CHAPTER TWO

Attention-Deficit/ Hyperactivity Disorder

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It is commonplace for children (especially preschoolers) to be active, energetic, and exuberant; to flit from one activity to another as they explore their environment and its novelties; and to act without much forethought, responding on impulse to events that occur around them, often with their emotional reactions readily apparent. But when children persistently display levels of activity that are far in excess of their age group; when they are unable to sustain attention, interest, or persistence as well as their peers do to their activities, longer-term goals, or the tasks assigned to them by others; or when their self-regulation lags far behind expectations for their developmental level, they are no longer simply expressing the *joie de vivre* that characterizes childhood. They are instead highly likely to be impaired in their social, cognitive, academic, familial, and eventually occupational domains of major life activities.

Highly active, inattentive, and impulsive youngsters will find themselves far less able than their peers to cope successfully with the universal developmental progressions toward self-regulation, cross-temporal organization, and preparation for their future so evident in our social species. And they will often experience the harsh judgments, punishments, moral denigration, and social ostracism reserved for those society views as lazy, unmotivated, selfish, thoughtless, immature, and willfully irresponsible. These heedless risk-taking children with the devil-may-care attitudes, and self-destructive ways have captured public and scientific interest for more than a century. Diagnostic labels for inattentive, impulsive children have changed numerous times over the last century; yet the actual nature of the disorder has changed little, if at all, from descriptions nearly a century ago (Still, 1902). This constellation of behavior problems may constitute one of the most well-studied childhood disorders of our time. Yet these children remain an enigma to most members of the public, who struggle to accept the notion that the disorder may be a biologically rooted developmental disability when nothing seems physically, outwardly wrong with them.

Children possessing the above-described attributes to a degree that is deviant for their developmental level sufficient to create impairments in major life activities are now diagnosed as having attention-deficit/hyperactivity disorder (ADHD; American Psychiatric Association, 1994). Their problematic behavior is thought to arise early in childhood, and to be persistent over development in most cases. This chapter provides an overview of the nature of this disorder; briefly considers its history; and describes its diagnostic criteria, its developmental course and outcomes, and its causes. Current critical issues related to these matters are raised along the way. Given the thousands of scientific papers on this topic, this chapter must of necessity concentrate on the most important topics in this literature. Readers

interested in more detail can pursue other sources (Accardo, Blondis, Whitman, & Stein, 2001; Barkley, 1998; Weiss & Hechtman, 1993). My own theoretical model of ADHD is also presented, providing a more parsimonious accounting for the many cognitive and social deficits in the disorder; this model points to numerous promising directions for future research, while rendering a deeper appreciation for the developmental significance and seriousness of ADHD. As will become evident, continuing to refer to this disorder as one involving attention deficits understates a more central problem with inhibition, self-regulation, and the cross-temporal organization of social behavior.

HISTORICAL CONTEXT

Literary references to individuals having serious problems with inattention, hyperactivity, and poor impulse control date back to Shakespeare, who made reference to a malady of attention in *King Henry VIII*. A hyperactive child was the focus of a German poem, "Fidgety Phil," by physician Heinrich Hoffman (see Stewart, 1970). William James (1890/1950), in his *Principles of Psychology*, described a normal variant of character that he called the "explosive will," which resembles the difficulties experienced by those who today are described as having ADHD. But, more serious clinical interest in children with ADHD first occurred in three lectures of the English physician George Still (1902) before the Royal Academy of Physicians.

Still reported on a group of 20 children in his clinical practice whom he defined as having a deficit in "volitional inhibition" (p. 1008), which led to a "defect in moral control" (p. 1009) over their own behavior. Described as aggressive, passionate, lawless, inattentive, impulsive, and overactive, many of these children today would be diagnosed as having not only ADHD but also oppositional defiant disorder (ODD) (see Hinshaw & Lee, Chapter 3, this volume). Still's observations were quite astute, describing many of the associated features of ADHD that would come to be corroborated in research over the next century: (1) an overrepresentation of male subjects (ratio of 3:1 in Still's sample); (2) high comorbidity with antisocial conduct and depression; (3) an aggregation of alcoholism, criminal conduct, and depression among the biological relatives; (4) a familial predisposition to the disorder, likely of hereditary origin; and yet (5) the possibility of the disorder's also arising from acquired injury to the nervous system.

Interest in these children arose in North America after the great encephalitis epidemics of 1917–1918. Children surviving these brain infections had many behavioral problems similar to those seen in contemporary ADHD (Ebaugh, 1923; Hohman, 1922; Stryker, 1925). These cases and others known to have arisen from birth trauma, head injury, toxin exposure, and infections (see Barkley, 1998) gave rise to the concept of a "brain-injured child syndrome" (Strauss & Lehtinen, 1947), often associated with mental retardation, that would eventually be applied to children manifesting these same behavior features but without evidence of brain damage or retardation (Dolphin & Cruickshank, 1951; Strauss & Kephardt, 1955). This concept evolved into that of "minimal brain damage" and eventually "minimal brain dysfunction" (MBD), as challenges were raised to the label in view of the dearth of evidence of obvious brain injury in most cases (see Kessler, 1980, for a more detailed history of MBD).

By the late 1950s, focus shifted away from etiology and toward the more specific behavior of hyperactivity and poor impulse control characterizing these children, reflected in labels such as "hyperkinetic impulse disorder" or "hyperactive child syndrome" (Burks, 1960; Chess, 1960). The disorder was thought to arise from cortical overstimulation, due to poor thalamic filtering of stimuli entering the brain (Knobel, Wolman, & Mason, 1959; Laufer, Denhoff, & Solomons, 1957). Despite a continuing belief among clinicians and researchers of this era that the condition had some sort of neurological origin, the larger influence of psychoanalytic thought held sway. And so, when the second edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-II) appeared, all childhood disorders were described as "reactions," and the hyperactive child syndrome became "hyperkinetic reaction of childhood" (American Psychiatric Association, 1968).

The recognition that the disorder was not caused by brain damage seemed to follow a similar argument made somewhat earlier by the prominent child psychiatrist Stella Chess (1960). It set off a major rift between professionals in North America and those in Europe, which

continues (to a lessening extent) to the present. Europe continued to view hyperkinesis for most of the latter half of the 20th century as a relatively rare condition of extreme overactivity, often associated with mental retardation or evidence of organic brain damage. This discrepancy in perspectives has been converging over the last decade, as evident in the similarity of the DSM-IV criteria (see below) with those of the *International Classification of Diseases*, 10th revision (ICD-10; World Health Organization, 1993). Nevertheless, the manner in which clinicians and educators view the disorder remains quite disparate; in North America, Canada, and Australia, such children are diagnosed with ADHD (a developmental disorder), whereas in Europe they are viewed as having a conduct problem or disorder (a behavioral disturbance believed to arise largely out of family dysfunction and social disadvantage).

By the 1970s, research emphasized the problems with sustained attention and impulse control in addition to hyperactivity (Douglas, 1972). Douglas (1980, 1983) theorized that the disorder involved major deficits in (1) the investment, organization, and maintenance of attention and effort; (2) the ability to inhibit impulsive behavior; and (3) the ability to modulate arousal levels to meet situational demands. Together with these deficits went an unusually strong inclination to seek immediate reinforcement. Douglas's emphasis on attention, along with the numerous studies of attention, impulsiveness, and other cognitive sequelae that followed (see Douglas, 1983; and Douglas & Peters, 1978, for reviews), eventually led to renaming the disorder "attention deficit disorder" (ADD) in 1980 (DSM-III; American Psychiatric Association, 1980). Historically significant was the distinction in DSM-III between two types of ADD: ADD with hyperactivity and without it. Little research existed at the time on the latter subtype that would have supported such a distinction being made in an official and increasingly prestigious diagnostic taxonomy. Yet, in hindsight, this bald assertion led to valuable research on the differences between these two supposed forms of ADD, which otherwise would never have taken place. That research may have been fortuitous, as it may be leading to the conclusion that a subset of those having ADD without hyperactivity may actually have a separate, distinct, and qualitatively unique disorder, rather than a subtype of ADHD (Milich, Balentine, & Lynam, 2001).

Even so, concern arose within a few years of the creation of the label ADD that the important features of hyperactivity and impulse control were being deemphasized,when in fact they were critically important to differentiating the disorder from other conditions and to predicting later developmental risks (Barkley, 1998; Weiss & Hechtman, 1993). In 1987, the disorder was renamed "attention-deficit hyperactivity disorder" in DSM-III-R (American Psychiatric Association, 1987), and a single list of items incorporating all three symptoms was specified. Also important here was the placement of the condition of ADD without hyperactivity, renamed "undifferentiated attention-deficit disorder," in a separate section of the manual from ADHD, with the specification that insufficient research existed to guide in the construction of diagnostic criteria for it at that time.

During the 1980s, reports focused instead on problems with motivation generally, and an insensitivity to response consequences specifically (Barkley, 1989a; Glow & Glow, 1979; Haenlein & Caul, 1987). Research was demonstrating that under conditions of continuous reward, the performances of children with ADHD were often indistinguishable from normal children on various lab tasks, but that when reinforcement patterns shifted to partial reward or to extinction (no-reward) conditions, the children with ADHD showed significant declines in their performance (Douglas & Parry, 1983, 1994; Parry & Douglas, 1983). It was also observed that deficits in the control of behavior by rules characterized these children (Barkley, 1989a).

Beginning in the late 1980s, researchers employed information-processing paradigms to study ADHD, and found that problems in perception and information processing were not so evident as were problems with motivation and response inhibition (Barkley, Grodzinsky, & DuPaul, 1992; Schachar & Logan, 1990; Sergeant, 1988; Sergeant & Scholten, 1985a, 1985b). The problems with hyperactivity and impulsivity also were found to form a single dimension of behavior (Achenbach & Edelbrock, 1983; Goyette, Conners, & Ulrich, 1978; Lahey et al., 1988), which others described as "disinhibition" (Barkley, 1990). All of this led to the creation of two separate lists of items and thresholds for ADHD when the DSM-IV was published later in the decade (American Psychiatric Association, 1994): one for inattention and another for hyper-

active–impulsive behavior. Unlike its predecessor, DSM-III-R, DSM-IV thus once again permitted the diagnosis of a subtype of ADHD that consisted principally of problems with attention (ADHD predominantly inattentive type). It also permitted, for the first time, the distinction of a subtype of ADHD that consisted chiefly of hyperactive–impulsive behavior without significant inattention (ADHD, predominantly hyperactive–impulsive type). Children having significant problems from both item lists were described as having ADHD, combined type. The specific criteria from DSM-IV are discussed in more detail below (see "Diagnostic Criteria and Related Issues").

Healthy debate continues to the present over the core deficits in ADHD, with increasing weight being given to problems with behavioral inhibition, self-regulation, and the related domain of executive functioning (Barkley, 1997a, 1997b, 2001c; Douglas, 1999; Nigg, 2001; Quay, 1997). The symptoms of inattention may actually be evidence of impaired working memory and not of perceptual, filtering, or selection (input) problems (Barkley, 1997b). Likewise, controversy continues to swirl around the place of a subtype composed primarily of inattention within the larger condition of ADHD (see *Clinical Psychology: Science and Practice*, 2001, Vol. 8, No. 4, for a debate on this issue): Some argue for its being a distinct disorder from ADHD (Barkley, 2001a; Milich et al., 2001), and others argue that this distinction may be premature (Hinshaw, 2001; Lahey, 2001) or not especially important to treatment planning (Pelham, 2001). Relatively consistent across viewpoints, however, is the opinion that a subset of children with only high levels of inattention probably have a qualitatively different problem in attention (deficient selective attention and sluggish cognitive processing) than is seen in children with ADHD (poor persistence, inhibition, and resistance to distraction).

DESCRIPTION AND DIAGNOSIS

The Core Symptoms

Research employing factor analysis has repeatedly identified two distinct behavioral dimensions underlying the various behavioral problems (symptoms) thought to characterize ADHD (Burns, Boe, Walsh, Sommers-Flanagan, & Teegarden, 2001; DuPaul, Powers, Anastopoulos, & Reid, 1997; Lahey et al., 1994; Pillow, Pelham, Hoza, Molina, & Stultz, 1998). These two dimensions have been identified across various ethnic and cultural groups, including Native American children (Beiser, Dion, & Gotowiec, 2000).

Inattention

Attention represents a multidimensional construct (Bate, Mathias, & Crawford, 2001; Mirsky, 1996; Strauss, Thompson, Adams, Redline, & Burant, 2000), and thus several qualitatively distinct problems with attention may be evident in children (Barkley, 2001a). The dimension impaired in ADHD reflects an inability to sustain attention or persist at tasks or play activities, remember and follow through on rules and instructions, and resist distractions while doing so. I have elsewhere argued that this dimension is more likely to reflect problems with the executive function of working memory than poor attention per se (Barkley, 1997b), and evidence is becoming available to support this contention (Oosterlan, Scheres, & Sergeant, in press; Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999; Wiers, Gunning, & Sergeant, 1998). Parents and teachers frequently complain that these children do not seem to listen as well as they should for their age, cannot concentrate, are easily distracted, fail to finish assignments, are forgetful, and change activities more often than others (DuPaul et al., 1998). Research employing objective measures corroborates these complaints through observations of exhibiting more "off-task" behavior and less work productivity, looking away more often from assigned tasks (including television), showing less persistence at tedious tasks (such as continuous-performance tasks), being slower and less likely to return to an activity once interrupted, being less attentive to changes in the rules governing a task, and being less capable of shifting attention across tasks flexibly (Borger & van der Meere, 2000; Hoza, Pelham, Waschbusch, Kipp, & Owens, 2001; Lorch et al., 2000; Luk, 1985; Newcorn et al., 2001; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Shelton et al., , 1998). This inattentive behavior distinguishes these children from those with learning disabilities (Barkley, DuPaul, & McMurray, 1990) or other psychiatric disorders (Chang et al., 1999; Swaab-Barneveld et al, 2000), and does not appear to be a function of other disorders often comorbid with ADHD (anxiety, depression, or oppositional and conduct problems)

(Murphy, Barkley, & Bush, 2001; Klorman et al., 1999; Newcorn et al., 2001; Nigg, 1999; Seidman, Biederman, Faraone, et al., 1995).

Hyperactive–Impulsive Behavior (Disinhibition)

Like attention, inhibition is a multidimensional construct (Nigg, 2000; Olson, Schilling, & Bates, 1999), and thus various qualitatively distinct forms of inhibitory impairments may eventually be found in children. The problems with inhibition seen in ADHD are thought to involve voluntary or executive inhibition of prepotent responses, rather than impulsiveness that may be more motivationally controlled, as in a heightened sensitivity to available reward (reward seeking) or to excessive fear (Nigg, 2001). Some evidence suggests that an excess sensitivity to reward or to sensation seeking may be more associated with severity of conduct disorder (CD) or psychopathy than with severity of ADHD (Beauchaine, Katkin, Strassberg, & Snarr, 2001; Daugherty & Quay, 1991; Fischer, Barkley, Smallish, & Fletcher, in press-a; Matthys, van Goozen, de Vries, Cohen-Kettenis, & van Engeland, 1998). Evidence is less clear about deficits in automatic or involuntary inhibition, as in eye blinking or negative priming, being associated with ADHD (Nigg, 2001).

More specifically, children with ADHD manifest difficulties with excessive activity level and fidgetiness, less ability to stay seated when required, greater touching of objects, moving about, running, and climbing than other children, playing noisily, talking excessively, acting impulsively, interrupting others' activities, and being less able than others to wait in line or take turns in games (American Psychiatric Association, 1994). Parents and teachers describe them as acting as if driven by a motor, incessantly in motion, always on the go, and unable to wait for events to occur. Research objectively documents them to be more active than other children (Barkley & Cunningham, 1979a; Dane, Schachar, & Tannock, 2000; Luk, 1985; Porrino et al., 1983; Shelton et al., 1998); to have considerable difficulties with stopping an ongoing behavior (Schachar, Tannock, & Logan, 1993; Milich, Hartung, Matrin, & Haigler, 1994; Nigg, 1999, 2001; Oosterlaan, Logan, & Sergeant, 1998); to talk more than others (Barkley, Cunningham, & Karlsson, 1983); to interrupt others' conversations (Malone & Swanson, 1993); to be less able to resist immediate temptations and delay gratification (Anderson, Hinshaw, & Simmel, 1994; Barkley, Edwards, Laneiri, Fletcher, & Metevia, 2001; Olson et al., 1999; Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986; Solanto et al., 2001); and to respond too quickly and too often when they are required to wait and watch for events to happen, as is often seen in impulsive errors on continuous-performance tests (Losier, McGrath, & Klein, 1996; Newcorn et al., 2001). Although less frequently examined, similar differences in activity and impulsiveness have been found between children with ADHD and those with learning disabilities (Barkley, DuPaul, & McMurray, 1990; Bayliss & Roodenrys, 2000; Klorman et al., 1999; Willcutt et al., 2001). Mounting evidence further shows that these inhibitory deficits are not a function of other psychiatric disorders that may overlap with ADHD (Barkley, Edwards, et al., 2001; Halperin, Matier Bedi, Sharpin, & Newcorn, 1992; Fischer et al., in press-a; Murphy et al., 2001; Nigg, 1999; Oosterlaan et al., 1998; Seidman Biederman, Faraone, et al., 1997).

Interestingly, recent research shows that the problems with inhibition arise first (at ages 3–4 years), ahead of those related to inattention (at ages 5–7 years), and that the sluggish cognitive tempo that characterizes the predominantly inattentive subtype of ADHD may arise even later (ages 8–10) (Hart, Lahey, Loeber, Applegate, & Frick, 1995; Loeber, Green, Lahey, Christ, & Frick, 1992; Milich et al., 2001). Whereas the symptoms of disinhibition in the DSM item lists seem to decline with age, perhaps owing to their heavier weighting with hyperactive than with impulsive behavior, those of inattention remain relatively stable during the elementary grades (Hart et al., 1995). They eventually decline by adolescence (Fischer, Barkley, Fletcher, & Smallish, 1993a), though not to normal levels. Why the inattention arises later than the disinhibitory symptoms and does not decline when the latter do over development remains an enigma. As noted above, it may simply reflect the different weightings of symptoms in the DSM. Those of hyperactivity may be more typical of preschool to early school-age children and are overrepresented in the DSM list, while those reflecting inattention may be more characteristic of school-age children. Another explanation comes from the theoretical model described below (Barkley, 1997b), in which inhibition and the two types of working memory (nonverbal and verbal) emerge at separate times in development.

Situational and Contextual Factors

The symptoms constituting ADHD are greatly affected in their level of severity by a variety of situational and task-related factors. Douglas (1972) commented on the greater variability of task performances by children with ADHD compared to control children. Many others since then have found that when a child with ADHD must perform multiple trials within a task assessing attention and impulse control, the range of scores around that child's own mean performance is frequently greater than in normal children (see Douglas, 1983). The finding is especially common in measures of reaction time (Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989; Fischer et al., in press-a; Kuntsi, Oosterlaan, & Stevenson, 2001; Murphy et al., 2001; Scheres, Oosterlaan, & Sergeant, 2001).

A number of other factors influence the ability of children with ADHD to sustain their attention to task performance, control their impulses to act, regulate their activity level, and/or produce work consistently. The performance of these children is worse (1) later in the day than earlier (Dane et al., 2000; Porrino et al., 1983; Zagar & Bowers, 1983); (2) in greater task complexity, such that organizational strategies are required (Douglas, 1983); (3) when restraint is demanded (Barkley & Ullman, 1975; Luk, 1985); (4) under low levels of stimulation (Antrop, Roeyers, Van Oost, & Buysse, 2000; Zentall, 1985); (5) under more variable schedules of immediate consequences in the task (Carlson & Tamm, 2000; Douglas & Parry, 1983, 1994; Slusarek, Velling, Bunk, & Eggers, 2001; Tripp & Alsop, 1999); (6) under longer delay periods prior to reinforcement availability (Solanto et al., 2001; Sonuga-Barke, Taylor, & Heptinstall, 1992; Tripp & Alsop, 2001); and (7) in the absence of adult supervision during task performance (Draeger, Prior, & Sanson, 1986; Gomez & Sanson, 1994).

Besides the aforementioned factors, which chiefly apply to task performance, variability has also been documented across more macroscopic settings. For instance, children with ADHD exhibit more problematic behavior when persistence in work-related tasks is required (chores, homework, etc.) or where behavioral restraint is necessary, especially in settings involving public scrutiny (in church, in restaurants, when a parent is on the phone, etc.), than in free-play situations (Altepeter & Breen, 1992; Barkley & Edelbrock, 1987; DuPaul & Barkley, 1992). Although they will be more disruptive when their fathers are at home than during free play, children with ADHD are still rated as much less problematic when their fathers are at home than in most other contexts. Fluctuations in the severity of ADHD symptoms have also been documented across a variety of school contexts (Barkley & Edelbrock, 1987; DuPaul & Barkley, 1992). In this case, contexts involving task-directed persistence and behavioral restraint (classroom) are the most problematic, with significantly fewer problems posed by contexts involving less work and behavioral restraint (at lunch, in hallways, at recess, etc.), and even fewer problems being posed during special events (field trips, assemblies, etc.) (Altepeter & Breen, 1992).

Associated Developmental Impairments

Children with ADHD often demonstrate deficiencies in many other cognitive and emotional abilities. Among these are difficulties with (1) physical fitness, gross and fine motor coordination, and motor sequencing (Breen, 1989; Denckla & Rudel, 1978; Harvey & Reid, 1997; Kadesjo & Gillberg, 1999; Mariani & Barkley, 1997); (2) speed of color naming (Tannock, Martinussen, & Frijters, 2000); (3) verbal and nonverbal working memory and mental computation (Barkley, 1997a; Mariani & Barkley, 1997; Murphy et al., 2001; Zentall & Smith, 1993); (4) story recall (Lorch et al., 2000; Sanchez, Lorch, Milich, & Welsh, 1999); (5) planning and anticipation (Grodzinsky & Diamond, 1992; Klorman et al., 1999); (6) verbal fluency and confrontational communication (Grodzinsky & Diamond, 1992; Zentall, 1988); (5) effort allocation (Douglas, 1983; Nigg, Hinshaw, Carte, & Treuting, 1998; Sergeant & van der Meere, 1994; Voelker, Carter, Sprague, Gdowski, & Lachar, 1989); (6) developing, applying, and self-monitoring organizational strategies (Clark, Prior, & Kinsella, 2000; Hamlett, Pellegrini, & Connors, 1987; Purvis & Tannock, 1997; Zentall, 1988); (7) internalization of self-directed speech (Berk & Potts, 1991; Copeland, 1979; Winsler, 1998; Winsler, Diaz, Atencio, McCarthy, & Chabay, 2000); (8) adhering to restrictive instructions (Danforth, Barkley, & Stokes, 1991; Roberts, 1990; Routh & Schroeder, 1976); and (9) self-regulation of emotion (Braaten & Rosen, 2000; Hinshaw, Buhrmeister, & Heller, 1989; Maedgen & Carlson, 2000). The last-mentioned difficulties, those with emotional control, may be especially salient in children having ADHD with comorbid

ODD (Melnick & Hinshaw, 2000). Several studies have also demonstrated that ADHD may be associated with less mature or diminished moral development (Hinshaw, Herbsman, Melnick, Nigg, & Simmel, 1993; Nucci & Herman, 1982; Simmel & Hinshaw, 1993). Many of these cognitive difficulties appear to be specific to ADHD and are not a function of its commonly comorbid disorders, such as learning disabilities, depression, anxiety, or ODD/CD (Barkley, Edwards, et al., 2001; Clark et al., 2000; Klorman et al., 1999; Murphy et al., 2001; Nigg, 1999; Nigg et al., 1998).

