CHILD AND ADOLESCENT PSYCHOPATHOLOGY

THIRD EDITION

EDITED BY
THEODORE P. BEAUCHAINE
STEPHEN P. HINSHAW

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Contents

Foreword

The remarkable third edition of *Child and Adolescent Psychopathology* represents an academic tour de force presenting the science of development associated with progressions to mental disorder. These processes are typically multiple and interacting. Indeed, their importance is clear, as neurodevelopmental models of psychopathology are dominant today. Sadly, both stigmatization—primarily from profound misunderstanding of mental disorders—and low economic status remain barriers to research and treatment (Martinez & Hinshaw, 2016; Merikangas et al., 2011).

The chapters show remarkable breadth, including the challenge of integrating genetics, brain imaging, brain trauma, and prenatal and physiological as well as environmental variables in a clinically meaningful way. Clinicians have already benefitted from studies detailing patterns of continuity and discontinuity. Indeed, such investigations can help to prevent premature prediction and labeling that in itself may be harmful. These models, as well as the transactional nature of many dysfunctional behaviors, preclude simplistic causal pathways.

Brain imaging has yet to contribute to clinical diagnosis and care, even though longitudinal and large-sample cross-sectional studies are starting to indicate subpopulation developmental brain phenotypes that have integrative potential for developmental psychopathology (Giedd et al., 2015; Gur, 2016). For example, it is possible that different developmental trajectories in attention-deficit/hyperactivity disorder reflect alternate clinical forms, as delayed cortical developmental may well relate to greater improvement in adolescence (Shaw et al., 2013).

In our sister science of developmental neurobiology, true "clinical breakthroughs" have emerged, such as the use of rapamycin for tuberous sclerosis (Franz et al., 2006), and magnesium infusion for prevention of cerebral palsy (Rouse et al., 2008). These are large-effect-size interventions of interest to child psychiatrists because of associated psychopathologies in these conditions. Both were serendipitous discoveries, which by definition cannot be planned. At the same time, it remains troubling how much risk remains embedded in political arenas of community infrastructure (e.g., support for schools, housing, and law enforcement). We must transcend psychobiology to incorporate multiple levels of analysis, as amply shown in the following chapters.

The Research Domain Criteria (RDoC; Cuthbert, 2014), highlighted in a number of chapters, do not represent a truly new approach. Dimensional as well as categorical measures have been hallmarks of NIH-funded psychiatric research for decades (Weinberger, Glick, & Klein, 2015), and neurobiologically founded, multiple-levels-of analysis research has contributed to key advances in our understanding of etiology since at least the mid-20th century (Beauchaine & Thayer, 2015). Evidence is mounting for age- and category-related interactions with dimensional brain MRI measures (e.g., Wiggins et al., 2016). In all, the RDoC provides a useful and surprisingly interactive set of measures.

Finally, I found inspiration in the several authors who reviewed the predictive and possible treatment implications of regulatory physiological measures for developmental psychopathology. Ultimately, these models will be judged on when and how these regulatory processes can be changed, given the complexity of initial measurements and the potential for highly individualized treatment plans. One might read this entire volume as a basis for future personalized therapies, paralleling the present movement in medicine. In all, the chapters herald considerable promise for the future.

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Preface

As we noted in the preface of the second edition of *Child and Adolescent Psychopathology* (Beauchaine & Hinshaw, 2013), global costs of mental illness—in terms of morbidity, mortality, and other forms of human suffering—are staggering. In many developed countries including the United States, over one third of individuals suffer from a major psychiatric disorder at some point in their lives (Kessler et al., 2009). In low- and middle-income countries, mental disorders account for 25% and 34%, respectively, of total years lived with disability, yet most of those affected receive no 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; Martinez & Hinshaw, 2016).

When the two of us met nearly 18 years ago, knowledge of the causes of mental illnesses was quite limited compared to today. Although behavioral genetics studies had shown that most psychiatric disorders are at least moderately heritable, little was known about molecular genetic, neural, or hormonal mechanisms of heritability. Moreover, neither epigenetic alterations in gene expression, nor rare structural variants, had been identified as possible mechanisms through which environment might confer vulnerability to psychopathology. Many prevailing models of mental illness still pitted nature and nurture against each other as competing causes of psychopathology. Transactional models, in which biological vulnerabilities are presumed to interact with environmental risk factors to eventuate in mental illness, were few in number and limited in specification of neurobiological mechanisms, as advanced neuroimaging was in still in its infancy.

Given limitations in technology, most of what we learned about mental illness has traditionally been obtained through observation and classification of symptoms (see Chapter 2 [Beauchaine & Klein]). Although useful in 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 particular disorders. In editing this book, we therefore sought authors with expertise in the developmental psychopathology perspective, which emerged only about 35 years ago (see Chapter 1 [Hinshaw]). 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 conditions such as attention-deficit/hyperactivity disorder, depression, schizophrenia, and substance dependence are shaped strongly by environmental influences, and effects of environmentally transmitted risks such as child maltreatment are moderated by genes and other biological predispositions (see e.g., Beauchaine & McNulty, 2013). Furthermore, through epigenetic mechanisms, the expression of several genes that are implicated in behavior regulation can be altered by experience, including exposure to stress and trauma—findings that defy anachronistic distinctions derived from reductionistic models. Thus, we asked all authors to identify *both* biological *and* environmental contributors to psychopathology and to discuss how these *interact* and *transact* across development to amplify risk.

This dynamic view of mental disorders served as the impetus for both the first and second editions of this book, and continues as a driving force behind the current third edition, which includes substantially updated material. Before the first edition was published, most graduate-level psychopathology texts were organized around the symptom-based approach to classifying mental illness, with limited consideration of the genetic and neural underpinnings of behavior or the interplay between biological vulnerabilities and environmental risk factors across development. However, in the nine 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 (see e.g., Beauchaine & Goodman, 2015).

Elucidating causes of mental illness is an international public health concern. 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 devise more effective prevention and intervention programs (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Thus, even though this text does not address treatment, we hope readers will keep in mind while digesting each chapter how important it is to identify causes of mental illness in our efforts to reduce human suffering. This motivation played a central role in the National Institute of Mental Health (2015) establishing the Research Domain Criteria (RDoC) project. RDoC is a collaboration between NIMH and researchers around the world to develop a neuroscience-informed system of characterizing psychopathology that identifies genetic, neural, hormonal, and social determinants of major behavioral systems that contribute to human function, and at the extremes, mental illness (see Chapter 2 [Beauchaine & Klein]).

Readers will likely note that some disorders that are often addressed in psychopathology texts are not included in this book. For example, we do not cover developmental disorders or intellectual disability. In omitting these disorders, we are not implying that they are unimportant. Rather, the vast literature on developmental disabilities makes it difficult to cover the topic adequately in a text that already includes 24 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., Burack, Hodapp, Iarocci, & Zigler, 2011) for excellent coverage of this domain.

We now invite you to join us in the quest for a deeper understanding of the development of mental disorders, which almost always 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 any preconceived notions you may have about what is "biological" and what is "environmental" in relation to normal and atypical development. We hope as well that our coverage will prompt the next 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.

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Child and Adolescent Psychopathology

PART I

THE DEVELOPMENTAL PSYCHOPATHOLOGY APPROACH TO UNDERSTANDING MENTAL ILLNESS

CHAPTER 1

Developmental Psychopathology as a Scientific Discipline

A 21st-Century Perspective

STEPHEN P. HINSHAW

Information continues to accumulate, at an increasingly rapid pace, about the complex processes and mechanisms underlying the genesis and maintenance of child and adolescent forms of mental disorder. Our major goal for this, the third edition of *Child and Adolescent Psychopathology*—in chapters written by international experts on the topics of interest—is to present current information, particularly surrounding core vulnerabilities and risk factors for major dimensions and categories of behavioral and emotional problems of youth. As in our prior editions (Beauchaine & Hinshaw, 2008, 2013), we emphasize psychobiological vulnerabilities in the active context of environmental forces that shape development. Framed somewhat differently, an important objective for each chapter is to delineate potential *ontogenic* processes in progressions to mental disorder, signifying mechanisms underlying individual development, with the realization that multiple vulnerabilities and risk factors interact and transact in case-specific yet ultimately predictable ways (Beauchaine & Hinshaw, 2016; Beauchaine & McNulty, 2013; Hinshaw, 2015). Parallel to the first two editions, we do not prioritize assessment or treatment-related information in this book, given that such coverage would necessitate a second or even third volume (e.g., Mash & Barkley, 2006, 2007).

Although the book's title focuses on children and adolescents, I note immediately that psychopathology, in many (if not most) cases, unfolds across the entire lifespan. Most so-called adult manifestations of mental disorder have origins, if not outright symptom presentations, prior to age 18. Moreover, even the earliest-appearing forms of behavioral and emotional disturbance typically portend escalating symptoms and impairments that can persist for decades (e.g., Kessler, Berglund, Demler, Jin, & Walters, 2005). Because resilience is also a possibility (Luthar, 2006), lifespan

approaches to the topics of interest in this book are increasingly mandated for thorough understanding, carrying profound clinical as well as scientific implications. The child is the father of the man—and the mother of the woman—given that adults emerge from a cascading set of processes set in motion years before.

Before delving further, I immediately acknowledge the major debt that Ted Beauchaine and I owe to all of our contributors, as each is a major force in the scientific literature. We asked them to integrate state-of-the-art knowledge into the chapters that follow. Indeed, given the fast-escalating sophistication of mechanistic accounts of the development of psychopathology—which are now integrating genetic vulnerability and brain architecture in the presence of contextual forces across development, providing unprecedented levels of synthesis (Hinshaw, 2015)—no current compendium can afford to rest on the laurels of previous editions. The field's work is emerging at ever-more-detailed levels of analysis, with the promise of accounts that should, in the future, better inform evidence-based practice in the context of validated knowledge structures that can be applied to the clinical phenomena under consideration. In this initial chapter, I delineate the clinical and policy-related importance of the subject matter at hand, explicate core principles of developmental psychopathology (DP), and provide a general overview of the sequence of the chapters and their contents. In so doing I aim to set the stage for the cutting-edge advances and wisdom provided in the remainder of the volume.

RELEVANCE AND IMPORTANCE

The subject matter under consideration in this volume is at once clinically compelling and conceptually fascinating. Mental disorders yield substantial impairment, pain, and suffering for individuals, families, communities, and even cultures. The levels of personal and family tragedy involved are often devastating (Hinshaw, 2008a). At the same time, multifactorial vulnerabilities and risk factors—along with the complex, transactional developmental progressions that produce symptoms and impairments—challenge investigators from disciplines as diverse as neuroscience, genomics, public health, psychology, psychiatry, and public policy to emerge with new insights and syntheses. Overall, the clinical need is urgent and the scientific motivation compelling.

I begin with the concept of impairment. As elaborated in nearly every working guide to psychopathology (e.g., American Psychiatric Association, 2013; Wakefield, 1992), a designation of mental illness mandates, beyond behavior patterns or symptoms, that the individual in question display impairment or "harm" before a diagnosis is made. Clinically, then, attention must be paid to the often-excruciating pain and suffering attending to conditions as diverse as autism-spectrum disorders, various sequelae of maltreatment, severe attention deficits and impulsivity, interpersonal aggression, significant anxiety and mood disorders, thought disorders (including schizophrenia), eating-related conditions, self-destructive behavior patterns and personality configurations, and substance use disorders. Each is

linked to setback and suffering, societal reverberations, and significant costs, the latter measurable in terms of huge expenditures borne by society, not related just to treatment per se but to the long-range outcomes of interpersonal, educational, and vocational failure that often attend to mental disorders (for an example of the huge costs linked to attention-deficit/hyperactivity disorder [ADHD], see Hinshaw & Scheffler, 2014).

Of course, impairment and harm—whether personal or experienced by others—are not sufficient for designating individuals as suffering from a mental disorder. In the view of Wakefield (1992), both harm (which involves a value-laden component) and dysfunction (a scientific construct) are required before mental illness should be diagnosed. Per Wakefield, dysfunction is "the failure of a mental mechanism to perform a natural function for which it was designed by evolution" (p. 373). Although mental health fields lack the objective markers and pathognomonic signs[1] as those found in medicine and neurology (see Chapter 2 [Beauchaine & Klein]), our aim for the accumulated work in the present volume is to propel knowledge of dysfunctional mechanisms related to child and adolescent psychopathology. At the same time, findings from each chapter remind us that the origins of mental health conditions are reciprocal, dynamic, multilevel, and fully linked with processes linked to environmental context.

Not every aspect of psychopathology is necessarily impairing. At the level of evolution, it cannot be the case that mental disorder is inevitably or inexorably linked to personal failure or reduced fecundity; otherwise, how would conditions such as severe thought and mood disorders have perpetuated across human history (for evolutionary psychological explanations of mental disorder, see Neese, 2005)? Partial genetic loadings or vulnerabilities in biological relatives may well carry adaptive advantage; at least some aspects of symptoms could yield inspiration or thriving. Still, clinical and population-level facts regarding impairment linked to mental illness are stark. Emotional and behavioral problems among children and adolescents are distressingly prevalent and often lead to serious impairments in such crucial life domains as academic achievement, interpersonal competencies, and independent living skills (for thorough accounts, see Mash & Barkley, 2014). These conditions incur intensive pain for individuals, families, and communities at large, delimiting life opportunities and triggering major burdens for caregivers, school districts, and health care systems. In short, far too many young lives are compromised by mental illness.

Moreover, child and adolescent conditions and mental-health-related issues are growing in impact. As just one harrowing example, recent data from the World Health Organization reveal that, worldwide, the number-one cause of death for girls aged 15–19 years is now suicide (World Health Organization, 2014).

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1. A pathognomonic sign is an indicator, usually biological, that at once (1) proves that a person suffers from a disease of known etiology, and (2) eliminates all other disease processes as potential causes. For a detailed discussion of the role of pathognomonic signs in medicine vs. psychiatry/psychology, see Beauchaine and Thayer (2015).

Rates of self-injury have escalated rapidly over the past decades, and conditions like autism and ADHD are undergoing huge increases in diagnosed prevalence (e.g., Visser et al., 2014). The age of onset of serious mood disorders appears to be dropping, signaling the importance of contextual "push" in unearthing vulnerability (Hinshaw, 2009). In both the developing and developed world, serious mental disorder in youth portends major life consequences and even tragedy (see, for example, Sawyer et al., 2002).

Moving beyond childhood and adolescence per se, each year the Global Burden of Disease findings convey that a number of mental health conditions (along with neurological and substance use disorders) are among the world's most impairing illnesses (Whiteford, Ferrari, Degenhardt, Feigin, & Vos, 2015). Indeed, the variable called "years lived with disability" is dominated by individuals with mental disorders in our current era, on par with and often surpassing so-called physical diseases. By the time of adulthood, economic costs related to mental illness escalate with respect to employment-related impairments, yielding huge public-entitlement expenditures and lack of productivity. In short, from a number of important lenses, mental disorders are tragically impairing, robbing individuals of opportunities to thrive and be productive, often in the prime of their lives. If readers sense a call to action in these words, they have read my intentions precisely.

