

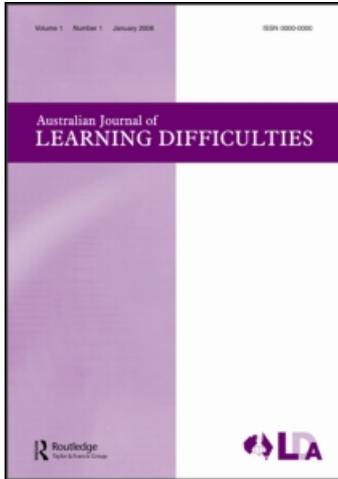
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A contemporary review of the assessment, diagnosis and treatment of ADHD

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Attention-deficit/hyperactivity disorder (ADHD) is a neuro-behavioural disorder that presents frequently in paediatric and mental health settings. Children with ADHD present with symptoms of hyperactivity, inattention, distractibility and impulsivity. The purpose of this paper is to provide clinicians with a summary of contemporary issues relevant to their clinical understanding, assessment, diagnosis and treatment of the disorder. Issues discussed include the primary and secondary symptoms of the disorder, developmental course, underlying causes, recognition in a clinical setting, diagnosis and treatment.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is the most frequently occurring behavioural disorder in childhood (American Academy of Pediatrics, 2000). It represents 26% of all referrals in paediatric clinical settings in Australia (Hewson et al., 1999). The purpose of this paper is to provide clinicians and other readers with a summary of contemporary issues relevant to their clinical understanding, assessment, diagnosis and treatment of the disorder.

Primary features of ADHD

The primary features of ADHD reflect hyperactivity, impulsivity, inattention and distractibility (Burns, Boe, Walsh, Sommers-Flanagan, & Teegarden, 2001; Du Paul, Power, Anastopoulos, & Reid, 1998).

Inattention/distractibility. The symptoms of inattention relate to persistence of effort or sustaining vigilance/effort on tasks (Newcorn et al., 2001). Difficulties with persistence are most salient in structured situations, such as independent schoolwork, where the child has to complete dull, repetitive tasks (Barkley, Du Paul, & McMurray, 1990; Shelton et al., 2000).

Another symptom associated with the inattentive construct has been termed 'distractibility'. Children with ADHD are often reported by parents and teachers to respond more readily to stimuli external to the task at hand than children without

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ADHD. What underlies this problem is still open for debate. However, current thinking suggests the problem may be found in a diminished capacity to inhibit behavioural responses to competing (and more rewarding) activities, rather than true problems with attention (Barkley, 1997, 2006a; Hoza, Pelham, Waschbusch, Kipp, & Owens, 2001).

Impulsivity/hyperactivity. The symptoms of hyperactivity can be manifested physically or verbally. Individuals with ADHD are therefore described as exhibiting excessive motor activity (either overt hyperactivity such as climbing or running inappropriately) or simply as being restless (squirming and fidgeting). They are also described as talking excessively and having difficulty playing quietly. The symptoms of impulsivity are generally manifested as failures in delaying gratification.

It is now common to refer to the impulsive and hyperactive symptoms that are the defining feature of ADHD as a deficit in behavioural inhibition (e.g. Barkley, 1997; 2006a; Nigg, 2001; Pennington & Ozonoff, 1996). Those with ADHD find it more difficult than other children to control and regulate their behaviour and to delay and inhibit their responses to environmental stimuli. They are therefore seen as overly impulsive, they have difficulty delaying gratification and working toward long-term goals and they have difficulty inhibiting inappropriate motor movements.

Behavioural disinhibition in those with ADHD may also underlie the inattentive symptoms of the disorder, such as distractibility (Barkley, 2006b). That is, the child with ADHD may not have a problem with attention per se. However, compared to a child without ADHD, they may be less able to inhibit a response to a more attractive or rewarding stimulus and therefore appear more inattentive.

Theories of ADHD

The heterogeneity of ADHD has led researchers to develop theories that have attempted to go beyond the descriptive, theoretical approach typified by the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV: American Psychiatric Association, 2000). At this time, there is no single universally accepted theory, although theories that refer to constructs such as behavioural inhibition (Barkley, 1997; Quay, 1997), working memory (Pennington, 1994), regulation of arousal/cognitive energy (Sergeant, 2000; Todd & Botteron, 2001), delay aversion (Sonuga-Barke, 2002) and aspects of the attentional networks (Posner & Petersen, 1990) are prominent.

These theories are briefly reviewed below, beginning with arguably the most prominent: Barkley's (1997) Unified Theory. Barkley's theory arose from three lines of experimental evidence: (1) that, beyond the descriptive symptoms of inattention and hyperactivity-impulsivity, almost all the cognitive deficits associated with the disorder fell into the category of self-regulation or executive functions (EF) (Barkley, 2006e); (2) earlier research into the neuropsychological function of the brain's pre-frontal lobes showed that behavioural inhibition and other EFs were mediated by these brain regions (e.g. Bronowski, 1977; Fuster, 1989); and (3) increasing evidence suggesting that ADHD might arise from deficiencies in the development, structure and function of the pre-frontal cortex and its networks with other brain areas.

Barkley (1997, 2006e) proposed that the initial EF of sensorimotor inhibition is an almost uniquely human trait. The development of inhibition permits young

children to put a pause between an event and a response and to selectively choose a response to sensory stimuli (Denckla, 2007), in other words, to stop and think. Sensorimotor or *behavioural* inhibition to which Barkley refers, provides the substrate for additional EF such as selective attention (Diamond, 2000); working memory; emotional regulation; arousal and activation mechanisms; and planning functions (Barkley, 2006e; Denckla, 2007). In the model, inhibition is the core deficit, which leads to 'downstream' deficits in the other EF. Barkley has suggested that ADHD represents a developmental delay of up to 30% in behavioural inhibition and the other EF (e.g. a 12-year-old child could be expected to have the self-regulatory capacity of a 9-year-old).

Others perceive the relation between inhibition and the other EFs somewhat differently, although readers should note the tendency for researchers to use distinct terminology to describe non-mutually exclusive constructs. For example, Pennington, Bennetto, McAleer and Roberts (1996) proposed deficits in the working memory system to be primary in ADHD. This model positions inhibition as a separate, but related, cognitive process that is disrupted by working memory deficits. However, Pennington et al.'s model includes inhibition as a component of working memory and it would follow that impairments in inhibition would adversely affect working memory.

