One

UNDERSTANDING ADHD

Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental condition marked by developmentally inappropriate levels of inattention, and/or impulsivity and hyperactivity that often significantly impair functioning across multiple domains and place children at elevated risk for a variety of adverse outcomes. It is important for clinicians who work with youth to possess a basic understanding of ADHD as it is one of the most frequently diagnosed disorders of childhood and among the most common reasons for child mental health referrals in both community and school settings. However, ADHD is frequently misunderstood even by mental health professionals. This is due in part to the confusing array of labels by which it is known, misinformation disseminated through the popular press, social media, and on the web, and to the complex, heterogeneous, and highly variable nature of the disorder itself.

Fortunately, ADHD has been subject to an enormous amount of scientific research (viz. more than 10,000 journal articles and over 100 textbooks) (Barkley, 2013). As a result, we know more about ADHD than any other mental health disorder beginning in childhood. The purpose of this chapter is to provide a brief but informative overview of ADHD, including current scientific knowledge. (Numerous resources exist for readers interested in more detailed descriptions of ADHD; see, for example, Barkley, 2006; DuPaul & Kern, 2011; Evans & Hoza, 2011; Goldstein & DeVries, 2011; Hinshaw & Scheffler, 2013; Jensen & Cooper, 2002). After a short summary of the history of ADHD, the chapter addresses core and associated features of the disorder, common comorbidities, etiology, and epidemiology. Although this book focuses on ADHD in children and adolescents, there is a growing body of literature about ADHD in adults (e.g., see Barkley, Murphy, & Fischer, 2008; Goldstein & Ellison, 2002; Surman, 2013; Weiss, Hechtman, & Weiss, 1999).

HISTORICAL PERSPECTIVE

ADHD has a long and somewhat controversial history (historical landmarks are summarized in Rapid Reference 1.1; for detailed accounts, see Antshel & Barkley, 2011; Taylor, 2011). Early clinical descriptions of the disorder, dating back over 200 years, came from physicians on the basis of children seen in their practices. These took numerous forms, ranging from book chapters (Weikard in 1775; see Barkley & Peters, 2012) and lengthy tomes (Crichton, 1798, 2008), to lectures (Still, 1902) and doggerel poems (Hoffmann's verses, "Fidgety Philip" and "Johnny Head-In-Air"; Hoffmann, 1844; English edition in 1848). Although the inclusion of inattentive, hyperactive, and impulsive symptoms has been relatively constant across clinical and scientific descriptions of the disorder over time, conceptualizations have evolved considerably with respect to presumed defining features, diagnostic labels, etiologic theories, and practice standards for assessment and treatment.

Early descriptions of ADHD often included inattention, but focused on hyperactivity as the core feature of the disorder. However, studies from Virginia Douglas' lab in the late 1960s and 1970s firmly reinstated the importance of deficits in sustained attention and impulse control in descriptions of the syndrome (Douglas, 1972, 1976). Influenced largely by this research, deficits in sustained attention rather than overactivity came to be viewed as central to the disorder by the early 1980s (American Psychiatric Association, 1980). Over recent decades, a neuroscience perspective has been applied to examining

"Let me see if Philip can
Be a little gentleman;
Let me see if he is able
To sit still for once at table":
Thus Papa bade Phil behave;
And Mamma looked very grave.
But fidgety Phil,
He won't sit still;
He wriggles,
And giggles,
And then, I declare,
Swings backwards and forwards,
And tilts up his chair,
Just like any rocking horse—
"Philip! I am getting cross!"

Figure 1.1. Excerpt from "The Story of Fidgety Philip," a cautionary poem about hyperactivity from the 1840s

Source: Hoffmann, 1844.

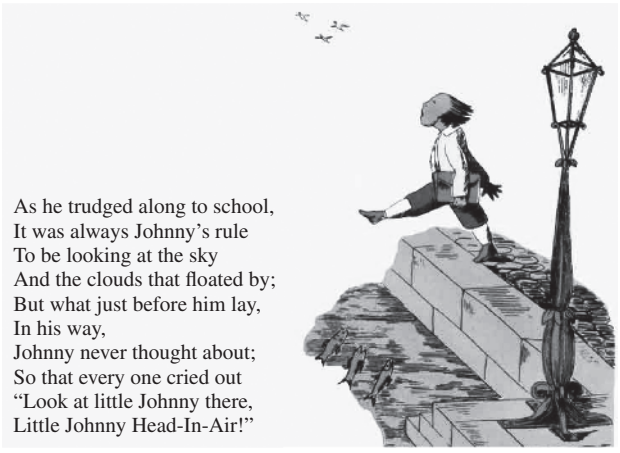


Figure 1.2. Excerpt from "The Story of Johnny Head-in-Air," an 1840s poem about pervasive inattention

Source: Hoffmann, 1844.

difficulties with motivation, response to reinforcement, inhibition, and executive functions as possible core problems underlying ADHD (Barkley, 1997; Brown, 2013; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg, 2013a; Nigg & Casey, 2005; Sagvolden, Aase, Zeiner, & Berger, 1998; Schachar, Tannock, & Logan, 1993).

Similarly, presumptions about the causes of ADHD have changed considerably over time, reflecting both research findings and prevailing scientific paradigms used to explain cognitive and behavioral functioning (Conners & Erhardt, 1998). Early views centering on defective "moral control" and presumed brain damage along with later environmental theories highlighting diet and child-rearing gradually gave way to more biologically based and data-driven explanations. At various times, research investigations have focused on psychophysiology, motivational deficits, neurotransmitter deficiencies, neuropsychological functioning, and genetic factors. More recently, studies employing increasingly rigorous and sophisticated methods (including brain imaging techniques) have elucidated potential structural and functional neurological bases for ADHD and illuminated how genetically based risk might interact with or be triggered by various environmental factors (e.g., pre- or post-natal environmental toxins such as alcohol, nicotine, and pesticides) to culminate in the self-regulation problems characteristic of ADHD (see the section on etiology below).

Rapid Reference 1.1

Historical Landmarks Related to ADHD in Children and Adolescents

1775 —Weikard's medical text has a chapter about "Attention Deficit," including his recommendations for treatment.

1798 —Crichton writes about disordered attention.

1840s—Hoffmann, a German physician, composes moralistic verses for his young son, including characters with features of hyperactivity and inattention.

1902 —Still describes patients with features of impulsivity and short attention span as suffering from an "abnormal defect of moral control . . . without general impairment of intellect and without physical disease."

1937 —Bradley documents benefits of the stimulant Benzedrine (dextro-amphetamine sulfate) for children with behavior disorders, marking the beginning of pharmacotherapy for this population.

1950s—Stimulants begin to be used regularly to treat hyperactivity.

1955 —FDA approves methylphenidate (Ritalin) for treatment of hyperactivity.

1963 —65—Eisenberg publishes studies documenting benefits of stimulant medication in treating hyperkinesis, in comparison to placebo and traditional psychotherapy.

1969 —Conners develops the first structured parent and teacher rating scales to reliably assess ADHD symptoms and treatment response.

1970s—Various environmental factors (e.g., food additives, societal tempo, poor parenting) proposed as causes of ADHD; stimulants emerge as treatment of choice for ADHD symptoms; efficacy studies support the use of behavioral treatment, via classroom-based modification and parent training, contributing to the emergence of combined treatments; studies by Douglas contribute to shifting view of attention deficits (rather than hyperactivity) as the defining feature of the disorder.

1975 —Public Law 94-142 mandates special education services for children with behavioral (as well as other) disabilities, though exclusion of terms specific to hyperactivity/ADD/ADHD would result in services being denied to many with ADHD.

1980s—Broadband and ADD-specific standardized rating scales published; computerized tests of attention developed; non-stimulants investigated for treatment of ADHD.

1986 —Seminal longitudinal study by Weiss & Hechtman demonstrates the persistence of inattention and impulsivity past childhood despite declines in hyperactivity, undermining the view that adolescents "outgrow" the disorder.

1987 —*Children and Adults with Attention-Deficit/Hyperactivity Disorder* (CHADD) founded; this information, support, and advocacy group would play an important role in psychoeducation and in securing access to special education services for youth with ADHD.

1990 —1991—Children with ADHD granted eligibility to special education services through the *Other Health Impaired* category of the *Individuals with Disabilities Education Act* (IDEA) and a memorandum from the U.S. Dept. of Education's Office of Special Education.

