PART I

THE DEVELOPMENTAL PSYCHOPATHOLOGY APPROACH TO UNDERSTANDING BEHAVIOR

CHAPTER 1

Developmental Psychopathology as a Scientific Discipline: Relevance to Behavioral and Emotional Disorders of Childhood and Adolescence

STEPHEN P. HINSHAW

A wealth of information has accumulated in recent years about the causes, correlates, and underlying mechanisms of child and adolescent mental disorders. At the same time, treatment strategies are becoming increasingly evidence based. The major goal of this volume is to provide up-to-date, conceptually and developmentally derived information about (a) risk factors for child and adolescent psychopathology and (b) the major conditions and disorders that come to clinical attention. Intervention strategies are not emphasized herein, as such information would require a set of chapters fully as long as the present contents. For a recent compendium of evidence-based treatments for child and adolescent disorders, see Silverman and Hinshaw (2008).

To contextualize and put into perspective why this topic area is so important, it is necessary to consider the levels of impairment and pain linked with child and adolescent psychopathology. Think, for instance, of the hopelessness and despair associated with depression in youth; the major limitations on life choices—and the sheer loneliness—so often imposed by many forms of Anxiety Disorder; the disorganization and chaos

related to Bipolar Disorder; the personal, family, school, and peer-related disruptions incurred by dysregulated attention and impulse control; the havoc wreaked by severely aggressive behavior on individuals and even entire communities; or the isolation and lost opportunities linked to Autism and other Pervasive Developmental Disorders. Similar portraits pertain to eating disorders, substance use and abuse, schizophrenia-spectrum conditions, and the beginnings of personality disorders. Overall, the personal and family confusion, grief, emptiness, and lost opportunities incurred by conditions such as these are deeply felt by all who are affected.

Furthermore, emotional and behavioral problems in 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, 2003; Wolfe & Mash, 2006). These conditions incur massive pain for individuals, families, and communities at large, triggering major economic burdens for caregivers, school districts, and health care systems. From a developmental perspective, not only are the major child and adolescent disturbances likely to persist across the lifespan, but the majority of mental disturbances experienced by adults have their origins in childhood and adolescence (Kessler, Berglund, Demler, Jin, & Walters, 2005).

Over and above the clinical and policy-related concerns raised by child and adolescent psychopathology, during the past century these conditions have begun to engage serious scientific efforts aimed at understanding their etiology, individual-level and systems-related maintaining factors, and empirically supported prevention and intervention efforts. After millennia of professional and scientific neglect of childhood psychopathology, we have now entered a time of rapid progress. The study of child and adolescent disorders is a major endeavor, and increasingly sophisticated efforts have begun to bear fruit in terms of scientific advances.

Conceptual bases for integrating developmental processes into the study of child and adolescent psychopathology have been present for several centuries, spanning fields such as embryology, systems theory, philosophy, and genetics (Cicchetti, 2006; Gottlieb & Willoughby, 2006). Yet it is only in the past several decades that developmental psychopathology (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 (for initial efforts, see Achenbach, 1974; Sroufe & Rutter, 1984). During this period, DP has exerted a major force on clinical child psychology, child psychiatry, developmental psychology, mental health services research, and a number of other disciplines in the behavioral and neurological sciences. New courses have been formed at major universities, journals have been created, and

governmental agencies have taken on the DP moniker to define their missions. It is remarkable how pervasive the DP perspective has become, galvanizing a host of clinical and scientific efforts.

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

In our instructions to the volume's contributors, we asked explicitly for coverage of historical context, epidemiologic factors, diagnostic issues, sex differences, cultural variables, developmental processes, and important psychobiological mechanisms that could illuminate the pathology under discussion. In providing these guidelines, we were clear 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 last century—interact and transact with biological risk variables to produce both maladaptation and healthy adaptation throughout development (for a compendium of integrative work focusing on adolescents, see Romer & Walker, 2007). Thus, we asked contributors to consider multilevel models, emphasizing transaction across a range of individual and contextual factors in the formation of psychopathology. Indeed, it is important to note that modern views of behavioral and molecular genetics have placed into sharp relief the unique and interactive roles that environmental and cultural forces exert on development (e.g., Cicchetti & Curtis, 2006; Rutter, Pickles, Murray, & Eaves, 2001).

Given page limitations and the desire for focused rather than exhaustive coverage, each chapter is relatively brief, with the goal of providing cogent, recent, and incisive commentary on conceptual issues, clinically relevant material, neuroscientific advances, and interactive models. It is our sincere hope that readers will use these contributions as a springboard for further exploration of conceptual frameworks, empirical research on etiology and mechanisms, and implications for prevention and treatment. Above all, we hope to provide a window into the integration of genetic, biological, psychological, and contextual forces that conspire to create costly and impairing patterns of maladaptive development. The utter complex-

ity of the enterprise is daunting and challenging; despite the considerable advances that have been made, the road ahead is long.

