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Attention-Deficit/Hyperactivity Disorder: A Neuropsychological Perspective Towards DSM-V

Gerry A. Stefanatos · Ida Sue Baron

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Abstract Neuropsychological methods and techniques have much to offer in the evaluation of the individual suspected as having Attention-Deficit/Hyperactivity Disorder (ADHD). After a review of the historical evolution of the ADHD concept, incidence and prevalence, and DSM-IV criteria for diagnosis, especially as regards omission related to gender differences, and other associated cultural, familial, socioenvironmental, and subject influences, this paper describes a number of dilemmas and obstacles encountered in clinical practice. Included are the confounds associated with the wide range of possible comorbidities, the insufficiency of current DSM-IV criteria, the emergence of subtype differentiation and its impact on diagnosis and treatment. The complex relationship between neuropsychological constructs and ADHD, and obstacles to valid assessment are also addressed. The complexities associated with a thorough ADHD evaluation are viewed within an impressive and expansive existing scientific framework and recommendations are made for future directions.

Keywords Attention deficit · Hyperactivity · Prevalence · Gender differences · Environmental influences · Comorbidity · Subtypes · Assessment · Neuropsychological impairment · DSM-IV · DSM-V

G. A. Stefanatos (✉)
Cognitive Neurophysiology Laboratory, Moss Rehabilitation Research Institute, Korman Research Pavilion, Albert Einstein Medical Center,
1200 W. Tabor Rd, Philadelphia, PA 19141, USA
e-mail: Stefanag@Einstein.edu

I. S. Baron
Independent Private Practice, Potomac, MD and Reston, VA and Department of Pediatrics, University of Virginia, Charlottesville, VA, USA

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a complex neurodevelopmental condition characterized by "a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development" (APA, 1994, Pg. 78). It is distinguished by excessive and situationally inappropriate motor activity (Halperin, Matier, Bedi, Sharma, & Newcorn, 1992; Kinsbourne, 1977), limited inhibitory control of responses (Barkley, 1997c; Chelune, Ferguson, Koon, & Dickey, 1986; Nigg, 2001), and impaired ability to focus, sustain, and switch attention (Cepeda, Cepeda, & Kramer, 2000; Douglas, 1972; Epstein, Conners, Erhardt, March, & Swanson, 1997; Levine, Busch, & Aufseeser, 1982; Seidel & Joschko, 1990). Considered the most common diagnosis for children seen in psychiatric clinics, ADHD is estimated to affect approximately 4.4 million children between the ages 4–17 years in the United States alone (CDC, 2003). While the disorder commonly emerges in the preschool years (Campbell, 1995; Connor, 2002), symptoms often persist into adolescence and adulthood (for 50–80% of cases) (Barkley, Fischer, Smallish, & Fletcher, 2002; Faraone, Biederman, Spencer et al., 2000; Hechtman, 2000; Wolraich et al., 2005), albeit in a modified presentation that likely reflects compensatory maturation, successful application of self-applied or formal treatment interventions, or some combination of these factors (Hechtman & Weiss, 1983; Wender, 1998). In approximately two-thirds (50–70%) of individuals diagnosed with ADHD, there is corollary evidence of clinical problems related to learning ability (Barry, Lyman, & Klinger, 2002; Mayes, Calhoun, & Crowell, 2000; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005), social adjustment and functioning (Carlson, Lahey, Frame, Walker, & Hynd, 1987; Pfiffner,

Calzada, & McBurnett, 2000), or emotional well-being (Abikoff & Klein, 1992; Accardo, Blondis, & Whitman, 1990; Jensen, Martin, & Cantwell, 1997; Shaywitz & Shaywitz, 1991). For some, these clinical issues, rather than attentional deficits or hyperactivity, initiate referral of children who will eventually be diagnosed with ADHD (Mulhern, Dworkin, & Bernstein, 1994; Weinberg & Emslie, 1991; Wilens et al., 2002). The presence of such comborbid problems can complicate diagnostic formulation, obscure the fundamental nature of the primary disorder, and limit appreciation for the full clinical manifestation (Milberger, Biederman, Faraone, Murphy, & Tsuang, 1995; Pliszka, 1998).

There are long-standing controversies related to ADHD. These extend across a broad cross-section of society, including clinicians, parents, teachers, policymakers, and the media. Despite ADHD's emergence as a coherent clinical entity over a century ago (Still, 1902), the enormous literature regarding this disorder is often contradictory, complicating consensus about etiology, diagnosis, behavioral characteristics, assessment procedures, and treatment. Widely varying opinions regarding the validity of current conceptions of the disorder confuse attempts to bring uniformity to the discussion. For some, the concept of ADHD is an artificial byproduct of modern societal or cultural pressures that impose strong restrictions on the expression of certain impulses or behaviors (Carey, 1998; Ruff, 2005; Timimi & Taylor, 2004). Others emphasize that the literature provides compelling evidence of physiological anomalies (Barry, Johnstone, & Clarke, 2003; Jonkman, Kenemans, Kemner, Verbaten, & van Engeland, 2004; Oades, DittmannBalcar, Schepker, Eggers, & Zerbin, 1996; Pliszka, Liotti, & Woldorff, 1999; Taylor, Voros, Logan, & Malone, 1993) and differences on structural and functional neuroimaging (Bush, Valera, & Seidman, 2005; Casey & Durston, 2006; Seidman, Valera, & Makris, 2005; Vaidya et al., 2005; Willis & Weiler, 2005) to support conceptualization of ADHD as a neurodevelopmental disorder (Castellanos, Glaser, & Gerhardt, 2006; Rauch, 2005; Sonuga-Barke, 2005a; Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998; Taylor, 1999). While there is mainstream agreement that the cluster of symptoms that define ADHD likely represents the final common behavioral pathway of diverse underlying problems, the nature of these problems and their basis remains inadequately specified (Kupfer et al., 2000). Correspondingly, there is continuing dissatisfaction with current taxonomies and diagnostic criteria (Applegate et al., 1997; Barkley, 2003; Faraone, 2005).

Our intent in this paper is to provide a concise review of ADHD, drawing particular attention to current controversies and dilemmas related to clinical conceptualizations of the disorder and its diagnosis. We emphasize that ADHD is multiply determined and must be assessed within its multifaceted context, that reasoned consideration of associated comorbid conditions is necessary as these have the potential to complicate definition, assessment, and treatment, and that it is crucial to consider a full range of factors or conditions that may be primarily responsible for attentional variability and hyperactivity/impulsivity but that may not lead to a formal ADHD diagnosis. In discussing these issues, we highlight a number of the thorny questions and sources of confusion that must be addressed to allow further progress in understanding and treating the disorder. Consideration of these issues may ultimately facilitate a more precise and clinically valid reformulation of the disorder. For more comprehensive reviews of neuropsychological aspects of ADHD, the reader is referred to a number of excellent integrative discussions (Barkley, 2003; Biederman, 2005; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg, 2005b; Seidman, 2006; Sergeant, 2005; Sonuga-Barke, 2005a; Voeller, 2004; Weyandt, 2005).

Historical evolution of the ADHD concept

The symptom complex of ADHD has not changed dramatically since George Frederic Still (1902) first highlighted the disorder's chief characteristics as manifested in 43 children from his clinical practice with "defects in moral control" (p. 1009). In three lectures to the Royal Academy of Physicians in London, he described children who demonstrated an "abnormal incapacity for sustained attention, restlessness, fidgetiness, violent outbursts, destructiveness, noncompliance, choreiform movements, and minor congenital anomalies" (p. 1166). He portrayed an overactive, passionate, and excessively emotional group of children who demonstrated little "inhibitory volition" (p. 1008), and who appeared resistant to discipline, spiteful, and sometimes cruel and dishonest. Still viewed their behavioral presentation as representing a fundamental deficit in the "control of action in conformity with the idea of the good of all" (p. 1008) and hypothesized a familial predisposition for the disorder, although he also considered the possibility of acquired nervous system insult.

Different aspects of this complex constellation of symptoms were emphasized in subsequent years. The encephalitis epidemic of 1917–1918 drew attention to the fact that similar behavioral problems (particularly hyperactivity, impulsivity, antisocial behavior, and emotional lability) could result from brain infection in childhood (Ebaugh, 1923; Hohman, 1922). While causal connections to encephalitis were often questionable (Barkley, 1998), this association prompted some to draw parallels between the behavioral problems of children with or without demonstrable brain damage. Despite acknowledgment of the inherent flaws (circularity) of this reasoning (see Kessler, 1980 for a review), it was argued that

the latter group demonstrated a "Minimal Brain Damage Syndrome." The implication that brain damage existed but could not be detected because of the neurological examination's fallibility (Strauss & Lehtinen, 1947) resulted in the emergence of the diagnostic concept of "minimal brain dysfunction (MBD)." However, the importance of environmental factors was also recognized, since post-encephalitic children successfully treated in special residential treatment centers often relapsed when returned to maladjusted parents (Bond & Smith, 1935). As further evidence accrued, it became apparent that a full spectrum of causality needed to be considered that included genetic factors, gestational and perinatal experiences, interactions with significant others, and the stresses and emotional trauma of later life (Clements & Peters, 1962).

Although speculations about the neurological basis for the disorder persisted (Knobel, Wolman, & Mason, 1959; Laufer, Denhoff, & Solomons, 1957a) and conceptions of MBD continued to evolve (Rie & Rie, 1980), later emerging theories focused to a greater extent on behavioral characterization of the syndrome. Initially, hyperactivity and poor impulse control were emphasized (Chess, 1960; Laufer, Denhoff, & Solomons, 1957b). Subsequently, the role of attentional deficits was highlighted, based on evidence that these children demonstrated behavioral inattention as well as performance deficits on laboratory measures of attention (Douglas, 1972; Dykman, Peters, & Ackerman, 1973). According to Douglas and Peters (1979), the key deficits involved: (1) organization, investment, and maintenance of attention and effort; (2) difficulties inhibiting impulsive behavior; (3) problems modulating arousal levels consistent with situational demands; and (4) a strong need for immediate reinforcement. This conceptualization was influential in transforming the name of the disorder from the psychodynamically oriented "hyperkinetic reaction of childhood" in DSM-II (APA, 1968) to "attention deficit disorder (ADD)" in DSM-III (APA, 1980). The resultant taxonomy considered ADD a multidimensional disorder with inattention as the central feature. Despite an absence of supportive empirical research, two distinct forms were described, one with hyperactivity (ADD-H) and one without (ADD-W).

Concern emerged that the introduction of the "ADD" label had shifted emphasis away from the important features of hyperactivity and poor impulse control, which were not only fundamental symptoms of the disorder but important to differentiate it from other conditions. Moreover, evidence accumulated that hyperactivity and impulsivity formed a single behavioral dimension (Achenbach & Edelbrock, 1983; Lahey et al., 1988). The disorder was then renamed "attention-deficit/hyperactivity disorder" in DSM-III-R (APA, 1987) and ADD without hyperactivity was designated "undifferentiated attention deficit disorder." This reformulation marked a shift to a unidimensional, polythetic classification strategy, premised on the view that three key symptom domains were implicated (inattention, hyperactivity, and impulsivity), without individual thresholds for each domain.