The commonality among most or all of these seemingly disparate abilities is that all have been considered to fall within the domain of "executive functions" in the field of neuropsychology (Barkley, 1997b; Denckla, 1994) or "metacognition" in developmental psychology (Flavell, 1970; Torgesen, 1994; Welsh & Pennington, 1988), or to be affected by these functions. All seem to be mediated by the frontal cortex, particularly the prefrontal lobes (Fuster, 1997; Stuss & Benson, 1986). "Executive functions" have been defined as those neuropsychological processes that permit or assist with human "self-regulation" (Barkley, 1997b, 2001a, 2001b), which itself has been defined as any behavior by a person that modifies the probability of a subsequent behavior by that person so as to alter the probability of a later consequence (Kanfer & Karoly, 1972). By classifying cognitive actions or thinking as private behavior, one can understand how these private, self-directed, cognitive (executive) actions fall within the definition of human self-regulation: They are private behaviors (cognitive acts) that modify other behaviors so as to alter the likelihood of later consequences for the individual. And when the role of the frontal lobes generally, and the prefrontal cortex particularly, in these executive abilities is appreciated, it is easy to see why researchers have repeatedly speculated that ADHD probably arises out of some disturbance or dysfunction of this brain region (Barkley, 1997b; Heilman, Voeller, & Nadeau, 1991; Levin, 1938; Mattes, 1980).

THEORETICAL FRAMEWORK

Many different theories of ADHD have been proposed over the past century to account for the diversity of findings so evident in this disorder (Barkley, 1999b). Some of these have been discussed above (see "Historical Context"), such as Still's (1902) notion of defective volitional inhibition and moral regulation of behavior; Douglas's (1972, 1983) theory of deficient attention, inhibition, arousal, and preference for immediate reward; and the attempts to view ADHD as a deficit in sensitivity to reinforcement (Haenlein & Caul, 1987) or rule-governed behavior (Barkley, 1981, 1989a). More recently, Quay (1997), relying on Gray's (1982) neuropsychological model of anxiety, has proposed that ADHD represents a deficit in the brain's behavioral inhibition system. Quay's hypothesis has resulted in increased research on inhibitory and activation (reinforcement) processes in both ADHD (Fischer et al., in press-a; Milich et al., 1994) and CD (see Hinshaw & Lee, Chapter 3, this volume). Relying on Logan's "race" model of inhibition, Schachar et al. (1993) have also argued for a central deficit in inhibitory processes in those with ADHD. In this model, an event or stimulus is hypothesized to trigger both an activating or primary response and an inhibitory response, creating a competition or race between the two as to which will be executed first. Disinhibited individuals, such as those with ADHD, are viewed as having slower initiation of inhibitory processes than normal children do.

There is little doubt that poor behavioral inhibition plays a central role in ADHD (see Barkley, 1997b, 1999a, and Nigg, 2001, for reviews). Although important in the progress of our understanding about ADHD, this conclusion still leaves at least two important questions on the nature of ADHD unresolved. First, how does this account for the numerous other associated symptoms found in ADHD (described above) and apparently subsumed under the concepts of motor control and executive functioning? Second, how does this account for the involvement of the separate problem with inattention (poor sustained attention) in the disorder? The theoretical model of ADHD I have developed over the past decade not only encompasses many of these earlier explanations, but may hold the answers to these questions as well as some unexpected directions that future research on ADHD might wish to pursue (Barkley, 1994, 1997a, 1997b, 2001b).

Inhibition, Executive Functions, and Time

The model of ADHD set forth below and in Figure 2.1 places behavioral inhibition at a central point in its relation to four other executive func-

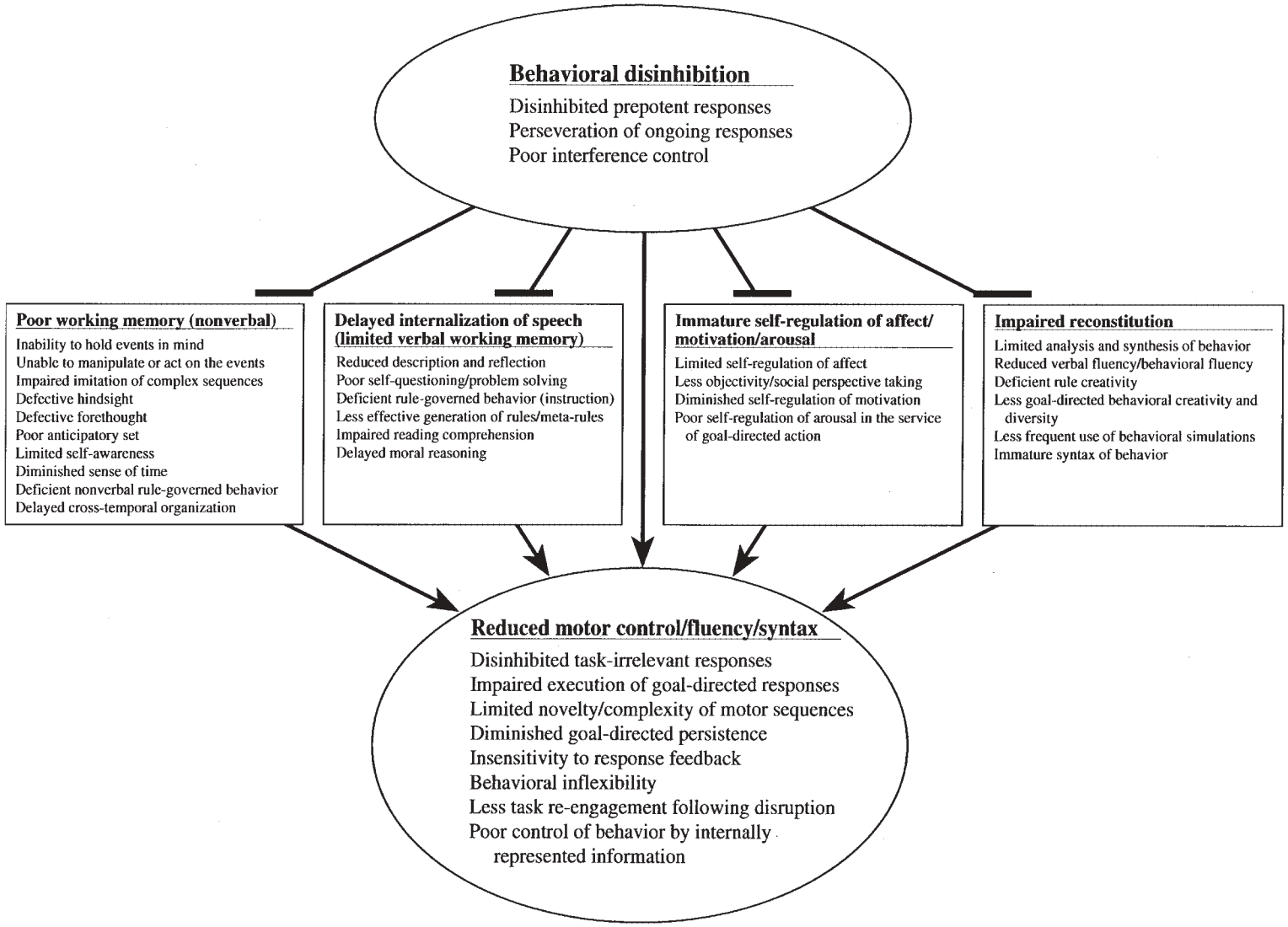

FIGURE 2.1. Diagram illustrating the complete hybrid model of executive functions (boxes) and the relationship of these four functions to the behavioral inhibition and motor control systems. From Barkley (1997b). Copyright 1997 by The Guilford Press. Reprinted by permission.

tions dependent upon it for their own effective execution. These four executive functions provide for self-regulation, bringing behavior progressively more under the control of time and the influence of future over immediate consequences. The interaction of these executive functions permits far more effective adaptive functioning toward the social future (social self-sufficiency).

Several assumptions are important in understanding the model as it is applied to ADHD: (1) The capacity for behavioral inhibition begins to emerge first in development, ahead of most or all these four executive functions but possibly in conjunction with the first, nonverbal working memory. (2) These executive functions emerge at different times in development, may have different developmental trajectories, and are interactive. (3) The impairment that ADHD creates in these executive functions is secondary to the primary deficit it creates in behavioral inhibition (improve the inhibition, and these executive functions should likewise improve). (4) The deficit in behavioral inhibition arises principally from genetic and neurodevelopmental origins rather than purely social ones, although its expression is certainly influenced by social factors over development. (5) The secondary deficits in self-regulation created by the primary deficiency in inhibition feed back to contribute further to poor behavioral inhibition, given that self-regulation contributes to the enhancement of self-restraint (inhibition). Finally, (6) the model does not apply to those having what is presently called the predominantly inattentive type of ADHD. The model has been derived from earlier theories on the evolution of human language (Bronowski, 1977), the internalization of speech (Vygotsky, 1966/1987), and the functions of the prefrontal cortex (Fuster, 1997). The evidence for the model as applied to ADHD is reviewed in detail elsewhere (Barkley, 1997b).

"Behavioral inhibition" is viewed as consisting of two related processes: (1) the capacity to inhibit prepotent responses, either prior to or once initiated, creating a delay in the response to an event (response inhibition); and (2) the protection of this delay, the self-directed actions occurring within it, and the goal-directed behaviors they create from interference by competing events and their prepotent responses (interference control). "Prepotent responses" are defined as those for which immediate reinforcement is available for their performance or for which there is a strong history of reinforcement in this context. Through the postponement of the prepotent response and the creation of this protected period of delay, the occasion is set for four other executive functions to act effectively in modifying the individual's eventual response(s) to the event. This is done to achieve a net maximization of temporally distant consequences rather than immediate consequences alone for the individual. The self-regulation is also protected from interference during its performance by a related form of inhibition (interference control).

The four executive functions are believed to develop via a common process. All represent private, covert forms of behavior that at one time in early child development (and in human evolution) were entirely publicly observable and were directed toward others and the external world at large. With maturation, this outer-directed behavior becomes turned on the self as a means to control one's own behavior. Such self-directed behaving then becomes increasingly less observable to others as the suppression of the public, peripheral, musculo-skeletal aspects of the behavior progresses. The child is increasingly able to act toward the self without publicly displaying the actual behavior being activated. This progressively greater capacity to suppress the publicly observable aspects of behavior is what is meant here by the terms "covert," "privatized," or "internalized." The child comes to be capable of behaving internally (in the brain) without showing that response through the peripheral muscles, at least not to the extent that it is visible to others. As I have discussed elsewhere (Barkley, 1997b, 2001c), this behavior-to-the-self can still be detected in very subtle, vestigial forms as slight shifts in muscle potential at those peripheral sites involving the muscles used in performing the public form of that behavior (e.g., when one engages in verbal thought, one still slightly moves the lips, tongue, larynx, etc.). In this sense, all of the executive functions follow the same general sequence as the internalization of speech (Diaz & Berk, 1992; Vygotsky, 1966/1987, 1978), which in this model forms the second executive function.

Each executive function is hypothesized to contribute to the following developmental shifts in the sources of control over human behavior:

- From external events to mental representations related to those events.
- From control by others to control by the self.
- From immediate reinforcement to delayed gratification.

- From the temporal now to the conjectured social future.

I have elsewhere asserted that the executive functions probably evolved in successive stages in our hominid ancestry from intraspecies competition for resources and reproduction in our group living speces. The sequence may resemble, to some extent, the same sequential development evident in children today. The first executive function (nonverbal working memory, which involves sensory–motor action to the self, especially visual imagery) begins its development so early in infancy that it must have been crucial to human survival. It may have evolved for the adaptive purposes of reciprocal altruism (social exchange) and generalized vicarious learning. These activities seem to be essential for the survival of our group-living species, contributing to cooperation, coalition formation (friendships), the construction of social hierarchies from these coalitions, and pedagogy (Barkley, 2001c). Vicarious learning can be considered a form of behavioral theft that, once having arisen in a species, would have set up strong selection pressure for the privatization of one's behavior—particularly during learning, rehearsal, and other forms of practice—so as not to have one's behavioral innovations readily appropriated by others (competitors). Other adaptive purposes that may have been served by this and the other three executive functions (which develop later) are verbal self-instruction, verbal self-defense against social manipulation by others, and self-innovation. Such evolutionary speculations permit this theory to hypothesize various social deficits that should be evident in ADHD, given the executive deficits associated with it, that can be tested in subsequent experiments. As is evident below, children with ADHD experience serious difficulties in their social relationships, some of which may arise from the deficits in executive functioning that interfere with reciprocal exchange, vicarious learning, social coalition formation, social self-defense, and self-innovation (improvement).

Nonverbal Working Memory (Sensory–Motor Action to the Self)

During the delay in responding created by inhibition, humans activate and retain a mental representation of events in mind (Bronowski, 1977), typically using visual imagery and private audition. The capacity for imagery may allow even infants to successfully perform delayed-response tasks to a limited degree (Diamond, 1990; Diamond, Cruttenden, & Niederman, 1994; Goldman-Rakic, 1987). As this capacity increases developmentally, it forms the basis for "nonverbal working memory," which has been defined as the ability to maintain mental information online so as to guide a later motor response. This activation of past images for the sake of preparing a current response is known as "hindsight" or the "retrospective function" of working memory (Bronowski, 1977; Fuster, 1997). It allows for the retention of events in a temporal sequence that contributes to the "subjective estimation of time" (Michon, 1985). Such temporal sequences can be analyzed for recurring patterns, and those patterns can then be used to conjecture hypothetical future events. Anticipating these hypothetical futures gives rise to a preparation to act, or "anticipatory set" (Fuster, 1997). This extension of hindsight forward into time also underlies "forethought" or the "prospective function" of working memory (Bronowski, 1977; Fuster, 1997). And from this sense of future probably emerges the progressively greater valuation of future consequences over immediate ones, which takes place throughout child development and early adult life (Green, Fry, & Meyerson, 1994).

Important in this model for understanding the linkage of inattention to disinhibition in ADHD is the critical role played by working memory in maintaining online (in mind) one's intentions to act ("plans"), so as to guide the construction and execution of complex goal-directed actions over time (Fuster, 1997). Such sustained chains of goal-directed actions create persistence of responding, giving rise to the capacity of humans to sustain attention (responding) for dramatically long periods of time in pursuit of future goals. As James (1890/1950) so eloquently described it: "The essential achievement of the will, in short, when it is most 'voluntary,' is to ATTEND to a difficult object and hold it fast before the mind" (p. 815); and "Everywhere then the function of the effort [voluntary or free will] is the same: to keep affirming and adopting a thought which, if left to itself, would slip away" (p. 818). Thus self-regulation relative to time arises as a consequence of inhibition acting in conjunction with nonverbal working memory. And since language is used in part to express cognitive content, references to time, sense of past, and sense of future can occur in verbal interactions with others; such references should become increasingly frequent in the de-

velopmental course of children as this sense of time develops.

As extrapolated to those with ADHD, the model predicts that deficits in behavioral inhibition lead to deficiencies in nonverbal working memory, and thus (1) particular forms of forgetfulness (forgetting to do things at certain critical points in time); (2) impaired ability to organize and execute actions relative to time (e.g., time management); and (3) reduced hindsight and forethought, leading to (4) a reduction in the creation of anticipatory action toward future events. Consequently, the capacity for the cross-temporal organization of behavior in those with ADHD is diminished, disrupting the ability to string together complex chains of actions directed, over time, to a future goal. The greater the degree to which time separates the components of the behavioral contingency (event, response, consequence), the more difficult the task will prove for those with ADHD, who cannot bind the contingency together across time so as to use it to govern their behavior as well as others.

Research is beginning to demonstrate some of these deficits in those with ADHD, such as nonverbal working memory, timing, and forethought (Barkley, 1997b; Barkley, Edwards, et al., 2001; Barkley, Murphy, & Bush, 2001; Murphy et al., 2001). Still unstudied is the prediction from this theory that children with ADHD will be delayed in making references to time, past, and future in their verbal interactions with others, relative to when normal children begin making such references in their development of sense of time.

Verbal Working Memory (Internalization of Speech)

One of the more fascinating developmental processes witnessed in children is the progressive internalization or privatization of speech (Diaz & Berk, 1992). During the early preschool years, speech, once developed, is initially employed for communication with others. By 3–5 years of age, language comes to be turned on the self. Such overt self-speech is readily observable in preschool and early school-age children. By 5–7 years of age, this speech becomes somewhat quieter and more telegraphic, and shifts from being more descriptive to being more instructive. Language is now a means of reflection (self-directed description), as well as a means for controlling one's own behavior. Self-directed speech progresses from being public to being subvocal to finally being private, all over the course of perhaps 6 to 10 years, thereby giving rise to verbal thought (Diaz & Berk, 1992; Kopp, 1982; Vygotsky, 1966/1987). I have conjectured (Barkley, 1997b) that this internalization of speech represents a larger process, in that various other forms of behavior may be internalized as well (sensory–motor action, emotion, and play).

For those with ADHD, the privatization of speech should be delayed, resulting in greater public speech (excessive talking), less verbal reflection before acting, less organized and rule-oriented self-speech, a diminished influence of self-directed speech in controlling one's own behavior, and difficulties following the rules and instructions given by others (Barkley, 1997b). Substantial evidence has accumulated to support this prediction of delayed internalization of speech (Berk & Potts, 1991; Landau, Berk, & Mangione, 1996; Winsler, 1998; Winsler et al., 2000). Given that such private self-speech is a major basis for verbal working memory, this domain of cognitive activity should be impaired in ADHD as well. Evidence suggests that this is so: Children with ADHD have difficulties with tasks such as backward digit span, mental arithmetic, paced auditory serial addition, paired-associate learning, and other tasks believed to reflect verbal working memory (Barkley, 1997b; Chang et al., 1999; Grodzinsky & Diamond, 1992; Kuntsi et al., 2001). Children with learning disabilities may also have difficulties with some of these tasks, making it unclear to what extent the deficits seen in working memory in ADHD are a function of the overlap of learning disabilities with this disorder (Cohen et al., 2000; Willcutt et al., 2001). ADHD may impair the actual internalization of speech, whereas reading disorders may reflect a normal internalization but of an impaired language ability.

Internalization and Self-Regulation of Affect

The inhibition of the initial prepotent response includes the inhibition of the initial emotional reaction that it may have elicited. It is not that the child does not experience emotion, but that the behavioral reaction to or expression of that emotion is delayed, along with any motor behavior associated with it. The delay in responding with this emotion allows the child time to engage in self-directed behavior that will modify both the eventual response to the event and the emotional

reaction that may accompany it. This permits a moderating effect on the emotion being experienced subjectively by the child, as well as on the child's eventual public expression of emotional behavior (Keenan, 2000). But it is not just affect that is being managed by the development of self-regulation, but the underlying components of emotion as well, these being motivation (drive states) and arousal (Fuster, 1997; Lang, 1995). This internalization and self-regulation of motivation permit the child to induce drive states that may be required for the initiation and maintenance of goal-directed, future-oriented behavior, thereby permitting greater persistence toward tasks and activities that may offer little immediate reinforcement but for which there may be substantial delayed reinforcement.

Extending this model to ADHD leads to the following predictions. Those with ADHD should display (1) greater emotional expression in their reactions to events; (2) less objectivity in the selection of a response to an event; (3) diminished social perspective taking, as these children do not delay their initial emotional reaction long enough to take the view of others and their own needs into account; and (4) diminished ability to induce drive and motivational states in themselves in the service of goal-directed behavior. Those with ADHD remain more dependent upon the environmental contingencies within a situation or task to determine their motivation than do others (Barkley, 1997b). Preliminary work has begun to demonstrate that those with ADHD do have significant problems with emotion regulation (Braaten & Rosen, 2000; Maedgen & Carlson, 2000; Southam-Gerow & Kendall, 2002) and that this may be particularly so in that subset having comorbid oppositional defiant disorder (Melnick & Hinshaw, 2000).

Reconstitution (Internalization of Play)

The use of private visual imagery as well as private language to mentally represent objects, actions, and their properties provides a means by which the world can be taken apart and recombined cognitively rather than physically. The delay in responding allows time for an event to be held in mind and then disassembled, so as to extract more information about the event before preparing a response to it. Internal imagery and speech permit analysis, and out of this process comes its complement—synthesis. Just as the parts of speech can be recombined to form new sentences, the parts of the world represented in speech and imagery are likewise recombined to create entirely new ideas about the world and entirely new responses to that world (Bronowski, 1977). The world is seen as having parts rather than inviolate wholes—parts capable of multiple, novel recombinations. This permits humans a far greater capacity for creativity and problem solving than is evident in our closest primate relatives. I believe that this process results from the internalization of play. Just as speech goes from being overt to self-directed and then covert, so does manipulative and verbal play. This process of mental play, or reconstitution, is evident in everyday speech in its fluency and generativity (diversity); yet it is also evident in nonverbal expression as well, such as in motor and design fluency. The need for reconstitution becomes obvious when obstacles must be surmounted to accomplish a goal. In a sense, reconstitution provides for planning and problem solving to overcome obstacles and attain goals. This mental module produces rapid, efficient, and often novel combinations of speech or action into entirely new messages or behavioral sequences, and so gives rise to behavioral innovation.

As applied to ADHD, the model predicts a diminished use of analysis and synthesis in the formation of both verbal and nonverbal responses to events. The capacity to mentally visualize, manipulate, and then generate multiple plans of action (options) in the service of goal-directed behavior, and to select from among them those with the greatest likelihood of succeeding, should therefore be reduced. This impairment in reconstitution will be evident in everyday verbal fluency when a person with ADHD is required by a task or situation to assemble rapidly, accurately, and efficiently the parts of speech into messages (sentences), so as to accomplish the goal or requirements of the task. It will also be evident in tasks where visual information must be held in mind and manipulated to generate diverse scenarios to help solve problems (Barkley, 1997b). Evidence for a deficiency in verbal and nonverbal fluency, planning, problem solving, and strategy development more generally in children with ADHD is limited, but what exists is consistent with the theory (Barkley, 1997b; Clark et al., 2000; Klorman et al., 1999; Nigg et al., 1998; Oosterlaan et al., in press).

Motor Control/Fluency

If the deficit in behavioral inhibition proposed in the current model is housed within the brain's motor or output system, then its effects should

also be evident in the planning and execution of motor actions. Complex fine and gross motor actions require inhibition to preclude the initiation of movements located in neural zones adjacent to those being activated. Inhibition provides an increasing "functional pruning" of the motor system such that only those actions required to accomplish the task are initiated by the individual. Lengthy, complex, and novel chains of goal-directed behavior can be constructed and protected from interference until they have been completed. The model stipulates that those with ADHD should display greater difficulties with the development of motor coordination, and especially in the planning and execution of complex, lengthy, and novel chains of goal-directed responses. There is substantial evidence already available for problems in motor development and motor execution in those with ADHD (see Barkley, 1997b; Harvey & Reid, 1997; Kadesjo & Gillberg, 2001). It remains to be determined whether those with ADHD have more difficulties in producing, executing, and sustaining lengthy and complex chains of novel responses toward goals.

Conclusion

I have recently theorized that this executive system may have evolved to support the social activities of reciprocal exchange and altruism, imitation and vicarious learning, self-sufficiency and innovation, and social self-defense (Barkley, 2001b). This theory implies that these larger, universally important domains of social development may be impaired by ADHD as well. If so, then deficits in adaptive functioning (self-sufficiency) more generally would be evident in ADHD, as seems to be the case (Barkley, Shelton, et al., 2002; Roizen, Blondis, Irwin, & Stein, 1994; Shelton et al., 1998; Stein, Szumowski, Blondis, & Roizen, 1995).

The present model of ADHD shows how the findings noted above under "Associated Developmental Impairments" can now be integrated into a more unifying theory of the disorder. Undoubtedly, this theory is imperfect. A great deal of research will be required to clarify the nature of each component in the model; to evaluate the strength of the relationship of each component to behavioral inhibition and to the other components; to elucidate the developmental progression of each component and their ordering; and to critically test some of the previously unexpected predictions of the model as applied to ADHD (e.g., diminished time management, reduced references to time in verbal interactions, the impact of ADHD on analysis/synthesis and self-innovation, etc.). All useful theories are imperfect and time-limited. What we ask of them is not perfection from birth, but the more pragmatic standard of greater utility than previously existing models or theories. Competing theories of ADHD have limited themselves to elucidating the nature of the inhibitory deficit (Quay, 1997; Sonuga-Barke, Lamparelli, Stevenson, Thompson, & Henry, 1994) while ignoring the associated cognitive, emotional, and social deficiencies associated with it and explaining why they exist. The present theory offers more utility, in that it addresses the origins of those associated problems, is more testable and hence falsifiable, provides a better link to normal child development, and yields a greater understanding of the basis for managing the disorder than do other extant models. Regardless of what theory may replace it in the future, that theory will likewise have to deal with the evidence that points to problems with inhibition and these four executive functions.

