CHAPTER 1

Toward a Developmental Psychopathology Approach for Understanding, Assessing, and Treating ADHD in Adolescents

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The two major topics addressed in this book—attention-deficit/hyperactivity disorder (ADHD) and adolescence—are steeped in lore and myth. Adolescence, for example, has been posited as a period of inevitable *sturm und drang* (storm and stress). ADHD has often been described as a social construction related to poor parenting and schooling. Far too many parents, teachers, clinicians, and member of the general public can become swayed by the inaccuracies.

In fact, although often challenging, adolescence is not inevitably tumultuous, and the psychobiological reality of ADHD is undoubted. Moreover, it is now clear that neither adolescence nor ADHD is a static category, but instead are highly dynamic processes shaped by the confluence of biology and transacting contextual forces. Official scientific and/or clinical recognition for both topics has emerged only within the past century-plus (e.g., Hall, 1904; Still, 1902). Finally, in recent years both adolescence and ADHD have been subjects of intense interest from the perspectives of science, clinical intervention, and policy, interest that has finally begun to place knowledge of each domain on a firm scientific foundation.

This volume marks the huge need for integration of findings related to ADHD—its manifestations, causal factors, development, and response to intervention—during the crucial developmental period of adolescence. Until quite recently, the intersection of these domains was extremely small. Indeed, psychiatry and clinical psychology assumed that the symptoms of ADHD (or what was formerly termed *minimal brain dysfunction, hyperkinesis,* or *hyperactivity*) largely vanished following puberty. Accumulating evidence, however, reveals that despite age-normative declines in overtly hyperactive behavior patterns, impairing levels of impulsivity and inattention persist in an overwhelming majority of children with diagnosable ADHD through the teenage years, and often beyond (Sibley, Mitchell, & Becker, 2016). In fact, the spiraling transactional patterns linked to ADHD during childhood often magnify with the onset of adolescence, resulting in an escalation and intensification of academic and social impairments, a broadening and deepening of comorbid psychopathology, and the presentation of major challenges for those interested in mounting and sustaining effective treatment strategies.

In this opening chapter, we provide a necessarily brief overview of basic points about both ADHD and adolescence before discussing the confluence of these crucial topics within the framework of developmental psychopathology. Such a framework is essential, we believe, given that reciprocal, interactive, and transactional forces typify the unfolding of ADHD-related problems—and, we hope, protective factors—during adolescence. We conclude with an overview of the volume's remaining contents.

At the outset, we are of the strong opinion that it is high time for increased consideration of ADHD and adolescence in tandem. Imagine entering the life-altering hormonal surge of puberty and the vast expansion of social expectations marking the adolescent years with a history of poor self-regulation and deficient impulse control, struggles in school, difficulties with peers, and strained family relationships. The challenges would be considerable indeed, requiring the best of scientific and clinical knowledge.

Attention-Deficit/Hyperactivity Disorder

Scientific findings about ADHD, along with controversies surrounding the entire topic, have grown exponentially in recent years (e.g., Barkley, 2015). Historically, following the advent of compulsory education during the 19th century, children's problems in attention span and behavioral deportment became salient to the medical community. Within the past 50 years, increasing academic pressures of postindustrial life and ready access to stimulant medications have prompted an explosion of concern and controversy over ADHD in national and international dialogue (Hinshaw & Scheffler, 2014). Overall, it is hard to conceive a clinical topic garnering the

amounts of interest, debate, and (too often) misinformation that ADHD has generated.

Readers should note that ADHD is a global phenomenon. Issues of dysregulated attention and impulsive behavior occur at surprisingly similar rates in youth around the world (see Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007; Polanczyk, Willcutt, Salum, Kieling, & Rohde, 2014). It can no longer be contended that this diagnostic condition is a sole product of U.S. values and cultural mores, a point that rings true for adolescent manifestations as well. The underlying behavioral features cluster into the domains of (1) inattention–disorganization and (2) hyperactivity–impulsivity (American Psychiatric Association, 2013). Developmentally extreme manifestations of these symptoms—when accompanied by clear functional impairments and not better accounted for by a different psychiatric condition—qualify the individual in question for a diagnosis. Yet without careful assessment procedures, ADHD can be both underdiagnosed and overdiagnosed (Hinshaw & Scheffler, 2014). Evidence-based evaluation strategies are therefore a priority, and the special considerations of adolescence with respect to assessment and diagnosis are featured in this book.

