CHAPTER 1

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

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

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

WHY DEVELOPMENTAL PSYCHOPATHOLOGY?

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

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

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

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

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

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

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

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

OVERVIEW OF APPROACH

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

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

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

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

DP CONCEPTS AND PRINCIPLES

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

Three related principles bear emphasis. The first is that multiple pathways
to pathology exist. Indeed, disparate routes may lead to a common condition
or outcome, exemplifying the construct of equifinality. For example, aggressive
behavior (or, diagnostically speaking, “conduct disorder”) can result from physical
abuse (Chapter 5), from a heritable tendency toward disinhibition (Chapter 3),
from injury to the frontal lobes (Chapter 10), from coercive parenting interchanges
with the developing child (Chapter 14), from prenatal and perinatal risk factors
acting in concert with early experiences of insecure attachment or parental rejection
(Chapter 9), or from different combinations of these vulnerabilities and risk factors
(e.g., Jaffee, Strait, & Odgers, 2012; Raine, Brennan, & Mednick, 1997; Tremblay, 2010).
In other words, separate—and in many cases interacting—causal influences can yield
similar clinical endstates. In addition, the concept of multifinality applies when a
given risk factor or initial state yields disparate outcomes across development. For
instance, abuse may or may not lead to severe maladaptation, depending on a host of
intervening factors; extremes of inhibited temperament may produce shyness and social withdrawal, but other, healthier outcomes are also possible, depending on the presence or absence of additional risk or protective factors (see Cicchetti & Rogosch, 1996). Both equifinality and multifinality imply that linear models of association and simplistic categorical conceptions of disorder are incapable of facilitating full understanding of child and adolescent psychopathology. Indeed, such simplistic models may actually be misleading.

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

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

As noted in the introductory chapter to the first edition (Hinshaw, 2008), a basic mathematical calculation may help to elucidate the underlying complexities here: Adults have approximately 100 billion neurons in their brains; children are born with even higher numbers, perhaps double that figure. Indeed a major developmental “task” over the earliest years of postnatal development is the pruning and migration of such neurons into a working, functional brain. But what is the rate of neural development during the 40 weeks of human gestation? To figure this out, one must divide 200 billion (a fair estimate of the number of neurons with which an infant is born) by the number of seconds in 40 weeks. The result—of dividing the numerator of 2 times 10 to the 11th power (the number of neurons) by the denominator of 2,419,2 times 10 to the 7th power (the number of seconds)—is the astonishing figure that, on average, the embryo and fetus are producing approximately 8,000 new neurons every second throughout the entire course of prenatal development. This average is not constant, of course, given that the neural tube and brain do not even form for some weeks. Thus, in some crucial periods, this figure is even higher (see Giedd, Shaw, Wallace, Gogtay, & Lenroot, 2006, for additional information on the
precise timing of neural development across pregnancy and childhood). Rates of connectivity in the developing brain yield numbers that are exponentially higher. As the cortex matures, revealing characteristic patterns of thickening and thinning, and as cortical neurons form rich and lasting connections with other brain regions, the number of synaptic connections goes into the trillions and beyond.

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

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

Normal and Atypical Development Are Mutually Informative

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

Yet the process is conceptualized as a two-way street, with the corresponding view that investigations of pathological conditions—sometimes referred to as adaptational failures in DP terms (see Sroufe, 1997)—can and should provide a unique perspective
on normal developmental mechanisms. In other words, the study of disrupted developmental progressions illuminates our understanding of what is normative.

Overall, this core tenet of DP—of the mutual interplay between the study of normality and pathology, along with the perspective that progress in each domain is dependent on progress in the other—is now widely held. Neurology abounds with relevant examples. For example, there is a long tradition utilizing the study of disrupted neural systems to enhance understanding of healthy brain functioning and vice versa. “Split-brain” patients (those who have had their cerebral hemispheres separated to provide relief from specific neurological disorders) provide unprecedented insights into normative brain processes, such that separable functions and even “personalities” subserved by the right versus left hemispheres become evident as a function of the pathology and resultant surgery. In parallel, famous case studies such as HM, in which key brain structures/regions have been surgically removed (in his case the hippocampus), greatly facilitate knowledge about human memory systems (see Gazzaniga, Ivry, & Mangun, 2009). Cicchetti and Curtis (2006) provide lucid detail on neuroscience-related approaches. For a specific example, the study of phenylketonuria (PKU) has implications for elaborating the normative development of executive functions (Diamond, Prevor, Callender, & Druin, 1997).

