CHILD AND ADOLESCENT PSYCHOPATHOLOGY

SECOND EDITION

EDITED BY
THEODORE P. BEAUCHAIN
STEPHEN P. HINSHAW
Child and Adolescent Psychopathology
# Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foreword</td>
<td>vii</td>
</tr>
<tr>
<td>Preface</td>
<td>xi</td>
</tr>
<tr>
<td>List of Contributors</td>
<td>xv</td>
</tr>
<tr>
<td><strong>Part I  THE DEVELOPMENTAL PSYCHOPATHOLOGY APPROACH</strong></td>
<td></td>
</tr>
<tr>
<td><strong>TO UNDERSTANDING MENTAL ILLNESS</strong></td>
<td></td>
</tr>
<tr>
<td>1 Development Psychopathology as a Scientific Discipline</td>
<td>3</td>
</tr>
<tr>
<td><em>Stephen P. Hinshaw</em></td>
<td></td>
</tr>
<tr>
<td>2 Development Psychopathology and the Diagnostic and Statistical</td>
<td>29</td>
</tr>
<tr>
<td>Manual of Mental Disorders</td>
<td></td>
</tr>
<tr>
<td><em>Theodore P. Beauchaine, Daniel N. Klein, Nora L. Erickson, and Alyssa L. Norris</em></td>
<td></td>
</tr>
<tr>
<td>3 Genetic and Environmental Influences on Behavior</td>
<td>111</td>
</tr>
<tr>
<td><em>Theodore P. Beauchaine and Lisa M. Gatzke-Kopp</em></td>
<td></td>
</tr>
<tr>
<td><strong>Part II  VULNERABILITIES AND RISK FACTORS FOR</strong></td>
<td></td>
</tr>
<tr>
<td><strong>PSYCHOPATHOLOGY</strong></td>
<td></td>
</tr>
<tr>
<td>4 Risk and Resilience in Child and Adolescent Psychopathology</td>
<td>143</td>
</tr>
<tr>
<td><em>Bruce E. Compas and Charissa Andreotti</em></td>
<td></td>
</tr>
<tr>
<td>5 Child Maltreatment and Risk for Psychopathology</td>
<td>171</td>
</tr>
<tr>
<td><em>Sara R. Jaffee and Andrea Kohn Maikovich-Fong</em></td>
<td></td>
</tr>
<tr>
<td>6 Impulsivity and Vulnerability to Psychopathology</td>
<td>197</td>
</tr>
<tr>
<td><em>Emily Neuhaus and Theodore P. Beauchaine</em></td>
<td></td>
</tr>
<tr>
<td>7 Behavioral Inhibition as a Temperamental Vulnerability to</td>
<td>227</td>
</tr>
<tr>
<td><em>Psychopathology</em></td>
<td></td>
</tr>
<tr>
<td><em>Jerome Kagan</em></td>
<td></td>
</tr>
<tr>
<td>8 Beyond Allostatic Load</td>
<td>251</td>
</tr>
<tr>
<td><em>Bruce J. Ellis, Marco Del Giudice, and Elizabeth A. Shirtcliff</em></td>
<td></td>
</tr>
<tr>
<td>9 Exposure to Teratogens as a Risk Factor for Psychopathology</td>
<td>285</td>
</tr>
<tr>
<td><em>Nicole A. Crocker, Susanna L. Fryer, and Sarah N. Mattson</em></td>
<td></td>
</tr>
</tbody>
</table>
Contents

10 Brain Injury as a Risk Factor for Psychopathology 317
Katherine E. Shannon Bowen and Lisa M. Gatzke-Kopp

11 Emotion Dysregulation as a Risk Factor for Psychopathology 341
Pamela M. Cole, Sarah E. Hall, and Nastassia J. Hajal

Part III EXTERNALIZING BEHAVIOR DISORDERS

12 Attention-Deficit/Hyperactivity Disorder 377
Joel Nigg

13 Oppositional Defiant Disorder, Conduct Disorder, and Juvenile Delinquency 411
Irwin D. Waldman and Benjamin B. Lahey

14 Development of Adult Antisocial Behavior 453
Thomas J. Dishion and Kristina Hiatt Racer

15 Substance Use Disorders in Adolescence 489
Sandra A. Brown, Kristin Tomlinson, and Jennifer Winward

Part IV INTERNALIZING BEHAVIOR DISORDERS

16 Anxiety Disorders 513
Carl F. Weems and Wendy K. Silverman

17 Depressive Disorders 543
Daniel N. Klein, Autumn J. Kujawa, Sarah R. Black, and Allison T. Pennock

18 The Development of Borderline Personality and Self-Inflicted Injury 577
Sheila E. Crowell, Erin A. Kaufman, and Mark F. Lenzenweger

Part V OTHER DISORDERS

19 Bipolar Disorder 613
Joseph C. Blader and Gabrielle A. Carlson

20 Autism Spectrum Disorders 649
Susan Faja and Geraldine Dawson

21 Childhood Schizophrenia 685
Robert F. Asarnow

22 Eating Disorders 715
Eric Stice and Cara Bohon

Author Index 739

Subject Index 765
Foreword

The field of developmental psychopathology first came into ascendance during the 1970s, predominantly by being highlighted as an important perspective by researchers conducting prospective longitudinal studies of children at risk for developing schizophrenia. Epidemiological investigations of families exhibiting discord and disruption (but where there was no parental mental disorder), studies of links between cumulative risk factors and developmental outcome, investigations of children with handicapping conditions, and research on cognitive and socio-emotional development in children with autism were among those areas that were influential in the field’s emergence. Conceptualizations of the nature of mental disorder, etiological models of risk and resilience, scientific questions that were posed, and research designs and data analytic strategies were all reexamined, challenged, and cast in a new light by developmental psychopathologists.