This appreciation of the linkage among the executive functions in the model, the self-regulation they permit, and the goal-directed persistence that derives from self-control explain several important findings about the link between disinhibition (hyperactive–impulsive behavior) and inattention. It is possible to see now why the problems with hyperactive–impulsive behavior arise first in the development of ADHD, to be followed within a few years by the problems with inattention. And it also explains the nature of that inattention as it arises. The inattention reflects a deficit in executive functioning, especially working memory, and so is really a form of intention deficit (attention to the future).

DIAGNOSTIC CRITERIA AND RELATED ISSUES

DSM-IV Criteria

The most recent diagnostic criteria for ADHD as defined in DSM-IV (American Psychiatric Association, 1994) are set forth in Table 2.1. These diagnostic criteria are some of the most rigorous and most empirically derived criteria ever available in the history of clinical diagnosis for this disorder. They were derived from a committee of some of the leading experts in the field, a literature review of ADHD, an informal survey of

TABLE 2.1. DSM-IV Criteria for Attention-Deficit/Hyperactivity Disorder (ADHD)

A. Either (1) or (2):

(1) six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

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

(2) six (or more) of the following symptoms of hyperactivity–impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity
(a) often fidgets with hands or feet or squirms in seat
(b) often leaves seat in classroom or in other situations in which remaining seated is expected
(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
(d) often has difficulty playing or engaging in leisure activities quietly
(e) is often "on the go" or often acts as if "driven by a motor"
(f) often talks excessively

Impulsivity
(g) often blurts out answers before the questions have been completed
(h) often has difficulty awaiting turn
(i) often interrupts or intrudes on others (e.g., butts into conversations or games)

B. Some hyperactive–impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

Code based on type:
314.01 Attention-Deficit/Hyperactivity Disorder, Combined Type: if both Criteria A1 and A2 are met for the past 6 months
314.00 Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive Type: if Criterion A1 is met but Criterion A2 is not met for the past 6 months
314.01 Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive–Impulsive Type: if Criterion A2 is met but Criterion A1 is not met for the past 6 months

Coding note: For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "In Partial Remission" should be specified.

Note. From American Psychiatric Association (1994, pp. 83–85). Copyright 1994 by the American Psychiatric Association. Reprinted by permission.

empirically derived rating scales assessing the behavioral dimensions related to ADHD by the committee, and from statistical analyses of the results of a field trial of the items using 380 children from 10 different sites in North America (Lahey et al., 1994).

Despite its empirical basis, the DSM criteria have some problems. As noted earlier, evidence is mounting that the predominantly inattentive type of ADHD (hereafter abbreviated as ADHD-PI) may be a diagnosis applied to a rather heterogeneous mix of children, a subset of whom have a qualitatively different disorder of attention and cognitive processing (Milich et al., 2001). This subset is probably not a subtype of ADHD, but may represent a separate disorder (Barkley, 1998, 2001a; Milich et al., 2001)—one manifesting a sluggish cognitive style and selective attention deficit; having less comorbidity with ODD and CD; demonstrating a more passive style of social relationship; involving memory retrieval problems; and, owing to the lower level of impulsiveness, probably having a different, more benign developmental course. Other children consigned to this subtype may be children who formerly met the criteria for ADHD, combined type (hereafter abbreviated as ADHD-C), but with age have had a sufficient decline in their hyperactive symptoms that they no longer qualify for this subtype. For example, in our follow-up study of hyperactive children, all of whom probably had ADHD-C in childhood, we found that 16% of these cases (or 27% of persistent cases) now met criteria only for ADHD-PI as young adults (Barkley, Fischer, Fletcher, & Smallish, 2002). Such individuals might better be thought of as having residual ADHD-C than as having ADHD-PI. Likewise, some children diagnosed with ADHD-PI place just a single symptom or two short of ADHD-C status yet resemble children with ADHD-C, albeit in milder form, in all other respects. Mixing these children formerly diagnosed with ADHD-C and ones currently diagnosed with subthreshold ADHD-C together into the ADHD-PI group is likely to constrain research on the distinctive features of this subtype, its etiology, its response to treatments, and its developmental course. In agreement with Milich et al. (2001), I believe that the subset of children with hypoactivity, lethargy, and sluggish cognitive tempo should be set aside as having a separate disorder from ADHD (Barkley, 2001a).

It is also unclear whether ADHD, predominantly hyperactive–impulsive type (hereafter abbreviated as ADHD-PHI) is really a separate type from ADHD-C or simply an earlier developmental stage of it. The DSM-IV field trial found that those diagnosed with ADHD-PHI were primarily preschool-age children, whereas those with ADHD-C were primarily school-age children. As noted above, this is what one would expect to find, given that the hyperactive–impulsive symptoms appear first and are followed within a few years by those of inattention. If one is going to require that inattention symptoms be part of the diagnostic criteria, then the age of onset for such symptoms will necessitate that ADHD-C have a later age of onset than ADHD-PHI. It seems that these two types may actually be developmental stages of the same type of ADHD.

Are the two separate symptom lists in DSM-IV important, rather than the one combined list used in DSM-III-R? Apparently. In the field trial (Lahey et al., 1994), significant levels of inattention mainly predicted additional problems with completing homework that were not as well predicted by the hyperactive–impulsive behavior. Otherwise, the latter predicted most of the other areas of impairment studied in this field trial. Other studies find that childhood symptoms of hyperactivity are related to adverse adolescent outcomes, such as antisocial behavior, substance abuse, and school disciplinary actions, such as suspensions/expulsions (Babinski, Hartsough, & Lambert, 1999). Symptoms of inattention seem to be primarily predictive of impairment in academic achievement (particularly reading) and school performance (DuPaul, Power, et al., 1998; Fischer, Barkley, Fletcher, & Smallish, 1993b; Weiss & Hechtman, 1993; Rabiner, Coie, & the Conduct Problem Prevention Research Group, 2000). Severity of hyperactive–impulsive behavior is often found to be the dimension of ADHD that more strongly predicts later CD, and so risk for various forms of substance use and abuse (Molina, Smith, & Pelham, 1999). A recent study suggests that adolescent inattention, however, may contribute further to the risk for tobacco use beyond that risk contributed by severity of CD alone (Burke, Loeber, & Lahey, 2001).

Another critical issue deserving consideration is how well the diagnostic thresholds set for the two symptom lists apply to age groups outside of those used in the field trial (ages 4–16 years, chiefly). This concern arises out of the well-known findings that the behavioral items in these lists, particularly those for hyperactivity, decline significantly with age (DuPaul, Power, et al., 1998;

Hart et al., 1995). Applying the same threshold across such a declining developmental slope could produce a situation where a larger percentage of young preschool-age children (ages 2–3 years) would be inappropriately diagnosed as having ADHD (false positives), whereas a smaller than expected percentage of adults would meet the criteria (false negatives). Support of just such a problem with using these criteria for adults was found in a study (Murphy & Barkley, 1996b) collecting norms for DSM-IV item lists on a large sample of adults, ages 17–84 years. The threshold needed to place an individual at the 93rd percentile for that person's age group declined to four of nine inattention items and five of nine hyperactive–impulsive items for ages 17–29 years, then to four of nine on each list for the 30- to 49-year age group, then to three of nine on each list for those 50 years and older. Studies of the utility of the diagnostic thresholds to preschool children younger than 4 years remain to be done. Until then, it seems prudent to utilize the recommended symptom list thresholds only for children ages 4–16 years.

The issue of selecting symptom cutoff scores raises a related conceptual problem for ADHD as well. Is ADHD a static psychopathology, the symptoms of which remain essentially the same regardless of age? Or is it a developmental disorder (delay in rate)? In the latter case, it must always be determined by comparison to same-age peers. Although the DSM criteria imply that ADHD is a developmental disorder (symptoms must be developmentally inappropriate), it also treats the disorder as a relatively static category by using fixed symptom cutoff scores across all age groups. Available research indicates that ADHD is most likely a dimensional disorder (Levy & Hay, 2001), representing an extreme of or delay in normal traits, and so is akin to other developmental disorders (e.g., mental retardation). If so, then, like all developmental disorders, ADHD reflects a delay in the *rate* at which a normal trait is developing—not an absolute loss of function, failure to develop, or pathological state. It needs to be diagnosed as a developmentally relative deficit, such as the 93rd or 98th percentile in severity of symptoms for age (DuPaul, Power, et al., 1998).

This notion of changing symptom thresholds with age raises another critical issue for developing diagnostic criteria for ADHD, and this is the appropriateness of the content of the item set for different developmental periods. Inspection of the item lists suggests that the items for inattention may have a wider developmental applicability across the school-age range of childhood, and even into adolescence and young adulthood. Those for hyperactive–impulsive behavior, in contrast, seem much more applicable to young children and less appropriate or not at all to older teens and adults. As noted above (Hart et al., 1995), the symptoms of inattention remain stable across middle childhood into early adolescence, whereas those for hyperactive–impulsive behavior decline significantly over this same course. Although this may represent a true developmental decline in the severity of the latter symptoms, and possibly in the severity and prevalence of ADHD itself, it could also represent an illusory developmental trend. That is, it might be an artifact of using more preschool-focused items for hyperactivity and more school-age-focused items for inattention.

An analogy using mental retardation may be instructive. Consider the following items that might be chosen to assess developmental level in preschool-age children: being toilet-trained, recognizing colors, counting to 10, repeating 5 digits, buttoning snaps on clothing, recognizing simple geometric shapes, and using a vocabulary repertoire of at least 50 words. Evaluating whether or not a child is able to do these things may prove to be very useful in distinguishing mental retardation in preschoolers. However, if one continued to use this same item set to assess children with mental retardation as they grew older, one would find a decline in the severity of the retardation in such children as progressively more items were achieved with age. One would also find that the prevalence of retardation would decline markedly with age as many formerly delayed children "outgrew" these problems. But we know this would be illusory, because mental retardation represents a developmentally relative deficit in the achievement of mental and adaptive milestones.

To return to the diagnosis of ADHD, if the same developmentally restricted item sets are applied throughout development with no attempt to adjust either the thresholds or, more importantly, the types of items developmentally appropriate for different periods, we might see the same results as with the analogy to mental retardation described here. Similar results are found in ADHD (see below), which should give one pause before interpreting the observed decline in symptom severity (and even the observed decline in apparent prevalence!) as being accurate.

As it now stands, ADHD is being defined mainly by one of its earliest developmental manifestations (hyperactivity) and one of its later (school-age) yet secondary sequelae (deficient goal-directed persistence), and only minimally by its central features (deficits in inhibition and executive functioning).

Also of concern is the absence of any requirement in the DSM for the symptoms to be corroborated by someone who has known the patient well, such as a parent, sibling, long-time friend, or partner. Most likely, this arises from the focus on children throughout much of the history of the ADHD diagnostic category. Children routinely come to professionals with people who know them well (parents). But, in the case of adults who are self-referred to professionals, this oversight could prove potentially problematic. For instance, available evidence suggests that children with ADHD (Henry, Moffitt, Caspi, Langley, & Silva, 1994) and teens with the disorder (Edwards, Barkley, Laneri, Fletcher, & Metevia, 2001; Fischer et al., 1993b; Mannuzza & Gittelman, 1986; Romano, Tremblay, Vitaro, Zoccolillo, & Pagani, 2001) significantly underreport the severity of their symptoms, relative to the reports of parents. If this occurs in adults with ADHD as well, it would mean that self-referred patients might underestimate the severity of their disorder, resulting in a sizable number of false-negative decisions being made by clinicians. There are good reasons why self-awareness might be limited by this disorder. Neuropsychological research indicates that self-awareness is relatively localized to the prefrontal lobes, and that disorders affecting this region (such as Alzheimer's disease) markedly reduce self-awareness (Fuster, 1997; Stuss & Benson, 1986). As evidence reviewed below suggests, underactivity and underdevelopment in these same regions of the brain are likely to be involved in ADHD, and so the disorder ought to restrict self-awareness.

These issues are not merely academic. My colleagues and I have been involved in follow-up research on children with ADHD into their adulthood and have been impressed at the chronicity of impairments created by the disorder, despite an apparent decline in the percentage of cases continuing to meet diagnostic criteria and an apparent decline in the severity of the symptoms used in these criteria (Barkley, Fischer, Edelbrock, & Smallish, 1990; Barkley, Fischer, Fletcher, & Smallish, 2002; Fischer et al., 1993a). Recently, we found that if these children, who are now adults, were interviewed using the DSM criteria, just 5% of them reported sufficient symptoms to receive the diagnosis (Barkley, Fischer, Fletcher, & Smallish, 2002)—a figure nearly identical to that for the New York longitudinal studies (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993, 1998). If instead the parents were interviewed, this figure rose to 46%—a ninefold difference in persistence of disorder as a function of reporting source. If instead of the recommended DSM symptom threshold, one were to substitute a developmentally referenced criterion (the 98th percentile) based on same-age control adults, then 12% of the probands would now have the disorder as adults based on self-reports, while the figure would climb to 66% based on parental reports. Whose reports of current functioning were more valid? We addressed this by examining the relationship of self-reports and parent reports to various domains of major life activities and outcomes (education, occupational functioning, friendships, crime, etc.). Parent reports made a substantially larger contribution to nearly all outcome domains and did so for more such domains than did self-reports, suggesting that the parent reports probably had greater validity. The higher rates of disorder parents reported at outcome were thus probably the more accurate ones. Such adjustments for age and source of reporting, however, do not correct for the potentially increasing inappropriateness of the item sets for this agng sample, and so it is difficult to say how many of those not meeting these adjusted criteria may still have had the disorder.

A different issue pertains to whether or not the criteria should be adjusted for the gender of the children being diagnosed. Research evaluating these and similar item sets demonstrates that male youngsters display more of these items, and do so to a more severe degree, than do female youngsters in the general population (Achenbach, 1991; DuPaul, Power, et al., 1998). Given that the majority of children in the DSM-IV field trial were boys (Lahey et al., 1994), the symptom threshold chosen in the DSM-IV is more appropriate to males. This results in girls' having to meet a higher threshold relative to other girls to be diagnosed as having ADHD than do boys relative to other boys. Gender-adjusted thresholds would seem to be in order to address this problem; yet this would evaporate the currently disproportionate male-to-female ratio of 3:1 found across studies (see below).

The DSM-IV requirement of an age of onset for ADHD symptoms (7 years) in the diagnostic criteria has also come under attack from its own field trial (Applegate et al., 1997); a longitudinal study (McGee, Williams, & Feehan, 1992); and a review of this criterion from historical, empirical, and pragmatic perspectives (Barkley & Biederman, 1997). Such a criterion for age of onset suggests that there may be qualitative differences between those who meet the criterion (early-onset) and those who do not (late-onset). Some results do suggest that those with an onset before age 6 years may have more severe and persistent conditions, and more problems with reading and school performance generally (McGee et al., 1992). But these were matters of degree and not kind in this study. The DSM-IV field trial also was not able to show any clear discontinuities in degree of ADHD or in the types of impairments it examined between those meeting and those not meeting the 7-year age of onset. It remains unclear at this time just how specific an age of onset may need to be for distinguishing ADHD from other disorders. Suffice it to say that no other mental disorder in the DSM-IV has so precise an age of onset; this suggests that ADHD should not as well.

A related potential problem for these criteria occurs in their failure to stipulate a lower-bound age group for giving the diagnosis, below which no diagnosis should be made. This is important because research on preschool children has shown that a separate dimension of hyperactive–impulsive behavior from aggression or defiant behavior does not seem to emerge until about 3 years of age (Achenbach & Edelbrock, 1987; Campbell, 1990). Below this age, these behaviors cluster together to form what has been called "behavioral immaturity," "externalizing problems," or an "undercontrolled pattern of conduct." This implies that the symptoms of ADHD may be difficult to distinguish from other early behavioral disorders until at least 3 years of age, and so this age might serve as a lower bound for diagnostic applications.

Similarly, research implies that a lower bound of IQ might also be important (IQ > 50), below which the nature of ADHD may be quite different. Minimal research seems to exist that speaks to the issue of a discontinuity or qualitative shift in the nature of ADHD in individuals with IQs below 50. Some indirect evidence implies that this may occur, however. Rutter and colleagues (Rutter, Bolton, et al., 1990; Rutter, Macdonald, et al., 1990) have concluded that children who fall below this level of IQ may have a qualitatively different form of mental retardation. This is inferred from findings that this group is overrepresented for its position along a normal distribution, and from findings that genetic defects contribute more heavily to this subgroup. Given this shift in the prevalence and causes of mental retardation below this level of IQ, a similar state of affairs might exist for the form of ADHD associated with it, necessitating its distinction from the type of ADHD that occurs in individuals above this IQ level. Consistent with such a view have been findings that the percentage of those responding positively to stimulant medication falls off sharply below this threshold of IQ (Demb, 1991).

Another issue pertinent to this discussion is the problem of the duration requirement's being set at 6 months. This has been chosen mainly out of tradition (because earlier DSMs have done this), with no research support for selecting this particular length of time for symptom presence. It is undoubtedly important that the symptoms be relatively persistent if we are to view this disorder as a developmental disability, rather than as a problem arising purely from context or out of a transient, normal developmental stage. Yet specifying a precise duration is difficult in the absence of much research to guide the issue. Research on preschool-age children may prove helpful here, however. Such research has shown that many children aged 3 years (or younger) may have parents or preschool teachers who report concerns about the activity level or attention of the children; yet these concerns have a high likelihood of remission within 12 months (Beitchman, Wekerle, & Hood, 1987; Campbell, 1990; Lerner, Inui, Trupin, & Douglas, 1985; Palfrey, Levine, Walker, & Sullivan, 1985). It would seem for preschoolers that the 6-month duration specified in the DSM-IV may be too brief, resulting in overidentification of children with ADHD at this age (false positives). However, this same body of research found that for those children whose problems lasted at least 12 months or beyond age 4 years, the behavior problems were highly persistent and predictive of continuance into the school-age range. Such research suggests that the duration of symptoms be set at 12 months or more.

The DSM-IV requirement that the symptoms be demonstrated in at least two of three environments, so as to establish pervasiveness of symptoms, is new to this edition and problematic. The DSM-IV implies that two of three sources of in-

formation (parent, teacher, employer) must agree on the presence of the symptoms. This confounds settings with sources of information. The degree of agreement between parents and teacher for any dimension of child behavior is modest, often ranging between .30 and .50 (Achenbach, McConaughy, & Howell, 1987). This sets an upper limit on the extent to which parents and teachers are going to agree on the severity of ADHD symptoms, and thus on whether or not a child has the disorder in that setting. Such disagreements among sources certainly reflect differences in the child's behavior as a function of true differential demands of these settings. But they also reflect differences in the attitudes and judgments of different people. Insisting on such agreement may reduce the application of the diagnosis to some children unfairly as a result of such well-established differences between parent and teacher opinions. It may also create a confounding of the disorder with, or issues of comorbidity with, ODD (Costello, Loeber, & Stouthamer-Loeber, 1991). Parent-only-identified children with ADHD may have predominantly ODD with relatively milder ADHD, whereas teacher-only-identified children with ADHD may have chiefly ADHD and minimal or no ODD symptoms. Children identified by both parents and teachers as having ADHD may therefore carry a higher likelihood of having ODD. They may also simply have a more severe form of ADHD than do the home- or school-only cases, being different in degree rather than in kind. Research is clearly conflicting on the matter (Cohen & Minde, 1983; Rapoport, Donnelly, Zametkin, & Carrougher, 1986; Schachar, Rutter, & Smith, 1981; Taylor, Sandberg, Thorley, & Giles, 1991). Considering that teacher information on children is not always obtainable or convenient, that parents can convey the essence of that information to clinicians, and that diagnosis based on parents' reports will lead to a diagnosis based on teacher reports 90% of the time (Biederman, Keenan, & Faraone, 1990), all imply that parent reports may suffice for diagnostic purposes for now. However, more recent evidence suggests that the best discrimination of children with ADHD from other groups may be achieved by blending the reports of parents and teachers, such that one counts the number of different symptoms endorsed across *both* sources of information (Crystal, Ostrander, Chen, & August, 2001; Mitsis, McKay, Schulz, Newcorn, & Halperin, 2000).

Many of these problematic issues are likely to be addressed in future editions of the DSM. Even so, the present criteria are actually some of the best ever advanced for the disorder; they represent a vast improvement over the state of affairs that existed prior to 1980. The various editions of DSM also have spawned a large amount of research into ADHD—its symptoms, subtypes, criteria, and even etiologies—that probably would not have occurred had such criteria not been set forth for professional consumption and criticism. The most recent criteria provide clinicians with a set of guidelines more specific, more reliable, more empirically based or justifiable, and closer to the scientific literature on ADHD than earlier editions. With some attention to the issues described above, the DSM criteria could be made to be even more rigorous, valid, and useful.

Is ADHD a "Real" Disorder?

Social critics (Breggin, 1998; Kohn, 1989; Schrag & Divoky, 1975) have charged that professionals have been too quick to label energetic and exuberant children as having a mental disorder. They also assert that educators may be using these labels as an excuse for simply poor educational environments. In other words, children who are diagnosed with hyperactivity or ADHD are actually normal, but are being labeled as mentally disordered because of parent and teacher intolerance (Kohn, 1989) or lack of love at home (Breggin, 1998). If this were actually true, then we should find no differences of any cognitive, neurological, genetic, behavioral, or social significance between children so labeled and normal children. We should also find that the diagnosis of ADHD is not associated with any significant risks later in development for maladjustment within any domains of adaptive functioning, or for problems with social, occupational, or school performance. Furthermore, research on potential etiologies for the disorder should likewise come up empty-handed. This is hardly the case, as evidence reviewed in this chapter attests. Differences between children with ADHD and normal children are too numerous to take these assertions of normality seriously. As will be shown later, substantial developmental risks await children meeting clinical diagnostic criteria for the disorder, and certain potential etiological factors are becoming consistently noted in the research literature.

Conceding all of this, however, does not automatically entitle ADHD to be placed within the

realm of valid ("real") disorders. Wakefield (1999) has argued that disorders must meet two criteria to be viewed as valid: They must (1) engender substantial harm to the individual or those around him or her, and (2) incur dysfunction of natural and universal mechanisms that have been selected in an evolutionary sense (i.e., have survival value). The latter criterion is based on the definition of an adaptation as used in evolutionary biology. Disorders are failures in adaptations that produce harm. In the case of psychology, these universal mechanisms are psychological ones possessed by all normally developing humans, regardless of culture. ADHD handily meets both criteria. Those with ADHD, as described in the theory above, have significant deficits in behavioral inhibition and inattention (the executive functions) that are critical for effective self-regulation. And those with ADHD experience numerous domains of impairment (risks of harm) over development, as will become evident below.

EPIDEMIOLOGY

Prevalence

The prevalence of ADHD varies across studies, at least in part due to different methods of selecting samples, the nature of the populations from which they are drawn (differing nationalities or ethnicities, urban vs. rural, community vs. primary care settings, etc.), the criteria used to define ADHD (DSM criteria vs. rating scale cutoff), and certainly the age range and sex composition of the samples. When only the endorsement of the presence of the behavior of hyperactivity (not the clinical disorder) is required from either parent or teacher rating scales, prevalence rates can run as high as 22–57% (Lapouse & Monk, 1958; McArdle, O'Brien, & Kolvin, 1995; Werry & Quay, 1971). This underscores the point made earlier that being described as inattentive or overactive by a parent or teacher does not in and of itself constitute a disorder in a child.

