychiatric disorders, identification
interviewer-based interviews, usage, 110
interviews, usage, 110
respondent-based interviews, usage, 110–111
psychiatric epidemiology
developmental approach, implications, 103–105
history, 106–111
origins, 106
psychopathology, 349–354
assessment, questionnaires, 109–110
empirically based models, 51–52
measurement, 108–111
reciprocal influences, 394–395
regulatory strategies, usage, 295
screening, increase, 37
setting, characteristics, 7
socioadjustment, adaptive/maladaptive patterns (development pathways), 697f
socioeconomic factors, 233
spatial preposition acquisition (representation), moving min-max graph (usage), 739f
temperament assessment methods, 35
temperament/personality, emotional security (contrast), 207–208
temperament, risk, 294
tutor (mathematics instruction), matches/mismatches/self-iterative utterances (changes), 690f
verbal abilities, 278
within-cohort/between-cohort predictive correlations. *See* Dutch children.
Children of alcoholics (COAs), 1048
Children of twins (CoT) design, 135. *See also* Extended children of twins design
Children's Depression Inventory (CDI), 1003–1004, 1032
Child-staff interaction, constraint, 703
China, one-child policy, 144–145
Chinese famine, 142
Chi-squared distribution, 1056
CHRM2 gene, association, 566
Chronic peer victims, sporadic peer victims (contrast), 547
Chronological age (CA), developmental metric, 48
Chronological time, 989
Circular causality, 721
Circularity, 588
Classification, validity, 14–15
Classroom Behavior Checklist, 1034
Client/therapist, attachment fit, 368–369
Clinical categories, operational definitions (GAP), 55
Clinical health strategies
early interventions, impact, 180–183
shaping, developmental models (impact), 179–185
Clinical targets/approaches, delineation, 250–251
Clinics, case classification, 98–99
Closed-loop feedback systems, 449
Cloud of unpleasantness, 619
Cluster analysis, 826–837, 847
discussion, 837
problem (specification), taxometry (usage), 827–828
Clustering
agglomerative clustering, divisive clustering (contrast), 829–830
decisions, 828
exhaustive clustering, nonexhaustive clustering (contrast), 830–831
stochastic clustering, deterministic clustering (contrast), 831–832
Clusters
base measures, selection, 832
convex clusters, nonconvex clusters (contrast), 832–833
disjunct clusters, overlapping clusters (contrast), 828
fear/joy/anxiety, density plots, 834f, 835f
hierarchical clusters, nonhierarchical clusters (contrast), 828–829
solutions
comparison, 836
cross-classification, 827t
three-cluster solutions, cross-tabulation, 835t
Coactive person-environment system, 670f
Coactive scaffolding, nature/importance, 688
Coalition, formation, 584
Cochrane Database of Systematic Reviews, 1048
Coded observational data, time-series (usage), 735
Cognitive-based intervention, impact, 182
Cognitive-behavioral therapy (CBT), 498
Cognitive-developmental processes, emergence, 477
Cognitive evaluation theory (CET), 400
Cohen's effect size estimate, 1048
Cohen's kappa, 829, 891, 901
Coherence, sense, 488
Cohesive families, defining, 614
Cohort effects, sequential designs (usage), 790f
Cohort-sequential data set, conversion, 789t
Coleman, 810
Collaborative Data Synthesis for Adolescent Depression Trials (CDSADT), 1080
Collective variable
developmental trajectories, mapping, 724
identification, 723

Combined resistance training and normative education plus information group (COMB), 1014
Communicating, impact, 592
Communication, problem solving, 598
Community, case classification, 98–99
Comorbidity, 644–645
  contrast, 59–60
  controls, presence/absence, 114t
  implications, 60–61
  issues, 59, 653
  rates, elevation, 78
Comorbidity, issues, 59–61
Compensation, concepts, 587
Competence support, 426
Complex interactions, 804–805
Complexity assessment, quantitative/qualitative methods (elaboration), 706–707
Complex research synthesis, 1062–1067
Complex systems, steady states (contrast), 702
Composite antitype (joint antitype), 875
Composite type, 896
Comprehensive Meta-analysis (CMA), 1079
Conceptual awareness, experiential awareness (contrast), 448–449
Conceptual primitives, 452–453
Conditional latent growth modeling, 1013
Conditional support, 472
Conditional types/antitypes, 912
Conduct disorder (CD), 55, 420–422
  association, 361–362
  development, 422
Conduct problems, 640
Conduct problems development
  pathways, 698–699
  trajectories, 668f
Confidence distribution (CD), 1062, 1066
Configural anticlusters, 871
Configural clusters, 871
Configural frequency analysis (CFA), 866–877. *See also* Trajectories
  alpha, protection, 876
  antitype, 868
  base models
    characteristics, 870
    classes, 871–873
    definition, 869–871
    frequency distribution, discrepancies, 900
    selection, 876
  cell frequencies, 894t
  CFA of runs, 895
  characteristics, 917
  configuration groups, testing, 874–876
  confirmatory CFA, application, 889–892
  correlation matrix, 905t
  cross-lagged CFA (BxMxB1XM1 cross-classification), 907t
  cross-lagged designs, 905–908
  cross-lagged relationship, 905f
  data examples, 879–883
  data usage, questions, 901–902
  designs, control group presence (treatment effects), 892–895
    data example, 893–895
  designs, no control group absence (treatment effects), 890–892
  development, end points (prediction), 912–916
    data example, 913–915
  differences, 878–883
    method, 879f
    structural zones, 881–883
  expected frequencies
    estimation, 876
    2x2 cross-classification, 902t
  extensions, 883–884
  first differences, 878
    calculation, 878t
  first-order CFA, 869t, 875t
    base model, 872
    example, 881t
    structural zeros, accounting (absence), 883t
    structural zeros, accounting (presence), 884t
  fourth differences, calculation, 878t
  individual/averaged developmental trajectories, 908f
  involvement, 830
  lags, 900–905
    autocorrelograms, 909f
  log-linear cross-lagged model, parameter estimates, 907t
  log-linear modeling, results, 904
  models, 877–912
  null hypothesis, 873
  observed frequencies, 2x2 cross-classification, 902t
  odds ratio, example, 894–895
  outcome pattern, 894
  prediction CFA, 897t
  prediction CFA (P-CFA), 912
  PRxVRxAR cross-classification, two-group CFA, 900t
  respondents, aggregate (autocorrelograms), 909f
  runs, 898–899
  second differences, 878–883
    calculation, 878t
  second-order CFA base model, 872
  significance testing, 873–876
  single-subject CFA, 895
  single-subject designs, 895–900
    data example, 895–900
    runs, CFA, 898–899
  steps, 876–877
  structural zones, differences, 881–883
    data, example, 882–883
  support, 76
  technical elements, 869–871
  tests, 873–874
    comparison, data example, 875–876
  third differences, calculation, 878t
  third-order CFA base model, 872
  translational research, relationship, 917–919
    limitations, 918–919
    value added, 919
  treatment, cross-classification, 894t
  two-group CFA, 880t
  two-group pattern shift, comparison (2x2 table), 893t
  two-group pattern shift, comparison (2x2x2 table), 893t
  two-variable pattern, comparison (2x2 table), 894t
  types/attributes, interpretation, 877
  variables, descriptive statistics, 880t
Configural Invariance Model, 1024
Configural invariance model, 1003
Configurations, 868
  groups, testing, 874–876
  substantive meaning, 877
Confirmatory CFA, application, 889–892
Confirmatory factor analysis, 860t
Confounding
  impact, 100–101
  variable, 129, 131
Congenital myotonic dystrophy, 275
Connectionism, 743
Connectionist modeling, 743–744
Conners Early Childhood (Conners-EC)
  Behavioral form1, 28
  summary score, absence, 29
Conscientiousness, levels, 460–463
Consciousness
  aim, 439–440
  feeling experience, origins, 440–442
  intersubjectivity, roots, 455–463
  meaning, 440–443
  representation, meaning, 442–443
  roots, 439

CONSORT flowchart, 1052
Constant change model, 1016
Constructs
  analysis, mesoanalytical level, 608
  interrelatedness, 615–616
  measurement, repetition (benefits), 933–934
Construct validity, 14
Content validity, 14
Contextual influences
  understanding
    growth mixture modeling studies, usage, 1032–1034
    latent change score modeling studies, usage, 1021–1027
    latent growth modeling studies, 1009–1015
  young children, 6–9
Contextualism, theories, 529
Contextual risk factors, 352
Continuity-discontinuity (developmental pathways), latent growth modeling studies (usage), 1003–1009
Continuous-time MCMC, 561
Control
  issues (perceptions), positive psychology (emphasis), 516–517
  parameters
    identification, 724–725
    manipulation, 725
  sense, 516
Controlled direct effect, 974, 975
Convergence, 773–774. *See also* Markov chain Monte Carlo
  term, usage, 814
Convergent thinking, 515
Convergent validity, 14
Convex clusters, nonconvex clusters (contrast), 832–833
Coordination
  growth, modeling, 684–686
  modeling, 686f
  trajectories, 685–686
Coparenting conflict, family process (relationship), 617–618
Coperception, 464
Core control variables
  identification, 704
  regulation attempts, coordination, 704
Core knowledge, 452–453
Correlation measures, 832
Corticotrophin-releasing hormone (CRH), 548
  source, 272
Cortisol responses, elevation, 355
CoT. *See* Children of twins
Co-twin, sex, 145
Coupled equations, 734–736
Covariance matrix, 855–856
Covariates, patterns, 1075t
Coy behavior, 206
C-reactive protein (CRP), 539
Creativity, 514–516. *See also* Emotional creativity
  Big-C version, 515
  small-c version, 515
CRH. *See* Corticotrophin-releasing hormone
Crime, cross-classification (first-order CFA), 875t
Criminality data, usage, 143
Critical ratio (CR), display, 1019f
Cross-classification cells, 868
Cross-cultural differences, theory, 602
Cross-domain relations
  latent change score modeling, usage, 1021–1022
  latent growth modeling, usage, 1009–1012
Cross-generational boundary violations, 616
Cross-informant agreement level, 78
Cross-lagged CFA, 895
  BxMxB1xM1 cross-classification, 907t
Cross-lagged designs, CFA, 905–908
  data example, 906–908
Cross-lagged model, log-linear cross-lagged model (parameter estimates), 907t
Cross-lagged regression, 05, 848
  model, 853f
Cross-population comparisons
  latent growth modeling, usage, 1012–1013
  usage, 1029–1032
Cross Study Project, 1055
CU. *See* Callous-unemotional
Cultural diversity. *See* Family systems
Cultural self, 476, 478
Cultural validity/norms, 16
Cultural values, capture, 234–235
Cumulative effects models, 160–161
Curiosity, impact, 516–517
Curves model, 1003
Cusp catastrophe model, 745f
  impact, 745f
Cyberbullying, 502–503
Cyber victimization, 545

Data
  absence, 760
    treatment, methods, 762t–763t
  accelerated longitudinal design, cohort-sequential data set conversion, 789t
  assumptions, 775–776
  augmentation, 770
  auxiliary variables, usage, 776–777
  bias, 761
  Borkenau data set, 858
  box, 854–858
    example, 855f
  change, reasons, 784
  considerations, 774–782
  examples, 858–861, 1067–1080. *See also* Configural frequency analysis; Stouffer's Z.
  full information maximum likelihood (FIML), 767–774
  longitudinal data, factor models, 855–857
  mediation analysis, 780–782
  missing at random (MAR), 765t, 766
  missing completely at random (MCAR), 764–766
  missing data
    mechanisms, 765t
    methods, 767
    practices, review, 782–784
  missingness, mechanisms, 763–767
  missing not at random (MNAR), 764, 766–767
  multiform planned missing protocols, 785–787
  multiple imputation with chained equations (MICE), 771–772
  Ostendorf data set, 858
  PANAS data, 858–861
  person-oriented data analysis, methods, 818–822
  planned missing data designs, 784–790
  pooled data, 1068–1071
  Rubin's rules, 772–773
  sequential designs, 790f
  sets, 858
  statistical issues, 761
  supermatrix technique, graphical representation, 781f
  synthesis, unification, 1062
  two-factor solution, positive/negative items, 859t
  two-method planned missing design, 787–789
  type I error, 761
  type II error/power, 761
  wave-missing designs, 789–790
DB-DOS. *See* Disruptive Behavior Diagnostic System
DBT. *See* Dialectical behavior therapy
Default selection, 554–555
Defense analogs, proliferation, 218–219
Defensive regulation, 408
Delay aversion, 686
Delinquent behavior
  longitudinal analyses, acceleration, 62–65

predictive pathways, 66f
six-year predictive pathways, 65, 67
Delinquent syndromes, 63
taxonomic properties, 67
Dendrogram, 830
example, 829f
Deoxyribonucleic acid (DNA) methylation, 167–168, 672
Dependence. *See* Social dependence
autonomous forms, 389
protracted dependence, 459
Dependency of tests, 876
Depressed affect, perceived directionality, 496
Depression
adolescents, 633–635
composite, pathways, 495–496
cross-classification, 894t
effects, 634
etiology, research, 417
measurements, repetition, 1010
pathways, 495–496
sadness/anger, combination, 496
self-esteem, relationship, 494
suicidal behaviors, relationship, 494–496
terms, usage, 97
variables, 888
young adults, 633–635
young children, 3–4
Depressive reaction, pathological condition, 472
Depressive symptoms
adolescents/young adults, 633–635
fitted growth curve models, comparisons, 1004t, 1006t
multivariate growth model, 1006f
pro-inflammatory cytokines, effect, 541
Descriptive developmental profile analysis, 738–740
Descriptive statistics, example, 880t
Detached awareness, 512
Deterministic clustering, stochastic clustering (contrast), 831–832
Deterministic models, 831
Developing students, arithmetic learning, 691–692
Development
autonomy disturbances, 413–426
change, relationship, 104
concept, 102–103
dynamic assessments, structural change (accounting), 707
dynamic systems analyses, psychological theory (importance), 706
end points, prediction, 912–916
data example, 913–915
epigenetic nature, 672f
goals
direction, 104–105
normality, 105
hierarchical levels. *See* Skill development.
infusion. *See* Family systems theory.
intersubjectivity, levels, 456t
pathways. *See* Aggression; Conduct problems; Inhibition; Social anxiety.
phase transitions, identification, 724
representational tier, levels, 674f
sociality/reciprocity, impact, 455–456
socioemotional adjustment. *See* Children.
study, 426–428
term, usage, 48
trajectories, 701
configural frequency analysis (CFA), 884–886
transactional models, 529
webs, 675
Developmental analysis, 989
comprehensiveness, 240
Developmental capacities, 598
Developmental cascade mechanisms, 217–223
approach, ethological systems (cascading effects), 221–223
affiliative system, 222–223
caregiving system, 223
exploratory system, 221–222
defense analogs, proliferation, 218–219
stress response systems, alterations, 219–221
Developmental domains, child functioning, 2
Developmental epidemiology, 94
contributions, 95f
defining, 102–105
development, concept, 102–103
future directions, 113–121
global mental health, 116
history, 106–111
homotypic/heterotypic continuity, comorbidity (presence/absence), 114t
impact studies, 117–119
intergenerational studies, 116–117
interventions, costs/effectiveness, 118
life-course studies, 115–116
longitudinal research, 114–117
longitudinal studies, 114–115
mental health
services, quantification (advantages), 81
surveillance, 118–119
role, 118
Developmental growth profiles, graphing process, 738
Developmental hypotheses, 162
early intervention, relationship, 183–184
Developmental immaturity, psychopathology (nonrelationship), 683–684
Developmentalists, dilemma, 717–719
resolution, 719
Developmental mechanisms, 163–170
child development, 175–177
clinical/public health application, 184–185
early/severe caregiving neglect/deprivation, 178–179
exposures (timing/duration/intensity assessment), experimental leverage (impact), 171
genetics, 167–168
human health/development, animal findings (translation), 174–175
individual differences, 172–173
investigation, conceptual/methodological considerations, 170–175
maternal immune activation paradigm, 177
neural development/circuitry, 166–167
neuroimmunology, 164–166
phenotypic/measurement stability, 171–172
prenatal stress/anxiety, 175–177
psychological mechanisms, 168–169
sex differences, 173–174
stress physiology, 163–164
studying, model systems/paradigms (usage), 175–179
Developmental models
child development, 175–177
clinical/public health application, 184–185
early interventions, impact, 180–183
early/severe caregiving neglect/deprivation, 178–179
exposures (timing/duration/intensity assessment), experimental leverage (impact), 171
human health/development, animal findings (translation), 174–175
impact, 179–185
individual differences, 172–173
investigation, conceptual/methodological considerations, 170–175
maternal immune activation paradigm, 177
phenotypic/measurement stability, 171–172
prenatal stress/anxiety, 175–177
sex differences, 173–174
studying, model systems/paradigms (usage), 175–179
Developmental origins of health and disease (DOHaD), 164
Developmental pathways, 241–243
self-organization, 667
turning points (study), latent growth modeling (usage), 1005–1006
Developmental pathways, understanding
latent growth modeling studies, usage, 1003–1009
latent growth modeling, usage, 1003–1004