The belief that the study of typical developmental processes informs understanding of pathological development and, conversely, that the study of pathological development informs the understanding of normative development, is one of the central tenets of developmental psychopathology—an interdisciplinary science that strives to reduce the schisms that so often separate scientific research from the application of knowledge to clinical populations. The field of developmental psychopathology owes its emergence and coalescence to a number of historically based endeavors in a variety of disciplines, including embryology, genetics, and the neurosciences, as well as psychoanalysis, psychiatry, and psychology. As such, developmental psychopathology provides an example of the synergistic contributions of previously disparate fields that result in the emergence of a new discipline. Somewhat surprisingly, given its historical roots, most of the research conducted on both the development of psychopathology and the processes contributing to resilience focused on relatively narrow domains of variables until the late 1990s and early years of the present millennium.

Over the past several decades, there has been a veritable explosion in our knowledge of developmental neurobiology, that area of neuroscience that focuses on factors regulating the development of neurons, neuronal circuitry, and complex neuronal organization systems, including the brain. Additionally, technological advances in the fields of neuroimaging and molecular genetics have contributed to progress in our understanding of normality, psychopathology, and resilience. Consequently, it has become increasingly acknowledged that the investigation of developmental processes, both normal and abnormal, is an inherently interdisciplinary enterprise.
It is now apparent from the nature of the questions addressed by developmental psychopathologists that progress toward a process-level understanding of mental disorders will require research designs and strategies that call for the assessment of multiple domains and multiple levels of variables, both within and outside the developing person. Likewise, research on the developmental pathways to resilience, the achievement of positive adaptation in the face of significant adversity, must follow these interdisciplinary multiple-levels-of-analysis perspectives. To comprehend typical development, psychopathology, and resilience fully, all levels of analysis must be examined and integrated. Multiple levels of analysis are necessary because no one level is sufficient to explain the complexity inherent in the study of development and psychopathology.

Developmental psychopathology is an exciting and complex field. A major goal of graduate and postdoctoral training in developmental psychopathology is to develop the next generation of scholars so that they can go on and launch their own research careers. An important early step in this process is student access to scholarly volumes that demonstrate the depth and breadth of the field in a clear and accessible way. Thus, this edited volume is a long-awaited, much needed, unique and innovative contribution to the field. It is organized around highlighting the principles and major tenets of developmental psychopathology into a work that does not shy away from presenting students, scholars, and clinicians with our current knowledge regarding the multilevel complexity of typical and atypical development.

The editors of this volume, Ted Beauchaine and Steve Hinshaw, are each leading theorists and researchers in developmental psychopathology. They both subscribe to multilevel research and also have engaged in impressive translational research through developing and implementing preventive interventions for high-risk youth that were influenced by basic research findings.

Ted Beauchaine has made seminal contributions to the understanding of the biological underpinnings of a number of mental and personality disorders in children and adolescents and has conducted exemplary research on the prevention of these conditions. Beauchaine also has utilized numerous psychophysiological measures in his research on attention deficit hyperactivity disorder, conduct problems, disinhibitory psychopathology, depression, and teen self-injury. Importantly, Ted also possesses superb quantitative skills that enable him to conduct data analyses across multiple levels of analysis.

Steve Hinshaw has made classic contributions on the role of the family and peer relationships to typical and atypical development. He also has completed impressive multilevel research on externalizing problems (i.e., conduct disorder, attention deficit disorder) and on behavior problems and psychopathology in girls. Furthermore, Hinshaw has implemented combinations of psychosocial and pharmacologic intervention for children with externalizing expressions of dysfunction. Finally, Steve has been a major advocate and contributor to the importance of destigmatizing mental illness. He has written two powerful volumes on this topic—one a personal account of his father’s lifelong struggle with bipolar disorder (*The Years of Silence Are Past: My Father’s Life with Bipolar Disorder*, 2002), the other a scholarly account of the history of the stigmatizing treatment of persons with mental illness.
that also reviews the extant research on stigma from various scientific perspectives and provides recommendations for future research, intervention, and social policy (The Mark of Shame: Stigma of Mental Illness and an Agenda for Change, 2007).

In order to have this volume adhere to the tenets of developmental psychopathology, Beauchaine and Hinshaw instructed the contributors to take a multiple-levels-of-analysis approach to their assigned chapter topic. All of the authors in this volume are world-class scholars in their area of expertise. Importantly, each contributor communicates clearly, thus enabling graduate students and professionals from a variety of disciplines to develop a firm grasp of psychopathology and resilience in their multi-system entirety.

Major high-risk conditions and mental disorders are given excellent coverage, as are processes contributing to the development of resilient functioning in individuals experiencing significant adverse experiences. The breadth and depth of each chapter’s content provides the reader with a deep appreciation of the complex nature of normality, psychopathology, and resilience. The topics and issues addressed in these chapters are immensely important – not only for the developmental sciences, but also for a number of other scientific fields.

I have been teaching graduate courses in developmental psychopathology for over 30 years. I have often searched for appropriate textbooks on psychopathology to assign to students that were undergirded by the principles of developmental psychopathology. Although there were a number of good textbooks available, few were truly developmental in their organization and content. One of the books that was guided by the tenets of developmental psychopathology was a multi-volume set that Donald Cohen and I co-edited on Developmental Psychopathology. These extensive volumes were not practical to assign to graduate students for a semester-long course. Thus, I have never assigned a required textbook for my courses on developmental psychopathology. My course syllabus was composed solely of journal articles and book chapters that the students were required to read.