1990s—Present—Significant research advances pertaining to the neurological basis of ADHD (via brain imaging studies), distinguishing neuropsychological factors with a particular emphasis on executive functions, and genetic contributions to the disorder; theories increasingly focus on behavioral inhibition and executive functioning as putative "core" deficits; increased attention to ADHD in females; the large-scale *Multimodal Treatment of Attention Deficit Hyperactivity Disorder* (MTA) study is conducted; introduction of new stimulant and non-stimulant medications along with marked increases in pharmacologic treatment of ADHD.

Sources: Antshel & Barkley (2011); Barkley (2006).

The labels used to describe the disorder now known as ADHD, both within and outside of official classification systems for mental disorders, have changed frequently over the years. These changes have generally paralleled shifts in prevailing views of defining features and causal factors discussed earlier. Thus, a non-exhaustive list of the terms that have been used to describe this syndrome includes *minimal brain damage (MBD)*, *hyperkinetic impulse disorder*, *hyperactive child syndrome, hyperkinetic reaction of childhood, minimal brain dysfunction*, *hyperkinesis, attention-deficit disorder*, and the current *attention-deficit/hyperactivity disorder* (see Rapid Reference 1.2). Efforts to more accurately and precisely label and diagnose this disorder have been accompanied by examinations of the most meaningful ways to subtype ADHD, particularly with respect to whether certain subtypes are not only durable over time but associated with clinically meaningful differences with respect to factors such as the nature of core and associated symptoms, causality, comorbidities, course, response to treatment, and outcomes. The most notable basis for such subtyping has involved whether inattentive features, hyperactive/impulsive features, or both are salient in the presentation of the disorder (a subtyping scheme that has appeared, disappeared, and reappeared over various editions of the DSM classification system). However, children with ADHD have also been meaningfully subtyped on the basis of the presence or absence of comorbid aggression, learning disorders, and anxious or depressive features (Barkley, 2006).

Among the most significant turn of events in the history of ADHD has been a shift in our understanding of the persistence and seriousness of the disorder. Spurred by results of numerous longitudinal studies (see, for example, Klein

Rapid Reference 1.2

Changing Labels

1950s—*Minimal brain damage*—presumed neurological damage.

1957 —*Hyperkinetic impulse disorder*—attributed high activity levels to central nervous system deficit leading to cortical overstimulation.

1960s—*Hyperactive child syndrome*—focused on high activity level as the defining feature; symptoms described as remitting in adolescence.
Hyperkinetic reaction of childhood (DSM-II)—initial inclusion of the disorder in DSM comprised a single-sentence description that noted hyperactivity, distractibility, and short attention span along with the assertion that features typically decline by adolescence.
Minimal brain dysfunction—symptoms accounted for by high and poorly regulated levels of activation, deficits related to the experience of pleasure and pain, and extroversion.

1980 —*Attention-deficit disorder (with or without hyperactivity) (DSM-III)*—reconceptualized ADHD with a focus on inattention as the defining feature; separate symptoms lists provided for inattention, hyperactivity, and impulsivity; subtypes, numerical cutoff scores, and guidelines for age of onset and duration of symptoms first introduced; ADD-Residual Type (ADD-RT) introduced for persistence of some symptoms after remission of hyperactivity.

1987 —present—*Attention-deficit/hyperactivity disorder* (ADHD).

1987 —DSM-III-R dropped subtypes (but added *undifferentiated ADD,* which resembles the current inattentive presentation); empirical basis as field trial results contributed to criteria; single-symptom list and cutoff score; added verbal manifestations of hyperactivity, introduced need to establish symptoms as developmentally inappropriate; grouped ADHD with ODD and CD in a disruptive behavior disorders category.

1994 —DSM-IV reintroduced subtypes, including *predominantly inattentive type, predominantly hyperactive-impulsive type,* and *combined type*; criteria become increasingly empirically based; separate symptom lists and cutoff scores for *inattention* and *hyperactivity-impulsivity* factors; used *ADHD not otherwise specified* to capture atypical presentations.

2013 —DSM-5 downgraded subtypes to "presentations," including *predominantly in-attentive presentation*, *predominantly hyperactive/impulsive presentation,* and *combined presentation*; ADHD included among *Neurodevelopmental Disorders* but no longer grouped with ODD and CD; age of onset raised from 7 to 12 years.

Sources: Antshel & Barkley (2011); Barkley (2006); Taylor (2011).

et al., 2012; Weiss & Hechtman, 1986, 1993a), the prevailing view of ADHD has evolved from a largely benign disorder that children generally outgrow by adolescence to a typically chronic and impairing condition. This recognition, along with greater clarity with respect to how the presentation of ADHD changes

over the life span, has led to increased efforts to refine and optimize our assessment and treatment practices.

Medication therapies have long been the most common treatment for ADHD and remain so today. The use of stimulant medications with behaviorally disordered youth (and, in fact, the origins of child psychopharmacology more generally) can be traced back more than 75 years, when the physician Charles Bradley reported that Benzedrine resulted in notable behavioral and academic improvements in a hospitalized group of such children (Bradley, 1937). Stimulant medications became routinely prescribed for ADHD in the 1950s, following additional reports noting positive effects of amphetamine and methylphenidate on children with what was then called minimal brain dysfunction or hyperactive child syndrome (Wolraich, 2011). The U.S. Food and Drug Administration (FDA) approved methylphenidate (Ritalin) for use in children with hyperactivity in 1955 (U.S. Food and Drug Administration, 2013). Research conducted by Leon Eisenberg and Keith Conners (Eisenberg et al., 1963) ushered in era of increased methodological rigor in pediatric studies documenting the benefits of stimulant medication. The results of numerous additional random controlled trials over subsequent years (Swanson, McBurnett, Wigal, & Pfiffner, 1993) led to the gradual emergence of stimulant medications as the treatment of choice for ADHD. Recent decades have witnessed the introduction of a host of new pharmacologic agents for ADHD (e.g., atomoxetine), novel ways to deliver and sustain their effects over longer periods of time (e.g., micro-beads, dermal patches, osmotic pumps), and rather dramatic increases in their prescription rates, their use across the age span (including preschoolers), and, consequently, ongoing controversy regarding their use (Greenhill, Halperin, & Abikoff, 1999; Greenhill et al., 2002; Greenhill et al., 2006; Kaplan, 2011; Mayes, Bagwell, & Erkulwater, 2009; Solanto, Arnsten, & Castellanos, 2001; Swanson & Volkow, 2009; Zuvekas, 2012).

Alongside the voluminous literature on stimulant medications, a significant evidence base has accrued over the past 40 years for the efficacy of behavioral-based treatments for ADHD, whether delivered in the context of classroom interventions, parent training, or therapeutic settings like specialized summer camps (Owens, Storer, & Girio-Herrera, 2011). Many other forms of treatment have been tried for ADHD through the years. With respect to improving primary symptoms of the disorder, some are ineffective (e.g., play therapy), some may help only a small portion of sufferers (e.g., special diets), and others have some evidence but have yet to accrue the type of strong, consistent research support needed to establish them as "proven" treatments (e.g., neurofeedback, computer-based cognitive training) (Hurt, Lofthouse, & Arnold, 2011; Lofthouse, McBurnett, Arnold, & Hurt, 2011; Melby-Lervag & Hulme, 2013).

Numerous studies have examined treatments that combine medication and behavioral interventions, leading to the general conclusion that while stimulants are the single most effective and possibly sufficient treatment for reducing core ADHD symptoms, combined treatments are most likely to normalize problem behavior and appear to be superior with respect to improving comorbid symptoms, building skills (academic, social, parenting), and reducing key life impairments (Conners et al., 2001; MTA Cooperative Group, 1999; Swanson et al., 2001). What has been elusive over many decades of intervention research has been the identification of treatments or treatment combinations yielding benefits that generalize across situations and time once acute treatment is discontinued or that fundamentally alter the core deficits of ADHD.

ADHD assessment practices have evolved considerably from an early reliance on clinical impressions (e.g., "I know it when I see it"), to the introduction of structured behavior rating scales by Keith Conners in the late 1960s (Conners, 1969), to the development of a host of scales, interviews, and objective tests designed to assist in the detection of the disorder (Pelham, Fabiano, & Massetti, 2005; Smith, Barkley, & Shapiro, 2007). This progression has led us to the current standard of care: A careful integration of interview, rating scale, testing, and observational data drawn from multiple sources and informants in order to identify ADHD and distinguish it from typical development and from other conditions that might produce similar symptoms (American Academy of Child and Adolescent Psychiatry Work Group on Quality Issues, 2007a). The *how*s and *why*s of such assessment practices for ADHD will be the focus of subsequent chapters.