Developmental patterns, types, 886
Developmental periods, intrinsic motivation (effects), 401–402
Developmental processes
  autonomy/autonomy support, 396–413
  research, configural frequency analysis (usage), 866
Developmental programming models, 157
Developmental psychopathology, 48, 107–108
  analysis, 989
    levels, 598
    multiple levels, 988
  approach, core theoretical principles, 986–989
  attachment, relationship, 325
  development, dynamic systems models (impact), 665
  dynamic systems methods, surveys, 717
  dynamic systems theories
    impact, 665–667
    relationship, 669–673
  ecological systems approach, 232
  emotional security theory, relationship, 199
  family systems theory, impact, 597–601
  framework, 528–530
  growth points, family systems perspectives, 621–622
  hierarchical linear modeling (HLM), application, 69
  implications, 463–465
  importance, examples, 112–113
  intraindividual variation, modeling (person-specific approaches), 846
  I-Self processes, impact, 470
  latent growth modeling, relationship, 986
  Me-self processes, impact, 470
  models, 238f
  open systems conceptualizations, 220–221
  pathways framework, 987
  peer relations, relationship, 527
  process, focus, 597–598
  research
    etiology, 1047–1049
    infrequently/rarely observed behaviors, 1049–1050
    integrative data analysis, usage, 1042
    moderation/mediation analyses, role, 923–924
    prevention/intervention, 1051
    screening/diagnostic tests, 1050–1051
    utilities, 1047–1051
  term, dual function, 665–666
Developmental research, CFA models, 877–912
Developmental susceptibility periods, identification (difficulty), 239–240
Developmental-time measures, 738–740
Developmental trajectories, 241–243, 837
  alternatives, 721f
  individual/averaged developmental trajectories, 908f
  mapping, 724
  study, multilevel modeling (usage), 1000–1015
  usage, 71–72
Developmental web, 675f
Development and Well-Being Assessment, 111
Development domains (young children), 9–17
Deviant talk, 728
  attractor, strength, 729t
Deviation, concept, 877
Diagnol-half sign test, 890
Diagnosis
  components, 77–78
  term, usage, 58
Diagnostic and Statistical Manual (DSM)
  criteria, operationalization (problem), 53
  diagnoses, relations, 83
  diagnostic system, family factors (inclusion), 593
  DSM-oriented scales, 27–28
  versions, 51
Diagnostic and Statistical Manual, Fifth Revision (DSM-5), 593, 799
  development, term (usage), 99
  relational diagnosis, usage, 596–597
Diagnostic and Statistical Manual for Primary Care (DSM-PC), family factors (inclusion), 593–594
Diagnostic and Statistical Manual, Third Revision (DSM-III), obsolescence, 73
Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood, Revised (DC:0–3R), family factors (inclusion), 594
Diagnostic formulations, 59, 77
Diagnostic grammar, 97–98
Diagnostic Infant and Preschool Assessment (DIPA), 30, 32, 33
  uniqueness, 34
Diagnostic interviews, 30–32
  interrater reliability, 33–34
  psychometric properties, 32–34
  test-retest reliability, 32–33
  types, 31t
  validity, 34
Diagnostic Interview Schedule, 1033
Diagnostic Interview Schedule for Children (DISC), 49
Diagnostic Interview Schedule for Children and Adolescents (DICA), 110
Diagnostic Interview Schedule (DIS-IV), usage, 1033
Diagnostic odds ratio (DOR), 1050
Diagnostic processes, 59, 77
Diagnostic systems, family factors (inclusion), 593–595
Diagnostic taxa, quantification, 86–87
  efforts, 82–85
Diagnostic tests, 1050–1051
Diagonal-half sign test, 890
Dialectical behavior therapy (DBT), 367–368
Diathesis-stress model, 162
DICA. *See* Diagnostic Interview Schedule for Children and Adolescents
Dichotomized variables, cross-classification (space-segment clusters), 836t
DID. *See* Dissociative identity disorder
Differences. *See* Configural frequency analysis
  first differences, 878
  method, 879f
  score modeling. *See* Latent change (difference) score modeling.
  structural zones, 881–883
Differential associations, mechanisms, 237
Differential item functioning (DIF), 822, 1055
Different me's, conflict, 507
Dimensional checklists, 24–29
  focus, 28–29
Dimensional identity, 808–809
Dimensional tools, usage, 23–24
DIPA. *See* Diagnostic Infant and Preschool Assessment
DISC. *See* Diagnostic Interview Schedule for Children
Discordant MZ twin design, power (increase), 134
Discordant sib pairs, 129
  natural experiments, 134–135
Discordant twin pairs (natural experiments), 133–134
Discriminant validity, 14
Discrimination types, 873
  finding, two-group CFA (impact), 900
Discriminator variables, 873
Disease
  definition, 95
  frequency, measurement, 96–97
  progression, process, 102–103
Disengaged families, defining, 614
Disjunct clusters, overlapping clusters (contrast), 828
Disorders. *See* Conduct disorders; Obsessive-compulsive disorders
  categorization, 60
  internalization, impairments. *See* Externalizing disorders.
  introjection/internally controlling states, impact, 414–420
  severe need thwarting. *See* Borderline personality disorders.
Disorder, self-organization, 704–707
Disorganization, DRD4 Gene (impact), 343
Disorganized attachment, determinants, 340
Disruptive Behavior Diagnostic System (DB-DOS), 35
Disruptive behavior disorders (young children), 4

Dissociation. *See* Isolating dissociation
first tier (agent isolation), 680–683
growth, modeling, 684–686
isolation, 679–683
skills, 679–680
modeling, 686f
passive/active splitting, relationship, 677–678
skills, trajectories, 685–686
Dissociative disorders, representation, 424
Dissociative identity disorder (DID), 422–425
Distress
internalization, 532–533
personal distress, 223
Distress-eliciting tasks, 338
Divergent thinking, 515
Divisive clustering, agglomerative clustering (contrast), 829–830
Divisive series, 828–829
DOHaD. *See* Developmental origins of health and disease
Domain gap, 339
example, 340
Domains. *See* Development domains
emphasis, variation, 29
Dominic-R, 110
Dopamine D4 receptor gene, polymorphism, 342–343
Dopamine D4 VNTR, intervention/variation, 356
Dopamine transporter (DAT1) gene, risk alleles, 555
Dorsal anterior cingulate cortex (dACC), activation, 550
Down syndrome, 106
DRD4 dopamine receptor, 177, 1012
DRD4 gene
impact, 343
long allele, impact, 566
Drinking Restraint Strategies (DRS) scale, 1070
Drinking Strategies (DS) scale, 1070
Drug Abuse Screening Test (DAST), 1032
Drug use, 640–641
DS. *See* Drinking Strategies; Dynamic systems
DSM. *See* Diagnostic and Statistical Manual
DSM-5. *See* Diagnostic and Statistical Manual, Fifth Revision
DSM-PC. *See* Diagnostic and Statistical Manual for Primary Care
Dunedin Multidisciplinary Health and Development Study, 1005
Duration-based data, 749
Dutch children
mean aggressive/delinquent behavior syndrome scores, 64f
within-cohort/between-cohort predictive correlations, 63f
Dutch famine, 142
Dyads
mixed dyad, preperturbation SSGs/postperturbation SSGs, 748f
recurrence plots (diagonal line structures), entropy (LCGA analysis results), 730f
study, 586
variables, parameter profiles, 742t
Dynamic factor model, 857–858
Dynamic growth modeling, 740–743
Dynamic parameters, 1017
Dynamic skills theory, tools, 673–675
Dynamic stability, 720
Dynamic systems (DS), 611, 803
analyses
data, suitability, 725–726
psychological theory, importance, 706
approaches, 702
attractors, description, 723–724
behavioral interaction components analysis (schematic diagram), event history analysis (usage), 731f
case studies, 727
catastrophe modeling, 744–746
clinical research, implications, 750–752
concepts, 726t
connectionist modeling, 743–744
control parameters
identification, 724–725
manipulation, 725
coupled equations, 734–736
descriptive developmental profile analysis, 738–740
developmental-time measures, 738–740
development, phase transitions (identification), 724
dyad recurrence plots (diagonal line structures), entropy (LCGA analysis results), 730f
dynamic growth modeling, 740–743
event history analysis, 731–738
interest, collective variable (identification), 723
Karnaugh maps, 733–734
latent class analysis, 740–746
longitudinal data, 727
meta-theoretical framework, 721
methodology, elaboration, 706–707
methods, 727–746
survey. *See* Psychopathology.
models, impact, 665
nonlinear change, 721–723
nonlinear dynamics, 736–738
null clines, 735f
observational data, 725–727
observation sessions, exemplary dyads (recurrence plots), 729f
organization, hierarchically embedded levels, 721
perspective, 529
perturbations, 721–723
phase transitions, 721–723
principles, 719–723
impact, 723–724
real-time measures, 727–731
recurrence quantification analysis (RQA), 729–731
state space grid analysis, 746–750
steady states, stability, 735f
techniques, 726t
term, usage, 720
time-series analysis, 727–729
translation, 701–704
Dynamic systems theories
action, adaptive/maladaptive forms (self-organization), 671–672
developmental psychopathology, relationship, 669–673
impact, 665–667
Dynamic trajectories, 667–669
averages/aggregates, problem, 667–669
Dyschronous interactions, repair, 6
Dysfunctional Attitudes Scale, perfectionism subscale, 1026
Dysfunction, conceptualization, 705–706
Dysphoria, 206, 215

Early adolescence (ages 11–13)
deviant talk attractor, strength, 729t
I-Self, impact, 478–479
Jamesian I-Self functions, impact, 479
self-enhancement, 484–485
Early adversity, definition, 285–286
Early attachment insecurity, effect, 352
Early caregiving neglect/deprivation, 178–179
Early childhood (ages 5–7)
adversity, exposure, 299
neuroatypical conditions, 275–276
peer interactions, 530–533
self-enhancement, 482–483
strategies, 481–482
Early Childhood Longitudinal Study—Kindergarten Cohort (ECLS-K)
data, 943
data examples, 924–925
data set, causal estimators, 978
mediation pathways, 957
two-predictor three-mediator, path diagram, 931f
Early Childhood Screening Assessment (ECSA), 23

Early experiences
attention system, 450
built-in motivational systems, 450
built-in sameness detection systems, 450–452
clinical/public health application, 184–185
conceptual primitives, 452–453
constraints, 447–455
core knowledge, 452–453
cumulative effects models, 160–161
developmental mechanisms, 163–170
developmental programming, 159–160
effects, developmental models/mechanisms (usage), 156
experiential awareness/conceptual awareness (contrast), 448–449
genetics, 167–168
infantile amnesia, 453–455
life span development, 160–161
mechanisms, 169–170
models, alternatives, 161–163
neural development/circuitry, 166–167
neuroimmunology, 164–166
newborns, functional propensities, 449–450
period models, sensitivity, 157–159
psychological mechanisms, 168–169
psychological research, developmental models/hypotheses (impact), 157–163
stress physiology, 163–164
Early institutional care, impact, 178
Early interventions
developmental hypotheses, relationship, 183–184
impact, 180–183
Early puberty, instrumental variable, 144
Early school intervention, impact, 182
Early stress exposure, animal studies, 174
Early to middle childhood (ages 5–7)
I-Self, impact, 475–477
Jamesian I-Self function, 476–477
Eating-disordered behaviors, 496–498
self-criticism, impact, 497
Eating-disordered behaviors, pathological condition, 472
Eating disorders, 418–419, 491, 590, 642–643
EB. *See* Empirical Bayesian
EBD. *See* Emotional-behavioral difficulties
ECBI. *See* Eyberg Child Behavior Inventory
ECLS-K. *See* Early Childhood Longitudinal Study—Kindergarten Cohort
Ecological fallacy, 1066
existence, 801, 838
Ecological momentary assessment (EMA), 862
data, 979
Ecologic data (contextual/individual effects), confounding (example), 811t
ECoT. *See* Extended children of twins
ECSA. *See* Early Childhood Screening Assessment
Effect modification, 101
Effect modifiers, operation, 101
Effect sizes (ES), 65
Effortful control, pathway, 295
Ego-control, mean growth trajectories (estimation), 1031f
Ego-involvement, 418
Egolessness, 511
Ego-logy, 440
Ego-resiliency, mean growth trajectories (estimation), 1031f
Elders, assessment, 47
EM. *See* Expectation-maximization
EMA. *See* Ecological momentary assessment
EMB children, mathematical performance (growth), 693f
Emergent structures, 704–705
Emerging reciprocation, products, 456–457
Emotion, 265
components, 516–517
context, 302–303
context-sensitive nature, 268
directions (future), 299–305
dynamics, 301–302
emotional expressiveness, development, 273
employment, 513
expression, 283
capacity, 270, 273
expressive control, development, 280–282
facial expressions, presence/ontogeny, 274
functionalist view, 266–267
infant self-regulation, 291
inhibition, 282
integrative regulation. *See* Negative emotions.
lability negativity, latent change score model, 1025f
management, 513
nature, 266–268
nonverbal emotional expressiveness, 274–277
perception, 513
personal relevance, 300–301
psychopathology, relationship, 268–269
role. *See* Positive emotions.
suppressive regulation, 409–410
talk, 277
time course, 301
translational implications, 303–305
understanding, 282–289, 513
development, 283–285
emotional dysfunction risk, association, 285–289
verbal emotional expressiveness, development, 277–280
Emotional action, direction, 696
Emotional awareness, 288
Emotional-behavioral difficulties (EBD)
development socioemotional adjustment. *See* Children.
neurobiological approaches, strengths/limitations, 687
prototypical responsive/nonresponsive learning sessions, interaction dynamics, 693f
self-sustaining learning trajectories, 686–695
students
school performance, modeling, 694–695
students, arithmetic learning, 691–692
Emotional-behavioral problems, neurobiological contributions, 687
Emotional competence, 269–270, 513–514
components, development, 273–282
development, 270–299
developmental pathways, 289–290
domains, 283
emotional dysfunction, relationship, 300
functional perspective, 304–305
prenatal period, 270–273
emotional dysfunction, risk (association), 272–273
Emotional control, 407
Emotional correlates, 501
Emotional creativity, 514–516
Emotional development, functional perspective, 265–266
Emotional disorders, natural kind designation (question), 57
Emotional dysfunction, 268
assessment, 303–304
pervasiveness, 269
risk, 288
avoidance, 276
sequential analyses, 301–302
Emotional dysfunction risk
emotional competence, association, 272–273
emotion regulation, association, 294–299
emotion understanding, association, 285–289
expressive control, association, 281–282
nonverbal emotional expressiveness, association, 275–277
verbal emotional expressiveness, association, 278–280
Emotional dysregulation, 407
Emotional efficacy, 513
Emotional expressiveness, 273. *See also* Nonverbal emotional expressiveness
capacity, 282

development. *See* Verbal emotional expressiveness.
modulation, ability, 280
Emotional functioning, 266
Emotional insecurity
interparental sources. *See* Children.
longitudinal studies, 237
signs, potency, 216–217
Emotional integration, 407
autonomy, antecedent, 408–409
development (social contexts), 409–411
Emotional intelligence, 513
Emotionally reactive children, regulatory strategies (usage), 295
Emotional problems, taxonomies (construction), 57–58
Emotional reaction, impact, 473
Emotional reactivity, 359–360
designation, 216–217
Emotional regulation, 273
SDT conceptualization, 407
Emotional reliance, 390
Emotional security
child characteristics, 229–232
child temperament/personality, contrast, 207–208
conceptualization, differences (EST-R/EST), 206–207
contextual characteristics, 223–235
ecological characteristics, 232–235
EST conceptualization, conceptual pitfalls/barriers, 203–204
ethological differentiation, 204–206
expansion, 246–247
family processes, 224–229
mediation, 209–223
role, research, 209, 212–214
mediator signs, studies/findings, 210t–212t
preservation, latent goal (bidirectional associations), 202f
substance, historical perspective, 201–209
system, characterization (advances), 204–209
Emotional security theory (EST), 199
centering, 247
clinical targets/approaches, delineation, 250–251
contextual characteristics, 223–235
developmental parameters, 235–243
developmental pathways/trajectories, 241–243
experiential histories, 237–239
insecurity, transactions, 236–237
sensitive periods, 239–241
developmental psychopathology, relationship, 199
directions (future), 243–251
emotional security, conceptualization (differences), 206–207
goal-corrected system, 201–203
integration, 233
intervention, 249–251
pathways/processes, proposal, 200f
person-based taxonomy, 243–246
pitfalls/barriers, 203–204
prevention, 249–251
process-oriented approach, 249
public policy, 249–251
Emotional security theory reformulation (EST-R), 201
emotional security
conceptualization, differences, 206–207
system, characterization (advances), 204–209
Emotional understanding, 273
Emotion-focused coping, 290
Emotion regulation, 283, 396, 406–410
capacities, development, 409
capacity, 360
development, 289–299, 387
caregiving, impact, 296–297
prenatal period, role, 290
emotional dysfunction risk, association, 294–299
growth, 293
impact, 1025f
styles, contrast, 406–407
Emotion Regulation Checklist (ERC), Lability/Negativity subscale, 1023–1024
Empirical Bayesian (EB) growth parameter estimates, 961–962
Empirically based constructs, nosologically based constructs (gaps), 83–85
Endogenous variables, 853
English and Romanian Adoptees (ERA) study, 139
Enmeshed families, defining, 614
Enmeshment, 589
profile, 228–229
Entity theory, 505
Entropy, 736–737, 1028
values, LCGA analysis results, 730f
Environment
change, adoption (impact), 139–140
differences, 669
facilitation, 390–396
nested levels, 809
sameness, 451
system, dynamics. *See* Person-environment system.
Environmentality, 669
Environmental mediation hypotheses
association, noncausal alternative explanations, 130–132
cause, meaning, 129–130
natural experiments, 132–148
testing, natural experiments (usage), 129
Environmental risk feature
environment (change), adoption (impact), 139–140
identification strategies, 138–142
risk
universal introduction, 142
universal removal, 142–144
time-series designs, 140–142
twin-singletown comparisons, 138–139
Epidemiology. *See* Shoe-leather epidemiology
defining, 94–96
definition, 94
developmental epidemiology, 94
developmental method, 111–113
exposure, 111–112
genetic epidemiology, 119–121
research, characteristics. *See* Psychiatric epidemiology.
risk, 111–112
time, meaning, 111–112
translational epidemiology, 121
Epigenesis. *See* Adaptive behavior; Maladaptive behavior
idea, comparison, 103
Epigenetic Relational Systems model, 591
Epigenetics, 120–121
processes, Wynne model, 592f
Equal Anger Effect model, 1018
Equal Behavior Problem Effect model, 1018
Equal Maltreatment Effects, 1006–1007
Equifinality, 130, 987
Equifinality-multifinality (developmental pathways), latent growth modeling studies (usage), 1003–1009
ERC. *See* Emotion Regulation Checklist
Ergodicity, 854
problem, 668–669
EST. *See* Emotional security theory
EST-R. *See* Emotional security theory reformulation
Ethnic diversity. *See* Family systems
Ethological parameters, interparental/parent-child relationships (security comparison), 204t
Ethological systems, cascading effects, 221–223
Etiological factor, 387
Euclidean distance, 832
Event history analysis, 731–738
interactive sequence, 731
usage, 731f
Events, prediction, 912
Evidence-based intervention, 701–704
Exemplary dyads, recurrence plots, 729f