The Cognitive Energetic Model (e.g. Sergeant, 2000) has used a model of information processing and its associated cognitive energetic model (arousal, activation and effort) in an attempt to identify the locus of the ADHD deficit within that paradigm (Sergeant, 2000). This model suggests that there may be some aspects of inhibition deficient in individuals with ADHD, but that external display of ADHD symptoms may also derive from low levels of cognitive arousal and deficits in the ability to recruit and allocate cognitive resources to tasks. The Delay Aversion hypothesis (e.g. Sonuga-Barke, 2002; Sonuga-Barke, Taylor, & Hepinstall, 1992; Sonuga-Barke, Taylor, Sembi, & Smith, 1992) takes a different approach in asserting that the impulsive behaviour seen in ADHD might be an attempt to reduce the subjective perception or experience of delay. Impulsivity is seen, not as a consequence of *inability* to inhibit behaviour as Barkley (1997) would suggest, but rather a consequence of a rational *choice* to avoid unpleasant delay. While both hypotheses have received limited support within the literature (e.g. Solanto et al., 2001; and see Luman, Oosterlaan, & Sergeant, 2005 for review), they predict circumstances under which symptoms of ADHD may be displayed (i.e. under circumstances of sub-optimal arousal or extended delay) rather than being theories that explain the heterogeneity of self-regulation deficits seen in ADHD (Barkley, 2006e).

Models of attention have attempted to link the three cognitive processes of attention, namely alerting (i.e. suppression of background neural noise by the suppression of ongoing or irrelevant activity to establish sustained attention), orienting (i.e. mobilising specific neural resources to prepare to establish selective attention) and executive control (i.e. to establish divided attention) to symptoms of ADHD. It has been proposed that these three aspects of attention are related to distinct neural networks, including an anterior network involving brain regions thought to be deficient in ADHD, such as the prefrontal cortex, the anterior cingulate and the basal ganglia (Fernandez-Duque & Posner, 2001; Posner & Dehaene, 1994). This model attempts to link the cognitive processes and neural networks involved in aspects of attention to symptom clusters of ADHD rather than to the disorder as a categorical entity itself.

Still other hypotheses, such as the Dynamic Development theory (Sagvolden, Johansen, Aase, & Russell, 2005) and the Energy Deficiency theory (Todd & Botteron, 2001) implicate hypofunctioning in dopamine pathways. The former proposes that hypofunctioning in three dopamine branches produces altered reinforcement of behaviour, such that only reinforcement delivered in close proximity to a response will be associated with that response, deficient extinction of previously learned behaviour, deficiencies in orienting attention, impairments in motor function, and habitual learning and memory (Sagvolden et al., 2005; see also *Behavioral and Brain Sciences*, 28, 2005 for critical reviews). The latter, Energy Deficiency theory (Todd & Botteron, 2001), proposes that some forms of ADHD are secondary to hypofunctionality in catecholamine projection pathways to prefrontal cortical areas, which results in decreased availability of neuronal energy. Although this model is yet to be tested it proposes an interesting hypothesis, which is commensurate with findings from genetic, structural and functional brain-based research. That is, a disruption of neuronal energy transfer, based on hypofunctionality in catecholamine projection pathways, results in reductions and disorganisation of neuronal function in prefrontal cortical areas, which may in turn lead to the primary symptoms of ADHD and the secondary EF delays.

In addition to the above theories and hypotheses that highlight the primary or secondary symptoms of ADHD or the conditions under which the symptoms may be manifest, there are a number of models that have emphasised the multifactorial etiology and multidimensional nature of ADHD (e.g. Castellanos & Tannock, 2002). These models have attempted to link etiological factors at the genetic level; neurobiological deficits in frontal and related brain circuitry, reward circuitry dysfunction and catecholaminergic dysregulation; psychological constructs such as delay aversion, executive/inhibition deficits and arousal/activation control; and diagnostic subgroups of ADHD and co-morbid disorders. Although these models are as yet untested they probably provide the way forward in describing a coherent pathway from etiology to diagnosis.

Secondary symptoms and executive functions

The one model that currently seems capable of encompassing both the primary and secondary symptoms of ADHD is Barkley's (1997) unified theory. This theory has also been thoroughly tested and supported by laboratory studies (e.g. Houghton, Cordin, Durkin, & Whiting, 2008) and studies conducted in ecologically-valid settings (e.g. Lawrence et al., 2002). As mentioned above, beyond the descriptive symptoms of inattention and hyperactivity-impulsivity, almost all the cognitive deficits associated with the disorder fall into the category of self-regulation or EF.

Executive functions are 'top-down' functions, mediated by the prefrontal cortex (Scheres, Oosterlaan, & Sergeant, 2001) that are often referred to as the overseer or conductor of cognitive operations within the brain. While an attractive explanation, it is overly simplistic and the EFs are better described as providing the infrastructure within which other human cognitions can operate. They provide the support necessary for humans to acquire new skills or to perform or produce already acquired skills and information (Denckla, 2007). A rather homely analogy that nicely encapsulates the interaction between EF and other cognitive and motor domains is as follows: to cook a meal one needs not only the ingredients, but also a recipe to follow. In this analogy the EF are the recipe providing a structure and guide while the other cognitive and motor domains represent the ingredients (Denckla,

2007). Just as a recipe is useless without the necessary ingredients, one cannot eat a cookbook full of recipes.

Barkley (1997) places executive function deficits (EFD) at the cognitive core of ADHD. Clinically, the following features and consequences can be observed:

Reduced verbal and non-verbal working memory capacity. Working memory is an EF that controls the mechanisms of short-term memory. The ability to perform memory tasks and complex operations that require memory is often weak in individuals with ADHD (Cornish, Wilding, & Grant, 2006). The deficits in working memory usually affect the ability to perform complex tasks; hence individuals with ADHD often have difficulty with reading comprehension even when oral language skills and word-reading are typical; they have trouble performing mathematical problem-solving tasks even when they have the knowledge of mathematical facts and operations to perform the required algorithms; they have difficulty learning new vocabulary and abstract academic concepts; they make place-finding errors and have difficulty maintaining a coherent set during performance of complex tasks; and, despite often having adequate short-term memory capacity, they can have difficulty organising and coding information sufficiently which leads to errors in memory recall.