Crucially, mental health and physical health are inexplicably intertwined. It is now well known that serious mental disorder is associated with reductions in life expectancy averaging from 10 to 25 years (e.g., Chang et al., 2011). The reasons here are plentiful: high-risk lifestyles, lack of access to medical care, suicide, homicide, co-occurring chronic (e.g., cardiovascular disease; diabetes), and infectious (e.g., HIV) illnesses, and related unhealthy practices such as smoking and substance abuse. Even nonpsychotic disorders (e.g., ADHD; many forms of depression) are linked to long-term health risks (e.g., Barkley, Murphy, & Fischer, 2008). Recent findings reveal links between a range of mental disorders and a startling list of chronic physical illnesses (Scott et al., 2016).

Given this set of enormously costly, persistent, and deeply human consequences and needs, why not rely on traditional clinical efforts in psychology and psychiatry for solutions, given their long, venerable histories? As detailed in earlier accounts, however, these efforts have led to static views of psychopathology, with priority given to categorical diagnoses that inevitably lump together individuals with substantially different etiologic pathways into the same "condition" (e.g., Cicchetti, 1984, 1990). Moreover, the reciprocally deterministic nature of development, both typical and atypical, is not well captured by such static diagnostic systems (or nosologies, see Chapter 2 [Beauchaine & Klein]). Because 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 rapidity, providing a call to investigators from a host of seemingly disparate disciplines regarding the promise of uncovering relevant mechanisms. Absent the multifaceted nature of DP models and paradigms, traditional perspectives are too often sterile

and impoverished, carrying huge potential for treatments and prevention efforts to be directed at the wrong targets.

Despite scientific and clinical urgency surrounding this entire topic, barriers stand in the way of increased scientific understanding and access to evidence-based treatment. Perhaps the primary issue is that mental disturbance, at any age, remains highly stigmatized (e.g., Hinshaw, 2007; Hinshaw & Stier, 2008; Martinez & Hinshaw, 2016). Intensive stigma and shame—related to the unpredictability of the behavior patterns in question, the threat they convey to perceivers' well-being, and their media-propelled linkages to violence and incompetence—too often preclude help seeking, prevent empathic responses, and serve to render mental health a lower priority than physical health, despite inextricable linkages between the two. Depressingly, although public knowledge of mental illness has grown considerably since the 1950s, the U.S. public is far *more* likely to link mental illness with dangerousness than in the past (see Phelan, Link, Stueve, & Pescosolido, 2000). Moreover, rates of stigma and social distance related to mental illness have not changed appreciably in recent decades (Pescosolido et al., 2010). 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 mandates for involuntary commitment to hospitals—along with frequent media attention linking mental illness to mass shootings, oftentimes inaccurately; and (c) the tenuousness of 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 Haslam & Kvaale, 2015; Martinez, Piff, Mendoza-Denton, & Hinshaw, 2011; Pescosolido et al., 2010). Indeed, although biological perspectives are a necessary antidote to the "blaming the family" and "castigating the individual" perspectives that dominated psychology and psychiatry for much of the 20th century, their reductionistic promotion is neither accurate nor aiding the cause of stigma reduction, in part because they appear to promote pessimism and dehumanization. Instead, DP perspectives offer complex as opposed to simplistic or reductionistic conceptions of mental disorder, potentially leading to appreciation of the multidetermined biological and contextual factors related to psychopathology instead of personal or family weakness or blame, or notions of genetic flaw (e.g., Haslam & Kvaale, 2015; Martinez & Hinshaw, 2016).

In all, despite major advances in both basic science and clinical applications in recent years, as highlighted in the following chapters, the field's knowledge of developing brains and minds in multiple, interacting contexts is still rudimentary. It is hard to imagine otherwise, given the sheer complexity of the subject matter under consideration. As noted in introductory chapters to the earlier editions of this volume (Hinshaw, 2008b, 2013), the trajectory of human prenatal neural development is nothing short of staggering, with literally thousands of new neurons proliferating during each second of development after the first few weeks following conception, as well as massive pruning and synaptogenesis in the first several years of life. Still, for those who enjoy a challenge and are excited by questions that will take both many

decades and many great minds and scientific teams to answer—with the potential payoff of bettering the human condition—the hope is that this volume will serve as a call to join the major scientific and clinical efforts so urgently needed. 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 and forthcoming generations of scholars and clinicians need to join the effort.

At this point, I provide a review of core axioms and principles of DP. These points reflect the multidisciplinarity and transactional nature of the field, signifying that static models and unidimensional conceptions are simply not able to explain the fascinating and troubling development of maladaptive behavior patterns comprising the domain of psychopathology.

PRINCIPLES OF DP

Many of the conceptual bases for integrating developmental principles and models into the study of child and adolescent psychopathology have been present for several centuries, spanning diverse fields and disciplines (e.g., Cicchetti, 1990). Yet it is only in the past 40 years that DP has taken formal shape as a perspective on behavioral and emotional disturbance throughout the lifespan, and as a major conceptual guidepost for the study of both normal and atypical development. During this period, DP has exerted a major force on clinical child psychology, child psychiatry, developmental psychology, developmental neuroscience, and a number of other disciplines in both behavioral and neurological sciences. Not only have new courses been formed at major universities, but journals have been created and new paradigms of conceptualizing mental disorder have gained traction (Insel et al., 2010; see Chapter 2 [Beauchaine & Klein]). It is remarkable how pervasive the DP perspective has become, galvanizing a host of clinical and scientific efforts and in the process becoming mainstream.

DP simultaneously comprises a theoretical model regarding the origins of mental disorders, a multidisciplinary approach linking principles of normative development to the genesis and maintenance of psychopathology, and a scientific discipline closely tied to clinical child and adolescent psychology and psychiatry but transcending the usual diagnosis-based emphases of these fields (Cicchetti, 2016; Lewis & Randolph, 2014). Through its focus on the dynamic interplay of biology and context, genes and environments, and transactional processes linking multilevel influences to the development of healthy and atypical functioning, DP has come to dominate current conceptual models of psychopathology. Many of its core ideas emerge from disciplines such as philosophy, systems theory, and embryology (see Gottlieb & Willoughby, 2006, for elaboration). The syntheses represented in this volume, reflecting DP's continuing growth into the first two decades of the 21st century, are cutting-edge, given the major knowledge explosion in recent years, related largely to greater understanding of psychobiological influences as they transact with contextual forces.

What characterizes a truly developmental view of psychopathology, versus descriptive, symptom-focused presentations dominating most classification systems? DP's originators contended with this core question (e.g., Achenbach, 1974; Cicchetti, 1990; Rutter & Sroufe, 1984; Sroufe & Rutter, 2000), and current syntheses still grapple with the fundamental issues involved (Cicchetti, 2016; Lewis & Rudolph, 2014). From my perspective the key issues constitute multidisciplinarity; acknowledgment of dynamic, multilevel processes; and appreciation of systems-level change in producing developmental transitions (whether the systems are biological or social). Despite the many gains that have been made, it is important to realize at the same time how far we must still travel to comprehend the development and maintenance of psychopathology via the tools and models of DP. The trail ahead is long and steep.

I list several core points that 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) exploring both risk and protective factors and their interplay, so that competence, strength, and resilience as well as pathology and impairment can be understood; (d) 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 (e) capturing the importance of both psychobiological vulnerabilities and social/cultural context in understanding the function of behavioral and emotional patterns.

Three related principles bear emphasis:

1. Multiple pathways to pathology exist. Indeed, disparate routes may lead to behaviorally indistinguishable conditions or outcomes, exemplifying the construct of *equifinality*. For example, aggressive behavior can result from physical abuse, from a heritable tendency toward disinhibition, from injury to the frontal lobes, from coercive parenting interchanges with the developing child, from prenatal and perinatal risk factors acting in concert with early experiences of insecure attachment or parental rejection, or—as is probably most often the case—from different combinations of these vulnerabilities and risk factors. A key problem with static nosologies is their assumption that everyone receiving a similar psychiatric diagnosis has the "same" underlying patterns and processes of psychopathology. Similarly, *multifinality* pertains when a given vulnerability, risk factor, or initial state fans out into disparate outcomes across different individuals (Cicchetti & Rogosch, 1996). Maltreatment may or may not lead to severe maladaptation, depending on a host of intervening factors. As another example, extremes of inhibited temperament may induce intense shyness and social withdrawal; but other, healthier outcomes are also possible, depending on the presence or absence of additional risk or protective factors.

2. DP models often 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 unique developmental journeys across the lifespan (see Bergman, von Eye, & Magnusson, 2006). 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., Hinshaw, 2002; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001), to ensure that (a) results are applicable to subsets of participants grouped on moderator variable of interest (males versus females, those from different ethnic groups, or those with different patterns of comorbidity) and (b) underlying mechanisms of change, gleaned from mediator variables, are taken into account.
3. Given the rapid growth in recent years of genomic models as well as brain imaging methods, DP researchers in the 21st century must pay increasing attention to the role of the brain, and neuroscientific principles in general, toward accounting for the wide range of extant pathologies and their devastating effects. The field has come a long way from the middle of the 20th century, when biological and temperamental factors were virtually ignored in accounts of child development and psychopathology. Again, however, progress will be stalled if the psychosocial reductionism of prior generations is replaced by biological and genetic reductionism in the current era. A key antidote is for students and investigators to embrace a multiple-levels-of-analysis approach, integrating across genes and gene products, neural systems, and temperamental traits and core behavioral patterns, in contexts of families, schools, and neighborhoods, including the general culture (Cicchetti, 2008; Insel et al., 2010). Isolated, single-factor or single-level models and paradigms are inadequate to the task.

In other words, the greatest potential for progress in the DP field is made when investigators travel back and forth between "micro" and "macro" levels—including intermediate steps or pathways—to understand mechanisms that underlie development of adjustment and maladjustment. The essential task is to link events at the level of genes (e.g., genetic polymorphisms; transcription and translation), neurotransmitters, and neuroanatomical development, into individual differences in temperament, social cognition, and emotional response patterns. At the same time, such bottom-up conceptions must be supplemented by top-down understanding of ways in which family interaction patterns, peer relations, school factors, and neighborhood/community variables influence the developing, plastic brain, even at the level of gene expression (see Chapter 3 [Beauchaine, Gatzke-Kopp, & Gizer]). Overall, progress toward understanding pathological behavior will require

multidisciplinary efforts in which investigators ranging from geneticists and biochemists, scientists focusing on basic psychological processes and individual psychopathology, experts on family and neighborhood processes, examiners of clinical service systems, and public health officials as well as policy experts must work collaboratively and in increasingly diversified ways. The phenomena under consideration are too complex, too dynamic, and too multifaceted to be understood by an exclusive focus on psychobiological processes, family factors, peer processes, or cultural factors in isolation. Performing the necessary kinds of investigations often mandates large-scale, complex, and interdisciplinary work, necessitating collaborations across traditional disciplinary boundaries.

Note that key concepts and principles of DP have been stated and restated across a large number of articles, chapters, and books. Indeed, detailed discussion could easily fill a volume unto itself. The challenge for the current chapter is to encapsulate several core tenets, in the service of foreshadowing and illuminating content on specific processes and specific mental dimensions and disorders.

Normal and Atypical Development Are Mutually Informative

As opposed to the study of discrete, mutually exclusive categories of disorder, DP models emphasize that nearly all relevant phenomena represent aberrations in continua of normal developmental pathways and processes—and, accordingly, that without understanding typical development, the study of pathology will remain incomplete and decontextualized. As just one example, related to a research area within my own expertise, illuminating the nature of ADHD requires thorough understanding of normative development of attention, impulse control, and self-regulation (e.g., Barkley, 2015; Hinshaw & Scheffler, 2014; Nigg, Hinshaw, & Huang-Pollack, 2006; Sonuga-Barke, Bitsakou, & Thompson, 2010; see also Chapter 13 [Nigg]). Similarly, investigations of autism must account for the development of interpersonal awareness and empathy, as well as social motivation—which typically takes place over the first several years of life—to gain understanding of the devastating consequences of failure to attain such development (Dawson & Toth, 2006; see also Chapter 22 [Faja & Dawson]). Additional examples exist across all forms of disordered emotion and behavior. Although considered set breaking at the outset of modern DP conceptions, this point is now taken for granted: Few would doubt the wisdom of understanding developmental sequences and processes associated with healthy outcomes as extremely relevant to elucidation of pathology.

Intriguingly, however, the process is conceptualized as bidirectional, as investigations of pathological conditions—sometimes referred to as *adaptational failures* in the language of DP (Sroufe, 1997)—can and should provide a unique perspective on normative development. Thus, the study of disrupted developmental progressions can and should facilitate our understanding of what is normative. This core tenet of DP, of mutual interplay between normality and pathology, is now

espoused widely. Examples abound in neurology, where the study of disrupted neural systems enhances understanding of healthy brain functioning (Gazzaniga, Ivry, & Mangun, 2014).

But just how appropriate is this perspective for DP? Outside of neurological formulations, where single lesions or single genes are investigated quite specifically, can studies of psychopathology inform normal development? It is commonly accepted that greater knowledge of basic emotion, cognition, attention, memory, social awareness, self-regulation, and so forth feeds into understanding of pathology. Indeed, almost no forms of mental disorder constitute clearly demarcated, qualitatively distinct categories or taxa, so processes applying to individuals near the peak of the bell curve are likely to apply to those further out on the continuum. Yet regarding the other direction—the informing of normal-range processes from study of the abnormal—we can legitimately ask what has been learned from far more complex developmental processes linked to mental disorder as regards application to normative development. In other words, in the absence of surgical lesions in certain brain tracts or single-gene forms of pathology such as phenylketonuria, can the far messier domain of psychopathology cycle back to inform developmental science?

Examples are becoming more apparent. The horrific experiments of nature that occurred when infants and toddlers in Eastern Europe were subjected to harsh, sterile institutionalization in large orphanages several decades ago, which included a bare minimum of human contact, provide important data (see O'Connor, 2006, for review). From accumulated research evidence, it is now clear that the more months—during infancy, toddlerhood, and the preschool years—a child is exposed to such conditions, the worse his or her developmental outcomes, both cognitively and socially. In short, the longer the periods of deprivation, the lower the chances for recovery. Intriguingly, the most common behavioral outcomes related to such early deprivation include inattention and overactivity, rather than conduct problems per se—a clear example of equifinality, given that heritable risk is the strongest contributor to such problems in more normative samples (see Kennedy et al., 2016; Kreppner et al., 2001).

Moreover, assignment to foster care can mitigate such developmental risk, if performed during the second or third year of life (Nelson et al., 2008). Indeed, for previously institutionalized girls, random assignment to foster care, compared to continued institutionalization, led to improvements in internalizing behavior patterns, mediated by the gaining of attachment security via change from institutional care to family placements (McLaughlin, Zeanah, Fox, & Nelson, 2012). Thus, even in a harshly abandoned and deprived sample, attachment processes were implicated in reductions of anxiety and depression. Whereas mediators of competence in more normative samples are still open to exploration, the extent of social and cognitive "catch-up" following removal from harsh institutional care is potentially informative about normal-range development of secure relationships and cognitive performance.