KEY DP CONCEPTS AND PRINCIPLES

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

Three related principles bear emphasis. The first is that multiple pathways to pathology exist. Indeed, disparate routes may lead to a common condition or outcome, exemplifying the construct of *equifinality.* For example, aggressive behavior could 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 from different combinations of these vulnerabilities and risk factors (e.g., Raine, Brennan, & Mednick, 1997). In other words, separate causal influences may well yield similar clinical endstates. In addition, the concept of *multifinality* pertains when a given risk factor or initial state leads to disparate outcomes during the course of development across different individuals. For instance, abuse

may or may not lead to severe maladaptation, depending on a host of intervening factors; extremes of inhibited temperament may produce shyness and social withdrawal, but other, healthier outcomes are also possible, depending on the presence or absence of additional risk or protective factors (for discussion, see Cicchetti & Rogosch, 1996).

Second, DP models place strong 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). Another way of putting this is that 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; Howe, Reiss, & Yuh, 2002; Kraemer et al., 2001), to ensure that (a) results are applicable to subsets of participants grouped on the basis of the moderator variable of interest (male versus female participants, those from different ethnic groups, or those with different patterns of comorbidity) and (b) underlying mechanisms of change, gleaned from mediator variables, are considered explicitly.

Third, given the rapid growth in recent years of genetic and genomic models as well as brain imaging methods, DP researchers in the twenty-first century must pay increasing attention to the role of the brain, and to neuroscientific principles in general, in order to account for the wide range of extant pathologies and their devastating impacts (see Cicchetti & Curtis, 2006). Clearly, we have come a long way from the mid-twentieth century, when biological and temperamental factors were virtually ignored in accounts of child development and psychopathology. To put into perspective just what a brain-based view entails, consider the following mathematical calculation: Adults have a "best estimate" of approximately 100 billion neurons in their brains; children are probably born with even higher numbers. Indeed a major developmental "task" over the earliest years of postnatal development is the pruning and migration of such neurons and their synaptic connections into a working, functional, and efficient brain.

As to the rate of neural development during the 40 weeks of human gestation, one can calculate the following quotient: Divide 200 billion (a fair estimate of the number of neurons with which an infant is born) by the number of seconds in 40 weeks. The result—of dividing 2 times 10 to the 11th power by this denominator, which is 2.4192 times 10 to the 7th power (i.e., the number of seconds)—is the astonishing figure that, on average, the embryo and fetus are producing around 8,000 new neurons *every sec-*

ond throughout the entire course of prenatal development. This average is not constant, of course, given that the neural tube and brain do not even form for some weeks; thus, in some crucial periods, this figure is far higher (see Giedd et al., 2006, for additional information on the precise timing of neural development across pregnancy and childhood).

Given such a staggering rate of development, a key question involves the joint influence of genes, hormones, nutrition, life experiences, and contextual influences on the plasticity of the brain's development—that is, the ultimate fate of this vast number of neurons—across childhood and adolescence. The number of potential synapses associated with any given neuron is large, making for an incalculably vast number of possible interconnections. Without transactional processes, multilevel models, computational frameworks, and a host of new information and technology related to developmental neuroscience, we will not be able to solve the problem of gaining deep understanding of relevant mechanisms (see also Romer & Walker, 2007).

All of the issues, terms, concepts, and principles described in the previous paragraphs have been stated and restated across a large number of articles, chapters, and books that promote and explicate DP models. Indeed, detailed discussion of any one of them could easily fill a volume unto itself. The challenge for the current chapter is to encapsulate these tenets, in order to foreshadow and illuminate the content of the remaining chapters on specific risk factors and specific disorders. In addition, explanations of these concepts too often remain at a rather global and abstract level, leaving unresolved precisely what they suggest for the investigation and treatment of behavioral and emotional disorders. In the following section, I therefore try to bring a number of these precepts to life. Note that in Chapter 2 Dante Cicchetti provides a treatise on the importance of investigating multiple levels of analysis in the DP enterprise. Because of this full coverage immediately following the current chapter, this topic is not emphasized herein, although integrating across multiple levels of analysis is essential to all work in DP (for a series of papers on this topic, see Cicchetti & Dawson, 2002).

Normal and Atypical Development Are Mutually Informative

As opposed to the study of discrete, mutually exclusive categories of *disorder,* DP models emphasize that phenomena defined as abnormal represent aberrations in normal developmental pathways and processes—and, accordingly, that without understanding typical development, the study of pathology will remain incomplete and decontextualized. For example, illuminating the nature of Attention-Deficit / Hyperactivity Disorder (ADHD)

requires thorough understanding of the normative development of attention, impulse control, and self-regulation (Nigg, 2006; Nigg, Hinshaw, & Huang-Pollack, 2006). Similarly, investigations of Autism must take into account the development of interpersonal awareness and empathy, 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). 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 the elucidation of pathology.

Intriguingly, however, the process is conceptualized as a two-way street, with the view that investigations of pathological conditions—sometimes referred to as *adaptational failures* in DP conceptualizations (e.g., Sroufe, 1997)—can and should provide a unique perspective on normal developmental mechanisms. In other words, it is posited explicitly that the study of disrupted developmental progressions can facilitate our understanding of what is normative.

This core tenet of DP—that mutual interplay between the study of normality and pathology, along with the perspective that progress in each domain depends on progress in the other—is now widespread. One of the best examples comes from neurology, which has a long tradition of utilizing the study of disrupted neural systems for enhancing understanding of healthy brain functioning and vice versa. For instance, "split-brain" patients (those who have had their cerebral hemispheres separated to provide relief from intractable seizures) provide unprecedented insights into normative brain processes and into the separable functions subserved by the right versus left hemispheres. This separation of functions becomes particularly evident with the severing of the large, interconnective structure known as the *corpus callosum* (see discussion in Gazzaniga, Ivry, & Mangun, 1998). Such induced hemispheric separation throws into sharp relief the typical interhemispheric communication and collaboration that takes place. Other neuroscience examples abound (see Cicchetti & Curtis, 2006; for a specific example, the study of phenylketonuria, or PKU, has implications for elaborating the normative development of executive functions; Diamond, Prevor, Callender, & Druin, 1997).