How might this perspective inform our understanding of psychological or psychiatric disorders that are the core subject matter of DP? In other words, beyond neurological conditions and formulations per se, can investigations of pathology inform normal development? Once again, it is now commonly accepted that the more we know about basic emotion, cognition, attention, memory, social awareness, self-regulation, and the like, the more investigations of psychopathology can benefit. Almost no form of mental disorder constitutes a clearly demarcated, qualitatively distinct category or taxon, meaning that processes applying to individuals near the center of the bell curve are likely to apply to those further out on the continuum as well. Nearly all forms of mental pathology appear consistent with a quantitative, dimensional perspective (Beauchaine, 2003), emphasizing the need for flow of information from normal developmental pathways to pathological functioning.

But what about the other direction? Specifically, what has been learned about normal developmental processes from studies of child and adolescent psychopathology? At first glance, the situation doesn’t seem to be as heuristic as that in neurology; it may be that we have not gained the kinds of dramatic insights about typical psychological development from studies of child and adolescent psychopathology that have been realized in “harder” scientific endeavors. In other words, the complexity of mental disorders may limit parallels to more simply caused neurological conditions. In short, there are few behavioral and emotional equivalents to the surgical procedures of creating lesions in certain brain tracts or single-gene forms of pathology such as PKU.

Yet consider the work on autism by Baron-Cohen (2000). Relevant findings suggest that the lack of social connectedness experienced by individuals with autism may relate to a failure in attainment of a basic “theory of mind,” which deals with the developing realization that other humans have mental states that differ from one’s own. Most normal 4-year-olds can master theory-of-mind tests, suggesting that basic
social understanding is predicated on a domain-specific cognitive module that, once operative, occurs almost automatically. On the other hand, a high percentage of youth with autistic disorder, even those with high levels of intellectual functioning, do not “pass” such psychological tests, suggesting that they have not come to the core realization that fellow humans have different minds and different psychological perspectives from their own.

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

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

A third instance, noted briefly, pertains to the horrific “experiments of nature” that occur when infants and toddlers are subjected to brutal institutionalization and lack of human contact during the earliest years of development (for review, see O’Connor, 2006). The development of specific symptom patterns (e.g., inattention
statistical strategies (e.g., growth mixture modeling; group-based trajectory modeling) are increasingly used to facilitate the search for separable trajectories or classes defined on patterns of change of the relevant dependent variable, leading to major insights about predictors, moderators, and mediators and emphasizing the major heterogeneity in multiple aspects of psychopathological functioning (see Muthén & Muthén, 2000; Nagin & Odgers, 2010a, 2010b).

**Multiple Levels of Analysis**

The greatest potential for progress (and complexity) 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 underlying the development of adjustment and maladjustment. 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; epigenetic influences) to neurotransmission and neuroanatomical development, and subsequently into individual differences in temperament, social cognition, and emotional response patterns (for detailed discussion, see Cicchetti, 2008). 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, invoking the concept of epigenetic influences. Overall, progress in understanding pathological behavior will require multidisciplinary efforts in which investigators ranging from geneticists and biochemists, clinicians focusing on individual pathology, experts on family and neighborhood processes, investigators 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 focusing exclusively 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-multidisciplinary work, necessitating collaborations across traditional academic boundaries. Although such collaboration is not simple, these kinds of efforts are undoubtedly where the payoffs will lie (for a key example, see Caspi, Hariri, Holmes, Uher, & Moffitt, 2010).

**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 potentators, 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 variables that are both malleable and potentially causal of the disorder in question (Kraemer et al., 1997).

Yet disordered behavior is not uniform, and risk factors are not inevitable predictors. Being female is a protective factor against most forms of psychopathology in the first decade of life but serves as a risk factor for internalizing conditions during adolescence (e.g., Hinshaw, 2009). As noted earlier, maltreatment is sometimes but not always a risk factor for later pathology. Furthermore, for most individuals with diagnosable forms of mental disorder, 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).

Resilience is the term often used to define unexpectedly good outcomes, or competence, despite the presence of adversity or risk (Luthar, 2006; Sapienza & Masten, 2011). Indeed, the concept of multifinality, noted earlier, directly implies that, depending on a host of biological, environmental, and contextual factors, variegated outcomes will emanate from common risk factors, with the distinct possibility of 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. Yet controversy surrounds the construct of resilience, the nature of protective factors, and the nature of competent functioning (see Luthar & Brown, 2007; Masten, Burt, & Coatsworth, 2006). In fact, some have claimed that there is no need to invoke a set of special processes that are involved, 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). Even so, 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.