Beyond the core symptom domains, underlying processes related to self-regulation, executive function, intrinsic motivation and reward sensitivity, and emotion regulation are salient in differing subgroups of individuals with ADHD (see Brown, 2013; Sonuga-Barke, Bitsakou, & Thompson, 2010). With regard to neural underpinnings, multiple brain regions and circuits are involved (Castellanos & Proal, 2012), and maturational deficits in prefrontal gray matter are highly implicated in relevant symptom patterns (Shaw et al., 2007). Heritability is strong for the relevant behavioral and attention-related symptoms. Even more, a host of additional early-onset, biologically related triggers are also salient, exemplifying the developmental psychopathology principle of equifinality, whereby several different etiological pathways may yield similar symptom profiles (Cicchetti & Rogosch, 1996; for findings related to ADHD, see Nigg, 2017). Crucially, transactions between biological vulnerabilities and contextual influences such as parenting practices, school settings, and wider sociocultural factors influence both rates of diagnosis and outcomes of individuals with underling vulnerabilities, either favorably or unfavorably (e.g., Beauchaine & McNulty, 2013; Fulton, Scheffler, & Hinshaw, 2015). In short, ADHD is simultaneously a biological, social, and cultural construct.

This lightning-round overview does not begin to do justice to the multiple perspectives and levels of analysis related to full understanding ADHD (see Hinshaw, 2018). In fact, ADHD comprises far more than a homogenous diagnostic category of children, adolescents, or adults. Every aspect of ADHD is multifactorial, developmentally complex, and transactional in nature, and at no time in life is this truism more accurate than the adolescent years.

Adolescence

Many cultures have recognized a period of development from the time of physical and sexual maturation to the full adoption of adult roles and financial independence. Yet it took the publication of G. Stanley Hall's two-volume book, *Adolescence,* at the beginning of the 20th century, to mark the official pronouncement of this developmental epoch (Hall, 1904). Prescient in key respects yet almost comically inaccurate in others, these volumes opened up the age period in question—roughly spanning puberty to the early 20s—for scientific study, as well as educational and psychological scrutiny (see Arnett, 2006, for a cogent overview of the strengths and weaknesses of Hall's conceptions). For a lucid and readable overview of a host of issues related to adolescence, see Steinberg (2014).

The time span of adolescence is a moving target. Given that (1) puberty occurs much earlier, on average, than it did 150 years ago; (2) full independence in industrialized nations does not automatically take place at age 18; and (3) brain development (particularly in the frontal lobes) does not become adult-like until the mid-20s or beyond, adolescence has an ever-increasing scope. In fact, the life period now termed *emerging adulthood,* stretching from the late teen years to the mid-20s (Arnett, 2000), may actually constitute an extended period of adolescence. Like ADHD, adolescence is a construct embedded in the worlds of both biology (e.g., puberty) and social-contextual forces (e.g., delayed entry into full independence).

A key paradox is that adolescence is a time period of unprecedented cognitive and physical prowess *and,* simultaneously, of greatly enhanced vulnerability for physical injury, risk-taking behaviors, sleep problems, and the onset or intensification of many forms of mental disorder. The World Health Organization (2016) has recently become quite invested in promoting adolescent physical and mental health, given the population surge of adolescents worldwide, particularly in developing nations.

In science and policy, the adolescent years mark a second "inflection point" in individual physical, cognitive, and social development, following the huge expansion of abilities during the first few years of life. Ellis et al. (2012) frame this shift in evolutionary terms: Following puberty, individuals become intensely oriented toward attaining social status and developing reproductive strategies, guided by natural selection to propagate the species. Casey, Jones, and Hare (2008) describe the cascade of brain-related changes in development during adolescence, discussing the mismatch of "bottom-up" motivations from limbic regions with slower-developing "top-down" cognitive control (see also Steinberg et al., 2008). More broadly, Forbes and Dahl (2010) argue that pubertal maturation spurs a reorientation of the individual's social and motivational tendencies, enhancing the drive for sensation seeking and pressing youth toward both peers and potential romantic partners. Newfound cognitive skills (e.g., the attainment of formal operations), changing attitudes toward authority and

conformity, surges in risky decision making and risky behavior, individuation from one's family of origin, and major shifts in identity development are hallmarks of this period of life (see also Crone & Dahl, 2012).