Exhaustive clustering, nonexhaustive clustering (contrast), 830–831
Exogenous variables, 853
Exosystem, 809
Expectation
  deviation, concept, 877
  discrepancies, 869
Expectation-maximization (EM), 762t
  bootstrapped EM, 770–771
Experience
  feeling, origins, 440–442
  primordial possession, 464–465
Experiential awareness. *See* Birth
  conceptual awareness, contrast, 448–449
Experiential histories, 237–239
Exploratory factor analyses, 331
Exploratory system, 221–222
Exponential decay latent growth curve model
  parallel process exponential decay latent growth curve model, 971f
  variables/direct paths, 970f
Exponential decay models
  moderation/mediation, 967–970
  usage, 969
Exponential decay structural equation model, 945–946
Exposures, 131
  duration, 113
  intensity, impact, 113
  timing, 112
  timing/duration/intensity, assessment, 171
Expressive control
  development, 280–282
  emotional dysfunction risk, association, 281–282
Extended children of twins (ECoT) design, 129, 135–136
Extended family, 604–605
Extensions. *See* Configural frequency analysis; Scores
External boundary, integration, 406
External information, usage, 877
Externalization, assessment, 695
Externalizing behaviors, 687
  development, 638
Externalizing children (EXT), 747–748
Externalizing disorders. *See* Adolescents; Young adults
  internalization, impairments, 420–422
Externalizing problems. *See* Adolescents; Young adults
  association, 361–362
Externalizing symptoms, 534–537
  comorbidity, 496
  development, insecurity (impact), 353
Externally controlling methods, 391
Extrafamilial stressors, 238
Extrinsic aspirations, 413
Extrinsic motivation, intrinsic motivation (contrast), 505
Extrinsic regulation, modes, 403–404
Eyberg Child Behavior Inventory (ECBI), 20

FACES. *See* Family Adaptability and Cohesion Evaluation Scale
Face-to-face exchanges, 457
Face-to-face proto-conversational exchanges, 460
Face validity, 14
Facial babbling, 274
Facial palsy, 275
Facilitative environments, 387–396
Factor analysis, 854–858
Factorial invariance, testing sequence, 857
Factor loading matrix, 999
Factor modeling, 847
Factor models. *See* Longitudinal data
  dynamic factor model, 857–858
  P-technique factor models, 857
Factor structure, 14
False self, 415
  behavior, prevalence, 487
Familial environment, impact, 421
Familism, values (cultural derivation), 603
Family (families)
  assessment, 5–6
  boundary conceptualizations, utility, 228–229
  coercion, 728–729
  cohesion, index. *See* Revised Family Cohesion Index.
  commitment, 591
  composition, diversity, 604–606
  constitution, 604
  context, long-term outcomes (intergenerational transmission/implications), 650–651
  coping, 599
  cross-cultural perspectives, 602–603
  defining, 604–606
  enmeshment profile, 228–229
  extended family, 604–605
  family-oriented assessment/interventions (psychopathology), 85–86
  family-oriented interventions, 47
  fictive kin, 604–605
  grandparent-headed households, 606
  instability/cohesion, 227–228
  interaction
    child, contribution, 609–610
    dimensions, 608
  observational research, analytic strategies, 610–613
  open systems, 590–590
  organizational patterns, 598
  organized systems, 588–589
  perspective. *See* Functional family perspective.
  projection process, 582
  psychosocial development, 584
  research, methodological advances (relevance), 621–622
  resilience, 598–599
  self-efficacy, 599
  social learning perspectives, 585
  structural types, 613–615
  structures, diversity, 604–606
  systemic constructs, 581
  systemic research methods, 606–613
  three-generational families, 605
  translational research, 621–622
  violence. *See* Hidden family violence.
  vulnerabilities, 598
Family Adaptability and Cohesion Evaluation Scale (FACES), 606
Family factors, inclusion, 593–595
  Diagnostic and Statistical Manual (DSM), 593
  Diagnostic and Statistical Manual for Primary Care (DSM-PC), 593–594
  Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood, Revised (DC:0–3R), 594
  Research Domain Criteria, 594–595
Family-level constructs, 586–587
Family Life History, 591
Family Life Impairment Scale, 36
Family processes, 224–229. *See also* Siblings
  analysis, level, 607–608
  coparenting conflict, relationship, 617–618
  family-level characteristics, 227–229
  fathering, 619–620
  gender dynamics, 619–620
  marriage, spillover, 615
  parental psychopathology, 226–227
  parent-child relationship properties, 224–226
  participant reactivity, 608–609
  self-reports, 606–607
  setting/task, 608–609
  spillover, 619–620
  theory, role, 607
  variations, 620
Family system constructs
  boundary dissolution, 618–619

empirical investigations, 613–619
global family qualities, 613–615
cohesion, 613
family structural types, 613–615
hierarchical organization, 613
marriage, spillover, 615–617
research, biological processes (incorporation), 621
triangulation, 615–617
Family systems
Borzormenyi-Nagy, contextual approach, 583
change processes, impact, 591–592
cultural/ethnic diversity, 601–603
diagnosis, alternative approaches (systemic perspective), 595–597
dynamic systems approaches, 611–613
fathers, involvement, 601
frameworks, impact, 228
global assessment of relational functioning (GARF), 595
historical changes, impact, 603–604
MRI Group, 582–583
observational coding, 607–610
perspective. *See* Psychopathology.
relational diagnoses, 595–596
relational disorders, classification (proposal), 595–596
relational syndromes, 596
Family systems, developmental psychopathology
convergence, 597–601
points, 597
divergence, 597–601
points, 597
emergent synthesis, 597–601
perspective, 580
Family systems theory, 580–581. *See also* Bowen family system theory
analysis, levels, 598
challenges, 601–606
developmental psychopathology, impact, 597–601
development, infusion, 599–601
feminist critiques, 601
historical origins/theoretical underpinnings, 581–586
impact. *See* Developmental psychopathology.
process, focus, 597–598
progenitors, 581–582
role, 607–608
Family theory. *See* Structural family theory
boundaries/subsystems, 589–590
circularity, 588
enmeshment, 589
homeostasis, 588
intrusiveness, 590
parentification, 589–590
relationships, relationships (effects), 587–588
spousification, 590
systemic characteristic, 586–592
Fathering, 619–620
Fathers
involvement, 601
teen fathers, 605–606
Father-son relationship, 616
Fear
density plots, 834f, 835f
dichotomized variable, cross-classification (space-segment clusters), 836t
FearD, cross-classification, 836t
Feelings, sameness, 452
Feeling structures, order/variability/disorder (self-organization), 704–707
FFT. *See* Functional Family Therapy
Fictive kin, 604–605
Field theory, 814–815
File drawer problem, 1053–1054
Filial responsibilities, 590
FIML. *See* Full information maximum likelihood
First differences, 878
First-order CFA, 869t, 871–872, 875t. *See also* Two-state anxiety questionnaire parallel forms
base model, 872
goodness-of-fit, 905
beer consumption, 904t
cross-classification, 911t
DV cross-classification, 913t
example, 881t
liquor consumption, 904t
monotonic trend information, cross-classification, 888t
mood ratings, 903t, 904t
numbers, cross-classification, 896t
subjective health, numbers (cross-classification), 896t
First-order cross-legged vector autoregression model, 854f
Fit indices, 1030t
Fit, meaning, 368–369
Fitted growth curve models, comparisons, 1004t, 1006t
5-HTTLPR, 177
alleles, 366
gene, short variant (possession), 167, 344
polymorphism, 74, 343
5-HTT serotonin transporter, 120
Five-factor model (FFM), 798
Fixed-effects model, 1059–1060
example, 1059f
random-effects model, contrast, 1061–1062
Fixed effects models, estimation, 140–141
Fixed forms, 667–669
averages/aggregates, problem, 667–669
psychopathology, 667
transformation, 669
Flow, concept, 506
FMI. *See* Fraction of missing information
fNIRS. *See* Functional near-infrared spectroscopy
Formal diagnoses, 59, 77
Fostering, usage, 147–148
Four-variable Karnaugh map, schematic, 733f
Four-wave exponential decay latent growth curve model, 946f
Four-wave latent change score model, 944f
Fraction of missing information (FMI), 774, 777
Fragile X syndrome, presence, 10
Fragmentation, 509
Fragmented self-functioning, 414
Friends, characteristics, 554–555
Friendship(s), 551–556
assessment, 559
buffering effects, 553
dark side, 555–556
existence, 527
groups. *See* Mixed-sex friendship groups.
number/quality, 647–648
quality, 552–554
Frustration, 543
anger (relation), cusp catastrophe model (impact), 745f
Full information maximum likelihood (FIML), 760, 767–774, 887
analysis, 780
multiple imputation, 768–774
preference, 782
usage, 778, 1003
timing, contrast, 782
Functional family perspective, 584–585
Functional Family Therapy (FFT), 584–585
critiques, 815
Functional flexibility, 508
Functional impairment, evaluation, 36
Functional magnetic resonance imaging (fMRI), 531
scans, examination, 553
studies, 363
usage, 401, 541
Functional near-infrared spectroscopy (fNIRS), 389
Functioning, psychological level, 666
Funnel plot, 1054

GABRA-6 (CC genotype) genes, 355
GARF. *See* Global Assessment of Relational Functioning
Gatekeeper item, 764
Gateway hypothesis, 134
Gay lesbian bisexual and transgender (GLBT) youth, self-descriptions, 646
Gender dynamics. *See* Family processes
Gene-environment correlation, 346, 621
General anxiety disorder (GAD), 635–637
Generalized learning curves, 995
Generalized linear model (GLM), 822–823
Generalized other, 491
Generalized partial credit model (GPCM), 1071
General Systems Theory, 718
Generation R Longitudinal Study ("R" = Rotterdam), 74–75
Genetic epidemiology
  epigenetics, 120–121
  mechanism, 119–121
  molecular genetics, 120
  psychiatric behavioral genetics, 119–120
Genetic mediation, 130
Genetic variations, impact, 270
Genome-wide association studies (GWAS), 344, 1053
GIMME, usage, 861–862
GLM. *See* Generalized linear model
Global Assessment of Relational Functioning (GARF), 595
Global CFA base models, 872
Global family qualities. *See* Family system constructs
  cohesion, 613
  family structural types, 613–615
  hierarchical organization, 613
Global mental health, 116
Global models (log-linear CFA base model), 871
Global self-esteem
  reduction, 501
  self-evaluations, 489–491
  subscale, usage, 493
Glucocorticoid receptor (GR), 540
Goal-directed change, concept, 105
Goodness-of-fit likelihood ratio, 869, 875, 890, 904
  impact, 910–911
Goodness-of-fit model, 1026
Grandparent-headed households, 606
Great Smoky Mountain Study, 113
Grid-to-grid Euclidian distance scores, 750
GridWare, 748, 750
Group-based individual differenced models, usage, 849–854
Group-based subjects x variables, results, 849
Group home, staff-child interaction (dynamics), 703f
Grouping variable, 911t
Group Iterative Multiple Model Estimation (GIMME), 861
Group Motivational Interview (GMI), 1068, 1073
Growth
  autonomy/psychopathology, relationship, 385–387
  linear growth, 1001
  quadratic growth, 1001
Growth curve
  bivariate growth curve model, 1010f
  designs, 67–70
  linear latent growth curve model, 851–852
  multilevel growth curve level, 850–851
  time-varying covariate growth curve model, 1011f
Growth mixture model (GMM), 70–72, 991, 1027–1029
Growth mixture modeling, 1027–1036
  analysis levels, 1034–1036
  implications, 1035–1036
  studies, 1029–1032
  usage, 1032–1034
Growth models, 1001–1002, 1028, 1036
  inherently nonlinear models, 1001–1002
  latent growth modeling framework, direct expansions, 1002
  linear growth, 1001
  multiple group growth model, 1002–1003
  quadratic growth, 1001
GWAS. *See* Genome-wide association studies
GxE predictions, 343

Habitual reactions, 867
Hamilton Rating Scale for Depression, 1026
Hamming distance, 734
Happy Couples, Happy Kids (HCHK) program, 250
Harter's Self-Perception Profile for Children, 1007
Harmonization, 1054–1055
Head Start effects (study), regression discontinuity (usage), 147
Health/illness, SDT pathways, 386–387
Health risk behaviors, peer acceptance/rejection (relationship), 541–542
Healthy differentiation, 511
  promotion, 510
Helplessness, 206
Heterogeneity, ignoring, 799
Heteronony, 387
Heterotelie, 813
Heterotypic continuity, comorbidity (presence/absence), 114t
Hidden family violence, 677, 679–683
Hierarchical clustering algorithms, 828–829
Hierarchical clusters, nonhierarchical clusters (contrast), 828–829
Hierarchical linear modeling (HLM), 740
  analysis, usage, 69–70
  benefits, 70
  usage, 68–69
Higher level scores, series (description), 887
  characteristics, 887
  early completion, 887
  qualitative characteristics, 887
  trend, 887
Higher-order abstractions, construction, 480
Histrionic exhibitionism, 487
HLM. *See* Hierarchical linear modeling
Holding therapies, 367
Holism, 586–587, 805
Holland-Copenhaver method, 884, 886, 890
Homeostasis, 588
Homicidal ideation, predictors (path-analytical model), 500f
Homogeneity, 828
Homophily, 559
Homophobia, 646–647
Homotypic continuity, comorbidity (presence/absence), 114t
Hope, impact, 516
Hopelessness, 206
  impact, 495
Horizontal relationships, 527
Hostility, 640
Hubert-Arabie-adjusted Rand index, 829
Human autonomy, spontaneous expression, 399–403
Human behavior, ecological context, 809
Human brain development, findings, 166
Human Connectome Project (HCP), 1081
Human genome, complexity, 270
Human health/development, animal findings (translation), 174–175
Human interaction, insufficiency (effects), 286
Human prenatal environment, research, 272
Human psychological functioning, complexity (comparison), 805
Human studies, causal effect (establishment), 176
Human symbolic psychology, 459–460
Humiliation, 473, 498–503. *See also* School system
  dimensions, 500–501
  emotional correlates, 501
  role, 499–502
Humility, role, 512
Hyperactivity, cross-classification (first-order CFA), 875t
Hypothalamic pituitary adrenal (HPA) activity, 69
Hypothalamic pituitary adrenal (HPA) axis, 172, 355, 538–539
  activity, stressors (impact), 548
  function, 176, 183
  inhibition, 540

modification, 164
recovery, 554
responses, peak, 549
stressor reaction, 554
Hypotheses, specificity (increase), 225