The term "attention-deficit/hyperactivity disorder" was retained in DSM-IV (APA, 1994), but differentiated into three defined types: *predominantly inattentive (PIA)*, *predominantly hyperactive/impulsive (PHI)*, and *combined (C)*. This conceptualization is consistent with factor analytic studies of parent and teacher symptom ratings that have fairly consistently identified two broad distinguishable behavioral dimensions that best characterize ADHD: inattention and hyperactivity-impulsivity (Bauermeister, Alegria, Bird, Rubio-Stipec, & Canino, 1992; Burns, Boe, Walsh, Sommers-Flanagan, & Teegarden, 2001; Collett, Crowley, Gimpel, & Greenson, 2000; DuPaul et al., 1997; Healey et al., 1993; Lahey et al., 1988; Pillow, Pelham, Hoza, Molina, & Stultz, 1998). Accordingly, beginning with DSM-IV, ADHD and its diverse manifestations are considered in terms of variations on separate continua of inattention and hyperactivity-impulsivity, or by a combination of problems in these two domains. These conventions apply to both males and females (Collett et al., 2000; Hudziak et al., 1998) and exist across ethnic and cultural groups (Beiser, Dion, & Gotowiec, 2000). The external validity of the two dimensions is also supported by evidence suggesting different developmental trajectories (Biederman, Mick, & Faraone, 2000), types of functional impairment, and mental health problems (Lahey & Willcutt, 2002).

Diagnosis according to DSM-IV criteria

The diagnosis of ADHD is based on observations and subjective reports of developmentally inappropriate behavior in the domains of inattention, hyperactivity, and/or impulsivity obtained from a variety of sources, including, but not necessarily limited to, the child, parents, and teachers. In order to meet DSM-IV diagnostic criteria for ADHD, a child must demonstrate six or more symptoms from either of two nine-item lists set forth in the DSM-IV-Text Revision (APA, 2000) manual and adapted here in Table 1. The lists were compiled by a committee of leading experts in the field, informed by literature review, an informal survey of available empirically derived behavior rating scales assessing ADHD symptomatology, and statistical analyses of data derived from a field trial of selected items conducted with 380 children at 10 different North American sites (Lahey et al., 1994). One list is comprised of items chosen to index problems related to "inattention," while the other contains items reflecting "hyperactivity-impulsivity." Of the latter, six items relate to hyperactivity and three to impulsivity. The type of ADHD diagnosed (PIA, PHI, or C) is determined by whether the six

Table 1 DSM-IV-TR criteria for ADHD

A. Either (1) or (2)

(1) Six (or more) of the following symptoms of inattention have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

—Often fails to give close attention to details or makes careless mistakes in school work, work, or other activities.
—Often has difficulty sustaining attention in tasks or play activities.
—Often does not seem to listen when spoken to directly.
—Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not to do to oppositional behavior or failure to understand instructions).
—Often has difficulty organizing tasks and activities.
—Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework).
—Often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools).
—Is often easily distracted by extraneous stimuli.
—Is often forgetful in daily activities.
—Symptom total

(2) Six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

—Often fidgets with hands or feet or squirms in seat.
—Often leaves seat in classroom or in other situations in which remaining seated is expected.
—Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, they are limited to subjective feelings of restlessness).
—Often has difficulty playing or engaging in leisure activities quietly.
—Is often "on the go" or often acts as if "driven by a motor."
—Often talks excessively.

Impulsivity

—Often blurts out answers before questions have been completed.
—Often has difficulty awaiting turn.
—Often interrupts or intrudes on others (e.g., butts into conversations or games).
— Symptom total

Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age seven years.
Some impairment from both symptoms is present in two or more settings (e.g., at school or work, and at home).
Clear evidence of clinically significant impairment in social, academic, or occupational functioning.

Note. Adapted from American Psychiatric Association (2000). Copyright 2000 by the American Psychiatric Association.

(or more) symptoms present in the last six months are on the inattention list, the hyperactivity-impulsivity list, or both.

The requisite threshold of six symptoms was ascertained with reference to scores obtained on the Children's Global Assessment Scale (CGAS) (Shaffer et al., 1983) during the field trials. In order to reach the CGAS cutoff of 60, which indicates a level of impairment requiring treatment, field trial data suggested five symptoms had to be present. However, to err conservatively and lessen the occurrence of false positives, the DSM-IV standard was set at six or more symptoms. This cut off also provided greatest discriminability and interjudge reliability. Formal diagnosis also specifies that symptom onset occurs prior to age seven years; symptoms exist for at least 6 months; are observable in more than one setting (e.g., school and home); and interfere with academic, social, or occupational functioning. The diagnosis is not appropriate if the symptoms occur exclusively in the course of pervasive developmental disorder, schizophrenia, or a psychotic disorder, or are better accounted for by another specific mental disorder, such mood disorder, anxiety disorder, dissociative disorder, or personality disorder.

ADHD incidence, prevalence, and persistence

Accurate estimation of the incidence and prevalence of ADHD has been hindered by several critical factors, including the lack of: (1) an objective diagnostic test for ADHD; (2) a "gold standard" measure of ADHD that is easily applicable in epidemiologic research; (3) a systematic means to monitor the diagnosis of ADHD; (4) consistency in case definition and how it is operationalized; and (5) consistency in reporting symptomology across age, gender, and informant source (Rowland, Lesesne, & Abramowitz, 2002). The disorder affects approximately 4–12% of school-age children (6–18 years) (AAP, 2000). However, estimates vary

considerably, ranging from 1.7 to 17.8% (Brown et al., 2001; Elia, Ambrosini, & Rapoport, 1999; Goldman, Genel, Bezman, & Slanetz, 1998). Clinic-based samples have generally yielded higher prevalence rate estimates in school-age children compared to population-based studies (Scahill & Schwab-Stone, 2000). Children suspected as having ADHD comprise as many as 30–50% of referrals to mental health agencies. By comparison, 3–5% of school-aged children are identified by academic institutional data, or approximately one child per class (DuPaul & Stoner, 1994; Evans, Vallano, & Pelham, 1995; Lorys-Vernon, Hynd, Lyytinen, & Hern, 1993; Teeter, 1998). A trend toward higher estimates from community samples (10.3%) compared to school samples (6.9%) has also been noted (Homer et al., 2000). In addition to differences in mode of ascertainment, variability in prevalence estimates is also likely due to changes in identification methods over time. For example, higher rates were obtained using DSM-III-R criteria (10.3%) compared to DSM-III criteria (6.8%) (Brown et al., 2001). In addition, changes in criteria and the increase in the number of ADHD types in DSM-IV resulted in increased prevalence estimates (Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996).

Prevalence estimates decline with age. While adult prevalence is estimated to be 2–7% (Dulcan et al., 1997; Wender, Wolf, & Wasserstein, 2001), these figures are difficult to interpret since studies commonly employ DSM-IV diagnostic criteria which were developed and field-tested with children and adolescents aged 6–14 years. Relatedly, rates of remission in ADHD may be linked to definitional criteria (Biederman et al., 2000). Nevertheless, a recent study employing an Adult ADHD Clinical Diagnostic Scale (Adler & Spencer, 2004) found a prevalence of 4.4% in the United States which was thought to be conservative (Kessler et al., 2006). By comparison, Kooij et al. (2005) estimated a prevalence between 1% and 2.5% in the Netherlands. Inclusion of adults in epidemiologic studies is challenging because of the need for retrospective childhood diagnosis and the likelihood of unreliable retrospective self-reports of symptoms (Barkley, Fischer et al., 2002; Mannuzza, Klein, Klein, Bessler, & Shrout, 2002). Many adults remain undiagnosed until such time as their own children are evaluated for the disorder and their personal, idiosyncratic behavioral features are determined to be of clinical significance, or when higher educational or vocational demands subject their own long-present weaknesses to closer scrutiny. Reporting source is also influential in adults, as it is in childhood prevalence estimation. For example, adult prevalence according to self-report was substantially lower than when parents report on symptoms in their adult children (Barkley, Fischer et al., 2002).

Complications also emerge when attempting to determine prevalence in preschoolers. A recent review by Egger, Kondo, and Angold (2006) concluded that studies using DSM diagnostic criteria and community or pediatric clinic referrals resulted in preschool ADHD prevalence estimates ranging from 2–7.9%. However, problems were evident when distinguishing between normal and "developmentally inappropriate" inattention, hyperactivity, and impulsivity in the preschool population, reflected in enormous variability of endorsed symptoms. For example, the number of preschoolers considered to demonstrate the symptom "always on the go, driven by a motor" ranged from 2% (Earls, 1982) to 72.7% (Pavuluri, Luk, & McGee, 1999). In addition, symptoms commonly endorsed by parents of preschoolers with ADHD were also frequently subscribed to by parents of young children without ADHD. For instance, "interrupts/intrudes" was endorsed by 100% of parents of preschoolers meeting criteria for ADHD and 44.7% of parents of preschoolers without ADHD. Overall, boys and older preschoolers (4–5 year-olds) were more likely than younger preschoolers (2–3 year-olds) to meet criteria for ADHD.

Given the large estimated prevalence, chronicity, and significant impairment of academic performance, social functioning, and overall quality of life (Sawyer et al., 2002), the Center for Disease Control and Prevention identified ADHD as a major public-health problem in 1999 (Lesesne, Abramowitz, Perou, & Brann, 1999). Affected families incur higher medical costs due to increased hospitalizations, outpatient visits, and pharmacy fills (Birnbaum et al., 2005). In addition, the disorder has implications for long-term safety and accident occurrence (Barkley, 2002). However, a meaningful indication of the full extent of the public health burden that ADHD poses on the individual and to society is unresolved.

Demographic, socioenvironmental, and contextual influences

Gender differences

Epidemiological studies indicate that ADHD is more commonly diagnosed among boys than girls (for reviews, see Lahey, Miller, Gordon, & Riley, 1999; Rowland et al., 2002). The gender difference in identification has often been attributed to complex issues surrounding neurobiological differences between males and females. However, a number of methodological issues, interacting with socioenvironmental influences, may also account for this pattern (Hartung & Widiger, 1998; Rutter, Caspi, & Moffitt, 2003). In particular, sampling biases may play a fundamental role in determining the magnitude of observed male-female gender ratios. In clinical samples, boys are six to ten times more likely than girls to be referred for the disorder (Arnold et al., 1997; Carlson, Tamm, & Gaub, 1997; Evans et al., 1995; Willcutt & Pennington, 2000) and three to four times more likely than

girls to be diagnosed (Cantwell, 1996). By contrast, gender differences in nonreferred samples are generally in the order of 1:1–3:1 (Biederman, Kwon et al., 2005; Cuffe et al., 2001). These findings indicate that boys are referred disproportionately more often than girls. Consistent with this observation, teachers given comparable fictional records (differing in gender) to review were more likely to refer boys than girls for consideration of ADHD (Sciutto, Nolfi, & Bluhm, 2004). While this bias was evident across symptom type, the largest gender differences were observed for children who exhibited aggression or hyperactivity without inattention.

It has been suggested that the considerably higher male-to-female ratio in clinic-referred children results from gender differences in the phenotypic expression of ADHD. Girls with ADHD are less likely than their male counterparts to manifest a comorbid disruptive behavior disorder (e.g., oppositional defiant or conduct disorder), major depression, or learning disability (Biederman et al., 2002; Gaub & Carlson, 1997). In addition, girls with ADHD are less likely to engage in rule-breaking or externalizing behaviors (Abikoff et al., 2002) or to demonstrate functional impairments that impact involvement in extracurricular activities (Biederman et al., 2002). Indeed, girls with ADHD demonstrate lesser impairment on a number of behavioral ratings (Newcorn et al., 2001) but may exhibit higher levels of inattentiveness, internalizing symptoms, or comorbid conditions (e.g., separation anxiety disorder generalized anxiety disorder) (Levy, Hay, Bennett, & McStephen, 2005), and social impairment (Biederman et al., 2002; Gaub & Carlson, 1997; Gershon, 2002). In comparison, boys with ADHD commonly exhibit higher levels of hyperactivity, conduct problems, aggression, and other externalizing symptoms that are regarded as more disruptive in the classroom (Gaub & Carlson, 1997; Gershon, 2002; Keenan & Shaw, 1997). To the extent that clinical referrals are frequently prompted by externalizing behavioral problems, such as aggression, and girls are at lower risk for comorbid disruptive behavior disorders, gender differences reported in the literature may well reflect a referral bias implicating males. These gender influences on the clinical manifestations of ADHD may consequently lead to underdiagnosis in girls (Arcia & Conners, 1998; Biederman, Kwon et al., 2005; Biederman et al., 2002). Though the gender ratio in adults is about 1:1 (Faraone, Biederman, Mick et al., 2000), gender differences persist into adulthood. Women with ADHD reported more problems and fewer assets, suggesting they have poorer self-perceptions than reported by ADHD men (Arcia & Conners, 1998).

