CHAPTER 1

Developmental Psychopathology as a Scientific Discipline
A 21st-Century Perspective

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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
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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 multi-level 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
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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 endocannabinoid 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 \times 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 \times 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 \times 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.

REFERENCES