Indeed, the influence of peers grows precipitously during adolescence. The classic experimental work of Gardner and Steinberg (2005) revealed that in the period of middle to late adolescence, the presence of agemates strongly magnifies risky behavior and risky decision making, a finding not present in adults. Given the peer-related difficulties surrounding youth with ADHD, this is a particularly fertile area for research—and clinical intervention.

The Confluence of ADHD and Adolescence

Synthesizing findings from these two domains, this volume features consideration of multiple aspects of ADHD during the adolescent years: clinical features, developmental trajectories, delineation of underlying mechanisms, evidence-based assessment practices, and effective and emerging intervention strategies. A focus on adolescent manifestations of ADHD is crucial given that ADHD beyond the childhood years has not received anywhere near the scientific and clinical scrutiny it so urgently requires.

In fact, the cumulative problems generated by ADHD-linked symptoms and problems often gain momentum following the major shifts in psychobiology and social expectations related to puberty. Consider, for example, the potential for escalating patterns of academic dysfunction—and resulting school disengagement—when academic material becomes more difficult and abstract, and when multiple teachers fill one's school day. Consider as well the pernicious social exclusion that can occur as peer groups coalesce and intimate relationships form during the teen years, along with growing opportunity for antisocial behavior and substance use during the transitional years of adolescence—particularly when children enter adolescence with preexisting vulnerabilities linked to impulse control. Risk for mood-related disorders and self-injury is also highly salient as emotion dysregulation becomes increasingly problematic during the teenage and early-adult years, particularly for youth with poor attentional filters and poor inhibitory control (e.g., Hinshaw et al., 2012; Owens et al., 2017). Moreover, these problems may all be compounded by increasingly shortened sleep duration across the adolescent years, in part due to early school start times in most high schools (American Academy of Pediatrics, 2014; Becker, Langberg, & Byars, 2015). In short, the passage through puberty, with its accompanying pressures for independence and self-sufficiency in the context of widening social networks—and in a world increasingly preoccupied with social media and academic pressure—makes adolescence a critical time period. It also prioritizes compiling and synthesizing the many scientific and clinical advances related to adolescents who have underlying problems in self-regulation.

The biological maturation linked to puberty pushes young teens to explore, seek thrills, and express themselves sexually at earlier ages than during most epochs of recent history. At the same time, the press for ever-greater educational and vocational training in much of the developed world—in an era of declining incomes for the majority of families and the unprecedented reality of lower income levels for young adults compared to their parents—means that adolescence appears to be extending longer than ever before. In the current era of rapid advances in our device-driven information landscape, with social media predominating many interpersonal exchanges, it is undoubtedly the case that intensive concentration needed for academic and social success may be both enhanced and hindered by the digital world in ways that still defy full comprehension. It is also likely that individuals with attention-related and impulsive styles may be quite vulnerable to the seduction of the digital world. The digital world may intersect in dangerous ways with the start of driving for many teens age 16 and upward, which is itself a key developmental challenge that becomes particularly salient when deficits in self-control and lapses in attention are part of the clinical picture (Barkley, Murphy, & Fisher, 2008; Narad et al., 2013).

Beyond ADHD per se, adolescent mental health has become a major topic. Data from the World Health Organization (2014) reveal that the leading cause of death, globally, for girls ages 15–19 years is now suicide. The age of onset of serious mood disorders appears to be dropping, signaling the importance of contextual "push" in unearthing vulnerability (Hinshaw, 2009). In both the developing and developed worlds, serious mental disorder in youth portends major life consequences and even tragedy (Sawyer et al., 2002). Even more, recent findings reveal links between a range of mental disorders and a long list of chronic physical illnesses (Scott et al., 2016). Emerging evidence suggests this linkage applies to ADHD as well, including predictions of lowered life expectancy (Barkley et al., 2008; Barkley & Fisher, 2019). Once again, adolescence marks a phase of life characterized by extreme promise and extreme risk.