But how accurate is this perspective for DP? In other words, outside of neurological formulations, can investigations of pathology inform normal development? To reiterate, it is now commonly accepted that the more we know about basic emotion, cognition, attention, memory, social awareness, self-regulation, and the like, the greater the benefit for investigations

of psychopathology. 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 as well. Indeed, nearly all forms of mental pathology appear consistent with a quantitative, dimensional perspective (Beauchaine, 2003), emphasizing the need for flow of information from normal developmental pathways to pathological functioning.

Yet regarding the other direction, what has been learned about normal developmental processes from studies of child and adolescent psychopathology? I have pondered this question for some time, prompted by a probing inquiry during a colloquium discussion from my eminent Berkeley colleague Alison Gopnik. My initial take was that we have *not* gained the kinds of dramatic insights about typical psychological development from studies of child and adolescent psychopathology that have been realized in neurology. Part of the reason is that pathological functioning is almost always multifaceted and complex, which makes it quite difficult to pinpoint areas of specific dysfunction that could inform how normal development occurs in their absence. In other words, there are few equivalents to the surgical procedures of creating lesions in certain brain tracts or to single-gene forms of pathology such as PKU.

Yet consider the work on Autism by Baron-Cohen (2000; see also Baron-Cohen, Leslie, & Frith, 1985, and the review in Dawson & Toth, 2006). Relevant findings suggest that the lack of social connectedness experienced by individuals with Autism may relate to a failure in attainment of a basic *theory of mind*, which deals with the developing realization that other humans have mental states that differ from one's own. Most normal 4- and 5-year-olds can master theory-of-mind tests, suggesting that basic social understanding is predicated on a domain-specific cognitive module that, once operative, occurs almost automatically. On the other hand, a high percentage of youth with Autistic Disorder, even those with high levels of intellectual functioning, do not "pass" such psychological tests, revealing that they have not come to the normally automatic realization that fellow humans have different minds and different psychological perspectives from their own.

Intriguingly, however, a number of individuals with high-functioning Autism can eventually learn to pass the kinds of experimental tests used to test for theory of mind. Through effortful processing, they can and do deduce that other children and adults have a different understanding than they do. Yet this theory-of-mind ability does not mean that their social interactions automatically become smooth and effortless. Indeed, the laborious kinds of calculations and inferences made by people with high-functioning Autism to understand interpersonal dynamics are not usually

accompanied by smooth, effortless social interactions (e.g., Grandin, 2006). A key implication is that "normal" social-cognitive and social functioning is highly automatic and intuitive, qualitatively distinct from the ability to deduce social situations analytically in Autism—which is time consuming, not perceived as very skillful by peers, and probably quite different from the way the process works in typically developing individuals. Thus, disruptions in social cognition and social performance by persons with Autism may help to clarify the automatic and highly developed nature of the social cognitions and processes that underlie skilled interpersonal performance in normal development.

Another example pertains to work on the reward sensitivity of individuals with ADHD (e.g., Sagvolden, Johansen, Aase, & Russell, 2005). Here, considerable evidence reveals that, in people with this condition, withholding of rewards leads to rather sudden decrements in task performance, presumably related to a dopaminergically mediated problem with responding during extinction. In other words, ADHD is associated with large performance decrements when rewards are suddenly stopped. This insight may help to understand the mechanisms—largely mediated by subcortical, dopaminergic brain structures—by which typically developing individuals can maintain behavior during extinction, with mutual enhancement of the understanding of basic developmental processes and mechanisms underlying dysregulated attention and impulse control.

A third instance, noted extremely briefly, pertains to the horrific "experiments of nature" that occur when infants and toddlers are subjected to brutal institutionalization and lack of human contact during the earliest years of development (for review, see O'Connor, 2006). Intensive study of this topic has revealed essential information about rates of recovery during placement into stable homes, implications for attachment theory, the development of specific symptom patterns (e.g., inattention and overactivity as opposed to aggression; see Kreppner, O'Connor, Rutter, & the English and Romanian Adoptees Study Team, 2001), and the presence of social and cognitive "catch-up"—all of which are extremely informative about the normal-range development of secure relationships, emotional and behavioral functioning, and cognitive performance. I urge readers to seek other parallels regarding the ways in which knowledge about pathological functioning might elucidate normative processes.

Developmental Continuities and Discontinuities

With this principle, it is commonly asserted that DP models must emphasize both continuous and discontinuous processes at work in the development of pathology. What precisely does this mean? Taking the 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, the 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 mean that the precise forms of externalizing, antisocial behavior remain constant? Clearly not, given that those children with extremes of temper tantrums and defiance during the toddler and preschool years are not especially likely to exhibit high rates of tantrums during adolescence. Rather, they have a high likelihood of displaying physical aggression in grade school, covert antisocial behaviors in preadolescence, and various forms of delinquency by their teen years, followed by adult manifestations of antisocial behavior after adolescence (e.g., Moffitt, 1993). In short, continuities exist, but these are *heterotypic* in nature, as the actual form of the underlying antisocial trait changes form with development.