As reviewed in introductory chapters of previous editions of this volume (Hinshaw, 2008b, 2013), further examples exist from the domains of ADHD and autism-spectrum disorders. For the former, information about disruptions to inhibitory control and reward-related mechanisms from individuals with clinical levels of the relevant symptoms informs developmental science about normative development of self-regulation and intrinsic motivation. Regarding autism, intensive investigation of social deficits has relevance to understanding normative development of "theory of mind" during the toddler and preschool years. Other examples abound outside the realm of neurodevelopmental disorders, in the areas of depression, anxiety, and response to trauma. Certainly, symptoms and systems at play in all such domains are more complex than in classic cases from neurology, but two-way communication between the atypical and typical is possible.

If our text had a "post-chapter quiz"—or suggestions for extra credit for readers and students—I would suggest there be mandated exploration, when examining relevant literature and pertinent clinical cases, of specific ways in which knowledge of pathological patterns can inform normative development. My guess is that this task could be an eye-opener for everyone involved.

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. Taking the specific example of externalizing and antisocial behavior, it is well known from a number of longitudinal investigations 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, rank order remains relatively preserved, such that the most aggressive individuals at early points in development remain highly aggressive, compared to others, across development. But does this well-replicated finding mean that the precise forms of externalizing, antisocial behavior remain constant? Clearly not, given that children who exhibit extreme temper tantrums and defiance during toddlerhood and preschool years are not especially likely to exhibit high rates of tantrums during adolescence. Rather, they have a strong likelihood of displaying early verbal aggression and then beyond-normative physical aggression in grade school, excessive covert antisocial behaviors in preadolescence, and high rates of delinquency by their teen years, followed by adult manifestations of antisocial behavior after adolescence, including partner abuse (e.g., Moffitt, 2006). In short, continuities exist, but these are *heterotypic* in nature, as the actual form of the underlying antisocial trait changes form with development. The implications are profound.

That is, investigators of continuity of psychopathology must take into account developmental progressions. Continuity may not be linear or static: During development, new life opportunities and brain maturation portend ascension of new

forms of pathological behavior. Predictability may well exist, but in complex and nonlinear fashion (see Hinshaw et al., 2012; Meza, Owens, & Hinshaw, 2016; and Swanson, Owens, & Hinshaw, 2014, for the example of emerging self-harm as girls with ADHD grow into their adult years).

Another important consideration is that patterns of continuity may differ considerably across separable subgroups with 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 noted in the paragraph above, the specific forms of the antisocial actions change across development. Yet not all early starters persist. In addition, a large subset of youth do not display major externalizing problems in childhood but instead shows a sharp increase with adolescence (Moffitt, 2006). Understanding such continuities and discontinuities via relatively homogeneous subgroups is likely to yield greater understanding than plots of overall curves or "growth." Sophisticated statistical strategies (for example, growth mixture modeling) are increasingly used to aid and abet this search for separable trajectories or classes defined on patterns of change of the relevant dependent variable (Muthén et al., 2002). In all, continuities abound across the course of development, but developmental associations of interest are not often simple or simplistic. The kinds of developmental perspectives emphasized in DP, and in this text, mandate examination of life trajectories, interactive and transactional processes, and multiple-levels-of-analysis perspectives. Without their consideration, relevant models are once again destined to oversimplification and a loss of relevant clinical information.

Risk and Protective Factors

A key focus of a discipline such as DP—with the term *psychopathology* embedded in its title—is to discover the nature of behavioral and emotional problems, syndromes, and disorders. Many different definitional schemes have been invoked to define and explain psychopathological functioning, with none able to provide a complete picture. Indeed, it is clear that biological vulnerabilities, psychological processes, environmental potentiators, and cultural-level norms and expectations all play major roles in defining and understanding behavioral manifestations that are considered abnormal and pathological in a particular social context.

Both biological vulnerabilities and environmental risk factors are antecedent variables that predict such dysfunction, and the ultimate goal is to discover which variables are both malleable and potentially causal of the disorder in question (Kraemer et al., 1997; see also Kraemer et al., 2001). Yet disordered behavior is not uniform, so vulnerabilities and risk factors are not inevitable predictors. Indeed, for most individuals with diagnosable forms of psychopathology, symptoms

and impairments wax and wane over time. It is often difficult to know when dysfunction precisely begins; it is also quite normative for periods of serious problems to be followed by healthier adjustment. In fact, the myth that mental disturbance is uniformly debilitating, handicapping, and permanent is a key reason for the continuing stigmatization of mental illness (Hinshaw, 2007; Hinshaw & Cicchetti, 2000). Crucially, not all individuals who experience vulnerabilities and risk factors for disorder develop subsequent pathology.

Resilience is the term used to define unexpectedly good outcomes, or competence, despite the presence of adversity or risk (Luthar, 2006; Masten & Cicchetti, 2016). Indeed, the concept of multifinality, noted previously, directly implies that, depending on a host of biological, environmental, and contextual factors, variegated outcomes may well emanate from common risk factors, with the distinct possibility of resilience and positive adaptation in some cases.

DP is therefore involved centrally in the search for what have been called *protective factors*: variables and processes that mitigate vulnerability/risk and promote more successful outcomes than would be expected in their presence. Controversy surrounds the construct of resilience, the nature of protective factors, and the definitions of competent functioning (see Burt, Coatsworth, & Masten, 2016). Some have claimed that there is no need to invoke a set of special, mysterious processes that are involved in resilience, given that a certain percentage of any sample exposed to a risk factor will show better-than-expected outcomes and that protective factors are all too often simply the opposite poles of what we typically think of as risk variables or vulnerabilities (e.g., higher rather than lower IQ; easier rather than more difficult temperament; warm and structured rather than cold and lax parenting). Still, it is crucial to examine processes that may be involved in promoting competence and strength rather than disability and despair, given that such processes may be harnessed for prevention efforts and may provide key conceptual leads toward the understanding of both pathology and competence.

In short, gaining understanding of why some children who are born into poverty fare well in adolescence and adulthood (see, for example, Wadsworth, Evans, Grant, Carter, & Duffy, 2016), why some individuals with alleles that tend to confer risk for pathological outcomes do not evidence psychopathology, why some youth with difficult temperamental features develop into highly competent adults, and why some people who lack secure attachments or enriching environments during their early years nonetheless show academic and social competence is essential for knowledge of both health and maladjustment. It is not just a luxury but a necessity to investigate positive developmental outcomes, given the inseparability of health and pathology. Competence can shed light on the pathways that deflect from pathology and, in so doing, may provide otherwise hidden insights into necessary developmental components of adjustment versus maladjustment (Luthar, 2006; Masten & Cicchetti, 2016).

Reciprocal, Transactional, Ontogenic Process Models

Linear models of causation, for which static psychological or psychobiological variables are assumed to respond in invariant ways to the influence of vulnerabilities and risk factors, are not adequate to the task of explaining psychopathology and its development. Richters (1997) provided detailed explication, highlighting that unique explanatory systems are needed to deal with "open systems" such as human beings. Pathways to adolescent and adult functioning are marked by reciprocal patterns or chains, in which children influence parents, teachers, and peers, who in turn shape the further development of the child (for an early, influential model, see Bell, 1968). Such mutually interactive processes propel themselves over time, leading to what are termed *transactional models*. Some developmental processes appear to operate via cascading, escalating chains (Masten et al., 2006) or even "symphonic" effects (Boyce, 2006). Indeed, nonlinear, dynamic systems models are needed to explicate core developmental phenomena (Granic & Hollenstein, 2006). Sensitive data-analytic strategies and innovative research designs are crucial tools for fostering greater understanding of such phenomena.

These kinds of models can be used to elucidate equifinal and multifinal processes, as described above. They also exemplify, once again, problems inherent in static, categorical models of pathology (e.g., American Psychiatric Association, 2013; see Chapter 2 [Beauchaine & Klein]). Recognition of such problems led the leadership of the National Institute of Mental Health to develop, several years ago, an alternative to categorical diagnosis, via an endeavor called the Research Domain Criteria (RDoC; see Insel et al., 2010). This dimensional means of accounting for psychopathology specifically embodies a multiple-levels-of-analysis approach by positing a number of core, dimensional behavioral systems, with clear biological substrates, shaped by context.

At the same time, ontogenic process models of psychopathology have witnessed a resurgence (see Beauchaine & Hinshaw, 2016; Beauchaine & McNulty, 2013), whereby heritable vulnerabilities transact with toxic contextual forces (e.g., coercive family interactions; violent neighborhoods) to yield psychopathology, particularly of the externalizing variety. Self-injury appears to fall in the same domain of relevant processes (see Chapter 19 [Crowell]). In all instances, static and/or linear models of influence must give way to reciprocal and transactional chains of influence.

Psychobiological Discoveries Intersect and Interact With Context

The genomic era has been upon us for some time, and advances in brain imaging research—despite criticisms of its methods and false-positive rates (Vul, Harris, Winkielman, & Pashler, 2009)—have made the developing brain far more accessible to scientific view than ever before. Although it is mistaken, as emphasized throughout, to give primacy to any single level of analysis (brain, context, or other), we have asked contributors to pay particular attention to psychobiological

factors and processes. Part of the reason is historical: Family systemic and environmental views dominated the field for much of the 20th century. Also, we now know that without understanding potential effects of genes, physiological processes, and biological vulnerabilities to psychopathology, there is little hope of understanding the most severe forms of disorder. Yet the brain is remarkably plastic and contexts influence biological unfolding. Thus, Ted Beauchaine and I have asked authors to emphasize contextualization of the psychobiological perspectives they present. In fact, reductionistic accounts of (a) the primacy of single genes, (b) the inevitable predictability of later functioning from early temperament, or (c) the placement of psychopathology completely inside brightly colored brain images are as short-sighted as the exclusively environmental accounts of psychopathology that dominated much of the 20th century.

Indeed, a key tenet of DP is that family, school-related, neighborhood, and wider cultural contexts are central for the unfolding of aberrant as well as adaptive behavior. This point cannot be overemphasized. What may have been adaptive, genetically mediated benefits at one point in human evolutionary history may be maladaptive in current times, given major environmental and cultural changes that render certain traits far less advantageous than previously (e.g., storage of fat in times of uncertain meals and sudden need for survival-related activity; presence of undue anxiety in relation to certain feared stimuli when conditions have markedly changed with respect to sedentary lifestyles). There are few absolutes in terms of behavior patterns that are inherently maladaptive or risk factors that inevitably yield dysfunction; cultural setting and context are all-important for understanding and creating healthy versus unhealthy adaptation.

Similarly, key environmental factors (such as parenting styles) are not always uniformly positive or uniformly negative in terms of their developmental effects. Deater-Deckard and Dodge (1997) showed that authoritarian parenting predicts antisocial behavior among White, middle-class children but not necessarily among African-American families. At the same time, many forms of mental disorder are present at roughly equivalent rates across multiple cultures, revealing key evidence for universality. Yet effects of risk or protective factors often differ markedly depending on developmental timing, family and social contexts, and niches that exist in given cultures for their expression and resolution (Serafica & Vargas, 2006). In short, the DP perspective tells us clearly that setting and context are all-important (see also Rutter et al., 1997).

The area of gene × environment interactions in DP provides an important, if contentious, case example. The underlying idea is that genotypes moderate the effects of environmental context on the development of psychopathology, and vice versa (i.e., environmental factors moderate genetic effects on mental disorder). With profound implications for DP, this subfield erupted, 15 years ago, with core publications by Caspi and colleagues (Caspi et al., 2002, 2003).

However, such widely cited findings have been subject to meta-analyses, which initially challenged the robustness of such results regarding interactions of the

serotonin transporter gene with maltreatment or stressful life events (e.g., Risch et al. 2009) and then subsequently upheld the initial results when all relevant investigations were included (Karg, Burmeister, Shedden, & Sen, 2011). Both statistical power and selection biases are major factors in all such investigations.

In a commentary, Caspi, Hariri, Holmes, Uher, and Moffitt (2010) made the point that interactive effects are accentuated in smaller-sample investigations that feature viable measures of environmental stress—highlighting the importance of precise measures of both the genetic (Dick et al., 2015) and the contextual side of the equation. Similar but greatly expanded perspectives have been provided by Dick et al. (2015), who outline essential recipes for avoiding the major issue of false positive findings in research on gene × environment interactions; and by Keller (2014), who adds to the cautionary note that many gene × environment researchers will overestimate such interactive power lest they explicitly take into account potentially confounding effects of passive gene-environment correlation. Furthermore, Bakermans-Kranenburg and van IJzendoorn (2015), Belsky and Pluess (2009), and Ellis, Boyce, Belsky, Bakermans-Kranenburg, and van IJzendoorn (2011) argue that some "vulnerability" genes are actually "susceptibility" genes, exquisitely responsive to either extremely good or poor environments—with the latter contentions also challenged by a range of artifacts that can produce false-positive findings.

In fact, the potential confounding of genetic and environmental contributions to behavior through gene-environment correlation is unquestioned, which is why contributions such as Harold et al. (2013)—who demonstrated reciprocal and transactional effects of child ADHD symptoms and negative parenting with respect to continuations of child behavior in adoptive samples, in which parents and children are biologically unrelated—are essential from a DP perspective. The bottom line is that increasingly sophisticated investigations, with careful attention paid to selection of genes, selection of environments, and careful consideration of a host of design and statistical issues, are needed to elucidate and validate specific ways in which genetic variation may be accentuated or unleashed in particular environmental contexts.

In cutting-edge research on DP, the *Journal of Abnormal Psychology* recently published a special section of articles on ontogenic process models in the field, with special emphasis on investigations focused on the integration of (a) gene-environment interplay, (b) neuroimaging correlates, and (c) contextual factors that may elicit pathological outcomes across development. I was asked to provide a commentary on these articles, and in doing so I noted that in many ways they represent the cutting edge of the field, largely related to such integration (Hinshaw, 2015). Commenting on only a subset (see also Hankin et al., 2015; LeMoult et al., 2015; Little et al., 2015; and Vrshek-Schallhorn et al., 2015), I first highlight that Carey et al. (2015) revealed an endocannibanoid polymorphism that interacted with childhood sexual abuse to predict development of cannabis dependence in adolescence. Upping the level of complexity and biological relevance, in one of their samples they also studied basolateral amygdala habituation.

This investigation added a dynamic neural measure to the usual Gene × Environment interaction paradigm, with findings suggestive of a plausible biological pathway leading to cannabis dependence symptoms.

Moreover, Pagliaccio et al. (2015) examined early life stress and genetic risk—indexed by a composite score of 10 polymorphisms in hypothalamic-pituitary-adrenal axis genes (see Nikolova, Ferrell, Manuck, & Hariri, 2011, for information on the amalgamation of "risky" alleles in polygenic risk indices), in relation to both (a) amygdala-related connectivity with other brain regions and (b) downstream anxiety symptoms and emotion regulation skills. Evidence was found for both moderation (of early stress by genetic vulnerability) related to low connectivity, and mediation (whereby such reduced connectivity was linked to poor emotion regulation).