Indeed, recent advances in the study of resilience show that some aspects of resilient functioning have psychobiological and even genetic underpinnings and that a systemic, transactional, and even epigenetic model is needed to understand the multipronged nature of resilience-enhancing processes (see review in Sapienza & Masten, 2011). Furthermore, Luthar and Brown (2007) remind us that relationships are central to any conception of resilience, despite current work in psychobiological undercurrents. In short, gaining understanding of why some children born into poverty fare well in adolescence and adulthood, why some individuals with alleles that tend to confer risk for pathological outcomes do not evidence
and overactivity, as opposed to aggression; see Kreppner, O'Connor, Rutter, & the English and Romanian Adoptees Study Team, 2001), and the extent of social and cognitive “catch-up” following removal from the institutions, are extremely informative about normal-range development of secure relationships and cognitive performance. Such work has even incorporated experimental methods to understand whether in-home foster placements can mitigate the effects of early deprivation in terms of cognitive growth (Nelson et al., 2007). Indeed, for the previously institutionalized girls in this randomized trial, foster care placement led to improvements in girls’ internalizing behavior patterns, mediated by the gaining of attachment security via the change from institutional care to family placements (McLaughlin, Zeanah, Fox, & Nelson, 2012). Thus, even in a harshly abandoned and deprived sample, attachment processes are implicated in reductions of anxiety and depression. Mediators of competence in more normative samples are still open to exploration.

Developmental Continuities and Discontinuities

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

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

**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 Richters, 1997, who highlights that quite different explanatory systems are needed to deal with open systems such as individual people). Pathways from childhood to adolescent and adult functioning are marked by reciprocal patterns or chains, in which children influence parents, teachers, and peers, who shape the further individual development of the child. Such mutually interactive processes can themselves escalate over time, leading to what are termed *transactional models*. Furthermore, some developmental processes appear to operate via cascading, escalating chains (Masten et al., 2006; Masten & Cicchetti, 2010), whereas others may, as just noted, be dampened or altered by mediating, protective factors. Given the strong potential for nonlinear change in all of the above processes, dynamic systems models are 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 Chapter 3, 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, through epigenetic processes. In addition, certain genotypes may become expressed only in the context of certain environmental factors, signifying the operation of gene-environment interactions (Rutter et al., 2006). Finally, as just noted, 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.
THE IMPORTANCE OF CONTEXT

A key tenet of DP, directly related to the above points, 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 behaviors at one point in human evolutionary history may be maladaptive in current times, given the major environmental and cultural changes that render certain genetically mediated traits far less advantageous than previously. As just two examples, consider [1] the storage of fat in times of uncertain meals and sudden need for survival-related activity, and [2] 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 either behavior patterns that are inherently maladaptive or risk factors that inevitably yield dysfunction. Cultural settings and context are all-important for shaping and even defining 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. (On the other hand, parenting that crosses the line into abuse is uniformly harmful.) 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 may differ markedly depending on their developmental timing, the family and social context 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).

This discussion throws into sharp relief the surge of interest, in recent years, regarding the specific effects environments play, once biological vulnerabilities are taken into account. Rutter, Pickles, Murray, and Eaves (2001) provide a masterful account of how to understand specific, causal environmental forces and factors; Jaffee et al. (2012) provide an extremely useful guide to how nonexperimental designs and recent statistical advances can help facilitate just such understanding. As noted by Caspi et al. (2010), the leap in knowledge between environmental correlates and environmental “pathogens” (i.e., environmental factors that are causal) is a large one.

Along these lines, the process known as gene-environment interaction, discussed in more detail in Chapter 3 (and in the comprehensive volume of Dodge & Rutter, 2011), remains a hot topic. After groundbreaking research a decade ago on this topic (e.g., Caspi et al., 2003) revealing that genotypes may yield pathological outcomes only in certain environmental contexts, meta-analyses have challenged the size and strength of such findings (e.g., Risch et al., 2009). However, in an incisive rejoinder, Caspi et al. (2010) examine animal as well as human models of gene products and genetically mediated vulnerabilities, laying the groundwork for much-needed psychobiological pathways through which such moderated effects may occur. They
also make the key point that many large-sample investigations—which receive the most “weight” in meta-analytic efforts—tend to utilize inferior assessments of environmental influences, such as those that are self-reported and retrospective. More precise environmental measures, in smaller studies, are thus discounted. Finally, Caspi et al. (2010) highlight that the serotonin transporter gene, implicated in a host of gene-environment interaction findings, exerts its effects not by conferring risk for any particular form of psychopathology but by enhancing sensitivity to key aspects of the environment—revealing the core DP principle that “nature” and “nurture” cannot fundamentally be separated.