Szatmari (1992) reviewed the findings of six large epidemiological studies that identified cases of ADHD within these samples. The prevalences found in these studies ranged from a low of 2% to a high of 6.3%, with most falling within the range of 4.2% to 6.3%. Other studies have found similar prevalence rates in elementary school-age children (4–5.5% in Breton et al., 1999; 7.9% in Briggs-Gowan, Horwitz, Schwab-Stone, Leventhal, & Leaf, 2000; 5–6% in DuPaul, 1991; and 2.5–4% in Pelham, Gnagy, Greenslade, & Milich, 1992). Lower rates result from using complete DSM criteria and parent reports (2–6% in Breton et al., 1999), and higher ones if just a cutoff on teacher ratings is used (up to 23% in DuPaul, Power, et al., 1998; 15.8% in Nolan, Gadow, & Sprafkin, 2001; 14.3% in Trites, Dugas, Lynch, & Ferguson, 1979). Sex and age differences in prevalence are routinely found in research. For instance, prevalence rates may be 4% in girls and 8% in boys in the preschool age group (Nolan et al., 2001), yet fall to 2–4% in girls and 6–9% in boys during the 6- to 12-year-old age period based on parent reports (Breton et al., 1999; Szatmari, Offord, & Boyle, 1989). The prevalence decreases again to 0.9–2% in girls and 1–5.6% in boys by adolescence (Breton et al., 1999; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; McGee et al., 1990; Romano et al., 2001; Szatmari et al., 1989). Even then, if both a symptom threshold and the requirement for impairment are used, the prevalence may decrease by 20–60% from that figure based on symptom thresholds alone (Breton et al., 1999; Romano et al., 2001; Wolraich, Hannah, Baumgaertel, & Feurer, 1998). As noted above, prevalence rates are routinely higher (sometimes more than double) when teacher reports are used in comparison to parent reports (Breton et al., 1999; DuPaul, Power, et al., 1998; Nolan et al., 2001). Switching from DSM-III-R criteria (used before 1994) to DSM-IV (in use since that time) may have resulted in a near-doubling in prevalence, owing to the inclusion of the new inattentive subtype (ADHD-PI), which was not included in DSM-III-R (Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996). Some segments of the population may also have greater levels of ADHD than others. For instance, Jensen et al. (1995), using DSM-III-R criteria, found a prevalence of 12% for ADHD among the children of military personnel—a figure more than double that found in other studies using these same criteria with general population samples (Szatmari, 1992).

Szatmari et al. (1989) found that the prevalence of ADHD in a large sample of children from Ontario, Canada also varied as a function of young age, male gender, chronic health problems, family dysfunction, low socioeconomic status (SES), presence of a developmental impairment, and urban living. Others have found similar conditions associated with the risk for ADHD (Lavigne et al., 1996; Velez, Johnson, & Cohen, 1989). Important,

however, was the additional finding in the Szatmari et al. (1989) study that when comorbidity with other disorders was statistically controlled for in the analyses, gender, family dysfunction, and low SES were no longer significantly associated with prevalence. Health problems, developmental impairment, young age, and urban living remained significantly associated with prevalence, however.

As noted above in the discussion of DSM-IV criteria, it may be that the declining prevalence of ADHD with age is partly artifactual. This could result from the use of items in the diagnostic symptom lists that are chiefly applicable to young children. This could create a situation where individuals remain impaired in the fundamental constructs of ADHD as they mature, while outgrowing the symptom list for the disorder, resulting in an illusory decline in prevalence (as was noted in my follow-up study discussed above). Until more age-appropriate symptoms are studied for adolescent and adult populations, this issue remains unresolved.

Sex Differences

As noted above, sex appears to play a significant role in determining prevalence of ADHD within a population. On average, male children are between 2.5 and 5.6 times more likely than female children to be diagnosed as having ADHD within epidemiological samples, with the average being roughly 3:1 (Breton et al., 1999; DuPaul, Power, et al., 1998; Lewinsohn et al., 1993; McGee et al., 1990; Szatmari, 1992). Within clinic-referred samples, the sex ratio can be considerably higher, suggesting that boys with ADHD are far more likely to be referred to clinics than girls. This is probably because boys are more likely to have comorbid ODD or CD. Szatmari's (1992) finding that sex differences were no longer associated with the occurrence of ADHD, once other comorbid conditions were controlled for in statistical analyses, implies that this may be the case. The sex ratio could also be an artifact of applying a set of diagnostic criteria developed primarily on males to females, as discussed above.

Studies of clinic-referred girls often find that they are as impaired as clinic-referred boys with ADHD, have as much comorbidity, and may even have greater deficits in intelligence, according to meta-analytic reviews of sex differences in ADHD (Gaub & Carlson, 1997; Gershon, 2001). Some studies suggest that these clinic-referred girls, at least as adolescents, may have more internalizing symptoms (e.g., depression, anxiety, and stress), greater problems with teacher relationships, and poorer verbal abilities (vocabulary) than boys with ADHD (Rucklidge & Tannock, 2001). Like the boys, girls with ADHD also manifest more CD, mood disorders, and anxiety disorders; have lower intelligence; and have greater academic achievement deficits than do control samples (Biederman, Faraone, et al., 1999; Rucklidge & Tannock, 2001). Males with ADHD had greater problems with cognitive processing speed than females in one study, but these differences were no longer significant after severity of ADHD was controlled for (Rucklidge & Tannock, 2001). No sex differences have been identified in executive functioning, with both sexes being more impaired than control samples on such measures (Castellanos et al., 2000; Murphy et al., 2001). In contrast, studies drawing their ADHD samples from the community find that girls are significantly less likely to have comorbid ODD and CD than boys with ADHD, and do not have greater intellectual deficits than these boys; however, they may be as socially and academically impaired as boys with the disorder (Carlson, Tamm, & Gaub, 1997; Gaub & Carlson, 1997; Gershon, 2001).

Socioeconomic Differences

Few studies have examined the relationship of ADHD to SES, and those that have are not especially consistent. Lambert, Sandoval, and Sassone (1978) found only slight differences in the prevalence of hyperactivity across SES when parent, teacher, and physician all agreed on the diagnosis. However, SES differences in prevalence did arise when only two of these three sources had to agree; in this instance, there were generally more children with ADHD from lower- than higher-SES backgrounds. For instance, when parent and teacher agreement (but not physician) was required, 18% of those identified as hyperactive were from high-SES, 36% from middle-SES, and 45% from low-SES backgrounds. Where only teachers' opinions were used, the percentages were 17%, 41%, and 41%, respectively. Trites (1979), and later Szatmari (1992), both found that rates of ADHD tended to increase with lower SES. However, in his own study Szatmari (Szatmari et al., 1989) found that low SES was no longer associated with rates of ADHD when other comorbid conditions, such as CD, were controlled for. For now, it is clear that

ADHD occurs across all socioeconomic levels. Variations across SES may be artifacts of the source used to define the disorder or of the comorbidity of ADHD with other disorders related to SES, such as ODD and CD.

Ethnic/Cultural/National Issues

Early studies of the prevalence of hyperactivity, relying principally on teacher ratings, found significant disparities across four countries (United States, Germany, Canada, and New Zealand)—ranging from 2% in girls and 9% in boys in the United States to 9% in girls and 22% in boys in New Zealand (Trites et al., 1979). Similarly, O'Leary, Vivian, and Nisi (1985), using this same teacher rating scale and cutoff score, found rates of hyperactivity to be 3% in girls and 20% in boys in Italy. However, this may have resulted from the use of a threshold established on norms collected in the United States across these other countries, where the distributions were quite different from those found in the United States.

Later studies, especially those using DSM criteria, have found the disorder across numerous countries. In a Japanese study (Kanbayashi, Nakata, Fujii, Kita, & Wada, 1994) using parent ratings of items from DSM-III-R, a prevalence rate of 7.7% of the sample was found. Baumgaertel (1994) used teacher ratings of DSM-III, DSM-III-R, and DSM-IV symptom lists in a large sample of German elementary school children and found rates of 4.8% for ADHD-C, 3.9% for ADHD-PHI, and 9% for ADHD-PI based on DSM-IV. In India, among over 1,000 children screened at a pediatric clinic, 5.2% of children ages 3–4 years were found to have ADHD by DSM-III-R criteria, whereas the rate rose to over 29% for ages 11–12 years (Bhatia, Nigam, Bohra, & Malik, 1991). This was not a true epidemiological sample, however. Differences in prevalence across ages could simply reflect cohort effects; children may be referred to this clinic for different reasons at different ages. Prevalence rates found in other countries more recently are as follows:

- 3.8% among 2,290 Dutch 6- to 8-year-olds in a study using parent-reported DSM criteria (Kroes et al., 2001).
- 5.3% among 2,936 Chinese 6- to 11-year-olds, falling to 3.9% for 1,694 Chinese 12- to 16-year-olds, in a study using teacher ratings (Liu et al., 2000).
- 5.8% among 1,013 Brazilian 12- to 14-year-olds, in a study using teacher ratings (Rhohde et al., 1999).
- 20% of boys and 12% of girls 4–17 years of age in 504 children randomly sampled from 80,000 Colombian children, in a study using just DSM-IV symptom thresholds with parent ratings (Pineda et al., 1999).
- 14.9% of 1,110 primary school children randomly chosen from more than 31,000 in the United Arab Emirates, in a study using teacher ratings (Bu-Haroon, Eapen, & Bener, 1999).
- 19.8% of 600 Ukrainian 10- to 12-year-old children, in a study using parent ratings of DSM-IV symptoms (Gadow et al., 2000).

Cultural differences in the interpretations given to symptoms of ADHD by teachers or parents and in expectations for child behavior undoubtedly exist and have probably contributed to the higher rates of disorder found in some of these countries compared to North American rates. Also, most of these studies used teacher or parent ratings rather than clinical diagnostic criteria. As already noted above, prevalence rates of hyperactivity or ADHD are typically higher when a threshold on a rating scale is the only criterion for establishing a case of the disorder. When clinical criteria are employed, rates are more conservative. Nevertheless, these studies together show that hyperactivity or ADHD is present in all countries studied to date. Although it may not receive the same diagnostic label in each, the behavior pattern constituting the disorder appears to be universal.

Differences among ethnic groups in rates of hyperactivity within the United States have been reported. Langsdorf, Anderson, Walchter, Madrigal, and Juarez (1979) reported that almost 25% of African American children and 8% of Hispanic American children met a cutoff score on a teacher rating scale commonly used to define hyperactivity, whereas Ullmann (cited in O'Leary et al., 1985) reported rates of 24% for African American children and 16% of European American children on a teacher rating scale. Lambert et al. (1978) found higher rates of hyperactivity among African American than European American children only when the teachers were the only ones reporting the diagnosis; Hispanic American children were not found to differ from European American children in this respect. Such differ-

ences, however, may arise in part because of socioeconomic factors that are differentially associated with these ethnic groups in the United States. Such psychosocial factors are strongly correlated with aggression and conduct problems. As noted above, those factors no longer make a significant contribution to the prevalence of ADHD when comorbidity for other disorders is controlled for (Szatmari, 1992). Doing the same within studies of ethnic differences might well reduce or eliminate these differences in prevalence among them. Thus it would seem that ADHD arises in all ethnic groups studied so far. Whether the differences in prevalence across these ethnic groups are real or are a function of the source of information about the symptoms of ADHD (and possibly socioeconomic factors) remains to be determined.

DEVELOPMENTAL COURSE AND ADULT OUTCOMES

Major follow-up studies of clinically referred hyperactive children have been ongoing during the last 25 years at five sites: (1) Montreal (Weiss & Hechtman, 1993), (2) New York City (Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Mannuzza et al., 1993), (3) Iowa City (Loney, Kramer, & Milich, 1981), (4) Los Angeles (Satterfield, Hoppe, & Schell, 1982), and (5) Milwaukee (Barkley, Fischer, et al., 1990). Follow-up studies of children identified as hyperactive from a general population have also been conducted in the United States (Lambert, 1988), New Zealand (McGee, Williams, & Silva, 1984; Moffitt, 1990), and England (Taylor et al., 1991), among others.

But before I embark on a summary of their results, some cautionary notes are in order. First, the limited number of follow-up studies does not permit a great deal of certainty to be placed in the specificity of the types and degrees of outcomes likely to be associated with ADHD. Even so, more can likely be said about the outcomes of ADHD than about those of most other childhood mental disorders. Second, the discontinuities of measurement that exist in these follow-up studies between their different points of assessments of their subjects make straightforward conclusions about developmental course difficult. Third, the differing sources of children greatly affect the outcomes to be found, with children drawn from clinic-referred populations having two to three times the occurrence of some negative outcomes and more diverse negative outcomes than those drawn from population screens (e.g., Barkley, Fischer, et al., 1990, vs. Lambert, 1988). Fourth, the differing entry/diagnostic criteria across follow-up studies must be kept in mind in interpreting and cross-referencing their outcomes. Most studies selected for children known at the time as "hyperactive." Such children are most likely representative of the course of ADHD-C from the current DSM taxonomy. Even then, the degree of deviance of the samples on parent and teacher ratings of these symptoms was not established at the entry point in most of these studies. These studies also cannot be viewed as representing ADHD-PI, for which no follow-up information is currently available. The descriptions of clinic-referred children with ADHD who are of similar age groups to those in the follow-up studies, but who are not followed over time, may help us understand the risks associated with different points in development. However, these may also be contaminated by cohort effects at the time of referral and so can only be viewed as suggestive. Such cohort effects may be minor; that is, adolescents with ADHD referred to clinics seem to have types and degrees of impairment similar to those of children with ADHD followed up to adolescence (Barkley, Anastopoulos, Guevremont, & Fletcher, 1991 vs. Barkley, Fischer, et al., 1990). In painting the picture of the developmental outcome of ADHD, then, broad strokes are permissible, but the finer details await more and better-refined studies. I concentrate here on the course of the disorder itself, returning to the comorbid disorders and associated conditions likely to arise in the course of ADHD in a later section of this chapter ("Comorbid Psychiatric Disorders").

The average onset of ADHD symptoms, as noted earlier, is often in the preschool years, typically at ages 3–4 (Applegate et al., 1997; Loeber et al., 1992; Taylor et al., 1991) and more generally by entry into formal schooling. Yet onset is heavily dependent on the type of ADHD under study. First to arise is the pattern of hyperactive–impulsive behavior (and, in some cases, oppositional and aggressive conduct), giving that subtype the earliest age of onset. ADHD-C has an onset within the first few grades of primary school (ages 5–8; Hart et al., 1995), most likely due to the requirement that both hyperactivity and in-

attention be present to diagnose this subtype. ADHD-PI appears to emerge a few years later (ages 8–12) than the other types (Applegate et al., 1997).

Preschool-age children who are perceived as difficult and resistant to control, or who have inattentive and hyperactive behavior that persists for at least a year or more, are highly likely to have ADHD and to remain so into elementary school years (Beitchman et al., 1987; Campbell, 1990; Palfrey et al., 1985) and even adolescence (Olson, Bates, Sandy, & Lanthier, 2000). Persistent cases seem especially likely to occur where parent–child conflict, greater maternal directiveness and negativity, and greater child defiant behavior exist (Campbell, March, Pierce, Ewing, & Szumowski, 1991; Olson et al., 2000; Richman, Stevenson, & Graham, 1982). More negative temperament and greater emotional reactivity to events are also more common in preschool children with ADHD (Barkley, DuPaul, & McMurray, 1990; Campbell, 1990). It is little wonder that greater parenting stress is associated with having preschool children with ADHD, and such stress seems to be at its highest with preschoolers relative to later age groups (Mash & Johnston, 1983a, 1983b). Within the preschool setting, children with ADHD will be found to be more often out of their seats, wandering the classroom, being excessively talkative and vocally noisy, and disruptive of other children's activities (Campbell, Schleifer, & Weiss, 1978; Schleifer et al., 1975).

By the time children with ADHD move into the elementary school-age range of 6–12 years, the problems with hyperactive–impulsive behavior are likely to continue and to be joined now by difficulties with attention (executive functioning and goal-directed persistence). Difficulties with work completion and productivity, distraction, forgetfulness related to what needs doing, lack of planning, poor organization of work activities, trouble meeting time deadlines associated with home chores, school assignments, and social promises or commitments to peers are now combined with the impulsive, heedless, and disinhibited behavior typifying these children since preschool age. Problems with oppositional and socially aggressive behavior may emerge at this age in at least 40–70% of children with ADHD (Barkley, 1998; Loeber et al., 1992; Taylor et al., 1991).

By ages 8–12 years, these early forms of defiant and hostile behavior may evolve further into symptoms of CD in 25–45% or more of all children with ADHD (Barkley, Fischer, et al., 1990; Gittelman et al., 1985; Loeber et al., 1992; Mannuzza et al., 1993; Taylor et al., 1991). Certainly by late childhood, most or all of the deficits in the executive functions related to inhibition in the model presented earlier are likely to be arising and interfering with adequate self-regulation (Barkley, 1997b). Not surprisingly, the overall adaptive functioning (self-sufficiency) of many children with ADHD (Stein, Szumowski, et al., 1995) is significantly below their intellectual ability. This is also true of preschoolers with high levels of these externalizing symptoms (Barkley, Shelton, et al., 2002). The disparity between adaptive functioning and age-appropriate expectations (or IQ) may itself be a predictor of greater severity of ADHD, as well as risk for oppositional and conduct problems in later childhood (Shelton et al., 1998). The disorder takes its toll on self-care, personal responsibility, chore performance, trustworthiness, independence, and appropriate social skills, as well as doing tasks on time specifically and moral conduct generally (Barkley, 1998; Hinshaw et al., 1993).

If ADHD is present in clinic-referred children, the likelihood is that 50–80% will continue to have their disorder into adolescence, with most studies supporting the higher figure (August, Stewart, & Holmes, 1983; Claude & Firestone, 1995; Barkley, Fischer, et al., 1990; Gittelman et al., 1985; Mannuzza et al., 1993). Using the same parent rating scales at both the childhood and adolescent evaluation points, Fischer et al. (1993a) were able to show that inattention, hyperactive–impulsive behavior, and home conflicts declined by adolescence. The hyperactive group showed far more marked declines than the control group, mainly because the former were so far from the mean of the normative group to begin with in childhood. Nevertheless, even at adolescence, the groups remained significantly different in each domain, with the mean for the hyperactive group remaining two standard deviations or more above the mean for the controls. This emphasizes a point made earlier: Simply because severity levels of symptoms are declining over development, this does not mean that children with ADHD are necessarily outgrowing their disorder relative to normal children. Like mental retardation, ADHD may need to be defined as a developmentally relative deficiency, rather than an absolute one, that persists in most children over time.

The persistence of ADHD symptoms across childhood as well as into early adolescence appears, again, to be associated with initial degree of hyperactive–impulsive behavior in childhood; the coexistence of conduct problems or oppositional hostile behavior; poor family relations, specifically conflict in parent–child interactions; and maternal depression, as well as duration of maternal mental health interventions (Fischer et al., 1993b; Taylor et al., 1991). These predictors have also been associated with the development and persistence of ODD and CD into this age range (12–17 years; Fischer et al., 1993b; Loeber, 1990; Mannuzza & Klein, 1992; Taylor et al., 1991).

Studies following large samples of clinic-referred children with hyperactivity, or ADHD, into adulthood are few in number. Only four follow-up studies have retained 50% or more of their original samples into adulthood and reported on the persistence of symptoms to that time. These are the Montreal study by Weiss, Hechtman, and their colleagues (see Weiss & Hechtman, in press); the New York City study by Mannuzza, Klein, and colleagues (see Mannuzza et al., 1993, 1998); the Swedish study by Rasmussen and Gillberg (2001); and my research with Mariellen Fischer in Milwaukee (Barkley, Fischer, Fletcher, & Smallish, 2002; Barkley, Fischer, Smallish, & Fletcher, in press; Fischer et al., in press-a, in press-b). The results regarding the persistence of disorder into young adulthood (middle 20s) are mixed, but can be better understood as being a function of reporting source and the diagnostic criteria used (Barkley, Fisher, Fletcher, & Smallish, 2002).

The Montreal study (n = 103) found that two-thirds of the original sample (n = 64; mean age = 25 years) claimed to be troubled as adults by at least one or more disabling core symptoms of their original disorder (restlessness, impulsivity, or inattention), and that 34% had at least moderate to severe levels of hyperactive, impulsive, and inattentive symptoms (Weiss & Hechtman, 1993). In Sweden (n = 50), Rasmussen and Gillberg (2001) obtained similar results, with 49% of probands reporting marked symptoms of ADHD at age 22 years compared to 9% of controls. Formal diagnostic criteria for ADHD, such as those in DSM-III or later editions, were not employed at any of the outcome points in either study, however. In contrast, the New York study has followed two separate cohorts of hyperactive children, using DSM criteria to assess persistence of disorder. That study found that 31% of the initial cohort (n = 101) and 43% of the second cohort (n = 94) met DSM-III criteria for ADHD by ages 16–23 (mean age = 18.5 years) (Gittelman et al., 1985; Mannuzza et al., 1991). Eight years later (mean age = 26 years), however, these figures fell to 8% and 4%, respectively (with DSM-III-R criteria now being used) (Mannuzza et al., 1993, 1998). Those results might imply that the vast majority of hyperactive children no longer qualify for the diagnosis of ADHD by adulthood.

The interpretation of the relatively low rate of persistence of ADHD into adulthood, particularly for the New York study, is clouded by at least two issues apart from differences in selection criteria. One is that the source of information about the disorder changed in all of these studies from that used at the childhood and adolescent evaluations to that used at the adult outcome. At study entry and at adolescence, all studies used the reports of others (parents and typically teachers). By midadolescence, all found that the majority of hyperactive participants (50–80%) continued to manifest significant levels of the disorder (see above). In young adulthood (approximately age 26 years), both the New York and Montreal studies switched to self-reports of disorder.

The rather marked decline in persistence of ADHD from adolescence to adulthood could stem from this change in source of information. Indeed, the New York study found this to be likely when, at late adolescence (mean age of 18–19 years), both the teenagers and their parents were interviewed about the teens' psychiatric status (Mannuzza & Gittelman, 1986). There was a marked disparity between the reports of parents and teens concerning the presence of ADHD (11% vs. 27%; agreement = 74%, kappa = .19). Other research also suggests that the relationship between 11-year-old children's self-reports of externalizing symptoms, such as those involved in ADHD, and those of parents and teachers is quite low (r = .16–.32; Henry et al., 1994). Thus changing sources of reporting in longitudinal studies on behavioral disorders can be expected to lead to marked differences in estimates of persistence of those disorders.

The question obviously arises as to whose assessment of the probands is more accurate. This would depend on the purpose of the assessment, but the prediction of impairment in major life activities would seem to be an important one in

research on psychiatric disorders. Our Milwaukee study examined these issues by interviewing both the participants and their parents about ADHD symptoms at the young adult follow-up (age 21 years). It then examined the relationship of each source's reports to significant outcomes in major life activities (education, occupation, social, etc.), after controlling for the contribution made by the other source. As noted earlier, another limitation in the earlier studies may reside in the DSM criteria, in that they grow less sensitive to the disorder with age. Using a developmentally referenced criterion (age comparison) to determine diagnosis may identify more cases than would the DSM approach. As discussed earlier, the Milwaukee study found that the persistence of ADHD into adulthood was heavily dependent on the source of the information (self or parent) and the diagnostic criteria (DSM or developmentally referenced). Self-report identified just 5–12% of probands as currently having ADHD (DSM-III-R), whereas parent reports placed this figure at 46–66%. Using the DSM resulted in lower rates of persistence (5% for proband reports and 46% for parents), whereas using a developmentally referenced cutoff (98th percentile) yielded higher rates of persistence (12% by self-reports and 66% by parent reports). The parent reports appeared to have greater validity, in view of their greater contribution to impairment and to more domains of current impairment, than did self-reported information (Barkley, Fischer, Fletcher, & Smallish, 2002). We have concluded that past follow-up studies grossly underestimated the persistence of ADHD into adulthood by relying solely on the self-reports of the probands.

COMORBID PSYCHIATRIC DISORDERS

Individuals diagnosed with ADHD are often found to have a number of other disorders besides their ADHD. What is known about comorbidity is largely confined to the ADHD-C subtype. In community-derived samples, up to 44% of children with ADHD have at least one other disorder, and 43% have at least two or more additional disorders (Szatmari et al., 1989). The figure is higher, of course, for children drawn from clinics. As many as 87% of children clinically diagnosed with ADHD may have at least one other disorder, and 67% have at least two other disorders (Kadesjo & Gillberg, 2001). The disorders likely to co-occur with ADHD are briefly described below.