Another important consideration is that patterns of continuity may differ considerably across separable subgroups 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 may well change with development. In addition, a large subset does not display major externalizing problems in childhood but instead shows a sharp increase with adolescence (for a review, see Moffitt, 2006). Understanding such continuities and discontinuities in the form of homogeneous subgroups is likely to yield greater understanding than mere 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).

Multiple Levels of Analysis

As noted previously, extensive coverage of this topic is found in Chapter 2. In short, 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 the mechanisms that underlie the development of adjustment and maladjust-

ment. The essential task for the next generations of DP investigators is to link events at the level of the gene (e.g., genetic polymorphisms; transcription and translation) to neurotransmission and neuroanatomical development, and subsequently 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 the 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. Overall, progress in understanding pathological behavior will require multidisciplinary efforts in which investigators ranging from geneticists and biochemists, scientists focusing on individual pathology, experts on family and neighborhood processes, examiners of clinical service systems, and public health officials 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.

Risk and Protective Factors

The 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 (see Hinshaw, 2007, Chapter 1). Indeed, it is clear that biological vulnerabilities, psychological handicaps, environmental potentiators, and cultural-level norms all play a major role in defining and understanding behavioral manifestations that are considered abnormal and/or pathological in a particular social context. Risk factors (and constitutional vulnerabilities) are those antecedent variables that predict such dysfunction, and the ultimate goal is to discover those risk variables that 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, and risk factors are not inevitable predictors. For most individuals with diagnosable forms of psychopathology, symptoms and impairments tend to 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, 2006, 2007).

Furthermore, and crucially, not all individuals who experience risk factors for disorder develop subsequent pathology. *Resilience* is the term often used to define unexpectedly good outcomes, or competence, despite the presence of adversity or risk (Luthar, 2006; Luthar, Cicchetti, & Becker, 2000; for a major research example, see Werner & Smith, 1982). 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 centrally involved in the search for what have been called *protective factors*—those variables and processes that mitigate risk and promote more successful outcomes than would be expected in the presence of risk factors. Controversy surrounds the construct of resilience, the nature of protective factors, and the definitions of competent functioning (see Masten, Burt, & Coatsworth, 2006). In fact, 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 born into poverty fare well in adolescence and adulthood, why some individuals with genetic 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 general inseparability of health and pathology. Competence can shed light on the pathways that deflect away from pathology and, in so doing, may pro-

vide otherwise hidden insights into necessary developmental components of adjustment versus maladjustment (Luthar, 2006; Masten et al., 2006).

Reciprocal, Transactional Models

Linear models of causation, in which static psychological variables are assumed to respond in invariant ways to the influence of risk or protective factors, are not adequate to the task of explaining psychopathology and its development (see detailed explication in Richters, 1997, who highlights that very different 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 individual development of the child. Such mutually interactive processes themselves propel themselves over time, leading to what are termed *transactional models.* Furthermore, some developmental processes appear to operate via cascading, escalating chains (Masten et al., 2006), whereas others may, as just noted, be dampened or altered by mediating, protective factors. Dynamic systems models are clearly needed to help explicate core developmental phenomena (see Granic & Hollenstein, 2006).

To be specific: It is now well known that a great many cognitive and personality outcomes are at least moderately heritable, meaning that genetic factors explain a sizable proportion of individual differences in the trait, attribute, or disorder in question. But via gene–environment correlations, environments (genetically associated with the trait in question) may amplify the expression of the trait, and individuals may seek or evoke environmental responses that further promote the trait's unfolding. Furthermore, as noted explicitly in the chapter of Beauchaine and Neuhaus (this volume, Chapter 5), early maturing brain regions that give rise to expression of key emotional and behavioral characteristics may influence the developmental maturation of other, later-maturing regions; environmental events and factors may actually aid in the "turning on" of genes that further reinforce similar neural and behavioral actions. In addition, certain genotypes may become expressed only in the context of certain environmental factors, signifying the operation of gene–environment interactions (Rutter, Moffitt, & Caspi, 2006; see also Chapter 2 for further elucidation). Finally, processes of development may operate in highly nonlinear ways, requiring a new set of tools and conceptual models for understanding change processes (Granic & Hollenstein, 2006). Sensitive data-analytic strategies and innovative research designs are crucial tools for fostering greater understanding of such nonlinear phenomena.

Context Matters

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 genetic effects at one point in human evolutionary history may be maladaptive in current times, given major environmental and cultural changes that render certain genetically mediated traits far less advantageous than previously (e.g., the storage of fat in times of uncertain meals and sudden need for survival-related activity; the presence of undue anxiety in relation to certain feared stimuli when conditions have markedly changed with respect to indoor, sedentary lifestyles). There are few absolutes in terms of behavior patterns that are inherently maladaptive or risk factors that inevitably yield dysfunction; the cultural setting and context are all-important for defining 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) have shown, for example, that harsh, authoritarian parenting predicts antisocial behavior in white, middle-class children but not necessarily in African-American families. Many forms of mental disorder are present at roughly equivalent rates across multiple cultures, revealing key evidence for universality; but the effects of risk or protective factors often differ markedly depending on their developmental timing, the family and social contexts in which they are experienced by the developing child, and the niche or *space* that exists in a given culture for their expression and resolution (see, for example, Serafica & Vargas, 2006). In short, the DP perspective tells us clearly that setting and context are all-important (Cicchetti, 2006).