Now that I have read this excellent volume edited by Ted Beauchaine and Steve Hinshaw, I believe that I have finally found the answer to my 30-plus-year search for a developmental psychopathology textbook. The volume is full of rich and exciting ideas that will help students develop a passion for the field of developmental psychopathology. I fully anticipate that my colleagues across the country will feel similarly and that the Beauchaine and Hinshaw text will play an important role in the training of future generations of developmental psychopathologists.

Dante Cicchetti, Ph.D.
McKnight Presidential Chair
William Harris Professor
and Professor of Child Psychology and Psychiatry
Institute of Child Development
University of Minnesota
Preface

SOCIETAL COSTS OF mental illness—in terms of both morbidity and mortality—are staggering. In 2002, the most recent year for which data are available, 154 million people worldwide suffered from depression, 106 million suffered an alcohol or drug use disorder, 25 million suffered from schizophrenia, and nearly 1 million died by suicide (World Health Organization, 2012). In low- and middle-income countries, mental disorders account for 25% and 34%, respectively, of total years lived with disability, yet the large majority of those affected receive no form of treatment (WHO World Mental Health Survey Consortium, 2004). Although treatment rates are slightly higher in wealthy countries, mental disorders continue to carry significant stigma. As a result, many avoid seeking help, and a lack of treatment parity remains for mental disorders vs. other health-related conditions (Hinshaw, 2007).

When we wrote the preface to the first edition of this book, we noted how important it is to elucidate the causes of mental illness. After all, the better we understand etiology across all relevant levels of analysis, including genetic, neural, familial, and cultural (to name a few), the better position we are in to formulate effective prevention and intervention programs (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Thus, even though this is not a text about treatment, we hope readers will keep in mind while digesting each chapter how important it is to identify the causes of mental illness in our efforts to reduce human suffering. In this regard, we live in an exciting and promising time. The Human Genome Project, completed only 10 years ago, sequenced 3 billion chemical base pairs that comprise human DNA. Following from this and major advances in psychiatric genetics, our understanding of molecular genetic vulnerabilities to mental illness continues to improve, even though new paradigms are constantly emerging. Similarly, the application of modern neuroimaging techniques—particularly fMRI—has advanced our understanding of neural vulnerabilities to psychopathology. Especially exciting is recent research demonstrating how neural functioning mediates links between genetic vulnerability and high risk personality traits that predispose individuals to psychopathology (e.g., Buckholtz et al., 2008).

Traditionally, most of what we learned about mental illness was obtained through observation and classification of symptoms (see Chapter 2). Although useful in the early stages of identifying different forms of mental illness, symptom classification often tells us little if anything about underlying causal processes—be they biological or environmental—that lead to a particular disorder. Accordingly, in editing
this book we sought authors with expertise in the developmental psychopathology perspective, which emerged only about 30 years ago (see Chapter 1). This perspective follows from the observation that human behavioral traits—including those that predispose to psychopathology—almost always arise from complex transactions between biological vulnerabilities and exposure to environmental risks across development. For example, heritable disorders such as depression, schizophrenia, and substance dependence are affected strongly by environmental experiences, and the effects of environmentally-transmitted risks such as child maltreatment are moderated by genes and other biological predispositions. Furthermore, through epigenetic mechanisms, the expression of several genes implicated in behavior regulation can be altered by experience including exposure to stress and trauma—findings that defy anachronistic distinctions between nature and nurture.

This shift in the scientific landscape—from a relatively static view of psychopathology based on specific clusters of behavior to a dynamic view of disorders emerging from complex transactions between vulnerabilities and risks across time—served as the impetus for the first edition of this book. It continues to drive the current second edition, which includes considerable new material. Before the first edition was published, most graduate-level psychopathology texts were organized around symptom-based approaches to classifying mental illness, with limited consideration of the genetic and neural underpinnings of behavior, or of unfolding of interactions between biological vulnerabilities and environmental risk across time. However, in the five years since the first edition was published, appreciation for the complexity of such transactions in the development of psychopathology has increased, and many new and exciting findings have emerged. The timing was therefore right for a second edition.

Some readers will likely note that a few disorders often covered in psychopathology texts are not included in this book. For example, we do not address developmental disabilities or mental retardation. In omitting these disorders, we are not implying that they are unimportant. Rather, the ever expanding literature addressing developmental disabilities makes it difficult to cover the topic adequately in a text that already includes 22 chapters. Thus, we were left with a difficult choice, and we decided not to limit coverage of the conditions contained herein. We refer interested readers to other sources (e.g., Jacobson, Mulick, & Rojahn, 2007) for excellent coverage of developmental disabilities and mental retardation.

We now invite you to join us in the quest for a deepened understanding of mental disorders and conditions that originate in childhood and adolescence. We hope that our emphases on genetic and other biological vulnerabilities, and how these interact with environmental risk factors and contexts, will challenge your preconceived notions as to what is “biological” and what is “environmental” in relation to normal and atypical development and psychopathology. We hope as well that our coverage will prompt a new generation of investigators, clinicians, and policymakers to pursue the daunting but essential goal of explaining, treating, and preventing the devastation that so often accompanies psychopathology.