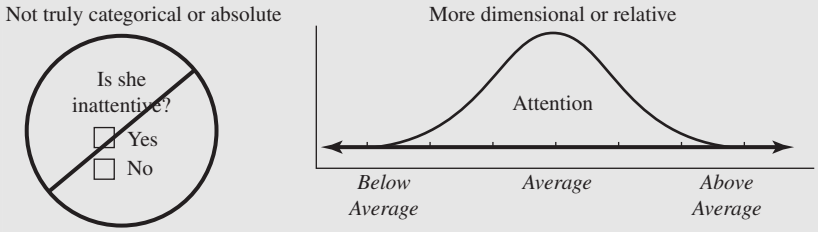
OVERARCHING PRINCIPLES

There are two central principles to remember when thinking about ADHD: *dimensionality* and *variability*. Although the practical realities of clinical diagnosis and the very nature of classification systems like the DSM lure us into thinking of ADHD (and other behavioral disorders) categorically ("either she *has* ADHD or she doesn't"), the reality is that ADHD (like many other mental health disorders) is best thought of dimensionally (Frick & Nigg, 2012; Lahey & Willcutt, 2002; Marcus & Barry, 2011) (see Chapter 3 for further discussion of this distinction). That is, the features of ADHD exist on a continuum along which every person can be placed, just like height, weight, or IQ. To illustrate this essential truth, consider how much typically developing children differ with respect to their ability to regulate their activity levels, sustain attention, and restrain their impulses. At each age, there is an average level of these abilities with a considerable range of variation

around that average. Younger children generally have a lower average and a broader range. In fact, the reality is that short attention spans, high activity levels, and impulsivity are a normal part of childhood for many youngsters. When diagnosing ADHD, we are identifying children who fall at the extreme end of the continuum, whose deficits lead to impairment. The challenge is that there is no magic dividing line on the continuum to separate "typical" from "ADHD." This lack of a natural boundary for the diagnosis of ADHD is why it is so important to be certain that symptoms are (1) excessive for a child's age and gender and (2) associated with significant impairment (important themes that will be emphasized throughout this book).

DON'T FORGET

Although diagnostic criteria are presented in a categorical format, ADHD is a dimensional disorder. In other words, rather than being an *absolute* "yes" or "no," assessing each symptom is a *relative* decision of "how much compared to peers."



In addition to dimensionality, variability is a hallmark feature of ADHD. The varied set of behavioral and cognitive problems involved in ADHD means that there is no single, unified presentation that fits all children who receive the diagnosis. Some children with ADHD exhibit lots of problems related to inattention but not many hyperactive or impulsive behaviors.[1] Others

[1] To further complicate matters, some contend that this group includes many children that may have a distinct attention disorder from that seen in ADHD. Referred to as *sluggish cognitive tempo* (SCT), the condition is marked by a "spacey" or "daydreamy" and lethargic presentation and is distinguishable from ADHD with respect to associated problems, executive functioning deficits, comorbidity patterns, and treatment response (Barkley, 2013; McBurnett, Pfiffner, & Frick, 2001). Although SCT was proposed for consideration (Hartman, Willcutt, Rhee, & Pennington, 2004), it is not included in the DSM-5 either as a presentation of ADHD or as a separate disorder.

(particularly among younger age groups) (Lahey, 1993; Willcutt, 2012) present with the opposite pattern. Most children diagnosed with ADHD have difficulties in both categories, even though one category may be dominant. The severity of symptoms and the nature and degree of associated impairment also vary across cases, adding further to the heterogeneity among youngsters with this disorder. Moreover, inattention, hyperactivity, and impulsivity are multifaceted constructs that can manifest in a wide variety of forms. Clearly, to say "not all children with ADHD look the same" is a gross understatement.

The variability seen in ADHD occurs not only *across* children with ADHD, but also *within* a given child (Castellanos et al., 2005). Symptoms of ADHD typically fluctuate across time, persons, tasks, situations, and settings (see Barkley, 2006, for a detailed discussion of the impact of these factors). It is this inconsistency from hour-to-hour, day-to-day, and task-to-task that often leads others to view those with ADHD as simply being "lazy" or as not trying hard enough. However, rather than suggesting the absence of disorder, such inconsistency is highly characteristic of ADHD. Indeed, some consider variability in performance across time and contexts to be the essence of ADHD (Brown, 2013). Although children with the disorder tend to stand out from their peers in most settings, the visibility and impact of symptoms can ebb and flow considerably depending upon the context and, in particular, the degree to which it requires sustained effort and focus, restraint, and self-control. Thus, free-play and other low-demand settings often reduce the expression of ADHD symptoms, as do novel, stimulating, and engaging tasks that provide clear and frequent feedback (whether reinforcing or corrective) closely tied to the child's performance. Video games are often cited as an example of an activity that elicits improved attention, though children with ADHD have been found to still be more restless and inattentive than their peers while playing such games and to perform less well on them (Tannock, 1997). In contrast, symptoms of ADHD are often very evident in highly familiar settings with low levels of individualized attention and feedback, as well as when tasks are of little interest but high demand (e.g., requiring planning, organization, focus, sustained effort). For example, a student who seems engaged during sports practice with a very involved coach may look inattentive during independent schoolwork and homework.

The variability associated with ADHD is further justification for seeking information about the child's functioning across multiple settings and tasks, with input from more than one observer. In addition to establishing pervasiveness and persistence of symptoms, multiple sources of input help determine if an example of good attention is an exception or the rule for a child. As explored in later chapters, it is important not to rule out ADHD on the basis of the child

performing adequately (or even well) in some settings or not displaying characteristic symptoms consistently across every context.

DON'T FORGET

Two Key Concepts for ADHD

1. The features of ADHD are dimensional (not categorical); they vary along a continuum in the general population.
2. Symptoms of ADHD have a variable presentation (across those with the diagnosis and within an individual). ADHD can look very different across children with the disorder. Inconsistency in symptom expression and in performance within an individual is a hallmark feature of ADHD.

CORE FEATURES

DON'T FORGET

Although the name has changed, the core features of ADHD have remained constant for over 100 years: inattention, hyperactivity, and impulsivity.

Clinical descriptions of ADHD have been remarkably stable for more than a century, including developmentally aberrant and impairing levels of inattention, impulsivity, and hyperactivity that emerge in childhood and persist over time and across situations. Each of these three core features is described briefly here, with greater detail about related diagnostic criteria provided in Chapter 2.

Although hyperactivity and impulsivity are discussed separately in what follows, it is important to note that it is difficult to separate these two constructs. In fact, there is wide consensus based on considerable research evidence that ADHD (at least among children and adolescents) is best defined in terms of two (rather than three) symptom domains: (1) *inattention* and (2) *hyperactivity/impulsivity* (accordingly, this is how ADHD is defined in the current edition of the DSM; American Psychiatric Association, 2013; see Chapter 2). The bases for this relate not only to the high correlation between hyperactive and impulsive features of the disorder (i.e., children who have symptoms of one tend to also display the other) and the replication of this same two-factor structure across ethnic and cultural groups (Bird, 2002; Reid, Casat, Norton, Anastopoulos, & Temple, 2001; Toplak et al., 2012), but also to the fact that the inattentive and hyperactive/impulsive dimensions differentially predict the types of impairments

children experience, their comorbid conditions, and neuropsychological findings. For example, children with inattentive features are more likely to have academic problems (including learning disorders), internalizing symptoms, and neuropsychological weaknesses related to working memory, processing speed, and response variability, whereas those with hyperactive/impulsive symptoms are more likely to show oppositional/disruptive behavior, conduct problems, aggression, peer rejection, and accidental injury (American Psychiatric Association, 2013; Barkley, 2006; Tannock & Brown, 2009; Willcutt & Bidwell, 2011; Willcutt et al., 2012).

DON'T FORGET

Clinical experience, research data, and diagnostic guidelines support two categories of ADHD symptoms: *inattentive* and *hyperactive/impulsive*. These two categories help predict associated problems and what interventions a child may need.