Equifinality and Multifinality

As suggested earlier, there is overwhelming evidence that multiple pathways exist to both health and illness. It is a myth to think that all individuals displaying symptoms of a given mental disorder "got there" through similar mechanisms and processes. We know, for instance, that the broad syndrome of depression may emanate from heritable risks in some cases, from severe life losses and stressors in others, from the interaction of the two in a great many more, and from other early or contemporaneous risk factors in still others. ADHD is substantially heritable, but the constituent symptomatology may also emerge from low birthweight, severe early deprivation, or effects of teratogens like nicotine or tobacco in utero (Barkley, 2006; Gatzke-Kopp & Shannon, this volume). In short, *equifinality*—

the presence of multiple pathways leading to apparently similar outcome states—clearly operates with regard to the major entitites of mental disturbance that we now recognize (see Cicchetti & Rogosch, 1996).

In parallel, although inhibited temperament in infancy and toddlerhood is clearly predictive of risk for subsequent social anxiety, there is far from a 1:1 correspondence. Other risk and protective factors, including the presence of childrearing environments that gently but firmly "push" the child out of inhibited, withdrawn behavior patterns, may deflect any inevitable association between early inhibition and later internalizing conditions (see Kagan, 1997; Kagan, this volume, Chapter 6). Similarly, child maltreatment does not lead to a uniform set of outcomes but may instead yield a range of subsequent behavioral and emotional patterns even when the type or severity of abuse is held constant (Cicchetti & Valentino, 2006). Hence, through processes of *multifinality*, complex causal chains of influence render the operation of early risk factors as probabilistic rather than deterministic.

Thus, although the presence of multiple risk factors is clearly linked to lowered chances of recovery, the DP model emphasizes malleability, flexibility, and plasticity in development. The core issues in this regard involve, first, the attempt to disentangle the many potential developmental influences that may tip the individual toward health and competence versus disorder and failure; and second, the necessity of incorporating what is termed *probabilistic epigenesis* (Gottlieb & Willoughby, 2006) into causal models. This term means that genes do not provide a one-way causal influence on neural structures and behavior, largely because of highly interactive, reciprocal, and bidirectional influences with epigenetic factors (e.g., other brain structures and products, behavioral patterns, environmental influences). Here we see that several DP principles—for example, nonlinear causal patterns, reciprocal/transactional models, and the importance of context—are closely linked together. In an elegant musical metaphor, Boyce (2006) presents the notion of *symphonic causation* to illustrate the confluence of biological and contextual influences on development.

Psychobiological Principles and Discoveries

The genomic era is upon us, and advances in brain imaging research have made the developing brain far more accessible to scientific view than ever before (see Giedd et al., 2006; Rende & Waldman, 2006). Although it is mistaken, as noted previously, to give primacy to any given level of analysis in a DP perspective—brain, contextual, or other—we have asked contributors to pay particular attention to psychobiological factors and processes in their coverage.

Part of the reason is historical: Family systemic and environmental views

dominated the field for much of the twentieth century, and recent work on a range of psychobiological processes is not always featured in reviews and texts (see Boyce, 2006). Another issue has to do with explanatory power, as we now know that without understanding the potential effects of genes, physiological processes, and biological risk factors on psychopathology we have little hope of understanding the most severe forms of disorder. Because the brain is remarkably plastic and because context influences biological unfolding, we have—as noted throughout this chapter—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 a half-century ago—e.g., the blaming of Autism on emotional refrigeration by parents or Schizophrenia by schizophregenic mothers.

Summary

All of the previous points converge on the core theme that the development of psychopathological functioning is multidetermined, complex, interactive, transactional, and in most instances nonlinear. It would be hard to imagine otherwise, given the staggering complexity of the brain and the myriad influences, ranging from the microsocial to the macrosocial, that impinge on the developing infant, toddler, and child. 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; those who are fascinated with how much remains to be learned about antecedent conditions and maintaining factors; those who are possessed by an intense "need to know" about the underlying mechanisms of eating disorders, conduct disturbance, substance abuse, anxiety and depression, Bipolar Disorder, Pervasive Developmental Disorders, ADHD, and other child and adolescent conditions; and 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 often required 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 is vast.

A GUIDE TO THE BOOK'S CONTENTS

Immediately following this introduction, Dante Cicchetti (Chapter 2) provides a multiple-levels-of-analysis framework for DP. This chapter is a true companion to the present one, as the multilevel framework is one of the central tenets in the field of developmental psychopathology. Linking genes to phenotypes—and understanding the key mediating steps of the process, which include the role of context and environment in shaping such linkages—is essential to gain understanding of both disordered functioning and strength, competency, and resilience. And in Chapter 3, Ted Beauchaine, Stephen Hinshaw, and Lisa Gatzke-Kopp discuss a number of core themes related to genetic factors and mechanisms underlying psychopathology, with a key focus on how these genetic factors interact with environments to increase risk for psychopathology. This chapter is explicit that it is no longer sensible to speak of nature versus nurture but rather that an integrated, dynamic conception of biology-environment interplay is the current paradigm of interest.