Gender differences may also reflect biases in assessment. In reviewing the literature, Hartung and Widiger (1998) noted that 81% of research participants in ADHD studies were male. Consistent with this bias, the criteria used to define ADHD were largely developed and tested on predominantly male samples. It remains questionable how appropriate or equivalent these criteria are for identifying ADHD in females. Indeed, normative studies on rating scales that sample ADHD symptomology demonstrate that male children in the general population are more likely than females to demonstrate behaviors pertinent to the diagnosis of ADHD (Achenbach, Howell, Quay, & Conners, 1991; DuPaul, 1991; Goyette, Connors, & Ulrich, 1978) without necessarily having ADHD. Given the gender differences in symptomology noted above, it could be argued that using the same criteria in females holds them to a higher threshold for being diagnosed as ADHD. Waschbusch and King (2006) demonstrated that a proportion of females who demonstrate elevated levels of inattentive or disruptive behavior weighed against same gender non-ADHD peers may not be detected as developmentally inappropriate if compared to the general population of age peers. This may result in underdiagnosis of ADHD in girls and particularly ADHD-PIA. In consideration of such biases, it has been argued that the accurate diagnosis of ADHD in females may require the use of gender appropriate age-based criteria (Kato, Nichols, Kerivan, & Huffman, 2001; Waschbusch & King, 2006).

Gender differences also appear to vary with ADHD type and age. The effect of ADHD type was noted in the DSM-IV field trial where male to female ratios appeared higher in ADHD-C type compared to ADHD-PIA (Lahey et al., 1994). In part, this may reflect inherent biases in the DSM-IV symptom list that emphasize externalizing behaviors, the kind of behaviors more closely associated with boys. Girls typically come to attention with internalizing behaviors not extensively sampled by DSM-IV, e.g., ineffective social behavior, poor self-esteem (Thurber, Heller, & Hinshaw, 2002). Correspondingly, several studies have suggested that clinic referred ADHD girls are more likely to receive the ADHD-PIA diagnosis (Biederman et al., 2002; Levy et al., 2005; Weiler, Bellinger, Marmor, Rancier, & Waber, 1999). By contrast, differences dissipate in nonreferred samples. Biederman et al. (2005) observed that nonreferred females and males with ADHD did not differ in extent of psychiatric comorbidity, treatment history, or diagnosed ADHD type. The most prevalent type in both groups was ADHD-C (58% of females and 61% of males), followed by ADHD-PIA (25% female and 27% male). ADHD-PHI was the least common, observed in only 13% of the female and 9% of the male ADHD group. The high proportion of ADHD-C and low frequency of ADHD-PHI is a common finding (Gomez, Harvey, Quick, Scharer, & Harris, 1999; Lahey et al., 1994; Nolan, Gadow, & Sprafkin, 2001).

Cultural and societal influences

Cultural and societal influences have received deserved attention. Across cultures, prevalence rates have varied from 1% to 16.1% (Spencer, Biederman, Wilens, & Faraone, 2002).

Early cross-cultural studies of hyperactivity found significantly different prevalence across four countries (United States, Germany, Canada and New Zealand) (Trites, 1979). The disorder was diagnosed nearly 50 times as often in North America as in Britain (Rutter, 1983). The lower prevalence rate in Britain was attributed to more restricted criteria (ICD-9) and the view that hyperactive children had conduct problems arising largely out of family dysfunction and social disadvantage rather than due to a developmental disorder. Despite substantial correspondence in diagnostic criteria between ICD-10 and DSM-IV, ADHD may continue to be underdiagnosed in the United Kingdom (Jick, Kaye, & Black, 2004).

While rate discrepancy has narrowed in recent years with increased criteria specificity, the manner in which clinicians and educators view the disorder may remain disparate. Clinicians often encounter referrals in which the referring educational professional suspects ADHD while parents remain relatively unconcerned (Wolraich et al., 2004). While there is moderate to high levels agreement between parents and teachers for disruptive behavior disorders (e.g., ODD and CD), concordance is lower for ratings of ADHD symptoms (Antrop, Roeyers, Oosterlaan, & Van Oost, 2002). However, when parental concerns are confirmed by an ADHD diagnosis, it is highly likely that a teacher report will concur (Biederman, Faraone, Milberger, & Doyle, 1993). The possibility that personal biases may be operative demands a cautionary approach to interpretation of the important historical information that is obtained through clinical interviews, record review, and teacher reports (de Ramirez & Shapiro, 1998).

Whether ADHD arises as a principal concern in a specific sociocultural context is often influenced by the degree of acceptance of externalizing behavioral traits by members of that sociocultural group. While a recent meta-analysis (Faraone, Sergeant, & Gillberg, 2003) indicated that prevalence is comparable, worldwide, higher rates were found in other than North American countries (e.g., 19.8% in the Ukraine (Gadow et al., 2000); 14.9% in the United Arab Emirates (Bu-Haroon, Eapen, & Bener, 1999). It therefore seems unclear whether differences in prevalence across ethnic groups are real or secondary to sociocultural factors and/or associated differences in sources of information about ADHD symptomatology. For example, higher rates of hyperactivity were diagnosed among African-Americans compared to Caucasian Americans, but mainly when identification was solely dependent on teacher report (Lambert, Sandoval, & Sassone, 1978; Nolan et al., 2001). That this may in part reflect the properties of behavioral rating scales that do not perform identically across racial groups (Reid et al., 1998) remains a significant consideration in clinical practice. Clarity regarding the influence of such factors is required to interpret not only the results of cross-cultural studies but also the conclusions in those investigations within a culture that may employ differential diagnostic criteria or interpretive schemas.

Family and socioenvironmental influences

Family and socioenvironmental factors influence both a child's behavior and informant agreement, and must be considered in the diagnostic process. Genetic predisposition is a highly relevant factor. High family concordance of ADHD is well documented through adoption, family, and twin studies (Cook, 1999; Doyle, Willcutt et al., 2005; Faraone & Doyle, 2001; Hechtman, 1996), as is a high ADHD incidence in children of parents diagnosed with ADHD (Alberts-Corush, Firestone, & Goodman, 1986; Biederman, Faraone et al., 1995; Epstein et al., 2000). One parent's familiarity with personally-experienced ADHD symptoms may make that parent more accepting of behaviors that are reminiscent of their own childhood symptomatology but unacceptable to the other parent. An absent or long-working parent may fail to appreciate the signficance of their child's behavior as well as the parent who has primary responsibility for behavioral management.

Awareness about the parent's state may also affect descriptions of the child. For example, depressed mood and parental stress may decrease informant agreement and thereby affect diagnosis (van der Oord, Prins, Oosterlaan, & Emmelkamp, 2006). Conflicting impressions may also arise between a parent whose expectations for behavior at home are at considerable variance with the teacher's expectations for classroom behavior. Other situational factors are also influential. For example, a child may mimic behaviors observed at home while in school, despite their being inappropriate in the academic setting. Additionally, socioenvironmental factors may be of considerable importance with regard to etiology of the behavors of concern. Caution is necessary to be sure such reactive transient states are not overly emphasized with respect to an ADHD diagnosis, and that a distinction between a formal ADHD diagnosis and transient attentional disorder is maintained.

Family factors have a broad impact on the diagnosis of ADHD. Despite widespread recognition in the lay public of the existence of ADHD and its associated symptoms, parents commonly and erroneously attribute ADHD behavior to volitional factors and noncompliance rather than to specific cognitive deficit or skill limitation (Harrison & Sofronoff, 2002). A recent study suggested that most (80%) parents of children with ADHD recognized their child had problems, but few (35%) considered the possibility of hyperactivity, and consequently few consulted a primary care physician for these problems (Sayal, Goodman, & Ford, 2006). When parents indicated concerns about inattention, impulsivity, and/or hyperactivity or used medical terms such as ADHD or ADD,

a sensitivity of 87% and a specificity of 47% for the subsequent diagnosis of ADHD was found (Mulhern et al., 1994). Indeed, the receipt of services for ADHD has been linked to informants describing the child's difficulties in medical terms (Bussing, Gary, Mills, & Garvan, 2003).

The impact of diverse circumstances related to the home or other socioenvironmental contexts or the child's psychological state, medical health, also may directly compromise the clinician's attempt to make sense of competing potential explanations for a child's functioning, which may in turn directly affect treatment decisions and application of interventions. Assessment of the familial context of ADHD is supported by evidence that family problems are often endemic to ADHD. Parents of children with ADHD are more likely to experience stress and marital discord, disagree about how their child learns, and have more negative parenting practices. In addition, they may demonstrate more psychopathology including but not limited to their own manifestation of ADHD symptomatology. The problems of the child with ADHD exacerbate these parental issues (Pelham & Lang, 1999; Pelham et al., 1998) and may be negatively impacted by them (Biederman, Milberger et al., 1995; Counts, Nigg, Stawicki, Rappley, & von Eye, 2005; Pressman et al., 2006). Family cohesion is inversely associated with ADHD ($p = 0.058$) (Cuffe et al., 2001). Parenting skills, in particular, are key targets for therapy given their importance in the prediction and mediation of the child's long-term outcome.

One of the most serious and intractable areas of impairment associated with ADHD concerns social relations or interpersonal relationships (Frederick & Olmi, 1994; Harpin, 2005; Pelham & Bender, 1982; Pfiffner et al., 2000). Children with ADHD often display substantial problems interacting with other children (Milich, Landau, Kilby, & Whitten, 1982; Mrug, Hoza, & Gerdes, 2001), and these problems are often debilitating with serious long-term implications (Greene, Biederman, Faraone, Sienna, & Garcia Jetton, 1997; Greene et al., 1999). Whalen and Henker (1997) identified impairment in five domains of social function: (1) response patterns; (2) style of approach; (3) social information processing; (4) peer appeal or social standing; and (5) social impact and influence. In addition to demonstrating problems with intrusiveness and immaturity, children with ADHD are frequently bossy and boastful, and often physically and verbally aggressive toward other children (Hinshaw & Melnick, 1995; Miller-Johnson, Coie, Maumary-Gremaud, & Bierman, 2002; Nolan & Gadow, 1997). As a consequence, negative social interactions with peers may be expected to very quickly result in peer rejection (Buhrmester, Whalen, Henker, MacDonald, & Hinshaw, 1992; Olson & Brodfeld, 1991), and these negative impressions or poor reputations within their peer group remain fairly stable over time, persisting even when behavioral improvements are evident (Buhrmester et al., 1992; Granger, Whalen, & Henker, 1993). Peers are not only critical of misbehaviors they observe but also have globally negative attributions about qualities that they may not witness firsthand (Whalen & Henker, 1992). These negative expectations of future behavior are surprisingly impervious to disconfirmation (Pfiffner et al., 2000). Social problems may therefore persist throughout adolescence and even into adulthood, whether or not individuals continue to meet criteria for the disorder (Slomkowski, Klein, & Mannuzza, 1995).