Developmental Psychopathology Framework

To provide a conceptual framework for the entire volume, we now convey several core principles of developmental psychopathology (DP), the optimal lens through which ADHD and associated impairments during adolescence should be viewed. Indeed, we believe it takes a theoretical model that encompasses reciprocal, dynamic, transactional models of influence to yield full understanding of an age period marked by intensive transactional processes that transpire on a daily basis.

For the past four decades, the perspectives of DP have provided an integrative framework for understanding typical and atypical development.

A small sampling of historical works includes Achenbach (1974), Cicchetti (1984, 1990), Rutter and Sroufe (2000), and Sroufe and Rutter (1984); for current volumes of state-of-the art conceptualizations, see Cicchetti (2016) and Lewis and Rudolph (2014). In a nutshell, DP models blend developmental psychology and the clinical disciplines in a unique synthesis, whereby atypical behavior emanates from early vulnerability (either biological or psychosocial), which is compounded by spiraling transactions with widening environmental influences to result in deviations from typical developmental paths. In other cases, protective factors and processes may deflect the at-risk individual toward a healthier set of outcomes. Inherent in the study of these processes is the need to consider *multiple levels of analysis,* spanning molecular, brain-based, intraindividual, familial, school-related, neighborhood, and wider societal–cultural influences. Our concise review of core axioms and principles shows clearly that static models, rigid categorical conceptions of diagnosis, and unidimensional perspectives are simply not up to the task of explaining mental disorders, including ADHD, in a convincing or clinically satisfactory manner.

DP perspectives are no longer considered radical, as they have become the mainstream models for understanding pathways to healthy versus less healthy developmental outcomes. In fact, the DP focus on the interplay between biology and context and on multilevel influences has come to dominate current models of psychopathology (e.g., Beauchaine & Hinshaw, 2016). Key guiding principles include cross-disciplinary perspectives, multilevel processes, and systems-level, often nonlinear change processes that ultimately lead to developmental aberrations (Hinshaw, 2017).

At the outset, it is essential to realize that multiple pathways to pathology exist. Indeed, a key problem with static nosologies is their implicit assumption that everyone receiving a similar psychiatric diagnosis has parallel, if not identical, risk factors for and underlying processes related to psychopathology. In fact, however, disparate routes may lead to behaviorally similar conditions or outcomes, exemplifying the construct of *equifinality* (Cicchetti & Rogosch, 1996). For example, ADHD may result from strong genetic liability, from prenatal alcohol exposure or low birthweight, from difficult temperament interacting with environmental risk, or even—rarely—from horrific deprivation in early caregiving. Similarly, *multifinality* pertains when a given vulnerability, risk factor, or initial state fans out into disparate outcomes across different individuals (Cicchetti & Rogosch, 1996). Thus, neither difficult temperament nor child abuse or neglect inevitably leads to maladaptation, depending on a host of intervening factors. In terms of the current subject matter, heritable vulnerability for ADHD does not inevitably portend clinical levels of symptoms. Moreover, ADHD is itself heterogeneous in its presentation, whereby any given vulnerability may lead to widely varying clinical presentations.

The following list of principles represents one means of synthesizing DP formulations, adapted and shortened from the discussion in Hinshaw

(2017). Other conceptualizations posit additional axioms and formulations; in the interest of brevity, we provide the following summation.

Normal and Atypical Development: Mutually Informative

DP models emphasize that clinical phenomena represent aberrations in continua of normal developmental pathways and processes—in other words, they constitute *adaptational failures* (Sroufe, 1997). Accordingly, without understanding typical development, the study of pathology will remain incomplete and decontextualized. For example, comprehending ADHD requires knowledge of the normative development of attention, impulse control, and self-regulation (e.g., Barkley, 2015; Hinshaw & Scheffler, 2014; Nigg, 2017; Owens & Hinshaw, 2016; Sonuga-Barke et al., 2010). One cannot study (or adequately treat) mental disorder without real understanding of normative developmental processes. At the same time, DP envisions a two-way street, as investigations of pathological conditions can and should, in parallel, provide a unique perspective on normative development. Atypical developmental processes may therefore provide an essential window on general models of human development.