Part I of the book features research on risk factors, those antecedent variables that help to set in motion the chains, pathways, or cascades of processes that emanate in psychopathological functioning. Bruce Perry (Chapter 4) elucidates the multiple ways that child abusive experiences set the stage for later maladaptation—although as emphasized, the risk is not absolute. Indeed, the concept of multifinality emphasizes that some children with high levels of maltreatment may nonetheless show resilient behavior. Next, Ted Beauchaine and Emily Neuhaus (Chapter 5) provide a richly contextualized perspective on the construct of impulsivity, alternately termed *disinhibition*. They highlight psychobiological, environmental, and interactive mechanisms that yield impulsive behavioral styles, which incur great risk for escalating impairments. Chapter 6 (Jerome Kagan) is a witty and engaging account of behavioral inhibition and its consequences for a range of internalizing behavior patterns. Emphasizing genetic risk factors, environmental potentiation of such risk, and the fascinating picture of heterotypic continuity across development in the face of inhibited temperament, Kagan's work strongly accentuates core DP principles and criticizes biological/genetic reductionism in accounting for all outcomes related to inhibited temperament. In Chapter 7, Susanna Fryer, Nicole

Crocker, and Sarah Mattson provide up-to-date information on teratogens—substances transmitted from a pregnant mother to her embryo or fetus—and their potential to promote dysfunction in the developing organism. This work emphasizes, once again, the probabilistic rather than the deterministic nature of this risk factor: maladaptive outcomes are not all or none in the face of this set of risk factors. Lisa Gatzke-Kopp and Kate Shannon write about various forms of brain injury (Chapter 8), with emphasis on risk for dysfunction and potential recovery. Of necessity, this chapter covers the specific "pathogens" of physical trauma and hypoxia, the construct of neural plasticity, and the potential for genetic factors to interact with trauma in the development of maladaptation. Chapter 9, written by Jim Coan and John Allen, features affective style, the patterns of emotion-related responding that may set the stage for maladaptive development. This dense, rich chapter challenges readers to consider levels of analysis not often dealt with in accounts of psychopathology. In Chapter 10, Pamela Cole and Sarah Hall discuss the "hot" topic of emotion regulation and, particularly, dysregulation, with its implications for disordered outcomes. In their view, emotions are inherently activating and organizing, but if attempts at their regulation are insufficient, if emotions are displayed in context-inappropriate fashion, or if emotions change either too quickly or too slowly, implications for dysfunction become apparent.

In Part II, the emphasis is on the externalizing (or disruptive) behavior disorders, those in which the salient symptomatology impinges on the personal space, rights, and integrity of others. Chapter 11, written by Joel Nigg and Molly Nikolas, features coverage of the core processes of inattention, impulsivity, and hyperactivity that characterize ADHD, and provides an intellectually rich account of key psychological and psychobiological processes and of both genetic and often-neglected environmental risk factors for this condition. Explicitly embracing the core tenets of DP, they emphasize the great need for integrated theoretical models in future work on this prevalent and distressing disorder. Chapter 12, by Benjamin Lahey, presents a comprehensive, multilevel, integrative view of the development of aggressive and antisocial behavior, with specific reference to the diagnostic categories of Oppositional Defiant Disorder and Conduct Disorder. A key issue for this domain of inquiry is the sheer number of pertinent risk factors, spanning intraindividual, familial, and wider contextual variables; Lahey's integrative account is a welcome antidote to the often-overwhelming feel of literature in this area. Kristi Hiatt and Tom Dishion, in Chapter 13, discuss principles and concepts underlying the development of antisocial personality, taking a lifespan perspective featuring interplay among the myriad risk factors (individual, parental, peer-related, neighborhood-level) that together yield adult antisocial behavior and, in

some cases, psychopathy. It provides a fine counterpoint and complement to Chapter 13. Chapter 14, by Sandy Brown, discusses developmental trajectories of alcohol and substance problems, emphasizing inherited, cognitive, and social risk and protection, providing an elegantly integrated model of the development of substance use disorders in adolescence.

Next, Part III features internalizing conditions. Chapter 15, by Carl Weems and Wendy Silverman, covers the range of anxiety disorders, providing an integrative perspective on their origins and maintenance. Building from dimensional conceptions of anxiety and fear, they emphasize genetic and psychophysiological factors, social learning and cognitive processes, social and interpersonal variables, and interactions across these levels. Daniel Klein, Dana Torpey, Sara Bufferd, and Margaret Dyson (Chapter 16) review comprehensively known risk factors for and developmental issues related to child and adolescent depression. Covering the wide-ranging precursors to depressive outcomes (e.g., maladaptive cognitive patterns, stress reactivity, genetic vulnerability, disrupted parent-child relationships, to name some of the more salient), they provide an integrative developmental model. In Chapter 17 Sheila Crowell, Ted Beauchaine, and Mark Lenzenweger provide essential commentary on Borderline Personality Disorder, self-injurious behavior, and their developmental antecedents. With the understanding that research in this domain is rather limited, these authors emphasize potential genetic and neural risk factors, the clear role of parenting disruptions, and their interactions, concluding that Borderline Personality Disorder and intentional self-injury constitute extremes of impulse control problems, particularly in relation to severe stressors. Note that the placement of this chapter in Part III is somewhat arbitrary, given the admixture of dysphoric, internalizing features and disinhibited, externalizing symptoms involved in these behavior patterns.