Subject and contextual influences

Within-subject response variability is one of the most robust characteristics of children with ADHD (Douglas, 1972; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005). Children with ADHD typically demonstrate greater variability than control children, especially on tasks assessing attention and inhibitory capacity, and requiring response over multiple trials (Douglas, 1983). This pattern is particularly evident on reaction time (RT) measures (Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989; Murphy, Barkley, & Bush, 2001; Scheres, Oosterlaan, & Sergeant, 2001). Although a meta-analysis found these effects were small (Huang-Pollock & Nigg, 2003), slower RT was often found when within-subject variability was greatest. Importantly, as such data show, identifying patterns of within-subject variability may augment our understanding of the nature of this observed variability (Castellanos et al., 2005) and in turn, result in more meaningful interpretations that influence clinical treatment interventions.

Contextual factors also influence the manifestation of ADHD symptomology. While DSM-IV criteria stipulate that ADHD symptoms must be present across different conditions it is clear, as noted above, that situational factors influence behavior and may in part account for some of the variance between parent and teacher reports of ADHD symptoms. For example, performance tends to be poorer in the afternoon compared to the morning (Dane, Schachar, & Tannock, 2000; Porrino et al., 1983); under low levels of stimulation (Antrop, Roeyers, Van Oost, & Buysse, 2000); when task complexity taxes organizational abilities (Douglas, 1983); and when restraint is required (Barkley & Ullman, 1975; Luk, 1985). Behavioral problems are far more likely to emerge when a child must persist in a work-related task (e.g., homework, household chore) or in a setting that constrains their behavior (e.g., church, restaurant) than in unrestricted, free play situations (Barkley & Edelbrock, 1987; Breen & Altepeter, 1990).

Reinforcement contingencies play a significant role in test performance. Performance worsens with intermittent rather than continuous reinforcement schedules, on fixed rather than variable schedules (Carlson & Tamm, 2000; Slusarek,

Velling, Bunk, & Eggers, 2001), and on delayed compared to immediate reinforcement schedules (Solanto et al., 2001; Tripp & Alsop, 2001). Such conditions have direct relevance to the assessment of children with ADHD, and to the application of interventional strategies.

The confound of comorbidity

Attention-deficit/hyperactivity disorder has long been associated with increased risk of comorbid problems such as oppositional defiant disorder (ODD), conduct disorder (CD), personality disorder, substance abuse, criminal behavior, and impairments in social adjustment, the latter three particularly evident in adolescence and adulthood (Angold, Costello, & Erkanli, 1999; Biederman, Newcorn, & Sprich, 1991; Jensen et al., 2001; Klein, 2002). Besides psychiatric and developmental disorders, a variety of health problems may co-exist and complicate assessment, diagnosis, and determination of the most appropriate intervention. Indeed, high rates of comorbid disorder have been observed in both ADHD and subthreshold ADHD, and it has been suggested that pure ADHD is relatively rare, even in samples derived from the general population (Kadesjo & Gillberg, 2001). Below, we discuss some of the substantial confounding issues engendered by comorbidity (Kessler, 2004; Pliszka, 1998).

Psychiatric disorder

A high frequency of comorbid psychiatric disorder in children with ADHD was documented through both clinical studies (Biederman et al., 2006; MTA, 1999b; Shekim, Asarnow, Hess, Zaucha, & Wheeler, 1990) and epidemiological surveys (August, Realmuto, MacDonald, Nugent, & Crosby, 1996; Pelham, Gnagy, Greenslade, & Milich, 1992). It has been estimated that approximately 59–87% of children and adolescents with ADHD may have at least one comorbid disorder (Fischer, Barkley, Smallish, & Fletcher, 2002; Kadesjo & Gillberg, 2001; McGough et al., 2005), and as many as 20% may have three or more comorbid disorders (Rowland et al., 2002). Many of the comorbid conditions present with several core symptoms of ADHD, including bipolar disorder (Biederman, Faraone, Mick, Wozniak et al., 1996; Biederman, Monuteaux, Kendrick, Klein, & Faraone, 2005; Jaideep, Reddy, & Srinath, 2006; Wozniak et al., 2004), anxiety disorder (March et al., 2000; Newcorn et al., 2001), mood disorder (Schmidt, Stark, Carlson, & Anthony, 1998; Wozniak et al., 2004), and childhood abuse/neglect (Glod & Teicher, 1996). Psychiatric comorbidities that involve externalizing disorder with an aggressive component, such as ODD (35.2%, 95% CI 27.2, 43.8) or CD (25.7%, 95% CI: 12.8, 41.3) tend to occur more frequently than those that involve internalizing disorders, such as anxiety (25.8%, 95% CI: 17.6, 35.3) or depressive/mood disorder (18.2%, 95% CI: 11.1, 26.6) (Barkley, Anastopoulos, Guevremont, & Fletcher, 1991; Bird, Gould, & Staghezza, 1993; MTA, 1999a; Shekim et al., 1985).

While many externalizing comorbid diagnoses are overtly apparent, internalizing disorders may be just as disruptive, yet less obvious. Such problems may come to attention through broad spectrum screening tools such as the Child Behavior Checklist (CBCL) (Biederman, Faraone, Mick, Moore, & Lelon, 1996; Biederman, Monuteaux et al., 2005) or structured interview (Biederman, Faraone, Doyle et al., 1993). However, depending on the specific comorbid condition and the age of the child, internalizing disorders may be detected only with specific inquiry, such as through careful parent interview. For example, anxiety-related disorders, such as simple phobias or separation anxiety, are easily overlooked or misidentified in early childhood when mistaken for normal features of a general developmental course.

Generalized anxiety disorders become more common with advancing age and are more likely to be reported for children whose parents or family members report shared symptomatology. Similarly, major depressive disorder (MDD) may not emerge until the adolescent years, e.g., subsequent to repeated exposure to socially and emotionally distressing circumstances such as physical trauma. Furthermore, MDD is a more common etiology in the presence of a history of maternal depression (Nigg & Hinshaw, 1998), when CD is present in the child or family or when a borderline or antisocial personality disorder is present (Fischer et al., 2002). Often, low self-esteem is an early manifestation (Treuting & Hinshaw, 2001). By adolescence, and in the extreme, the combination of ADHD and comorbid MDD may result in a significant increase in suicidal ideation or attempts (Barkley & Fisher, 2005; James, Lai, & Dahl, 2004). Therefore, it is necessary to carefully screen for internalizing symptoms that may be less obvious than externalizing symptoms in order to thoroughly consider the range of potential comorbities.

Comorbidity in ADHD is further complicated by a tendency for comorbid disorders to be comorbid with each other. In general, there is a trend for externalizing disorders to be comorbid with other externalizing disorders and for internalizing disorders to be comorbid with other internalizing disorders (Lewinsohn, Shankman, Gau, & Klein, 2004). These relationships can follow distinctly different temporal or developmental courses. Some comorbid disorders may be concurrent, co-occuring over a relatively short space of time, while others may co-exist over several years. In children with ADHD, for example, the co-occurrence of CD ranges from 20 to 50%, but increases to 44 to 50% in adolescents (Barkley, 1998; Barkley, Fischer, Edelbrock, & Smallish, 1990; Lahey, McBurnett, & Loeber, 2000). Alternatively, some disorders may be successively comorbid (Angold et al., 1999). It has been suggested, for example, that ODD frequently precedes

and may be a precursor for a later occurring CD (Lahey et al., 2000; Loeber, Burke, Lahey, Winters, & Zera, 2000). In addition, a quarter or fewer of adults with ADHD continue to have CD, but 12–21% meet criteria for an antisocial personality disorder (APD) (Biederman et al., 2006; Fischer et al., 2002; Loeber, Burke, & Lahey, 2002). Given the close relationship, ADHD has been cited as one of the most reliable early predictors of the later emergence of these externalizing disorders (Fischer, Barkley, Fletcher, & Smallish, 1993; Lahey et al., 2000). Earlier age of onset and more severe ADHD are associated with more severe externalizing and internalizing symptoms (Connor et al., 2003).

Bipolar disorder (BPD) occurs in approximately 10% of the ADHD individuals (Wilens et al., 2003), and is an especially controversial comorbid diagnosis, in part because of the considerable overlap in symptoms used for both diagnoses (hyperactivity, distractibility) (Carlson, 1990; Geller & Luby, 1997; West et al., 1995). A BPD diagnosis may differ from ADHD in its episodic course, mania, disturbed thinking, grandiosity, severe mood instability and irritability associated with inordinate responses to minor frustrations. Mick et al. (2005), for example, found that while the "mad/cranky irritability" characteristic of ODD was frequent in ADHD, "super-angry/grouchy/cranky" was common only to ADHD children with BPD. Comorbidity of ADHD and BPD is more likely the earlier the onset of BPD. However, recent studies suggest that the frequency of ADHD in children with juvenile BPD may be lower than previously thought (Jaideep et al., 2006) and their subective self-report of difficulties in attentional/problem solving ability in BPD may not be supported by their performance on objective measures (Robertson, Kutcher, & Lagace, 2003). The problems with sustained attention, working memory, and processing speed in BPD remain present even after after controlling for comorbid ADHD (Doyle, Wilens et al., 2005). The disorders are most likely to co-occur in individuals with significant family histories of bipolar disorder, and onset may occur earlier than in bipolar disorder alone (Faraone, Biederman, Mennin, Wozniak, & Spencer, 1997; Faraone, Biederman, & Monuteaux, 2001). This subgroup may therefore represent a genetically distinct subset of ADHD.

Tourette syndrome (TS) may represent one-way comorbidity; 35–80% of children with TS demonstrate ADHD (Comings, 2001; Kadesjo & Gillberg, 2000). In comparison, less than 2% of children with ADHD are diagnosed with TS, although 10–15% may be expected to demonstrate simple tics (Spencer et al., 2001).

In contrast to the frequency with which the above comorbid diagnoses occur, relatively few children with ADHD (2–4%) will also demonstrate a comorbid obsessive-compulsive disorder (OCD). Obsessive-compulsive disorder risk increases slightly with age and obsessive-compulsive behaviors are typically more frequent in families with a minimal history of ADHD. Children with OCD may demonstrate better attention and perfectionistic tendencies while at school, but more obvious detrimental obsessive compulsive behaviors within the home (Arnold, Ickowicz, Chen, & Schachar, 2005).

Developmental and health related problems

Learning disorder is an especially common comorbid diagnosis (20–25%), irrespective of learning disability type (Pliszka, 2000). Problems with academic functioning may begin as early as the preschool years (Barkley, Shelton et al., 2002) and affect pre-academic skills acquisition (DuPaul, McGoey, Eckert, & VanBrakle, 2001). Preschool children with ADHD on average scored one standard deviation below a control group on the Batelle Developmental Index (Newborg, Stock, & Wnek, 1988). The influence on academic performance and productivity may unfold over time as deficiencies in attention, working memory, and intellectual development combine with ODD/CD problems (Rapport, Scanlan, & Denney, 1999) to impede the acquisition of basic academic skills. Difficulty with basic mathematics, and pre-reading skills is often noted during the first school year (Mariani & Barkley, 1997; Spira & Fischel, 2005) and continues through later grades. Risk for reading disorders (16–39%) is similar to, or slightly above risk for developing spelling problems (24–27%) or math difficulties (13–33%) (August & Garfinkel, 1990; Casey, Rourke, & DelDotto, 1996; Semrud-Clikeman et al., 1992). Reading and listening comprehension deficits are not uncommon and may be related to the adverse impact of inattention (Aaron, Joshi, Palmer, Smith, & Kirby, 2002), working memory (Martinussen & Tannock, 2006) or executive problems of ADHD (Miranda, Soriano, & Garcia, 2005; Samuelsson, Lundberg, & Herkner, 2004). By late childhood ($\sim$ 11 years), as many as 80% of children with ADHD may fall two or more grades below grade level (Cantwell & Baker, 1992). In addition, writing deficits are fairly common particularly among children with ADHD-C (Marcotte & Stern, 1997).