I^2 test, 1060
ICC. *See* Intraclass correlation
ICD-11. *See* International Classification of Diseases, Eleventh Revision
IDA. *See* Integrative data analysis
Identified regulation, 403
Identity
development, fostering, 412
formation, 387, 396, 410–413
SDT viewpoint, 410–411
types, qualitative differences, 412–413
Idiographic Filter (iFACE), 861
iFFA. *See* Inventory of the Forms and Functions of Aggression
Illness, burdens, 118
Imagining, process, 459
Immaturity, uses, 451
Impairment (young children), assessment, 35–36
Implicit/explicit attitudes/motives, alignment/congruence, 408–409
Imputations. *See* Multiple imputation
number, 774
Inattention
latent change score model, example, 1020f
moderation, latent change score model (usage), 1019f
Incipient synthesia, 447
Inclusion criteria, 1052–1053
Inclusive strategy, 776
Independence
autonomy, contrast, 389–390
deviation concept, selection, 876
family pattern, 602
Indicator matrix, 870
Indicator variable, 689
Individual, concept, 805
Individual configurations, CFA tests (usage), 873–874
Individual growth curve analysis, 68
Individual growth modeling, 995
Individuality, concept, 805
Individual participant-level data (IPD), 1043
AD, contrast, 1044
availability, 1044–1045, 1048
discussion, 1043
involvement, 1052
meta-analysis, 1044, 1051, 1061
challenge, 1055
studies, 1046
usage, 1065–1066
multivariate meta-analysis
covariates, patterns, 1075t
parameter estimates, 1075t
pooled IPD, analysis, 1047
usage, 1044f, 1052
Individual problem behaviors, prevalences, 108
Individual trajectories, 706
base models, 912
comparison, 908–912
data example, 910–912
extensions, 912
individual developmental trajectories, 908f
Individuation, increase (indication), 389
Infancy
cognitive science, impact, 453
peer interactions, 530–533
Infant-caregiver interaction quality, 276
Infantile amnesia, 453–455
Infants
action, predisposition, 448
attachment, 396–398
differences, 329
autonomy, 396–398
caregivers, interactions, 274
contrast sensitivity/grating acuity, 448–449
differences, conceptualization, 290–291
emotion, self-regulation, 291
interpersonal conversation, 455
resistance, 329
self-objectification, 457
temperament, 298
difficulty, 341
visual world, attendance, 450
Infant-Toddler Social Emotional Assessment (ITSEA), 12, 28
functioning assessment, 24
response format, 17
Inferior frontal gyrus (IFG), 364
Inferiority, 206
Inflammation, impact, 540–541
Information processing, psychological mechanisms, 169
Ingratiation, 206
Inhelder, B., 724
Inherently nonlinear change patterns, 999–1000
Inherently nonlinear models, 1001–1002
Inhibited-victor aggressor pathway, 700–701
Inhibition, development pathways, 699–701
Insecurity. *See* Emotional security
cascade mechanisms, 244
multilevel cascade models, complexity, 220
outcomes, 217
protection/resilience, 247–249
signs, methodological approaches, 216
Insecurity, transactions, 236–237
Intrinsic interests, impact, 411
Instruction
class-based nature, 692
short-term continuity, 693
Instrumental variables, 129
Integration
enhancement, 404
movement, 676–677
Integrative data analysis (IDA)
aggregated data (AD), usage, 1044f
analytical approaches, 1046f
data examples, 1067–1080
directions/limitations, 1080–1081
individual participant-level data (IPD), usage, 1044f
pooled data, 1068–1071
research synthesis, 1051–1067
methods, 1045t
translational implications, 1080–1081
usage, 1042, 1065–1066
Integrative emotion regulation, capacity, 408
Intelligence. *See* Emotional intelligence
Intelligence quotients (IQs), relative rankings, 669
Intentional communication, emergence, 461
Interaction dynamics, 693f
Interaction models, proposals, 226–227
Interaction sequences, graphical depiction, 733–734
Interactive effect model, 1008
Interdependence
family pattern, 602
focus, 805
Interest, collective variable (identification), 723
Intergenerational boundaries, rating, 613
Intergenerational studies, 116–117
Intergenerational transmission, 650–651
Intergrid distance score, 750
Interindividual changes, 1004
Interindividual differences, 800

Interindividual longitudinal analysis
autoregressive latent trajectory mediation models, 963–964
autoregressive mediation model, longitudinal/contemporaneous mediation, 954t, 957t
autoregressive model, 942–943
cross-lagged paths, 943f
autoregressive panel models, longitudinal mediation, 953–958
causal inference, 973–978
controlled direct effect, 975
Early Childhood Longitudinal Study—Kindergarten Cohort (ECLS-K) data examples, 924–925
exponential decay structural equation model, 945–946
four-wave latent change score model, 944f
latent change model, 944–945
latent growth curve (LGC) mediation model, 958–961
multilevel model estimation, 961–963
latent growth curve (LGC) model, 943–944
longitudinal data, 933–934
moderation/mediation models, 946–973
multilevel modeling approaches, 939–941
structural equation models, 942–946
longitudinal mediation models, 973–978
mediated effect over time, significant/nonsignificant coefficients (combinations), 952t
moderation/mediation, 922
latent change score models, 964–967
multilevel growth models, time-varying covariate interactions, 948–950
multilevel modeling, 934–939
multilevel models
mediation, 937–939
moderation, 936–937
multiple-indicator latent change score model, 945f
natural direct/indirect effects, assumptions, 975
outcomes model, 974–975
Interindividual variation
modeling, group-based individual differenced models (usage), 849–854
Interindividual variation, intraindividual variation (contrast), 848–849
Interleukin-1 (IL-1), impact, 165
Interleukin-6 (IL-6), impact, 539, 541
Intermodal systems, existence, 446
Internal boundary, integration, 406
Internal consistency, 12–13
Internalization, 387, 396, 403–406
assessments, 695
autonomy, relationship, 404
continuum, 403–404
enhancement, 404
impairments, 413–414, 420–422
promotion, 406
social contexts, relationship, 404–406
Internalizing behavior, 687
Internalizing disorders, 414
Internalizing symptoms, 537–538
biological pathway, 538–541
comorbidity, 496
early insecurity, 353
Internally controlling regulation, 413
Internally controlling states, involvement, 414–420
Internal states, 475
Internal working models (IWMs), 332, 349–350
International Classification of Diseases, Eleventh Revision (ICD-11), relational diagnosis (usage), 594, 596–597
Interparental conflict, 200
child adjustment, emotional security mediation, 209–223
signs, studies/findings, 210t–212t
child emotional insecurity, interparental sources, 214–216
child insecurity
temporal dimensions, interplay, 242
child insecurity, sequelae, 217
children vulnerability, 229
destructiveness, 224–225, 612–613
developmental cascade mechanisms, 217–223
approach, ethological systems (cascading effects), 221–223
defensive analogs, proliferation, 218–219
stress response system, alterations, 219–221
dysphoric reactivity, 217
emotional security, mediational role (research), 209, 212–214
interplay, systematic analysis, 215
mediational pathways, emotional insecurity signs (potency), 216
parent-child boundary dissolution, 619
parent-child dysfunction, association, 587–588
responses, organization, 244
temporally ordered lagged analyses, 212–213
Interparental discord, 616
Interparental dyad, emotional security (expansion), 246–247
Interparental/parent-child relationships (security comparison), 204t
Interpartner conflict, exposure, 287
Interpersonal contexts, threats, 247
Interpersonal distance, 602
Interpersonal process, 409
Interpersonal Psychotherapy—Adolescent Skills Training (IPT-AST), 654
Interpersonal Psychotherapy for Adolescents (IPT-A), 654
Interrater reliability, 13
diagnostic interviews, 33–34
Interrelational level, 475
Intersubjectivity. *See* Primary intersubjectivity; Secondary intersubjectivity; Tertiary intersubjectivity
levels, 456t
roots, 455–463
tertiary level, 456
Intervention. *See* Parenting intervention
adult attachment, 367–368
attachment, 364–369
comparisons, multivariate meta-analysis, 1078f
framework, 325–326
groups/procedures, 1068
programs, autonomy/autonomy support, 425–426
reflective functioning, 367–368
study designs, impact, 171
Intervention science
growth mixture modeling, implications, 1035–1036
latent change score modeling, implications, 1026–1027
latent growth modeling, usage, 1013–1015
Interviewer-based interviews, 110
Interviews
interviewer-based interviews, 110
reliability, 30
respondent-based interviews, 110–111
usage. *See* Adolescents; Children.
Intimate partner violence (IPV), 638, 640–641, 653
alcohol/drug use, 640–641
anger, 640
antisocial behavior, 640
conduct problems, 640
experience, biopsychological profiles, 807f
hostility, 640
Intraclass correlation (ICC), 934–935
Intraindividual change
determinants, 995
interindividual variability, 994–995
modeling, 993–994
Intraindividual differences, 800
Intraindividual variation
definition, 848
interindividual variation, contrast, 848–849
modeling, person-specific approaches, 846
Intrinsic motivation, 387, 396, 399–403
contextual sensitivity, 402
definition, 399
effects, 401–402
extrinsic motivation, contrast, 505
free-choice measure, 400

significance, 402
social contexts, relationship, 399–401
Introjection, 417–418
basis, 403
involvement, 414–420
Introjects, understanding, 415
Introspection, tendency, 491
Intrusiveness, 590
INUS, 130
Invariant model, 1003
Inventory of the Forms and Functions of Aggression (iFFA) survey, 786–787
Inverse probability to treatment weighting (IPTW), 131, 141
Inverse probability weighting, longitudinal data (usage), 977
Invulnerability, sense, 485
Inward unity, sense, 508
Iowa Family Interaction Rating Scales, 608
IPD. *See* Individual participant-level data
Ipseity, 465
IPT. *See* Interpersonal Psychotherapy
IPTW. *See* Inverse probability to treatment weighting
IPV. *See* Intimate partner violence
Irons, D.E., 145
Irritability, 867
IRT. *See* Item response theory
I-Self, 471
cognitive acquisitions, 480
conspiracy, 481–488
functions, 489–491. *See also* Jamesian I-Self functions; James's I-Self functions; Self-coherence.
impact, 473–481
perspective, 518
processes, 470, 476. *See also* Self-efficacy.
Buddhist perspective. *See* Mindfulness.
developmental prerequisites, 481
emergence, 479
role, 503
Isolating agents, 680–682
Isolating dissociation, 678
Isolating personalities, 682–684
Item response theory (IRT), 800, 1055
Iteration, 689
Iterative feedback components, representation, 741f
ITSEA. *See* Infant-Toddler Social Emotional Assessment

Jamesian I-Self functions, 474–483
compromise, socialization practices (impact), 488
impact, 475–481
James's I-Self functions, 474–475
Johnson-Neyman technique, Pothoff extension, 927
Joint antitype (composite antitype), 875
Joint problem solving, 592
Journal article reporting standards (JARS), 1052
Joussemet, M., 416, 421
Joy
density plots, 834f, 835f
dichotomized variable, cross-classification (space-segment clusters), 836t
JoyD, cross-classification, 836t
Juvenile-onset MDD, 115

Karnaugh maps, 733–734
example, 612f
schematic, 733f
Kiddie Schedule for Affective disorders and Schizophrenia (Kiddie-SADS), 53, 110
k independent studies, 2x2 tables (model setting), 1056t
k-means method, 829
Knock-out mouse model, usage, 165
Kolmogorow-Smirnow test, 740
K-SADS. *See* Kiddie Schedule for Affective disorders and Schizophrenia; Schedule for Affective Disorders and Schizophrenia
K-SADS-PL. *See* Schedule for Affective Disorders and Schizophrenia—Present and Lifetime

Lability/Negativity subscale, 1023
Lag as moderator model (LAM), 946–948
approach, 923
multilevel LAM models, coefficients/standard errors, 948t
Lagged regression models, 852–853
Lags
associations, 905
autocorrelograms, 909f
beer consumption, cross-classification, 902t
CFA, 900–905
results, 904–905
contemporaneous associations, 905
correlation matrix, 905t
cross-lagged designs, CFA, 905–908
data example, 901–904
liquor consumption, cross-classification, 902t
log-linear modeling, results, 904
moderator model, 946–948
mood ratings, cross-classification, 903t
series, 901t
LAM. *See* Lag as moderator model
Language, 670
development, 277
emergence, 482
Late adolescence (ages 17–19)
I-Self, impact, 480
Jamesian I-Self function, impact, 480–481
self-enhancement, 486–488
Latent basis, 999
growth model, 1004
Latent change model, 944–945
four-wave latent change score model, 944f
multiple-indicator latent change score model, 945f
Latent change score (LCS)
mediation models, coefficients/standard errors, 967t
modeling, 996
studies, usage, 1018–1027
usage, 1022–1023
parallel process latent change score mediation model, 965f
parallel process mediation model, coefficients/standard errors, 966t
Latent change (difference) score modeling, 1015–1027
Latent change score (LCS) models
bivariate latent change score models, 1017
example, 1019f
moderation/mediation, 964–967
univariate latent change score models, 1015–1017
Latent change, true scores (paths), 967t
Latent class analysis (LCA), 740–746, 833
Latent class mixture models (LCMMs), 833
Latent curve modeling, usage, 1011–1012
Latent growth curve (LGC) mediation
estimation, multilevel model, 961–963
model, 958–961
example, 960f
Latent growth curve (LGC) model, 943–944
example, 997f
exponential decay latent growth curve model, variables/direct paths, 970f
four-wave exponential decay latent growth curve model, 946f
L1/L2 predictions, example. *See* Structural equation modeling.
parallel process exponential decay latent growth curve model, 971f
Latent growth curve (LGC), structural equation model, 996–997
Latent growth modeling, 995–1000
analysis
levels, latent curve modeling (usage), 1011–1012
multiple levels, 988
approach, core theoretical principles, 986–989
developmental psychopathology, relationship, 986

Latent growth modeling, *(Continued)*
framework, direct expansions, 1002
height, changes (longitudinal plots), 992f
historical development, 995–996
implications, 1013–1015
interindividual change, 993–994
interindividual variability, 994–995
intraindividual change
determinants, 995
modeling, 993–994
issues, 990–995
longitudinal descriptive analyses, 989–995
longitudinal research, primary goals, 989
longitudinal study, planning/sampling, 989–990
measurement, 990–991
pathways framework, 987
questions, 990–991
resilience, 987–988
studies, usage, 1003–1015
time metrics, 991–993
usage, 1003–1012
Latent growth models
inherently nonlinear change patterns, 999–1000
latent basis, 999
linear change, 998
multiphase change, 1000
quadratic change, 998
time, transformation, 998–999
types, 997–1000
unstructured change, 999
Latent variable grouping, manifest variable grouping (contrast), 833
LCA. *See* Latent class analysis
LCGA analysis results, 730f
LCMMs. *See* Latent class mixture models
LCS. *See* Latent change score
Learning
dynamics, 687–689
self-sustaining trajectories. *See* Suboptimal learning dynamics.
embodiment/coaction/initial conditions, 688–689
goals, 505
interactive/microdevelopmental process, 689
LGC. *See* Latent growth curve
LGM mediation model, coefficients/standard errors, 961t
Life-course epidemiology, 116
Life-course studies, 115–116
Life satisfaction, 493
Life span
applications, 72
development, 160–161
perspectives, 47
Likelihood ratio tests (LRTs), 1029
Limbic system, occurrence, 558
Linear growth, 1001
model, 1003–1004
Linear latent growth curve model, 851–852
Linear trend, 885
Linkage
clusters, 833
types, 830
Liquor consumption
first-order CFA, 904t
lag cross-classification, 902t
LISREL, 820, 956
model, usage, 853
notation, 819
usage, 851
Local association, 868, 896
concept, 905–906
Local nonlinear systems, dynamics (understanding), 703
Log-linear CFA base models, 871
Log-linear cross-lagged model, parameter estimates, 907t
Log-linear modeling, 822–823
Log-linear models
observed/expected frequencies, 825t
types, 822–823
Log-linear notation, 870–871
Longitudinal data. *See* Interindividual longitudinal analysis
autoregressive mediation model, longitudinal/contemporaneous mediation, 954t
benefits, 933–934
factor models, 855–857
moderation/mediation models, 946–973
usage. *See* Inverse probability weighting; Potential outcome mediation model.
Longitudinal descriptive analyses, 989–995
Longitudinal developmental context (validity), 14
Longitudinal mediation, 953–958
models, 973–978
Longitudinal research, 114–117
primary goals, 989
Longitudinal study, planning/sampling, 989–990
Longitudinal twins and parents (LTaP) design, 129, 136
Low power, issues, 1066
Low self-esteem
perceived directionality, 496
perceptions, alteration, 491–493
Low self-esteem, pathological condition, 472
LRTs. *See* Likelihood ratio tests
LTaP. *See* Longitudinal twins and parents
Lurking variables, 802

Macrolevel coding, disadvantages, 607
Macrosystem, 809
MAGIC. *See* Missouri Assessment of Genetics Interview for Children
Major Depressive Disorder (MDD), 4, 115, 1032–1033
Maladaptive behavior, structure (epigenesis), 672–673
Maladaptive explanatory styles, usage, 537
Maladaptive functioning, involvement, 269
Maladaptive Index (ITSEA), 12
Maladaptive interaction pattern, 563
Maladaptive patterns, development pathways, 696–701. *See also* Children
Maladaptive psychological functioning, 265
Maladaptive socioemotional adjustment, dynamics, 696
Maltreated children
abuse, 678–679
abusive relationships, 679
affective dissociation, development, 673–686
attachment-based intervention, effectiveness, 366
coordination
growth, modeling, 684–686
modeling, 686f
depressive symptoms, fitted growth curve models (comparisons), 1004t, 1006t
development, webs, 675
dissociation
growth, modeling, 684–686
modeling, 686f
hidden family violence, 679–683
isolating dissociation, 679–683
passive/active splitting, dissociation (relationship), 677–678
personality processes/behavioral problems, univariate growth mixture modeling (fit indices), 1030t
psychological activity, natural fractionation, 675–677
psychopathology, developmental immaturity (nonrelationship), 683–684
self-esteem, fitted growth curve models (comparison), 1004t, 1006t
skilled action, structure (developmental changes), 673–675
Maltreatment Classification System (MCS), 1006, 1023
Maltreatment effects model, 1006
Manchester Child Attachment Story Task (MCAST), 358–359
Mania without delirium, description, 106–107
Manifest variable grouping, latent variable grouping (contrast), 833
Manifest/variables interaction, SEM (impact), 942f