In addition, Chhangur et al. (2015) examined interactions of two dopamine receptor alleles with core aspects of parenting (high control, low support) to predict adolescent delinquency, using five waves of adolescent data. One genetic variant (DRD2), in interaction with low parental support, showed the expected interaction. Intriguingly, the shape of the interaction was curvilinear, such that the combination of the DRD2 allele in question (A2A2) with low parental support was associated with quick increases in delinquency across early to mid-adolescence, followed by sharp decreases by late adolescence. It may be the case that different configurations of genes and family environments are needed to explain the pernicious group of youth with persistent antisocial behavior patterns (see Gizer, Otto, & Ellingson, 2016). Finally, as highlighted above with respect to gene × environment research in general, most such investigations are seriously underpowered, so only replication can reveal strong evidence for interactive effects (Dick et al., 2015).

Throughout this special section of articles, it was openly admitted by authors that interactive effects are typically of small size regarding typical effect-size metrics. It is noteworthy that Chhangur et al. (2015) were diligent in following the strong advice of Keller (2014) to adjust for potential gene-environment correlations before claiming significant effects of Gene × Environment interactions.

In all, the possibility that genetically induced variation in vulnerability to psychopathology is moderated by stressful or downright harmful environmental factors—and conversely, that contextual influences on key outcomes are moderated by genotype—remains a tantalizing and theoretically fascinating possibility, with considerable supportive research evidence amidst a sea of controversy about the entire endeavor (e.g., Bakermans-Kranenburg & van IJzendoorn, 2015; Dick et al., 2015; Keller, 2014). This example of the intersection of biology and context is emblematic of the promise—and problems—of the field in the second decade of the 21st century.

In sum, recent investigations in the field are explicitly tying in gene-environment interplay with (a) sensitive measures of brain function and (b) randomized clinical trials (Bakermans-Kranenburg & van Ijzendoorn, 2015), in the attempt to elucidate developmental pathways to psychopathology of various forms. The progenitors of

DP would probably not, a generation and more ago, have envisioned the extent to which technological advances and conceptual sophistication have propelled the gene-environment field along the lines of core DP axioms and principles, nor the wholesale questioning of the endeavor.

SUMMARY

Each of the previous points converges on the core theme that the development of psychopathological functioning is multidetermined, complex, interactive, transactional, and in many instances nonlinear. For those who like problems and solutions wrapped in neat packages, the study of DP will undoubtedly be a frustrating if not unfathomable endeavor. On the other hand, for those who are intrigued by the diverse clinical presentations of various pathological conditions in childhood and adolescence; for those who are fascinated with how much remains to be learned about antecedent conditions and maintaining factors; for those who are possessed by an intense "need to know" about underlying mechanisms of child and adolescent forms of mental illness; and for those who realize the need to consider healthy outcomes and competence as well as maladaptation, the DP perspective is a necessary guide to and framework for the rapidly growing scientific enterprise linking normal and atypical development.

Longitudinal, multilevel investigations are typically mandated to gain the types of knowledge needed to understand psychopathology (and competence) from a developmental perspective, with potentially high yield for basic developmental science; for elucidation of highly impairing behavioral, emotional, and developmental conditions; and for informing prevention and intervention efforts. The study of DP is ever expanding, engaging scientists from multiple disciplines and perspectives. Progress is emerging quickly, but the territory to explore remains vast.

CHAPTER CONTENTS

In our instructions to the volume's contributors, we asked for up-to-date material that is simultaneously developmentally based, clinically relevant, and directly inclusive of the types of psychobiological formulations gaining ascendancy in the mental-health enterprise. In other words, our aim for each chapter was presentation of state-of-the-art, DP-laden information, full of complexity but presented in a manner facilitating comprehension and integration.

Specifically, for chapters dealing with particular disorders and dimensions of psychopathology, we requested coverage of historical context, epidemiology, diagnostic issues, sex differences, etiology (including psychobiological *and* contextual factors, as well as RDoC considerations when possible), developmental processes, cultural variables, and synthetic comments to illuminate the pathology under discussion. We clarified that emphasis on neural and neurophysiological processes must not be reductionistic. Indeed, psychosocial and family factors—which served

as the predominant modality throughout much of the past century—interact and transact with biological vulnerabilities to produce both maladaptation and healthy adaptation throughout development (Beauchaine & Hinshaw, 2016; Beauchaine & McNulty, 2013). There is no escaping the need for integrative and integrated models as the field moves forward.

Thus, we asked contributors to consider multilevel models and transactional processes. Indeed, as noted above, 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., Belsky & Pluess, 2009; Dodge & Rutter, 2011; Hyde, 2015). Given page limitations and our desire for focused rather than exhaustive coverage, each chapter is relatively brief. Our goal is that readers can use these contributions as a springboard for additional exploration of conceptual frameworks, empirical research on mechanisms of interest, and building blocks for a new generation of evidence-based prevention and treatment efforts.

As can be seen, the early chapters pertain to core conceptual and developmental issues and factors, and later chapters cover specific dimensions and disorders of interest.

Immediately following this introductory chapter, Theodore Beauchaine and Daniel Klein (Chapter 2) provide crucial material spanning categorical (i.e., DSM) empirically based (e.g., the Child Behavior Checklist; Achenbach, 2009), and continuous (i.e., RDoC) methods and models for conceptualizing psychopathology. Certainly, dimensional/continuous accounts are gaining traction, yet at the same time clinical needs call for categorical diagnoses. Integrating these overarching frameworks is therefore necessary. The material in this chapter provides needed context for each of the remaining entries.

Next, in Chapter 3 Beauchaine, Lisa Gatzke-Kopp, and Ian Gizer discuss crucial concepts related to gene-environment interplay in the genesis of psychopathology. This chapter exemplifies what is now a truism: genes and environments must not be viewed as separable, independent factors influencing mental disorders, as their effects are tightly intertwined in reciprocal and transactional fashion. In keeping with current trends in DP, this chapter conveys core material from both behavioral genetic and molecular genetic perspectives and discusses rapidly evolving research on epigenetic processes through which environmental experiences alter DNA expression, with possible implications for psychological adjustment. It does not shy away from either promise or controversy regarding this endeavor.

Bruce Compas, Meredith Gruhn, and Alexandra Bettis (Chapter 4) present essential material on risk and resilience, providing a needed set of concepts and principles related to the potential for better-than-expected outcomes for subsets of vulnerable and high-risk youth. We must remember that not all children who express biological vulnerabilities and/or grow up with exposure to environmental risk develop pathological outcomes; indeed, one of the core DP principles noted above pertains to multifinal outcomes resulting from adverse early experiences. This chapter challenges conceptions of inevitable pathology from early vulnerability and risk.

In Chapter 5, Sara Jaffee covers the crucial area of child maltreatment, providing needed integration of psychosocial and psychobiological mechanisms through which maltreatment confers risk for a wide range of pathological outcomes. This chapter is a paragon of integrated and integrative perspectives on this prevalent and potentially devastating set of risk factors; compared to earlier formulations on maltreatment, her coverage of biological processes shows an explosion of growth in this arena.

Chapter 6, written by Emily Neuhaus and Theodore Beauchaine, covers impulsivity and vulnerability to psychopathology, viewing impulse-control problems as an underlying dimension that confers vulnerability to a range of mental disorders. Such risk is "expressed," however, in the context of often-toxic environments, whether in the form of maladaptive parenting, less-than-responsive schools, or violent neighborhoods. In other words, transactional models, spanning biological vulnerability and environmental risk, are necessary for considerations of the development of psychopathology, particularly for the next generation of ontogenic process models in the DP field.

Chapter 7, written by Jerome Kagan, deals with the temperamental construct of behavioral inhibition, emphasizing its predictive power for pathological outcomes in some but not all cases. Written with flair, it provides both historical and current perspectives on links between temperament and environment.

In Chapter 8, Bruce Ellis, Marco Del Giudice, and Elizabeth Shirtcliff cover the highly relevant constructs of allostasis and biological sensitivity to context, topics that are receiving increasing coverage in the research literature each year. Notable here are both the complexity of the relevant biological mechanisms involved and the inherent interplay between genes, biological substrates, and environmental inputs intricately involved in these phenomena. They contrast their adaptive calibration model to the earlier construct of allostatic load per se, arguing for the greater predictive and explanatory power of adaptive calibration.

Chapter 9, written by Lauren Doyle, Nicole Crocker, Susanna Fryer, and Sarah Mattson, covers the important area of exposure to teratogens (chemicals ingested by pregnant mothers) that confer risk for physical malformations as well as behavioral and emotional sequelae for the child, once born. As all students of pharmacology know, the placenta provides a completely permeable border for any and all drugs ingested by the mother, and the fetus's organs for metabolizing foreign substances are slow to develop—potentially providing for a host of teratogenic exposures. Consequences for developmental psychopathology are profound.

Next, in Chapter 10, Peter Arnett, Jessica Meyer, Victoria Merritt, Lisa Gatzke-Kopp, and Katherine Shannon Bowen write about brain injury as a risk factor for psychopathology. The multiple ways in which the developing brain can receive insults—and the complex pathways through which such injury affects development—are staggering. This chapter provides information about which many readers will have relative unfamiliarity; we are glad to have included these essential perspectives in our third edition.

Immediately following is Chapter 11 by Pamela Cole, Sarah Hall, and Nastassia Hajal on the still-growing topic of emotion regulation and dysregulation. Clearly, this chapter moves "up" a level from Chapters 9 and 10 in terms of levels of analysis, as the former chapters are heavily biological. Indeed, the ways in which intraindividual vulnerability and contextual risk shape individuals' abilities to recognize, process, and act on emotions (their own and those of others) are fascinating and of real importance to psychopathology.

Finally, rounding out the early "conceptual" chapters, in Chapter 12 Wesley Jennings and Nicholas Perez move up another level again, considering effects of neighborhoods on psychopathology, particularly externalizing behaviors. As in each of the other chapters, transactional processes are highly salient, as this analysis clarifies ways in which systems-level influences represented by neighborhood-level effects interact with individual vulnerabilities and risk factors to shape the most pronounced cases of antisocial behavior.

Beginning the section of chapters on disorders and dimensions of salience to psychopathology, Joel Nigg (Chapter 13) presents an elegant, integrative view on the development of attention-related and impulse-control problems (categorized as ADHD). Despite the strongly heritable nature of such symptoms, other biological-level influences as well as contextual processes are central to their developmental unfolding, as portrayed in this state-of-the-art chapter.

Then, in Chapter 14, Benjamin Lahey and Irwin Waldman present, in a parallel framework, interconnected processes related to development of aggression and antisocial behavior—which are tremendously costly to property, lives, and the economy as a whole. Once again, multiple levels of analysis and transactional processes are on center stage in this synthetic chapter, which features intensive discussion of important subfacets of externalizing behavior patterns.

In Chapter 15, Sandra Brown, Kristin Tomlinson, and Jennifer Winward discuss the topic of substance use disorders in adolescence and beyond. Because the major impairments—physical, emotional, economic—linked to substance abuse are legion, this chapter will be of interest to readers from multiple disciplines and perspectives. In addition to elucidating developmental pathways and mechanisms, the chapter authors also feature biological effects of substances on the developing brain, a vital issue not often sufficiently emphasized.

Next, Carl Weems and Wendy Silverman use Chapter 16 to convey essential, developmentally relevant information on anxiety disorders, which are prevalent and frequently devastating in the impairments they "carry." As the field moves from a multiple-categories conception of anxiety conditions, embodied by the DSM approach, to more current formulations informed by developmental psychopathology and transactional models, this chapter provides essential reading.

Chapter 17, by Emily Ricketts, Deepika Bose, and John Piacentini, covers obsessive-compulsive conditions and disorders, including OCD, body dysmorphic disorder, hair-pulling disorder, hoarding disorder, and skin-picking disorder. As noted by their placement in a separate chapter, these conditions reveal different

developmental processes and pathways from other anxiety-related disorders. Biological and environmental mechanisms underlying symptom display are emphasized.

In Chapter 18, authored by Daniel Klein, Brandon Goldstein, and Megan Finsaas, the subject matter is the highly prevalent and severely impairing spectrum of depressive disorders. The evolving picture of biological vulnerability and psychosocial risk related to depression in youth—operating transactionally and in equifinal fashion—provides fertile testing ground for many core tenets of DP. Indeed, the chapter features the heterotypically continuous manifestations of depressive disorders across the lifespan, shaped by biological vulnerability and contextual risk.

Erin Kaufman, Sheila Crowell, and Mark Lenzenweger (Chapter 19) write about the related but partially independent topics of borderline personality configurations and self-injury. In intriguing ways, these areas signify the confluence of internalizing and externalizing tendencies in the same youth; massive increases in rates of self-harm, along with its undoubted psychobiological and psychosocial roots, make this chapter another fulcrum point for a large number of DP principles and processes.

Chapter 20 features the contentious and clinically important topic of trauma-related disorders, authored by Bruce Perry. Here again is an area in which genetic vulnerabilities are accentuated in the face of traumatic life events—and in which long-term consequences of trauma are experienced in both biological systems and a range of psychological and emotional symptoms.

Then, in Chapter 21, Joseph Blader, Donna Roybal, Colin Sauder, and Gabrielle Carlson take on the controversial topic of bipolar-spectrum disorders, which continue to be a source of contention in the field (i.e., does bipolar illness exist in children—and if so, what forms does it take)? Issues of heritability along with psychosocial stressors, and of "kindling" across the lifespan—such that episodes potentially become more self-generating and frequent over time—are salient in this chapter.

Chapter 22 authored by Susan Faja and Geraldine Dawson, features the crucial topic of autism spectrum disorders. The fast rise in diagnosed prevalence, the serious impairments accruing from the symptoms, the early age of onset in most cases, and the controversies over effective intervention strategies render many issues in this area contentious—and of major clinical and scientific importance. The biological explosion of knowledge about this area is featured in this chapter.

Robert Asarnow and Jennifer Forsyth, in Chapter 23, deal with the low-prevalence but clinically and scientifically fascinating area of schizophrenia spectrum disorders in children and adolescents, long a source of diagnostic controversy. Their formulations, steeped in psychobiological vulnerability in transaction with stressful family environments, provide an authoritative account, revealing the importance of this topic for modern conceptions of early-onset schizophrenia.

Finally, Chapter 24, authored by Eric Stice and Deanna Linville, takes on the area of eating disorders. In writing about an area associated with intensive pain

for individuals and family members alike, the authors add binge eating disorder to the traditional syndromes of anorexia nervosa and bulimia nervosa for this current synthesis.

In sum, each chapter features complex, interactive processes spanning psychobiological vulnerabilities and psychosocial risk factors, while providing strong emphasis on a developmental neuroscience perspective.

Overall, the study of atypical development is fascinating, complex, and clinically as well as scientifically essential. It carries major potential for elucidating processes through which normal development occurs, at the same time that it highlights both expected and unexpected pathways to potentially devastating behavioral and emotional outcomes. As the 21st century continues its lightning-fast progressions into multilevel, integrative models of risk and resilience (and of health and pathology), it is heuristic to consider, simultaneously, the major progress made each year in the field along with the fundamental ignorance the field still possesses of the relevant variables, principles, and pathways linked to impairing mental disorders. We hope that you, the readers, are enticed by the clinical and scholarly puzzles that remain to be solved as well as humbled by the huge clinical need that remains in place for every single child, adolescent, family, and community experiencing the isolation, pain, and impairment related to mental disorder. The best minds of the next generations of scientists, clinicians, and policy makers need to become deeply engaged in the long journey that remains in front of us.