Speech and language disorders commonly co-occur with ADHD (Baird, Stevenson, & Williams, 2000; Bruce, Thernlund, & Nettelbladt, 2006; Cantwell & Baker, 1992; Cohen et al., 2000; Damico, Damico, & Armstrong, 1999) and are reported to be as high as 40% and 64% in some samples (Gross-Tsur, Shalev, & Amir, 1991; Humphries, Koltun, Malone, & Roberts, 1994; Szatmari, Offord, & Boyle, 1989a; Taylor, Sandberg, Thorley, & Giles, 1991). This appears to be a two-way comorbidity as children with speech and language disorders also have higher-than-expected prevalence of ADHD (30–58%) (Tannock & Brown, 2000). In addition, recent studies identify coexistence of central auditory processing problems (Breier, Gray, Klaas, Fletcher, & Foorman,

2002; Gomez & Condon, 1999; Riccio, Hynd, Cohen, Hall, & Molt, 1994).

Neurological soft signs indexed by sluggish or otherwise poor motor coordination and excessive-for-age "overflow" or "mirror movements" are common in ADHD (Denckla, Rudel, Chapman, & Krieger, 1985). Motor coordination problems were found in 60% of children with ADHD, compared to 35% of controls (Kadesjo, Kadesjo, Hagglof, & Gillberg, 2001; Mariani & Barkley, 1997). However, simple repetitive motor coordination or speed is less affected than the execution of complex sequences (Klimkeit, Mattingley, Sheppard, Lee, & Bradshaw, 2005; Marcotte & Stern, 1997; Seidman et al., 1995). Interestingly, connections exist between ADHD and developmental coordination disorder (Sergeant, Piek, & Oosterlaan, 2006). The rate of comorbidity between the disorders is close to 50% (Barkley, 1990) and they may share genetic etiology (Martin, Piek, & Hay, 2006).

Comorbid diagnoses in some instances may represent common outcomes of those neurological circumstances that were the etiological factors responsible for ADHD, i.e., comorbidity may imply a common underlying etiology that subsequently results in the expression of two or more different disorders (Gillberg et al., 2004). For example, childhood tic disorder is a disturbance of dopaminergic metabolism that may also impact cognitive function and result in a constellation of problems characteristic of children with ADHD. In other cases, ADHD may contribute to the emergence of the comorbid disorder. For example, ODD in some cases may be an emergent property of the impact of ADHD on emotional self-regulation. In these cases, the causal ties may be exemplified by observations that pharmacologic treatment of ADHD often will reduce ODD symptomatology. In other cases, the comorbidity of ODD may imply underlying pathophysiology that predicts the persistence of ADHD over the course of development. Similarly, early emergence of CD may represent a unique subtype associated with more severe and persistent antisocial behavior. Often this emerges in families with significant psychopathology, including antisocial behavior, substance abuse, or major depression. By contrast, CD emerging post-pubertally (>12 years) is more often related to social disadvantage, familial conflict, or the influence of deviant peers. Hostile, inconsistent or indiscriminant parenting may contribute to the emergence of ADHD and CD (Drabick, Gadow, & Sprafkin, 2006). Parenting styles that are driven by emotional extremes, e.g., episodically harsh or permissive, are particularly implicated. Clinical expertise is necessary to resolve the predicament of the conflictual relationship between two or more disorders, and to enable meaningful interpretations that are most likely to result in application of appropriate intervention.

While ADHD does not appear to follow a uniform developmental course, several trends and patterns are noted. Clinical observations and empirical studies find a decline in total ADHD symptoms of hyperactivity, impulsivity, and inattention with increasing age (Biederman et al., 2000), while the probability of a comorbid diagnosis increases with age. By adulthood, only 13% of individuals with ADHD are free of comorbid diagnosis (McGough et al., 2005). The most common comorbid diagnosis in adulthood is general anxiety disorder (53%), followed by alcohol abuse/dependence (34%), drug abuse (30%), cyclothymia (25%), dysthymia (25%), panic disorder (15%), and obsessive-compulsive disorder (13%).

Aside from these widely recognized psychiatric comorbid disorders, the diagnosis of ADHD is complicated by the existence of a variety of social and medical conditions that can mimic characteristics of the disorder. While children with ADHD are twice as likely to experience sleep disorder than controls (Corkum, Rimer, & Schachar, 1999; Gruber, Sadeh, & Raviv, 2000; Marcotte et al., 1998), some sleep problems such as obstructive sleep apnea (Marcotte et al., 1998; O'Brien et al., 2003), and restless legs syndrome (Wagner, Walters, & Fisher, 2004) may be associated with difficulties similar to ADHD. In addition, ADHD symptomology can occur in the context of medication side effects, hypothyroidism (Rovet & Hepworth, 2001; Stein, Weiss, & Refetoff, 1995), anemia, chronic adenoidal/tonsillar hypertrophy (Miller & Castellanos, 1998).

Comorbidity and subtype

A presumed benefit of specifying clinically meaningful subtypes of ADHD is facilitation of the prediction of the extent and character of associated functional impairments (McBurnett et al., 1999). Consistent with this goal, the DSM-IV field trials found differences in ADHD type relative to age of onset, gender balance, and level of academic and social impairment. Academic deficits and peer unpopularity appeared more common for individuals with ADHD-PIA whereas individuals with ADHD-PHI were more likely to experience peer rejection and accidental injuries. In addition, the individuals with ADHD-PHI were more strongly associated with conduct problems (Lahey et al., 1994).

Continued investigation supported suggestions that the pattern of functional impairment may differ according to ADHD type. There is evidence of increased symptom severity in ADHD-C (Levy et al., 2005), but with few differences observed on measures of neurodevelopmental, academic, and cognitive functioning (Tripp, Luk, Schaughency, & Singh, 1999). Different ADHD types were associated with different rates of psychiatric disorder comorbidity (Eiraldi, Power, & Nezu, 1997; Faraone, Biederman, Weber, & Russell, 1998; Power, Costigan, Eiraldi, & Leff, 2004; Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999). In general, ADHD's impact in terms of comorbid psychiatric

symptoms, social dysfunction, associated cognitive difficulties, and impairment in academic function is greatest for the ADHD-C type, followed by ADHD-PIA and then ADHD-PHI. However, the relationship between level of impairment and ADHD type is dependent on the specific functional domain under consideration. For example, while ADHD-PIA and ADHD-C may demonstrate greater internalizing problems and academic impairment (Bauermeister et al., 2005; Power et al., 2004), disruptive behavioral problems tend to be more evident in ADHD-PHI and ADHD-C (Gadow et al., 2004; Nolan, Volpe, Gadow, & Sprafkin, 1999).

Several attempts have been made to further fractionate the DSM types into subtypes by incorporating information about developmental course and comorbid conditions. ADHD-C may be comprised of two subtypes distinguishable on the basis of the presence or absence of CD (Fischer, Barkley, Smallish, & Fletcher, 2005). Approximately 20–45% of children with ADHD-C will demonstrate early-onset CD symptoms, and these children are especially likely to demonstrate psychopathic tendencies as adolescents or adults. The presence of comorbid CD symptoms differentiates this subtype from ADHD-C without CD (Barkley, Shelton et al., 2002). Overall, children with ADHD-C appear more likely to experience severe anxiety and depressive symptoms than other types (Power et al., 2004; but see Volk, Neuman, & Todd, 2005; Willcutt et al., 1999).

In summary, comorbid conditions are commonly associated with ADHD but their nature and significance are the subject of continued interest. While some argued that a comorbid diagnosis has little or no effect on treatment outcome or approaches (Kolko, Bukstein, & Barron, 1999; MTA, 1999a) others contend that comorbid conditions have a significant impact on life course and associated functional impairments (Barkley, 2002). In general, it appears that comorbid conditions may reasonably be expected to have significant implications for treatment planning although direct one-to-one correspondence between treatment type and functional outcomes remains to be resolved empirically.

Diagnostic dilemmas related to DSM-IV criteria

While ADHD has good clinical validity (Faraone, 2005; Lahey & Willcutt, 2002), there is widespread recognition of the need for continued refinement of the operational criteria used to diagnose the disorder (Achenbach, 2000; Levy, Hay, McLaughlin, Wood, & Waldman, 1997). The empirically derived diagnostic criteria for ADHD as set forth in DSM-IV represented a significant advance over previous taxonomies, but a number of persistent problems have emerged with clinical application of this diagnostic scheme. These relate in part to limitations in symptom specification, insufficient consideration of developmental course, age, gender, and maturational stage, heterogeneity of subtypes, unspecified influence regarding non-empirically based age demarcation for both diagnosis and duration, and indifference to environmental contextual considerations.

A number of the concerns surrounding the DSM-IV approach to the diagnosis of ADHD stem from the framework's underemphasis on developmental differences and situational factors. The same criteria are used irrespective of chronological age, and adjustment is not made for age appropriate behavioral change. Due to lack of developmental gradation of ADHD symptoms, children may cross subtype boundaries as they mature, resulting in considerable instability of the DSM typology. For example, DSM-IV field trial data indicated that while ADHD-C was primarily evident in school-aged children, ADHD-PHI was primarily diagnosed in preschool children. Recent longitudinal studies (Lahey, Pelham, Loney, Lee, & Willcutt, 2005) have suggested that children who met criteria for ADHD-PHI at baseline were less likely to meet criteria for ADHD in subsequent years than children diagnosed with ADHD-C. Attention-Deficit/Hyperactivity Disorder-PHI identified early in life may therefore be a transient problem in a proportion of children, one that is eventually outgrown. However, of those who continue to meet criteria for ADHD, many (76%) meet criteria for ADHD-C at some point in subsequent assessments. This suggests that that ADHD-PHI may be a precursor to ADHD-C later in life (Lahey et al., 1994). Similarly, ADHD-C may evolve into ADHD-PIA with abatement of hyperactivity symptoms due to developmental change or treatment (DuPaul & Stoner, 1994; Marsh & Williams, 2004). Relatedly, whether or not a child is categorized as ADHD-C or ADHD-PHI may reflect the developmental stage at which symptoms become evident. Based on developmental trends, PHI symptoms are more likely to occur earlier in development followed by problems related to inattention (Hart, Lahey, Loeber, Applegate, & Frick, 1995; Loeber, Green, Lahey, Christ, & Frick, 1992). Accordingly, as the full expression of the disorder takes time to unfold, children brought to attention early may be diagnosed as ADHD-PHI whereas later the same children may meet criteria for ADHD-C. While the instability of ADHD types may result from developmental changes in symptomology, they may also entail measurement error and regression to the mean or changes in situations or informants (teachers).

The lack of stability of ADHD types, as currently defined, contributes to the substantial heterogeneity that exists within each type. Although the DSM-IV field trial provided little evidence to support ADHD-PHI as a distinct clinical entity (Lahey et al., 1994), subsequent research has argued that these children may differ in important ways from children diagnosed with another ADHD subtype (Gadow et al., 2004). According to parent report, these children

demonstrate milder internalizing symptoms of inattention and depression, have poor peer relations compared to ADHD-C children, and exhibit more oppositionality than ADHD-PIA children. Barkley and colleagues (Barkley, 2006) posit at least three subtypes of ADHD-PHI; a majority of these children (~90%) comprise a subgroup who develop into a C type; a second subtype includes preschool children who present mainly with ODD; and, a third subtype remains classified as ADHD-PHI because ADHD-C symptomatology remains at sub-threshold levels.