Importantly, current models are moving away from the idea that certain genetic configurations are inevitably “risk” factors for psychopathology. Some, in fact, may yield better-than-expected outcomes in optimal contexts. In other words, they may serve as plasticity factors as opposed to risk factors per se, yielding differential susceptibility to both positive and negative environmental settings (e.g., Belsky & Pluess, 2009).

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, for example, is substantially heritable but the constituent symptomatology may also emerge from low birthweight, severe early deprivation, effects of teratogens like nicotine or tobacco in utero, or even from what were formerly thought to be low levels of environmental lead (Barkley, 2006; Nigg, 2006). In short, equifinality—the presence of multiple pathways leading to apparently similar outcome states—clearly operates with regard to the major entities of mental disturbance that we now recognize (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 child-rearing 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, and Chapter 7). 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. In cases of pathology, the chain tends to intensify and widen, in a process termed developmental cascades (Masten & Cicchetti, 2010).
Thus, the DP model emphasizes malleability, flexibility, and plasticity in development, although the presence of multiple risk factors is clearly linked to lowered chances of recovery. 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, because of highly interactive, reciprocal, and bidirectional influences with epigenetic factors (e.g., other brain structures and products, behavioral patterns, and 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. 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 and clinical techniques have made the developing brain far more accessible to scientific view than ever before (e.g., Giedd et al., 2006; Rende & Waldman, 2006). Although it is mistaken, as noted earlier, to give primacy to any given level of analysis in a DP perspective—brain, contextual, or other (Cicchetti & Toth, 2009)—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 20th century, and recent work on a range of psychobiological processes is not always featured in reviews and texts (Boyce, 2006). Another issue pertains to explanatory power: 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 the primacy of single genes, the inevitable predictability of later functioning from early temperament, or the “placement” of psychopathology completely inside brightly colored brain images on journal or chapter pages are as short-sighted as the exclusively environmental accounts of psychopathology that dominated a half-century ago, such as the blaming of autism on emotional refrigeration by parents or schizophrenia by schizophrenic mothers.

**Summary**

Clearly, the development of psychopathological functioning is multidetermined, complex, interactive, transactional, and in many if not most instances nonlinear. It is nearly impossible 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 endeavor, perhaps even unfathomable. On the other hand, for those who are intrigued by the diverse clinical presentations of various pathological conditions in childhood and adolescence; who are fascinated with how much remains to be learned about antecedent conditions and maintaining factors; who are possessed by an intense “need to know” about the underlying mechanisms of the conditions discussed in this volume; and who realize the need to consider healthy outcomes and competence as well as maladaptation, the DP perspective is a vital 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 the potential of high yield for developmental science and for informing prevention and intervention efforts. The study of DP is ever expanding, engaging scientists from large numbers of disciplines and perspectives. Progress is emerging quickly, but the territory to explore is vast and the road ahead is long.

A GUIDE TO THE BOOK’S CONTENTS

Immediately following this chapter, Theodore P. Beauchaine, Daniel N. Klein, Nora L. Erickson, and Alyssa L. Norris (Chapter 2) provide a history of attempts to classify the vast domain of child and adolescent psychopathology, culminating in the newest editions of the Diagnostic and Statistical Manual of Mental Disorders and the International Classification of Diseases. This historical perspective throws into sharp relief the tensions between categorical versus continuous conceptions of psychopathological behavior and the still-vast territories to be investigated in order to reconcile basic science and current classifications. In Chapter 3, Theodore P. Beauchaine and Lisa M. Gatze-Kopp provide rich detail on gene-environment interplay and on psychobiological forces more generally, given their strong implications in the genesis of child and adolescent psychopathology. This material is essential background for many of the disorder-specific chapters that follow later in the volume.