Theodore P. Beauchaine
Stephen P. Hinshaw
REFERENCES


List of Contributors

Charissa Andreotti
Vanderbilt University, TN

Robert F. Asarnow
UCLA School of Medicine

Theodore P. Beauchaine
Washington State University

Sarah R. Black
University at Stony Brook, NY

Joseph C. Blader
Stony Brook University School of Medicine, NY

Cara Bohon
University of California, Los Angeles

Katherine Shannon Bowen
University of Wisconsin

Sandra A. Brown
Veterans Affairs San Diego Healthcare System, CA

Gabrielle A. Carlson
Stony Brook University School of Medicine, NY

Pamela M. Cole
Pennsylvania State University

Bruce E. Compas
Vanderbilt University, TN

Nicole A. Crocker
SDSU/UCSD Joint Doctoral Program in Clinical Psychology, CA

Sheila E. Crowell
University of Utah

Geraldine Dawson
Autism Speaks, NY

Marco Del Giudice
Center for Cognitive Science, Department of Psychology, University of Turin, Italy

Thomas J. Dishion
Arizona State University

Bruce J. Ellis
John and Doris Norton School of Family and Consumer Sciences, AZ

Nora L. Erickson
Washington State University

Susan Faja
University of Washington

Susanna L. Fryer
University of California, San Francisco

Lisa M. Gatzke-Kopp
Pennsylvania State University

Nastassia J. Hajal
Pennsylvania State University
List of Contributors

Sarah E. Hall
Wheaton College, IL

Stephen P. Hinshaw
University of California, Berkeley

Sara R. Jaffee
Institute of Psychiatry, London

Jerome Kagan
Harvard University

Erin A. Kaufman
University of Utah

Daniel N. Klein
University at Stony Brook, NY

Autumn J. Kujawa
University at Stony Brook, NY

Benjamin B. Lahey
University of Chicago

Mark F. Lenzenweger
State University of New York at Binghamton

Andrea Kohn Maikovich-Fong
Colorado Blood Cancer Institute at Presbyterian/St. Luke’s Hospital

Sarah N. Mattson
San Diego State University, CA

Emily Neuhaus
University of Colorado School of Medicine

Joel Nigg
Oregon Health and Science University

Alyssa L. Norris
Washington State University

Allison T. Pennock
University at Stony Brook, NY

Kristina Hiatt Racer
University of Oregon

Elizabeth A. Shirtcliff
University of New Orleans, LA

Wendy K. Silverman
Florida International University

Eric Stice
Oregon Research Institute

Kristin Tomlinson
University of California, San Diego

Irwin D. Waldman
Emory University, GA

Carl F. Weems
University of New Orleans, LA

Jennifer Winward
University of California, San Diego
CHAPTER 1

Developmental Psychopathology as a Scientific Discipline
Rationale, Principles, and Advances

STEPHEN P. HINSHAW

From its “launch” between three and four decades ago (see Achenbach, 1974; Cicchetti, 1984; Sroufe & Rutter, 1984), developmental psychopathology (DP) has become a force to be reckoned with. DP is at once a perspective on the origins of mental disorders that begin during childhood and adolescence, a multidisciplinary conceptual approach linking normative development to psychopathology, and a scientific discipline closely tied to clinical child/adolescent psychology and psychiatry but transcending the usual diagnosis-based emphasis of these fields (Cicchetti & Cohen, 2006; Cicchetti & Toth, 2009). Through its focus on the dynamic interplay of biology and context, genes and environments, and “inner” versus “outer” influences on the development of healthy and atypical functioning, it has come to dominate current thinking and research on psychopathology. Some of its core ideas are not new, having emerged in the context of embryology, systems theory, philosophy, and genetics long ago (see Cicchetti, 2006; Gottlieb & Willoughby, 2006, for elaboration). Yet the syntheses represented in this volume, reflecting DP’s continuing growth, are truly cutting edge, given the relatively recent emergence of DP and given the knowledge explosion in recent years related to psychobiological influences as they transact with contextual forces. Today, scientists from diverse disciplines contribute to ever-expanding knowledge of this enterprise while clinicians benefit from and utilize its core principles. The underlying perspectives are no longer revolutionary; instead, they have come to comprise the dominant paradigm.

In this, our second edition of a graduate-level compendium on core aspects of this vast topic, we continue our tradition of providing current, conceptually based, clinically relevant, and developmentally informed information on causal mechanisms underlying child and adolescent psychopathology. Leading scientists across the entire field have contributed state-of-the-art summations of their particular
areas of expertise; we owe them a great debt for their efforts toward translating the complex findings into each chapter synthesis. Indeed, every entry in this edition is brand new, which is entirely necessary given how much the science has advanced across the several years since the first edition (Beauchaine & Hinshaw, 2008).

WHY DEVELOPMENTAL PSYCHOPATHOLOGY?
To contextualize and put into perspective why this entire area is so important, one must first consider the high levels of suffering involved in child and adolescent psychopathology, including the severe pain and restricted life opportunities experienced by not only affected children and adolescents but also by families, schools, and in some cases communities and society at large. Emotional and behavioral problems in youth are not only distressingly prevalent but also hugely impairing, leading to serious problems in such crucial domains as academic achievement, interpersonal competencies, and independent living skills. Distress is often intense; individuals may engage in behavior patterns that are highly destructive to their own development as well as the well-being of others.

For example, depression is associated with high degrees of hopelessness and despair, anxiety disorders with severe restrictions on exploration, bipolar disorder with disruption and chaos as well as high risk for suicidality, attention-deficit hyperactivity disorder with major deficits in academic and social arenas as well as risk for accidental injury, conduct problems with both violence and victimization, eating disorders with threats to physical well-being and healthy self-image, autism and other pervasive developmental disorders with severe isolation and major skill deficits, and substance use/abuse with squandered opportunities and major health risks. Child maltreatment is linked, in too many instances, to tragic developmental consequences, and the origins of personality disturbance are linked to major risk for self-harm and interpersonal disasters. Although lifelong pain and impairment are not inevitable, as we know from investigations of resilience (e.g., Luthar & Brown, 2007; Sapienza & Masten, 2011), mental disorders are quite likely to “up the ante” for devastation.