Heterogeneity in ADHD-PIA is also recognized and the PIA diagnosis may be reached via different routes (Barkley, 2006). One PIA subtype may demonstrate sub-threshold C type symptoms and is best viewed as a milder or sub-threshold C type (Milich, Balentine, & Lynam, 2001). Relatedly, another subtype may meet criteria for ADHD-C early in life but evolve into a diagnosis of ADHD-PIA, given that hyperactivity-impulsivity symptoms decrease over time while inattention persists (Ingram, Hechtman, & Morgenstern, 1999; Wolraich et al., 2005). A third subgroup of children with ADHD-PIA may be distinct from the other two subgroups (Barkley, 2001; Hartman, Willcutt, Rhee, & Pennington, 2004; Milich et al., 2001). This group, possibly comprising 30–50% of children diagnosed with ADHD-PIA, is characterized by sluggish cognitive tempo and a clinical presentation that includes easy confusion, daydreaming, staring into space, and mental spaciness along with hypoactive, slow-moving, lethargic, and sluggish motor function (Carlson & Mann, 2002; McBurnett, Pfiffner, & Frick, 2001). Whereas children with ADHD-C demonstrate distractibility and impaired persistence of effort, these children with ADHD-PIA process information in a slow and error-prone manner with poor focused or selective attention and have passive learning styles (Carlson, Booth, Shin, & Canu, 2002). Socially, they are withdrawn or introverted and are less prone to initiate social interactions (Bauermeister et al., 2005). Some suggested that ADHD-PIA children are more likely to demonstrate math disorder (Marshall, Schafer, O'Donnell, Elliott, & Handwerk, 1999), but others report equal impairment across a number of different academic skills (Zentall & Ferkis, 1993). Several reports suggest that these children demonstrate right hemisphere implicating profiles on neuropsychological assessment (Landau, Auerbach, Gross-Tsur, & Shalev, 2003; Stefanatos & Wasserstein, 2001). Given the distinctive behavioral profile, pattern of comorbidity, and response to medication (Carlson & Mann, 2000; Hynd et al., 1991), some have suggested that these children may not be a different type of ADHD, but a separable disorder with an alternative underlying neurobiology (Hinshaw, 2001; Milich et al., 2001). A recent meta-analysis by Waldman and Faraone (2002), for example, suggests that the dopamine transporter gene DAT1 is more closely associated with ADHD-C than ADHD-PIA.

A corollary problem of the failure of the diagnostic algorithm to accommodate developmental changes is that reductions in symptom presentation (i.e., remission rates) may reflect the static nature of the criteria rather than the true course of the disorder (Biederman et al., 2000). Of those diagnosed in childhood, 20% to 30% will continue to meet criteria for ADHD during late adolescence (Muglia, Jain, Macciardi, & Kennedy, 2000), and fewer in adulthood (Dulcan et al., 1997; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998). There are sample data that the core symptoms of hyperactivity-impulsivity decrease over time (DuPaul & Stoner, 1994), while inattention may persist (Ingram et al., 1999; Wolraich et al., 2005). While some individuals experience functional remission (full recovery), most are predicted to demonstrate qualitatively similar problems at older ages while failing to meet diagnostic criteria that do not accommodate to maturational change (Biederman et al., 2000).

Relatedly, both the cutoff and specific items chosen for inclusion in the the DSM-IV symptom list may have limited generalizability to age groups outside the 4–16 years age range. For children younger than 4 years, the cutoff has the potential for an increased false positive rate since symptom list items are developmentally inappropriate at these younger ages. Conversely, application of these thresholds to adolescents and adults may result in underdiagnosis and a greater false negative rate because hyperactivity decreases significantly with increasing age (Barkley, Fischer et al., 2002; Hart, Lahey, Loeber, Applegate, & Frick, 1995; Hill & Schoener, 1996; Lobar & Phillips, 1995). Moreover, item content does not transition well across age ranges. For example, failing "to finish schoolwork, chores, or duties in the workplace" is inapplicable to young children, and "often having difficulty playing ... quietly" is not especially relevant for adults. DSM-IV's implied static view of psychopathology is not empirically supported. As a result, there is growing support for a dimensional view, one that proposes that ADHD represents an extreme of, or delay in, a normal trait and therefore needs to be diagnosed as a relative deficit compared to normal developmental expectations (Levy, Hay, McStephen, Wood, & Waldman, 1997). There are no explicit guidelines on how to accommodate or make adjustments when literal interpretation of symptoms are not developmentally appropriate.

Another apparent weakness regarding the DSM-IV criteria is the failure to acknowledge gender difference in the manifestation of ADHD symptomatology. As discussed above, young males are more likely to demonstrate behaviors consistent with DSM symptom criteria than are young girls (Gershon, 2002), perhaps a reflection of the fact that a majority of children in the DSM field trial were male (Lahey et al., 1994). As a consequence, some recommend that symptom cutoff scores be sex referenced (Kato et al., 2001; Waschbusch & King, 2006).

The DSM-IV's age of onset cutoff of seven years appears to lack historical or empirical justification (Barkley & Biederman, 1997). Qualitative differences are not found between children who display symptomatology before or after this cutoff age. Data suggest an earlier onset may reflect a more severe disorder with more persistent symptoms and secondary problems (McGee, Williams, & Feehan, 1992). Interestingly, while nearly all of a cohort who met behavioral criteria for ADHD-PHI demonstrated an age of onset prior to seven years (Applegate et al., 1997), 18% of a cohort who met symptom criteria for ADHD-C, and 43% of youths who met symptom criteria for ADHD-PIA failed the age of onset criteria. The application of the age cutoff reduced the accuracy of identification. Given that ADHD symptoms may be difficult to distinguish from other behavioral problems that manifest early, until at least age three years (Egger et al., 2006) there may be substantial benefit for establishment of a lower bound for the age at which a diagnosis is appropriate. There is wide consensus that the current age cutoff appears inappropriate, ill-founded, and in need of revision (Rohde et al., 2000).

An additional problem regarding the DSM-IV diagnostic schema is the requirement for six months symptom duration. The problems experienced by a child with ADHD often become noticeable soon after preschool enrollment (Barkley, 1990; Campbell, 1995) or by early elementary school grades, when they fail to comply with behavioral expectations imposed by the novel classroom structure. Such problems may persist for an extended time, particuarly if the child adapts slowly or poorly to the new situation. It is clearly important to monitor symptoms for a sufficient length of time to rule out transient or contextual determinants of the symptomatology. Confounding circumstances may exist, such as illness or death of a family member or pet, parental discord, parental job stressors, sibling conflicts, anxious concerns unresolved, sleep problems, reaction to negative news reports, personality style or temperament inconsistent with teacher expectation, developmental delay such as for writing neatly as teacher expects, peer bullying or peer pressure. Each may be a stressor that prompts or potentiates ADHD-like behaviors but not necessarily indicative of a diagnosable disorder. As a result, some argue that six months may be an insufficient term and suggest a duration of 12 months or longer (Barkley, 2006; Beitchman, Wekerle, & Hood, 1987; Palfrey, Levine, Walker, & Sullivan, 1985).

Criticism has also arisen regarding the DSM-IV requirement that symptoms be demonstrable in at least two of three environments. This requirement implies that agreement is reached by at least two of three informational sources (parent, teacher, or employer). Yet, the research often finds only modest concordance between parent and teacher observations (0.3–0.5), in part because informational source is confounded with setting (Achenbach, McConaughy, & Howell, 1987; Lambert et al., 1978; Mitsis, McKay, Schulz, Newcorn, & Halperin, 2000; Schachar, Rutter, & Smith, 1981). For example, a teacher may initiate complaints about a child's behavior in the absence of overt parental concern. Common complaints include a high frequency of: (1) off task behavior; (2) poor and inaccurate completion of assignments; (3) poor completion of assignments in a timely fashion; (4) higher frequency of transgression of classroom rules (e.g., play with "off-limits toys"; and (5) disruptive classroom behavior (Atkins, Pelham, & Licht, 1985; Byrne, DeWolfe, & Bawden, 1998). Such behaviors are associated with poorer academic achievement, higher rates of retention, greater need for academic tutoring or special class placement, referral for disciplinary action, expulsion and a higher dropout rate (DuPaul, 2006; Faraone et al., 1993; Hinshaw, 1992; LeFever, Villers, Morrow, & Vaughn, 2002). Many of these behaviors will not be noted in a home environment that may be relatively unstructured and where there are fewer expectations and more lax rules. One alternative is to integrate parent and teacher reports or a history of symptoms in order to more accurately document the number of different symptoms endorsed by both sources (Crystal, Ostrander, Chen, & August, 2001; Mitsis et al., 2000). Based on DSM-IV field trials, "requiring two corroborating sources (e.g., parent and teacher) to make a diagnosis (i.e., pervasive model) may be too restrictive" (August & Garfinkel, 1998, pg. 446). The requirement of agreement across parent, teacher, and clinician severely reduces the diagnosis, particularly for PIA and PHI types (Mitsis et al., 2000; Szatmari, Offord, & Boyle, 1989b).

Neuropsychological constructs and ADHD

One of the challenges to current conceptions of ADHD within a medical "disease construct" model is to document in a scientifically rigorous manner an identifiable nexus of dysfunction involving psychological or biological mechanisms that can account for the behavioral phenotype and justify a "disorder" designation (Wakefield, 1992). It was suggested that neuropsychological impairment may serve as that explanatory mechanism (Barkley, 1997a), since deficiencies in specific neuropsychological processes can potentially elucidate why the disorder develops and how it is expressed. Candidate processes include deficiencies in aspects of executive function (EF) including effortful attention (Douglas, 1983), inhibitory control (Barkley, Grodzinsky, & DuPaul, 1992; Nigg, 2001; Schachar & Logan, 1990; Sergeant & Scholten, 1985), working memory (Karatekin & Asarnow, 1998; Mariani & Barkley, 1997; Tannock, 1998), planning or set shifting (Harrier & DeOrnellas, 2005; Nigg, Hinshaw, Carte, & Treuting, 1998) and delay aversion (Sonuga-Barke, 2003). More recently, models have attempted to explain ADHD in less dimensionally-constrained terms, proposing

involvement of multiple developmental pathways in ADHD (Nigg, 2005b; Sonuga-Barke, 2005a). These models help to account for the heterogeneity of this disorder, and have potentially profound implications for both clinical practice and research methodology.

The origins of current theory regarding the neuropsychological basis of ADHD can be traced to Douglas' (1972) contention that disturbances of sustained attention or vigilance comprised one of a constellation of closely-related deficits that had a broad impact on the behavior, learning ability and cognitive function in "hyperactive" children. Attention was recognized as a complex, multidimensional construct related to a diverse range of fundamental abilities such as concentration, exertion of mental effort, staying alert and watchful, focusing, and ignoring distractions. Attentional problems could therefore be construed to account for diverse parental or teacher complaints of "off task" behavior such as poor concentration, reduced persistence on tedious tasks such as homework assignments, susceptibility to distraction, forgetfulness, poor listening skills, and difficulty remaining alert and focused, among others. Inattentive behavior in ADHD did not appear secondary to the existence of other potential comorbid conditions (e.g., anxiety, depression, conduct disorder, oppositional defiant disorder) (Klorman et al., 1999; Murphy et al., 2001; Nigg, 1999) and could to some extent distinguish children with ADHD from other childhood psychiatric disorders (Barkley, DuPaul, & McMurray, 1990; Chang et al., 1999; Swaab-Barneveld et al., 2000). However, the concept of inattention has proven challenging to operationalize due to its multidimensional nature. It is difficult to separate general, superordinate attentional mechanisms from domain or task-specific processes, and it has been difficult to dissociate these systems experimentally in a manner that maintains relevance to real-world functioning. Consequently, the ecological validity of laboratory measures has been questioned (DuPaul, Anastopoulos, Shelton, Guevremont, & Metevia, 1992; Weis & Totten, 2004). Nevertheless, data support this theory and find children with ADHD repeatedly not performing as well as matched controls on neurocognitive tests of vigilance and sustained attention, such as the continuous performance task or CPT (Berwid et al., 2005; Halperin et al., 1990; Inoue et al., 1998; Seidman et al., 1998) where inattentiveness is indexed as errors of omission. Performance on such tasks appears positively correlated with ADHD symptoms (Anderson, Anderson, & Anderson, 2006; Epstein et al., 2003; Mahone, Pillion, Hoffman, Hiemenz, & Denckla, 2005; Marks et al., 2005; Nichols & Waschbusch, 2004). Correlations with behavioral measures of inattention vary and are at best moderate. However, when combined with other measures, these adjunctive data appear effective to identify and categorize ADHD (Marks, Himelstein, Newcorn, & Halperin, 1999; Schatz, Ballantyne, & Trauner, 2001).