Mantel-Haenszel (MH) method, 1056–1057
  estimates, 1059
  usage, 1058t
MAOA. *See* Monoamine oxidase A
MAP-DB. *See* Multidimensional Assessment Profile of Disruptive Behavior
MAR. *See* Missing at random
Marginal-dependent measure, 876
Marginal-free measure, 876
Markov chain Monte Carlo (MCMC), 561, 1071
  assessment, 773
  Bayesian MCMC, 770
  continuous-time MCMC, usage, 561
  convergence, 773
  simulation techniques, 770
  usage, 773
Marriage, spillover, 615
MARS. *See* Meta-analysis reporting standards
Mastery goals, 505
Mastery motivation, 399, 504–505
MATCH approach, 654
Matches
  changes, mismatches/self-iterations changes (relation), 692f
  growth, analysis, 691f
Matches/mismatches/self-iterative utterances (changes), child/tutor interaction (mathematics instruction), 690f
Maternal Behavior Q-set (MBQS), usage, 338–339
Maternal immune activation (MIA) paradigm, 177
Maternal smoking effects, paternal smoking effects (contrast), 137–138
Mathematical performance, growth, 692f. *See also* EMB children
Mathematics instruction (child/tutor interaction), matches/mismatches/self-iterative utterances (changes), 690f
Maximum likelihood estimator (MLE), 1061
MBQS. *See* Maternal Behavior Q-set
MCAR. *See* Missing completely at random
MCAST. *See* Manchester Child Attachment Story Task
McClelland cascade model, 744
M-CHAT-R/F. *See* Modified Checklist for Autism in Toddlers, Revised with Follow-Up
McMaster Family Assessment Device, 606–607
MCMC. *See* Markov chain Monte Carlo
M-COT. *See* Mediation change over time
MCS. *See* Maltreatment Classification System
Mean aggressive/delinquent behavior syndrome scores (Dutch children), 64f
Mean growth trajectories, estimation, 1031f
Mean separation anxiety symptoms, age/information source basis, 100f
Means Model, 1003
Means+Variances Model, 1003
Measles mumps and rubella (MMR), impact, 143
Measurement error, levels (elevation), 338
Measurement occasion, 989
Measurement stability, 171–172
Measures (series), description (polynomial usage), 884–885
Medial prefrontal cortex (MPFC), activity, 389
Mediated effect over time, significant/nonsignificant coefficients (combinations), 952t
Mediated moderation
  coefficients, 933t
  Fairchild/MacKinnon approach, path diagram, 932f
  standard errors, 933t
  tests, 933t
Mediating process (understanding), latent growth modeling (usage), 1007–1008
Mediation
  analysis, 780–782
    role, 923–924
  autoregressive mediation model, longitudinal mediation, 955t
  coefficients, 931t
  establishment, 929
  genetic mediation, 130
  impact, 941
  lag as moderator model (LAM), 946–948
  latent change score models, 964–967
  latent growth curve (LGC) mediation, estimation, 961–963
  longitudinal mediation, 953–958
  moderated mediation, Fairchild/MacKinnon approach (path diagram), 932f
  moderation
    combination, 931–933
    contrast, 922–923
  standard errors, 931t
  tests, 931t
  usage, 937–939
Mediational pathways
  documentation, 212–213
  emotional insecurity signs, potency, 216–217
Mediation change over time (M-COT) model, 950–953
  coefficients, 953t
  mediated effect trends, 953t
  standard errors, 953t
Mediation model, 928–931
  alternate parallel process latent change score mediation model, 966f
  autoregressive latent trajectory mediation models, 963
    example, 964f
  example, 942f
  fit, 781
  latent growth curve mediation model, 958–961
  parallel process latent change score mediation model, 965f
  path diagrams, 928f
Mediators, risk/protective mechanism function, 1018–1021, 1029–1032
Meehl's paradox, 803
Memory, absence, 454
Mendelian randomization (MR), 129, 145–146
Mental health
  implications, positive psychology movement (role), 512–516
  I-Self
    impact, 473–474
    processes, impact, 470
  Me-self processes, impact, 470
  promotion, I-Self role, 503
  services, quantification (advantages), 81
  surveillance, 118–119
Mental Research Institute (MRI), 582–583, 588
Mental retardation, psychiatric disorder (contrast), 106
Mental synthesis, levels, 443
Me's, conflict, 507
Me-Self
  conspiracy, 481–488
  constructions, 504
  deficiencies, 472
  descriptions, 475–476
  developmental construction, I-Self (positive contribution), 474–481
  functions, 489–491
  processes, 470
Mesoanalytical level analysis, 608
Mesosystem, 809
Messenger RNA (mRNA), 672
  levels, increase, 539
Meta-analysis, 63, 351
  classical approaches, 1055–1056
  IPD/AD, usage, 1044f
  model-based meta-analysis, 1072–1073
  multivariate meta-analysis, 1062–1064, 1073–1079
  network meta-analysis, 1064–1065
  software programs, examples, 1079t
  studies, citing (proportion), 1046f
  term, usage, 1052
Meta-analysis reporting standards (MARS), 1052–1053
Metacognitive abilities, link, 458
Metacognitive self-awareness, 476
Metafor (package), 1079

MetAnalysis, 1079
MFAM. *See* Multicultural Family Assessment Module
MH. *See* Mantel-Haenszel
MI. *See* Multiple imputation
MIA. *See* Maternal immune activation
MICE. *See* Multiple imputation with chained equations
Michigan Alcoholism Screening Test (MAST), 1032
Michigan Longitudinal Study (MLS), 1048
Microsystem, 809
Midadolexcence (substance abuse), regression results (impact), 729t
Middle adolescence (ages 14–16)
  I-Self, impact, 479–480
  Jamesian I-Self functions, impact, 480
  self-enhancement, 485–486
Middle childhood, emotion regulation (growth), 293
Middle-to-late childhood (ages 8–10)
  I-Self, impact, 477–478
  Jamesian I-Self functions, impact, 477–478
Middle to late childhood (ages 8–10), self-enhancement, 483–484
Migration studies, 129, 138
Mind. *See* Theory of Mind
  modularity, 448
  viewpoints, 443
Mindfulness
  cultivation, 511
  I-Self process, Buddhist perspective, 511–512
Min-max graph, example, 739f
Minnesota Multiphasic Personality Inventory-2 (MMPI-2), 362–363
Minnesota Multiphasic Personality Inventory (MMPI) Scores, 67
Mirror game, 455
Mirror mark test, 457
Mirror self-experience, negative affective connotation, 458
MIS. *See* Mother-Infant Study
Mismatches
  changes, matches/self-iterations changes (relation), 692f
  growth, analysis, 691f
Missing at random (MAR), 765t, 766
Missing completely at random (MCAR), 764–766
Missing data
  designs, 784–790
  mechanisms, 765t
    example, 765f
  model fit, assessment, 777–780
  practices, review, 782–784
Missing data methods, 767
Missing information. *See* Fraction of missing information
Missingness
  causes, 776
  mechanisms, 763–767
Missing not at random (MNAR), 764, 766–767
Missing value, distribution (plausibility), 769
Missouri Assessment of Genetics Interview for Children (MAGIC), 110
MIXED (children comorbid), 747–748
Mixed dyad, preperturbation SSGs/postperturbation SSGs, 748f
Mixed models, 935
Mixed-sex friendship groups, 639
MLE. *See* Maximum likelihood estimator
MLM. *See* Multilevel modeling
MMPI. *See* Minnesota Multiphasic Personality Inventory
MNAR. *See* Missing not at random
Möbius syndrome, 275
Model-based approaches, 1057
Model-based estimates, 1076f, 1077f
Model-based meta-analysis, 1072–1073
Model fit, 778
  assessment. *See* Missing data.
Model-implied trajectories, 1013
Moderated mediation
  coefficients, 933t
  Fairchild/MacKinnon approach, path diagram, 932f
  standard errors, 933t
  tests, 933t
  usage, 970–973
Moderated nonlinear factor analysis (MNLFA), 1055
Moderating processes (understanding), latent growth modeling (usage), 1008–1009
Moderation, 925–928
  analysis, role, 923–924
  effects (testing), HLM analyses (usage), 69–70
  forms, 238–239
  lag as moderator model (LAM), 946–948
  latent change score models, 964–967
    example, 1020f
  mediated moderation, Fairchild/MacKinnon approach (path diagram), 932f
  mediation
    combination, 931–933
    contrast, 922–923
  models, coefficients/standard errors, 927t
  potential outcome model, 976
  usage, 936–937, 941–942
Moderators
  gap, 339
  moderator model, lag, 946–948
  risk/protective mechanism function, 1018–1021, 1029–1032
  types, 565
Modernism, 508
Modified Checklist for Autism in Toddlers, Revised with Follow-Up (M-CHAT-R/F), 19
Molecular genetics, 120
  research, 167
Monitoring the Future National Survey, 1034
Monoamine oxidase A (MAOA)
  expression, 120
  impact, 103
Monotonicity, 887
Monotonic trend, 887
Monotonic trend information, cross-classification
  first-order CFA, 888t
  two-group CFA, 888t
Monte Carlo permutation analysis, 690
Montemayor, 474
Mood
  beer consumption, cross-lagged relationship, 906f
  numbers cross-classification, first-order CFA, 896t
Mood ratings
  first-order CFA, 903t, 904t
  lag cross-classification, 903t
Moral insanity, 106–107
Moral, term (usage), 107
Moral transgressions, 500
Mother-child attachment security, stability, 346–347
Mother-child transactions, Karnaugh map, 612f
Mother-father-child triads, observation, 619
Mother-Infant Study (MIS), 868–869, 915
Motivation. *See* Mastery motivation
  contrast, 505
Motivational goals, 505
Motive-relevant changes, 670
Moving min-max graph, 738–739
Moving to opportunity (MTO) housing voucher experiment, 148
MPFC. *See* Medial prefrontal cortex
Mplus software, 778
MR. *See* Mendelian randomization
MRI. *See* Mental Research Institute
MTO. *See* Moving to opportunity
Multculturalism, attention, 567
Multi-causality, theory (evolution), 814–815
Multicultural Family Assessment Module (MFAM), 86
Multicultural norms
  applications, 77
  construction, 76–77
Multidimensional Assessment Profile of Disruptive Behavior (MAP-DB), 24, 29

Multifinality, 130
concept, 924
developmental pathways. *See* Equifinality-multifinality.
principle, 217
Multiform planned missing protocols, 785–787
Multigroup CFA, 873
Multigroup models, focus, 819–820
Multilevel autoregressive (AR) model, 852
Multilevel autoregressive (AR) modeling, 847
Multilevel growth
curve model, 850–851
curve modeling, 849
models, time-varying covariate interactions, 948–950
Multilevel LAM models
coefficients/standard errors, 948t, 950t
time-varying predictor, 950t
Multilevel modeling (MLM), 989
growth models, 1001–1002
specification, alternative, 1001
usage, 1000–1015
Multilevel models, 935
Multiphase change, 1000
Multiple developmental domains, direct assessment (importance), 10
Multiple end points, 1062
Multiple group growth model, 1002–1003
Multiple imputation (MI), 760, 768–774
implementation, software packages (usage), 771t
usage, 778
timing, contrast, 782
Multiple imputation with chained equations (MICE), 771–772
Multiple-indicator latent change score model, 945f
Multiple-mediator models, bootstrap confidence intervals, 931t
Multiple me's, conflict, 508
Multiple selves
functional flexibility, 508
integration/differences, measurement/assessment, 509f
Multiple sensory channels, openness, 444
Multisource data, usage, 87
Multivariate analysis of variance (MANOVA), 831, 1063
Multivariate growth model, example, 1006f
Multivariate LGM, associations, 1021–1022
Multivariate meta-analysis, 1062–1064, 1073–1079
example, 1078f
term, usage, 1052
Multivariate twin designs, 129
natural experiments, 133
Multlinomial sampling scheme, 870
Mutual blind mindedness, 463
Mutual invisibility, 463
Mutuality, 592
Mutual recognition, centrality, 463–464
Mvtmeta (package), 1079
MZ paris, 133–134

Naive comparison, 1071
Naive dialecticism, 510
Narcissism, 517
danger, 485
normative characteristic, 483
pathological condition, 472
treatment, 368
Narratives, construction, 476–477
Narrowed consciousness, cross-classification (first-order CFA), 875t
National Comorbidity-Adolescent Supplement (NCS-A), 644
National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA), 1081
National Health and Nutrition Examination Survey (NHANES-IV), 119
National Health Interview Survey (NHIS), 119
National Institute of Mental Health, Research Domain Criteria (RDoC) framework, 24, 37–38
National Longitudinal Survey of Youth (NLSY), 1009
National Longitudinal Survey of Youth—Children and Young Adults (NLSY-CYA), 991
female participants, sample, 992f
Natural experiments, 132–148
adoption studies, 136–137
assisted reproduction technology (ART), 137
children of twins design, 135
discordant sib pairs, 134–135
discordant twin pairs, 133–134
environment (change), adoption (impact), 139–140
environmental risk feature
identification strategies, 138–142
twin-singleton comparisons, 138–139
extended children of twins (ECoT) design, 135–136
genetically sensitive designs, 133–138
Head Start effects (study), regression discontinuity (usage), 147
instrumental variables, 144–146
early puberty, 144
one-child policy (China), 144–145
limitation, 149
longitudinal twins and parents (LTaP) design, 136
maternal smoking effects, paternal smoking effects (contrast), 137–138
methodology, overview, 149
migration strategies, 138
multivariate twin designs, 133
natural disaster, relationship, 177
prenatal/postnatal effects (separation), adoption/fostering (usage), 147–148
requirements, question, 148–149
schooling influences (assessment), regression discontinuity (usage), 146–147
selection bias
designs, impact, 142–144
Mendelian randomization (MR), 145–146
risk, universal introduction, 142
risk, universal removal, 142–144
unmeasured confounders, regression discontinuity designs (usage), 146–147
usage, 129
Natural fractionation. *See* Psychological activity
Natural killer (NK) cell functioning, enhancement, 166
NCS-A. *See* National Comorbidity-Adolescent Supplement
Need
frustration, psychopathology (relationship), 413–414
need-supportive socialization, 390–396
support, role, 412
Needed scale, provision, 1051
Negative affectivity, dimensions (impact), 231
Negative emotionality, pathway, 295
Negative emotions
integrative regulation, 409–410
naturalistic contexts, 280
regulation, 298
socioemotional adjustment, dynamics, 696f
young children, regulation, 292
Negative predictive value (NPV), 15
Negative rule-break bout slope, time-series (example), 728f
Negative self-evaluations, pathological condition, 472
Negative self-processes, role, 473
Negativity bias, 678–679
Neglect. *See* Parental involvement/neglect
Neglecting responsibilities (NR), 1072
latent trait scores, model-based estimates, 1076f
Nested chi-square difference tests, 1003
*Neta*i, 744
Netherlands Twin Registry (NTR), 75
Network meta-analysis, 1062, 1064–1065
example, 1064f
Neural growth, 441
Neuroimmunology, 164–166
application, 165
Neurophysiological processes, genetic influences, 271

Neurotransmitters, impact, 170
Newborns
  experiential awareness, 448
  feeling, description, 444–447
  functional propensities, 449–450
  self-awareness, 463
NHANES-IV. *See* National Health and Nutrition Examination Survey
NICHD day care study, 183
NLSY-CYA. *See* National Longitudinal Survey of Youth—Children and Young Adults
NMDA gene expression, 167
No-coupling model, 1026
No-growth model, 1003–1004
Nonconscious appraisals, 670
Nonconvex clusters, convex clusters (contrast), 832–833
Nonexhaustive clustering, exhaustive clustering (contrast), 830–831
Nonhierarchical clusters, hierarchical clusters (contrast), 828–829
Nonlinear change, 721–723
Nonlinear dynamics, 736–738, 740
Nonmaltreated children
  depressive symptoms, fitted growth curve models (comparisons), 1004t, 1006t
  dissociation/coordination, modeling, 686f
  personality processes/behavioral problems, univariate growth mixture modeling (fit indices), 1030t
  self-esteem, fitted growth curve models (comparison), 1004t, 1006t
Nonnormative development (complexity assessment), quantitative/qualitative methods (elaboration), 706–707
Nonreferred children, sample, 55
Nonverbal children (infans), 440
Nonverbal emotional expressiveness, 274–277
  emotional dysfunction, risk (association), 275–277
Nonverbal emotional information, recognition ability, 283–284
Normal behavior, abnormal behavior (contrast), 108
Normality
  psychopathology, relationship, 988–989
  viewpoint, 105
Normative data
  consideration, 16
  obtaining, 83
Normative development, 327
  complexity assessment, quantitative/qualitative methods (elaboration), 706–707
  phase transitions, 751
Normative liabilities, transience, 508
Normative narcissism, 484
Normative talk, 728
Normatization, 15–16
Norms, purpose, 15–16
Nosologically based constructs, empirically based constructs (gaps), 83–85
Nosologically based models. *See* Psychopathology
Nosologies, 50
NPV. *See* Negative predictive value
NR. *See* Neglecting responsibilities
NR3C1 (glucocorticoid receptor gene), impact, 168
NTR. *See* Netherlands Twin Registry
Nuclear factor kappa-B (NF-kB), 538–539
Null clines, 735f
Null hypothesis significance testing (NHST), 1080–1081
Null model, 891