Second, the costs of mental disturbance are huge in other ways. Health care expenditures rise dramatically, educational and occupational milestones are likely to be hugely delayed or lost altogether, and deficits in later employability are often staggering, with major economic consequences (e.g., Murray & Lopez, 1996; Robb et al., 2011). Thus, beyond personal and family suffering, disabling skill deficits and harsh economic realities frequently accrue from mental disorder.

Third, not only do behavioral, emotional, and developmental disturbances in childhood and adolescence typically persist into adulthood, but what are often considered to be “adult” mental disorders often have precursors in the early years of development (Kessler et al., 2005). All too often, symptoms and impairments start early and remain problematic for years to come.

Given this set of deeply human, enormously costly, and persistent needs, why not rely on traditional clinical efforts in psychology and psychiatry, with their long, venerable histories? As detailed in earlier treatises, these efforts have too often
yielded overly broad and static categories of mental disorders, with insufficient attention paid to biological vulnerabilities, contextual influences, multilevel chains of causation, dynamic and transactional influences, and divergent life-course pathways within a given diagnostic category (e.g., Cicchetti, 1990). The reciprocally deterministic nature of development, both typical and atypical, is not well captured in such diagnostic systems. As a function of the huge expansion of knowledge in a host of related fields and subfields, the complex yet compelling perspectives offered by DP have taken hold with increasing pace. Without them, traditional models seem sterile and impoverished.

Yet despite the utter scientific and clinical urgency surrounding this topic, important barriers stand in the way of increased scientific understanding and access to evidence-based treatment. Perhaps the primary reason is that mental disturbance at any age is still highly stigmatized (e.g., Hinshaw, 2007; Hinshaw & Stier, 2008). Intensive stigma and shame prevent help seeking and serve to render mental health a lower priority than physical health, despite the inextricable linkages between the two. Intriguingly, although we appear to be a far more open and accepting culture regarding mental health than half a century ago—and although public knowledge of mental illness has grown considerably since the 1950s—the U.S. public is more likely to link mental illness with dangerousness, and it wishes for greater social distance from those with mental disorders, than in the past (see Phelan, Link, Stueve, & Pescosolido, 2000). The reasons are complex but may relate to (a) increased numbers of seriously impaired individuals on the streets, without needed community services and resources; (b) enhanced public awareness that “dangerousness” is one of the few reasons that can still lead to involuntarily commitment; and (c) the tenuousness of the evidence that biogenetic ascriptions to mental illness (i.e., that it is a “brain disease” or a “disease like any other”) can eliminate stigmatization (see Hinshaw, 2007; Jorm & Griffiths, 2008; Martinez, Piff, Mendoza-Denton, & Hinshaw, 2011; Pescosolido et al., 2010). DP perspectives promote complex as opposed to simplistic or reductionistic conceptions of mental disorder, leading to both enhanced scientific progress and, it is hoped, a more realistic view on the part of the general public, emphasizing multidetermined pathways but not personal weakness or blame.

In all, despite the major advances in basic science and clinical applications in recent years, which we highlight in the following pages, the field’s knowledge of developing brains and minds in multiple, interacting contexts is still rudimentary. How could it be otherwise, given the sheer complexity of our topic matter? Still, for those who enjoy a challenge—and those who are excited by questions that will take many years and many great minds to answer, with the potential for a payoff of bettering the human condition—we sincerely hope that our chapters serve as a call to join the major scientific and clinical efforts needed in the decades ahead. Indeed, if the field is to continue to make headway toward understanding, treating, and preventing the serious clinical conditions that emerge during childhood and adolescence, the best minds of the current generation are required.

We admit that the multilayered nature of the topic at hand, paired with the huge numbers of risk factors (biological, experiential, and contextual) that promote disturbed functioning and the many protective factors that might mitigate such
risk, can serve to delay needed translational efforts from DP-related insights to evidence-based treatment strategies (Cicchetti & Toth, 2009). Although this book is, by design, not a volume on intervention, our ultimate hope is that the intentional application of advances in basic science to clinical practice and prevention will occur at an ever-increasing pace.

OVERVIEW OF APPROACH

In the chapters that follow, our core objective is to bring to life DP’s core tenets and principles into a useful guide for students, clinicians, and scholars, in order to facilitate deepened understanding of the major forms of child and adolescent behavioral and emotional disturbance. To meet this aim, we have asked leaders in the field to present up-to-date material that is simultaneously developmentally based, clinically relevant, and directly inclusive of the types of psychobiological formulations that are gaining ascendancy in the entire mental health enterprise. In other words, we aim to supplement the kinds of developmental, process-oriented constructs typically linked to DP with appreciation of core findings in behavioral and molecular genetics, neural pathways, and brain plasticity that have risen to prominence in recent years.

Thus, in our instructions to the volume’s contributors, we asked explicitly for coverage of historical context, epidemiologic factors, diagnostic issues, sex differences, cultural variables, developmental processes, and important psychobiological mechanisms that can illuminate the forms of pathology under discussion in their particular chapter. At the same time, we emphasized that neurobiological processes must not be represented in reductionistic fashion. Indeed, those contextual factors—familial, cultural, school-related—viewed as the predominant causal mechanisms throughout much of the last century are now known to interact and transact with biological vulnerabilities and risk variables to produce both maladaptation and healthy adaptation across development. Thus, we urged our authors to consider vulnerability and risk across multiple levels of analysis, emphasizing transaction across a range of individual and contextual factors in the genesis of (or desistance from) psychopathology. Indeed, modern views of behavioral and molecular genetics have placed into sharp relief the unique and interactive roles that environmental and cultural forces exert on development (e.g., Cicchetti & Curtis, 2006; Dodge & Rutter, 2011; Rutter, Moffitt, & Caspi, 2006).