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CHAPTER 2

Classifying Psychopathology

The *DSM*, Empirically Based Taxonomies, and the Research Domain Criteria

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All scientific disciplines have rules for classifying phenomena and events that fall within their purview. Chemistry, for example, among the more advanced physical sciences, has fundamental laws that describe what constitutes an element (i.e., the number of protons in an atomic nucleus), what gives rise to similarities among elements (e.g., common bonding properties), how elements differ from one another (e.g., solubility vs. inertness), and how elements interact across levels of analysis to create what might otherwise be inexplicable phenomena (e.g., the high boiling point of water conferred by hydrogen bonds). For chemistry, these and other properties are summarized in the periodic table, which represents a taxonomy of elements. Although issues of taxonomy in chemistry are far more complex than this brief description implies, the example illustrates how important precise classification is in any discipline. Accurate classification ultimately leads to better prediction and control of external events, which are primary objectives of science (Braithwaite, 1953; see also Beauchaine, Gatzke-Kopp, & Mead, 2007). In chemistry, control of chemical reactions and molecular compounds has led to astounding advances in processes such as water purification, improving quality of life for millions. As outlined in Chapter 1 [Hinshaw], a major goal of developmental psychopathology is to improve prediction and control of mental illness, which should ultimately lead to more effective prevention and intervention programs, alleviating considerable human suffering (see also Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008).

Taxonomies of diseases, including psychopathology, are often referred to as *nosologies*. In this chapter we describe the predominant classification system of psychopathology in the United States—the *Diagnostic and Statistical Manual of*

Mental Disorders (5th ed.; *DSM-5*; American Psychiatric Association [APA], 2013). In doing so we (a) outline the history of the *DSM*; (b) highlight important issues and difficulties that emerge when diagnosing psychopathology; and (c) juxtapose the *DSM-5* and its limitations with alternative perspectives and theoretical orientations, including empirically derived taxonomies and the Research Domain Criteria (RDoC). The latter is a fairly new approach to characterizing psychopathology that is currently being developed by the National Institute of Mental Health (2015a).

HISTORICAL CONTEXT

Unlike the physical sciences, such as physics, chemistry, and geology, clinical psychology and psychiatry are relatively new. In fact, the first well-organized attempt in the United States at devising a classification system of psychopathology occurred only 64 years ago with publication of the first edition of the *DSM* (APA, 1952). As a result, psychology and psychiatry still struggle with unresolved taxonomic issues,[1] some of which are specific to children and adolescents (see e.g., Achenbach & Rescorla, 2006; Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Eaton, Krueger, South, Simms, & Clark, 2011; Jensen, Knapp, & Mrazek, 2006; Krueger et al., 2011; World Health Organization, 1996). These issues are described in sections to follow.

Early Versions of the *DSM*

The current version of the *DSM* is the *DSM-5* (APA, 2013), which is actually the eighth in a series of *DSM*s, including both major and minor revisions, dating to 1952 (*DSM-I*, 1952; *DSM-II*, 1968; *DSM-II*, seventh printing, 1974; *DSM-III*, 1980; *DSM-III-R*, 1987; *DSM-IV*, 1994; *DSM-IV-TR*, 2000; *DSM-5*, 2013). Below we provide brief descriptions of each *DSM*, list the primary objectives of the American Psychiatric Association in undertaking each revision, and outline major changes in each new edition.

DSM-I. The *DSM-I* (APA, 1952) was an effort by the APA to produce a single nomenclature for psychopathology. Prior to the *DSM-I*, there were several alternative classification systems, none of which was used consistently across the United States (see Blashfield, 1998). The *DSM-I* was influenced strongly by Adolph Meyer's psychobiology, which characterized psychopathology as a reaction

1. We are not suggesting that taxonomic questions have been resolved in other sciences. In fact, issues of classification continue to be debated in many other fields, including evolutionary biology (see e.g., Laurin, 2010) and paleontology (see Beauchaine, 2003).

to stress (e.g., Meyer, 1934). Hence, all disorders included "reaction" in their titles (e.g., depressive reaction). In formulating the *DSM-I*, the APA relied on the collective opinion of its membership. To do so, it sent detailed questionnaires to 10% of members, from which proposed categories of psychopathology were derived. Three broad classes of psychopathology emerged, including organic brain syndromes, functional disorders, and mental deficiency. Within these broad classes, 108 specific diagnoses were created (depending on the method of counting), only one of which could be applied specifically to children (adjustment reaction of childhood/adolescence). Final approval of psychiatric classes and specific diagnoses was obtained through a vote of the full APA membership. As this description implies, the *DSM-I* had little if any basis in empirical research.

DSM-II. The *DSM-II* (APA, 1968), which contained about 182 diagnoses (again, depending on the method of counting), was published with few changes in process or philosophy. A major goal in formulating the *DSM-II* was to improve communication among mental health professionals—especially psychiatrists (e.g., Scotti & Morris, 2000). The *DSM-II* had strong psychoanalytic overtones, reflecting the training of most psychiatrists at the time. Major diagnostic classes of psychopathology were expanded from 3 to 11, and a number of childhood and adolescent disorders were added, including group delinquent reaction, hyperkinetic reaction, overanxious reaction, runaway reaction, unsocialized aggressive reaction, and withdrawing reaction.

Since publication of the *DSM-II*, international treaty has dictated that the *DSM* and the International Classification of Diseases (ICD) be compatible. The ICD, published by the World Health Organization (WHO), is the classification system used in most other countries to diagnose mental illness. Some changes made to the *DSM-II* were needed to render it more similar to the ICD-8 (WHO, 1966). Currently, the ICD is in its 10th edition—revised (ICD-10; WHO, 2008). The ICD-11 is expected in 2018 (WHO, 2015).

DSM-II, Seventh Printing. In the seventh printing of the *DSM-II* (APA, 1974), homosexuality was removed as a mental disorder, following protests by gay rights activists at the 1970 Annual Convention of the APA in San Francisco and a subsequent vote of the membership. This landmark event illustrates several important and interrelated points about diagnosis of mental illness. First, diagnostic systems such as the *DSM*, which are constructed by social institutions, always reflect social values (see e.g., McCarthy & Gerring, 1994). Second, psychiatry and related disciplines at times reinforce prevailing social value systems, which can lead to stigmatization of certain members of society, with considerable potential for negative effects on mental health (see e.g., Prilleltensky, 1989). Finally, as a social institution, the APA is not indifferent to sociopolitical influence.

Removing homosexuality from the *DSM-II* also foreshadowed struggles to deal with validity of psychiatric diagnosis more broadly, a major issue confronted in later revisions of the *DSM*, as described below.

Reliability, Validity, and Subsequent Versions of the *DSM*

In contrast to the *DSM-I* (APA, 1952) and the *DSM-II* (APA, 1968), the *DSM-III* (APA, 1980) was designed to be descriptive and largely atheoretical, so it would appeal and be useful to professionals from disciplines and conceptual orientations beyond psychiatry. Research on clinical features and etiologies of major forms of psychopathology were also weighted heavily in formulating the *DSM-III*—a major shift from the consensus opinion approach to constructing its earlier versions (see above). Thus, introduction of the *DSM-III* in 1980 was a watershed event in modern classification of psychopathology. Prior to 1970, most mental health professionals in the United States were not especially concerned with psychiatric diagnosis. The dominant paradigm was psychoanalysis, which did not place much stock in diagnosis. However, in the 1960s a new paradigm, often referred to as biological psychiatry, challenged and ultimately supplanted psychoanalysis as the dominant perspective in the United States. One agenda of biological psychiatry proponents was to make the discipline more scientific by increasing its emphasis on empirical research, particularly on the biological bases and treatment of psychopathology, thereby bringing psychiatry into mainstream modern medicine.

Diagnosis played a central role in this agenda, as a reliable and valid classification system was necessary for the enterprise. Indeed, how successful could research on biological causes/correlates of psychopathology be if the major independent variable—diagnosis—was unreliable or invalid? Because diagnosis was a cornerstone of well-developed specialties in medicine (e.g., Engel, 1977), emphasis on reliable diagnosis was paramount. However, there was a major obstacle: limited evidence of interrater reliability of psychiatric diagnosis (e.g., Spitzer & Fleiss, 1974).

Problems with reliability were hard to ignore. First, rates of various diagnoses differed dramatically between the United States and most European countries. For example, the rate of schizophrenia was many times higher in the United States than in the United Kingdom. In order to address this issue, a team of researchers in the United States and United Kingdom launched the Cross-National Diagnostic Project (for a description see Gurland, 1976). Using the same diagnostic criteria and assessment procedures, they found that differences in clinical diagnoses between hospitals in New York and London were attributable entirely to different diagnostic practice; patients' symptoms were virtually identical in both cities. Furthermore, clinical diagnoses by British psychiatrists corresponded more closely to patients' actual clinical presentations than those by American psychiatrists, who greatly overdiagnosed schizophrenia and underdiagnosed mood disorders.

Second, almost all studies that addressed diagnostic reliability during that era indicated very low interrater agreement. Spitzer and Fleiss (1974) aggregated

data from these studies, and calculated interrater reliability using the kappa (κ) statistic, which measures the degree of association between categorical constructs such as presence vs. absence of a diagnosis, correcting for chance agreement. In general, κs ranging from 0 to .20 indicate slight agreement, .21 to .40 fair agreement, .41 to .60 moderate agreement, .61 to .80 substantial agreement, and .81 to 1.0 excellent agreement (Landis & Koch, 1977). Spitzer and Fleiss reported that κs from previous interrater reliability studies were .41 for depression, .33 for mania, .45 for anxiety neurosis, .57 for schizophrenia, and .71 for alcoholism. Only the latter could be considered adequate.

Spitzer and Fleiss (1974) attributed low interrater reliability to two sources: *criterion variance* and *information variance*. Criterion variance refers to diagnosticians' reliance on different criteria when making a diagnosis, whereas information variance refers to collection of different data (see below).

With respect to criterion variance, if one clinician diagnoses schizophrenia on the basis of even mild indications of cognitive slippage (a form of thought disorder), whereas another reserves the diagnosis only for patients who exhibit severe delusions or hallucinations, agreement will be low. In this regard, the *DSM-I* (APA, 1952) and *DSM-II* (APA, 1968) were not helpful because their diagnostic criteria were vague. Each diagnosis was described in several sentences listing characteristic signs and symptoms, yet there was no specification of how many symptoms were required, how long a symptom had to be present, or whether other symptoms might rule out a diagnosis (e.g., in a patient with visual hallucinations, could schizophrenia be diagnosed in the context of acute alcohol withdrawal?).

Operationalizing Diagnostic Criteria: Reducing Criterion Variance

The criterion variance problem was addressed initially by Mandel Cohen, who was interested in developing a more empirical approach to studying psychopathology. Cohen conducted several pioneering studies of mood, anxiety, and somatoform disorders. These involved formulating very careful criteria for diagnosis, applying them to what at the time were large samples of patients, and examining patients' clinical presentations, family histories, and clinical course (see Healy, 2002). Psychiatric journals were not particularly interested in this work, so most of Cohen's papers were published in medical journals (e.g., Cohen, Cassidy, Flanagan, & Spellman, 1937; Cohen, Robins, & Purtell, 1952), with very little effect on psychiatry or psychology.

One of Cohen's students was Eli Robins, who became chair of the Psychiatry Department at Washington University in St. Louis. Throughout the 1960s, Robins and several colleagues, including Samuel Guze and George Winokur, applied Cohen's approach in a series of landmark studies of psychopathology (e.g., Arkonac & Guze, 1963; Reich, Clayton, & Winokur, 1969). One of the hallmarks of the Washington University approach was development of systematic operational (i.e., explicit) diagnostic criteria for a selected group of diagnoses.

This approach was explicated by E. Robins and Guze (1970), who published a brief yet highly influential paper in which they advanced a five-step process toward ensuring that psychiatric classes were specific, objective, and nonarbitrary. Using the example of schizophrenia, Robins and Guze suggested that diagnostic validity can be established only when a clinical syndrome is characterized by (1) a cluster of covarying symptoms and etiological precursors (obtained from clinical description); (2) reliable physiological, biological, and/or psychological markers (obtained from laboratory studies); (3) readily definable exclusionary criteria; (4) a predictable course (assessed through follow-up studies); and (5) increased rates of the same disorder among first-degree relatives (assessed through family studies). The Robins and Guze method was soon used by Feighner et al. (1972) to develop the first set of psychiatric disorders that were validated systematically. Associated symptom lists are now referred to as the *Feighner Criteria*. Although the primary motivation in formulating the Feighner Criteria was to validate psychiatric disorders (see Kendler, Munoz, & Murphy, 2009), doing so required specification of explicit operational criteria, as noted above.

Soon after the Feighner Criteria (1972) were published, the NIMH sponsored the Collaborative Study of the Psychobiology of Depression, a multisite investigation of the clinical features, family history, biological correlates, and course of depression (see Katz, Secunda, Hirschfeld, & Koslow, 1979). As part of this study, the NIMH contracted with Spitzer and Endicott to develop a revised version of the Feighner criteria, which came to be known as the Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978). Thus, by the late 1970s, the importance of specifying operational criteria for psychiatric disorders was widely recognized among the psychopathology research community, which strongly influenced development of the *DSM-III* (APA, 1980) and all subsequent versions of the *DSM* (see e.g., Cloninger, 1989; Kendler et al., 2009), including the *DSM-5* (APA, 2013).

Structured Interviews: Reducing Information Variance

With the goal of reducing information variance, a major task of the US-UK Cross-National Project was to standardize collection of data on symptoms, assessed by British and American clinicians. Accordingly, Wing, Cooper, and Sartorius (1974) developed a standardized clinical interview that provided (a) specific questions to be asked by the interviewer, (b) specific rating scales for each symptom, (c) conventions for making ratings, and (d) a detailed glossary defining each symptom. This instrument was called the Present State Examination (PSE), which was designed to allow experienced clinicians to obtain a systematic assessment of patients' current symptoms. It did not collect information on previous course or history and therefore could not be used to make diagnoses. However, it was an important advance in standardizing collection of information across clinicians and sites.

At the same time, psychiatrists at Washington University developed a semistructured diagnostic interview for use in various research projects being conducted in their department. Like the PSE, it included standardized questions and rating scales. However, it also provided a systematic assessment of the development and course of psychopathology, rather than focusing only on the patient's current state (Woodruff, Goodwin, & Guze, 1974). Thus, it included all information necessary to make diagnoses according criteria established at the time (see above).

Soon afterward, as part of their role in the NIMH Collaborative Study of the Psychobiology of Depression Study, Endicott and Spitzer (1978) developed a semistructured diagnostic interview called the Schedule for Affective Disorders and Schizophrenia (SADS). This interview allowed trained clinicians to collect systematic and reliable data on both current symptoms and history of most major psychiatric disorders. Thus, use of the SADS also allowed clinicians to make specific diagnoses.