OABCL. *See* Older Adult Behavior Checklist
OAD. *See* Overanxious disorder
OASR. *See* Older Adult Self-Report
Observational assessment (young children), 35
Observational coding, 607–610
Observational data (dynamic systems), 725–726
Observational research, analytic strategies. *See* Family
Observational studies, limits (circumvention), 71
Observation sessions, exemplary dyads (recurrence plots), 729f
Obsessive-compulsive disorders (OCDs), 415–417
Obsessive-compulsive personality (OCP), 415–416
  regulation, internally controlling nature, 416
O'Connor, T.G., 341
ODD. *See* Oppositional defiant disorder
Odds ratio (OR), 895, 1033
  combining, Peto's odds ratio (usage), 1049, 1057–1058
  estimation, 1057
  example, 894–895
  measurement, 1058
O-E method, 1057
Older Adult Behavior Checklist (OABCL), 72
Older Adult Self-Report (OASR), 72
OLS. *See* Ordinary least squares
Omnipotence
  fantasies, 481
  feelings, 485
One-child policy (China), 144–145
One-way ANOVA, 847, 925
Open functioning, 408
Opportunities, opening, 599
Opposites, construction (ability), 476
Oppositional defiant disorder (ODD), 55
  CD, precedence, 80
  scale, 82
  test-retest reliability statistics, 33
Optimism, impact, 516
Optimx (R package), 1079
OR. *See* Odds ratio
Orbitofrontal cortex (OFC)
  deactivation, 363–364
Order, self-organization, 704–707
Ordinal scores, series (description), 887
  characteristics, 887
  early completion, 887
  qualitative characteristics, 887
  trend, 887
Ordinary least squares (OLS) regression
  analyses, 928
  model, 952
Oregon Youth Study (OYS), 638–639, 652
Oregon Youth Study-Couples Study, 638–640
Organization, hierarchically embedded levels, 721
O'Rorke, D.J., 402
Orthogonal polynomials
  coefficients, calculation requirement (absence), 885
  usage, impact, 885
Ostendorf data set, 858
Other, role, 501
Others, actions, 496
Other-sex friendships, frequency (increase), 639
Outcome analysis, 1071–1079
Outcomes mediation analyses, comparison, 975–976
Outcomes model, 974–975
Overanxious disorder (OAD), 120
Overcontrol, concept, 695
Overlapping clusters, disjunct clusters (contrast), 828

PAAP. *See* Physical aggression against peers; Physically aggressive against peers
PANAS. *See* Positive and Negative Affect Scale
PANDAS. *See* Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal
PAPA. *See* Preschool Age Psychiatric Assessment
Paradoxical interventions, family therapist usage, 591
Parallel analysis strategy, 1047
Parallel cross-classification, creation, 835
Parallel process exponential decay latent growth curve model, 971f
Parallel process latent change score mediation model, 965f
  alternate parallel process latent change score mediation model, 966f
Parallel process mediation model. *See* Latent change score
Parameter interpretation, 823–826
Parental autonomy support/control, 391–392

Parental chaos, role, 392–393
Parental conditional regard (PCR), 391–392
  negative repercussion, 405
  student perceptions, 405
Parental control, outcomes, 416–417
Parental involvement, 390–391
  impact, 393–394
Parental need support/thwarting, antecedents (role), 394f
Parental neglect, impact, 393–394
Parental psychopathology, 226–227
Parental structure, 390–391
  role, 392–393
Parental subsystem, 584
Parent-child attachment relationship, normative development, 327
Parent-child boundary dissolution. *See* Interparental conflict
Parent-child dysfunction, interparental conflict (association), 587–588
Parent-Child Early Relational Assessment (PCERA), 35
Parent-child interaction, structured/unstructured conditions, 3
Parent-child/interparental relationships, security (comparison), 204t
Parent-child relationship
  context, 350
  processes, model, 226f
  properties, 224–226
  security, preservation, 205
Parent-child security, ethological differentiation, 204–206
Parent-child subsystems, 584
Parent Development Interview, 339
Parent-driven family processes, 609
Parent-driven triangulation, 616–617
Parentification, 583, 589–590
Parenting. *See* Siblings
  conflict, family process (relationship). *See* Coparenting conflict.
  control, SDT formulation, 391
  intervention, 364–367
  marriage, spillover, 615
Parenting Stress Index, 36
Parent Management Training Oregon (PMTO) model, 622
Parents
  behavior. *See* Attachment.
  confidence, 244
  personality factors, 395–396
  psychological needs, contextual/reciprocal influences, 394–396
  report tools, characteristics, 11t
  supports, 395
Participant reactivity, 608–609
Partners, online communication, 649–650
PAS. *See* Preschool Anxiety Scale
Passive splitting, dissociation (relationship), 677–678
Paternal smoking effects, maternal smoking effects (contrast), 137–138
Patient-focused analysis, 86–87
Pattern complexity, problem, 810
Pattern parsimony, 806
Patterns, 868
Pattern summary, 805
PBS. *See* Protective behavioral strategies
PBSS. *See* Protective Behavioral Strategies Scale
PCERA. *See* Parent-Child Early Relational Assessment
P-CFA. *See* Prediction CFA
PCGC. *See* Philadelphia Child Guidance Clinic
PCR. *See* Parental conditional regard
PDD. *See* Pervasive developmental disorder
Peabody Picture Vocabulary Test (PPVT)-Revised Form L, 1009
Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal (PANDAS) infections, 165
Peer acceptance/rejection
  externalizing symptoms, 534–537
  health risk behaviors, relationship, 541–542
  internalizing symptoms, 537–538
Peer Belief Inventory, 1007
Peer influence. *See* Adolescents
  contexts, 559–562
  effects (multilevel investigations), 565–566
  mechanisms, 562–564
  moderators, 564–565
Peer rejection, 535
  depressive symptoms, links, 537
  immune system activity, relationship, 538–541
  role, importance, 542
Peer relations
  constructs, 528
  developmental psychopathology, relationship, 527
  research, 528
  science, 567
Peers
  aggression, assessment, 544
  cliques, operationalization (difficulty), 562
  competence, 533
  conformity, prototype/willingness model proposal, 563
  influence, 542
    mechanisms, 564
    occurrence, 562
  interaction. *See* Early childhood; Infancy.
    simulation model, 741t
  online communication, 649–650
  peer-perceived popularity, 542
  selection, 648–649
  status, 527, 533–542. *See also* School-age children.
  victimization, impact, 649
  victims, contrast, 547
Peer victimization, 533, 542–551
  aggression, 543–544
  assessment/perceptions, 544
  bullying, 543–544
  determinants/consequences, 546
  dynamic systems framework, 547–548
  effects, moderators, 546–547
  HPA axis, association, 548
  impact. *See* Psychological symptoms.
  psychophysiological correlates, 548–549
  relevance, 547–548
  social pain, relationship, 549–551
Percentile bootstrap, usage, 969
Percent missing (PM), 777
Performance-contingent monetary reward, provision, 401
Performance, optimal level, 675
Perrine, M.W., 908
Perseverance, 867
Personal causation, absence (perception), 424
Personal distress, 223
Personality
  characteristics, 867
  models, construction, 867f
  processes, univariate growth mixture modeling (fit indices), 1030t
Personal relevance, 301
Person-based taxonomy, 243–246
  demobilizing patterns, 245–246
  dominant patterns, 245
  mobilizing patterns, 244
  secure patterns, 243–244
Person-environment system, 676. *See also* Coactive person-environment system
  dynamics, 669–671
Person-oriented approaches, 797, 804–812, 857
  Block, Jack, 815–818
  cluster analysis, 826–837
  coherence/structure, 805
  complexity, 804–805
  data example, 833–836
  description, 799–812
  differences, varieties, 805–808
  dimensional identity, 808–809
  discussion, 837
  early protagonists, 812–818
  generation, difficulty, 809

Person-oriented approaches, *(Continued)*
human behavior, ecological context, 809
Lewin, Kurt, 814–815
limitations, 809–812
log-linear modeling, 822–823
parameter interpretation, 823–826
process
occurrence, 805
specificity, 804
Q-set, 817–818
Q-technique, 816–817
statistical approaches, 818–837
Stern, William, 813–814
Person-oriented data analysis, methods, 818–822
Person-oriented research, 866
Person oriented, term (usage), 867–868
Person's correlation, 832
Person-specific approaches, 846
Borkenau data set, 858
confirmatory factor analysis, 860t
data box, 854–858
data examples, 858–861
discussion, 861–862
factor analysis, 854–858
interindividual variation (modeling), group-based individual differenced models (usage), 849–854
Ostendorf data set, 858
PANAS data, 858–861
two-factor solution, positive/negative items, 859t
Perturbations, 721–723
Pervasive developmental disorder (PDD), 84
Peto's odds ratio, 1057–1058
usage, 1058t
Phase transitions, 721–723, 751
generation, control parameters (manipulation), 725
identification, 724
Phenomena, term (usage), 813
Phenomenological analysis, 388
Phenotypic stability, 171–172
Philadelphia Child Guidance Clinic (PCGC), 583–584
Physical abuse (PA), 286
testing effects, 1031
Physical aggression against peers (PAAP), 879, 899
development
log-linear model, observed/expected frequencies, 825t
model, parameters, 825t
path model, 821f
two-group model, 821f, 827f
Physically aggressive against peers (PAAP), 820
Physical pathology, determination, 705–706
Physical self-conservation, 506
Physiological mediators. *See* Attachment
PICA, 12
PKBS-2. *See* Preschool and Kindergarten Behavior Scales
Planned missing data designs, 784–790
two-method planned missing data design, candidate constructs (usage), 788t
Planned missing design
example, 784t
three-form planned missing design, example, 785t
two-method planned missing design, 787–789
Planned missing protocols, multiform planned missing protocols, 785–787
Plant, R., 419
PNDE. *See* Pure natural direct effect
PNIE. *See* Pure natural indirect effect
Polynomials
approximations, usage, 885
order/degree, 884
orthogonal polynomial coefficients, calculation requirement (absence), 885
parameters, independence, 885
scores, description, 884–885
usage, 884–885
Pooled data, 1068–1071
intervention groups/procedures, 1068
measures, 1068
protective behavioral strategies, 1068, 1070–1071
sample, 1068
Pooling data (2x2 tables), 1058t
Popularity. *See* Peers
term, usage, 542
Positive affectivity, 513
concept, invocation, 513
Positive and Negative Affect Scale (PANAS) data, 858–861
Positive and Negative Consequences Experiences (PNCE) questionnaire, 1071
Positive emotions, role, 512–513
Positive predictive value (PPV), 15
Positive psychology
emphasis. *See* Control.
movement, role, 512–516
Postmodernism, 508
Postperturbation SSGs, example, 747f, 748f
Post-traumatic stress disorder (PTSD), 361
cross-classification, 894t
first-order CFA, 869t
diagnosis, absence, 367
empirical/conceptual work, 3
symptoms, 806
unresolved loss/trauma, association, 361
variables, 869, 888
young children, 3
Potential outcome mediation model, longitudinal data (usage), 976–977
Potential outcomes mediation analysis, traditional outcome mediation analysis (comparison), 975–976
Potential outcomes model, 974–975
moderation, usage, 976
Pothoff extension, 927
Poverty, exposure (duration), 113
PPV. *See* Positive predictive value
PPVT. *See* Peabody Picture Vocabulary Test
PRE. *See* Proportionate reduction in error
Preacher, K.J., 968
Prediction CFA (P-CFA), 870–873, 912
Predictive validity, 14
Predictors, risk/protective mechanism function, 1018–1021, 1029–1032
Preece, M.A., 994
Preferred Reporting Items for Systematic reviews and Meta-Analysis (PRISMA), 1052
Prenatal/postnatal effects (separation), adoption/fostering (usage), 147–148
Prenatal risk factors, design, 134
Prenatal stress/anxiety, 175–177
Preperturbation SSGs, example, 747f, 748f
Pre-post designs, CFA, 889–892
Pre-post study, schizophrenics (neuroleptic drug usage), 890t
Preschool Age Psychiatric Assessment (PAPA), 20
interview, 30
usage, 30–32, 33
Preschool and Kindergarten Behavior Scales (PKBS-2), 28, 29
Preschool Anxiety Scale (PAS), 20, 28–29
Prescott, C.A., 133
Pressure, internal forms, 395
Prevention
categories, 117
framework, 325–326
growth mixture modeling, implications, 1035–1036
latent change score modeling, implications, 1026–1027
latent growth modeling, implications, 1013–1015
Prevention/intervention
effort, steps, 703–704
unit, examination, 702
Prevention/practice, dynamic systems (translation), 701–704

Primary intersubjectivity, 455
Primordial possession, 464–465
Private audiences, emergence, 398
Problem ratings, comparison, 76
Problems (internalizing), latent change score model, 1020f
Proc CALIS, 956
Product-multinomial sampling designs, 826
Product-multinomial scheme, 870
Profiles, 868
Programmability, application, 160
Project INTEGRATE, 1067–1068, 1069t, 1071, 1078
  intervention groups, 1070t
Proportional change model, 1016
Proportionate reduction in error (PRE) coefficients, 891, 901
PROSPER STUDY, 639
Protection, foundational focus, 397
Protective behavioral strategies (PBS), 1068, 1070–1072
  follow-up example, 1072f, 1074f
Protective Behavioral Strategies Scale (PBSS), 1070
Protective-enhancing factors, 248
Protective factors
  identification, 248
  moderating effects, 1009
  understanding, latent growth modeling (usage), 1006–1007
Protective mechanisms (understanding)
  growth mixture modeling studies, usage, 1029–1032
  latent change score modeling studies, usage, 1018–1021
Protective mechanisms (influences), growth mixture modeling studies (usage), 1029–1032
Protective processes, investigation, 598–599
Protective-reactive processes, 988
Protective-stabilizing factors, 248
Prototype/willingness model, proposal, 563
Prototypical responsive/nonresponsive learning session, interaction dynamics, 693f
Psychiatric behavioral genetics, 119–120
Psychiatric disorder
  distinctions, 106–107
  identification, interviews (usage). *See* Adolescents; Children.
  mental retardation, contrast, 106
Psychiatric epidemiology. *See* Children
  case identification
    ascertainment bias, 99–100
    developmental issues, 99
    question-and-answer methods, usage, 98
  cases
    classification, 98–99
    defining, 97
  developmental approach, implications, 103–105
  disease, frequency (measurement), 96–97
  instrumentation, 97–98
  mean separation anxiety symptoms, age/information source basis, 100f
  mechanisms, 97–98
  normal behavior, abnormal behavior (contrast), 108
  protective factors, measurement, 101–102
  research, characteristics, 96–102
  risk factors, impact
    estimation, bias (control), 100–102
    measurement, 101–102
  taxonomy, 97–98
Psychiatric patients, facility/research diagnoses (cross-classification), 892t
Psychiatrists (cartoon), 51f
Psychoanalytic theory, 107–108
Psychodynamic theory, content (problems), 108
Psychological activity
  adaptive/maladaptive patterns, 686
  natural fractionation, 675–677
Psychological development
  cumulative effects models, 160–161
  developmental mechanisms, 163–170
  developmental programming, 159–160
  early experiences (psychological research), developmental models/hypotheses (impact), 157–163
  early experiences (effects), developmental models/mechanisms (usage), 156
  genetics, 167–168
  life span development, 160–161
  mechanisms, 169–170
  neural development/circuitry, 166–167
  neuroimmunology, 164–166
  psychological mechanisms, 168–169
  sensitive period models, 157–159
  stress physiology, 163–164
Psychological independence, 602–603
Psychological needs, contextual/reciprocal influences. *See* Parents
Psychological processes, genetic factors (impact), 167
Psychological research
  developmental models/hypotheses, impact, 157–163
  missing data practices, review, 782–784
Psychological symptoms (predictors), peer victimization (impact), 545–546
Psychological theory, importance, 706
Psychology. *See* Human symbolic psychology
  movement, role. *See* Positive psychology movement.
Psychometric properties, understanding, 12
Psychometric theory, limitation, 56
Psychoneuroimmunology (PNI)
  application, 165
  impact, 165
Psychopathology. *See* Developmental psychopathology; Parental psychopathology
  adolescent parenthood, association, 645–646
  adults, multi-informant data, 54
  aggressive/delinquent behavior
    longitudinal analyses, acceleration, 62–65
    six-year predictive pathways, 65, 67
  assessment
    advancement, 77–79, 79t
    bottom-up approach, 52f
    developmental considerations, 49
    impact, 56–57
    questions, 48. *See also* Children.
    role, 56–58
    taxonomy/diagnosis, relations, 77f
    tools, 10
    top-down approach, 51f
  autonomy disturbances, 385, 413–426
    graphic overview, 414f
  autonomy relationship, 385–387
  between-cohort predictive correlations, 63f
  biological research, 84
  bottom-up strategy, variations, 52
  challenges, 78–79
    quantitative aids, usage, 79–81
    SDI studies, usage, 78
  children
    multi-informant data, 53–54
  children, risk
    identification, 249–250
    increase, interparental conflict (impact), 226f
  classification, family systems perspective, 593–597
  comorbidity
    Berkson's bias, 60
    implications, 60–61
    issues, 59–61
  component, 50–52
  conceptualization, quantification (advantages), 80
  criterial features, quantification (advantages), 81t
  cross-domain relations
    growth mixture modeling, usage, 1032–1034
    latent change score modeling, usage, 1021–1022
    latent growth modeling, usage, 1009–1012
  cross-population comparisons