Given page limitations and our desire for focused rather than exhaustive coverage, each chapter is relatively brief, with the goal of providing cogent, recent, and incisive commentary on conceptual issues, clinically relevant material, neuroscientific advances, and interactive models. We strongly hope that readers use these contributions as a springboard for further exploration of conceptual frameworks, empirical research on etiology and mechanisms, and bases for prevention and treatment.

As noted earlier, despite the considerable advances that have been made, the road ahead is long. One can only wonder at what scholars a century from now will make of our preliminary attempts to model the hugely complex developmental pathways,
processes, and trajectories linked to psychopathology. After all, the human genome was decoded only a decade ago, and high-resolution brain scanning is still a relatively young field of endeavor. Still, throughout the following pages, I highlight key advances that have been made in recent years regarding DP processes, methods, and models, signifying that the enterprise has already yielded unprecedented insights and discoveries.

**DP CONCEPTS AND PRINCIPLES**

What characterizes a truly developmental view of psychopathology, as opposed to the kinds of descriptive, symptom-focused presentations that still dominate most classification systems and that still permeate many ideas in the field? As discussed in key readings (e.g., Cicchetti & Cohen, 2006; Cicchetti & Toth, 2009; Mash & Dozois, 2003; Rutter & Sroufe, 2000; Sameroff, Lewis, & Miller, 2000), several core points are commonly viewed as central to the DP perspective. These include the necessity of (a) interweaving studies of normal development and pathological functioning into a true synthesis; (b) examining developmental continuities and discontinuities of traits, behavior patterns, emotional responses, and disorders; (c) evaluating evidence across multiple levels of analysis (from genes to cultures, including the intermediate levels of individuals, families, schools, and neighborhoods); (d) incorporating distinct perspectives, including clinical and developmental psychology, child and adolescent psychiatry, genetics, neurology, public health, philosophy of science, and many others, into a truly multidisciplinary effort; (e) exploring both risk and protective factors and their interplay, so that competence, strength, and resilience as well as pathology and impairment can be understood; (f) involving reciprocal, transactional models of influence in the field’s causal models, through which linear patterns of association and causation are replaced by probabilistic, dynamic, nonlinear, and complex conceptual models; and (g) capturing the importance of social and cultural context both in understanding the function and meaning of behavioral and emotional patterns and in interacting with biological predisposition to yield disordered functioning.

Three related principles bear emphasis. The first is that multiple pathways to pathology exist. Indeed, disparate routes may lead to a common condition or outcome, exemplifying the construct of *equifinality*. For example, aggressive behavior (or, diagnostically speaking, “conduct disorder”) can result from physical abuse (Chapter 5), from a heritable tendency toward disinhibition (Chapter 3), from injury to the frontal lobes (Chapter 10), from coercive parenting interchanges with the developing child (Chapter 14), from prenatal and perinatal risk factors acting in concert with early experiences of insecure attachment or parental rejection (Chapter 9), or from different combinations of these vulnerabilities and risk factors (e.g., Jaffee, Strait, & Odgers, 2012; Raine, Brennan, & Mednick, 1997; Tremblay, 2010). In other words, separate—and in many cases interacting—causal influences can yield similar clinical endstates. In addition, the concept of *multifinality* applies when a given risk factor or initial state yields disparate outcomes across development. For instance, abuse may or may not lead to severe maladaptation, depending on a host of
The Developmental Psychopathology Approach to Understanding

Intervening factors; extremes of inhibited temperament may produce shyness and social withdrawal, but other, healthier outcomes are also possible, depending on the presence or absence of additional risk or protective factors (see Cicchetti & Rogosch, 1996). Both equifinality and multifinality imply that linear models of association and simplistic categorical conceptions of disorder are incapable of facilitating full understanding of child and adolescent psychopathology. Indeed, such simplistic models may actually be misleading.

Second, DP models place emphasis on person-centered research designs, in which the typical practice of examining global effects of one or more risk/protective variables across an entire sample or population is supplemented by consideration of unique subgroups—whether defined by genotypes, personality variables, socialization practices, neighborhoods, or other key factors—and their particular developmental journeys across the life span (Bergman, von Eye, & Magnusson, 2006). Framed from a slightly different perspective, developmental continuities and discontinuities may well differ across homogeneous subgroups of participants. Even in variable-centered research, key moderator variables and mediator processes must always be considered (e.g., Fairchild & MacKinnon, 2009; Hinshaw, 2002; Howe, Reiss, & Yuh, 2002; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001) to ensure that (a) results are applicable to subsets of participants grouped on the basis of the moderator variable of interest (e.g., male versus female participants, those from different ethnic groups, those with different patterns of comorbidity) and (b) underlying mechanisms of change are considered explicitly.

Third, given the rapid growth in recent years of genetic and genomic models and brain imaging methods, DP researchers in the 21st century must pay increasing attention to the role of the brain—and to neuroscientific principles in general—to account for the wide range of extant pathologies and their devastating effects (Cicchetti & Curtis, 2006). Clearly, we have come a long way from the mid-20th century, when biological and temperamental factors were virtually ignored in accounts of child development and psychopathology.