Inattention

Attention is a multifaceted construct (Stauss, Thompson, Adams, Redline, & Burant, 2000). It includes components such as arousal and alertness, selective or focused attention and distractibility (the ability to attend to particular stimuli while ignoring competing, irrelevant stimuli), attention span (the amount of information that one can attend to at one time), and sustained attention or vigilance (the persistence of focus over time), among others. Of these components, children with ADHD appear to have the greatest difficulty with sustained attention (Douglas, 1983; Newcorn et al., 2001; Swaab-Barneveld et al., 2000). As a consequence, they often struggle to maintain their concentration and effort and to persist with tasks, particularly those they experience as boring, tedious, or repetitious. They are also more distractible than other children. Not only are they more likely than peers to respond to irrelevant events around them, but they also take longer to get back on-track (if they do at all). This distractibility can be compounded by a tendency to become bored quickly and to actively seek opportunities to escape the current task. People with ADHD, in general, are highly drawn to activities that seem more appealing than what they are engaged in at the moment.

Impulsivity

Another core feature of ADHD is difficulty with impulse control (Gordon, 1979; Newcorn et al., 2001; Nigg, 2001; Scheres et al., 2004). More so than inattention,

poor inhibition of impulsive responding has been found to distinguish those with ADHD both from typically developing children and from children with other disorders (Barkley, 1997; Nigg, 2001). This poor inhibition impacts behavior, speech, and cognition. People with ADHD may appear to act upon whatever comes to mind without prior consideration for the likely consequences for themselves or others. This may be reflected in their taking shortcuts in their work (often leading to mistakes), things they want from others (without permission), and unnecessary risks (often on a whim or in response to a dare). The propensity to take chances where others would think twice is particularly troubling, as it increases risk for a variety of serious consequences, ranging from accidental injuries to car accidents (see discussion in "Course and Outcome," section). Children with ADHD often begin tasks before instructions have been completed and without proper consideration of what is required (with predictable consequences for their performance).

Poor inhibition makes waiting highly aversive for many people with ADHD (Solanto et al., 2001). A youngster with ADHD might have difficulty waiting for her turn in a game or conversation, for others to finish what they are saying, when lining up for lunch or recess, or for a meal, class, or religious service to end. Delaying gratification is also a challenge. When given the choice, youth with ADHD tend to opt for a smaller but immediately available reward (whether it be a toy, snack, or money) rather than waiting for a larger payoff (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986; see Willcutt & Bidwell, 2011, for a summary of these "delay aversion" studies).

Verbal impulsivity in ADHD may include blurting out answers prematurely, interrupting others' conversations, and voicing things best left unsaid. These behaviors often lead to hurt feelings, anger, and the perception that the person with ADHD is rude and insensitive (which in turn exacerbates social problems often associated with the disorder). Some of the attention problems described earlier can also be related to inhibitory deficits, in that individuals with ADHD may struggle to stay on task due to difficulties inhibiting unrelated thoughts (Shaw & Giambra, 1993).

Hyperactivity

Hyperactivity is the third core feature of ADHD. Like other symptoms of the disorder, it tends to vary depending upon setting and prevailing demands (e.g., worsening in less stimulating environments). Many (though not all) children with ADHD exhibit excessive motor movement. Studies using

objective measures of activity level show them to be, on average, significantly more active than their peers, including during sleep (Porrino et al., 1983; Teicher, Ito, Glod, & Barber, 1996). In addition to higher levels of activity, many children with ADHD have difficulty adjusting their activity level to meet the demands of the setting they are in (e.g., a school assembly) or the tasks before them (e.g., walking in an orderly line while transitioning between classrooms).

Like inattention and impulsivity, hyperactivity can take many forms, ranging from the restless child who taps his fingers and feet, fidgets, and plays with objects while remaining seated to the whirling dervish who blazes chaotically through space with a driven, accelerated quality. Hyperactivity can also be expressed vocally, via excessive talking (often saying too much, too quickly, and too loudly), humming, singing, or making other sounds in situations that call for quiet. A quality shared by many hyperactive symptoms is that they lack direction or purpose and are extraneous to the task at hand.

ASSOCIATED FEATURES AND COMORBIDITY

In addition to the core features of the disorder, children with ADHD experience a range of additional problems to a greater degree than typically developing peers. These associated features, reviewed below, span cognitive academic, behavioral, emotional, social, developmental, and medical issues. Although neither always present nor part of the diagnosis, these features add to the heterogeneity of ADHD and often reflect or contribute to the impairments associated with the disorder.

Problems associated with ADHD can also be reflected in comorbid diagnoses. Children with ADHD are highly likely to present with one or more additional mental health disorders (for a review, see Brown, 2008; Rosen, Froehlich, Langberg, & Epstein, 2011; Taurines, Schmitt, Renner, Conner, Warnke, & Romanos, 2010). Some studies estimate that 80% or more of children with ADHD have one or more coexisting disorders (Kadesjo & Gillberg, 2001; Pfiffner et al., 1999; Wilens et al., 2002; Willcutt & Bidwell, 2011). Although figures vary across studies, comorbidity is the rule rather than the exception among children with ADHD who seek clinical services. These high comorbidity rates make it crucial that any evaluation of ADHD include assessment for possible comorbid conditions (American Academy of Child and Adolescent Psychiatry Work Group on Quality Issues, 2007b; American Academy of Pediatrics, 2011). Common comorbidities are described in the following sections, and summarized in Rapid Reference 1.3.

DON'T FORGET

Comorbidity refers to the co-occurrence of two or more different disorders. The high comorbidity rates for children with ADHD and the potential for other disorders to "mimic" the symptoms of ADHD mean that ADHD evaluations must include assessment for other possible conditions.

Cognitive and Academic

Children with ADHD fall along the range of intellectual ability, from gifted to typical to intellectually impaired. Core difficulties related to attention and inhibition are intertwined with the neurocognitive processes subsumed under the umbrella term *executive functions*. (For a review of the literature on executive functions and deficits, see Hunter & Sparrow, 2012.) Although no consensus exists as to the definition of executive functions, they are generally thought to include such processes as working memory (keeping information in mind until it is needed or consolidates), planning, response suppression, mental flexibility, set shifting, and the monitoring of one's actions and performance over time, among others. In addition to their deficits in the core symptom domains of vigilance and response inhibition (factors that some include among the executive functions), children with ADHD have been found to have particular difficulties related to working memory and somewhat smaller weaknesses in other executive functions, such as set shifting and some measures of planning (Martinussen, Hayden, Hog-Johnson, & Tannock, 2005; Nigg, 2013a; Willcutt & Bidwell, 2011; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).[2] They have also been found to be impaired relative to peers in their abilities to estimate, track, and manage time (Barkley, 2006). These executive functioning impairments may contribute to the modest but significant decrements (averaging 9 points) children

[2] Because executive functions are thought to underlie many of the abilities that enable self-regulation (e.g., planning how we approach tasks as well as for the future, maintaining focus and organization over time, suppressing responses that impede goals or task completion, monitoring our performance, shifting approaches when needed), some contend that they represent the foundation of ADHD (Barkley, 2012; Brown, 2013). Failing to support this view, however, is the fact that substantial numbers of individuals with ADHD do not demonstrate executive functioning deficits, as measured by objective tests. It is also worth noting that current tests of executive functioning are neither sensitive nor specific enough to use as diagnostic indicators of ADHD (Smith, Barkley, & Shapiro, 2007; Willcutt & Bidwell, 2011).

with ADHD demonstrate on standardized intelligence tests (Frazier, Demaree, & Youngstrom, 2004).

Academic underachievement is typical for children with ADHD. Nearly all underperform relative to their ability levels and many (particularly among those who are referred for services) are doing poorly in school. Students with ADHD tend to be both less productive and less accurate in their schoolwork than other children (Pfiffner & Barkley, 1990) and, on average, they score lower on standardized academic achievement tests than their classmates (Brock & Knapp, 1996; Casey, Rourke, & Del Dotto, 1996; Fischer, Barkley, Fletcher, & Smallish, 1990; Frazier, Youngstrom, Glutting, & Watkins, 2007; Semrud-Clikeman et al., 1992). Children with ADHD receive special education and related services, repeat grades, and drop out of school at much higher rates than youth without the disorder, and fewer go on to post-secondary education (Barkley, DuPaul, & McMurray, 1990; Barkley, Fischer, Edelbrock, & Smallish, 1990; Hinshaw, 1992; Klein & Mannuzza, 1991).

The research literature consistently indicates high rates of comorbidity between ADHD and specific learning disorders. A careful literature review found that children with ADHD are three to four times more likely to have a learning disorder than their peers in the general population (DuPaul & Stoner, 2003). An estimated 12–60% of students with ADHD are likely to have a math disorder, 8–40% a reading disorder, up to 60% a disorder of written expression, and 12–27% a spelling disorder (see reviews in Barkley, 2006, and Tannock & Brown, 2009). More globally, recent estimates suggest that 25% or more of children with ADHD have a comorbid learning disorder (Tannock & Brown, 2009; Willcutt et al., 2012). This common comorbidity highlights the importance of considering cognitive testing when assessing students for ADHD (see Chapter 4 for discussion).