By the time the *DSM-III* was published in 1980, structured diagnostic interviews were accepted as state-of-the-art in psychiatric assessment. However, both the PSE and SADS were quite time consuming, and neither matched the *DSM-III*. Hence, Spitzer and Williams (1983) developed a new instrument, the Structured Clinical Interview for *DSM-III* (SCID), which eventually assessed all major disorders in the *DSM-III* and later the *DSM-III-R* (e.g., Spitzer, Williams, Gibbon, & First, 1990), *DSM-IV* (e.g., First, Spitzer, Gibbon, & Williams, 2002), and *DSM-5* (First, Williams, Karg, & Spitzer, 2015). One objective was that the SCID be sufficiently user-friendly to be adopted in routine clinical practice in addition to research, although such adoption is extremely limited.

Another major development in structured interviewing was construction of the Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981), by Lee Robins (not to be confused with E. Robins, her spouse), a sociologist at Washington University who pioneered research on antisocial personality disorder (see, e.g., Dishion & Hiatt Racer, 2013). The impetus for development of the DIS was a report by the Carter Administration's Presidential Commission on Mental Health, which stressed the need to collect better data on the prevalence of mental disorders in the United States. This report led to the NIMH Epidemiological Catchment Area (ECA) survey, the largest epidemiological study of mental disorders ever conducted at that time (see Regier et al., 1984). When designing this study, it was clear that hiring trained clinicians to conduct diagnostic interviews with over 18,000 participants would be prohibitively expensive. L. Robins and colleagues therefore developed the DIS so it could be used by lay interviewers with no previous training in psychopathology. Because it was designed for use by nonclinicians, it is much more structured than other diagnostic interviews, and, unlike the PSE, SADS, and SCID, it leaves no room for interviewer judgment in formulating questions and rating symptoms. With these latter instruments, the interviewer is expected to probe

respondents' answers until confident they understand the question and are reporting a clinically significant experience that is relevant to the construct being assessed. In contrast, with the DIS, interviewers take the respondents' report at face value. Thus, it is a respondent-based, as opposed to an interviewer-based interview (Angold & Fisher, 1999). Diagnoses are derived by computer using *DSM* criteria.

In order to assess rates of psychopathology in large epidemiological samples of children and adolescents, the NIMH later developed the Diagnostic Interview Schedule for Children (DISC; Costello, Edelbrock, Dulcan, Kalas, & Klaric, 1984). The current version of the DISC assesses 30 *DSM-IV-TR* (APA, 2000) psychiatric disorders. It is designed for use with parents of children, ages 6–17, and with both children and adolescents, ages 9–17. There is currently no DISC for the *DSM-5*, although one is being constructed. This tardiness may be of limited consequence for most childhood disorders, as changes to the *DSM-5* were minimal (see below). Two exceptions are disruptive mood dysregulation disorder and intermittent explosive disorder—new diagnoses that are not represented in previous instantiations of the *DSM* (see e.g., Beauchaine & McNulty, 2013; Leibenluft & Stoddard, 2013). Like the DIS, the DISC is respondent-based, and can be administered by lay interviewers (Shaffer, Fischer, Lucas, Dulcan, & Schwab-Stone, 2000). Both the DIS and DISC have been controversial, with some questioning the validity of diagnoses so completely based on self-report—especially among youth (see e.g., Renou, Hergueta, Flament, Mouren-Simeoni, & Lecrubier, 2004). Indeed, adolescents who suffer from externalizing behavior disorders such as ADHD and conduct disorder often underreport their symptoms (e.g., Sibley et al., 2010). It is therefore routinely recommended that adult informants (parents) also provide data for such conditions. Nevertheless, considerable evidence points toward reliability of the DISC (see Shaffer et al., 2000), and its use in research settings is now commonplace.

Finally, semistructured, interviewer-based diagnostic interviews have also been developed to assess psychopathology among children and adolescents (Dougherty, Klein, Olino, & Laptook, 2008). The most widely used of these is a downward extension of the SADS—the Kiddie SADS (Kaufman et al., 1997).

The *DSM-III*, *DSM-III-R*, *DSM-IV*, and *DSM-5*

DSM-III. Following from his extensive work on psychiatric diagnosis outlined above, Spitzer was chosen to lead on revisions to the *DSM-III*. Rather than continuing with tradition, he looked toward the Feighner et al. (1972) criteria and the RDC (Spitzer, Endicott, & Robins, 1978) as a means of solving the problem of criterion variance. The *DSM-III* therefore became the first official classification system in psychopathology that used specific symptoms, including inclusion, exclusion, and duration criteria for each diagnosis. This effort required a major expansion of the

Feighner criteria and the RDC, which at the time covered no more than about 15 disorders.

The *DSM-III* (APA, 1980) also introduced multiaxial classification. Thus, in addition to classifying major psychiatric syndromes (Axis I), separate axes were created for personality disorders (Axis II); physical conditions that are relevant to understanding a person's presenting problem (Axis III); psychosocial and environmental stressors and problems (Axis IV); and overall severity, or global assessment of functioning (GAF; Axis V). Use of multiple axes was a means of addressing patients' uniqueness in making a diagnosis: not every patient with the same diagnosis is the same in all respects. This is a particularly important consideration in developmental psychopathology research (see Chapter 1 [Hinshaw]), which emphasizes equifinality and contextual influences on the development of mental illness (see Chapters 1 [Hinshaw] and 4 [Compas, Gruhn, & Bettas]).

DSM-III-R. A revised version of the *DSM-III* (APA, 1987) was published only seven years later. In large part because so little new research was available, changes were minimal, and the revision was not extensive enough to warrant being called a fourth edition. The rationale for the revision was that some diagnostic criteria were inconsistent, unclear, or contradicted by subsequent research (APA, 1987).

Despite almost no alterations to diagnostic criteria, one set of changes had major consequences. Following publication of the *DSM-III* (APA, 1980), several studies were published questioning widespread use of *exclusion criteria*. Exclusion criteria are a means of implementing diagnostic hierarchies, which serve to simplify diagnosis. Patients typically present with a wide array of symptoms. Traditionally, a major task of diagnosing has been *differential diagnosis*—deciding what the most appropriate diagnosis is among many possibilities suggested by the patient's clinical presentation. Diagnostic hierarchies are useful in differential diagnosis because they indicate which symptoms should receive priority. Prior to the *DSM-III-R*, *organic* mental disorders (syndromes attributable to central nervous system disease, brain trauma, or significant substance abuse) were at the top of the diagnostic hierarchy. Next came schizophrenia. Then came major mood disorders, with neurotic and personality disorders at the bottom. Thus, in the absence of organic factors, schizophrenia symptoms were accorded priority in diagnosis, regardless of the presence of major mood, neurotic, and/or personality disorder features. In the absence of both organic factors and schizophrenia symptoms, mood disorder symptoms took precedence regardless of neurotic and personality disorder features. Finally, neurotic and personality disorder diagnoses were only considered if organic, schizophrenia, and mood disorder features were absent.

Several studies in the early 1980s demonstrated that exclusion criteria in the *DSM-III* (APA, 1980) were often arbitrary and caused a loss of significant information. For example, family histories of patients with major depression and panic

disorder differed from those of patients with major depression alone (Leckman, Weissman, Merikangas, Pauls, & Prusoff, 1983). Hence, comorbid panic disorder appeared to be important, and excluding the panic disorder diagnosis among patients with major depression represented a loss of potentially important information. In light of these considerations, exclusion criteria were largely abandoned from the *DSM-III-R* (APA, 1987) onward, except those used to rule out organic (general medical or substance-induced) causes of disorder.

As might be expected, eliminating exclusion criteria led to a significant increase in rates of comorbidity—the co-occurrence of two or more disorders (see Klein & Riso, 1993). As a consequence, understanding comorbidity has been a top agenda item in psychopathology research ever since (see e.g., Angold, Costello, & Erkanli, 1999; Beauchaine & Cicchetti, 2016a, 2016b; Beauchaine, Hinshaw, & Pang, 2010; Klein & Riso, 1993). At the same time, reduction of hierarchical exclusion criteria has resulted in a diminished role for differential diagnosis in diagnostic practice.

DSM-IV. In 1994 the *DSM-IV* (APA, 1994) was published. One motivation for publishing a new version so soon was the international treaty requirement that the *DSM* be consistent with the ICD (see above), which was undergoing revision. Although content changes were again relatively minor, the process through which *DSM-IV* revisions were derived witnessed a marked change. Revisions were driven much more by data than before, and the process was more systematic and better documented. As outlined in the *DSM-IV* itself: (a) review papers were commissioned by the APA addressing relevant literature for almost all existing and proposed categories; (b) the NIMH funded 12 multisite field trials to collect data to inform decisions about revisions to criteria; (c) the MacArthur Foundation provided funding for several investigators to reanalyze existing data sets, thereby providing additional data relevant to proposed revisions, and; (d) the literature reviews, results from field trials, reanalyses, and rationales for all revisions were published in a multivolume *DSM-IV* Sourcebook (e.g., APA, 1996). A similar process was carried forward to the *DSM-5*, as described below.

DSM-IV-TR. In the text revision to the *DSM-IV*, published in 2000 (APA, 2000), diagnostic categories and their criteria were left almost completely unchanged. Instead, factual errors were corrected; sections of text describing each diagnostic category, associated features, advances in laboratory and clinical research, and so on were revised based on new research; and diagnostic codes that had changed in the latest edition of the ICD were updated.

DSM-5. The revision process for the *DSM-5* (APA, 2000) began in 1999 with an informal discussion about the need to improve validity of psychiatric diagnosis between Steven Hyman, director of the NIMH; Steven Mirin, medical director of

the APA; and David Kupfer, chair of the APA Committee on Psychiatric Diagnosis and Assessment at the NIMH (APA, 2012a). This discussion spawned the initial *DSM-5* Research Planning Conference in 1999, sponsored by both the APA and the NIMH. Participants invited to this conference included experts in behavioral genetics, molecular genetics, neuroscience, life-span development, cognition, and behavior. Notably, many of those involved in the *DSM-IV* revision were not invited, with the explicit purpose of encouraging new thinking. The Committee commissioned a series of white papers to identify (a) areas of needed research, (b) cross-cutting unresolved issues in psychiatric diagnosis, (c) ways in which the burgeoning research base in neuroscience could inform psychiatric diagnosis, and (d) issues of culture in psychopathology, among others. Soon after the conference, Darrel Regier was recruited to coordinate development of the *DSM-5*. Regier became vice chair of the *DSM-5* Task Force, which was chaired by David Kupfer. A first set of white papers appeared in 2002 (Kupfer, First, & Regier, 2002), and a second set appeared in 2007 (Narrow, First, Sirovatka, & Regier, 2007). These edited volumes identified specific areas in which new research was needed.

Between 2004 and 2008, 13 conferences were held among experts at the NIMH, the APA, the WHO, the American Psychiatric Institute for Research and Education, the National Institute on Drug Abuse, and the National Institute on Alcoholism and Alcohol Abuse. Participants from both the United States and other nations wrote a series of review papers, from which more specific research agendas were developed (APA, 2012b).

In 2006, Kupfer and Regier nominated chairs of the diagnostic work groups for the *DSM-5* Task Force, who were approved by the APA Board of Trustees in 2007. These chairs then recruited leading experts in their fields to populate individual work groups, which were approved by the APA in 2008, after they had begun meeting. Thirteen work groups were formed, representing major diagnostic categories in the *DSM-IV-TR* (APA, 2000).

As with previous revisions (see above), the *DSM-5* Task Force implemented a series of field trials, this time to ascertain the validity, reliability, feasibility, and clinical utility of proposed criteria, including new dimensional indices—an approach never used in previous versions of the *DSM*. A goal of the field trials was to develop diagnostic criteria that are useful in both research and clinical settings. However, the design and implementation of the field trials were controversial, and the reliability of a number of criterion sets proved to be disappointing (Frances & Widiger, 2012; Regier et al., 2013), although they led to some revisions of criteria (APA, 2012c).

The personality disorders (PD) section was one of the most controversial parts of *DSM-IV*, and significant changes to PDs were anticipated in *DSM-5*. Indeed, the PD Work Group proposed a hybrid categorical/dimensional approach to diagnosis that required meeting overarching criteria for PD including impairment in self and interpersonal functioning. It also added five higher-order pathological trait dimensions

(negative affectivity, detachment, antagonism, disinhibition, psychoticism) and 25 lower-order facets on which all individuals would be rated. In addition, they recommended retaining only six of the 10 specific PD diagnoses: obsessive-compulsive, narcissistic, schizotypal, avoidant, antisocial, and borderline, with revisions of specific criteria for these diagnoses to reflect the pathological traits noted above (see Klein, Bufferd, Dyson, & Danzig, 2014 for a discussion of the application of these criteria in youth). The four PDs with the smallest databases—paranoid, schizoid, histrionic, and dependent—were to be dropped. These changes would have been a marked departure from the *DSM-IV-TR*, which used a categorical system in which PDs were grouped into three clusters (Cluster A, paranoid, schizoid, schizotypal; Cluster B, antisocial, borderline, histrionic, narcissistic; Cluster C, avoidant, dependent, obsessive-compulsive) and did not include overarching criteria for PD or trait dimensions. Despite recommendations of the PDs Work Group, these changes were not implemented, and the PDs section of the *DSM-5* was left unchanged from *DSM-IV-TR*. Proposed changes offered by the *DSM-5* PDs Work Group appear in Section III of the manual (emerging measures and models) and are being used by researchers, but it is unlikely that this system will be used in clinical practice.

In contrast, changes were made to a number of other sections. Here we focus on the most notable of these changes. Interested readers are referred to Beauchaine and Hayden (2016), and to specific chapters in this volume, for more detailed accounts. A major change was elimination of the *DSM-IV* multiaxial system of diagnosis (see above). The rationale for this change stemmed from the conceptual overlap between the major Axis I clinical syndromes and the Axis II personality disorders, as many Axis I disorders share the hallmarks of personality disorders—early-onset, persistence, and pervasive impact on functioning (Klein et al., 2014). In addition, Axes III, IV, and V were often if not usually ignored in applied settings.

Several changes, albeit minor, were made to ADHD. The *DSM-IV-TR* included three ADHD subtypes, including primarily hyperactive-impulsive, primarily inattentive, and combined. This subtyping scheme was dropped from the *DSM-5* in favor of *presentations*, which specify whether criteria have been met for hyperactivity/impulsivity, inattention, or both (i.e., combined)—specifically in the past 6 months. This change follows from recognition that many children move in and out of subtypes over time (e.g., Todd et al., 2008). In addition, the *DSM-5* no longer includes ADHD among the disruptive behavior disorders, but instead moves it to the neurodevelopmental disorders section, which includes intellectual disabilities, communication disorders, autism spectrum disorder, specific learning disorder, and motor disorders. This decision was based on (a) evidence for aberrant neural responding and functional connectivity across several brain regions/networks among children, adolescents, and adults with ADHD (see e.g., Chapter 13 [Nigg]; Diamond, 2005; Fair et al., 2013; Plichta & Scheres, 2014; Rubia, 2011), and (b) hope that classifying ADHD as a neurodevelopmental disorder will lead to early

diagnosis, more thorough assessment, easier access to intervention, and more research on effects of comorbid inattention and learning disabilities on academic achievement (see Tannock, 2013). In addition, the age of onset criterion for impairing symptoms was increased from under 7 to under 12 years of age, and symptom thresholds were reduced somewhat for adult diagnoses. More radical changes, such as expanding the number of impulsivity-related symptoms, were not adopted.