Working memory dysfunction often contributes to apparent deficits in hindsight and forethought. Individuals with ADHD may not learn as well from previous behaviour or consequences and they find it more difficult to plan and regulate future behaviour on the basis of previous successful or unsuccessful experiences. The ability to monitor task performance and to make changes in response to error or feedback can also be delayed. Put another way; individuals with ADHD seem to repeat the same mistakes.

Reduced working memory capacity may also affect the individual's sense of time (Barkley, 2006e). They have difficulty planning behaviour across time and using time to manage task performance. Children who fail to consider time and the future before acting show less goal-directed behaviour, particularly for long-term goals. They are also more likely to be motivated by smaller, shorter, more immediate goals.

Working memory dysfunction may also make the child with ADHD less able to utilise internally represented information, such as rules, teacher/parent expectations and social rules, to guide behaviour.

Reduced self-regulation of emotion/motivation. Individuals with ADHD experience the same emotions as other individuals, however, they have more difficulty inhibiting the external expression of those emotions (Barkley, 2006e). In other words, they are less emotionally inhibited.

When they are not responding impulsively, individuals with ADHD often have difficulty initiating action. They often require assistance to prompt them to begin tasks. Once started, tasks often remain incomplete due to the combined effects of difficulty sustaining mental arousal, working memory deficits and inhibitory deficits that lead to distraction.

Individuals with ADHD tend towards having more difficulty than others in using internal mechanisms to motivate and drive behaviour. They tend to be more influenced by the potential of immediate and external rewards. In a classroom setting an individual with ADHD is most likely to respond to, or be distracted by, the stimulus that is potentially the most rewarding. In the absence of an alternative external reward for completing a standard academic task (e.g. mathematical problems; see Wright [2007] for an example of how to provide structure of academic

tasks for students with ADHD) it is to be expected that the student will shift their attention to a more rewarding stimulus (e.g. the noise made by a peer).

Poor planning and problem solving. Effective performance of complex tasks requires considerable EF demands. Beyond inhibiting responses to irrelevant stimuli and inhibiting impulsive response to the task itself, one has to first deconstruct the task and develop a coherent strategy for performance. This EF may operate in much the same way as a Managing Director of a business who might manage an incoming job from a client by breaking it down and allocating sections of work to different work teams, by setting deadlines for each piece of work, by recruiting new team members if sufficient skill is not available within the business, by checking and monitoring ongoing performance and making changes where necessary, and by finally bringing the job to completion by reconstituting the individual pieces.

Individuals with ADHD often display dysfunction in these complex problem-solving skills. They tend to respond without planning and tend to have difficulty inhibiting and changing inefficient strategies during the course of the task. Not only does this EFD affect academic performance but it may adversely affect social problem solving. Because replication of complex motor sequences often requires the individual to deconstruct the sequence into individual movements, EFD may also underlie the co-occurrence of motor coordination weaknesses in ADHD.

Co-morbidity. As a group, children with ADHD are more likely to have co-morbid psychiatric disorders than their peers. Between 12 and 35% of children with ADHD are likely to have a co-existing anxiety disorder (Angold, Costello, & Erkanli, 1999; Biederman, Newcorn, & Sprich, 1991; Tannock, 2000). Symptoms of depression are often elevated in those with ADHD (Treuting & Hinshaw, 2001) and studies have reported that 9–32% of children with ADHD have a co-morbid mood disorder (e.g. Biederman et al., 1991).

Attention-deficit/hyperactivity disorder also co-occurs with oppositional and conduct disordered behaviours. The average across studies is that at least 55% of children with ADHD also meet criteria for Oppositional Defiant Disorder (ODD) (Biederman et al., 1991). Conduct Disorder (CD) rarely occurs alone in children with ADHD; being more likely to exist in the context of ODD (Barkley, 2006c). For example, Pfflner et al. (1999) found that just 1% of a sample of boys with ADHD had ADHD+CD, whereas 43% of the sample had ADHD+ODD+CD.

Up to 40% of children with ADHD have significant academic difficulties (Frick et al., 1991). Many children with ADHD also have problems with coordination, reduced physical fitness and a higher incidence of developmental co-ordination disorder is observed. Children with ADHD have little or no elevated risk for tic disorders or an autism spectrum disorder (Barkley, 2006c; Peterson, Pine, Cohen, & Brook, 2001). However, up to 60% of individuals with Tourette's Syndrome have ADHD (Comings, 2000) and up to 59% of children on the autistic spectrum have co-morbid ADHD (Goldstein & Schwebach, 2004).

Prevalence and course

The prevalence rates for ADHD vary greatly in the literature. This is at least partly because of changing diagnostic criteria over time, but also because of differences across studies in the instrument used to define deviance, the requirement for

teacher/parent agreement and the sample (e.g. community versus referred). Three recent reviews of the prevalence literature have placed prevalence rates at between 5 and 10% (Faraone, Sergeant, Gillberg, & Biederman, 2003; Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007; Skounti, Philalithis, & Galankis, 2007). The only two studies of prevalence in Australia have placed rates in a similar range of 6.8 to 9.9% (Gomez, Harvey, Quick, Scharer, & Harris, 1999; Graetz, Sawyer, Hazell, Arney, & Baghurst, 2001). The National Health and Medical Research Council (1997) report on ADHD reported prevalence rates of 2.3 to 6%.

Gender differences in prevalence across studies put the ratio at 3.4:1 males to females (Barkley, 2006a). Girls and boys present with similar symptoms. However, symptom severity may be less in girls and girls are less likely to manifest aggressive behaviour and may experience a reduced risk of depression. Clinically, girls may manifest hyperactivity verbally more so than boys.

Although ADHD was once thought to abate with age, it is now known that symptoms and impairment persist (Fischer, Barkley, Smallish, & Fletcher, 2004; Barkley, Anastopoulos, Guevremont, & Fletcher, 1991; Weiss & Hechtman, 1993). There may be symptom reduction, but 70% to 80% of children with ADHD are likely to continue to have the disorder into adolescence and adulthood (e.g. Barkley et al., 1991; Fischer et al., 2004; Weiss & Hechtman, 1993). As many as half of these children will display oppositional, anti-social or conduct disordered behaviour (Biederman et al., 1996) and many will have significant academic skill deficits (e.g. Fischer, Barkley, Edelbrock, & Smallish, 1990). There is also an increased risk of car accidents, accidental injury (Barkley, Guevremont, Anastopolous, DuPaul, & Shelton, 1993; Weiss & Hechtman, 1993) and substance abuse (Barkley, Fischer, Edelbrock, & Smallish, 1990).