Conduct Problems and Antisocial Disorders

The most common comorbid disorders with ADHD-C are ODD and, to a lesser extent, CD. Indeed, the presence of ADHD increases the odds of ODD/CD by 10.7-fold (95% confidence interval [CI] = 7.7–14.8) in general population studies (Angold, Costello, & Erkanli, 1999). Studies of clinic-referred children with ADHD find that between 54% and 67% will meet criteria for a diagnosis of ODD by 7 years of age or later. ODD is a frequent precursor to CD, a more severe and often (though not always) later-occurring stage of ODD (Loeber, Burke, Lahey, Winters, & Zera, 2000). The co-occurrence of CD with ADHD may be 20–50% in children and 44–50% in adolescence with ADHD (Barkley, 1998; Barkley, Fischer, et al., 1990; Biederman, Faraone, & Lapey, 1992; Lahey, McBurnett, & Loeber, 2000). By adulthood, up to 26% may continue to have CD, while 12–21% will qualify for a diagnosis of antisocial personality disorder (ASPD) (Biederman et al., 1992; Fischer, Barkley, Smallish, & Fletcher, in press; Mannuzza & Klein, 1992; Rasmussen & Gillberg, 2001; Weiss & Hechtman, 1993b). Similar or only slightly lower degrees of overlap are noted in studies using epidemiologically identified samples rather than those referred to clinics. ADHD therefore has a strong association with conduct problems and antisocial disorders, such as ODD, CD, and ASPD, and has been found to be one of the most reliable early predictors of these disorders (Fischer et al., 1993b; Hinshaw & Lee, Chapter 3, this volume; Lahey et al., 2000). Recent longitudinal research suggests that severity of early ADHD is actually a contributing factor to risk for later ODD, regardless of severity of early ODD (Burns & Walsh, 2002), perhaps due to the problems with poor emotion (anger) regulation in ADHD noted above. Familial associations among the disorders have also been consistently found, whether across boys and girls with ADHD or across European American and African American samples (Biederman et al., 1992; Faraone et al., 2000; Samuel t al., 1999). This suggests some underlying causal connection among these disorders. Evidence from twin

studies indicates a shared or common genetic contribution to the three disorders, particularly between ADHD and ODD (Coolidge, Thede, & Young, 2000; Silberg et al., 1996). When CD occurs in conjunction with ADHD, it may represent simply a more severe form of ADHD having a greater family genetic loading for ADHD (Thapar, Harrington, & McGuffin, 2001). Other research, however, also suggests a shared environmental risk factor may also account for the overlap of ODD and CD with ADHD beyond their shared genetics (Burt, Krueger, McGue, & Iacono, 2001), that risk factor likely being family adversity generally and impaired parenting specifically (Patterson, Degarmo, & Knutson, 2000). To summarize, ODD and CD have a substantial likelihood of co-occuring with ADHD, with the risk for ODD/CD being mediated in large part by severity of ADHD and its family genetic loading and in part by adversity in the familial environment.

One of the strongest predictors of risk for substance use disorders (SUDs) among children with ADHD upon reaching adolescence and adulthood is prior or coexisting CD or ASPD (Burke et al., 2001; Chilcoat & Breslau, 1999; Molina & Pelham, 1999; White, Xie, Thompson, Loeber, & Stouthamer-Loeber, 2001). Given the heightened risk for ODD/CD/ASPD in ADHD children as they mature, one would naturally expect a greater risk for SUDs as well. Although an elevated risk for alcohol abuse has not been documented in follow-up studies, the risk for other SUDs among hyperactive children followed to adulthood ranges from 12% to 24% (Fischer et al., in press-b; Gittelman et al., 1985; Mannuzza et al., 1993, 1998; Rasmussen & Gillberg, 2001). One longitudinal study of hyperactive children suggested that childhood treatment with stimulant medication may predispose youths to develop SUDs (Lambert, in press; Lambert & Hartsough, 1998). Most longitudinal studies, however, find no such elevated risk, and in some cases even a protective effect if stimulant treatment is continued for a year or more or into adolescence (Barkley, Fischer, Smallish, & Fletcher, in press; Biederman, Wilens, Mick, Spencer, & Faraone, 1999; Chilcoat & Breslau, 1999; Loney, Kramer, & Salisbury, in press). The basis for the conflicting findings in the Lambert study was probably not examining or statistically controlling for severity of ADHD and CD at adolescence and young adulthood (Barkley, Fischer, Smallish, & Fletcher, in press).

Anxiety and Mood Disorders

The overlap of anxiety disorders with ADHD has been found to range from 10% to 40% in clinic-referred children, averaging to about 25% (see Biederman, Newcorn, & Sprich, 1991, and Tannock, 2000, for reviews). In longitudinal studies of children with ADHD, however, the risk of anxiety disorders is no greater than in control groups at either adolescence or young adulthood (Fischer et al., in press-b; Mannuzza et al., 1993, 1998; Russo & Beidel, 1994; Weiss & Hechtman, 1993). The disparity in findings is puzzling. Perhaps some of the overlap of ADHD with anxiety disorders in children is due to referral bias (Biederman et al., 1992; Tannock, 2000). General population studies of children, however, do suggest an elevated odds ratio of having an anxiety disorder in the presence of ADHD of 3.0 (95% CI = 2.1–4.3), with this relationship being significant even after controlls for comorbid ODD/CD (Angold et al., 1999). This implies that the two disorders may have some association apart from referral bias, at least in childhood. The co-occurrence of anxiety disorders with ADHD has been shown to reduce the degree of impulsiveness, relative to ADHD without comorbid anxiety disorders (Pliszka, 1992). Some research suggests that the disorders are transmitted independently in families and so are not linked to each other in any genetic way (Biederman, Newcorn, & Sprich, 1991; Last, Hersen, Kazdin, Orvaschel, & Perrin, 1991). This may not be the case for ADHD-PI: Higher rates of anxiety disorders have been noted in some studies of these children (see Milich et al., 2001, for a review; Russo & Beidel, 1994), though not always (Barkley, DuPaul, & McMurray, 1990), and in their first- and second-degree relatives (Barkley, DuPaul, & McMurray, 1990; Biederman et al., 1992), though again not always (Lahey & Carlson, 1992; Milich et al., 2001). Regrettably, research on the overlap of anxiety disorders with ADHD has generally chosen to consider the various anxiety disorders as a single group in evaluating this issue. Greater clarity and clinical utility from these findings might occur if the types of anxiety disorders present were to be examined separately.

The evidence for the co-occurrence of mood disorders, such as major depression or dysthymia (a milder form of depression), with ADHD is now fairly substantial (see Faraone & Biederman, 1997; Jensen, Martin, & Cantwell, 1997; Jensen, Shervette, Xenakis & Richters,

1993; and Spencer, Wilens, Biederman, Wozniak, & Harding-Crawford, 2000, for reviews). Between 15% and 75% of those with ADHD may have a mood disorder, though most studies place the association between 20% and 30% (Biederman et al., 1992; Cuffe et al., 2001; Fischer et al., in press-b). The odds ratio of having depression, given the presence of ADHD in general population samples, is 5.5 (95% CI = 3.5–8.4) (Angold et al., 1999). Some evidence also suggests that these disorders may be related to each other, in that familial risk for one disorder substantially increases the risk for the other (Biederman, Newcorn, & Sprich, 1991; Biederman et al., 1992; Faraone & Biederman, 1997), particularly in cases where ADHD is comorbid with CD. Similarly, a recent follow-up study (Fischer et al., in press-b) found a 26% risk of major depression among children with ADHD by young adulthood, but this risk was largely mediated by the co-occurrence of CD. Likewise, a meta-analysis of general population studies indicated that the link between ADHD and depression was entirely mediated by the linkage of both disorders to CD (Angold et al., 1999). In the absence of CD, ADHD was not more likely to be associated with depression.

The comorbidity of ADHD with bipolar (manic–depressive) disorder is controversial (Carlson, 1990; Geller & Luby, 1997). Some studies of ADHD children indicate that 10–20% may have bipolar disorder (Spencer et al., 2000; Wozniak et al., 1995)—a figure substantially higher than the 1% risk for the general population (Lewinsohn, Klein, & Seeley, 1995). Follow-up studies, have not documented any significant increase in risk of bipolar disorder in children with ADHD followed into adulthood (Fischer et al., in press-b; Mannuzza et al., 1993, 1998; Weiss & Hechtman, in press); however, that risk would have to exceed 7% for these studies to have sufficient power to detect any comorbidity. A 4-year follow-up of children with ADHD reported that 12% met criteria for bipolar disorder in adolescence (Biederman, Faraone, Milberger, et al., 1996). Children with ADHD but without bipolar disorder do not have an increased prevalence of bipolar disorder among their biological relatives (Biederman et al., 1992; Faraone, Biederman, & Monuteaux, 2001; Lahey et al., 1988), whereas children with both ADHD and bipolar disorder do (Faraone et al., 1997, 2001); this suggests that where the overlap occurs, it may represent a familially distinct subset of ADHD. Children and adolescents diagnosed with childhood bipolar disorder often have a significantly higher lifetime prevalence of ADHD, particularly in their earlier childhood years (Carlson, 1990; Geller & Luby, 1997). Where the two disorders coexist, the onset of bipolar disorder may be earlier than in bipolar disorder alone (Faraone et al., 1997, 2001; Sachs, Baldassano, Truman, & Guille, 2000). Some of this overlap with ADHD may be partly an artifact of similar symptoms in the symptom lists used for both diagnoses (hyperactivity, distractibility, poor judgment, etc.) (Geller & Luby, 1997). In any cse, the overlap of ADHD with bipolar disorder appears to be unidirectional: A diagnosis of ADHD seems not to increase the risk for bipolar disorder, whereas a diagnosis of childhood bipolar disorder seems to dramatically elevate the risk of a prior or concurrent diagnosis of ADHD (Geller & Luby, 1997; Spencer et al., 2000).

Tourette's Disorder and Other Tic Disorders

Up to 18% of children may develop a motor tic in childhood, but this declines to a base rate of about 2% by midadolescence and less than 1% by adulthood (Peterson, Pine, Cohen, & Brook, 2001). Tourette's disorder, a more severe disorder involving multiple motor and vocal tics, occurs in less than 0.4% of the population (Peterson et al., 2001). A diagnosis of ADHD does not necessarily appear to elevate these risks for a diagnosis of tics or Tourette's disorder, at least not in childhood or adolescence (Peterson et al., 2001). Among clinic-referred adults diagnosed with ADHD, there may be a slightly greater occurrence of tic disorders (12%; Spencer et al., 2001). In contrast, individuals with obsessive–compulsive disorder or Tourette's disorder have a marked elevation in risk for ADHD, averaging 48% or more (range = 35–71%; Comings, 2000). Complicating matters is the fact that the onset of ADHD often seems to precede that of Tourette's disorder in cases of comorbidity (Comings, 2000). Yet Pauls et al. (1986) have shown that Tourette's disorder and ADHD occur independently among relatives of those with each disorder; this suggests that a "Berkson's bias" (comorbidity with ADHD leads to clinic referral) may be operating in clinical referrals for Tourette's disorder such that comorbid cases are more likely to get referred.

ASSOCIATED DEVELOPMENTAL AND SOCIAL PROBLEMS

Apart from an increased risk for various psychiatric disorders, children and teens with ADHD-C are also more likely to experience a substantial array of developmental, social, and health risks; these are discussed in this and the next section. Far less is known about the extent to which these correlated problems are evident in ADHD-PI, particularly the subgroup having problems with sluggish cognitive tempo described above. The various types of problems most likely to occur in children with ADHD-C are briefly listed in Table 2.2.

Motor Incoordination

As a group, as many as 60% of children with ADHD, compared to up to 35% of normal children, may have poor motor coordination or developmental coordination disorder (Barkley, DuPaul, & McMurray, 1990; Hartsough & Lambert, 1985; Kadesjo & Gillberg, 2001; Szatmari et al., 1989; Stewart, Pitts, Craig, & Dieruf, 1966). Neurological examinations for "soft" signs related to motor coordination and motor overflow movements find children with ADHD to demonstrate more such signs (as well as generally sluggish gross motor movements) than control children, including those with "pure" learning disabilities (Carte, Nigg, & Hinshaw, 1996; Denckla & Rudel, 1978; Denckla, Rudel, Chapman, & Krieger, 1985; McMahon & Greenberg, 1977). These overflow movements have been interpreted as indicators of delayed development of motor inhibition (Denckla et al., 1985).

Studies using tests of fine motor coordination, such as balance assessment, tests of fine motor gestures, electronic or paper-and-pencil mazes, and pursuit tracking, often find children with ADHD to be less coordinated in these actions (Hoy, Weiss, Minde, & Cohen, 1978; Mariani & Barkley, 1997; McMahon & Greenberg, 1977; Moffitt, 1990; Shaywitz & Shaywitz, 1985; Ullman, Barkley, & Brown, 1978). Simple motor speed, as measured by finger-tapping rate or grooved pegboard tests, does not seem to be as affected in ADHD as is the execution of complex, coordinated sequences of motor movements (Barkley, Murphy, & Kwasnik, 1996a; Breen, 1989; Grodzinsky & Diamond, 1992; Mariani & Barkley, 1997; Marcotte & Stern, 1997; Seidman, Benedict, et al., 1995: Seidman, Biederman, et al., 1995). The bulk of the available evidence therefore supports the existence of deficits in motor control, particularly when motor sequences must be performed, in those with ADHD.

Impaired Academic Functioning

The vast majority of clinic-referred children with ADHD have difficulties with school performance, most often underproductivity. Such children frequently score lower than normal or control groups of children on standardized achievement tests (Barkley, DuPaul, & McMurray, 1990; Fischer, Barkley, Edelbrock, & Smallish, 1990; Hinshaw, 1992, 1994). These differences are likely to be found even in preschool-age children with ADHD (Barkley, Shelton, et al., 2002; Mariani & Barkley, 1997), suggesting that the disorder may take a toll on the acquisition of academic skills and knowledge even before entry into first grade. This makes sense, given that some of the executive functions believed to be disrupted by ADHD in the model presented earlier are also likely to be involved in some forms of academic achievement (e.g., working memory in mental arithmetic or spelling; internalized speech in reading comprehension; verbal fluency in oral narratives and written reports, etc.).

Between 19% and 26% of children with ADHD are likely to have any single type of learning disability, conservatively defined as a significant delay in reading, arithmetic, or spelling relative to intelligence and achievement in one of these three areas at or below the 7th percentile (Barkley, 1990). If a learning disability is defined as simply a significant discrepancy between intelligence and achievement, then up to 53% of hyperactive children could be said to have such a disability (Lambert & Sandoval, 1980). Or, if the criterion of simply two grades below grade level is used, then as many as 80% of children with ADHD in late childhood (age 11 years) may have learning disorders (Cantwell & Baker, 1992). Studies suggest that the risk for reading disorders among children with ADHD is 16–39%, while that for spelling disorders is 24–27% and for math disorders is 13–33% (August & Garfinkel, 1990; Barkley, 1990; Casey, Rourke, & Del Dotto, 1996; Frick et al., 1991; Semrud-Clikeman et al., 1992).

Although the finding that children with ADHD are more likely to have learning disabilities

TABLE 2.2. Summary of Impairments Likely to Be Associated with ADHD

Cognitive
Mild deficits in intelligence (approximately 7–10 points below average)
Deficient academic achievement skills (range of 10–30 standard score points below average)
Learning disabilities: Reading (8–39%), spelling (12–26%), math (12–33%), and handwriting (common but unstudied)
Poor sense of time; inaccurate time estimation and reproduction
Decreased nonverbal and verbal working memory
Impaired planning ability
Reduced sensitivity to errors
Possible impairment in goal-directed behavioral creativity (??)

Language
Delayed onset of language (up to 35%, but not consistent)
Speech impairments (10–54%)
Excessive conversational speech (commonplace); reduced speech to confrontation
Poor organization and inefficient expression of ideas
Impaired verbal problem solving
Co-existence of central auditory processing disorder (minority, but still uncertain)
Poor rule-governed behavior
Delayed internalization of speech (30+% delay)
Diminished development of moral reasoning

Adaptive functioning: 10–30 standard score points below normal

Motor development
Delayed motor coordination (up to 52%)
More neurological "soft" signs related to motor coordination and overflow movements
Sluggish gross motor movements

Emotion
Poor self-regulation of emotion
Greater problems with frustration tolerance
Underreactive arousal system

School performance
Disruptive classroom behavior (commonplace)
Underperforming in school relative to ability (commonplace)
Academic tutoring (up to 56%)
Repeating a grade (30% or more)
Placement in one or more special education programs (30–40%)
School suspensions (up to 46%)
School expulsions (10–20%)
Failure to graduate from high school (10–35%)

Task performance
Poor persistence of effort/motivation
Greater variability in responding
Decreased performance/productivity under delayed rewards
Greater problems when delays are imposed within the task and as they increase in duration
Decline in performance as reinforcement changes from being continuous to intermittent
Greater disruption when non-contingent consequences occur during the task

Medical/health risks
Greater proneness to accidental injuries (up to 57%)
Possible delay in growth during childhood
Difficulties surrounding sleeping (up to 30–60%)
Greater driving risks: Vehicular crashes and speeding tickets

Note. Adapted from Barkley (1998).

(Gross-Tsur, Shalev, & Amir, 1991; Tannock & Brown, 2000) might imply a possible genetic link between the two disorders, more recent research (Doyle, Faraone, DuPre, & Biederman, 2001; Faraone et al., 1993; Gilger, Pennington, & DeFries, 1992) shows that the two sets of disorders are transmitted independently in families. Some subtypes of reading disorders associated with ADHD may share a common genetic etiology (Gilger et al., 1992). This may arise from the finding that early ADHD may predispose children toward certain types of reading problems, whereas early reading problems do not generally give rise to later symptoms of ADHD (Chadwick, Taylor, Taylor, Heptinstall, & Danckaerts, 1999; Rabiner et al., 2000; Velting & Whitehurst, 1997; Wood & Felton, 1994). The picture is less clear for spelling disorders; a common or shared genetic etiology to both ADHD and spelling disorder has been shown in a joint analysis of twin samples from London and Colorado (Stevenson, Pennington, Gilger, DeFries, & Gillis, 1993). This may result from the fact that early spelling ability seems to be linked to the integrity of working memory (Mariani & Barkley, 1997; Levy & Hobbes, 1989), which may be impaired in those with ADHD (see the discussion of the theoretical model, above). Writing disorders have not received as much attention in research on ADHD, though handwriting deficits are often found among children with ADHD, particularly those having ADHD-C (Marcotte & Stern, 1997).

Rapport, Scanlan, and Denney (1999) provide some evidence for a dual-pathway model of the link between ADHD and academic underachievement. Briefly, ADHD may predispose to academic underachievement through its contribution to a greater risk for ODD/CD and conduct problems in the classroom more generally, the net effect of which is an adverse impact on productivity and general school performance. But ADHD is associated with cognitive deficits not only in attention, but general intelligence (see below) and working memory (see above), all of which may have a direct and adverse impact on academic achievement. Supportive of this view as well are findings that the inattention dimension of ADHD is more closely associated with academic achievement problems than is the hyperactive–impulsive dimension (Faraone, Biederman, Weber, & Russell, 1998; Hynd, Lorys, et al. 1991; Marshall et al., 1997). According to this dual-pathway model, both pathways will require interventions if the marked association of ADHD with school underachievement is to be addressed.

A higher prevalence of speech and language disorders has also been documented in many studies of children with ADHD, typically ranging from 30% to 64% of the samples (Gross-Tsur et al., 1991; Hartsough & Lambert, 1985; Humphries, Koltun, Malone, & Roberts, 1994; Szatmari et al., 1989; Taylor et al., 1991). The converse is also true: Children with speech and language disorders have a higher than expected prevalence of ADHD (approximately 30–58%), among other psychiatric disorders (see Tannock & Brown, 2000, for a review on comorbidity with ADHD).

Reduced Intelligence

Clinic-referred children with ADHD often have lower scores on intelligence tests than control groups used in these same studies, particularly in verbal intelligence (Barkley, Karlsson, & Pollard, 1985; Mariani & Barkley, 1997; McGee et al., 1992; Moffitt, 1990; Stewart et al., 1966; Werry, Elkind, & Reeves, 1987). Differences in IQ have also been found between hyperactive boys and their normal siblings (Halperin & Gittelman, 1982; Tarver-Behring, Barkley, & Karlsson, 1985; Welner, Welner, Stewart, Palkes, & Wish, 1977). The differences found in these studies often range from 7 to 10 standard score points. Studies using both community samples (Hinshaw, Morrison, Carte, & Cornsweet, 1987; McGee et al., 1984; Peterson et al., 2001) and samples of children with behavior problems (Sonuga-Barke et al., 1994) also have found significant negative associations between degree of ADHD and intelligence (r's = $-.25$ – $-.35$). In contrast, associations between ratings of conduct problems and intelligence in children are often much smaller or even nonsignificant, particularly when hyperactive–impulsive behavior is partialed out of the relationship (Hinshaw et al., 1987; Lynam, Moffitt, & Stouthamer-Loeber, 1993; Sonuga-Barke et al., 1994). This implies that the relationship between IQ and ADHD is not likely to be a function of comorbid conduct problems (see Hinshaw, 1992, for a review).

Social Problems

ADHD is classified in DSM-IV as an "attention-deficit and disruptive behavior disorder" because of the significant difficulties it creates in social

conduct and general social adjustment. The interpersonal behaviors of those with ADHD, as noted earlier, are often characterized as more impulsive, intrusive, excessive, disorganized, engaging, aggressive, intense, and emotional. And so they are "disruptive" of the smoothness of the ongoing stream of social interactions, reciprocity, and cooperation, which is an increasingly important part of the children's daily life with others (Whalen & Henker, 1992).

Research finds that ADHD affects the interactions of children with their parents, and hence the manner in which parents may respond to these children (Johnston & Mash, 2001). Those with ADHD are more talkative, negative and defiant; less compliant and cooperative; more demanding of assistance from others; and less able to play and work independently of their mothers (Barkley, 1985; Danforth et al., 1991; Gomez & Sanson, 1994; Johnston, 1996; Johnston & Mash, 2001). Their mothers are less responsive to the questions of their children, more negative and directive, and less rewarding of their children's behavior (Danforth et al., 1991; Johnston & Mash, 2001). Mothers of children with ADHD have been shown to give both more commands and more rewards to sons with ADHD than to daughters with the disorder (Barkley, 1989b; Befera & Barkley, 1984), but also to be more emotional and acrimonious in their interactions with sons (Buhrmester, Camparo, Christensen, Gonzalez, & Hinshaw, 1992; Taylor et al., 1991). Children and teens with ADHD seem to be nearly as problematic for their fathers as their mothers (Buhrmester et al., 1992; Edwards et al., 2001; Johnston, 1996; Tallmadge & Barkley, 1983). Contrary to what may be seen in normal mother–child interactions, the conflicts between children and teens with ADHD (especially boys) and their mothers may actually increase when fathers join the interactions (Buhrmester et al., 1992; Edwards et al., 2001). Such increased maternal negativity and acrimony toward sons in these interactions has been shown to predict greater noncompliance in classroom and play settings and greater covert stealing away from home, even when the level of the sons' own negativity and parental psychopathology are statistically controlled for in the analyses (Anderson et al., 1994). The negative parent–child interaction patterns also occur in the preschool age group (Cohen, Sullivan, Minde, Novak, & Keens, 1983; DuPaul, McGoey, Eckert, & VanBrakle, 2001) and may be even more negative and stressful (to the parents) in this age range (Mash & Johnston, 1982, 1990) than in later age groups. With increasing age, the degree of conflict in these interactions lessens, but remains deviant from normal into later childhood (Barkley, Karlsson, & Pollard, 1985; Mash & Johnston, 1982) and adolescence (Barkley, Anastopoulos, Guevremont, & Fletcher, 1992; Barkley, Fischer, Edelbrock, & Smallish, 1991; Edwards et al., 2001). In families of children with ADHD, negative parent–child interactions in childhood have been observed to be significantly predictive of continuing parent–teen conflicts 8–10 years later in adolescence (Barkley, Fischer, et al., 1991). Few differences are noted between mothers' interactions with their children who have ADHD and their interactions with the siblings of these children (Tarver-Behring et al., 1985).