Psychopathology *(Continued)*
latent change score modeling, usage, 1023–1026
latent growth modeling (usage), 1012–1013
definition, 87
development, 265
emotion, role, 270–271
developmental component, 48–50
developmental differences, 52
developmental immaturity, relationship (absence), 683–684
developmental methodology, 49–50
developmental study, assessment/taxonomy (roles), 56–58
developmental study, framework, 52–56
data sources, 52–54
epidemiological aspects, 54
multivariate aspects, 55
operational definitions, 55–56
developmental study, longitudinal design
acceleration, 61–65
growth curve designs, 67–70
growth mixture model (GMM), 70–72
hierarchical linear modeling (HLM), usage, 68–70
path analysis, structural equation modeling (usage), 67
path designs, 65–67
problems (externalization), developmental trajectories (usage), 71–72
strategy, acceleration, 62–65
developmental study, longitudinal designs, 61–72
developmental theories, 49
diagnosis, 47, 58–61
advancement, 77–79, 79t
assessment/taxonomy, relations, 77f
comorbidity issues, 59–61
developmental considerations, 49
formal diagnoses, 59
formulations, 59
processes, 59
Diagnostic and Statistical Manual (DSM) diagnoses, relations, 83
disease, treatment, 705–706
dynamic trajectories, 667–669
emotional/behavioral disorders, natural kind designation (question), 57
emotion, relationship, 268–269
empirically based models, 51–52
family-oriented assessment/interventions, 85–86
fixed forms, 667–669
forced choices, avoidance, 80
Generation R Longitudinal Study ("R" = Rotterdam), 74–75
genetic research, 84
information, preservation, 80
interventions (evaluation), norms (usage), 87
life span applications, 72
longitudinal research designs, 84
longitudinal studies, assessment/taxonomic models, 72–75
measurement. *See* Adolescent psychopathology; Children.
mechanisms, 363–364
multicultural findings/applications, 75–77
multicultural norms, construction, 76–77
multicultural perspectives, 47–48
multisource data, usage, 87
need frustration, relationship, 413–414
Netherlands Twin Registry (NTR), 75
normality, relationship, 988–989
nosologically based models, 50–51
person-oriented approaches, 797
prototypes, quantitative aspects, 80–81
quantification
advantages, 81
precision, increase, 80
research
continuation, directions, 85–87
multicultural directions, 86
quantification, advantages, 80
rethinking, 667–669
risk, 553–554
scale scores, multicultural comparisons, 76
secular trends, research, 84–85
study, 426–428
symptoms, 545
syndromes, multicultural tests, 75–76
taxonomy
advancement, 77–79, 79t
assessment/diagnosis, relations, 77f
bottom-up approach, 52f
constructs, advantages, 81t
development considerations, 49
impact, 57
role, 56–58
top-down approach, 51f
term, usage, 705–706
top-down strategy, 51
TRacking Adolescents' Individual Lives Survey (TRAILS) study, 74
variable-oriented approach, 797
within-cohort predictive correlations, 63f
Zuid Holland Longitudinal Study, 73–74
Psychosexual development theory, 445
Psychosis, origin, 581
Psychotherapy, autonomy/autonomy support, 425–426
P-technique, 848, 855
factor models, 857
PTSD. *See* Post-traumatic stress disorder
Puberty. *See* Early puberty
Publication bias, 1053–1054, 1066
Public health epidemiology, 96
Public health strategies
early interventions, impact, 180–183
shaping, developmental models (impact), 179–185
Pulsion theory, 445
Pure natural direct effect (PNDE), 974
Pure natural indirect effect (PNIE), 974
P-value combination, 1056

Q-set, 816–817. *See also* Attachment Q-Set; California Q-Set
deficiency, 818
Q-sort, 816
Q statistic, 1073
Q-technique, 815–817
Q test, 1060
Quadratic growth, 1001
Quadratic model, 998
Quality scores, 1054
Quantitative diagnostic scores, advantages, 80
Question-and-answer methods, usage, 98
Questionnaire approach, advantages, 109–110
Quiet ego, 489

Radar-and-response system, 265, 297
Radiation, durations (risk factors), 112
Rand Index, 829, 837
Random-effects model, 1060–1061
example, 1060f
fixed-effects model, contrast, 1061–1062
Random effects modeling, 68
Randomized controlled trials (RCTs), 132, 356
alternative, 147
RB. *See* Rule-break
RDC. *See* Research diagnostic criteria
RDoC. *See* Research Domain Criteria
Reactive aggression, 698
Reactive attachment disorder, 31
Real-time longitudinal studies, cost, 50
Real-time measures (DS methods), 727–731
Reciprocal influences, 394–395
Reciprocation, products, 456–457

Reciprocity, impact, 455–456
Recurrence quantification analysis (RQA), 729–731
Referred children, 55
Reflective functioning (RF), 367–368. *See also* Intervention
  description, 368
Reflexes, 674
  movements, closed-loop feedback systems, 449
Regional CFA base models, 872–873
Regional models (log-linear CFA base model), 871
Regression, 798
  analysis, 718
  cross-lagged regression model, 853f
  lagged regression models, 852–853
  multilevel autoregressive model, 852
  results, impact, 729t
Regression discontinuity (RD) designs, 129
  usage, 146–147
Regulations, internalization/ownership (promotion), 406
Relational disorders, classification (proposal), 595–596
Relational functioning, global assessment, 595
Relational functions, 584
Relational risk factors, 596
Relational self-esteem, 491
Relational syndromes, 596
Relational systems, epigenetic processes (Wynne model), 592f
Relationship conflict, intergenerational transmission, 650
Relationship education (RE) programs, development, 654–655
Relationship quality, 493
Relationships. *See* Abusive relationships
  behavior/experiences, 641–642
  relationships, effects, 587–588, 619
Reliability
  interpretation rules, 13t
  measure consistency/stability, 12–13
  statistics, 13t
  types, 12t
REML. *See* Restricted maximum likelihood
Repellor, presence, 720f
Repellors, 720
Representation
  defining, 442–443
  term, usage, 442
Representational incompetence, 454
Representational levels, 440
Representational process, 442
Representations, 674
  impact, 439–440
Research design strategies, DS principles (impact), 723–725
Research diagnostic criteria (RDC), 52–53
Research Domain Criteria (RDoC), 839
  family factors (inclusion), 594–595
  framework (National Institute of Mental Health), 24, 37–38
Research synthesis, 1051–1067
  change, model-based estimates, 1077f
  complexity, 1062–1067
  data example, 1058–1059
    outcome analysis, 1071–1079
    pooled data, 1068–1071
    software programs/packages, 1079–1089
  data pooling, 2x2 tables (impact), 1058t
  data synthesis, approach (unification), 1062
  directions, 1053f
  discoveries, 1066–1067
  effects, moderation, 1066–1067
  fixed-effects model, 1059–1060
  groups/measures, harmonization, 1054–1055
  Mantel-Haenszel method, 1056–1057
  methods, 1045t
  model-based approaches, 1059–1062
  multivariate meta-analysis, 1062–1064
  Peto's odds ratio, 1057–1058
  publication bias, 1053–1054
  p-value combination, 1056
  random-effects model, 1060–1061
  selection bias, 1053–1054
  software programs/packages, 1079–1080
  studies, inclusion criteria, 1052–1053
  subgroups, 1066–1067
  2x2 tables, combination, 1056–1059
  variables, selection, 1054–1055
  variance, estimators, 1061
Residuals, labeling, 817–818
Resilience, 987–988
Resource depletion models, usage, 221
Respiratory sinus arrhythmia (RSA), 354
Respondent-based interviews, 110–111
Restricted maximum likelihood (REML), 1061, 1066
  usage, 1079–1080
Restrictive strategy, 776
Reverse causation, 129
  possibility, 131
Revised Family Cohesion Index, 614f
RF. *See* Reflective functioning
Ribonucleic acid (RNA), 672
Rigidity, 867
Rigid triangles, 584
Risk, 249–250
  alleles, impact, 555
  conditioning, 282
  cross-domain relations
    growth mixture modeling, usage, 1032–1034
    latent change score modeling, usage, 1021–1022
    latent growth modeling, usage, 1009–1012
  cross-population comparisons
    latent change score modeling, usage, 1023–1026
    latent growth modeling, usage, 1012–1013
  exposure, length, 112
  factors, 351
  genetic mediation, 130
  impact, reduction, 599
  influences (understanding), growth mixture modeling studies (usage), 1029–1032
  moderating effects, 1009
  multistage models, 113
  removal, 142–143
  understanding
    growth mixture modeling studies, usage, 1029–1032
    latent change score modeling studies, usage, 1018–1021
    latent growth modeling, usage, 1006–1007
  universal introduction, 142
  universal removal, 142–144
Risk factors
  distortion, confounding (impact), 100–101
  impact
    estimation, 100–102
    measurement, 101–102
RMSEA. *See* Root mean square error of approximation
Role-specific self-esteem, 491
Romantic relationships, 631–633
  peer selection/socialization processes, 648–649
Root mean square error of approximation (RMSEA), 778–779, 820–821, 941, 1003
  values, 966, 1007–1011, 1024–1027
R package semTools, 778
RQA. *See* Recurrence quantification analysis
R-technique, 815
  examples, 854
  factor analytic approach, 857
Rubin's rules, 772–773, 779
Rule-break (RB) bout slope, examples, 728f
Rule-break (RB) talk, 728

Runs
CFA, 895, 898–899
single scores, 898
Rutgers Alcohol Problem Index (RAPI), 1071

Sadness, anger (combination), 496
Sameness, sense, 451–452
Same-sex relationships, 646–647
Sample-size adjusted Bayesian information criterion (SABIC), 1030, 1033
Sampling variability, 1059
SAS Proc Mixed, 962
SAS, usage, 740, 831, 851
Scaffolding
dynamics, 690. *See also* Arithmetic lessons.
nature/importance. *See* Coactive scaffolding.
SCAN. *See* Schedules for Clinical Assessment in Neuropsychiatry
Schedule for Affective Disorders and Schizophrenia (K-SADS), 33
Schedule for Affective Disorders and Schizophrenia DBDS (K-SADS-DBDS), 34
Schedule for Affective Disorders and Schizophrenia—Present and Lifetime (K-SADS-PL), 30
Schedules for Clinical Assessment in Neuropsychiatry (SCAN), 54
Schermerhorn/Cummings's model, example, 612f
Schizophrenia, 464
research, focus, 581–582
Schizophrenic mother
psychosis, origination, 581
targeting, 583
Schizophrenics, neuroleptic drug usage (treatment evaluation), 890t
School-age children, 533–556
friendship, 551–556
peer acceptance/rejection
externalizing symptoms, 534–537
health risk behaviors, relationship, 541–542
internalizing symptoms, 537–538
peer-perceived popularity, 542
peer rejection, immune system activity (relationship), 538–541
peer status, 533–542
peer victimization, 542–551
Schooling influences (assessment), regression discontinuity (usage), 146–147
School performance, modeling. *See* Emotional-behavioral difficulties
School system, humiliation/violence, 501–502
Scientific epidemiology, 95–96
SCIFF. *See* System for Coding Interactions and Family Functioning
Scores (series)
cross-classification, 901t
description, polynomial usage, 884–885
interruption, absence, 898
length, differences (analysis), 886–889
data example, 887–889
extensions, 889
ordinal/higher level scores series, description, 887
polynomial approximation, usage, 885
Scores, uninterrupted series, 898
Screening
methods, 18–19
characteristics, 19–20
tests, 1050–1051
tools, 20, 23
SCSQ. *See* Self-Control Strategies Questionnaire
SDIs. *See* Standardized diagnostic interviews
SDQ behavioral screening tool, 23
SDS. *See* Social defense system
SDT. *See* Self-determination theory
Secondary intersubjectivity, 452
Secondary intervention programs (high-risk intervention programs), 117
Second-order CFA base model, 872
Security
developmental model, 238
foundational focus, 397
models, temperament (impact), 231
theory, impact, 328–329
Selection bias, 1053–1054
designs, impact, 142–144
Self
abstractions, 479
adolescent cognitive development (Piaget theory), application, 478
conflicts, normalization, 508
continuity/coherence, 471
developmental psychopathology, impact, 473
differentiation, 582
discrepancies, 491
identities, anchoring, 411–412
impoverished self, 507
integration/differences, measurement/assessment. *See* Multiple selves.
multiple selves, functional flexibility, 508
others, actions, 496
processes, role, 498–503
SDT viewpoint, 410–411
theory, 474
Self-acceptance, display, 418–419
Self-agency, 474–476
root, 440
sense, 477
Self-aggrandizement, 517
pathological condition, 472
Self-attributes, 479–480
Self-awareness, 471, 474–475
development, 457–459
I-Self function, 480
minimum, 445, 463
Self-coherence, 474–475
cross-cultural differences, 510–511
I-Self function, 507–510
Self-concept
formation, developmental changes, 557–558
problem, pathological condition, 472
Self-conscious emotions, 499
Self-conservation, 506
Self-continuity, 474–476, 506
absence, 507
search, 480
sense, 477
Self-Control Strategies Questionnaire (SCSQ), 1070
Self-critical depression, 417–418
Self-critical perfectionism, 417
Self-criticism, role, 497
Self-deception, 415, 517
pathological condition, 472
Self-determination, impact, 516
Self-determination theory (SDT), 386
argument, 397
autonomy
extension, 391
support, 391
conceptualization, 407
development, study, 426–428
emphasis, 401
eudaimonic viewpoint, 407
framework, 427–428
hypothesis, 403
independence extension, 389
parenting research, 390–391
pathways, 386–387
principles, 386
psychological needs, satisfaction, 412
psychopathology, study, 426–428
representation, 390
research, 387
viewpoint, 410–411

Self-development, 478
  autonomy/autonomy disturbances, 385
  autonomy support/attachment, interrelation, 397
  disturbance, 427
Self-disclosure, excess, 556
Self-disparagement, 415, 417–418
Self-effacing behavior, 489
Self-efficacy, 474, 478, 509
  I-Self process, 504
Self-enhancement. *See* Early adolescence; Early to middle childhood; Late adolescence; Middle adolescence; Middle to late childhood
  biases, 481–488
  cross-cultural differences, 488–489
  necessity, 503
  normative perspective, 481
  strategies, 487, 488. *See also* Early childhood.
Self-esteem, 471. *See also* Low self-esteem
  cross-cultural differences, 493–494
  depression, relationship, 494
  fitted growth curve models, comparisons, 1004t, 1006t
  inflation, 487
  looking-glass self model, 490
  multivariate growth model, 1006f
  problems, 498–499
    perceived directionality, 496
    perceptions, alteration, 491–493
    persistence, 490
  term, usage, 490
Self Esteem Inventory (SEI), 1003–1004
Self-esteem, self-evaluations. *See* Global self-esteem
Self-evaluation, 458, 563
  purpose, 482
Self-evidence, 464
Self-evident mine-ness, loss, 464
Self-focused goal orientation, development, 422
Self-functioning, active facilitation, 397–398
Self-integration, breakdown, 424
Self-iterations (changes), matches/mismatches (relation), 692f
Self-management, display, 418–419
Self-narratives, 475
  creation, 506
Self-organization, 671–672
  process, 666, 671
Self-perception
  discrepancy, 607
  inflation, 481
Self-processes, pathological conditions, 472–473
Self-protective strategies, 483
  usage, 488
Self-recognition, 475
Self-regulation
  attachment, relationship, 359–360
  fostering, 277
Self-reported externalizing problems, trajectories, 535
Self-reported friendship quality, validity, 552
Self-serving biases, 481–489, 517
  pathological condition, 472
Self-silencing, 635
Self-sufficiency, illusion (preservation), 418
Self-sustaining learning trajectories, 686–695
Self-unity, 464
  root, 440
Self-validation, 505
SEM. *See* Structural equation modeling
Semiparametric mixture model fitting, usage, 71–72
Senses, unitary/common functioning, 446
Sensitive period models, 157–159
Sensitivity
  conception, 336
  effectiveness, meta-analysis, 365
  parent scoring, 336
  period, 158
  term, usage, 15
Sensorimotor acts, 674
Separation, 828
Separation-reunion situations, physiological function, 355–356
Sequential ignorability, 975
Series. *See* Scores
SES. *See* Socioeconomic status
Setting, characteristics, 7
Severe caregiving neglect/deprivation, 178–179
Sex differences, 173–174
Sex steroid involvement, biological evidence, 174
Sexting, impact, 653
Sexual abuse (SA), testing effects, 1031
Sexually transmitted infections, risk (increase), 647
Shannon's entropy, 736–737
Shapiro-Wild test, 740
Sharing
  equity, developmental trend, 462
  levels, 460–463
Shoe-leather epidemiology, 96
Siblings, parenting/family process, 615
Significance testing, usage, 873–876
Significance tests, 825
  performing, 877
  selection, 876–877
Sign patterns, combinations (impossibility), 882
Single-mediator models, bootstrap confidence intervals, 931t
Single Nucleotide Polymorphisms (SNPs), 173, 344
Single-subject CFA, 895
Skewed distribution, 739
Skewness, 739
Skill development, hierarchical levels, 674f
Skilled action, structure (developmental changes), 673–675
Sleeper effect, 162
SNPs. *See* Single Nucleotide Polymorphisms
Sobel's approximate significance tests, 1024
Sociable behaviors, periods, 458
Social anxiety, 636
  development pathways, 699–701
  romantic relationship difficulties, relationship, 637
Social anxiety disorder (SAD), 635–637
Social-avoidance motivation, 531
Social awareness, 479
  development, 457–458
Social-cognitive mediators. *See* Attachment
Social comparison
  emphasis, 484
  engagement, 483
Social competence, age effects (illustration), 16f
Social contexts
  emotional integration, development, 409–410
  internalization, relationship, 404–406
  intrinsic motivation, relationship, 399–401
  support, 412
Social cues, attention (increase), 558
Social de-escalation strategies, 205–206
Social defense system (SDS), 204
  activation, 221
    recurrence, 223
  dialectical opposition, 222
  external goal, 205
  impact, 235
    model, 222f
  operation, 209, 221
  prioritization, 205–206
  protective function, analysis, 206–207
  trigger, 205
Social dependence, 459–460
Social-emotional problems
  assessment, dimensional tools (usage), 23–24
  checklist/questionnaire measures, 21–22t, 25t
  dimensional checklists, 24–29