This core tenet—of the mutual interplay between normality and pathology—is espoused widely. Indeed, examples are prevalent in neurology, where the study of disrupted neural systems enhances understanding of healthy brain functioning, and vice versa (Gazzaniga, Ivry, & Magnun, 2014). In neurology, however, single lesions or single genes can often be isolated, whereas mental disorder is inherently multifactorial (see Kendler, 2005). Still, accumulating evidence suggests that at the levels of genes, harshly aberrant environments, reward mechanisms, empathy "modules," and many more examples, examination of pathological development may yield key clues to normative pathways.

Developmental Continuities and Discontinuities

Here, a key conception is that, across development, traits and behavior may unfold in lawful ways, but the surface behavioral manifestations may well change with maturity and opportunity; that is, continuity of behavior may be *heterotypic*. In the realm of antisocial behavior, for example, extremes of difficult temperament (e.g., intense and frequent tantrums) may give way to verbal aggression in preschool, physical assaults in elementary school, sexual aggression and robbery during adolescence, and partner violence by adulthood. The "surface" behaviors change as an underlying antisocial trait changes form with time. In other words, brain maturation, growing cognitive skills, and widening social and cultural opportunities converge to propel the emergence of heterotypically continuous behavior patterns over time, in keeping with the dynamic tenets of DP models.

With ADHD, for example, defiance and overt hyperactivity early in

development typically yield academic problems and peer rejection during grade school, with multiple forms of impulsivity and disorganization becoming salient by the teen years. Even more, other disorders (e.g., substance abuse, antisocial personality) may become apparent. By adulthood, pathways may branch even further. Of extreme importance, girls with ADHD (particularly those with early-emerging impulsivity) are at high risk for displaying clinically significant levels of self-harm by adolescence (see Hinshaw et al., 2012; for additional information on predictor and mediator variables, see Guendelman, Owens, Galan, Gard, & Hinshaw, 2016; Meza, Owens, & Hinshaw, 2016; Swanson, Owens, & Hinshaw, 2014). Indeed, Guendelman et al. (2016) showed that childhood ADHD in the presence of maltreatment yields extraordinarily high rates of suicide attempts by late adolescence in girls. Thus, (1) heterotypic continuity may encompass behavior patterns that are dissimilar to their *phenotypic* precursors but lawfully emerge from core *developmental* precursors; (2) the adolescent years are likely to witness important examples of the emergence of heterotypically continuous problems in youth with ADHD; (3) the individual's biological sex may serve as a moderator of differing patterns of heterotypic continuity; and (4) biological vulnerability (e.g., impulsivity) is likely to interact with psychosocial risk (e.g., maltreatment) to yield particularly salient impairment and pathology.

Risk and Protective Factors

A key focus of DP—with the term *psychopathology* embedded in its title—is discovery of the nature and roots of behavioral and emotional problems. But many argue that it is equally (if not more) important to uncover those protective influences that may transform risk into *resilience,* defined as unexpectedly good outcomes, or competence, in the face of adversity or risk (see Luthar, 2006; Masten & Cicchetti, 2016). Indeed, the concept of multifinality, noted previously, directly implies that depending on a host of biological, environmental, and contextual factors, variegated outcomes may well emanate from the same risk factor across individuals, with the distinct possibility of resilience and positive adaptation in some cases.

DP is therefore involved centrally in the search for what have been called *protective factors:* variables and processes that mitigate vulnerability/risk and promote more successful outcomes than would be expected in their presence. Controversy surrounds the construct of resilience, the nature of protective factors, and the definitions of competent functioning (see Burt, Coatsworth, & Masten, 2016). Perhaps there is no need to invoke a set of special processes that are involved in resilience, as a certain percentage of any high-risk sample is likely to show better-than-expected outcomes. Also, protective factors are too often viewed as the opposite poles of 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). Yet it is essential to examine processes involved in promoting competence and strength rather than disability and despair; such processes may be harnessed for prevention efforts and may provide key conceptual leads toward the understanding of both pathology and competence.