Externalizing/Behavioral

Externalizing behavior problems associated with ADHD involve a host of oppositional behaviors including willful noncompliance or defiance in response to adult directives or rules, disruption of ongoing activities, argumentativeness, stubbornness, temper outbursts, verbal hostility, and physical aggression (Connor, Steeber, & McBurnett, 2010; Loney & Milich, 1982). A minority of children with ADHD develop more serious conduct problems such as lying, stealing, fighting, truancy, and vandalism (Pfiffner et al., 1999). Disruptive behavior disorders are the most common coexisting conditions among children with ADHD; about 50% of children with ADHD have oppositional defiant disorder (ODD) and over 20% have conduct disorder (CD) (Nigg, 2013b). Even in the

absence of volitional oppositionality, problems with attention and impulsivity can result in poor compliance with rules and instructions at home, school, and in recreational settings (Barkley, 2013).

Internalizing/Emotional

Children with ADHD frequently struggle with emotional regulation. They are often described as "wearing their emotions on their sleeves," and as being prone to more intense and labile displays of emotions than children without the disorder (Skirrow, McLoughlin, Kuntsi, & Asherson, 2009). Their difficulties regulating emotions are often particularly salient with regard to managing frustration or disappointment. They have also been found to display higher levels of anger and more symptoms of anxiety and depression than other children (Barkley, 2006; Carlson & Meyer, 2009; Tannock, 2009).

Approximately 25–35% of children with ADHD are estimated to have an anxiety disorder, with high rates of generalized anxiety disorder, separation anxiety disorder, and social phobia (Langberg, Froehlich, Loren, Martin, & Epstein, 2008; Tannock, 2009). Estimates of the risk for depressive disorders among youth with ADHD vary widely but often average around 25–30% (Barkley, 2006), with the vast majority of these cases involving major depressive disorder rather than dysthymic disorder (Wilens et al., 2002). The relationship between ADHD and bipolar disorder in children is controversial, in part due to unresolved questions related to the nature of bipolarity in children, the symptom overlap between the two disorders, and the dramatic increases in the number of children being diagnosed with bipolar disorder over recent decades (see Carlson & Meyer, 2009, for a discussion). The new diagnosis of disruptive mood dysregulation disorder (American Psychiatric Association, 2013) was introduced in part as a response to the apparent overdiagnosis of bipolar disorder among youth. It appears likely that many who receive this diagnosis will also meet criteria for ADHD, but the prevalence of disruptive mood dysregulation disorder among children with ADHD is currently unknown.

Interpersonal/Social

For children with ADHD, interactions with parents and teachers tend to be marked by elevated rates of negativity, conflict, and stress (DuPaul, McGoey, Eckert, & VanBrakle, 2001; Johnson & Mash, 2001). Problems with peer relations are also common, including difficulties forming and maintaining friendships (see Hoza, 2007; McQuade & Hoza, 2008; Tomb, Linnea, McQuade, & Hoza, 2011, for reviews).

Children with ADHD are poorly accepted or actively rejected by peers at much higher rates than non-disordered children and children with other psychiatric disorders (Asarnow, 1988; Blachman & Hinshaw, 2002; Gaub & Carlson, 1997; Hinshaw & Melnick, 1995; Hoza et al., 2005; Mikami, 2003; Milich & Landau, 1982; Pelham & Bender, 1982). Indeed, 50–80% of children with ADHD appear to be rejected by peers (Hoza, 2007; Hoza et al., 2005; Tomb et al., 2011). Moreover, the peer rejection often experienced by children with ADHD usually emerges after very brief periods of interaction (Erhardt & Hinshaw, 1994; Pelham & Bender, 1982), remains stable over time, and predicts adverse long-term outcomes (e.g., academic failure, school dropout, criminality, or psychopathology) (Bagwell, Schmidt, Newcomb, & Bukowski, 2001; Parker & Asher, 1987). In addition to continuing to experience elevated rates of peer rejection (Bagwell, Molina, Pelham, & Hoza, 2001), adolescents with ADHD have been found to be more likely to bully others and to be bullied than those without the disorder (Unnever & Cornell, 2003).

Substance Use and Abuse

Early childhood behaviors associated with ADHD (e.g., hyperactivity, impulsivity, poor persistence, "novelty seeking") have been found to predict early onset substance use among teenagers and alcohol dependence in early adulthood (Caspi, Moffitt, Newman, & Silva, 1996; Masse & Tremblay, 1997). Few studies have looked at alcohol use among adolescents with ADHD and their results have been inconsistent. However, those using developmentally sensitive measures tend to suggest more frequent and heavy drinking compared to peers without ADHD by the late teenage years (see Molina, 2011, for a summary). Adolescents (as well as young adults) with ADHD are roughly twice as likely to smoke cigarettes than their peers without the disorder (Molina, 2011) and also appear to be more likely to abuse drugs or alcohol (see, for example, Katusic et al., 2005). Furthermore, longitudinal studies following children with ADHD into adulthood show ADHD to be associated with an earlier onset and overall higher risk for substance use disorders (Hechtman & Weiss, 1986; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Molina & Pelham, 2003; Molina et al., 2007; Wilens et al., 2011). Although coexisting conditions such as conduct disorder and bipolar disorder increase this risk substantially, ADHD represents an independent risk factor for substance use disorders in the absence of these comorbidities (Kollins, 2008; Wilens, 2009; Wilens et al., 2011).

Considerable controversy has surrounded the issue of whether the treatment of ADHD with stimulant medications increases the risk for future substance abuse. Interestingly, the available data suggest not only that stimulant therapy does not

increase the risk of substance use disorders, but that it may protect against them, particularly among adolescents (Katusic et al., 2005; Kollins, 2008; Wilens, 2009; Wilens, Faraone, Biederman, & Gunawardene, 2003).

Developmental and Medical

Developmental and health problems occurring at elevated rates among children with ADHD include delays in self-help and other adaptive functioning skills (Stein, Szumowski, Blondis, & Roizen, 1995), poor motor coordination (Barkley et al., 1990; Harvey & Reid, 2003; Kadesjo & Gillberg, 2001), speech and language deficits (Barkley et al., 1990; Tannock & Brown, 2009), sleep difficulties (Miano, 2012; Owens, Brown, & Modestino, 2009), obesity (Panzer, 2006), and various forms of accidental injury (Barkley, 2001). Children with ADHD have been found to utilize medical services in general and emergency room services in particular at higher rates than youth without the disorder (Leibson, Katusic, Barbaresi, Ransom, & O'Brien, 2001).

With respect to comorbidities, developmental coordination disorder (Kadesjo & Gillberg, 2001) and, to a lesser extent, tic disorders (Peterson, Pine, Cohen, & Brooks, 2001; Spencer et al., 1999) appear to occur at higher rates among children with ADHD than those without the disorder. Information on the prevalence of autism spectrum disorder among children with ADHD is lacking, in part because the presence of a pervasive developmental disorder (PDD) generally precluded the DSM diagnosis of ADHD until the recently released DSM-5 (American Psychiatric Association, 2013).

Rapid Reference 1.3

Comorbidity Rates in ADHD

High comorbidity rates are known to be common among children with ADHD. Precise estimates of how frequently specific disorders accompany ADHD are elusive because results vary considerably across studies (likely reflecting differences in their samples and diagnostic methods). Nonetheless, the following ranges of estimates (from Willcutt & Bidwell, 2011) provide a sense of the relative frequency of comorbid disorders within the ADHD population:

- ODD: 30–60%
- CD: 20–50%

(*continued*)

(*Continued*)

- Learning disorders: 20–40%
- Anxiety disorders: 15–30%
- Depression: 15–30%

As you might imagine, comorbidity rates for some disorders vary by ADHD presentation. The following table presents estimated frequencies of comorbid disorders by DSM-IV-TR subtypes, based on a large meta-analysis of studies (Willcutt et al., 2012). Estimated frequencies of these disorders in children without ADHD are also provided for comparison purposes.