Etiology

Evidence from a range of imaging, pharmacologic and psychophysical studies implicates structures in frontostriatal cortex, the basal ganglia and the cerebellum vermis in the pathophysiology of the disorder (Barkley, 2006d). A clear role for the neurotransmitters dopamine and norepinephrine has been established and genetic factors play an important role (Rappley, 2005). There is also a role for unique environmental events, such as exposure to lead, low birth weight, maternal smoking pregnancy complication and post-natal brain trauma (Barkley, 2006d; Rappley, 2005).

Recognition and diagnosis

An evaluation for ADHD should be initiated when a child presents with distractibility or inattentiveness, hyperactivity, impulsivity, disruptive behaviour, academic underachievement relative to general cognitive ability, poor self-esteem or problems with developing and maintaining social relationships (American Academy of Pediatrics, 2000). Consensus guidelines (American Academy of Pediatrics, 2000; National Health & Medical Research Council, 1997) require the child to meet the diagnostic criteria specified in the DSM-IV and its text revision (American Psychiatric Association, 2000). The DSM-IV-TR makes a distinction between three subtypes of ADHD:

- (1) Predominantly Inattentive (ADHD-PI)
- (2) Predominantly Hyperactive-Impulsive (ADHD-PHI)
- (3) Combined Type (ADHD-C)

Meeting symptom criteria is not enough for a diagnosis of ADHD. Additional criteria state that symptoms must begin prior to seven years of age and have been present for at least six months. Importantly, the symptoms must also be developmentally deviant and produce clinically significant impairment across at least two settings (home, school, social). As a final qualifier, the DSM-IV-TR requires that the symptoms cannot be better accounted for by another mental disorder.

Although the merits of the DSM-IV-TR criteria are not for debate in this paper, clinicians should be aware that a number of issues have been raised in the literature. For example, there is little or no research supporting six months as a critical time frame for duration of symptoms. This is particularly so in young children, for whom symptoms of over activity and inattention have a high likelihood of remission within 12 months (Campbell, 1990; Palfrey, Levine, Walker, & Sullivan, 1985). Many experts have proposed that the duration of symptoms would be better set at 12 months in order to improve accuracy of diagnosis (e.g. Barkley, 2006e).

The requirement for symptom pervasiveness across at least two settings is also problematic. Research has shown that the degree of teacher-parent agreement on a number of behavioural dimensions is modest ($r = .3 - .5$) (Achenbach, McConaughy, & Howell, 1987; Mitsis, McKay, Schulz, Newcorn, & Halperin, 2000). Clinicians may more accurately make diagnoses by establishing a *history* of impairment across settings rather than relying on parent-teacher agreement at one point in time.

There is also some debate regarding the threshold for developmental inappropriateness of symptoms. Currently, the cutoff for deviance is typically defined as 1.5 standard deviations above the mean on a well normed rating scale (Achenbach, 2001; Barkley & Edwards, 2006; Barkley & Murphy, 2006). Such a cutoff corresponds to the 93rd percentile. However, clinicians should avoid applying the threshold dogmatically. Cases falling near, but not over the threshold, should be treated as borderline or sub-threshold cases and the symptoms treated accordingly.

Another issue currently debated in the literature concerns the recent finding that a separate aspect of inattentive symptoms may exist among a sub-set of clinically referred children (e.g. Milich, Balentine, & Lynam, 2001). Children with these recently identified symptoms are described as having “sluggish cognitive tempo” (SCT). They are typically hypoactive, drowsy and sluggish (both physically and cognitively) (Milich et al., 2001). Clinically, it is observed that these children, although possessing adequate intelligence and basic skills, fall behind their peers academically and socially because of the effort required to maintain the same workload and speed of processing. They typically become socially withdrawn and social problems worsen with age. The symptoms observed in these children may be the very opposite of what a clinician would expect in a child with ADHD. They are therefore the children most in danger of remaining unidentified. To avoid this, the clinician may take the symptoms of SCT into account when deciding if a child is appropriate for evaluation of ADHD.

For further discussions on the merits of the DSM-IV-TR criteria, the reader's attention is drawn to the excellent reviews provided by Barkley (2006a) and also see the special issue of *Clinical Psychology: Science and Practice* (2001) (e.g. Lahey, 2001; Milich et al., 2001).

The evaluation

Consensus guidelines have been published regarding the diagnosis and management of ADHD (American Academy of Pediatrics, 2000; American Academy of Child and

Adolescent Psychiatry, 1997; National Health & Medical Research Council, 1997). Guidelines recommend core ADHD diagnostic procedures that include a structured interview with parents/caregivers, medical examination, teacher reports, rating scales, educational assessment and vision and hearing assessment. Parental perceptions indicate between 45 and 91% compliance with these recommendations (Concannon & Yang, 2005).

A review of the major developmental domains (motor, language, intellectual, academic, emotional and social functioning) and childhood psychiatric disorders is essential for differential diagnosis and documentation of co-morbid disorders (Barkley & Edwards, 2006). Prenatal and birth events with the potential to affect development need to be established (Rappley, 2005). Questioning both the parent(s) and child regarding the symptoms of the common childhood psychiatric disorders is also necessary, although clinicians should be cautious in attributing too much weight to child self-reports. Clinicians should also note that an unstandardised observation of children's behaviour in a clinic setting is an unreliable diagnostic tool and should not be used as a primary tool for diagnosis.

A family history is necessary to identify acute stressors that may be causing symptoms; while a history of certain psychiatric conditions in the extended family (particularly ADHD in parents and siblings) can lend weight to a diagnostic formulation of ADHD (Barkley & Edwards, 2006). The symptoms of ADHD must be explored in detail, along with symptom duration and pervasiveness. At least one well-normed ADHD rating scale should be administered. Rating scales are essential to establish the developmental deviance of symptoms relative to same age and sex peers (Barkley & Edwards, 2006).