The presence of comorbid ODD is associated with the highest levels of interaction conflicts between parents and their ADHD children and adolescents (Barkley, Anastopoulos, et al., 1992; Barkley, Fischer, et al., 1991; Edwards et al., 2001; Johnston, 1996). In a sequential analysis of these parent–teen interaction sequences, investigators have noted that the immediate or first lag in the sequence is most important in determining the behavior of the other member of the dyad (Fletcher, Fischer, Barkley, & Smallish, 1996). That is, the behavior of each member is determined mainly by the immediately preceding behavior of the other member, and not by earlier behaviors of either member in the chain of interactions. The interactions of the comorbid ADHD/ODD group reflected a strategy best characterized as "tit for tat," in that the type of behavior (positive, neutral, or negative) of each member was most influenced by the same type of behavior emitted immediately preceding it. Mothers of teens with ADHD only and of normal teens were more likely to utilize positive and neutral behaviors regardless of the immediately preceding behavior of their teens; this has been characterized as a "be nice and forgive" strategy, which is thought to be more mature and more socially successful for both parties in the long run (Fletcher et al., 1996). Even so, those with ADHD alone are still found to be deviant from normal in these interaction patterns, though less so than the comorbid ADHD/ODD group. The presence of comorbid ODD has also been shown to be associated with greater maternal stress and psychopathology, as well as parental marital/couple difficulties (Barkley, Anastopoulos, et al.,

1992; Barkley, Fischer, et al., 1991; Johnston & Mash, 2001).

These interaction conflicts in families of children with ADHD are not limited to parent–child interactions. Increased conflicts have been observed between children with ADHD and their siblings, relative to normal child–sibling dyads (Mash & Johnston, 1983a; Taylor et al., 1991). Research on the larger domain of family functioning has shown that families of children with ADHD experience more parenting stress and decreased sense of parenting competence (Fischer, 1990; Johnston & Mash, 2001; Mash & Johnston, 1990); increased alcohol consumption in parents (Cunningham, Benness, & Siegel, 1988; Pelham & Lang, 1993); decreased extended family contacts (Cunningham et al., 1988); and increased marital/couple conflict, separations, and divorce, as well as maternal depression (Befera & Barkley, 1984; Cunningham et al., 1988; Barkley, Fischer, et al., 1990; Johnston & Mash, 2001; Lahey et al., 1988; Taylor et al., 1991). Again, the comorbid association of ADHD with ODD or CD is linked to even greater degrees of parental psychopathology, marital/couple discord, and divorce than is ADHD only (Barkley, Fischer, et al., 1990, 1991; Lahey et al., 1988; Taylor et al., 1991). Interestingly, Pelham and Lang (1993) have shown that the increased alcohol consumption in these parents is in part a direct function of their stressful interactions with their children with ADHD.

Research has demonstrated that the primary direction of effects within these interactions is from child to parent (Danforth et al., 1991; Johnston & Mash, 2001; Mash & Johnston, 1990), rather than the reverse. That is, much of the disturbance in the interaction seems to stem from the effects of the child's excessive, impulsive, unruly, noncompliant, and emotional behavior on the parent, rather than from the effects of the parent's behavior on the child. This was documented primarily through studies that evaluated the effects of stimulant medication on the behavior of such children and their interaction patterns with their mothers. Such research found that medication improves the compliance of those with ADHD and reduces their negative, talkative, and generally excessive behavior, so that their parents reduce their levels of directive and negative behavior as well (Barkley & Cunningham, 1979b; Barkley, Karlsson, Pollard, & Murphy, 1985; Danforth et al., 1991; Humphries, Kinsbourne, & Swanson, 1978). These effects of medication are noted even in preschool-age children with ADHD (Barkley, 1988) as well as in those in late childhood (Barkley et al., 1985), and in children of both sexes (Barkley, 1989b). Besides a general reduction in the negative, disruptive, and conflictual interaction patterns between children with ADHD and their parents as a result of stimulant medication, general family functioning also seems to improve when these children are treated with stimulant medication (Schachar, Taylor, Weiselberg, Thorley, & Rutter, 1987). None of this is to say that parental reactions to disruptive child behavior, parental skill and competence in child management and daily rearing, and parental psychological impairment are unimportant influences on children with ADHD. Evidence certainly shows that parental management, child monitoring, parental antisocial activity, maternal depression, father absence, and other parent and family factors are exceptionally important in the development of ODD, CD, major depression, ad other disorders likely to be comorbid with ADHD (Johnson, Cohen, Kasen, Smailes, & Brook, 2001; Johnston & Mash, 2001; Pfiffner, McBurnett, & Rathouz, 2001; Patterson et al., 2000). But it must be emphasized, as the behavioral genetic studies described below strongly attest, that these are not the origins of the impulsive, hyperactive, and inattentive behaviors or the related deficits in executive functioning and self-regulation.

The patterns of disruptive, intrusive, excessive, negative, and emotional social interactions that have been found between children with ADHD and their parents have been found to occur in the children's interactions with teachers (Whalen, Henker, & Dotemoto, 1980) and peers (Clark, Cheyne, Cunningham, & Siegel, 1988; Cunningham & Siegel, 1987; DuPaul et al., 2001; Whalen, Henker, Collins, McAuliffe, & Vaux, 1979). It should come as no surprise, then, that those with ADHD receive more correction, punishment, censure, and criticism than other children from their teachers, as well as more school suspensions and expulsions, particularly if they have ODD/CD (Barkley, Fischer, et al., 1990; Whalen et al., 1980). In their social relationships, children with ADHD are less liked by other children, have fewer friends, and are overwhelmingly rejected as a consequence (Erhardt & Hinshaw, 1994), particularly if they have comorbid conduct problems (Gresham, MacMillan, Bocian, Ward, & Forness, 1998; Hinshaw & Melnick, 1995). Indeed, among such comorbid cases, up to 70%

may be rejected by peers and have no reciprocated friendships by fourth grade (Gresham et al., 1998). These peer relationship problems are the results not only of these children's more active, talkative, and impulsive actions, but also of their greater emotional, facial, tonal, and bodily expressiveness (particularly anger), more limited reciprocity in interactions, use of fewer positive social statements, more limited knowledge of social skills, and more negative physical behavior (Casey, 1996; Erhardt & Hinshaw, 1994; Grenel, Glass, & Katz, 1987; Madan-Swain & Zentall, 1990). Those with ODD/CD also prefer more sensation-seeking, fun-seeking, and trouble-seeking activities, which further serve to alienate their normal peers (Hinshaw & Melnick, 1995; Melnick & Hinshaw, 1996). Furthermore, children with ADHD seem to process social and emotional cues from others in a more limited and error-prone fashion, as if they were not paying as much attention to emotional information provided by othrs. Yet they do not differ in their capacity to understand the emotional expressions of other children (Casey, 1996). However, in those with comorbid ODD/CD, there may be a greater misperception of anger and a greater likelihood of responding with anger and aggression to peers than normal children (Cadesky, Mota, & Schachar, 2000; Casey, 1996; Matthys, Cuperus, & van Engeland, 1999). Little wonder, then, that children with ADHD perceive themselves as receiving less social support from peers (and teachers) than do normal children (Demaray & Elliot, 2001). The problems with aggression and poor emotion regulation are also evident in the sports behavior of these children with their peers (Johnson & Rosen, 2000). Once more, stimulant medication has been observed to decrease these negative and disruptive behaviors toward teachers (Whalen et al., 1980) and peers (Cunningham, Siegel, & Offord, 1985; Wallander, Schroeder, Michelli, & Gualtieri, 1987; Whalen et al., 1987), but it may not result in any increase in more prosocial or positive initiatives toward peers (Wallander et al., 1987).

HEALTH OUTCOMES

Once again, caution should be used in extending the findings below beyond the ADHD-C subtype, given that very little research exists on the health outcomes of ADHD-PI.

Physical Health

The postnatal course of those with hyperactivity has been shown to be subject to more stress and complications in several studies (Hartsough & Lambert, 1985; Stewart et al., 1966; Taylor et al., 1991). Chronic health problems, such as recurring upper respiratory infections, asthma, and allergies, have also been documented in the later preschool and childhood years of hyperactive children (Hartsough & Lambert, 1985; Mitchell, Aman, Turbott, & Manku, 1987; Szatmari et al., 1989). And children with atopic (allergic) disorders have been shown to have more symptoms of ADHD (Roth, Beyreiss, Schlenzka, & Beyer, 1991). Yet more careful research using better control groups, longitudinal samples, or analysis of the familial aggregation of disorders has not shown a specific association of these disorders with hyperactivity (Biederman, Milberger, Faraone, Guite, & Warburton, 1994; McGee, Stanton, & Sears, 1993; Mitchell et al., 1987; Taylor et al., 1991).

One study suggests that ADHD may be associated with growth deficits, particularly in height, during childhood and early adolescence (Spencer et al., 1996). These deficits did not exist in older adolescents, suggesting that the problem with growth is one of delayed maturation.

Accident-Proneness and Injury

In one of the first studies of the issue, Stewart et al. (1966) found that four times as many hyperactive children as control children (43% vs. 11%) were described by parents as accident-prone. Later studies have also identified such risks; up to 57% of children with hyperactivity or ADHD are said to be accident-prone by parents, relative to 11% or fewer of control children (Mitchell et al., 1987; Reebye, 1997). Interestingly, knowledge about safety does not appear to be lower in overactive, impulsive children than in control children. And so simply teaching more knowledge about safety may not suffice to reduce the accident risks of hyperactive children (Mori & Peterson, 1995).

Most studies find that children with ADHD experience more injuries of various sorts than control children. In one study, 16% of the hyperactive sample had at least four or more serious accidental injuries (broken bones, lacerations, head injuries, severe bruises, lost teeth, etc.),

compared to just 5% of control children (Hartsough & Lambert, 1985). Jensen, Shervette, Xenakis, and Bain (1988) found that 68% of children with DSM-III ADD, compared to 39% of control children, had experienced physical trauma sufficient to warrant sutures, hospitalization, or extensive/painful procedures. Several other studies likewise found a greater frequency of accidental injuries than among control children (Taylor et al., 1991), as did I when I analyzed data from research Terri Shelton and I had done (Shelton et al., 1998) and found that more than four times as many children with ADHD as control children (28.4% vs. 6.4%) had an accident related to their impulsive behavior. One of my own studies, however, did not find a higher proportion of children with ADHD as having accidents (Barkley, DuPaul, & McMurray, 1990). Sample sizes in this study were small, however, and may not have been able to detect moderate to small effect sizes with adequate statistical power.

Head trauma is not overrepresented among children with hyperactivity or ADHD (Stewart et al., 1966; Szatmari et al., 1989). As for burns, only one study of children with ADHD has been done, and it did not find a significantly elevated incidence (2.0% vs. 2.4% for controls) (Szatmari et al., 1989). Bone fractures, in contrast, seem to be somewhat more common in children with ADHD than in control children (23.5% vs. 15.1%) (Szatmari et al., 1989). Children with ADHD may be two to three times more likely to experience accidental poisonings (21% vs. 8% in Stewart, Thach, & Friedin, 1970; 7% vs. 3% in Szatmari et al., 1989). Jensen et al. (1988) found that 13% of children with ADD and 8% of control children had ingested poisonous substances.

Driving Risks and Auto Accidents

The most extensively studied form of accidents occurring among those with hyperactivity or ADHD is motor vehicle crashes. Evidence emerged years ago that hyperactive teens as drivers had a higher frequency of vehicular crashes than control subjects (1.3 vs. 0.07; $p < .05$) (Weiss & Hechtman, 1993). Also noteworthy in their driving histories was a significantly greater frequency of citations for speeding.

Subsequently, my colleagues and I (Barkley, Guevremont, Anastopoulos, DuPaul, & Shelton, 1993) found that teens with ADHD had more crashes as drivers (1.5 vs. 0.4) than did control teens over their first few years of driving. Forty percent of the group with ADHD had experienced at least two or more such crashes, relative to just 6% of the control group. Four times more teens with ADHD were deemed to have been at fault in their crashes as drivers than controls (48.6% vs. 11.1%), and these teens were at fault more frequently than the controls (0.8 vs. 0.4). In keeping with the Weiss and Hechtman (1993) initial report, teens with ADHD were more likely to get speeding tickets (65.7% vs. 33.3%) and got them more often (means = 2.4 vs. 0.6). Two studies in New Zealand using community samples suggest a similarly strong relationship between ADHD and vehicular accident risk (Nada-Raja et al., 1997; Woodward, Fergusson, & Horwood, 2000). Adults diagnosed with ADHD also manifest more unsafe motor vehicle operation and crashes. More adults with ADHD in one study had their licenses suspended (24% vs. 4.0%) than in the control group, and reported having received more speeding tickets (means = 4.9 vs. 1.1) than control adults (Murphy & Barkley, 1996a). The difference in the frequency of vehicular crashes between the groups was only marginally significant (means = 2.8 vs. 1.8, $p < .06$), however.

Later, in a more thorough examination of driving (Barkley, Murphy, & Kwasnik, 1996b), we found that the group with ADHD reported having had more vehicular crashes than the control group (means = 2.7 vs. 1.6), and that a larger proportion of this group had been involved in more severe crashes (resulting in injuries) than the control subjects (60% vs. 17%). Again, speeding citations were overrepresented in the self-reports of the subjects with ADHD (100% vs. 56%) and occurred more frequently in this group than in the control group (means = 4.9 vs. 1.3).

The most thorough study to date of driving performance among young adults with ADHD (Barkley, Murphy, DuPaul, & Bush, 2002) used a multimethod, multisource battery of measures. More than twice as many young adults with ADHD as members of the control group (26% vs. 9%) had been involved in three or more vehicular crashes as drivers, and more had been held at fault in three or more such crashes (7% vs. 3%). The ADHD group had also been involved in more vehicular crashes overall than the control group (means = 1.9 vs. 1.2) and had been held to be at fault in more crashes (means = 1.8 vs. 0.9). The dollar damage caused in their first accidents

was estimated to be more than twice as high in the ADHD group as in the control group (means = $4,221 vs. $1,665). As in the earlier studies, the group with ADHD reported a greater frequency of speeding citations (3.9 vs. 2.4), and a higher percentage had had their licenses suspended than in the control group (22%vs. 5%). Both the greater frequency of speeding citations and license suspensions were corroborated through the official state driving records for these young adults.

These studies leave little doubt that ADHD, or its symptoms of inattention and hyperactive–impulsive behavior, are associated with a higher risk for unsafe driving and motor vehicle accidents than in the normal population. In view of the substantial costs that must be associated with such a higher rate of adverse driving outcomes, prevention and intervention efforts are certainly called for to attempt to reduce the driving risks among those having ADHD.

Sleep Problems

Many studies have suggested an association between ADHD and sleep disturbances (Ball, Tiernan, Janusz, & Furr, 1997; Gruber, Sadeh, & Raviv, 2000; Kaplan, McNichol, Conte, & Moghadam, 1987; Stewart et al., 1966; Trommer, Hoeppner, Rosenberg, Armstrong, & Rothstein, 1988; Wilens, Biederman, & Spencer, 1994). The problems are mainly more behavioral problems at bedtime, a longer time to fall asleep, instability of sleep duration, tiredness at awakening, or frequent night waking. For instance, Stein (1999) compared 125 psychiatrically diagnosed children with 83 pediatric outpatient children and found moderate to severe sleep problems in 19% of those with ADHD, 13% of the psychiatric controls, and 6% of pediatric outpatients. Treatment with stimulant medication increased the proportion of children with ADHD and sleep problems to 29%—a not unexpected finding, given the well-known stimulant side effect of increased insomnia (see Barkley, 1998). Sleep electroencephalograms (EEGs) have typically not revealed differences in the quality of sleeping, however (Ball & Kolonian, 1995). Other research implies that the comorbid disorders (ODD, anxiety disorders, etc.) associated with ADHD may contribute to the increased risk for some of these sleep problems (Corkum, Beig, Tannock, & Moldofsky, 1997). Indeed, a later study by Corkum and associates (Corkum, Moldofsky, Hogg-Johnson, Humphries, & Tannock, 1999) found that sleep problems occurred twice as often in ADHD than in control children. These problems could be reduced to three general factors: (1) dyssomnias (bedtime resistance, sleep onset problems, or difficulty arising); (2) sleep-related involuntary movements (teeth grinding, sleeptalking, restless sleep, etc.); and (3) parasomnias (sleep walking, night wakings, sleep terrors). Dyssomnias were primarily related to comorbid ODD or treatment with stimulant medication, whereas parasomnias were not significantly different from the control group. However, involuntary movements were significantly elevated in children with ADHD-C.

Within normal populations, quantity of sleep is inversely associated with an increased risk for school behavioral problems (Aronen, Paavonen, Fjallnerg, Soinen, & Torronen, 2000), particularly daytime sleepiness and inattention rather than hyperactive–impulsive behavior (Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001). The direction of effect, then, between ADHD and sleep problems is unclear. It is possible that sleep difficulties increase ADHD symptoms during the daytime, as the research on normal children implies. Yet some research finds that the sleep problems of children with ADHD are not associated with the severity of their symptoms; this suggests that the disorder, not the impaired sleeping, is what contributes to impaired daytime alertness, inattention, and behavioral problems (Lecendreux, Konofal, Bouvard, Falissard, & Mouren-Simeoni, 2000).

ETIOLOGIES

Since the first edition of this text was published, considerable research has accumulated on various etiologies for ADHD. Notably, virtually all of this research pertains to the ADHD-C subtype, or what was previously considered hyperactivity in children. Readers should not extend these findings to the ADHD-PI subtype, especially the subset noted above to have sluggish cognitive tempo and (probably) a qualitatively different disorder. But for ADHD-C, there is even less doubt now among career investigators in this field that although the disorder may have multiple etiologies, neurological and genetic factors are likely to play the greatest role in causing it. These two areas, along with the associated field of the neuropsychology of ADHD, have witnessed enormous growth in the past decade, further

refining our understanding of the neurogenetic basis of the disorder. Our knowledge of the final common neurological pathway through which these causes produce their effects on behavior has become clearer from converging lines of evidence employing a wide array of assessment tools, including neuropsychological tests sensitive to frontal lobe functioning; electrophysiological measures (EEG, quantitative EEG [QEEG], and evoked response potentials [ERPs); measures of cerebral blood flow; and neuroimaging studies using positron emission tomography (PET), magnetic resonance imaging (MRI), and functional MRI. Several recent studies have even identified specific protein abnormalities in specific brain regions that may be linked to possible neurochemical dysregulation in the disorder. Precise neurochemical abnormalities that may underlie this disorder have proven extremely difficult to document with any certainty over the past decade, but advancing psychopharmacological, neurological, and genetic evidence suggests involvement in at least two systems—the dopaminergic and noradrenergic systems. Neurological evidence is converging on a highly probable neurological network for ADHD, as discussed below. Nevertheless, most findings on etiologies are correlational in nature and do not provide direct, precise, immediate molecular evidence of primary causality. But then that is the case for all psychiatric disorders (and, indeed, many medical ones as well), so ADHD is in good company. In fact, our understanding of causal factors here may be far more advanced than is the case in most other psychopathologies of childhood.

Neurological Factors

Various neurological etiologies have been proposed for ADHD. Brain damage was initially proposed as an initial and chief cause of ADHD symptoms (Still, 1902), whether it occurred as a result of known brain infections, trauma, or other injuries or complications occurring during pregnancy or at the time of delivery (see Barkley, 1998, for more on the history of ADHD). Several studies show that brain damage, particularly hypoxic/anoxic types of insults, is associated with greater attention deficits and hyperactivity (Cruickshank, Eliason, & Merrifield, 1988; O'Dougherty, Nuechterlein, & Drew, 1984). ADHD symptoms also occur more often in children with seizure disorders (Holdsworth & Whitmore, 1974) that are clearly related to underlying neurological malfunction. However, most children with ADHD have no history of significant brain injuries or seizure disorders, and so brain damage is unlikely to account for the majority of children with ADHD (Rutter, 1977).

Throughout the century, investigators have repeatedly noted the similarities between symptoms of ADHD and those produced by lesions or injuries to the frontal lobes more generally and the prefrontal cortex specifically (Barkley, 1997b; Benton, 1991; Heilman et al., 1991; Levin, 1938; Mattes, 1980). Both children and adults suffering injuries to the prefrontal region demonstrate deficits in sustained attention, inhibition, regulation of emotion and motivation, and the capacity to organize behavior across time (Fuster, 1997; Grattan & Eslinger, 1991; Stuss & Benson, 1986).

Neuropsychological Studies

Much of the neuropsychological evidence pertaining to ADHD has been reviewed above in relation to the particular forms of cognitive impairment seen in ADHD, especially as regards the theory described earlier. A large number of studies have used neuropsychological tests of frontal lobe functions and have detected deficits on these tests, albeit inconsistently (Barkley, Edwards, et al., 2001; Conners & Wells, 1986; Chelune, Ferguson, Koon, & Dickey, 1986; Fischer et al., 1990; Heilman et al., 1991; Mariani & Barkley, 1997; Murphy et al., 2001; Seidman, Biederman, Faraone, et al., 1997). I have reviewed much of this literature up to 1997 (Barkley, 1997b), but it has nearly doubled in volume since that time. Where consistent, the results suggest that poor inhibition of behavioral responses, or what Nigg (2001) has called "executive inhibition," is solidly established as impaired in this disorder, at least the ADHD-C and ADHD-PHI types. As noted earlier, evidence has mounted for difficulties as well with nonverbal and verbal working memory, planning, verbal fluency, response perseveration, motor sequencing, sense of time, and other frontal lobe functions. Adults with ADHD have also been shown to display similar deficits on neuropsychological tests of executive functions (Barkley, Murphy, & Bush, 2001; Murphy et al., 2001; Seidman, Biederman, Faraone, et al., 1997). One recent study of adults found diminished olfactory identification in adults with ADHD—a finding predicted on the basis of the fact that both executive functions

and olfactory identification are mediated by prefrontal regions (Murphy et al., 2001).

Moreover, recent research shows not only that do siblings of children with ADHD who also have ADHD show similar executive function deficits, but even that siblings who do not actually manifest ADHD themselves appear to have milder yet significant impairments in these same excutive functions (Sedman, Biederman, Weber, Monuteaux, & Faraone, 1997). Such findings imply a possible genetically linked risk for executive function deficits in families of children with ADHD, even if symptoms of ADHD are not fully manifested in those family members. Supporting this implication is evidence that the executive deficits in ADHD arise from the same substantial shared genetic liability as do the ADHD symptoms themselves and as does the overlap of ADHD with ODD/CD (Coolidge et al., 2000). Important in recent studies in this area has been the demonstration that these inhibitory and executive deficits are not the result of comorbid disorders, such as ODD, CD, anxiety, or depression, thus giving greater confidence to their affiliation with ADHD itself (Barkley, Edwards, et al., 2001; Barkley, Murphy, & Bush, 2001; Bayliss & Roodenrys, 2000; Chang et al., 1999; Clark et al., 2000; Klorman et al., 1999; Murphy et al., 2001; Nigg et al., 1998; Oosterlaan et al., in press; Wiers et al., 1998). This is not to say that some other disorders, such as learning disabilities or autism, do not affect some executive function tasks, such as those of verbal working memory, perhaps owing to their associated deficits in language development; still, the pattern of deficits associated with ADHD is not typical of these other disorders (Pennington & Ozonoff, 1996). The totality of findings in the neuropsychology of ADHD is impressive in further suggesting that some dysfunction of the prefrontal lobes (inhibition and executive function deficits) is involved in this disorder.

Neurological Studies

Early research in the 1960s and 1970s focused on psychophysiological measures of nervous system (central and autonomic) electrical activity, variously measured (EEGs, galvanic skin responses, heart rate deceleration, etc.). These studies were inconsistent in demonstrating group differences between children with ADHD and control children in resting arousal. But where differences from normal were found, they were consistently in the direction of diminished reactivity to stimulation, or arousability, in those with ADHD (see Hastings & Barkley, 1978, for a review). Recent research continues to demonstrate differences in skin conductance and heart rate parameters in response to stimulation in those with ADHD (Borger & van der Meere, 2000), which may distinguish them from children with CD or those with comorbid ADHD and CD (Beauchaine et al., 2001; Herpertz et al., 2001).