One of the most critical EF components considered deficient in ADHD is inhibitory control (Barkley, 1997c; Fischer et al., 2005; Nigg, 2001; Schachar, Mota, Logan, Tannock, & Klim, 2000), i.e., the ability to stop (completely and suddenly) an activity or planned course of action (Logan & Cowan, 1984). Inhibitory control is considered central in controlling responses to environmental events in everyday life, and a breakdown of inhibitory control serves as a potent marker for ADHD (Barkley, 1997c; Schachar et al., 2000). Among the many clinical manifestations of ineffective inhibition are inability to remain seated, impulsive object touching, persistent propensity to interrupt others (Malone & Swanson, 1993), impatience waiting in line or taking turns in games, and resistance to delayed gratification (Anderson, Hinshaw, & Simmel, 1994; Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001). Response inhibition difficulty may also contribute to motor overactivity, evidenced by fidgetiness, appearing "motor-driven," and loquaciousness. Performance-based measures particularly sensitive to deficiencies in inhibitory control demand that children wait and watch for events to occur (Losier, McGrath, & Klein, 1996; Newcorn et al., 2001). Responses that occur too quickly or too often signal a problem (Berwid et al., 2005; Fischer et al., 2005; Gordon & Mettelman, 1988; Ossmann & Mulligan, 2003; Riccio, Reynolds, Lowe, & Moore, 2002). Poor performance on the stop signal test and CPT commission errors distinguish children with ADHD from controls (Nigg, 2001; Oosterlaan & Sergeant, 1998; Pennington & Ozonoff, 1996; Schachar, Tannock, & Logan, 1993).

A developmental perspective leads to the presumption that early-appearing processes involved in inhibitory control are precursors of, or are foundational for, general problems with executive function (Barkley, 1997c; Barkley et al., 1992; Nigg, 2001; Oosterlaan, Logan, & Sergeant, 1998; Quay, 1997). Barkley (1997c) specifically linked poor inhibitory control to problems in four executive neuropsychological functions: (1) working memory; (2) self-regulation of affect-motivation-arousal; (3) internalization of speech; and (4) reconstitution (behavioral analysis and synthesis). Data from several studies found inhibitory control deficits were not explained by differences in IQ, comorbid psychiatric disorder (Barkley, Murphy, & Bush, 2001; Halperin et al., 1992; Nigg, 1999; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997), or learning disability (Accardo et al., 1990; Fletcher, Shaywitz, & Shaywitz, 1999; Klorman et al., 1999). However, inhibitory control deficits are not unique to ADHD, are observed in other disruptive disorders (Oosterlaan et al., 1998; Sergeant, Geurts, & Oosterlaan, 2002), and worsen when ADHD is comorbid with other disorders (Purvis & Tannock, 2000; Willcutt et al., 2001). In addition, data suggest that with at-risk preschoolers problems with inhibitory function and other EF capabilities may be mediated by difficulties in nonexecutive abilities and

appear unrelated to ratings of ADHD symptoms or objective indices of activity level (Marks et al., 2005).

It should be noted that disinhibition does not easily account for some experimental findings in children with ADHD, such as slower response production on the Stop Signal test (Nigg, 1999; Purvis & Tannock, 2000; Schachar & Logan, 1990; Schachar et al., 2000; Schachar, Tannock, Marriott, & Logan, 1995). Slowed response inhibition may be better interpreted as a reflection of general constraints in processing speed rather than as a specific response inhibition deficit (Oosterlaan et al., 1998; Overtoom et al., 2002; Tannock, 1998). Others found no substantial variability in response speed for those with ADHD without significant differences in response inhibition (Kuntsi, Oosterlaan, & Stevenson, 2001; Scheres et al., 2001). Nigg (2001) has argued that inhibitory deficits mainly emerge when task performance requires suppression of a prepotent motor response (e.g., basic go/no go tasks, stop signal test) and are variably evident in other circumstances in which performance requires suppression of a conflicting response (e.g., flanker tests, interference condition on the Stroop test). Supportive meta-analyses found larger effect sizes for the stop signal reaction time (0.54–0.85) (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Lijffijt et al., 2005) compared to Stroop interference scores (0.13–0.35) (Hervey, Epstein, & Curry, 2004; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

Working memory is an aspect of EF that is assumed to play an important role in ADHD, although comprehensive and systematic studies are lacking. Working memory refers to the capacity to maintain information or a goal in mind in order to take action despite interference (Baddeley & Hitch, 1974), providing an essential "interface between perception, attention, memory, and action" (Baddeley, 1996, pg. 13472). Deficits in verbal working memory were observed on measures such as digit span, (Biederman, Faraone, Milberger et al., 1996; Karatekin, 2000), with worse backward than forward span (Mariani & Barkley, 1997; McInnes, Humphries, Hogg-Johnson, & Tannock, 2003). Spatial working memory task results have been inconsistent (see Karatekin, 2004 for review). Barkley (1997c) has argued that many problems attributed to inattention could be more accurately conceptualized as problems involving working memory. However, tasks cited to index working memory (recalling past information, formulation of plans) were complex and would involve central executive and possibly strategic components. However, recent meta-analyses support the existence of working memory problems in ADHD (Boonstra et al., 2005; Hervey et al., 2004; Willcutt, Doyle et al., 2005). Moreover, Martinussen et al. (2005) found larger effect sizes for various measures of spatial working memory (0.85–1.06) compared to verbal working memory (0.47–0.56). Interestingly, these findings are consistent with neuropsychological profiles implicating greater right versus left hemisphere involvement in ADHD (Heilman, Voeller, & Nadeau, 1991; Landau et al., 2003; Stefanatos & Wasserstein, 2001) and with neuroimaging findings (Casey et al., 1997; Giedd, Blumenthal, Molloy, & Castellanos, 2001; Makris et al., 2006).

Executive function deficits involving effortful attention, response disinhibition, and working memory have been reported in numerous studies of children (Fischer et al., 2005), adolescents (Seidman, Biederman, Faraone, Wever, & Ouellette, 1997), and adults (Hervey et al., 2004; Lovejoy et al., 1999; Seidman et al., 2004). That performance deficiencies across several measures can distinguish children with ADHD from controls with reasonable precision suggests that such tasks can contribute substantially to the identification of ADHD (Berlin, Bohlin, Nyberg, & Janols, 2004; Kalff et al., 2002) However, inconsistencies exist in ascribing priority to each of these processes, and the relationships among them. For example, while Barkley (1997c) posits a "core" inhibitory deficit impairs development in several areas of executive function including working memory, Denney and Rapport (2001) suggest that working memory deficit is the primary causal process, resulting in secondary deficits in disinhibition and impulsivity. In addition, considerable ambiguity exists regarding the concept of EF itself and the often undifferentiated manner in which it is assessed.

One of the great challenges inherent to all ADHD conceptualizations is the necessity that one define what is meant by a term and operationalize it in a manner that allows for clear hypothesis testing and replication. Not only is there disagreement as to which component of executive function is crucially related to ADHD, but problems are inherent to concept definition and test development to measure these constructs. As a result, it is difficult to compare component control functions. Indeed, the tendency to define executive function with reference to purported measurement instruments may be especially problematic. For example, the Stroop Color-Word Test (SCWT, Golden, 1978), and its modifications, is often considered a sensitive EF measure and poor SCWT performance in individuals with ADHD is often taken as evidence of poor interference control. However, a number of investigators have reported performance on the interference condition without controlling for performance on the colour naming condition, despite the importance of this distinction and a means for calculation of this variable. Performance on the Interference condition may be deficient in ADHD for reasons that have little to do with EF (Tannock, 1998). According to a recent meta-analysis, children with ADHD no longer demonstrated poor interference control on the SCWT when non-interference aspects of the task were taken into account (van Mourik, Oosterlaan, & Sergeant, 2005).

Confounding efforts to identify fundamental neuropsychological processes that account for ADHD is the

disorder's inherent heterogeneity. Most neuropsychological studies pertain to ADHD-C while comparatively little attention was devoted to ADHD-PIA. Efforts to characterize ADHD types on the basis of neuropsychological patterns of performance or, alternatively, to validate existing behaviorally-defined subtypes using neuropsychological data had mixed results. Chhabildas et al. (2001) tested the hypothesis that ADHD-PHI symptomatology may be associated with behavioral inhibition deficits, whereas ADHD-PIA symptomatology may be associated with deficiencies in processing speed and vigilance. Children with ADHD-C may be expected to demonstrate deficits in both areas. Contrary to predictions, similar patterns of neuropsychological impairment were apparent across all three groups and symptoms of inattention best predicted performance across all measures and ADHD types.

Recent views have posited that problems with inattention and disinhibition may be conceptualized as manifestations of a disorder related to critical aspects of self-regulation (Nigg, 2001), state regulation (Berwid et al., 2005), or dysregulation or dyscontrol of cognitive energetic resources (Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003). Self-regulation problems can be considered to have at least three components: an inhibitory component, an attention component and an organizational or strategic component (Douglas, 2005). According to state-regulation deficit models (Douglas & Peters, 1979; Sanders, 1983; Sergeant, 2005; Van der Meere, 1996), ADHD results from failure to sufficiently modulate physiological state to meet task demands, events, and/or circumstances. Deficiencies can potentially occur at three distinct levels: (1) lower-level cognitive processes such as response organization; (2) an "energetic pool" such as arousal, activation and effort; and (3) executive function related to activation of pooled resources. Consistent with this notion, children with ADHD are often slower and less likely to return to activities once interrupted and they are less proficient at flexibly shifting attention across tasks (Borger & van der Meere, 2000; Hoza, Pelham, Waschbusch, Kipp, & Owens, 2001; Seidman, Biederman, Faraone, Weber et al., 1997).

Overall, these conceptualizations attempt to integrate findings derived from different levels of analysis. The physiological basis considered to underlie a number of these disturbances implicates the fronto-dorsal striatal circuitry and associated dopaminergic innervation (e.g., mesocortical) (Arnsten, 2006; Barkley, 1997b; Biederman & Faraone, 2005; Castellanos, 1997; Wender et al., 2001; Woods, Lovejoy, & Ball, 2002), although noradrenergic systems have also been implicated in animal studies, genetic investigations, and stimulant medication trials (Arnsten & Dudley, 2005; Biederman, 2005; Biederman & Spencer, 1999; Levy & Swanson, 2001; Oades et al., 2005; Schmitz et al., 2006; Viggiano, Ruocco, Arcieri, & Sadile, 2004). In addition, disruption in other prefrontal regions or related pathways could lead to apathy and failures in initiation, both also sometimes evident in ADHD. Symptoms of inattention may also relate to a breakdown in the distributed attentional systems known to be preferentially located in right cerebral regions (Booth et al., 2005; Stefanatos & Wasserstein, 2001; Voeller & Heilman, 1988).