Clinicians should also gather a detailed school history in order to establish a history of ADHD symptoms and evidence for impairment in academic functioning. Where possible, clinicians should also conduct a functional behaviour analysis to aid in treatment planning. Because the DSM-IV-TR criteria require impairment across multiple domains, information and objective data in the form of behaviour rating scales should be routinely obtained from teachers. School records should be examined when available. Teacher interviews can also serve as an additional opportunity to screen for the presence of a learning disability.

Neuropsychological tests, such commonly administered intelligence measures, are not diagnostic of ADHD. However, an estimate of intelligence is useful for ruling out unusual giftedness or global impairment (Gordon, Barkley, & Lovett, 2006). When there is evidence or concern of language and learning difficulties, a specialist allied health evaluation of skills relating to the presenting problem may be required to provide adequate information for treatment planning.

The child's past medical history and physical examination should be completed. The examination may need to be repeated in 12 months if there have been any significant changes. The use of blood tests is not justifiable unless there are specific clinical pointers to a disease or condition (e.g. lead poisoning, hypothyroidism). There is insufficient data to justify the routine clinical use of hair analysis, zinc levels, EEG, computerised EEG, neuroimaging or computerised tests of attention and vigilance (American Academy of Pediatrics, 2000; American Academy of Child and Adolescent Psychiatry, 1997; Barkley & Edwards, 2006; Loo & Barkley, 2005; Rappley, 2005).

Research (Barkley, 1997) has demonstrated that child behavioural disorders, and their response to treatment, is a function of the child, parental characteristics and

factors affecting the family unit. An evaluation for ADHD should therefore routinely gather relevant information about parental physical and mental health, relationship issues within the family, external stressors and other factors relating to the family's ability to cope and manage a child with ADHD.

Treatment

Pharmacology. Stimulant medications, such as methylphenidate and dexamphetamine, have been in use since 1937 and provide a safe and effective treatment for the symptoms of ADHD. They are some of the most widely studied medications in use today. No studies have revealed any long-term adverse side-effects. Stimulant medications should always be commenced on a trial basis only, usually for a period of one month using different doses. Careful evaluation of the positive effects and negative effects of each dose should be made at the end of the trial period before selecting the most effective dosage level. Where possible, clinicians should employ standardised methodology, such as rating scales, to collect data on the child's function during the trial period. Commonly reported side-effects of stimulant medication include decreased appetite and low growth rates. Less commonly reported are difficulty falling asleep, headaches and stomach aches. Most of these can be managed by changes in dose or dose timing.

Effects on growth have been reported and indicate a small reduction in height in children treated with stimulants compared to controls (American Academy of Child & Adolescent Psychiatry, 1997). Baseline data should be collected on growth (weight and height) and monitored regularly at follow-up. There is a theoretical risk of stimulant medications lowering seizure thresholds, but this is not supported in clinical practice (Hemmer, Pasternak, Zecker, & Trommer, 2001). Stimulant medication has been found to lower the risk of illicit substance abuse when compared to controls (Biederman, 2003). Recent evidence suggests that stimulant use is neither contraindicated in, or thought to increase the incidences of, and may well be beneficial for, tic disorders (Kurlan, 2002; 2003).

Potential adverse cardiovascular effects have been prominent in the popular press recently but are not supported by available evidence. Stimulants are associated with a small average increase in both heart rate and blood pressure in children and adults but this is extremely unlikely to have any significance for children or adults with a normal heart (Findling et al., 2005; Weisler, Biederman, Spencer, & Wilens, 2005). More frequent monitoring of blood pressure should occur in the early phase of treatment. Children with known cardiovascular abnormalities should have a cardiology evaluation before commencing stimulants.

Psychosocial therapy. More than 100 studies have reported improvements in child compliance, reduced disruptive behaviours and improvements in parent/teacher-child relations following parent and teacher training programs (Diller & Goldstein, 2006). Yet the role for psychosocial interventions in treatment of ADHD has been questioned by the results of the National Institute of Mental Health Multimodal Treatment Study of ADHD (MTA Cooperative Group, 1999a). The MTA study reported that psychosocial therapy was less effective than long-term treatment with stimulant medication in managing and alleviating the primary and some comorbid symptoms of ADHD. Furthermore, the use of psychosocial therapy and medication in combination resulted in outcomes no better than when medication was used alone.

As a result, a number of prominent recent reviews have concluded that psychosocial therapy should not be a front line treatment for children with ADHD (e.g. Rappley, 2005). However, this conclusion is rather limited in its scope and fails to take into account the criticisms that have been leveled at the MTA study (Klein, 2001). In fact, when other areas of function have been examined, such as anxiety, academic performance, oppositional behaviour, parent-child relations and social skills, a combined medication/psychosocial approach to treatment has been shown to produce small, but significant improvements over medication alone (MTA Cooperative Group, 1999b, 2004). These gains were also made with significantly lower stimulant dosage. This finding may have important implications for treatment, as side-effects are a function of dosage.

Further analysis of the MTA data has also indicated that the superiority of medication treatment over a combined treatment was true only for the minority of children with uncomplicated ADHD. The combined treatment resulted in significantly better outcomes for children with complicated ADHD (i.e. co-morbid anxiety and/or ODD) (Conners et al., 2001). A recent study has also demonstrated that combined medication/psychosocial treatment leads to greater gains in positive parenting when objective, rather than self-report measures are used (Wells et al., 2006).

A further criticism against the practice of employing only medication in the management of ADHD is that the symptoms of the disorder are dimensional rather than categorical. Those selected for research studies are likely to be more symptomatic and impaired than those seen in typical community clinic situations, where children are more likely to present with mild or sub-threshold ADHD (Diller & Goldstein, 2006). In the absence of published data on best-practice treatment for these children, psychosocial intervention would seem to be indicated. Finally, stimulant medication works on the primary symptoms of the disorder. Medication does not help the child, family and educators manage the secondary executive deficits that typically cause impairment; nor does it provide the understanding of the child's condition that appears necessary for satisfactory adjustment. For this reason alone, psycho-education and psychosocial training should be presented as an adjunct therapy and, in some mild or sub-threshold cases, physicians may choose psycho-education and psychosocial training as the primary treatment option.

Alternative interventions. A range of alternative therapies such as psychotherapy, diet supplements and restricted diets, herbal medicines, reflexology, neurofeedback, vestibular and sensory-motor integration and rapid auditory processing training have been promoted at substantial expense to families. None of these treatments currently meets satisfactory scientific criteria for efficacious treatment (American Academy of Pediatrics, 2000; Rappley, 2005).