Changes were also made to the mood disorders section. In contrast to the *DSM-IV-TR*, which had one mood disorders section, the *DSM-5* differentiates between unipolar and bipolar disorders by parsing the categories into two sections, in order to acknowledge the link between bipolar disorder and schizophrenia spectrum disorders. In addition, the exclusionary criterion for bereavement is removed for major depressive disorder (MDD), given little evidence for meaningful differences between depressive episodes following loss compared with those that occur in other contexts (e.g., Kendler, Myers, & Zisook, 2008; although see Wakefield, 2013 for an opposing view). A new category of mood disorder, *persistent depressive disorder*, subsumes *DSM-IV-TR* chronic MDD and dysthymic disorder, given limited evidence of meaningful differences between the two syndromes (e.g., Klein, 2010; Klein, Shankman, Lewinsohn, Rohde, & Seeley, 2004).

More fundamental changes were made to the anxiety disorders section. Panic disorder and agoraphobia are now separate disorders, and posttraumatic stress disorder is moved from the anxiety disorders chapter into a new section, *trauma and stressor-related disorders*, given evidence of partially distinct etiologies (e.g., Stein, Craske, Friedman, & Phillips, 2011). Perhaps the largest change is elimination of OCD from the anxiety disorders section, which follows from emerging evidence that anxiety disorders and OCDs exhibit different patterns of comorbidity and arise from partially independent neural substrates (e.g., Stein et al., 2010; although see Abramowitz & Jacoby [2015] for a dissenting view). Finally, the *DSM-5* no longer distinguishes between anxiety disorders of childhood vs. adulthood, given limited evidence validity of such distinctions (e.g., Bögels, Knappe, & Clark, 2013). Thus, separation anxiety can be diagnosed at any age.

In addition to changes made to existing disorders, several new disorders were added to the *DSM-5*, a few of which are especially relevant for children and adolescents (although most also apply to adults). Disruptive mood dysregulation disorder (DMDD), which is characterized by severe tantrums accompanied by persistent dysphoric mood, was added to the depressive disorders section in *DSM-5*. This diagnosis was created, in large part, to reduce rampant overdiagnosis of pediatric bipolar disorder (see e.g., Batstra et al., 2012), given evidence that most children with severe mood dysregulation are not on the bipolar spectrum (see Chapter 21 [Blader, Roybal, Sauder, & Carlson]; Carlson & Klein, 2014). However, studies of the course and validity of DMDD are only beginning to appear (e.g., Dougherty et al., 2014).

Another new diagnosis is intermittent explosive disorder (IED), which is characterized by severe emotional lability (particularly anger and aggression). IED differs

from DMDD in that it does not require persistent dysphoria between outbursts, or a childhood onset. IEE has a lifetime prevalence rate of almost 8% among adolescents (McLaughlin et al., 2012).

A third addition to the *DSM-5* is nonsuicidal self-injury (NSSI), which is listed as a condition for further study. Adding NSSI follows from recognition that (a) its prevalence rate has increased in recent years (Nock 2010); (b) it is exhibited by a large proportion of depressed adolescents, especially girls (e.g., Wilkinson, Kelvin, Roberts, Dubicka, & Goodyer, 2011); (c) it is often a developmental precursor to borderline personality disorder (e.g., Crowell, Beauchaine, & Linehan, 2009); (d) it is associated with altered patterns of central nervous system activity (e.g., Sauder, Derbidge, & Beauchaine, 2015), peripheral nervous system activity (e.g., Crowell et al., 2005), neuroendocrine responding (Beauchaine, Crowell, & Hsiao, 2015), and serotonergic function (e.g., Crowell et al., 2008); and (e) it marks considerable functional impairment, both concurrently and prospectively, and predicts future suicide attempts better than any other independent variable (e.g., Klonsky, May, & Glenn, 2012; Nock 2010).

THE *DSM* AND DEVELOPMENTAL PSYCHOPATHOLOGY

Although it is important for any student of psychopathology to understand the history behind, rationale for, and use of the predominant classification system of mental disorders in the United States, it is equally important to understand limitations of that system. Indeed, several departures in philosophy between the *DSM* approach and the developmental psychopathology approach to characterizing mental health are apparent. Historically, criticisms of the *DSM* have come from both within and outside psychiatry (see e.g., McCarthy & Gerring, 1994; van Praag, 2010), with developmental psychopathologists providing some of the most incisive critiques (e.g., Richters & Cicchetti, 1993). We and others have summarized these critiques, and provided a few of our own elsewhere (e.g., Beauchaine, 2003; Beauchaine et al., 2009; Cummings, Davies, & Campbell 2000; Hinshaw & Park, 1999; Hudziak, Achenbach, Altoff, & Pine, 2007). Here we provide an overview of such criticisms, some of which are specific to the *DSM-5*, but most of which apply to the overall philosophy that undergirds—oftentimes implicitly—categorical diagnostic systems.

Problems With Changes to the *DSM-5*

Even though most changes to the *DSM-5* were minor, it will take years of research to determine how effective this newest revision will be in increasing the validity of psychiatric diagnosis—a major objective of the *DSM-5* Task Force, the APA, and other interested parties (see e.g., Kraemer, Kupfer, Narrow, Clarke, & Regier, 2010). It is

likely, however, that several decisions made by the *DSM-5* Task Force will interfere with this objective. Although the Task Force explicitly charged *DSM-5* workgroups with proposing changes that were founded in empirical research, the Task Force ultimately ignored several of these recommendations. For example, despite strong evidence that several PDs can be diagnosed reliably in adolescence and that developmental precursors to these PDs exist (see e.g., Chapter 19 [Kaufman, Crowell, & Lenzenweger]; Beauchaine et al., 2009; Crowell, Kaufman, & Beauchaine, 2014; Klein et al., 2014), the *DSM-5* proscribes PD diagnoses among those who are under age 18 years. Second, the *DSM-5* retains all *DSM-IV-TR* PDs, despite little evidence for the validity of several and almost no evidence for validity of the A, B, and C clustering structure outlined above (see Beauchaine et al., 2009).

The decision to move ADHD into the neurodevelopmental disorders section and out of the disruptive behavior disorders section is also problematic in some ways. As noted earlier, this decision was based largely on practical grounds, such as hopes for earlier diagnosis, more thorough assessment, easier access to intervention, and more research on effects of comorbid inattention and learning disabilities on academic achievement (Tannock, 2013). Notably, such considerations were not applied to other disorders. If they had been, one could argue convincingly that conduct disorder (CD) should have also been moved, since ADHD and CD share common neurodevelopmental substrates and psychopathological endpoints (see Chapter 13 [Nigg]; Beauchaine & McNulty, 2013; Diamond, 2005; Fair et al., 2013; Gatzke-Kopp, 2011; Gatzke-Kopp et al., 2009; Kopp & Beauchaine, 2007; Rubia, 2011). Thus, moving ADHD to a different section of the *DSM* obscures its etiological connections with CD and other disruptive behavior disorders (see Beauchaine & Hayden, 2016; Beauchaine & Hinshaw, 2016; Beauchaine, Zisner, & Sauder, 2017). Of course, there is not complete correspondence between (a) ADHD and (b) CD and other antisocial-spectrum conditions (for a historical overview, see Hinshaw, 1987; see also Ahmad & Hinshaw, 2016), but placing ADHD in the neurodevelopmental disorders section may not be conceptually clarifying in all respects.

From a developmental psychopathology perspective, the decision to drop the multiaxial structure that characterized the *DSM-III*, *DSM-III-R*, *DSM-IV*, and *DSM-IV-TR*, particularly Axis IV (psychosocial and environmental stressors), is also unfortunate. De-emphasizing psychosocial and contextual factors downplays the important role that environment plays in shaping almost all forms of mental illness—even those with strong genetic underpinnings (see Chapters 1 [Hinshaw] and 3 [Beauchaine, Gatzke-Kopp, & Gizer] Beauchaine et al., 2017).

Additional Criticisms of the *DSM* Approach

Problems With Construct Validity. Although application of the Feighner Criteria and the RDC to some (though not nearly all) disorders represents an attempt to ensure

diagnostic validity (see above), reliability has been of far greater concern from the *DSM-III* onward (APA, 1980; see e.g., Kraemer, Kupfer, Narrow, Clarke, & Regier, 2010). It is important to note that reliability is necessary for validity but does not ensure validity. To use a somewhat hyperbolic example, separate raters can agree with very high precision that a person is over 6′5″ (reliability), but such agreement says nothing about height being a symptom of mental illness (validity). Indeed, any such assertion would be fully arbitrary—a situation that applied to sexual orientation before the seventh printing of the *DSM-II*, when homosexuality was considered a mental disorder (see above).

In developmental psychopathology research, *construct validity* refers to the extent to which symptoms of a diagnosis mark an objective, nonarbitrary entity that relates to mental health outcomes. Construct validity should be considered whenever the cause of a trait cannot be observed directly (Cronbach & Meehl, 1955), which is usually the case for psychopathology. To borrow an example we have used elsewhere (Beauchaine & Marsh, 2006), consider the difference between a medical syndrome such as pancreatic cancer and a common psychiatric condition such as MDD. In the former case, a patient presents at his/her physician's office with a collection of symptoms, which might include weight loss, dark urine, nausea, and abdominal pain. This collection of symptoms, or *manifest indicators*, leads to a hypothesis on the part of the physician regarding its unobserved, or *latent* cause. Importantly, for a medical condition such as pancreatic cancer, the hypothesis is confirmed or disconfirmed by a biopsy or other diagnostic test. If the biopsy is positive, the cause of the disorder becomes known. If the biopsy is negative, a new hypothesis is generated and tested.

Compare this with a depressed individual, who also presents with a collection of symptoms, including depressed mood, anhedonia, fatigue, weight loss, and insomnia. In contrast to the case of pancreatic cancer, there are no diagnostic tests that can identify most causes of depression (although certain medical conditions such as hypothyroidism can be identified and should therefore be ruled out). Thus, we are left with a somewhat tautological definition of depression: The patient is depressed because s/he presents with a collection of symptoms, and the patient presents with a collection of symptoms because s/he is depressed. We are therefore forced to infer psychopathology with no gold standard or pathognomonic sign of disease state (see Beauchaine & Thayer, 2015).

Under such conditions, difficulties posed for construct validation of psychiatric disorders are often formidable. Prior to publication of the *DSM-III* (APA, 1980), almost no evidence existed for the construct validity of any diagnostic category (Kendell, 1989), because all were derived clinically rather than through systematic research (see above). At present, even after decades of relevant research, unanswered questions about the construct validity of many psychiatric disorders abound. For example, in research on pediatric bipolar disorder, issues regarding proper diagnostic cutoffs and delimitation from other disorders including

ADHD have not been addressed fully (see Chapter 21 [Blader, Roybal, Sauder, & Carlson] Carlson & Klein, 2014).

Heterogeneity Within Diagnostic Classes. A related issue follows from the observation that diverse etiologies often result in what appears to be a single disorder, a phenomenon known as *equifinality* (see Chapter 1 [Hinshaw]). For example, impulsivity may arise from one of several sources, each of which may be expressed behaviorally as ADHD (see Chapters 6 [Neuhaus & Beauchaine], 13 [Nigg], & 10 [Arnett et al.]; Castellanos-Ryan & Séquin, 2015; Zisner & Beauchaine, 2015). However, since *DSM* diagnoses are all derived syndromally (i.e., from symptoms with little if any regard to etiology or pathophysiology), different underlying causes of a disorder may never be ascertained, even when it is possible to do so.

Both treatment and prevention are improved when pathophysiological and etiological diagnosis are used rather than syndromal diagnosis (see Beauchaine et al., 2008; Preskorn & Baker, 2002). For example, if hypothyroidism is identified in the pathophysiology of depression, treatment follows a very different course (synthetic thyroxine treatment) than antidepressant use and/or psychotherapy. Although this example may seem extreme, potentially meaningful distinctions among depression subtypes are underemphasized in the *DSM-5*. For example, melancholia—a subtype of depression that appears to arise from different etiological mechanisms than nonmelancholic depression (see Leventhal & Rehm, 2005)—may confer increased risk of adverse long-term functional outcomes including suicide (e.g., Carroll, Greden, & Feinberg, 1980; Coryell & Schlesser, 2001), yet it is not classified as a separate mood disorder, even though some argued ardently for doing so in the *DSM-5* (e.g., Parker et al., 2010).

Categorical Versus Dimensional Measurement. One of the most persistent criticisms of the *DSM* is that all disorders are diagnosed categorically (i.e., present vs. absent), even though overwhelming research evidence indicates that most forms of psychopathology (a) reflect extreme expressions of continuously distributed traits (see e.g., Haslam, Holland, & Kuppens, 2012; Hudziak et al., 2007; Krueger & Tackett, 2015; Krueger, Watson, & Barlow, 2005; Trull & Durrett, 2005), and (b) are rooted in interactions among neural systems that subserve overlapping behavioral and emotional functions (see e.g., Beauchaine, 2015; Beauchaine & Thayer, 2015). Even in rare exceptions when psychiatric vulnerability may be distributed categorically (e.g., schizotypy; see Lenzenweger, McLachlan, & Rubin, 2007), individual differences in symptom expression are nevertheless observed and meaningful functionally (Beauchaine, Lenzenweger, & Waller, 2008). They also provide key information about current functioning and long-term prognosis.

Other adverse consequences of categorizing dimensions include difficulty ascertaining optimal diagnostic cutoffs (e.g., 95th percentile? 98th percentile?

see Meehl, 1995), and loss of statistical information (see MacCallum, Zhang, Preacher, & Rucker, 2002). Individuals in need of intervention may also be turned away because they fail to meet diagnostic criteria even though they suffer considerable impairment. To address such problems, hybrid classification systems have been proposed in which both presence vs. absence *and* severity of psychopathology are assessed (e.g., Hudziak et al., 2007). As outlined above, such an approach was recommended by the PDs Work Group for the *DSM-5*, but was ultimately rejected. Notably, dimensional assessment has long been used in child psychopathology research, even when applying *DSM* criterion sets (e.g., Achenbach & Edelbrock, 1991; Conners, Sitarenios, Parker, & Epstein, 1998; Gadow & Sprafkin, 1997; Robinson, Eyberg, & Ross, 1980). Such is not the case in adult psychopathology research.