Reciprocal, Transactional, and Ontogenic Process Models

Linear models of causation, whereby static psychological or psychobiological risk factors respond in invariant ways to the influence of additional variables, cannot fully explain psychopathology and its development (see Richters, 1997, for elaboration). Pathways to adolescent and adult functioning are marked by reciprocal patterns or chains, in which children influence parents, teachers, and peers, who in turn shape the further development of the child (for a classic formulation, see Bell, 1968). Such mutually interactive processes evolve over time into *transactional models*. It is little wonder that static categories of mental disorder are hard-pressed to do justice to such dynamic, interactive processes. Sensitive data-analytic strategies and innovative research designs are crucial essential for fostering greater understanding of these phenomena.

Incorporation of these processes can elucidate patterns of equifinality and multifinality, as described earlier. Moreover, recognition of the problems with current categorical nosologies prompted the genesis of the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC; Insel et al., 2010), a dimensional means of accounting for psychopathology that embraces a multiple-levels-of-analysis approach and emphasizes neural circuitry in interaction with contextual influences. Clearly, the field is seeking the kinds of models and paradigms that can optimally incorporate the complexity and multifaceted nature of mental disorder.

In parallel, *ontogenic* process models of psychopathology have witnessed a resurgence (see Beauchaine & Hinshaw, 2016; Beauchaine & McNulty, 2013). Here, heritable vulnerabilities transact with environmental forces (e.g., coercive family interactions; violent neighborhoods) to yield psychopathology. In such models, what appear to be the emergence of discrete, "comorbid" disorders across development may in fact represent heterotypic continuity. Indeed, apparent *comorbidity* (the joint presence of two or more independent conditions) may in many cases be artifactual, representing instead the unfolding of transactional, DP-related processes. Once again, static and/or linear models of influence must yield to reciprocal, dynamic chains of influence.

Psychobiological Vulnerability and Context

The genomic era fully emerged in 2001, when the strands of DNA comprising the human genome were finally decoded. At the same time, progressions

in brain imaging research have made the developing brain far more accessible to scientific view than ever before. These advances flew in the face of the predominant models of the 20th century, which emphasized parenting and other aspects of socialization as the core drivers of development. Rather than pitting biology against context, DP prioritizes interactive and transactional models of the ways in which early biological risk is potentiated (or redirected) by complex, multilevel contextual influences (e.g., see Hyde, 2015).

For example, gene–environment correlation recognizes the inextricable confounding of genes and contexts in shaping development within biological families. Genetically informative research investigations are therefore crucial to understanding risk and protection. Regarding ADHD, Harold et al. (2013) leveraged two adoption designs to disentangle heritable from contextual influences. In brief, even in adoptive families, which remove the influence of gene–environment correlation, children's disruptive behaviors evoked harsh maternal responses, which in turn predicted subsequent ADHD behavior patterns. Thus, although the heritable nature of ADHD is clear, psychosocial processes within families are still highly influential in terms of development and clinical manifestation.

The area of gene × environment interplay provides an additional, if sometimes contentious, example. The underlying idea is that genotypes moderate the effects of environmental context on the development of psychopathology, such that contextual risk is most pronounced for only certain configurations of genetic–biological vulnerability (for elaboration, see Dodge & Rutter, 2011). Might it be the case that risk for ADHD-related impairment is most pronounced for only certain genotypes in the context of certain environments? Core critiques of this concept have been argued. For instance, Dick et al. (2015) outline essential recipes for avoiding the major issue of false-positive findings in research on gene × environment interactions; Keller (2014) adds the cautionary note that many gene–environment researchers will overestimate such interactive power unless they take into account the potentially confounding effects of passive gene–environment correlation. Crucially, it may also be the case that some "vulnerability" genes are actually "susceptibility" genes, which means that they are particularly responsive to either extremely good or poor environments in yielding optimal versus pathological outcomes (see Belsky & Pluess, 2009; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). The potential for resilience is fascinating to consider within this framework.