Conclusion

Attention-deficit/hyperactivity disorder is a neuro-behavioural disorder that presents frequently in paediatric and mental health settings. Assessment of ADHD involves taking a careful medical, developmental and behavioural history. Use of standardised questionnaires to assess the severity of symptoms and resulting impairment should be standard. A review of developmental and psychiatric disorders is necessary for differential diagnosis and to identify co-morbid disorders. A family history should be taken to identify psychiatric disturbances or other factors that

affect management of ADHD. Referral to an expert may be necessary if learning disorders are suspected. If the evaluation reveals behaviours meeting the DSM-IV-TR criteria for ADHD, best-practice involves a management plan that includes a trial of a stimulant medication and parent education on the primary and secondary symptoms and course of ADHD. Psychological therapy is indicated in the case of co-existing psychiatric or behavioural problems. In many cases a learning expert may be required to assist with cognitive weaknesses affecting academic performance.

References

- Achenbach, T.M. (2001). *Child Behavior Checklist*. Burlington, VT: Research Center for Children, Youth and Families.
- Achenbach, T.M., McConaughy, S.H., & Howell, C.T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross informant correlations for situational specificity. *Psychological Bulletin*, *101*, 213–232.
- American Academy of Child and Adolescent Psychiatry. (1997). Practice parameters for the assessment and treatment of children, adolescents and adults with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*(10 suppl.), S85–S121.
- American Academy of Pediatrics. (2000). Clinical practice guideline: Diagnosis and evaluation of the child with attention-deficit/hyperactivity disorder. *Pediatrics*, *105*(5), 1158–1170.
- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders Text Revision* (4th ed.). Washington, DC: American Psychiatric Association.
- Angold, A., Costello, E.J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, *40*, 57–88.
- Barkley, R.A. (1997). ADHD, self-regulation and time: Toward a more comprehensive theory of ADHD. *Journal of Developmental and Behavioural Pediatrics*, *18*, 271–279.
- Barkley, R.A. (2006a). Primary symptoms, diagnostic criteria, prevalence, gender differences. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder* (3rd ed., pp. 76–121). New York: The Guilford Press.
- Barkley, R.A. (2006b). Associated cognitive, developmental and health problems. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder* (3rd ed., pp. 122–183). New York: The Guilford Press.
- Barkley, R.A. (2006c). Comorbid disorders, adjustment and subtyping. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder* (3rd ed., pp. 184–218). New York: The Guilford Press.
- Barkley, R.A. (2006d). Etiologies. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder* (3rd ed., pp. 219–247). New York: The Guilford Press.
- Barkley, R.A. (2006e). A theory of ADHD. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder* (3rd ed., pp. 297–334). New York: The Guilford Press.
- Barkley, R.A., Anastopoulos, A.D., Guevremont, D.G., & Fletcher, K. (1991). Adolescents with attention deficit hyperactivity disorder: Patterns of behavioural adjustment, academic functioning and treatment utilization. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*, 752–761.
- Barkley, R.A., Du Paul, G.J., & McMurray, M.B. (1990). A comprehensive evaluation of attention deficit disorder with and without hyperactivity. *Journal of Consulting and Clinical Psychology*, *58*, 775–789.
- Barkley, R.A., & Edwards, G. (2006). Diagnostic interview, behaviour rating scales and the medical examination. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder* (3rd ed., pp. 337–368). New York: The Guilford Press.
- Barkley, R.A., Fischer, M., Edelbrock, C.S., & Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria. I: An eight-year prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 546–557.
- Barkley, R.A., Guevremont, D.G., Anastopoulos, A.D., DuPaul, G.J., & Shelton, T.L. (1993). Driving-related risks and outcomes of attention deficit hyperactivity disorder in adolescents and young adults: A 3–5 year follow-up survey. *Pediatrics*, *92*, 212–218.

- Barkley, R.A., & Murphy, K.R. (2006). *Attention-deficit hyperactivity disorder: A clinical workbook* (3rd ed.). New York: The Guilford Press.
- Biederman, J. (2003). Pharmacotherapy for attention-deficit/hyperactivity disorder (ADHD) decreases the risk for substance abuse: Findings from a longitudinal follow-up of youths with and without ADHD. *Journal of Clinical Psychiatry*, *64*(10 Suppl.), S3–S8.
- Biederman, J., Faraone, S.V., Milberger, S., Curtis, S., Chen, L., Marris, A., et al. (1996). Predictors of persistence and remission of ADHD into adolescence: Results from a four-year prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*, 343–351.
- Biederman, J., Newcorn, J., & Sprich, S. (1991). Co-morbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety and other disorders. *American Journal of Psychiatry*, *148*, 564–577.
- Bronowski, J. (1977). Human and animal languages. In P.E. Ariotti (Ed.), *A sense of the future* (pp. 104–131). Cambridge, MA: MIT Press.
- Burns, G.L., Boe, B., Walsh, J.A., Sommers-Flanagan, R., & Teegarden, L.A. (2001). A confirmatory factor analysis on the DSM-IV ADHD and ODD symptoms: What is the best model for the organization of these symptoms? *Journal of Abnormal Child Psychology*, *29*, 339–349.
- Campbell, S.B. (1990). *Behavior problems in preschool children: Clinical and developmental issues*. New York: The Guilford Press.
- Castellanos, F.X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: The search for endophenotypes. *Nature Reviews Neuroscience*, *3*, 617–628.
- Comings, D.E. (2000). Attention-deficit/hyperactivity disorder with Tourette's syndrome. In T.E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents and adults* (pp. 363–392). Washington, DC: American Psychiatric Press.
- Concannon, P.E., & Yang, Y.P. (2005). The management of attention deficit hyperactivity disorder: A parental perspective. *Journal of Paediatric and Child Health*, *41*(12), 625–630.
- Conners, C.K., Epstein, J.N., March, J.S., Angold, A., Wells, K.C., Klaric, J., et al. (2001). Multimodal treatment of ADHD in the MTA: An alternative outcome analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 159–167.
- Cornish, K., Wilding, J., & Grant, C. (2006). Deconstructing working memory in developmental disorders of attention. In S.J. Pickering (Ed.), *Working memory and education* (pp. 58–184). Burlington, MA: Elsevier.
- Denckla, M.B. (2007). Executive function: Binding together the definitions of attention-deficit/hyperactivity disorder and learning disabilities. In L. Meltzer (Ed.), *Executive function in education: From theory to practice* (pp. 5–18). New York: The Guilford Press.
- Diamond, A. (2000). Close interrelation of motor development and cognitive development and of the cerebellum and prefrontal cortex. *Child Development*, *71*, 44–56.
- Diller, L., & Goldstein, S. (2006). Science, ethics and the psychosocial treatment of ADHD. *Journal of Attention Disorders*, *9*(4), 571–574.
- Du Paul, G.J., Power, T.J., Anastopoulos, A.D., & Reid, R. (1998). *The ADHD Rating Scale – IV: Checklists, norms and clinical interpretation*. New York: Guilford Press.
- Faraone, S.V., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The worldwide prevalence of ADHD: Is it an American condition? *World Psychiatry*, *2*(2), 104–113.
- Fernandez-Duque, D., & Posner, M.I. (2001). Brain imaging of attentional networks in normal and pathological states. *Journal of Clinical & Experimental Neuropsychology. Special issue: Functional neuroimaging and neuropsychology: Convergence, advances and new directions*, *23*(1), 74–93.
- Findling, R.L., Biederman, J., Wilens, T.E., Spencer, T.J., McGoug, J.J., Lopez, F.A., & Tulloch, S.J. (2005). Short- and long-term cardiovascular effects of mixed amphetamine salts extended release in children. *Journal of Pediatrics*, *147*(3), 348–54.
- Fischer, M., Barkley, R.A., Edelbrock, C.S., & Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria, II: Academic, attentional and neuropsychological status. *Journal of Consulting and Clinical Psychology*, *58*, 580–588.
- Fischer, M., Barkley, R.A., Smallish, L., & Fletcher, K. (2004). Hyperactive children as young adults: Deficits in inhibition, attention and response perseveration and their relationship to severity of childhood and current ADHD and conduct disorder. *Developmental Neuropsychology*, *27*, 107–133.