In the next section (Part II), Bruce E. Compas and Charissa Andreotti (Chapter 4) lead off by writing about cultural and contextual factors linked to the development of psychopathology. As discussed in the present chapter, such influences deserve equal billing with psychobiological factors and forces in current DP conceptualizations. In Chapter 5, Sara R. Jaffee and Andrea Kohn Maikovich-Fong cover maltreatment, which is known to be a clearly “environmental” contributor to psychopathology but one that (a) correlates and interacts with biological vulnerabilities and (b) may well produce biological as well as psychological consequences. Then, in Chapter 6, Emily Neuhaus and Theodore P. Beauchaine discuss the role of impulsivity as a risk factor for diverse forms of externalizing psychopathology. Providing rich historical coverage, they debunk earlier myths about the neural underpinnings of impulsivity and bring to life a host of core DP constructs. Chapter 7, by Jerome Kagan, is a witty and engaging account of behavioral inhibition and its consequences for
a range of internalizing behavior patterns. This work also highlights many core DP
principles, serving as a needed antidote to reductionism, either environmental or bi-
ological. In Chapter 8 Bruce J. Ellis, Marco Del Giudice, and Elizabeth A. Shirtcliff detail
how exquisitely adaptive the human stress response system is, and the implications
of this adaptability for both vulnerability to and protection from psychopathology.
In Chapter 9, Nicole A. Crocker, Susanna L. Fryer, and Sarah N. Mattson discuss
teratogens—substances either (a) transmitted from a pregnant mother to her embryo
or fetus, or (b) encountered in the postnatal environment—that promote dysfunction
in the developing organism. This work emphasizes the probabilistic rather than the
deterministic nature of this pernicious risk factor. In Chapter 10, Katherine Shannon
Bowen and Lisa M. Gatzke-Kopp cover various forms of brain injury, with emphasis
on risk for dysfunction and potential recovery. This chapter deals with 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. In Chapter 11, Pamela M. Cole, Sarah E. Hall, and Nastasia J. Hajal
write a theoretically rich account of emotion dysregulation and its implications
for psychopathological 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 too quickly
or too slowly, implications for dysfunction become apparent.

Part III covers disruptive or externalizing conditions. In Chapter 12, Joel Nigg
provides rich coverage on the core processes of inattention, impulsivity, and
hyperactivity that characterize ADHD, with an integration of psychological and
psychobiological processes—and of both genetic and often-neglected environmen-
tal risk factors for this condition. Explicitly embracing the core tenets of DP, this
chapter emphasizes the great need for integrated theoretical models in future work
on this prevalent and distressing disorder. Chapter 13, by Irwin D. Waldman and
Benjamin B. 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 intra-individual, familial, and wider contextual variables; Waldman and
Lahey's integrative account is a welcome antidote to the often-chaotic feel of litera-
ture in this area. Thomas J. Dishion and Kristina Hiatt Racer, in Chapter 14, discuss
principles and concepts underlying the development of antisocial personality, taking
a life-span perspective as they do so, and featuring interplay among the myriad risk
factors (individual, parental, peer-related, neighborhood level) that together yield
adult antisocial outcomes and in some cases psychopathy. Chapter 15, by Sandra A.
Brown, Kristin Tomlinson, and Jennifer Winward, discusses developmental trajectories
of alcohol and substance problems, an area ripe for all of the DP principles
discussed in this chapter, with major public health implications given the scope and
consequences of early drug use and abuse.

Next, Part IV features internalizing conditions. Chapter 16, by Carl F. Weems
and Wendy K. Silverman, covers the range of anxiety disorders, providing an in-
tegrative 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 N. Klein, Autumn J. Kujawa, Sarah R. Black, and Allison T. Pennock comprehensively review, in Chapter 17, 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 18, Shelis E. Crowell, Erin A. Kaufman, and Mark F. Lenzenweger provide essential commentary on borderline personality, self-injurious behavior, and their developmental antecedents. They emphasize genetic and neural risk factors, the clear role of parenting disruptions, and interactions among them, concluding that borderline personality 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 IV is somewhat arbitrary, given the admixture of dysphoric, internalizing features, and disinhibited, externalizing symptoms involved in these behavior patterns.

Finally, the coverage in Part V focuses on several additional, extremely important disorders. Bipolar disorder—also representing an extreme blend of externalizing and internalizing features—is the topic of Chapter 19, written by Joseph C. Blader and Gabrielle A. 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 20, written by Susan Faja and Geraldine Dawson, features autism spectrum disorder, an extremely "hot" area of both basic and applied research. This chapter presents a balanced and detailed perspective on the most promising of recent conceptual models, along with the promise of early intervention. Schizophrenia can and does exist in children, with major increases in prevalence throughout adolescence. In Chapter 21, Robert F. Asarnow tackles the important developmental issues related to, and the strong psychobiologic roots of, schizophrenia-spectrum conditions. Finally, Chapter 22, authored by Eric Stice and Cara Bohon, covers anorexia nervosa, bulimia nervosa, and binge eating disorder, once again featuring processes spanning psychobiological and psychosocial factors in such conditions while providing strong emphasis on a developmental neuroscience perspective.

As readers begin the rest of this volume, we highlight that 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. The study of atypical development is complex and clinically relevant, with the potential for elucidating the processes by which normal development occurs and for informing sorely needed intervention and prevention strategies. The questions herein are among the most important issues in all of science. Welcome to the journey ahead!
REFERENCES