Far more consistent have been the results of QEEG and ERP measures, sometimes taken in conjunction with vigilance tests (Frank, Lazar, & Seiden, 1992; Klorman, 1992; Klorman, Salzman, & Borgstedt, 1988; Rothenberger, 1995). Although results have varied substantially across these studies (see Tannock, 1998, for a review), the most consistent pattern for EEG research is increased slow-wave or theta activity, particularly in the frontal lobe, and excess beta activity, all indicative of a pattern of underarousal and underreactivity in ADHD (Baving, Laucht, & Schmidt, 1999; Chabot & Serfontein, 1996; Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996; Monastra, Lubar, & Linden, 2001). Children with ADHD have been found to have smaller amplitudes in the late positive and negative components of their ERPs. These late components are believed to be a function of the prefrontal regions of the brain, are related to poorer performances on inhibition and vigilance tests, and are corrected by stimulant medication (Johnstone, Barry, & Anderson, 2001; Pliszka, Liotti, & Woldorff, 2000; Kuperman et al., 1996). Thus psychophysiological abnormalities related to sustained attention and inhibition indicate an underresponsiveness of children with ADHD to stimulation that is corrected by stimulant medication.

Several studies have also examined cerebral blood flow using single-photon emission computed tomography (SPECT) in children with ADHD and normal children (see Tannock, 1998, and Hendren, DeBacker, & Pandina, 2000, for reviews). They have consistently shown decreased blood flow to the prefrontal regions (most recently in the right frontal area), and to pathways connecting these regions with the limbic system via the striatum and specifically its anterior region known as the caudate, and with the cerebellum (Gustafsson, Thernlund, Ryding, Rosen, & Cederblad, 2000; Lou, Henriksen, & Bruhn, 1984; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989; Sieg, Gaffney, Preston, & Hellings, 1995). Degree of blood flow in the right frontal region has

been correlated with behavioral severity of the disorder, while that in more posterior regions and the cerebellum seems related to degree of motor impairment (Gustafsson et al., 2000).

Within the last few years, a radioactive chemical ligand known as [I^{123}] Altropane has been developed that binds specifically to the dopamine transporter protein in the striatum of the brain, and thus can be used to indicate level of dopamine transporter activity within this region. Following intravenous injection of the ligand, SPECT is used to detect the binding activity of Altropane in the striatum. The dopamine transporter is responsible for the reuptake of extracellular dopamine from the synaptic cleft after neuronal release. Several pilot studies found that adults with ADHD had significantly increased binding potential of Altropane and thus greater dopamine transporter activity (Dougherty et al., 1999; Krause, Dresel, Krause, Kung, & Tatsch, 2000). A third pilot study replicated this difference in binding potential and found that degree of transporter activity was significantly associated with severity of ADHD symptoms, but not with comorbid anxiety or depression (Barkley et al., 2002). These findings are interesting because research suggests that the drug methylphenidate, which is often used to treat ADHD, has a substantial effect on activity in this brain region and may produce its therapeutic effect by slowing down this dopamine transporter activity (Krause et al., 2000; Volkow et al., 2001).

Studies using PET to assess cerebral glucose metabolism have found diminished metabolism in adults with ADHD, particularly in the frontal region (Schweitzer et al., 2000; Zametkin et al., 1990), and in adolescent females with ADHD (Ernst et al., 1994), but have proven negative in adolescent males with ADHD (Zametkin et al., 1993). An attempt to replicate the finding in adolescent females with ADHD in younger female children with ADHD failed to find such diminished metabolism (Ernst, Cohen, Liebenauer, Jons, & Zametkin, 1997). Such studies are plagued by their exceptionally small sample sizes, which result in very low power to detect group differences and considerable unreliability in replicating previous findings. However, significant correlations have been noted between diminished metabolic activity in the anterior frontal region and severity of ADHD symptoms in adolescents with ADHD (Zametkin et al., 1993). Also, using a radioactive tracer that indicates dopamine activity, Ernst et al. (1999) found abnormal dopamine activity in the right midbrain region of children with ADHD, and discovered that severity of symptoms was correlated with the degree of this abnormality. These demonstrations of an association between the metabolic activity of certain brain regions on the one hand, and symptoms of ADHD and associated executive deficits on the other, is critical to proving a connection between the findings pertaining to brain activation and the behaviors constituting ADHD.

More recent neuroimaging technologies offer a more fine-grained analysis of brain structures using the higher-resolution MRI devices. Studies employing this technology find differences in selected brain regions in those with ADHD relative to control groups. Much of the initial work was done by Hynd and his colleagues (see Tannock, 1998, for a review). Initial studies from this group examined the region of the left and right temporal lobes associated with auditory detection and analysis (planum temporale) in children with ADHD, children with reading disorders, and normal children. The first two groups were found to have smaller right-hemisphere plana temporale than the control group, but only the reading-disordered subjects had a smaller left plana temporale (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990). In the next study, the corpus callosum was examined in those with ADHD. This structure assists with the interhemispheric transfer of information. Those with ADHD were found to have a smaller callosum, particularly in the area of the genu and splenium and that region just anterior to the splenium (Hynd, Semrud-Clikeman, et al., 1991). An attempt to replicate this finding, however, failed to show any differences between children with ADHD and control children in the size or shape of the entire corpus callosum, with the exception of the region of the splenium (posterior portion), which again was significantly smaller in the subjects with ADHD (Semrud-Clikeman et al., 1994).

The various brain regions often implicated in ADHD in the most recent MRI research are illustrated in Figure 2.2. Here the right hemisphere of the brain is shown, but the left hemisphere has been cut away to expose the location of the striatum in relation to the prefrontal regions controlling movement specifically and behavior generally.

In a later study by Hynd and colleagues (Hynd et al., 1993), children with ADHD had a significantly smaller left caudate nucleus, creating a reversal of the normal pattern of left > right asym-

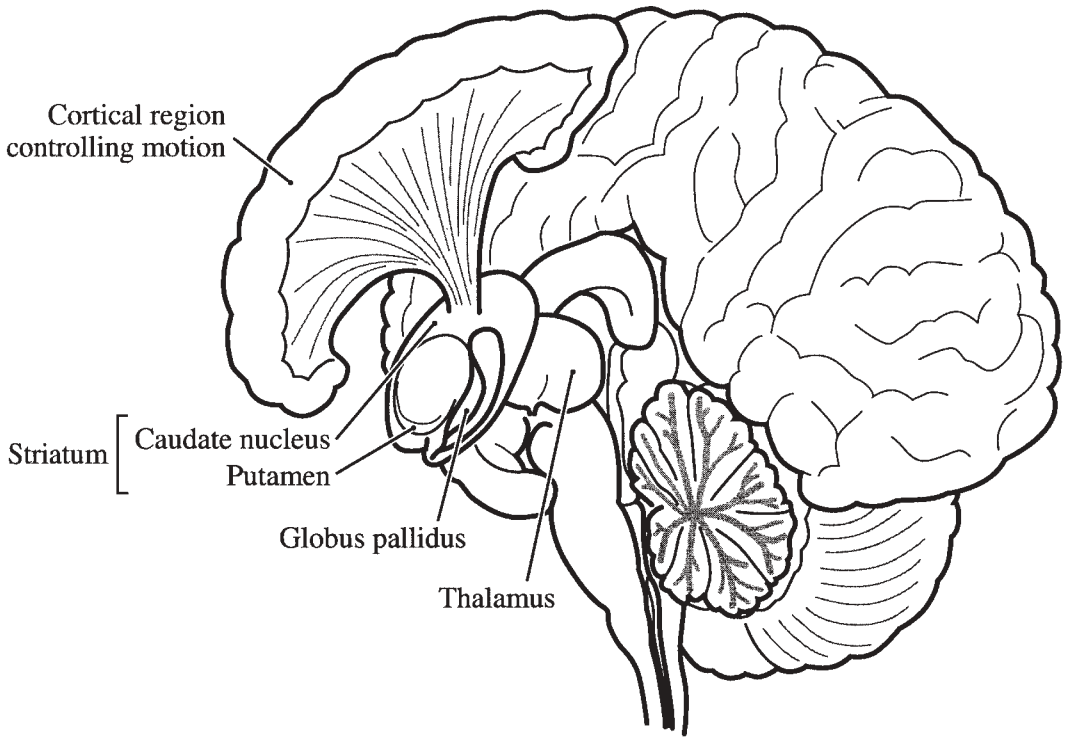

FIGURE 2.2. Diagram of the human brain showing the right hemisphere, and particularly the location of the striatum, globus pallidus, and thalamus. Most of the left hemisphere has been cut away up to the prefrontal lobes to reveal the striatum and other midbrain structures. Adapted from an illustration by Carol Donner in Youdin & Riederer (1997). Copyright 1997 by *Scientific American*. Adapted by permission.

metry of the caudate. This finding is consistent with the earlier blood flow studies of decreased activity in this brain region. Several more recent studies, using quantitative MRI technology, have used larger samples of subjects with ADHD and control subjects. These studies have indicated significantly smaller anterior right frontal regions, smaller size of the caudate nucleus, reversed asymmetry of the head of the caudate, and smaller globus pallidus regions in children with ADHD compared to control subjects (Aylward et al., 1996; Castellanos et al., 1994, 1996; Filipek et al., 1997; Singer et al., 1993). Important as well have been the findings that the size of some of these regions, particularly the structures in the basal ganglia and right frontal lobe, has been shown to correlate with the degree of impairment in inhibition and attention in the children with ADHD (Casey et al., 1997; Semrud-Clikeman et al., 2000). The putamen, however, has not been found to be smaller in children with ADHD (Aylward et al., 1996; Castellanos et al., 1996; Singer et al., 1993), or to be associated with behavioral inhibition deficits in these children (Casey et al., 1997).

Interestingly, the study by Castellanos et al. (1996) also found smaller cerebellar volume in those with ADHD. This would be consistent with recent views that the cerebellum plays a major role in executive functioning and the motor-presetting aspects of sensory perception that derive from planning and other executive actions (Diamond, 2000).

No differences between groups on MRI were found in the regions of the corpus callosum in either of the studies by Castellanos et al. (1994, 1996), as had been suggested in the small studies discussed above or as had been found in a prior study by this same research team (Giedd et al., 1994). However, the study by Filipek et al. (1997) did find smaller posterior volumes of white matter in both hemispheres in the regions of the parietal and occipital lobes, which might be consistent with the earlier studies showing smaller

volumes of the corpus callosum in this same area. Castellanos et al. (1996) suggest that such differences in corpus callosal volume, particularly in the posterior regions, may be more closely related to learning disabilities (which are found in a large minority of children with ADHD) than to ADHD itself.

The results for the smaller size of the caudate nucleus are quite consistent across studies, but are inconsistent in indicating which side of the caudate may be smaller. The work by Hynd et al. (1993) discussed earlier found the left caudate to be smaller than normal in their subjects with ADHD. The more recent studies by Filipek et al. (1997) and Semrud-Clikeman et al. (2000) found the same result. However, Castellanos et al. (1996) also reported a smaller caudate, but found this to be on the right side of the caudate. The normal human brain demonstrates a relatively consistent asymmetry in volume, in favor of the right frontal cortical region's being larger than the left (Giedd et al., 1996). This led Castellanos et al. (1996) to conclude that a lack of frontal asymmetry (a smaller than normal right frontal region) probably mediates the expression of ADHD. However, whether this asymmetry of the caudate (right side > left side) is true in normal subjects is debatable, as other studies found the opposite pattern in their normal subjects (Filipek et al., 1997; Hynd et al., 1993). More consistent across these studies are the findings of smaller right prefrontal cortical regions, smaller caudate volume, and smaller regions of the cerebellar vermis (again, more likely on the right than on the left side).

With the advent of even more advanced MRI technology, researchers can now evaluate functional activity in various brain regions while administering psychological tests to subjects being scanned. These studies find children with ADHD to have abnormal patterns of activation during attention and inhibition tasks than do normal children, particularly in the right prefrontal region, the basal ganglia (striatum and putamen), and the cerebellum (Rubia et al., 1999; Teicher et al., 2000; Vaidya et al., 1998). Again, the demonstrated linkage of brain structure and function with psychological measures of ADHD symptoms and executive deficits is exceptionally important in such research, to permit causal inferences to be made about the role of these brain abnormalities in the cognitive and behavioral abnormalities constituting ADHD.

Neurotransmitter Deficiencies

Possible neurotransmitter dysfunction or imbalances have been proposed in ADHD for quite some time (see Pliszka, McCracken, & Maas, 1996, for a review). Initially, these rested chiefly on the responses of children with ADHD to differing drugs. These children respond remarkably well to stimulants, most of which act by increasing the availability of dopamine via various mechanisms, and by producing some effects on the noradrenergic pathways as well (DuPaul, Barkley, & Connor, 1998). These children also respond well to tricyclic antidepressants, giving further support to a possible noradrenergic basis to ADHD (Connor, 1998). Consequently, it seemed sensible to hypothesize that these two neurotransmitters might be involved in the disorder. The finding that normal children show a positive (albeit lesser) response to stimulants (Rapoport et al., 1978), however, partially undermines this logic. Other, more direct evidence comes from studies of cerebrospinal fluid in children with ADHD and normal children, which indicate decreased brain dopamine in the children with ADHD (Raskin, Shaywitz, Shaywitz, Anderson & Cohen, 1984). Similarly, other studies have used blood and urinary metabolites of brain neurotransmitters to infer deficiencies in ADHD, largely related to dopamine regulation. Early studies of this sort proved conflicting in their results (Shaywitz, Shaywitz, Cohen, & Young, 1983; Shaywitz et al., 1986; Zametkin & Rapoport, 1986). A subsequent study continued to find support for reduced noradrenergic activity in ADHD, as inferred from significantly lower levels of a metabolite of this neurotransmitter (Halperin et al., 1997). The limited evidence from this literature thus seems to point to a selective deficiency in the availability of both dopamine and norepinephrine, but this evidence cannot be considered conclusive at this time.

Pregnancy and Birth Complications

Some studies have not found a greater incidence of pregnancy or birth complications in children with ADHD compared to normal children (Barkley, DuPaul, & McMurray, 1990), whereas others have found a slightly higher prevalence of unusually short or long labor, fetal distress, low forceps delivery, and toxemia or eclampsia (Hartsough & Lambert, 1985; Minde, Webb, &

Sykes, 1968). Nevertheless, though children with ADHD may not experience greater pregnancy complications, prematurity, or lower birthweight as a group, children born prematurely or who have markedly lower birthweights are at high risk for later hyperactivity or ADHD (Breslau et al., 1996; Nichols & Chen, 1981; Schothorst & van Engeland, 1996; Sykes et al., 1997; Szatmari, Saigal, Rosenbaum, & Campbell, 1993). It is not merely low birthweight that seems to pose the risk for symptoms of ADHD or the disorder itself (among other psychiatric disorders), but the extent of white matter abnormalities due to birth injuries, such as parenchymal lesions and/or ventricular enlargement (Whittaker et al., 1997). These findings suggest that although certain pregnancy complications may not be the cause of most cases of ADHD, some cases may arise from such complications, especially prematurity associated with minor bleeding in the brain.

Several studies suggest that mothers of children with ADHD are younger when they conceive these children than are mothers of control children, and that such pregnancies may have a greater risk of adversity (Denson, Nanson, & McWatters, 1975; Hartsough & Lambert, 1985; Minde et al., 1968). Since pregnancy complications are more likely to occur among young mothers, mothers of children with ADHD may have a higher risk for such complications, which may act neurologically to predispose their children toward ADHD. However, the complications that have been noted to date are rather mild and hardly compelling evidence of pre- or perinatal brain damage as a cause of ADHD. Furthermore, large-scale epidemiological studies have generally not found a strong association between pre- or perinatal adversity (apart from prematurity as noted above) and symptoms of ADHD once other factors are taken into account—such as maternal smoking and alcohol use (see below) as well as socioeconomic disadvantage, all of which may predispose offspring to perinatal adversity and hyperactivity (Goodman & Stevenson, 1989; Nichols & Chen, 1981; Werner et al., 1971).

One study found that the season of a child's birth was significantly associated with risk for ADHD, at least among those subgroups of children who either also had a learning disability or did not have any psychiatric comorbidity (Mick, Biederman, & Faraone, 1996). Birth in September was overrepresented in this subgroup of children with ADHD. The authors conjecture that the season of birth may serve as a proxy for the timing of seasonally mediated viral infections to which these mothers and their fetuses may have been exposed, and that such infections may account for approximately 10% of cases of ADHD.

Genetic Factors

Evidence for a genetic basis to this disorders comes from three sources: family studies, twin studies, and (most recently) molecular genetic studies identifying individual candidate genes. Again, nearly all of this research applies to the ADHD-C subtype.

Family Aggregation Studies

For years, researchers have noted the higher prevalence of psychopathology in the parents and other relatives of children with ADHD. Between 10% and 35% of the immediate family members of children with ADHD are also likely to have the disorder, with the risk to siblings being approximately 32% (Biederman et al., 1992; Biederman, Faraone, Keenan, & Tsuang, 1991: Pauls, 1991; Welner et al., 1977). Even more striking is the finding that if a parent has ADHD, the risk to the offspring is 57% (Biederman et al., 1995). Thus, ADHD clusters significantly among the biological relatives of children or adults with the disorder, strongly implying a hereditary basis to this condition. Subsequently, these elevated rates of disorders have been noted in African American samples with ADHD (Samuel et al., 1999) as well as in girls with ADHD compared to boys (Faraone et al., 2000).

These studies of families further suggest that ADHD with CD may be a distinct familial subtype of ADHD. In research separating children with ADHD into those with and without CD, it has been shown that conduct problems, SUDs, and depression in the parents and other relatives are related more to the presence of CD in the children with ADHD than to ADHD itself (August & Stewart, 1983; Biederman, Faraone, Keenan, & Tsuang, 1991; Faraone, Biederman, et al., 1995; Faraone, Biederman, Mennin, Russell, & Tsuang, 1998; Lahey et al., 1988). Rates of hyperactivity or ADHD remain high even in relatives of children with ADHD but not CD (Biederman, Faraone, Keenan, & Tsuang, 1991); however, depression and antisocial spectrum disorders are most likely to appear in the comorbid group. Using sibling pairs in which both siblings had ADHD, Smalley et al. (2000) have

also recently supported this view through findings that CD significantly clusters among the families of only those sibling pairs having CD.

Some research has also suggested that girls who manifest ADHD may need to have a greater genetic loading (higher family member prevalence) than do males with ADHD (Smalley et al., 2000). Faraone et al. (1995) also found some evidence in support of this view, in that male siblings from families with one affected child were more likely to have ADHD than were female siblings from these families. They also reported that the gender difference noted earlier for ADHD (a 3:1 male-to-female ratio) may apply primarily to children from families in which either an affected child or a parent has antisocial behavior.

Interestingly, research by Faraone and Biederman (1997) suggests that depression among family members of children with ADHD may be a nonspecific expression of the same genetic contribution that is related to ADHD. This is based on their findings that family members of children with ADHD are at increased risk for major depression, while individuals having major depression have first-degree relatives at increased risk for ADHD. Even so, as noted above, the risk for depression among family members is largely among those children having ADHD with CD.

Adoption Research

Another line of evidence for genetic involvement in ADHD has emerged from studies of adopted children. Cantwell (1975) and Morrison and Stewart (1973) both reported higher rates of hyperactivity in the biological parents of hyperactive children than in the adoptive parents of such children. Both studies suggest that hyperactive children are more likely to resemble their biological parents than their adoptive parents in their levels of hyperactivity. Yet both studies were retrospective, and both failed to study the biological parents of the adopted hyperactive children as a comparison group (Pauls, 1991). Cadoret and Stewart (1991) studied 283 male adoptees and found that if one of the biological parents had been judged delinquent or had an adult criminal conviction, the adopted-away sons had a higher likelihood of having ADHD. A later study (van den Oord, Boomsma, & Verhulst, 1994), using biologically related and unrelated pairs of international adoptees, identified a strong genetic component (47% of the variance) for high scores on the Attention Problems dimension of the Child Behavior Checklist, a rating scale commonly used in research on ADHD. More recently, a study of three groups of children (adopted children with ADHD, children with ADHD living with their biological parents, and a control group) and their families showed the same pattern of an elevated prevalence of ADHD among just the biological parents of the children with ADHD (6% vs. 18% vs. 3%, respectively) (Sprich, Biederman, Crawford, Mundy, & Faraone, 2000). Thus, like the family association studies discussed earlier, the adoption studies point to a strong possibility of a significant hereditary contribution to hyperactivity.

Twin Studies

Since the first edition of this text, the number of twin studies of ADHD and its underlying behavioral dimensions has increased markedly. More exciting has been the striking consistency across all of these studies. This research strategy provides a third avenue of evidence for a genetic contribution to ADHD. But it also provides a means of testing any competing environmental theories of the disorder (e.g., that ADHD is due to poor parenting, adverse family life, excessive TV viewing, etc.). This is because twin studies can not only compute the proportion of variance in a trait that is genetically influenced (heritability), but also the proportion that results from common or shared environment (things twins and siblings have in common growing up in the same family) and that which results from unique environment (all nongenetic factors or events that are unique or specific to one child and not to others in the family) (Plomin, Defries, McClearn, & Rutter, 1997).

Early research on ADHD using twins looked only at twin concordance (likelihood of twins' sharing the same disorder) and did not compute these estimates of heritability, shared environment, and unique environment. These early studies demonstrated a greater agreement (concordance) for symptoms of hyperactivity and inattention between monozygotic (MZ) twins than between dizygotic (DZ) twins (O'Connor, Foch, Sherry, & Plomin, 1980; Willerman, 1973). Studies of very small samples of twins (Heffron, Martin, & Welsh, 1984; Lopez, 1965) found complete (100%) concordance for MZ twins for hyperactivity, and far less agreement for DZ twins. Gilger et al. (1992) found that if one twin was diagnosed as having ADHD, the concordance for the disorder was 81% in MZ twins and 29% in DZ

twins. Sherman, McGue, and Iacono (1997) found that the concordance for MZ twins having ADHD (mother-identified) was 67%, as opposed to 0% for DZ twins.

Later research has computed heritability and environmental contributions to ADHD. One such study of a large sample of twins (570) found that approximately 50% of the variance in hyperactivity and inattention in this sample was due to heredity, while 0–30% may have been environmental (Goodman & Stevenson, 1989). The relatively limited number of items assessing these two behavioral dimensions, however, may have reduced the sensitivity of the study to genetic effects. Later and even larger twin studies have found an even higher degree of heritability for ADHD, ranging from .75 to .97 (see Levy & Hay, 2001, and Thapar, 1999, for reviews) (Burt et al., 2001; Coolidge et al., 2000; Gjone, Stevenson, & Sundet, 1996; Gjone, Stevenson, Sundet, & Eilertsen, 1996; Levy, Hay, McStephen, Wood, & Waldman, 1997; Rhee, Waldman, Hay, & Levy, 1999; Sherman, Iacono, & McGue, 1997; Sherman, McGue, & Iacono, 1997; Silberg et al., 1996; Thapar et al., 2001; Thapar, Hervas, & McGuffin, 1995; van den Oord, Verhulst, & Boomsma, 1996). Thus twin studies indicate that the average heritability of ADHD is at least .80, being nearly that for human height (.80–.91) and higher than that found for intelligence (.55–.70). These studies consistently find little if any effect of shared (rearing) environment on the traits of ADHD, while sometimes finding a small significant contribution for unique environmental events. In their totality, shared environmental factors seem to account for 0–6% of individual differences in the behavioral trait(s) related to ADHD. This is why I have stated at the opening of this section that little attention is given here to discussing purely environmental or social factors as involved in the causation of ADHD.