- Frick, P.J., Kamphaus, R.W., Lahey, B.B., Loeber, R., Christ, M.A.G., Hart, E.L., et al. (1991). Academic underachievement and the disruptive behavior disorders. *Journal of Consulting and Clinical Psychology, 59*, 289–294.
- Fuster, J.M. (1989). *The prefrontal cortex: Anatomy, physiology and neuropsychology*. New York: Raven Press.
- Goldstein, S., & Schwabach, A.J. (2004). The comorbidity of pervasive developmental disorder and attention deficit hyperactivity disorder: Results of a retrospective chart review. *Journal of Autism and Developmental Disorders, 34*, 329–339.
- Gomez, R., Harvey, J., Quick, C., Scharer, I., & Harris, G. (1999). DSM-IV AD/HD: Confirmatory factor models, prevalence, gender and age differences based on parent and teacher ratings of Australian primary school children. *Journal of Child Psychology and Psychiatry, 40*, 265–274.
- Gordon, M., Barkley, R.A., & Lovett, B.T. (2006). Tests and observational measures. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder* (3rd ed., pp. 369–377). New York: The Guilford Press.
- Graetz, B.W., Sawyer, M.G., Hazell, P.L., Arney, F., & Baghurst, P. (2001). Validity of DSM-IV ADHD subtypes in a nationally representative sample of Australian children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*, 1410–1417.
- Hemmer, S.A., Pasternak, J.F., Zecker, S.G., & Trommer, B.L. (2001). Stimulant therapy and seizure risk in children with ADHD. *Pediatric Neurology, 24*(2), 99–102.
- Hewson, P.H., Anderson, P.K., Dinning, A.H., Jenner, B.M., McKellar, W.J.D., Weymouth, R.D., et al. (1999). A 12-month profile of community paediatric consultations in the Barwon region. *Journal of Paediatrics and Child Health, 35*, 16–22.
- Houghton, S., Cordin, R., Durkin, K., & Whiting, K. (2008). Salience and temporal sequencing of time-related actions in boys with attention-deficit/hyperactivity disorder. *Child Neuropsychology, 14*, 60–70.
- Hoza, B., Pelham, W.E., Waschbusch, D.A., Kipp, H., & Owens, J.S. (2001). Academic task performance of normally achieving ADHD and control boys: Performance, self-evaluations and attributions. *Journal of Consulting and Clinical Psychology, 69*, 271–283.
- Klein, R.G. (2001). MTA findings fail to consider methodological issues. *Archives of General Psychiatry, 58*, 1184–1185.
- Kurlan, R. (2002). Methylphenidate to treat ADHD is not contraindicated in children with tics. *Movement Disorders, 17*(1), 5–6.
- Kurlan, R. (2003). Tourette's syndrome: Are stimulants safe? *Current Neurology & Neuroscience Reports, 3*(4), 285–288.
- Lahey, B.B. (2001). Should the combined and predominantly inattentive types of ADHD be considered distinct and unrelated disorders? Not now, at least. *Clinical Psychology: Science and Practice, 8*(4), 494–501.
- Lawrence, V., Houghton, S., Tannock, R., Douglas, G., Durkin, K., & Whiting, K. (2002). ADHD outside the laboratory: Boys' executive function performance on tasks in video game play and on a visit to the zoo. *Journal of Abnormal Child Psychology, 30*(5), 447–462.
- Loo, S.K., & Barkley, R.A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Applied Neuropsychology, 12*, 64–76.
- Luman, M., Oosterlaan, J., & Sergeant, J.A. (2005). The impact of reinforcement contingencies on AD/HD: A review and theoretical appraisal. *Clinical Psychology Review, 25*, 183–213.
- Milich, R., Balentine, A.C., & Lynam, D. (2001). ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. *Clinical Psychology Science and Practice, 8*, 463–488.
- Mitsis, E., McKay, K.E., Schulz, K.P., Newcorn, J.H., & Halperin, J.M. (2000). Parent-teacher concordance from DSM-IV attention-deficit/hyperactivity disorder in a clinic referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 308–313.
- MTA Cooperative Group. (1999a). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 56*, 1073–1086.
- MTA Cooperative Group. (1999b). Moderators and mediators of treatment response for children with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 56*, 1088–1096.