As noted in the introductory chapter to the first edition (Hinshaw, 2008), a basic mathematical calculation may help to elucidate the underlying complexities here: Adults have approximately 100 billion neurons in their brains; children are born with even higher numbers, perhaps double that figure. Indeed a major developmental “task” over the earliest years of postnatal development is the pruning and migration of such neurons into a working, functional brain. But what is the rate of neural development during the 40 weeks of human gestation? To figure this out, one must divide 200 billion (a fair estimate of the number of neurons with which an infant is born) by the number of seconds in 40 weeks. The result—of dividing the numerator of 2 times 10 to the 11th power (the number of neurons) by the denominator of 2.4192 times 10 to the 7th power (the number of seconds)—is the astonishing figure that, on average, the embryo and fetus are producing approximately 8,000 new neurons every second throughout the entire course of prenatal development. This average is not constant, of course, given that the neural tube and brain do not even form for some weeks. Thus, in some crucial periods, this figure is even higher (see Giedd, Shaw, Wallace, Gogtay, & Lenroot, 2006, for additional information on the
precise timing of neural development across pregnancy and childhood). Rates of connectivity in the developing brain yield numbers that are exponentially higher. As the cortex matures, revealing characteristic patterns of thickening and thinning, and as cortical neurons form rich and lasting connections with other brain regions, the number of synaptic connections goes into the trillions and beyond.

Given such staggering statistics, a key question involves the joint influence of genes, hormones, nutrition, life experiences, and contextual influences on the plasticity of the brain’s development across childhood and adolescence. Without consideration of transactional processes, multilevel models, computational frameworks, gene-environment interplay, and a host of technological and conceptual advances related to the overall field of developmental neuroscience, we will not be able to solve the problem of gaining deep understanding of relevant mechanisms (see also Blakemore, Burnett, & Dahl, 2010; Romer & Walker, 2007; and Steinberg, 2010, for considerations of adolescent brain and behavioral development).

Key concepts and principles related to DP have been stated and restated across a large number of articles, chapters, and books. Indeed, detailed discussion of any one of them could easily fill a volume unto itself. The challenge for the current chapter is to encapsulate these tenets, in the service of foreshadowing and illuminating content on specific risk factors and specific disorders that fill the rest of the book. Because explanations of these concepts too often remain at a rather global and abstract level, leaving unresolved precisely what they suggest for the investigation and treatment of behavioral and emotional disorders, I try, in the following section, to bring these percepts to life.

Normal and Atypical Development Are Mutually Informative

As opposed to the study of discrete, mutually exclusive categories of “disorder,” DP models emphasize that phenomena defined as abnormal represent aberrations in normal developmental pathways and processes. Hence, without a full understanding of typical development, the study of pathology will remain incomplete and decontextualized. Taking just one example, illuminating the nature of attention-deficit/hyperactivity disorder (ADHD) requires thorough understanding of the normative development of attention, impulse control, and self-regulation (Nigg, 2006; Nigg, Hinshaw, & Huang-Pollack, 2006; Chapter 12). Similarly, investigations of autism must be fully integrated with the development of interpersonal awareness and empathy, which typically takes place over the first several years of life. Without such developmental templates, understanding autism may become an empty exercise of counting symptoms (for a developmental approach, see Dawson & Toth, 2006; Chapter 20). Additional instances exist across all forms of disordered emotion and behavior. Currently, few doubt the wisdom of understanding developmental sequences and processes associated with healthy outcomes as extremely relevant to the elucidation and explication of pathology.

Yet the process is conceptualized as a two-way street, with the corresponding view that investigations of pathological conditions—sometimes referred to as adaptation failures in DP terms (see Sroufe, 1997)—can and should provide a unique perspective
The Developmental Psychopathology Approach to Understanding normal developmental mechanisms. In other words, the study of disrupted developmental progressions illuminates our understanding of what is normative. Overall, this core tenet of DP—of the mutual interplay between the study of normality and pathology, along with the perspective that progress in each domain is dependent on progress in the other—is now widely held. Neurology abounds with relevant examples. For example, there is a long tradition utilizing the study of disrupted neural systems to enhance understanding of healthy brain functioning and vice versa. “Split-brain” patients (those who have had their cerebral hemispheres separated to provide relief from specific neurological disorders) provide unprecedented insights into normative brain processes, such that separable functions and even “personalities” subserved by the right versus left hemispheres become evident as a function of the pathology and resultant surgery. In parallel, famous case studies such as HM, in which key brain structures/regions have been surgically removed (in his case the hippocampus), greatly facilitate knowledge about human memory systems (see Gazzaniga, Ivry, & Mangun, 2009). Cicchetti and Curtis (2006) provide lucid detail on neuroscience-related approaches. For a specific example, the study of phenylketonuria (PKU) has implications for elaborating the normative development of executive functions (Diamond, Prevor, Callender, & Druin, 1997). How might this perspective inform our understanding of psychological or psychiatric disorders that are the core subject matter of DP? In other words, beyond neurological conditions and formulations per se, can investigations of pathology inform normal development? Once again, it is now commonly accepted that the more we know about basic emotion, cognition, attention, memory, social awareness, self-regulation, and the like, the more investigations of psychopathology can benefit. Almost no form of mental disorder constitutes a clearly demarcated, qualitatively distinct category or taxon, meaning that processes applying to individuals near the center of the bell curve are likely to apply to those further out on the continuum as well. Nearly all forms of mental pathology appear consistent with a quantitative, dimensional perspective (Beauchaine, 2003), emphasizing the need for flow of information from normal developmental pathways to pathological functioning. But what about the other direction? Specifically, what has been learned about normal developmental processes from studies of child and adolescent psychopathology? At first glance, the situation doesn’t seem to be as heuristic as that in neurology; it may be that we have not gained the kinds of dramatic insights about typical psychological development from studies of child and adolescent psychopathology that have been realized in “harder” scientific endeavors. In other words, the complexity of mental disorders may limit parallels to more simply caused neurological conditions. In short, there are few behavioral and emotional equivalents to the surgical procedures of creating lesions in certain brain tracts or single-gene forms of pathology such as PKU. Yet consider the work on autism by Baron-Cohen (2000). Relevant findings suggest that the lack of social connectedness experienced by individuals with autism may relate to a failure in attainment of a basic “theory of mind,” which deals with the developing realization that other humans have mental states that differ from one’s own. Most normal 4-year-olds can master theory-of-mind tests, suggesting that basic
social understanding is predicated on a domain-specific cognitive module that, once operative, occurs almost automatically. On the other hand, a high percentage of youth with autistic disorder, even those with high levels of intellectual functioning, do not “pass” such psychological tests, suggesting that they have not come to the core realization that fellow humans have different minds and different psychological perspectives from their own.