Finally, the coverage in Part IV focuses on several additional, extremely important disorders. Bipolar Disorder—also representing an extreme blend of externalizing and internalizing features—is the topic of Chapter 18, written by Joseph Blader and Gabrielle Carlson. This detailed chapter lays out the complex interactions and transactions (and strong heritability) of Bipolar Disorder, dealing directly with core developmental issues related to assessment, diagnosis, and symptom presentation. Chapter 19 features Autism spectrum conditions; its authors are Geraldine Dawson and Susan Faja. Major psychobiological theorizing is occurring in relation to Autism spectrum disorders; this chapter presents a balanced perspective on the most promising of the latest conceptual models. Schizophrenia can and does exist in children, with major increases in prevalence throughout adolescence. In Chapter 20, Robert Asarnow and Claudia Kernan tackle the important developmental issues related to, and the strong psychobiologic

roots of, schizophrenia-spectrum conditions, emphasizing developmental continuities and discontinuities. Finally, Chapter 21, authored by Eric Stice and Cindy Bulik, covers the eating disorders of Anorexia Nervosa, Bulimia Nervosa, and Binge Eating Disorder, once again featuring complex, interactive processes spanning psychobiological and psychosocial factors in such conditions while providing strong emphasis on a developmental neuroscience perspective.

The study of atypical development is fascinating, complex, and clinically relevant, with the potential for elucidating the processes by which normal development occurs. Progress in this field is accumulating rapidly, but there is still a huge distance to travel, given the almost unfathomable complexity of developmental processes and pathways that exist.

Recall the discussion, earlier in this chapter, of the prenatal rate of development of neurons. How does this unfathomable number of neurons, and the incalculable number of synapses that are shaped before and well after birth, produce consciousness, attentional deployment, memory, emotion regulation, and both healthy and atypical developmental patterns? Which models do we still need to construct if we are to begin to answer such questions with accuracy? These are among the most important issues in all of science, and the amount that needs to be learned is staggering. Yet with the tools of DP—and the new techniques and conceptual models that are necessary to develop to keep up the momentum—additional progress is in sight.

We wish you well as you begin your journey into the many aspects of developmental psychopathology and into the key risk factors for and manifestations of child and adolescent mental disorders that are presented in the following chapters. Smooth, packaged, easily digestible accounts are not found within these pages, as the kinds of reciprocal, interactive, cascading, and integrative models needed to facilitate further understanding are far from simple or linear. Yet, for the next generation of investigators, clinicians, and policy-makers—who, we hope, will carry with them an appreciation of the systemic models and transactional processes embedded in DP conceptualizations—there can be no more fascinating venture.

REFERENCES

Achenbach, T. M. (1974). *Developmental psychopathology*. New York: Ronald Press.

Barkley, R. A. (2006). *Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed.). New York: Guilford.

Baron-Cohen, S. (2000). Theory of mind and autism: A fifteen-year review. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.). *Understanding other minds: Perspectives from developmental cognitive neuroscience* (2nd ed., pp. 3–20). Oxford, UK: Oxford University Press.

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition, 21,* 37–46.

Beauchaine, T. P. (2003). Taxometrics and developmental psychopathology. *Development and Psychopathology, 15,* 501–527.

Bergman, L. R., von Eye, A., & Magnusson, D. (2006). Person-oriented research strategies in developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 1: Theory and method* (2nd ed., pp. 850–888). New York: Wiley.

Boyce, T. (2006). Symphonic causation and the origins of childhood psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 2: Developmental neuroscience* (2nd ed., pp. 797–817). New York: Wiley.

Cicchetti, D. (2006). Developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 1: Theory and method* (2nd ed., pp. 1–23). New York: Wiley.

Cicchetti, D., & Cohen, D. J. (Eds.). (2006). *Developmental psychopathology.* New York: Wiley.

Cicchetti, D., & Curtis, W. J. (2006). The developing brain and neural plasticity: Implications for normality, psychopathology, and resilience. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 2: Developmental neuroscience* (2nd ed., pp. 1–64). New York: Wiley.

Cicchetti, D., & Dawson, G. (Eds.). (2002). Multiple levels of analysis. *Development and psychopathology, 14,* Special Issue.

Cicchetti, D., & Rogosch, F. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology, 8,* 597–600.

Cicchetti, D., & Valentino, K. (2006). An ecological-transactional perspective on child maltreatment: Failure of the average expectable environment and its influence on child development. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation,* (2nd ed., pp. 317–357). New York: Wiley.

Dawson, G., & Toth, K. (2006). Autism spectrum disorders. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 317–357). New York: Wiley.

Deater-Deckard, K., & Dodge, K. A. (1997). Externalizing behavior problems and discipline revisited: Nonlinear effects and variation by culture, context, and gender. *Psychological Inquiry, 8,* 161–175.

Diamond, A., Prevor, M. B., Callender, G., & Druin, D. P. (1997). Prefrontal cortex cognitive deficits in children treated early and continuously for PKU. *Monographs of the Society for Research in Child Development, 62* (Serial No. 4).

Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (1998). *Cognitive neuroscience: The biology of the mind.* New York: Norton.