- MTA Cooperative Group. (2004). National Institute of Mental Health Multimodal Treatment Study of ADHD follow-up: 24-month outcomes of treatment strategies for attention-deficit/hyperactivity disorder. *Pediatrics*, *113*, 754–761.
- National Health and Medical Research Council. (1997). *Attention deficit hyperactivity disorder*. Canberra: Australian Government Publishing Service.
- Newcorn, J.H., Halperin, J.M., Jensen, P., Abikoff, H.B., Arnold, E., Cantwell, D.P., et al. (2001). Symptom profiles in children with ADHD: Effects of comorbidity and gender. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 137–146.
- Nigg, J.T. (2001). Is ADHD an inhibitory disorder? *Psychological Bulletin*, *125*, 571–596.
- Palfrey, J.S., Levine, M.D., Walker, D.K., & Sullivan, M. (1985). The emergence of attention deficits in early childhood: A prospective study. *Journal of Developmental and Behavioral Pediatrics*, *6*, 339–348.
- Pennington, B.F. (1994). The working memory function of the prefrontal cortices: Implications for developmental and individual differences in cognition. In M.M. Haith & J.B. Benson (Eds.), *The development of future-oriented processes. The John D. and Catherine T. MacArthur Foundation series on mental health and development* (pp. 243–289). Chicago, IL: University of Chicago Press.
- Pennington, B.F., Bennetto, L., McAleer, O., & Roberts, R.J., Jr. (1996). Executive function and working memory. In *Attention, memory and executive function* (pp. 327–348). Baltimore, MD: Paul H. Brookes.
- Pennington, B.F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *37*, 51–87.
- Peterson, B.S., Pine, D.S., Cohen, P., & Brook, J.S. (2001). Prospective, longitudinal study of tic, obsessive-compulsive and attention-deficit/hyperactivity disorders in an epidemiological sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 685–695.
- Pfiffner, L.J., McBurnett, K., Lahey, B.B., Loeber, R., Green, S., Frick, P.J., et al. (1999). Association of parental psychopathology to the comorbid disorders of boys with attention-deficit hyperactivity disorder. *Journal of Consulting and Clinical Psychology*, *67*, 881–893.
- Polanczyk, G., de Lima, M.S., Horta, B.L., Biederman, J., & Rohde, L.A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry*, *164*(6), 942–948.
- Posner, M.I., & Dehaene, S. (1994). Attentional networks. *Trends in Neurosciences*, *17*(2), 75–79.
- Posner, M.I., & Petersen, S.E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*, 25–42.
- Quay, H.C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Psychology*, *25*(7), 7–13.
- Rappley, M.D. (2005). Attention-deficit/hyperactivity disorder. *New England Journal of Medicine*, *352*(2), 165–175.
- Sagvolden, T., Johansen, E.B., Aase, H., & Russell, V.A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined types. *Behavioral and Brain Sciences*, *28*, 397–468.
- Scheres, A., Oosterlaan, J., & Sergeant, J.A. (2001). Response execution and inhibition in children with AD/HD and other disruptive disorders: The role of behavioral activation. *Journal of Child Psychology and Psychiatry*, *42*(3), 347–357.
- Sergeant, J. (2000). The cognitive-energetic model: An empirical approach to attention-deficit hyperactivity disorder. *Neuroscience and Behavioural Reviews*, *24*, 7–12.
- Shelton, T.L., Barkley, R.A., Crosswait, C., Moorehouse, M., Fletcher, K., Barrett, S., et al. (2000). Multimethod psychoeducational intervention for preschool children with disruptive behaviour: Two-year post-treatment follow-up. *Journal of Abnormal Child Psychology*, *28*, 253–266.
- Skounti, M., Philalithis, A., & Galanakis, E. (2007). Variations in prevalence of attention deficit hyperactivity disorder worldwide. *European Journal of Pediatrics*, *166*(2), 117–123.
- Solanto, M.V., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G.D., Wigal, T., et al. (2001). The ecological validity of delay aversion and response inhibition as measures of impulsivity in AD/HD: A supplement to the NIMH Multimodal Treatment study of AD/HD. *Journal of Abnormal Child Psychology*, *29*(3), 215–228.

- Sonuga-Barke, E.J. (2002). Psychological heterogeneity in AD/HD: A dual pathway model of behavior and cognition. *Behaviour and Brain Research*, *130*, 29–36.
- Sonuga-Barke, E.J.S., Taylor, E., & Hepinstall, E. (1992). Hyperactivity and delay aversion: II. The effect of self- versus externally-imposed stimulus presentation periods on memory. *Journal of Child Psychology and Psychiatry*, *33*, 399–409.
- Sonuga-Barke, E.J.S., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion: I. The effect of delay on choice. *Journal of Child Psychology and Psychiatry*, *33*, 387–398.
- Tannock, R. (2000). Attention-deficit/hyperactivity disorder with anxiety disorders. In T.E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents and adults* (pp. 125–170). Washington, DC: American Psychiatric Press.
- Todd, R.D., & Botteron, K.N. (2001). Is attention-deficit/hyperactivity disorder an energy deficiency syndrome? *Biological Psychiatry*, *50*, 151–158.
- Treuting, J.J., & Hinshaw, S.P. (2001). Depression and self-esteem in boys with attention-deficit/hyperactivity disorder: Associations with comorbid aggression and explanatory attributional mechanisms. *Journal of Abnormal Child Psychology*, *29*, 23–39.
- Weisler, R.H., Biederman, J., Spencer, T.J., & Wilens, T.E. (2005). Long-term cardiovascular effects of mixed amphetamine salts extended release in adults with ADHD. *CNS Spectrums*, *10*(12, Suppl. 20), 35–43.
- Weiss, G., & Hechtman, L. (1993). *Hyperactive children grown up* (2nd ed.). New York: Guilford Press.
- Wells, K.C., Chi, T.C., Hinshaw, S.P., Epstein, J.N., Pfiffner, L., Nebel-Schwalm, M., et al. (2006). Treatment-related changes in objectively measured parenting behaviors in the multimodal treatment study of children with attention-deficit/hyperactivity disorder. *Journal of Consulting and Clinical Psychology*, *74*(4), 649–657.
- Wright, C. (2007). ADHD in the classroom. *Special Education Perspectives*, *15*(2), 3–8.