Summary

These principles converge on the major theme that the development of psychopathological functioning, including ADHD, is multidetermined, complex, interactive, transactional, and in many instances nonlinear. When the

life period of adolescence becomes part of the mix, with its inherent complexities and dynamics, the need for DP models becomes even more apparent. In fact, we consider the DP perspective an essential framework for readers who are intrigued by the diverse clinical presentations of various pathological conditions, like ADHD, across childhood and adolescence; for those who are fascinated with how much remains to be learned about risk and maintaining factors; and for those looking for ways to assess and treat adolescent forms of ADHD with maximum impact. With this consideration in mind, we now turn to providing an overview of the following chapters that examine the confluence of ADHD and adolescence within a DP framework.

Overview of the Book's Contents

The contributors to this book were encouraged to carefully consider the previously discussed review on the DP of ADHD in adolescence as they prepared their chapters. The result is a compendium of chapters on ADHD that each take seriously the developmental context of adolescence. This book is divided into two sections. Part I focuses on ADHD in the context of adolescence and adolescent development, and Part II focuses on assessing and treating ADHD in adolescence. Immediately following this chapter, Henrik Larsson (Chapter 2) provides an overview of the developmental course of ADHD across adolescence and into early adulthood. He covers important questions regarding the trajectory of ADHD across adolescence, including both diagnostic persistence and symptom dimensions, and also reviews the recent controversial literature on the possibility of an adult-onset ADHD that is distinct from childhood-onset ADHD. In Chapter 3, Erik G. Willcutt summarizes behavioral genetic studies of ADHD with a particular focus on adolescence, including new analyses from two twin samples conducted for this chapter. He also reviews literature on molecular genetic and environmental risk factors, observing the need for etiologically informative longitudinal designs that may help identify factors associated with the persistence or decline of ADHD symptoms across adolescence. Next, Joshua Doidge, Wafa Saoud, and Maggie E. Toplak (Chapter 4) provide a review of the executive function and decision-making research in ADHD. Particular attention is given to temporal discounting, risky decision making, and ADHD presentations/co-occurring psychopathologies. In Chapter 5, Nóra Bunford covers the area of emotion regulation and its importance to conceptualizations of ADHD. In doing so, she reviews key definitional and measurement issues, considers overlapping biological and psychosocial vulnerabilities, and emphasizes the important role of emotion regulation for functioning in adolescents with ADHD.

The remaining chapters in Part I shift to the social experiences,

co-occurring difficulties, and adolescent-salient domains of functioning. In Chapter 6, Judith Wiener considers the family context of adolescents with ADHD. She builds on the larger literature on families of children with ADHD before reviewing the extant literature on adolescents specifically, including bidirectional associations, parenting cognitions, parental ADHD, and parent involvement in interventions for adolescents with ADHD. Chapter 7, by Julia D. McQuade, shifts to the peer relationships that take on increased priority and consume more time during adolescence. In reviewing the literature on peer relationships in adolescents with ADHD, attention is given to the larger peer group, dyadic friendships, and romantic relationships, in addition to both in-person and online relationships. She also summarizes the very limited intervention research focused on the peer relationships of adolescents with ADHD, an area ripe for much-needed attention. Steven W. Evans, Saskia Van der Oord, and Emma E. Rogers (Chapter 8) then turn our attention to the school setting in which adolescents spend much of their day and frequently struggle. The authors consider pathways to academic difficulties in adolescents with ADHD and review the rapidly growing body of intervention research specifically targeting academic impairments in adolescents with ADHD. In Chapter 9, Stephen P. Becker and Nicholas D. Foglеman review psychopathologies that frequently co-occur with ADHD (comorbidity). Prevalence rates of co-occurring internalizing and externalizing psychopathologies in adolescence are reviewed, in addition to risk factors and developmental pathways. Attention is given to domains of psychopathology that need additional scrutiny in adolescence, including autism spectrum disorder (ASD), sluggish cognitive tempo (SCT), eating pathology, and self-harm and suicide. Research examining co-occurring psychopathologies as outcomes, predictors, or moderators of intervention for adolescents with ADHD are also reviewed. Sleep difficulties are also common in youth with ADHD, and sleep is the focus of Chapter 10 by Melissa Mulraney, Emma Sciberras, and Stephen P. Becker. Although the sleep problems of children with ADHD have been documented for some time, only recently has there been significant interest in the sleep of adolescents with ADHD. Mulraney and colleagues review recent research in this area and possible biological and psychosocial contributors to poor and insufficient sleep in adolescents with ADHD, in addition to likely transactional processes and assessment–intervention considerations. Next, Traci M. Kennedy, Kirsten M. P. McKone, and Brooke S. G. Molina (Chapter 11) review the extensive literature on substance use in adolescents with ADHD. The authors give important attention to assessment and measurement issues before reviewing evidence for problematic substance use in adolescents with ADHD. Developmental pathways, moderators and protective effects, and treatment implications are carefully considered. The final chapter (Chapter 12) in Part I focuses on a key domain that first emerges in adolescence: driving. Annie A. Garner