Giedd, J. N., Shaw, P., Wallace, G., Gogtay, N., & Lenroot, R. K. (2006). Anatomic brain imaging studies of normal and abnormal brain development in children

and adolescents. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 2: Developmental neuroscience* (2nd ed., pp. 127–196). New York: Wiley.

Gottlieb, G., & Willoughby, M. T. (2006). Probabilistic epigenesis of psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 1: Theory and method* (2nd ed., pp. 673–700). New York: Wiley.

Granic, I., & Hollenstein, T. (2006). A survey of dynamics systems methods for developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 1: Theory and method* (2nd ed., pp. 889–930). New York: Wiley.

Grandin, T. (2006). *Thinking in pictures: And other reports from my life with Autism* (2nd ed.). New York: Vintage.

Hinshaw, S. P. (2002). Intervention research, theoretical mechanisms, and causal processes related to externalizing behavior patterns. *Development and Psychopathology, 14,* 789–818.

Hinshaw, S. P. (2006). Stigma and mental illness: Developmental issues and future prospects. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 317–357). New York: Wiley.

Hinshaw, S. P. (2007). *The mark of shame: Stigma of mental illness and an agenda for change.* New York: Oxford University Press.

Howe, G. W., Reiss, D., & Yuh, J. (2002). Can prevention trials test theories of etiology? *Development and Psychopathology, 14,* 673–694.

Kagan, J. (1997). Temperament and the reactions to unfamiliarity. *Child Development, 68,* 139–143.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey replication. *Archives of General Psychiatry, 62,* 593–602.

Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry, 54,* 337–343.

Kraemer, H. C., Stice, E., Kazdin, A., Offord, D., & Kupfer, D. (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry, 158,* 848–856.

Kreppner, J. M., O'Connor, T. G., Rutter, M., & the English and Romanian Adoptees Study Team. (2001). Can inattention/overactivity be a deprivation disorder? *Journal of Abnormal Child Psychology, 29,* 513–528.

Luthar, S. S. (2006). Resilience in development: A synthesis of research across five decades. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 739–795). New York: Wiley.

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development, 71,* 543–562.

Mash, E. J., & Barkley, R. A. (Eds.). (2003). *Child psychopathology* (2nd ed.). New York: Guilford.

Mash, E. J., & Dozois, D. J. A. (2003). Child psychopathology: A developmental-systems perspective. In E. J. Mash & R. A. Barkley (Eds.), *Child psychopathology* (2nd ed., pp. 3–71). New York: Guilford.

Masten, A. S., Burt, K. B., & Coatsworth, J. D. (2006). Competence and psycho-

pathology in development. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 696–738). New York: Wiley.

Moffitt, T. E. (1993). "Life-course-persistent" and "adolescence-limited" antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674–701.

Moffitt, T. E. (2006). Life course persistent versus adolescence limited antisocial behavior. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 570–598). New York: Wiley.

Muthén, B. O., Brown, C. H., Masyn, K., Jo, B., Khoo, S. T., Yang, C. C., et al. (2002). General growth mixture modeling for randomized prevention trials. *Biostatistics, 3,* 459–475.

Nigg, J. T. (2006). *What causes ADHD? Understanding what goes wrong and why.* New York: Guilford Press.

Nigg, J. T., Hinshaw, S. P., & Huang-Pollack, C. (2006). Disorders of attention and impulse regulation. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 358–403). New York: Wiley.

O'Connor, T. G. (2006). The persisting effects of early experiences on psychological development. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 202–234). New York: Wiley.

Raine, A., Brennan, P., & Mednick, S. A. (1997). Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: Specificity to serious, early-onset violence. *American Journal of Psychiatry, 154,* 1265–1271.

Rende, R., & Waldman, I. (2006). Behavioral and molecular genetics and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 2: Developmental neuroscience* (2nd ed., pp. 427–464). New York: Wiley.

Richters, J. E. (1997). The Hubble Hypothesis and the developmentalist's dilemma. *Development and Psychopathology, 9,* 193–229.

Romer, D., & Walker, E. F. (Eds.). (2007). *Adolescent psychopathology and the developing brain.* New York: Oxford University Press.

Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry, 47,* 226–261.

Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. *Psychological Bulletin, 127,* 291–324.

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

Sagvolden, T., Johansen, E. B., Aase, H., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined types. *Behavioral and Brain Sciences, 28,* 397–419.

Sameroff, A. J., Lewis, M., & Miller, S. M. (Eds.). (2000). Handbook of developmental psychopathology (2nd ed.). New York: Kluwer Academic/Plenum.

Serafica, F. C., & Vargas, L. A. (2006). Cultural diversity in the development of child psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 1: Theory and method* (2nd ed., pp. 588–626). New York: Wiley.

Silverman, W., & Hinshaw, S. P. (2008). Evidence-based psychosocial treatments for children and adolescents. The ten year update. *Journal of Clinical Child and Adolescent Psychology, 37(2), Special Issue.*

Sroufe, L. A. (1997). Psychopathology as an outcome of development. *Development and Psychopathology, 9,* 251–268.

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

Werner, E., & Smith, R. (1982). *Vulnerable but invincible: A study of resilient children.* New York: McGraw-Hill.

Wolfe, D. A., & Mash, E. J. (Eds.) (2006). *Behavioral and emotional disorders in adolescents.* New York: Guilford.