There is growing recognition that the symptom complex characteristic of ADHD cannot be exclusively accounted for by cognitive dysregulation but must incorporate dysfunction of pathways involving motivation or alterations in reward processes (Barkley, 1997b; Nigg, 2005b; Sonuga-Barke, 2003). So-called "dual route" models of ADHD emerged to account for ADHD heterogeneity in part as a result of observations that children with ADHD and matched controls differed depending on whether the dependent variable was speed or accuracy, and which of these was specifically emphasized in the test instructions (Manly et al., 2001). Findings from several studies (Sonuga-Barke, 2002; Sonuga-Barke, Dalen, & Remington, 2003) suggested that impulsive behaviors may be reconceptualized as a functional response aimed at avoiding delay, i.e., delay aversion. From a biological standpoint, this may result from abnormalities related to reward processes (Castellanos & Tannock, 2002; Douglas & Parry, 1994; Ernst et al., 2003; Iaboni, Douglas, & Baker, 1995; Sagvolden, Aase, Zeiner, & Berger, 1998; Tripp & Alsop, 2001) and hypofunctioning of dopaminergic fronto-ventral striatal reward circuits and meso-limbic branches that terminate in the ventral striatum, particularly the nucleus accumbens (Sonuga-Barke, 2005a). These separate routes are thought to make distinctive contributions to the emergence of ADHD through their influence on individual adaptation to developmental constraints.

Direct comparison of predictions based on these different accounts have not favored a "single theory of ADHD" (Solanto et al., 2001). Indeed, the search for a single or common core dysfunction in ADHD may be a "fool's errand" (Sonuga-Barke & Castellanos, 2005). While studies of normal children or adults allowed for fractionation of EF into separable components (Miyake et al., 2000; Rogers, Andrews, Grasby, Brooks, & Robbins, 2000), attempts to identify similar disassociations in ADHD children often found deficits "across-the-board" (Chhabildas et al., 2001; Kempton et al., 1999). Such concerns have led to caution that "peeling off the concept of executive function leaves us with a concept with homuncular properties that is neither observable, nor testable" (Band & Scheres, 2005, pp. 518), and it has been argued that to invoke executive system dysfunction as a key causal factor in ADHD may be so general an explanation as to account for either everything or nothing (Band & Scheres, 2005; Wilding, 2005). Clearly, the neuropsychological difficulties associated with ADHD are not likely confined to executive functioning nor

do all individuals with ADHD demonstrate problems with EF as currently measured (Biederman et al., 2004; Crosbie & Schachar, 2001).

Notwithstanding the need for more precisely defined concepts and better-designed, psychometrically-sound behavioral tasks, the field is in urgent need of methodological improvements and innovations. There needs to be greater recognition that neuropsychological constructs of interest in ADHD are multifaceted and need to be examined with the view that performance on any one task may have multiple determinants. Therefore, in order to establish a differential deficit affecting a particular ability, one must control for possible artifacts and demonstrate that effects converge across different measures of the same ability (Nigg, 2005a). Tests need to be more carefully and specifically designed with developmental concepts given primary emphasis, and not simply utilize downward extensions of adult measures. For example, the Wisconsin Card Sorting Test is an often used test regarded as a paradigmatic measure of frontal lobe function. However, effect sizes have generally been rather modest (0.35) (Frazier, Demaree, & Youngstrom, 2004). It would be useful to incorporate features (such as varying perceptual load (Huang-Pollock, Nigg, & Carr, 2005; Lavie & Tsal, 1994) that ensure that the same function is being assessed and that a comparable challenge level is engaged across different developmental levels. In addition, results need to be evaluated within a developmental context and in consideration of gender influences. Comparison groups beside the "normal control" would be useful. One strategy to address the known heterogeneity of current DSM-IV defined subtypes of ADHD in experimental designs is to utilize comorbid disorders to facilitate subgrouping and reduce within group heterogeneity. An alternative strategy would be to separate groups on the basis of poor performance on a particular set of neuropsychological measures, and then examine the external correlates of these so divided groups. Nigg et al. (2004), for example, found that children grouped according to whether or not they had the attentional impairments had family members with differential cognitive functioning.

Obstacles to assessment

The complexity of ADHD assessment is often not fully appreciated when erroneously considered merely a matter of establishing whether DSM-IV criteria are accurately met. An ADHD diagnosis is not reached as a result of a single objective test result since no independent test exists that will confirm an ADHD diagnosis (NIH Consensus Statement, 1998). However, objective test results can be interpreted with respect to their respective contributions to discrimination among comorbid diagnoses, and with respect to probable and typical patterns of ADHD subtype performance. Often, ADHD is a diagnosis of exclusion, once competing potential etiologies are considered and discarded. Yet, determinations need to be made in order to provide appropriate treatment interventions. How best to accomplish this is a source of continued controversy. While some guidelines support primary reliance on behavioral features and rating scales (American Academy of Pediatrics, 2000), others support reliance on standardized neuropsychological evaluation along with behavioral report, records review, and detailed partitioning of features that are not specifically addressed in DSM-IV but which clarify comorbidity and enhance diagnostic accuracy. A number of important references exist regarding neuropsychological assessment and findings in presumptive ADHD (Dige & Wik, 2005; Fischer et al., 2005; Gallagher & Blader, 2001; Nigg, 2005b; Perugini, Harvey, Lovejoy, Sandstrom, & Webb, 2000; Schoechlin & Engel, 2005; Seidman, 2006) for those wishing to review assessment concepts and methods in greater detail.

Nonetheless, it is worth noting that ADHD associated behavioral variability is both supportive of diagnosis and a complicating factor. Overall, ADHD assessment is only at a preliminary stage in addressing these many varied and complex issues. Clinical practitioners are acutely aware of the moment-to-moment variability possible in the behavior of a child suspected as having ADHD, and how clinical observations may not necessarily correlate with standardized test data obtained in an artificial structured test environment, thereby limiting confirmation of behavioral impressions obtained through objective means and generalization to the natural, real-world environment. Furthermore, the impact of diverse circumstances related to the home or other socioenvironmental contexts, or the child's psychological state and medical health, also may directly compromise the clinician's attempt to make sense of competing potential explanations for a child's functioning, which in turn may directly affect treatment decisions and application of interventions. The evidence base for an evaluation may vary depending on whether the purpose is diagnosis, treatment planning, determination of prognosis, or outcomes assessment (Mash & Hunsley, 2005). The fundamental reason for assessment in most clinical settings goes well beyond establishment of a diagnosis and, instead, involves case conceptualization, determination of need for treatment, delineation of treatment goals, development of treatment targets, and monitoring of progress and outcome. Documentation of symptoms of attention, impulsivity, and hyperactivity to determine whether a child meets DSM-IV criteria for ADHD diagnosis only partially addresses the clinical evaluation's purpose and the concerns that brought the child to attention. In order to develop an adequate treatment plan, it is also necessary to evaluate various impairments affecting daily life functioning in order to determine if there are any deficiencies in adaptive skills This entails an assessment of a host of other potential targets for intervention including, but not limited to, academic

achievement, peer relations, and parenting skills. Indeed, these ought to be a central focus of an assessment (Pelham, Fabiano, & Massetti, 2005).

Rating scales are used prominently in ADHD assessment and are an efficient means of measuring the severity of behavioral symptoms as perceived by informants familiar with the child's behavior (e.g., parents and teachers). Standardized parent and teacher rating scales are commonly used to rapidly screen for symptoms relevant to ADHD. The most frequently used questionnaires are the Achenbach Child Behavior Checklist (Parent and Teacher report forms) (Achenbach & Edelbrock, 1983), the Revised Conners Parent and Teacher Rating Scales (Conners, Sitarenios, Parker, & Epstein, 1998), the ADHD Checklist (DuPaul, Power, Anastopoulos, & Reid, 1998) and the Swanson, Nolan and Pelham (SNAP) Teacher and Parent Rating Scales (see www.adhd.net). The Achenbach Child Behavior Checklist, Parent and Teacher Report Forms sample a broader spectrum of behaviors than the others and is designed to stratify "externalizing behaviors" such as hyperactivity and aggression and "internalizing behaviors" related to anxiety and mood concerns. Scores along several behavioral dimensions (e.g., attention, social withdrawal, thought disorder) vary along a continuum from normal to "clinically elevated" and are not linked to specific psychiatric diagnoses. By contrast, the Conners, SNAP-IV, and similar scales are specifically linked to the DSM-IV criteria for ADHD, oppositional defiant disorder, and conduct disorder. However, rating scales are insufficient for diagnosis. Comprehensive evaluations, such as those performed in research clinics, typically will also rely on structured or semi-structured psychiatric interviews. Highly structured interviews do not entail interpretation of the informant's response; while more reliable, they may be less valid. Alternatively, semi-structured interviews require interpretation by a clinically trained and knowledgeable interviewer. These interviews tend to be more valid but less reliable. Both formats provide information regarding the presence of comorbid psychiatric or behavioral disorder. In addition, a comprehensive assessment should include a psychological or neuropsychological evaluation to ascertain the presence of comorbid learning or cognitive disorders.

Therefore, the purpose of ADHD assessment as currently practiced appears to be twofold: (1) assessment of DSM-IV symptoms, and (2) assessment of impairment. The former is a source of controversy, given the above noted weaknesses. While it is clearly important to establish whether a child meets DSM-IV criteria, this step alone is typically not the most appropriate referral basis (Angold et al., 1999) nor does it allow for prediction of long-term outcome (Mannuzza & Klein, 2000). The assessment of impairment needs to entail an analysis of the impact of ADHD as it relates to (1) difficulties and family functioning; (2) peer relations; and (3) academic functioning. Problems in these three domains are predictors of negative long-term outcome and comprise the targets of therapeutic intervention (Angold et al., 1999; Chamberlain & Patterson, 1995).

Conclusion

The prevalence of ADHD is substantial and an ADHD diagnosis carries with it a predictably enormous long-term impact of this major public health problem on personal goals, family and interpersonal relationships, educational learning objectives, and integration in society. The extensive research on neuroanataomic linkage, subtype partitioning, and treatment efficacy continues to be impressive and expanding at considerable speed. Yet a clinical perspective always needs to be maintained to temper the voluminous detail that emerges from these experimental studies, and to continue to serve as a pragmatic guide to finer delineation of whether aspects of ADHD can be firmly endorsed for the individual and lead to an effective therapeutic regimen. The substantial database can serve to re-focus the clinician on the individual. It is obligatory that the clinician consider many viable alternative explanations that may result in an exhibition of behavior that group-based population statistics might characterize as ADHD, when in fact the very personal circumstances related to the individual may prove to be merely a transient or situational feature that is also amenable to therapy, but of a different sort. Or, an alternative diagnosis or comorbidity is masked by rapid endorsement of a more common feature characteristic of those with ADHD. A system is needed to better evaluate whether an individual meets accepted criteria or whether, instead, personal circumstances or idiosyncracies are responsible. The existing diagnostic criteria may often fail to capture the full spectrum of disorder, and fail to lead to appropriate alternative diagnostic considerations. There are obstacles to diagnosis that must be surmounted or, if unchanged, they will serve to continue the clinical conundrum commonly encountered by clinicians. Toward that end, the rich database that continues to grow regarding ADHD further underscores the weaknesses inherent to our current clinical diagnostic taxonomy and the necessity that we do better in describing and identifying true ADHD.

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