	Estimated Percentage of ADHD Cases with the Comorbid Disorder			Estimated Percentage of Non-ADHD Cases with the Comorbid Disorder
	Combined	Inattentive	Hyperactive/ Impulsive	
ODD	51.8%	4.9%	42.9%	4.6%
CD	21.6%	7.1%	14.9%	1.3%
GAD	11.3%	10.4%	14.6%	2.9%
SAD	13.5%	8.7%	10.6%	2.0%
MDD	9.8%	9.5%	7.6%	1.5%
Bipolar	6.9%	3.2%	6.4%	< 1.0%
LD	24.2%	29.1%	17.9%	8.4%
Speech/language	14.8%	17.8%	13.9%	10.7%
Tic disorder	15.8%	12.1%	22.6%	4.7%

ETIOLOGY

The cause of ADHD has been a matter of considerable speculation, study, and debate since it was first recognized. Although definitive causes have yet to be established, scientific investigation into the etiology of ADHD over recent decades has unearthed considerable (though still indirect) evidence that permits certain conclusions to be drawn while identifying promising paths for future research (for detailed discussions, see Nigg, 2006, 2013b; Thapar, Cooper, Eyre, & Langley, 2013).

ADHD Has Multiple Causes

There is consensus among scientists who study ADHD that the disorder has multiple causes. In this sense, the heterogeneity and complexity that mark the

presentation (or phenomenology) of the disorder apply to its etiology as well. Like many disorders, the causes of ADHD are multifactorial both in the sense that various factors may converge to lead to ADHD in a given child (e.g., genetic vulnerability interacting with prenatal malnutrition and exposure to alcohol) and that different children with ADHD may have developed the disorder via highly distinct pathways (e.g., an extreme temperament vs. early life exposure to environmental pesticides).[3]

ADHD Is Neurobiological

Scientific studies on etiology converge on the fact that ADHD is a neurobiological disorder. Considerable evidence now points to both structural and functional differences between the brains of those with and without ADHD, particularly involving the prefrontal region and its connections with various neural circuits implicated in self-regulation and executive functioning. With respect to brain structure, overall brain volume and numerous brain regions representing key frontal and subcortical structures have been found to be smaller in individuals with ADHD (e.g., prefrontal cortex, basal ganglia/striatum, cerebellum, caudate nucleus, corpus callosum) (Nakao, Radua, Rubia, & Mataix-Cols, 2011; Nigg, 2013b; Valera, Faraone, Murray, Seidman, 2007; Willcutt et al., 2012). Not only do key brain structures appear to be smaller and to remain so throughout development (Castellanos et al., 2002), but also children with ADHD appear to lag (on the order of 2 to 3 years) behind their age-mates in their brain maturation, particularly with respect to their frontal lobe regions (Shaw et al., 2007).

With regard to neural function, numerous parts of the brain implicated in self-regulation have been demonstrated to be less active in individuals with ADHD. Multiple lines of evidence, including studies of electrical activity (as measured by EEG devices) (Klorman et al., 1988; Loo & Barkley, 2005), blood flow (Lou, Henriksen, & Bruhn, 1984), and neural images (via PET and fMRI scans) (Dickstein, Bannon, Castellanos, & Milham, 2006; Paloyelis, Mehta, Kuntsi, & Asherson, 2007) demonstrate lower activity in the prefrontal area of the brains of people with ADHD as compared to those without the disorder. In fact, these lower activity levels, or brain-activation deficits, have been found in virtually all regions of the prefrontal cortex (as well as other areas of the brain) (Dickstein, Bannon, Castellanos, & Milham, 2006). The pattern of brain-activation deficits revealed to date suggests that ADHD involves aberrant functioning in both

[3] The term *equifinality* is sometimes used to capture the idea that there can be multiple pathways to a particular outcome (e.g., ADHD) (Hinshaw, 2013).

fronto-striatal and frontal-parietal neural circuits (Durston, Tottenham, Thomas, Davidson, Eigsti, Yang, Ulug, & Casey, 2003; Nigg, 2013b). Findings from multiple studies using a newer imaging technique (known as *diffusion tensor imaging* or DTI) implicate altered white matter microstructure in ADHD, suggesting that widespread brain processes (such as synaptic signaling and myelin formation) rather than isolated neural circuits might be involved in the disorder (Konrad & Eickhoff, 2010; Nigg, 2013b).

Factors That Change the Brain

What causes these abnormalities in the structure, function, and maturation of prefrontal areas of the brains of persons who develop ADHD? Investigations over recent decades have identified a number of possibilities.

Genes

Genetic factors have emerged as the single largest contributor to ADHD. Family aggregation, twin, and adoption studies have shown ADHD to be a highly heritable condition, with genes accounting for approximately 75% or more of the differences among people with respect to their ADHD symptoms (Faraone et al., 2005; Nikolas & Burt, 2010; Thapar et al., 2013; Willcutt, in press). Moreover, ADHD is polygenetic; many genes contribute to its expression. The search for these genes comprises an active area of research that to date has identified multiple genes associated with ADHD (Gizer, Ficks, & Waldman, 2009). A number of these implicated genes impact pathways related to dopamine (a neurotransmitter believed to be critical to the regulation of attention and impulsivity) (Nigg, 2013b). Other genes of interest for ADHD research are involved in brain growth, neuronal migration, and neuronal connections (Barkley, 2013), including those that impact norepinephrine, serotonin, acetylcholine, GABA, and histamine (Aboitz & Castellanos, 2011), as well as the MAOs and nicotinic receptors (Gizer, Ficks, & Waldman, 2009).

Genes play an important (though by no means the only) role in influencing temperament (Neuhaus & Beauchaine, 2013). A certain percentage of those with ADHD appear to represent individuals at the extreme high end of the temperament continuum with respect to traits such as activity level, impulsivity, and sensation seeking (or, conversely, at the extreme low end with respect to self-regulation) (Marcus & Barry, 2011). Although not inherently pathological, such genetically shaped extreme temperaments become impairing in a broad social context that emphasizes academic and occupational achievement and expects conformity to indoor, sedentary, desk-based work (see Hinshaw &

Scheffler, 2013, for a compelling discussion of how the advent of compulsory education and intensifying performance pressures have spurred dramatic increases in ADHD).

Environmental Insults

Numerous environmental factors spanning pre- and perinatal events, neurotoxic substances, dietary factors, and various forms of psychosocial adversity have been found to be associated with ADHD (see Froehlich et al., 2011, for a review). Although determining causality is difficult, some have been well established as risk factors for ADHD. These include maternal alcohol, tobacco, and substance use during pregnancy, maternal stress during pregnancy, low birth weight and prematurity, exposure to environmental toxins such as pesticides and lead, and severe deprivation with respect to early care giving (other environmental factors, such as malnutrition and family adversity have been found to correlate with ADHD but are not yet considered risk factors for the disorder) (Thapar et al., 2013).

Some ADHD cases might emerge due to brain injuries suffered during early pre- or postnatal development (e.g., from central nervous system infections, pregnancy or birth complications, and head trauma). However, such demonstrable brain damage is thought to account for only a small subset of children with the disorder (Barkley, 2013).

The Interplay of Genes and Experience

In some cases, the influence of multiple genes may be adequate to produce ADHD symptoms of sufficient severity to merit a diagnosis. In other instances, environmental insults to the brain may be sufficient to cause ADHD. However, the causal models that are receiving the most attention (and which may account for the most cases) are those that consider both genetic and biologically compromising environmental factors and, importantly, how they influence one another. Such models invoke the concepts of *gene–environment interaction* (wherein either environmental factors moderate the effects of genes on behavior or genes moderate the effects of environmental factors) and *epigenetics* (wherein environmental and experiential factors alter the ways genes are expressed) (Beauchaine & Gatzke-Kopp, 2013).

Gene–Environment Interactions

There are a number of possible ways in which genetic and environmental factors might interact to produce ADHD. First, genes may convey a risk for ADHD but exposure to certain biological or experiential stressors is necessary to activate that

risk and set the child on a path toward the disorder (Nigg, 2013b). Thus, two children may be born with the same genetic vulnerability to ADHD. One has the misfortune of being exposed prenatally to alcohol, which triggers his vulnerability and sets him on a course toward ADHD. The other, spared of such exposure, develops normally.

Second, genetic and environmental factors may *combine* to elevate one's risk for ADHD. For example, genes associated with ADHD and maternal smoking during pregnancy each independently increase the risk for the disorder. However, the combination of both factors increases the likelihood of ADHD substantially (Neuman et al., 2007).