The twin studies cited above have also been able to indicate the extent to which individual differences in ADHD symptoms are the result of nonshared environmental factors. Such factors include not only those typically thought of as involving the social environment, but also all biological factors that are nongenetic in origin. Factors in the nonshared environment are those events or conditions that will have uniquely affected only one twin and not the other. Besides biological hazards or neurologically injurious events that may have befallen only one member of a twin pair, the nonshared environment also includes those differences in the manner in which parents may have treated each child. Parents do not interact with all of their children in an identical fashion, and such unique parent–child interactions are believed to make more of a contribution to individual differences among siblings than do those factors about the home and child rearing that are common to all children in the family. Twin studies to date have suggested that approximately 9–20% of the variance in hyperactive–impulsive–inattentive behavior or ADHD symptoms can be attributed to such nonshared environmental (nongenetic) factors (Levy et al., 1997; Sherman, Iacono, & McGue 1997; Silberg et al., 1996). A portion of this variance, however, must be attributed to the error of the measure used to assess the symptoms. Research suggests that the nonshared environmental factors also contribute disproportionately more to individual differences in other forms of child psychopathology than do factors in the shared environment (Pike & Plomin, 1996). Thus, if researchers are interested in identifying environmental contributors to ADHD, these studies suggest that such research should focus on those biological and social experiences that are specific and unique to the individual and are not part of the common environment to which other siblings have been exposed.

Molecular Genetic Research

Although a quantitative genetic analysis of the large sample of families studied in Boston by Biederman and his colleagues suggested that a single gene may account for the expression of the disorder (Faraone et al., 1992), most investigators suspect multiple genes, given the complexity of the traits underlying ADHD and their dimensional nature. The focus of research was initially on the dopamine type 2 gene, given findings of its increased association with alcoholism, Tourette's disorder, and ADHD (Blum, Cull, Braverman, & Comings, 1996; Comings et al., 1991), but others have failed to replicate this finding (Gelernter et al., 1991; Kelsoe et al., 1989). More recently, the dopamine transporter gene (DAT1) has been implicated in two studies of children with ADHD (Cook et al., 1995; Cook, Stein, & Leventhal, 1997; Gill, Daly, Heron, Hawi, & Fitzgerald, 1997). Again, however, other laboratories have not been able to replicate this association (Swanson et al., 1997).

Another gene related to dopamine, the DRD4 (repeater gene), has been the most reliably found

in samples of children with ADHD (Faraone et al., 1999). It is the seven-repeat form of this gene that has been found to be overrepresented in children with ADHD (Lahoste et al., 1996). Such a finding is quite interesting, because this gene has previously been associated with the personality trait of high novelty-seeking behavior; because this variant of the gene affects pharmacological responsiveness; and because the gene's impact on postsynaptic sensitivity is primarily found in frontal and prefrontal cortical regions believed to be associated with executive functions and attention (Swanson et al., 1997). The finding of an overrepresentation of the seven-repeat DRD4 gene has now been replicated in a number of other studies—not only of children with ADHD, but also of adolescents and adults with the disorder (Faraone et al., 1999).

Thyroid Disorder

Resistance to thyroid hormone (RTH) represents a variable tissue hyposensitivity to thyroid hormone. It is inherited as an autosomal dominant characteristic in most cases. It has been associated with mutations in the thyroid hormone beta receptor gene; thus a single gene for the disorder has been identified. One study (Hauser et al., 1993) found that 70% of individuals with RTH had ADHD. Other research has suggested that 64% of patients with RTH display hyperactivity or learning disabilities (Refetoff, Weiss, & Usala, 1993). A later study was not able to corroborate a link between RTH and ADHD, however (Weiss et al., 1993). In a subsequent study, Stein, Weiss, and Refetoff (1995) did find that half of their children with RTH met clinical diagnostic criteria for ADHD. Even so, the degree of ADHD in patients with RTH is believed to be milder than that seen in clinic-referred and diagnosed cases of ADHD. The patients with RTH often have more learning difficulties and cognitive impairments than do the children with ADHD but without RTH. Given that RTH is exceptionally rare in children with ADHD (prevalence of 1:2,500) (Elia et al., 1994), then thyroid dysfunction is unlikely to be a major cause of ADHD in the population. An interesting recent finding is that children with both RTH and ADHD may show a positive behavioral response to liothyronine, with decreased impulsiveness, than do children with ADHD who do not have RTH (Stein, Weiss, & Refetoff, 1995).

Environmental Toxins

As the twin and quantitative genetic studies have suggested, unique environmental events may play some role in individual differences in symptoms of ADHD. This should not be taken to mean only those influences within the realm of psychosocial or family influences. As noted above, variance in the expression of ADHD that may be due to "environmental sources" means all nongenetic sources more generally. These include pre-, peri, and postnatal complications, as well as malnutrition, diseases, trauma, toxin exposure, and other neurologically compromising events that may occur during the development of the nervous system before and after birth. Among these various biologically compromising events, several have been repeatedly linked to risks for inattention and hyperactive behavior.

One such factor is exposure to environmental toxins, specifically lead. Elevated body lead burden has been shown to have a small but consistent and statistically significant relationship to the symptoms of ADHD (Baloh, Sturm, Green, & Gleser, 1975; David, 1974; de la Burde & Choate, 1972, 1974; Needleman et al., 1979; Needleman, Schell, Bellinger, Leviton, & Alfred, 1990). However, even at relatively high levels of lead, fewer than 38% of children in one study were rated as having the behavior of hyperactivity on a teacher rating scale (Needleman et al., 1979), implying that most lead-poisoned children do not develop symptoms of ADHD. And most children with ADHD likewise, do not have significantly elevated lead burdens, although one study indicates that their lead levels may be higher than those of control subjects (Gittelman & Eskinazi, 1983). Studies that have controlled for the presence of potentially confounding factors in this relationship have found the association between body lead (in blood or dentition) and symptoms of ADHD to be .10–.19; the more factors are controlled for, the more likely the relationship is to fall below .10 (Fergusson, Fergusson, Horwood, & Kinzett, 1988; Silva, Hughes, Williams, & Faed, 1988; Thomson et al., 1989). Only 4% or less of the variance in the expression of these symptoms in children with elevated lead is explained by lead levels. Moreover, two serious methodological issues plague even the better-conducted studies in this area: (1) None of the studies have used clinical criteria for a diagnosis of ADHD to determine precisely what percentage of lead-burdened children actually have the disorder (all have simply

used behavior ratings comprising only a small number of items of inattention or hyperactivity); and (2) none of the studies have assessed for the presence of ADHD in the parents and controlled its contribution to the relationship. Given the high heritability of ADHD, this factor alone could attenuate the already small correlation between lead and symptoms of ADHD by as much as a third to a half of its present leels.

Other types of environmental toxins found to have some relationship to inattention and hyperactivity are prenatal exposure to alcohol and tobacco smoke (Bennett, Wolin, & Reiss, 1988; Denson et al., 1975; Milberger, Biederman, Faraone, Chen, & Jones, 1996a; Nichols & Chen, 1981; Shaywitz, Cohen, & Shaywitz, 1980; Streissguth et al., 1984; Streissguth, Bookstein, Sampson, & Barr, 1995). It has also been shown that mothers of children with ADHD do consume more alcohol and smoke more tobacco than control groups even when they are not pregnant (Cunningham et al., 1988; Denson et al., 1975). Thus it is reasonable for research to continue to pursue the possibility that these environmental toxins may be causally related to ADHD. However, most research in this area suffers from the same two serious methodological limitations as the lead studies discussed above: the failure to utilize clinical diagnostic criteria to determine rates of ADHD in exposed children, and the failure to evaluate and control for the presence of ADHD in the parents. Until these steps are taken in future research, the relationships demonstrated so far between these toxins and ADHD must be viewed with some caution. In the area of maternal smoking during pregnancy, at least, such improvements in methodology were used in a recent study, which found the relationship between maternal smoking during pregnancy and ADHD to remain significant even after symptoms of ADHD in the mothers were controlled for (Milberger et al., 1996a).

Psychosocial Factors

A few environmental theories of ADHD were proposed over 20 years ago (Block, 1977; Willis & Lovaas, 1977), but they have not received much support in the available literature since then. Willis and Lovaas (1977) claimed that hyperactive behavior was the result of poor stimulus control by maternal commands and that this poor regulation of behavior arose from poor parental management of the children. Others have also conjectured that ADHD results from difficulties in the parents' overstimulating approach to caring for and managing the children, as well as parental psychological problems (Carlson, Jacobvitz, & Sroufe, 1995; Jacobvitz & Sroufe, 1987; Silverman & Ragusa, 1992). But these conjectures have not articulated just how the deficits in behavioral inhibition, executive functioning, and other cognitive deficits commonly associated with clinically diagnosed ADHD as described above could arise purely from such social factors. Moreover, many of these studies proclaiming to have evidence of parental characteristics as potentially causative of ADHD have not used clinical diagnostic criteria to identify children as having ADHD; instead, they have relied merely on elevated parental ratings of hyperactivity or laboratory demonstrations of distractibility to classify the children as having ADHD (Carlson et al., 1995; Silverman & Ragusa, 1992). Nor have these purely social theories received much support in the available literature that has studied clinically diagnosed children with ADHD (see Danforth et al., 1991; Johnston & Mash, 2001).

In view of the twin studies discussed above, which show minimal, nonsignificant contributions of the common or shared environment to the expression of symptoms of ADHD, theories based entirely on social explanations of the origins of ADHD are difficult to take seriously any longer. This is not to say that the family and larger social environment do not matter, for they surely do. Despite the large role heredity seems to play in ADHD symptoms, they remain malleable to unique environmental influences and nonshared social learning. The actual severity of the symptoms within a particular context, the continuity of those symptoms over development, the types of comorbid disorders that will develop, the peer relationship problems that may arise, and various outcome domains of the disorder are likely to be related in varying degrees to parental, familial, and larger environmental factors (Johnson et al., 2001; Johnston & Mash, 2001; Milberger, 1997; Pfiffner et al., 2001; van den Oord & Rowe, 1997). Yet even here, care must be taken in interpreting these findings as evidence of a purely social contribution to ADHD. This is because many measures of family functioning and adversity also show a strong heritable contribution to them, largely owing to the presence of the same or similar symptoms and disorders (and genes!) in the parents as in the children (Pike & Plomin, 1996; Plomin, 1995). Thus there is a genetic con-

tribution to the family environment—a fact that often goes overlooked in studies of family and social factors involved in ADHD.

Summary

It should be evident from the research reviewed here that ADHD arises from multiple factors, and that neurological and genetic factors are substantial contributors. Like Taylor (1999), I envision ADHD as having a heterogeneous etiology, with various developmental pathways leading to this behavioral syndrome. These various pathways, however, may give rise to the disorder through disturbances in a final common pathway in the nervous system. That pathway appears to be the integrity of the prefrontal cortical–striatal network. It now appears that hereditary factors play the largest role in the occurrence of ADHD symptoms in children. It may be that what is transmitted genetically is a tendency toward a smaller and less active prefrontal–striatal–cerebellar network. The condition can also be caused or exacerbated by pregnancy complications, exposure to toxins, or neurological disease. Social factors alone cannot be supported as causal of this disorder, but such factors may exacerbate the condition, contribute to its persistence, and (more likely) contribute to the forms of comorbid disorders associated with ADHD. Cases of ADHD can also arise without a genetic predisposition to the disorder, provided that children are exposed to significant disruption of or injury to this final common neurological pathway, but this would seem to account for only a small minority of children with ADHD. In general, then, research conducted since the first edition of this text was published has further strengthened the evidence for genetic and developmental neurological factors as likely causal of this disorder while greatly reducing the support for purely social or environmental factors as having a role. Even so, environmental factors involving family and social adversity may still serve as both exacerbating factors, determinants of comorbidity, and contributors to persistence of disorder over development.

THE INATTENTIVE SUBTYPE

Mounting research on the predominantly inattentive subtype of ADHD (ADHD-PI) suggests that it differs in many important respects from the combined subtype (ADHD-C) of the disorder. Children with the ADHD-C manifest more oppositional and aggressive symptoms, a greater likelihood of having ODD and CD, and more peer rejection than children with ADHD-PI (Crystal et al., 2001; Milich et al., 2001; Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999). Those with ADHD-PI also may have a qualitatively different impairment in attention (selective attention and speed of information processing) (see Milich et al., 2001, for a thorough review). More than twice as many children with ADHD-C as with ADHD-PI were diagnosed as having ODD (41% vs. 19%) in a study using DSM-III-R criteria, and more than three times as many were diagnosed as having CD (21% vs. 6%) (Barkley, DuPaul, & McMurray, 1990). The children with ADHD-C may also be more likely to have speech and language problems (Cantwell & Baker, 1992). Children with ADHD-C are described as more noisy, disruptive, messy, irresponsible, and immature; in contrast, children with ADHD-PI are characterized as more daydreamy, hypoactive, passive, apathetic, lethargic, confused, withdrawn, and sluggish (Edelbrock, Costello, & Kessler, 1984; Lahey, Shaughency, Strauss, & Frame, 1984; Lahey, Schaughency, Hynd, Carlson, & Nieves, 1987; McBurnett, Pfiffner, & Frick, 2001; Milich et al., 2001). Research suggests that these symptoms of sluggish cognitive tempo in ADHD-PI form a separate dimension of inattention from that in the DSM-IV (McBurnett et al., 2001), which may have resulted in their being prematurely discarded from the DSM-IV inattention list (Milich et al., 2001). A recent study by Carlson and Mann (2002) indicates that if the subset of children with ADHD-PI characterized by sluggish cognitive tempo are separated from children with this subtype who are not so characterized, then greater problems with anxiety/depression, social withdrawal, and general unhappiness and fewer problems with externalizing symptoms may be more evident in this former subset.

Social passivity and withdrawal have been reported in other studies of children with ADHD-PI as well, when parent and teacher ratings of social adjustment are used (Maedgen & Carlson, 2000; Milich et al., 2001). Direct observations of the peer interactions of these subtypes tend to corroborate these ratings, finding that children with ADHD-C are more prone to fighting and arguing, whereas children with ADHD-PI are more shy (Hodgens, Cole, & Boldizar, 2000).

Research using objective tests and other lab measures has met with mixed results in identifying consistent distinctions between these subtypes. When measures of academic achievement and neuropsychological functions have been used, most studies have found no important differences between the groups (Carlson, Lahey, & Neeper, 1986; Casey et al., 1996; Lamminmaki, Aohen, Narhi, Lyytinen, & Todd de Barra, 1995); both groups have been found to be more impaired in academic skills and in some cognitive areas than normal control children. A more recent study suggests that children with ADHD-C are more impaired in response inhibition (Nigg, Blaskey, Huang-Pollack, & Rappley, 2002), but otherwise manifest comparable deficits on executive function tasks. As in many studies of this issue, however, sample sizes were low, so that statistical power may have compromised the sensitivity of the study to all but large effect sizes. Hynd and colleagues (Hynd, Lorys, et al., 1991; Morgan, Hynd, Riccio, & Hall, 1996) found greater academic underachievement, particularly in math, and a higher percentage of learning disabilities (60%) in their samples of children with ADHD-PI compared to children with ADHD-C. My colleagues and I, however, were not able to find any differences between the subtypes on measures of achievement or in rates of learning disabilities (Barkley, 1990). Nor were Casey et al. (1996) able to find such differences in achievement or rates of learning disabilities, using the same means to define the subtypes and to classify children as learning-disabled. Both groups of children with ADHD were impaired in their academic achievement. Our own study also found both subtypes to have been retained in grade (32% in each group), and placed in special education considerably more often than our normal control children (45% vs. 53%). We did find that children with ADHD-C were more likely to have been placed in special classes for behavior-disordered children (emotionally disturbed) than children with ADHD-PI (12% vs. 0%), whereas the children with ADHD-PI were more likely to be in classes for learning-disabled children than the children with ADHD-C (53% vs. 34%). Others have also found that children with ADHD-PI needed more remedial assistance in school than children with ADHD-C (Faraone, Biederman, Weber, & Russell, 1998). We have found that both groups seem to have equivalent rates of learning disabilities, but that the additional problems with conduct and antisocial behavior are likely to result in the children with ADHD-C being assigned to the programs for behavioral disturbance rather than the programs for learning disabilities. Only one study has examined handwriting problems among subtypes of children with ADHD (Marcotte & Stern, 1997); these were found to be greatest in children with ADHD-C, but present to some extent in children with ADHD-PI compared to control children.

Unfortunately, few of these studies have directly addressed the issue of whether these subtypes differ in the components of attention they disrupt. This would require a more comprehensive and objective assessment of different components of attention in both groups. But the results of some studies suggest that their attentional disturbances are not identical (see Milich et al., 2001). Children with ADHD-PI may have more deficits on tests of selective or focused attention (such as the Coding subtest of the Wechsler Intelligence Scale for Children—Revised), problems in the consistent retrieval of verbal information from memory, and even more visual–spatial deficits than children with ADHD-C (Barkley, DuPaul, & McMurray, 1990; Garcia-Sanchez, Estevez-Gonzalez, Suarez-Romero, & Junque, 1997; Johnson, Altmaier, & Richman, 1999). Children with ADHD-C, in contrast, have more problems with motor inhibition, sequencing, and planning (Barkley, Grodzinsky, & DuPaul, 1992; Marcotte & Stern, 1997; Nigg et al., 2002). These findings intimate a qualitative difference in the attention deficits of children with ADHD-PI, which may fall more in the realms of perceptual–motor speed and central cognitive processing speed.

Studies of family psychiatric disorders are also limited and inconsistent. Some have found children with ADHD-C to have families with greater discord between their parents, and more maternal psychiatric disorders generally (Cantwell & Baker, 1992). We found a greater history of ADHD among the paternal relatives and of SUDs among the maternal relatives of children with ADHD-C (Barkley, DuPaul, & McMurray, 1990). In contrast, Frank and BenNun (1988) did not find such differences in family histories. Moreover, we noted a significantly greater prevalence of anxiety disorders among the maternal relatives of children with ADHD-PI, which was not reported by the Frank and BenNun study. That finding, however, also was not replicated in another study of family history (Lahey & Carlson, 1992), suggesting that anxiety disorders may not

be more common among the relatives of children with ADHD-PI.

In general, these results suggest that children with ADHD-PI and those with ADHD-C have considerably different patterns of psychiatric comorbidity. Children with ADHD-C are at significantly greater risk for ODD and CD, academic placement in programs for behaviorally disturbed children, school suspensions, and psychotherapeutic interventions than are children with ADHD-PI. The research also appears to indicate that children with ADHD-PI can be distinguished in a number of domains of social adjustment from those with ADHD-C. Cognitive differences are less consistently noted, but this may have to do with sample selection procedures in which the children with ADHD-PI are chosen solely on the basis of the DSM inattention list, rather than focusing more on symptoms of sluggish cognitive tempo (which are not represented in that list). *Based on the evidence available to date, I concur with Milich et al. (2001) that we should begin considering these two subtypes as actually separate and unique childhood psychiatric disorders, and not as subtypes of an identical attention disturbance.*

A survey (Szatmari et al., 1989) indicates that the prevalence of these two disorders within the general population is different, especially in the childhood years (6–11 years of age). ADHD-PI appeared to be considerably less prevalent than ADHD-C in this epidemiological study. Only 1.4% of boys and 1.3% of girls had ADHD-PI, whearas 9.4% of boys and 2.8% of girls had ADHD-C. These figures changed considerably in the adolescent age groups, where 1.4% of males and 1% of females had ADHD-PI, while 2.9% of males and 1.4% of females had ADHD-C. In other words, the rates of ADHD-PI remained relatively stable across these developmental age groupings, whereas ADHD-C (especially in males) showed a considerable decline in prevalence with age. Among all children with either type, about 78% of boys and 63% of girls had ADHD-C. Baumgaertel, Wolraich, and Dietrich (1995) found a considerably higher prevalence rate for ADHD-PI among German school children. According to the DSM-III definitions for these subtypes, 3.2% had ADD without hyperactivity (corresponding to ADHD-PI), while 6.4% had ADD with hyperactivity (corresponding to ADHD-C). In contrast, when the more recent DSM-IV criteria for subtyping were employed, 9% percent of the children met criteria for ADHD-PI, while 8.8% fell into the ADHD-PHI and ADHD-C categories. The differences in these studies are difficult to reconcile, as both employed rating scales to define their subtypes. However, the Szatmari et al. (1989) study did not use DSM symptom lists but constructed their subtypes based on rating scale items, whereas Baumgaertel et al. (1995) employed symptom lists from the past three versions of the DSM.

It remains to be seen just how stable ADHD-PI is over development. No follow-up studies have focused on this subtype of ADHD, and so the long-term risks associated with it remain unknown.

FUTURE DIRECTIONS

A number of the issues raised in this chapter point the way to potentially fruitful research. The theoretical model discussed above, alone, suggests numerous possibilities for studying working memory; time and its influence over behavior; the internalization of language; creativity and fluency; the self-regulation of affect and motivation; and motor fluency in those with ADHD. Such research will not only be theory-driven, but should have the laudable outcome of linking studies of a child psychopathological condition with the larger literature of developmental psychology, developmental neuropsychology, information processing, and behavior analysis—linkages already being examined in a general way for commonalities among their paradigms and findings (Lyon, 1995).

Certainly, the diagnostic criteria developed to date, even though the most rigorous and empirical ever provided, may still suffer from problems. The fact that such criteria are not theory-driven and developmentally referenced, despite being empirically derived, risks creating several difficulties for understanding the disorder and clinically applying these criteria. Among these are the following: (1) Apparent developmental declines in the disorder and its symptoms may be more illusion than fact; (2) subtypes of a disorder are created that may simply be developmental stages of the same disorder (ADHD-PHI and ADHD-C) or are different disorders entirely (ADHD-PI); (3) female subjects may be underidentified, given that current criteria were developed predominantly from male populations; and (4) a criterion for pervasiveness that confounds the source of

information with its setting may be resulting in overly restrictive criteria. These are just a few of the difficulties.

Important in future research will be efforts to understand the nature of the attentional problems in ADHD, given that extant research seriously questions whether these problems are actually within the realm of attention at all, and that the subtypes of ADHD may have qualitatively different attentional disturbances. Most studies point to impairment within the motor or output systems of the brain rather than the sensory processing systems in ADHD-C; this is not as evident in ADHD-PI. The theoretical model presented here hypothesizes that even this supposed problem with sustained attention represents a deficiency in a more complex, developmentally later form of goal-directed persistence associated with working memory and executive functioning. It arises out of poor self-regulation, rather than representing a disturbance in the more basic and traditional form of sustained responding that is contingency-shaped and maintained. Our understanding of the very nature of the disorder of ADHD is at stake in how research comes to resolve these issues.

That the field of behavioral and especially molecular genetics offers exciting prospects for future research on ADHD goes without saying. Evidence available to date shows a strong hereditary influence in the behavior patterns constituting ADHD, as well as the clinical disorder itself. As of this writing, the race seems to be on to identify the very genes that give rise to it. Such exciting prospects also exist within the domain of neurobiological and neuroimaging studies, in view of present (albeit limited) evidence that diminished metabolic activity and even minute structural differences in brain morphology within highly specific regions of the prefrontal and midbrain systems may be associated with this disorder. The increasing availability, economy, safety, and sensitivity of modern neuroimaging devices should result in a plethora of new studies on ADHD, given the promising starts to date.

Key to understanding ADHD may be the notion that it is actually a disorder of performance, rather than skill; of how one's intelligence is applied in everyday effective adaptive functioning, rather than intelligence itself; of doing what you know, rather than knowing what to do; and of when, rather than how, in the performance of behavior generally. The concept of time, how it is sensed, and particularly how one uses it in self-regulation may come to be critical elements in our understanding of ADHD, as they are coming to be in our understanding of the unique role of the prefrontal cortex more generally (Fuster, 1997). Likewise, the study of how events are mentally represented and prolonged in working memory, and of how private thought arises out of initially public behavior through the developmental process of internalization, are likely to hold important pieces of information for the understanding of ADHD itself. And as the evolutionary (adaptive) purposes of the prefrontal lobes and the executive functions they mediate come to be better understood (Barkley, 2001c), it is highly likely that these findings will yield a rich vein of insights into the sorts of adaptive deficits caused by ADHD.

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