I note that a number of individuals with high-functioning autism can eventually learn to pass the kinds of experimental tests used to test for theory of mind. Through effortful processing, they come to deduce that other children and adults have a different understanding of events in the world than they do. Yet this effortful skill does not mean that their social interactions become smooth, effortless, and “automatic.” Indeed, the laborious kinds of calculations and inferences made by people with high-functioning autism to understand interpersonal dynamics are not usually accompanied by perfectly functional social interactions (Grandin, 2006). A key implication is that “normal” social-cognitive and social functioning is highly automatic and qualitatively distinct from the ability to deduce social situations analytically more typical in autism—which is time consuming and not perceived as skillful by peers. Thus, disruptions in social cognition and social performance in persons with autism may help to clarify the automatic and highly developed nature of the social cognitions and processes that underlie skilled interpersonal performance in normal development. Parenthetically, I note that current views of theory of mind posit that at least some aspects of understanding false beliefs appear far earlier in life, even toddlerhood and infancy, further challenging developmental models of both normative and atypical development (see, for example, Sodian, 2011).

Another example pertains to work on the reward sensitivity of individuals with ADHD (e.g., Sagvolden, Johansen, Aase, & Russell, 2005). Here, considerable evidence reveals that in people with this condition, large performance decrements occur when rewards are suddenly stopped, presumably related to a dopaminergically mediated problem with responding during extinction. More recent research (Volkow et al., 2009) reveals, in fact, that never-medicated adults with ADHD have markedly deficient numbers of dopamine receptors and transporters in reward and motivational brain pathways than do non-ADHD comparison subjects. This finding has served to revive “motivational” theories regarding the origins of ADHD, revealing a biologically driven undersensitivity to reward. Not only is extrinsic reward necessary to enhance task performance of affected individuals, but reward cessation would be expected to lead to larger-than-normal drop-off of task performance. In all, these insights foster understanding of basic developmental processes and mechanisms underlying dysregulated attention and impulse control, such that ADHD-related reward processes may well elucidate normative patterns of motivation, persistence, and effort.

A third instance, noted briefly, pertains to the horrific “experiments of nature” that occur when infants and toddlers are subjected to brutal institutionalization and lack of human contact during the earliest years of development (for review, see O’Connor, 2006). The development of specific symptom patterns (e.g., inattention
and overactivity, as opposed to aggression; see Kreppner, O’Connor, Rutter, & the English and Romanian Adoptees Study Team, 2001), and the extent of social and cognitive “catch-up” following removal from the institutions, are extremely informative about normal-range development of secure relationships and cognitive performance. Such work has even incorporated experimental methods to understand whether in-home foster placements can mitigate the effects of early deprivation in terms of cognitive growth (Nelson et al., 2007). Indeed, for the previously institutionalized girls in this randomized trial, foster care placement led to improvements in girls’ internalizing behavior patterns, mediated by the gaining of attachment security via the change from institutional care to family placements (McLaughlin, Zeanah, Fox, & Nelson, 2012). Thus, even in a harshly abandoned and deprived sample, attachment processes are implicated in reductions of anxiety and depression. Mediators of competence in more normative samples are still open to exploration.

**Developmental Continuities and Discontinuities**

With this principle, it is commonly asserted that DP models must emphasize both continuous and discontinuous processes at work in the development of pathology. What precisely does this mean? Taking the example of externalizing and antisocial behavior, it is well known that antisocial behaviors show strong stability across time—meaning that correlations are substantial between early measures of aggressive and antisocial tendencies and those made at later times. In other words, the rank order remains relatively preserved, such that the most aggressive individuals at early points in development remain high in such behavior patterns across development. But does this mean that the precise forms of externalizing antisocial behavior remain constant? Clearly not, given that those children with extremes of temper tantrums and defiance during the toddler and preschool years are not especially likely to exhibit high rates of tantrums during adolescence. Rather, they have a high likelihood of displaying physical aggression in grade school, covert antisocial behaviors in preadolescence, and various forms of delinquency by their teen years (e.g., sexual assault, property crime, violence), followed by adult manifestations of antisocial behavior after adolescence (e.g., Moffitt, 1993, 2006). In short, continuities exist, but these are *heterotypic* in nature, as the actual form of the underlying antisocial trait changes form with development.

Another important consideration is that patterns of continuity may differ considerably across separable subgroups that display different developmental patterns or trajectories. Not all highly aggressive or antisocial children remain so, as some are prone to desist with the transition to adolescence. Others, however—the so-called early starter or life-course-persistent subgroup—maintain high rates through at least early adulthood (although, as just noted, the specific forms of the antisocial actions may well change with development). In addition, a large subset does not display major externalizing problems in childhood but instead shows a sharp increase with adolescence (for a review, see Moffitt, 2006). Understanding such continuities and discontinuities via homogeneous subgroups is likely to yield greater understanding than basic plots of overall curves of “growth” across the population. Sophisticated