first provides an important backdrop on driving risk and driving problems in adolescence broadly, before reviewing the literature examining driving behaviors and outcomes in adolescents with ADHD specifically. Risk and protective factors are thoroughly reviewed, and possible interventions to improve the driving of adolescents with ADHD are discussed.

In Part II, we turn our attention more directly to the assessment and treatment of adolescents with ADHD. George J. DuPaul, Arthur D. Anastopoulos, and Kristen Kipperman (Chapter 13) start this section off with a comprehensive review of the process, methods, and measures used to assess and diagnose ADHD in adolescence. Their chapter also includes several case examples that highlight key issues in the assessment of ADHD in adolescents and young adults. In Chapter 14, Margaret H. Sibley addresses the key issue of motivation when treating adolescents with ADHD. She reviews the complex causal pathways implicated in the complex pathways of ADHD that often give rise to organization, time management, and planning problems as well as volitional, self-efficacy, and/or motivation difficulties. Various treatment approaches to target these multifaceted difficulties are discussed, including the Supporting Teens' Autonomy Daily (STAND) intervention, which is a parent–adolescent therapy targeting executive function and motivation deficits. Intervention approaches targeting homework problems are the focus of Chapter 15 by Joshua M. Langberg, Zoe R. Smith, and Cathrin D. Green. Using the homework completion cycle as their guide, the authors consider homework problems across development, the assessment of homework problems, and core treatment strategies for improving homework problems in adolescents with ADHD. Research on treating these problems in adolescents with ADHD is reviewed, including interventions implemented in afterschool, in-school, and clinic settings: the Challenging Horizons Program (CHP) and the Homework, Organization, and Planning Skills (HOPS) and STAND interventions. Next, Susan E. Sprich and Jennifer A. Burbridge (Chapter 16) briefly review psychosocial treatments for adult ADHD and the theoretical and developmental rationale to extend cognitive-behavioral approaches down to adolescence. They describe modifying their adult cognitive-behavioral therapy (CBT) treatment protocol for use with adolescents with ADHD, which consists primarily of individual sessions augmented by two parent–adolescent sessions and optional parent-only sessions. Two case examples offer insights for implementing a CBT approach when working with adolescents with ADHD. In Chapter 17, Naomi Ornstein Davis and John T. Mitchell provide an overview of mindfulness-based interventions and their extension to adolescents before providing a rationale for applying these interventions to individuals with ADHD. They then review the small but growing body of research evaluating mindfulness-based interventions for adolescents with ADHD and provide important considerations for this nascent area. In the final chapter (Chapter 18), William B. Brinkman, Tanya E. Froehlich, and Jeffery N. Epstein shift

our focus to the important area of medication treatment in adolescents with ADHD. The authors review the evidence for medication treatment in this population, including effects on ADHD symptoms and functional impairments, as well as side effects and possible adverse consequences such as misuse and diversion. Issues related to medication adherence are detailed, and recommendations are provided for clinicians working with adolescents with ADHD, with two case examples illustrating how clinicians can help support medication continuity while also fostering autonomy.

Together, the chapters in this book integrate a large volume of research to help us better understand, assess, and treat adolescents with ADHD. Yet each chapter also points to the need for far more research devoted to ADHD during this crucial developmental period. A DP approach is especially well-suited to examining the inherent complexities of ADHD in adolescence, and it is our hope that others will join us in this endeavor of major empirical and clinical importance.

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