Third, certain genes (or gene combinations) can render some individuals susceptible to particular neurotoxic factors in the environment (Nigg, Nikolas, & Burt, 2010). This can help to explain why established environmental risk factors do not affect all children.[4] Take, for example, two children who are born close to commercial farms where pesticides associated with ADHD (organophosphates) are used. One child's genes make her vulnerable to the toxic effects of these chemicals in ways that lead to the development of ADHD symptoms. The other, exposed to the same pesticides but lacking that genetic susceptibility, escapes harm.

Epigenetics

Epigenetic changes represent an aspect of gene–environment interaction that has been the subject of increasing scientific focus. Epigenetics concerns how experiences (e.g., significant stressors, diet, exposure to environmental toxins) can alter the expression of genes, impacting outcome for both the individual and future generations (Nigg, 2012). Timing is a crucial variable in epigenetics, as it seems that periods of rapid development (e.g., gestation) are associated with greater epigenetic vulnerability (Mill & Petronis, 2008). Thus, experiential factors occurring during particular developmental periods may change DNA structure in ways that alter, for better or worse, the expression of genes related to the development of ADHD symptoms. Evidence for epigenetic effects on psychopathology (including ADHD) is only beginning to emerge (with much of it based on animal models) (Kubota, Miyake, & Hirasawa, 2012; Neuhaus & Beauchaine, 2013). However, this line of research holds great promise for deepening our understanding of how environmental and contextual forces may often enable, preclude, and shape the expression of this biologically based disorder.

[4] This phenomenon illustrates the concept of *multifinality*, wherein a given risk factor may lead to many different outcomes depending on a variety of intervening factors (e.g., developmental level, genetic susceptibilities) (Cicchetti & Rogosch, 1996).

SPECIAL TOPIC: WHAT DOES NOT CAUSE ADHD?

Although the etiology of ADHD is complex and not fully understood, scientific evidence suggests that certain factors do not represent significant causes of the disorder (Barkley, 2013; Thapar et al., 2013). Speculation that hormonal deficiencies, vestibular problems, and yeast sensitivity are causes of ADHD has not been supported by research findings. Beliefs that family environment factors (e.g., poor parenting, negative parent–child interaction patterns, chaotic homes) cause ADHD have likewise not received empirical support (noteworthy, however, is that such factors are among the known causes of ODD and CD). Some have conjectured that high levels of television exposure might cause ADHD. Although ADHD may be associated with watching more TV, such correlation does not prove causation (e.g., it may well be that ADHD is associated with more TV watching since it requires less effort and sustained attention than most alternative activities). In any case, convincing data that TV exposure causes ADHD are currently lacking.

Various dietary factors have been proposed to cause ADHD or influence its symptoms. Although severe malnutrition can adversely affect the developing brain, the effects of less extreme dietary deficiencies are less clear (Sinn, 2008). Deficiencies related to zinc, magnesium, and polyunsaturated fatty acids have all been investigated in relation to ADHD, but there is not sufficient evidence to conclude that they play a causal role (Thapar et al., 2013). Despite popular lore, the evidence does not support sugar as a cause of ADHD. For many years, hypotheses that synthetic food additives might contribute to ADHD were dismissed as lacking scientific credibility. However, respected recent meta-analyses documenting that food coloring can influence ADHD symptoms and that a subset of children with ADHD may respond to dietary interventions are likely to revive interest in this area (Nigg, Lewis, Edinger, & Falk, 2012; Schab & Trinh, 2004).

EPIDEMIOLOGY

Epidemiology concerns the distribution patterns of a disorder or disease in the population and various factors related to those patterns. It is important to understand the patterns of ADHD, including how common it is, how it varies with gender and culture, and its typical course and outcome. Knowing the prevalence rates of ADHD and other disorders helps you consider how likely a diagnosis might be for a given child (see also Chapter 5, "Command of Child Psychopathology"). Understanding differences in how ADHD presents in boys versus girls guides where and how you look for symptoms and impairment. It is also important to be aware of cultural factors impacting ADHD, including geography, social class, and ethnicity, as these dictate certain changes in your assessment, interpretations of results, and communication of findings (see Chapter 3). Finally, knowledge of the typical course for ADHD will help you

recognize when the diagnosis is appropriate; outcome data suggest possible impairments to consider and affect your prognostic statements.

Prevalence

ADHD is a common condition, with a worldwide prevalence in children and adolescents of around 5.3% (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). One U.S. survey reported a 1-year prevalence rate for adolescents of 8.7% (Merikangas et al., 2010), suggesting that in excess of 3 million minors in the United States could have the disorder.

It is important to distinguish between prevalence rates and diagnostic rates. In the United States, the number of children being diagnosed with ADHD has been rising rather dramatically over recent decades (e.g., the percentage of 4- to 7-year-olds who had ever received a diagnosis of ADHD climbed from 7.8% in 2003 to 11% by 2011–2012, which translates to over 6 million youth) (Boyle et al., 2011; Hinshaw & Scheffler, 2013; Schwarz & Cohen, 2013). Combined data from multiple U.S. federal agencies indicates ADHD to be the most common mental health diagnosis received by youth ages 3 through 17 (Perou et al., 2013). Although the trend in diagnosing ADHD has been dramatically upward, it is unclear if this reflects a true rise in prevalence of the disorder.

Gender

ADHD, like nearly all of the neurodevelopmental disorders, is more common in boys than girls. Figures vary but the best estimates suggest that ADHD is diagnosed about three times more often among boys relative to girls during childhood (Hinshaw & Blachman, 2005; Polanczyk & Jensen, 2008). However, for reasons that are unclear, this gender disparity declines over development such that, by adulthood, the rates of ADHD among males and females begin to even out (American Psychiatric Association, 2013; Kessler et al., 2006).

After decades of scientific neglect, studies have begun to examine ADHD as it presents in females (see, for example, Hinshaw et al., 2012). Overall, the expression of ADHD in males and females is quite similar, although girls have been found to have generally lower rates of core symptoms (Gershon, 2002; Newcorn et al., 2001). In comparison to males, females are also more likely to show predominantly inattentive features (American Psychiatric Association, 2013). Although girls certainly display symptoms from the hyperactive-impulsive domain, those features appear somewhat more likely to appear in boys (Hinshaw & Blachman, 2005).

With respect to associated problems, girls exhibit less aggressive behavior (and possibly less substance abuse) than boys but appear to show more internalizing problems, including depression (Hinshaw & Scheffler, 2013). Rates of externalizing disorders (Rucklidge, 2010), specifically disruptive behavior disorders, appear to be lower in girls with ADHD than boys with ADHD, which may lead to lower referral rates for girls than boys (Biederman et al., 2002). Otherwise, girls and boys appear to experience similar levels of serious and persistent academic, behavioral, and social problems (Hinshaw et al., 2012).

Culture

ADHD is universal; it has been found to exist in every country and in every ethnic group studied to date (Bird, 2002; Faraone, Sergeant, Gillberg, & Biederman, 2003; Polanczyk et al., 2007). Moreover, there is no evidence to suggest that ADHD appears to differ in significant biological ways by race or ethnicity (Bussing & Gary, 2011). The cross-cultural validity of ADHD is supported by findings that its prevalence, factor structure, and biological correlates are similar in developed and undeveloped nations (see Rohde et al., 2005, for a review). The previously discussed two-factor (viz., Inattention and Hyperactivity/Impulsivity) structure of ADHD has been found to be similar not only across nations (Toplak et al., 2012) but also across African-American and European-American children in the United States (Reid et al., 1998).

Nonetheless, the rates at which ADHD is diagnosed (and treated), at least in the United States, vary by geography, social class, and ethnicity (Hinshaw & Scheffler, 2013). In contrast to previous trends, (1) children at or near the poverty level are now more likely to receive an ADHD diagnosis than those from higher-income families, (2) African-American children are just as likely as, if not more likely than, Caucasian youth to receive an ADHD diagnosis; and (3) Hispanic youth are still less frequently diagnosed with ADHD than either African-American or Caucasian youngsters, but these differences have begun to decrease (Getahun et al., 2013; Visser, Bitsko, Danielson, & Perou, 2010). Although reduced economic and ethnic disparities in the detection and treatment of ADHD are certainly a welcome development, it will be important to examine whether the quality of evaluations and accuracy of diagnoses are comparable across groups, particularly those who experience systemic (as opposed to within-child) problems that can lead to behaviors that mimic ADHD symptoms.

