Attention-Deficit/Hyperactivity Disorder (ADHD) is characterized by persistent and chronic inattention and/or excessive motor restlessness and impulsivity. Inattentive symptoms include poor organizational skills, making careless errors, forgetfulness, trouble listening, and distractibility. Hyperactive/impulsive symptoms include restlessness, excessive talking, and interrupting. For the purpose of diagnosis, symptom manifestation should be developmentally inappropriate and exhibited in two or more settings (e.g., home and school). Much of the research in the past decade has focused on deficits associated with the disorder in regard to executive function, response inhibition, cognitive control, and motivational dysfunction in response to delayed reinforcers.

The manifestation of ADHD and its associated core problems vary with development. ADHD in the combined subtype is characterized in the preschool and prepubescent period by high rates of gross motor activity, difficulty sitting in one’s seat, academic difficulties, and peer-interaction problems. ADHD in adolescence is a period associated with high risk-taking behavior, and teenagers and young adults with ADHD are likely to have more traffic accidents, substance abuse, treatment for sexually transmitted diseases (Barkley et al., 2006), and earlier initiation of sexual activity and intercourse (Flory, Pelham, et al., 2006). ADHD in adulthood is recognized as a period with less observable gross motor hyperactivity. However, problems with sustaining attention and impulsive behavior continue to result in poor work performance, greater unemployment, higher divorce rates, and engagement in criminal behavior (Barkley, 2006).

According to the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR, 2000), there are three ADHD subtypes: predominantly inattentive, predominantly hyperactive-impulsive, and a combined subtype that is the most prevalent. Individuals diagnosed with the combined subtype exhibit both inattentive and hyperactive/impulsive symptoms to a significant degree. Individuals identified with the inattentive or hyperactive/impulsive diagnoses present with predominant inattention or hyperactive and impulsive symptoms, respectively. There is also recent interest in a subset of the ADHD, inattentive, which is characterized by slow or sluggish cognitive tempo (McBurnett et al., 2001). Children with this inattentive type of ADHD appear hypoactive and seem to be in a fog or daydreaming.

There is evidence to suggest that there are differences in the genetic profile and treatment response among the ADHD subtypes. Consistent, robust differences on a neuropsychological level among the ADHD subtypes are difficult to identify, however. Neuropsychological studies that have found differences have been contradictory in nature or apply primarily to a subset of the subjects. In general, however, it appears that children with the ADHD combined subtype perform poorly on planning, cognitive flexibility, and response inhibition tasks, whereas children with the ADHD inattentive subtype display difficulty using cues to guide their behavior and demonstrate slowed motor output, impaired vigilance, and altered arousal effects.

ADHD is frequently comorbid with other disorders, most commonly with disruptive behavior disorders.
From 42.7% to 56% of children meeting criteria for ADHD in community samples meet criteria for conduct disorder as well. It is the ADHD-type symptoms rather than the conduct disorder symptoms that are most likely to predict academic achievement, thus indicating that the attentional symptoms need to be addressed as robustly as the conduct problems in children who have both. Other frequently occurring comorbid disorders in childhood ADHD include learning disabilities and anxiety disorders. Children with ADHD are also more vulnerable than young people in general to develop a substance abuse disorder and smoke nicotine.

**Prevalence**

Prevalence rates of ADHD in the childhood population vary, with expert opinion most often citing an incidence of approximately 3%–7% (American Psychiatric Association, 2000). The majority of children with the disorder continue to exhibit some symptoms in adulthood. Prevalence rates in adults are estimated to be about 4.4% (Kessler et al., 2006). The disorder is more common in males, with 6:1 frequency among clinic-referred samples and a 3.4:1 frequency among nonclinic-referred samples for males to females (Barkley, 2006).

**Etiology**

There is considerable evidence to suggest a genetic basis for ADHD. The disorder is highly heritable, with a risk of 57% of occurrence from parents with ADHD to their children. Most of the genetic research has focused on candidate genes involved in dopaminergic transmission, although noradrenergic and serotonin systems are also studied. The most reliable findings in ADHD are associated with the DRD4 repeat polymorphism (Barkley, 2006). Other associations for candidate genes have included DAT1, DBH, DRD5, SNAP-25, 5HTT, and HTR1B. ADHD also has been associated with markers at several chromosomes including, but not limited to, regions 5p, 6q, 9q, 11q, 16q, and 17p. Additional factors thought to contribute to ADHD include low birth weight, environmental toxins such as prenatal exposure to maternal smoking and alcohol consumption, and postnatal exposure to lead.

Neuroimaging research into brain structure and function in ADHD repeatedly finds significant differences between ADHD and controls in frontal, anterior cingulate, basal ganglia, and cerebellar anatomy and function. Reviews of the literature from structural MRI methods (e.g., Krain & Castellanos, 2006) reveal perhaps the most consistent findings in regard to ADHD imaging studies. Comparisons in global brain volume between children with ADHD and healthy comparison children have repeatedly found evidence of significantly reduced total brain volume, frontal cortical volume, and reductions in frontal regions in ADHD using gray-white matter segmentation techniques. Measures of cortical thickness via a recent longitudinal study (Shaw et al., 2007) revealed a delay of approximately three years in an ADHD group compared to controls in attaining peak thickness throughout the cerebrum, with delays ranging from two to five years in the prefrontal regions. Interestingly, the ADHD group had slightly earlier peak thickness (about four months) in the primary motor cortex compared to the control group.