Social inferiority trait, 812–813
Social information processing, 357–358
  models, relevance, 541–542
Sociality, impact, 455–456
Socialization. *See* Need-supportive socialization
  effects, 559
  practices, impact, 488
  processes, 648–649
Social learning perspectives. *See* Family
Social pain, peer victimization (relationship), 549–551
Social partners, display, 457
Social problems, taxonomies (construction), 57–58
Social Rating Scale, 924
Social regulations, integration, 387
Social regulations/values, assimilation, 403–406
Social selection, 129
  possibility, 130–131
Social Skills Improvement System (SSIS), 29
Social values/regulations, internalization (impairments), 413–414
Social withdrawal, 530–533
  distress, internalization, 532–533
Society for Research in Child Development (SRCD), 178
Sociocultural factors, impact (examples), 8t
Socioeconomic adjustment, dynamics, 696
Socioeconomic status (SES), 48, 61, 215, 494, 1012
  dropout, linear correlate, 775
  effects, examination, 136
  neighborhood aggregation, 803
  samples, 340
  variance, 137
Socioemotional adjustment, dynamics, 696f
  adaptive/maladaptive patterns, development pathways, 696–701
Socio-emotional behaviors, assessment tools, 10
Solomon four-group design (planned missing design), 784t
Somesthetic sensitivity, 441–442
Space-segment clusters, 836t
Space-segmenting method, usage, 836
Spatial preposition (child acquisition), moving min-max graph (representation), 739f
Specific Affect Coding System, usage, 642
Specificity, 15
  increase, 225
Specific reactions, 867
Spillover. *See* Family processes
  concepts, 587
Splitting, 677
  prominence, 680
Spontaneous speech, observations, 278
Sporadic peer victims, chronic peer victims (contrast), 547
Spousification, 590
SPSS, usage, 739, 740
SPT. *See* Supportive psychotherapy
SRS. *See* Stress response system
SSGs. *See* State space grids
SSIS. *See* Social Skills Improvement System
SSRIs, usage, 170
Stable unit treatment value (SUTVA), 134
Staff-child interaction, dynamics, 703f
Stage-salient task framework, 241
Standard deviation (SD) measure, 739
Standardized diagnostic interviews (SDIs)
  development, 52–53
  diagnoses, 78
  studies, usage, 78
  usage, 73
Standardized Pearson residual, 874
State regulation deficit, 686
State space, 720, 746
  attractors/repellor, presence, 720
  model, example, 671f
State space grids (SSGs), 746–748
  analysis, 746–749
  between-grid analysis, 750
  examples, 747f, 749f
  within grid analysis, 748–749
  methodology, 746–747
  preperturbation SSGs/postperturbation SSGs, 747f
  SSG-to-SSG change, 750
State synesthesia, 445–447
Steady states, stability, 735f
Steenbeck/van Geert peer interaction simulation model, 741f
Stereotypes, content, 476
Still Face Procedure, usage, 291
Stimulations, impact, 449
Stochastic actor-based modeling, application, 561
Stochastic clustering, deterministic clustering (contrast), 831–832
Stouffer's Z, 1056
  data example, 874–875
Stranger Situation. *See* Ainsworth Stranger Situation
Strange Situation, 339
  infant attachment security, 342
  modification, 333
  mother-child attachment security, stability, 346–347
  separation episode, 354
  stress responses, 356
  usage, 330
Strange Situation Procedure, 332
Strengths and Difficulties Questionnaire, 119
Stress
  exposure, animal studies, 174
  physiology, 163–164
  prenatal stress/anxiety, 175–177
  research, 272
  response, 174
  susceptibility model, 239
Stress response system (SRS)
  alterations, 219–221
  evolutionary conceptualization, 219
  reactivity/activity, delineation, 220
Structural equation modeling (SEM), 67, 819–822, 941–942
  data, example, 820–821
  discussion, 821–822
  exponential decay structural equation model, 945–946
  impact, 942f
  latent growth curve (LGC), 996–997
    model, L1/L2 predictions, 943f
  mediation
    impact, 941
    model, 941f
  moderated mediation, usage, 970–973
  moderation, usage, 941–942
  usage, 67, 132, 740, 925
Structural family theory (Minuchin), 583–584
Structural patterns, 598
Structural zeros, accounting
  absence, 883t
  presence, 884t
Structural zeros, declaration, 881
Structural zones, differences (CFA), 881–883
Structured Interview of Parent Management Skills and Practices, Parent Version, 1034
Students
  arithmetic learning. *See* Developing students; Emotional-behavioral difficulties.
  school performance, modeling. *See* Emotional-behavioral difficulties.
  self-iterations, growth (analysis), 691f
Student's *t* distribution, 773
Study of Early Child Care and Youth Development (SECCYD), 331, 337–338, 347–348, 1018
  analysis, 352
  data, usage, 353, 360
  longitudinal sample, 348
Subgenual anterior cingulate (subACC), 550
Subjective health, numbers cross-classification

first-order CFA, 896t
prediction CFA, 897t
Submissive disengagement, 205
Suboptimal learning dynamics, self-sustaining trajectories, 692–693
Subramanian, S.V., 801, 803
Substance, complexity (problem), 810
Success, importance, 483
Suicidal behaviors
depression, relationship, 493–496
pathological condition, 472
Suicidal ideation, predictors (path-analytical model), 500f
Supermatrix technique, graphical representation, 781f
Supportive friendships, 537
Supportive psychotherapy (SPT), 367–368
Surgency dimensions, 231–232
Survey of Wellbeing of Young Children (SWYC), 23
SUTVA. *See* Stable unit treatment value
Symons, D.K., 358
Sympathetic-adreno-medullary (SAM) system, 548
Sympathetic nervous system (SNS), 538–539, 550
Symptomatic factor, 387
Symptomatology, internalization, 1025f
Symptom expression, transformations, 600
Syndromes
multicultural tests, 75–76
term, usage, 51
Synesthesia, 446, 447
System destabilization, 751–752
System for Coding Interactions and Family Functioning (SCIFF), 608
Systemic-level interrelations, 598
Systems changes, monitoring/response, 704
Systems thinking, mind-set shift, 702

Taxa, quantification. *See* Diagnostic taxa
Taxometry, usage, 827–828
Taxonomy (taxonomies), 97–98. *See also* Psychopathology
advancement, 77–79, 79t
assessment/diagnosis, relations, 77f
construction, 57–58
constructs, advantages, 81t
models, 72–75
prototypes, 68
thinking, categories (impact), 58
Taylor series expansion, 929–930
TBSI. *See* Toddler Behavior Screening Inventory
Teacher Observation of Classroom Adaptation—Revised, 1034
Teacher self-iterations, growth (analysis), 691f
Teacher's Report Form (TRF), 65, 353, 1030
completion, 73
ratings, 85
Teaching, dynamics, 687–689
Teen fathers, 605–606
Teleonomic activities, 104
Temperament
impact, 231
relative/conjoint contributions, 296
Temper tantrums, 31–32
Temporal comparison, usage, 482, 483
Tertiary intersubjectivity, 461
Test-retest attenuation, 78
Test-retest reliability, 13, 32–33, 33t
statistics (ODD), 33
TFP. *See* Transference-focused psychotherapy
Theory of Mind, 358–359
Therapist/client, attachment fit, 368–369
Thinking structures, order/variability/disorder (self-organization), 704–707
Third-order CFA base model, 872
Thought disturbance, cross-classification (first-order CFA), 875t
Three-cluster solution, creation, 836t
Three-form design, 787
Three-form planned missing design, example, 785t
Three-generational families, 605
Time
meaning, 111–112
metrics, 991–993
transformation, 998–999
Time of measurement effects, sequential designs (usage), 790f
Time scales, interrelations, 720–721
Time-series analysis, 727–729
Time-series approach, examples, 141
Time-series designs, 140–142
Time-varying covariate, 732
growth curve model, 1011f
TLI, 778–779
Toddler Behavior Screening Inventory (TBSI), 20
Toddlers, emotion lexicon (development), 284
Total effect (TE), 974
Total natural direct effect (TNDE), 974
Total natural indirect effect (TNIE), 974
Total Problems scores, analysis, 77
TRacking Adolescents' Individual Lives Survey (TRAILS) study, 74
Traditional outcomes mediation analysis, potential outcome mediation analysis (comparison), 975–976
Traits, contrast, 812
Trajectories. *See* Conduct problems; Dynamic trajectories
alternatives. *See* Developmental trajectories.
atypical development, configural frequency analysis (CFA), 884–886
data example, 885–886
atypical trajectories, 877
autoregressive latent trajectory mediation models, 963
comparison, 908–912
individual trajectories, 706
mean growth trajectories, estimation, 1031f
prediction, 915–916
data example, 915–916
simulation, 685–686
two-group CFA, 881
typical development, configural frequency analysis (CFA), 884–886
data example, 885–886
typical trajectories, 877
Transactional family dynamics, Schermerhorn/Cummings's model, 612f
Transactions, construct, 588
Transference-focused psychotherapy (TFP), 367–368
Transformations, idea, 600
Translational efforts, 653
Translational epidemiology, 121
Translational research. *See* Family
configural frequency analysis, relationship, 917–919
Translational science, developmental epidemiology (contributions), 95f
Translation, concept, 174–175
Transmission gap, 336
Trauma therapies, 367
TRF. *See* Teacher's Report Form
Triangle. *See* Rigid triangles
idea, 582
Triangular capacity, 600
Triangulation, 614–617
dimensions, 616–617
T-tests, 718
Tukey range tests, 1032
Tumor necrosis factor-α (TNF-α), 539
Turning points (study), latent growth modeling (usage), 1005–1006
Tutor/child interaction (mathematics instruction), matches/mismatches/self-iterative utterances (changes), 690f
Twins
children of twins design, 135
co-twin, sex, 145
discordant twin pairs (natural experiments), 133–134
extended children of twins (ECoT) design (natural experiments), 135–136
longitudinal twins and parents (LTaP) design, 136

Twins *(Continued)*
multivariate twin designs (natural experiments), 133
Netherlands Twin Registry (NTR), 75
twin-singleton comparisons, 138–139
Twin-singleton comparisons, 129
Two-factor solution, positive/negative items, 859t
Two-group CFA, 880t
base model, estimation, 888
cross-classification, 911t
monotonic trend information, cross-classification, 888t
PRxVRxAR cross-classification, 900t
Two-group latent growth modeling analyses, 1013
Two-method planned missing data design, 789
candidate constructs, usage, 788t
Two-method planned missing data design, candidate constructs (usage), 788t
Two-method planned missing design, 787–789
Two-method planned missing model, variations, 788f
Two-parameter logistic (2-PL) IRT analysis, 1055
Two-state anxiety questionnaire parallel forms, means/linear trends (first-order CFA), 886t
Typical developmental patterns, 886
Typical trajectories, 877

UK Millennium Cohort study, 134
Unconditional model, 68
Undermining effect, 399
Unidirectional comorbidity, bidirectional comorbidity (contrast), 59–60
Unified individual, understanding, 813
Uniformity myths, 799
Univariate growth mixture modeling, fit indices, 1030t
Univariate latent change score models, 1015–1017
Universal mental health screening, importance, 19
Unmeasured confounders, regression discontinuity designs (usage), 146–147
Unstructured change, 999
U.S. Preventive Services Task Force (USPSTF), hypertension screening declaration, 172

Validity
cultural validity/norms, 16
diagnostic interviews, 34
longitudinal developmental context, 14
measurement ability, 13–14
types, 14
Values, 446
Vanderbilt Therapeutic Alliance Scale, 1026
VAR. *See* Vector autoregressive
Variability
impact, 724
self-organization, 704–707
Variable-oriented approach, 797, 800–803
description, 799–812
limitations, 801–803
Variable oriented, term (usage), 867
Variable x variable correlation matrix, 855
Variance, estimators, 1061
Vector autoregression model, 853–854
Vector autoregressive (VAR) process, 858
Verbal aggression against adults (VAAA), 820, 879, 899
path model, 821f
two-group model, 821f, 827f
Verbal communication, research, 279–280
Verbal emotional expressiveness, 277–278
emotional dysfunction risk, association, 278–280
Vertical-integrative neurobiological model, 290
Very early childhood (ages 2–4)
I-Self, impact, 475
Jamesian I-Self functions, 475
Victimization. *See* Peer victimization
assessment, 544
experiences, 546
forms, 544–545
study, 641
Vignettes, creation, 501
Vineland Adaptive Behavior Scales II, 36
Violence. *See* School system
frequency, cross-classification, 894t
Violent revenge, 498–503
negative self-processes, role, 473
Virginia Twin Study of Adolescent Behavioral Development (VTSABD), 120
Vuong-Lo-Mendell-Rubin likelihood ratio test (VLMR-LRT), 1029

Ward's method, 830, 834t, 835t
usage, 836t
Wave-missing designs, 789–790
Weak monotonicity, 887
Wechsler Memory Scores (WMS), 1022
Well-being, I-Self (impact), 473–474
Whole-family processes, 616
Willi-Prader syndrome, 275
WinBUGS, 996
Within-cohort predictive correlations (Dutch children), 63f
Within-family risk dynamics, 69
Within-grid analyses, 749
Within-individual changes, 960
Within-study correlation, 1062
Within-study variability, 1059
Woodcock-Johnson Applied Projects subtest, usage, 991
World Mental Health Survey, 116
Worthlessness, 417
Wouters, 393

Young Adult Alcohol Problems Screening Test (YAAPST), 1071
Young adults
antisocial peers/peer victimization, involvement, 649
anxiety symptoms/disorders, 635–637
attention-deficit/hyperactivity disorder (ADHD), 643–644
cognitive dispositions, 538
comorbidity, 644–645
depression/depressive symptoms, 633–635
eating disorders, 642–643
externalizing behaviors, development, 638
externalizing problems/disorders, 637–642
friendships, number/quality, 647–648
internalizing problems/disorders, 633–637
intimate partner violence (IPV), 640–641
mental health (promotion), I-Self (role), 639
methodological issues, 652–653
measurement issues, 652–653
mediating/moderating variables, 653
psychological outcomes/comorbidity issues, 653
public health implications/translational efforts, 653
sample issues/study populations, 652
study designs, 652
mixed-sex friendship groups, 639
Oregon Youth Study (OYS)/Oregon-Couples Study, 638–639
peer context, 647–650
peers/partners, online communication, 649–650
relationships, behavior/experiences, 634–635, 636–637
romantic involvement, 633–634, 636, 639–640
romantic relationships, 631–633
peer selection/socialization processes, 648–649
same-sex relationships, 646–647
theory development, issues, 651
translational implications, 653–655
Young children
assessment, parent-caregiver reports, 12
behavioral problems, checklist/questionnaire measures, 21t–22t, 25t
caregiver report tools, characteristics, 11t
caregiving contexts, 6–9
changes, 1
contextual influences, sensitivity, 6–9
depression, 3–4

development, domains, 9–17
disruptive behavior disorders, 4
early problems, importance, 2–4
emotional information, perception, 285
impairment, assessment, 35–36
information, caregivers (reliance), 4–6
Major Depressive Disorder, 4
negative emotion regulation, 292
observational assessment, 35
parent report tools, characteristics, 11t
post-traumatic stress disorder (PTSD), 3
problems, identification, 16–17
psychiatric diagnosis, progress, 3–4
psychometric properties, understanding, 12
psychopathology, assessment, 1
screening methods, 18–19
characteristics, 19–20
screening tools, 20, 23
self-initiated distraction, 292–293
social competence, age effects (illustration), 16f
social-emotional problems, checklist/questionnaire measures, 21t–22t, 25t
sociocultural factors, 7–9
Young children, assessment
approach/tool, selection, 11–17
checklist measures, coverage (breadth/depth), 26t–27t
classification, validity, 14–15
considerations, 4–9
diagnostic approaches, 29–30
Diagnostic Infant and Preschool Assessment (DIPA), 32
diagnostic interviews, 30–32
interrater reliability, 33–34
psychometric properties, 32–34
test-retest reliability, 32–33
types, 31t
validity, 34
findings, sociocultural factors (impact), 8t
multidomain checklists, 25, 27–28
normatization, 15–16
Preschool Age Psychiatric Assessment (PAPA), usage, 30–32
reliability, 12–13
interpretation rules, 13t
statistics, 13t
research, continuation, 37–38
response formats, 17
tools, types, 11–12
validity
cultural validity/norms, 16
longitudinal developmental context, 14
measurement ability, 13–14
Youth Risk Behavior Surveillance System (CDC), 119
Youth Self-Report (YSR), 65, 82, 1032
Yule's Q, representation, 610

Z test, 874
Zuid Holland Longitudinal Study, 73–74

WILEY END USER LICENSE AGREEMENT

Go to www.wiley.com/go/eula to access Wiley’s ebook EULA.