Failure to Consider Development. Developmental psychopathologists have been especially critical of the *DSM* because it fails to consider issues of development in diagnosis (e.g., Beauchaine et al., 2017; Richters & Cicchetti, 1993; Sroufe, 1997). With few exceptions (e.g., early-onset conduct disorder; see Chapter 14 [Lahey & Waldman]), child and adolescent psychopathology are assessed and diagnosed without consideration of normative developmental trends in behavior, and without acknowledgement that single behavioral traits—including those that confer vulnerability to psychopathology—may be expressed differently at different ages. *Heterotypic continuity* refers to such changes in the behavioral expression of psychopathology across development (see Chapter 1 [Hinshaw]). As an example, we have known for over 50 years that delinquent adult males almost invariably traverse a developmental pathway that begins with severe hyperactivity/impulsivity as early as toddlerhood, followed in rough temporal sequence by oppositional defiant disorder (ODD; Chapter 14 [Lahey & Waldman]) in preschool, early-onset conduct disorder (CD; Chapter 14 [Lahey & Waldman]) in elementary school, substance use disorders (SUDs; Chapter 15 [Brown, Tomlinson, & Winward]) in adolescence, and antisocial personality disorder (ASPD) in adulthood (see e.g., Beauchaine & Hinshaw, 2016; Beauchaine & McNulty, 2013; Beauchaine et al., 2017; Loeber & Hay, 1997; Lynam, 1998; Robins, 1966). Thus, even though continuity in externalizing conduct is common among those on this trajectory, specific behaviors vary considerably across development (Ahmad & Hinshaw, 2016; Beauchaine, Shader & Hinshaw, 2015; Hinshaw, Lahey, & Hart, 1993). Among other consequences, failure to consider heterotypic continuity results in (a) a research literature that is fractionated based on topographies of behavior (e.g., tantrums in toddlerhood, truancy in elementary school, substance use in adulthood) rather than etiology, (b) alternative treatment strategies for conditions such as CD and SUDs that are not informed by one another when they would benefit from being so (see Beauchaine et al., 2008), and (c) faulty conclusions about etiology and comorbidity of externalizing disorders (see Beauchaine et al., 2010). Finally, there is growing evidence that many

preschool-aged children meet *DSM* criteria for psychiatric disorders (Bufferd, Dougherty, Carlson, Rose, & Klein, 2012). However, it is often unclear where to draw the line between developmentally normative and pathological behavior in early childhood, and whether diagnostic criteria developed for older children, adolescents, and adults are appropriate for preschoolers (Bufferd, Dyson, Hernandez, & Wakschlag, 2016).

Failure to Consider Culture and Other Contextual Issues. In general, the *DSM* is indifferent to both (a) culturally induced individual differences in behavior that might be mistaken for psychopathology (see e.g., Marsella & Yamada, 2010), and (b) cultural, socioeconomic, and other contextually driven individual differences in the expression of psychopathology (see e.g., Lewis-Fernández et al., 2010; Gone & Kirmayer, 2010). As a result, strict adherence to *DSM* criterion sets without consideration of race, ethnicity, and class can lead to both false positive and false negative conclusions regarding the presence versus absence of psychopathology. One objective of the developmental psychopathology approach is to construct a discipline that acknowledges the role of context in shaping behavior, and that does not assume—even implicitly—that group differences in behavior between members of the dominant social class and other cultural subgroups always imply deficits in functioning among the latter (e.g., Garcia-Coll, Akerman, & Cicchetti, 2000; Cicchetti & Toth, 2009; see also Chapter 1 [Hinshaw]).

EMPIRICALLY DERIVED CLASSIFICATION SYSTEMS

Early on, the *DSM* was, and in many ways remains, a top-down, *deductive* approach to classifying psychopathology. Opinions of experts are still weighed heavily in the revision process, and empirical findings are sometimes eschewed, despite explicit calls for, both within and outside *DSM* workgroups, a research-based taxonomy of mental illness (see above). In stark contrast to this approach, developmental psychopathologists have a long history of constructing and using, in both research and clinical settings, bottom-up, *inductive* systems of classification and assessment that derive almost fully from empirical interrelations among symptoms of psychopathology. The earliest and most renowned of these is the parent-report Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983), which was later expanded to include both teacher (Teacher Report Form [TRF]; Achenbach & Edelbrock, 1986) and self-report versions (Youth Self-Report [YSR]; Achenbach & Edelbrock, 1987). Collectively, these instruments, along with more newly developed adult versions, comprise the Achenbach System of Empirically Based Assessment (ASEBA; Achenbach, 2009).

The CBCL and its successors were derived from factor analyses of large sets of symptoms of psychopathology. These studies, and subsequent factor-analytic evaluations of adult psychopathology (e.g., Krueger, 1999), demonstrated a remarkably consistent hierarchical latent structure of mental illness in which two higher-order,

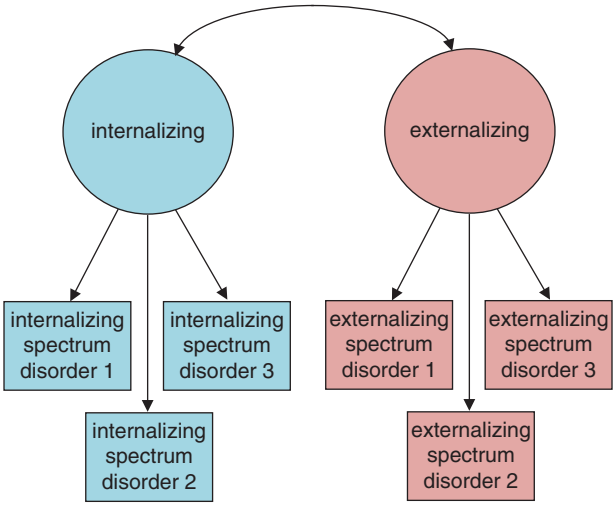

Figure 2.1 A simplified depiction of the hierarchical latent structure of psychopathology.
Adapted from Beauchaine and Thayer (2015).

latent factors, *internalizing* and *externalizing*, account for much of the covariation among first-order factors (i.e., behavioral syndromes).[2] This hierarchical latent structure of psychopathology is depicted in Figure 2.1. First-order internalizing factors include constructs such as anxious/depressed, withdrawn/depressed, and somatic complaints, whereas first-order externalizing factors include constructs such as impulsivity, rule-breaking behavior, and aggression. When using the CBCL and related empirically based assessment instruments, children and adolescents (and/or parent and teachers) rate each symptom, and these ratings are summed to provide scores on individual first-order syndromes. Syndrome scores are then added to compute broad-band (i.e., higher-order) internalizing and externalizing scores.

There are several advantages of empirically based assessment, compared with the approach to diagnosis represented in the DSM. First, raters are not forced to render dichotomous diagnostic decisions. Rather, each individual receives a set of scale scores, the severity of which can be evaluated vis-à-vis national norms. Oftentimes, children who score at or above the 95th percentile are considered to be clinically impaired. Lower but elevated scores, such of those above 85th percentile, may also be flagged for concern. Second, empirically based assessment does not

2. Factor analysis is a mathematical approach to reducing large numbers of items (in this case, symptoms), into a smaller number of *factors*, each of which consists of items that share common variance. Although most factor analyses of psychopathology allow for correlated factors, correlations of items within factors exceed correlations of items across factors. Interested readers are referred elsewhere for detailed accounts of factor analysis (e.g., Thompson, 2004).

force diagnosticians, even implicitly, to choose one disorder over others. Rather, elevated scores both within and across internalizing and externalizing domains are observed and expected, which "carves nature at its joints" more effectively than assigning a single disorder. For example, adolescents with conduct disorder (CD), although likely to experience symptoms of ADHD, are often diagnosed only with the former disorder, which may interfere with treatment and obscure etiological relations between the two conditions (e.g., Beauchaine et al., 2010; 2017). Third, such systems are more sensitive to capturing heterotypic comorbidity, whereby an individual with a primary externalizing disorder, for example, also displays—often subclinically—symptoms of an internalizing disorder (see, e.g., Zisner & Beauchaine, in press). Based on these considerations and others, empirically based assessment is used in almost all research contexts among developmental psychopathologists, even when *DSM*-derived diagnoses are also evaluated.

THE RESEARCH DOMAIN CRITERIA

In 2009, the NIMH, as part of its Strategic Plan (NIMH, 2015a), launched a new initiative, the Research Domain Criteria (RDoC; e.g., Cuthbert & Insel, 2013; NIMH, 2015b; Sanislow et al., 2010), to provide an alternative framework, particularly for research purposes, of studying and ultimately classifying psychopathology. RDoC was developed out of frustration with the slow pace in understanding the etiopathogenesis of, and development of effective treatments for, mental disorders, and a sense that the *DSM* has not adequately facilitated and may have hindered such progress (Insel et al., 2010). RDoC acknowledges that nearly all current *DSM*-defined clinical phenotypes are etiologically heterogeneous and lack neurobiological validity, and that information about core etiological mechanisms is needed to identify more homogeneous, biologically valid phenotypes (see also Beauchaine & Thayer, 2015)—a precondition for specifying molecular genetic substrates of psychopathology (see Chapter 6 [Neuhaus & Beauchaine]). Furthermore, RDoC assumes that key etiological influences, and ultimately clinical phenotypes, take the form of dimensions rather than discrete classes, an observation that has proven almost axiomatic in psychopathology research (see e.g., Forbes, Tackett, Markon, & Krueger, in press; Krueger & Tackett, 2015; Krueger et al., 2002).

RDoC descends from biobehavioral motivational systems perspectives, which were advanced initially in the mid- to late 20th century by distinguished investigators including Jeffrey Gray (see e.g., Gray, 1987) and Peter Lang (e.g., Lang, Bradley, & Cuthbert, 1992). These investigators identified broad, neurally mediated activation/approach and inhibition/withdrawal systems, which predispose to individual differences in dispositional responding to specific classes of stimuli (e.g., Beauchaine & Thayer, 2015; Fowles, 1988). RDoC, which is intended to be an evolving project that integrates research across human and infrahuman species, posits the existence of five major domains of behavior, which should be studied across multiple units of analysis, ranging from genes to molecules to cells to neural

circuits (e.g., emotion-modulated startle) to physiology (e.g., heart rate) to behavior (naturalistic observation or in particular tasks) to self-reports (interviews, questionnaires). These five domains, each of which includes a number of subdomains, were selected for their potential relevance to psychopathology, and because aspects of their neural circuitry are already understood. The domains include negative valence systems (acute threat [or fear], potential threat [or anxiety], sustained threat, loss, and frustrative nonreward); positive valence systems (e.g., initial responsiveness to reward, sustained responsiveness to reward, reward learning); cognitive systems (e.g., attention, perception, cognitive control, working memory), systems for social processes (e.g., affiliation and attachment, social communication, perception and understanding of the self, perception and understanding of others), and arousal and regulatory systems (arousal, circadian rhythms, sleep and wakefulness) (NIMH, 2015b).

These five domains and their subdomains are presented in a series of rows, and units of analysis head a series of columns, which together comprise the RDoC matrix (Morris & Cuthbert, 2012). Ultimately, cells in the matrix will be filled with measures of constructs in each domain, at each unit of analysis (e.g., fear-potentiated startle is a measure of the acute fear subdomain at the unit of circuits). Following from Cronbach and Meehl's (1955) classic construct validation framework, the goal is to develop and test a "nomological network" of hypotheses about interrelations among measures at various levels of analysis for each construct represented in the domains and subdomains.

The RDoC matrix also includes a column for paradigms, referring to tasks that are particularly useful in assessing the domain construct (National Advisory Mental Health Council Workgroup on Tasks and Measures for Research Domain Criteria, 2016). Finally, two important dimensions that are recognized as being critically important but are not formally included in the matrix are environmental influences and development (Casey, Oliveri, & Insel, 2014).

Despite its significant effect on funding priorities in the United States, RDoC is still very much under development and faces a number of questions and challenges. First, it is not clear how thoroughly and systematically development, the course of psychopathology, and environmental influences (including culture) will be incorporated, given that these are not formally represented in the matrix. Second, the construct validity of the domains and subdomains is only partially established. For example, it must be determined whether RDoC should include all of the most crucial domains and subdomains, and whether the convergent and discriminant validity of the domains and subdomains are consistent with the structure posited in the matrix. Third, even if phenotypes are defined on the basis of underlying processes rather than clinical presentation, it is likely that complex behaviors reflect interactions among multiple domains and subdomains (multifinality), and that particular domains and subdomains contribute to many different patterns of behavior (equifinality) (Beauchaine & Thayer, 2015). Fourth, measures for many of the cells in the matrix have yet to be identified, and the construct validity of many (if not most) of the candidate measures is only partially established. Moreover, related

to the previous point, it is likely that most of the endophenotypes/intermediate phenotypes that populate the cells are themselves highly complex (Iacono, Vaidyanathan, Vrieze, & Malone, 2014; see Chapter 3 [Beauchaine, Gatzke-Kopp, & Gizer]), and may reflect effects of multiple domains and subdomains. Fifth, magnitudes of associations between measures at different levels of analysis are often very modest, making it difficult to demonstrate construct validity (Patrick et al., 2013). Sixth, there are significant conceptual challenges to understanding relationships between and across units of analysis (Cicchetti, 2008; Meehl, 1977; Miller, 2010). Seventh, despite efforts not to privilege lower units of analysis, there are concerns that it may be susceptible to biological reductionism (e.g., Beauchaine et al., 2017; Berenbaum, 2010). Finally, and perhaps most importantly, the RDoC matrix does not include clinical phenotypes to classify patients and provide targets for clinical research and treatment.

Although this omission raises questions about clinical relevance, it is central to the entire endeavor. RDoC assumes that biologically valid phenotypes are likely to be narrower than, or cut across, diagnostic constructs in the *DSM*. Thus, a major goal of the RDoC initiative is to identify phenotypes that are related to impairment in core domains of biobehavioral functioning. Just as 35 years ago the field assumed that introduction of operational diagnostic criteria in *DSM-III* (APA, 1980) would increase reliability, thereby leading to more valid phenotypes, enhanced understanding of etiopathogenesis, and the development of more effective treatments (see above), proponents of RDoC are wagering that research elucidating core biobehavioral systems across multiple units of analysis will yield more valid phenotypes and better understanding of the causes and treatment of mental disorders.

CONCLUSIONS

In this chapter, we reviewed historical developments in psychiatric diagnosis and identified core issues confronted by those who seek to classify psychopathology. As our review indicates, the history of the *DSM*, RDoC, and the complexities behind their development are far more intricate than might be surmised at first glance. Although considerable efforts of many talented scientists have contributed to revising the *DSM*, longstanding issues of validity (and to a lesser extent reliability) remain to be addressed fully. Among the most important limitations of the *DSM* framework are its failures to (a) capture developmental processes underlying current and future risk for psychopathology, (b) specify pathophysiological and etiological mechanisms of psychopathology, (c) map broad biobehavioral traits that predispose to psychopathology across traditional diagnostic boundaries, and (d) account fully for contextual influences such as ethnicity and culture on the development of psychopathology. Although the RDoC initiative addresses some of these limitations, it ignores others—particularly those related to development, environment, and culture. These and other issues, which are central to the developmental psychopathology perspective (Chapter 1 [Hinshaw]), are addressed in chapters to follow.

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