In general, our understanding of ethnic and other sociocultural influences on the detection and treatment of ADHD symptoms is lacking (Miller, Nigg, &

Miller, 2009). This knowledge gap is particularly problematic as the U.S. population becomes increasingly diverse (Bussing & Gary, 2011). Cultural differences in a variety of factors, including attitudes toward ADHD and help seeking for mental health concerns, access to care, and even the meanings attached to behaviors of potential relevance to ADHD (e.g., calling out vs. remaining quiet in groups) may all impact the degree to which the disorder is accurately detected in diverse populations (see Chapter 3 for further discussion of cultural issues) (Lee & Humphreys, 2011; Nigg, 2013b).

Course and Outcome

The variability that characterizes so many aspects of ADHD also applies to its course and outcome. However, considerable research supports the conclusion that it is typically a chronic disorder associated with elevated risk for impairment across a wide range of functional domains.

Parents are likely to notice symptoms of ADHD by the time their child is 3 or 4 years old, if not earlier. (Some parents recall active, moody, and generally challenging temperaments as early as infancy.) Notably, symptoms of ADHD during the preschool years tend to be highly unstable and do not persist for many 3- and 4-year-olds (Willoughby, 2013). However, those preschoolers who have sufficiently severe symptoms to warrant a diagnosis, whose symptoms persist for at least a year, and whose interactions with parents are characterized by high degrees of negativity are likely to continue to have the disorder into their childhood and adolescent years (Barkley, 2013; Hinshaw & Scheffler, 2013).

ADHD was long regarded as a disorder that was "outgrown" in adolescence. This false conclusion that the disorder was self-remitting was likely due to the fact that its most visible feature, motoric hyperactivity, often declines with age. Nonetheless, problems with inattention, poor impulse control, subjective restlessness, poor planning, disorganization, and overall self-regulation (along with their associated impairments) are very likely to extend through the teen years and into adulthood (American Psychiatric Association, 2013; see Barkley, 2006, for a review).

Indeed, results of a number of prospective longitudinal studies (extending to as long as 33 years) have converged to reveal that despite changing presentations over time, ADHD is usually a chronic disorder (Barkley et al., 1990; Barkley, Fischer, Smallish, & Fletcher, 2002; Barkley, Fischer, Smallish, & Fletcher, 2006; Klein & Mannuzza, 1991; Klein et al., 2012; Weiss & Hechtman, 1993b). ADHD appears to persist into adolescence for up to 80% of youth who were diagnosed in

childhood and into adulthood for 50–65% (figures that increase if one considers ADHD in partial remission) (Barkley, 2013).

A large body of research shows that individuals with ADHD are at elevated risk for serious and wide-ranging impairments (some of which overlap with the associated problems discussed previously) (Barkley, 2006; Hinshaw, 2002; Hinshaw et al., 2012; Klein et al., 2012). Children and adolescents with ADHD have higher-than-average rates of accidental injuries, academic underachievement, school failure (e.g., expulsion, dropout), behavioral disturbance, including noncompliance, defiance, aggression, and serious conduct problems, peer rejection, family disharmony, and substance use. Serious additional risks affecting adolescents include earlier initiation of sexual activity, possibly lower use of birth control, more involvement in teen pregnancies, and higher rates of speeding tickets and auto accidents (Barkley et al., 2006; Thompson, Molina, Pelham, & Gnagy, 2007).

The increased responsibilities and expectations of adulthood appear to only expand the range of impairments experienced by individuals with ADHD. Compared to those without the disorder, adults with ADHD have been found to have lower levels of educational attainment and socioeconomic status, occupational challenges (lower job status, more instability, greater likelihood of being fired), problems managing money, marital and other relationship difficulties, and increased rates of substance use disorders, antisocial personality disorder, suicidal ideation and attempts, incarcerations, and hospitalizations (Barkley et al., 2006; Impey & Heun, 2012; Klein et al., 2012). Females with ADHD have been underrepresented in most of the follow-up studies conducted to date. However, recent longitudinal research with adolescent and young adult females with the disorder suggests rates of academic, social, and peer problems generally comparable to males, accompanied by alarming levels of self-injurious behaviors and suicide attempts (Hinshaw et al., 2012).

Although sobering, it is important to note that these adverse outcomes are not inevitable for those with ADHD. Approximately 10–33% of youth with ADHD show improved functioning by early adulthood, and about 50% of youth are much improved by midlife (some to the point of being symptom-free and indistinguishable from their peers) (Barkley, 2013; Klein et al., 2012; Weiss & Hechtman, 1993b). Clearly, ADHD is not disadvantageous in every setting and some, particularly once freed from the rigid structure of formal schooling, leverage their energy, curiosity, and comfort with risk into significant achievements and success. Identifying the factors that predict such resilience will be an important area for future research, with implications for enhancing both our assessment and treatment practices.

SUMMARY

Before approaching the assessment and diagnosis of ADHD, it is critical to be knowledgeable about the disorder. In addition to the core diagnostic constructs of inattention, impulsivity, and hyperactivity, there are a number of associated features and comorbidities that tend to occur. Your awareness of these common concerns across cognitive/academic, behavioral, emotional, social, developmental, and medical domains will improve your assessment of children for possible ADHD. These associated features can help identify impairment as well as suggest possible competing diagnoses or comorbidities. Your familiarity with current research on the causes of ADHD will enhance your differential diagnosis and improve your communications with others about the disorder (e.g., separating truth from myth). Finally, knowing the epidemiology of ADHD (including prevalence, gender- and culture-related differences, and typical course and outcome) enables you to more accurately diagnose this disorder.

TEST YOURSELF

1. **Hyperactivity and inattention are modern problems that emerged only in the past 100 years.**
   a. True
   b. False
2. **Which of the following statements are *true*? (Mark all that apply.)**
   a. ADHD is characterized by variability, both across children with the disorder and within a given child who has ADHD.
   b. All children with ADHD look the same.
   c. "Sluggish cognitive tempo" is a DSM-5 subtype of ADHD.
   d. The DSM-5 is primarily a categorical approach to ADHD, with some dimensional elements like severity.
   e. The features of ADHD exist on a continuum along which every person can be placed.
3. **Hyperactivity and impulsivity (mark all that apply):**
   a. Are associated with increased risk of injury
   b. Are highly correlated with each other
   c. Are two different subtypes of DSM-5 ADHD
   d. Can be expressed verbally and motorically
   e. Cluster together in factor analyses

4. **Sustained attention is a major problem for children with ADHD.**
   a. True
   b. False
5. **Researchers estimate that 80% or more children with ADHD have at least one comorbid disorder.**
   a. True
   b. False
6. **Which of the following disorders occur at higher-than-average rates among children with ADHD? (Mark all that apply.)**
   a. Anxiety disorders
   b. Depressive disorders
   c. Oppositional defiant disorder
   d. Specific learning disorders
   e. Tic disorders
7. **Which of the following statements about the associated features of ADHD are *true*? (Mark all that apply.)**
   a. Adaptive functioning can be impaired in children with ADHD.
   b. Children with ADHD can show poor compliance with rules, even when they are not trying to be oppositional.
   c. Executive dysfunction is an essential feature of ADHD, with good diagnostic sensitivity and specificity.
   d. Problems with peer relations indicate that an autism spectrum disorder is more likely than ADHD.
   e. When emotional regulation problems are present, you can eliminate ADHD as a diagnostic possibility.
8. **ADHD has multiple causes. Which of the following have strong scientific data supporting them as likely factors in the etiology of ADHD? (Mark all that apply.)**
   a. Gene-environment interactions
   b. Genes
   c. Prenatal exposure to toxins
   d. Television exposure
   e. Vestibular deficits
9. **Mark all of the *true* statements about the epidemiology of ADHD:**
   a. ADHD is largely a condition of childhood and adolescence, and most people outgrow the disorder by adulthood.
   b. ADHD is one of the most common mental health diagnoses in children.
   c. Both boys and girls can have ADHD, although boys are more likely to be diagnosed.

d. Cultural factors can impact accurate detection of ADHD in some populations.
e. Lifetime symptoms of ADHD are usually episodic rather than chronic.

10. On average, children with ADHD are *more* likely than the general population to: (mark all that apply)

a. Attend college or other post-secondary education
b. Experience peer rejection and be bullied
c. Have higher rates of speeding tickets and auto accidents
d. Score lower on standardized academic achievement tests
e. Utilize emergency room services

Answers: 1. b; 2. a, d, & e; 3. a, b, d, & e; 4. a; 5. a; 6. a, b, c, d, & e; 7. a & b; 8. a, b, & c; 9. b, c, & d; 10. b, c, d, & e

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