Studies that use a variety of functioning imaging techniques suggest decreased brain activation in the frontal cortex in ADHD, although a number of studies have also shown relative increased brain activation in ADHD (see Fassbender & Schweitzer, 2006). Accumulating evidence also suggests that the anterior cingulate cortex is implicated in ADHD and is often hypoactive in both child and adult participants. The basal ganglia are another structure of inherent interest in ADHD, given the functional attributes of this structure and the fact that it is a clear target of the most common treatment for ADHD, which is

[https://search.credoreference.com/content/topic/attention_deficit_hyperactivity_disorder](https://search.credoreference.com/content/topic/attention_deficit_hyperactivity_disorder)
stimulant medication. Methylphenidate appears to increase dopamine levels in the basal ganglia via blocking its reuptake. Alterations are consistently found in the basal ganglia during functional brain-imaging studies, with the evidence mixed about whether the alterations in basal ganglia activation are increased or decreased (see Fassbender & Schweitzer, 2006). Stimulants appear to increase basal ganglia activation in children with ADHD.

Another subcortical structure, the cerebellum, also appears to be altered in structure and function in ADHD. The cerebellar vermis and hemispheres are smaller in children with ADHD than in comparison groups of healthy controls. Methylphenidate appears to increase cerebellar vermis activity, but only in a nontask-related fashion. Rates of hyperactivity and response to methylphenidate treatment also appear to be linked to the degree of activity in the cerebellar vermis (Fassbender & Schweitzer, 2006). Associations between the cerebellum, the basal ganglia, and the PFC suggest that abnormalities found in these regions may reflect a circuit-wide dysfunction in prefrontal-basal ganglia-cerebellar loops in ADHD. Ultimately, methods combining behavioral, imaging, and genetic techniques should help identify ADHD subgroups and potentially develop more targeted treatment efforts for the subgroups.

**Diagnosis**

Practice parameters for ADHD have been identified by several organizations including the American Academy of Pediatrics (AAP) and the American Academy of Child and Adolescent Psychiatry. These organizations include recommendations for the evaluation of ADHD and, via AAP, access to rating scales for evaluating ADHD and monitoring treatment response. A comprehensive evaluation of ADHD in adults or children should assess the presence or absence of symptomatology, differential diagnosis from other disorders that mimic ADHD, and the possibility of comorbid psychiatric disorders. At a minimum, the evaluation should include a clinical interview, a medical evaluation conducted within the past year, standardized behavior-rating scales from parents and teachers, and a direct observation of the patient. Evaluations for both children and adults also should include a family history as well as documentation regarding developmental, social, and academic functioning.

An evaluation for adults also ought to include information regarding childhood via academic records and retrospective-childhood ratings by the adult patient and a parent or another individual who knew the patient as a child. An assessment of intellectual, academic, neuropsychological, and attentional functioning is desirable for purposes of differential diagnosis, as well as for pointing out individual strengths and weaknesses. Psychoeducational testing can also be useful when a low level of intellectual functioning or a learning disability mimics or coexists with ADHD. Preferably, the evaluation should be individualized to address areas of concern for each patient.

**Treatment**

Although the evidence for the efficacy of pharmacological treatments for ADHD has strong empirical support (Jensen, 2002; MTA Cooperative Group, 1999; MTA Cooperative Group, 2004), there is also a role for behavioral interventions in the treatment of the disorder. Behavioral interventions are necessary for the 20%–30% of patients who do not respond to stimulants and for those who experience significant side effects from pharmacological agents. Perhaps most importantly, these interventions can also address comorbidity such as anxiety, academic performance, parent-child conflict, and stress in the parent (MTA Cooperative Group, 1999). Behavioral interventions for children include social skills training, school interventions, and parent training in contingency management. There is recent support for the use of behavioral and cognitive-behavioral treatments for adults with ADHD.
(e.g., Solanto et al., 2008) as well. The adult interventions teach compensatory strategies, organizational skills, planning, and the development of new habits to help resist distracters. Adults and adolescents may also benefit from “coaching” to monitor progress toward academic, career, and social goals.

The use of pharmacological interventions is warranted if the symptoms are interfering significantly with functioning. Most medications for ADHD appear to affect the dopaminergic and/or noradrenergic system. The majority of individuals (children and adults) are responsive to psychostimulant medications (e.g., methylphenidate and dextroamphetamine), and these compounds are considered safe and effective treatments for ADHD. Stimulants, typically considered the first line of defense, can produce improvements in impulse control, attention, on-task behavior, and social behavior. A number of fairly new delivery systems (including a pill and a transdermal “patch”) for psychostimulant medications are commercially available, including several with long-acting or extended-release options, such as Concerta, Metadate, Ritalin SR, and Ritalin LA. A recently released drug, Vyvanse (lisdexamfetamine dimesylate and mixed amphetamine salts), is an extended-release formulation of d-amphetamine that becomes therapeutically active following oral ingestion. Thus, Vyvanse should be less likely to be abused than other stimulants. Atomoxetine, a nonstimulant compound that is a highly selective inhibitor of the presynaptic noradrenergic transporter, has FDA approval for treatment of ADHD in adults and children. Other pharmacological options include bupropion and tricyclic antidepressants, but these drugs are prescribed less frequently than the stimulants. The most common side effects for stimulants and atomoxetine include decreased appetite, mild stomachaches or headaches, insomnia, increased anxiety, and/or irritability. All medications need to be monitored on a frequent and regular basis, given that children’s needs may change as they mature.

See also
Psychostimulant Treatment for Children.

References


Suggested Reading

JULIE B. SCHWEITZER
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