

Attention deficit hyperactivity disorder

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterised by executive dysfunction occasioning symptoms of inattention, hyperactivity, impulsivity and emotional dysregulation that are excessive and pervasive, impairing in multiple contexts, and developmentally-inappropriate. [8]

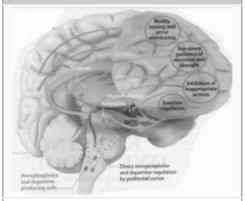
ADHD symptoms arise from executive dysfunction, and emotional dysregulation is often considered a core symptom. Difficulties in self-regulation such as time management, inhibition and sustained attention may cause poor professional performance, relationship difficulties and numerous health risks, collectively predisposing to a diminished quality of life and a direct average reduction in life expectancy of 13 years. ADHD is associated with other neurodevelopmental and mental disorders as well as non-psychiatric disorders, which can cause additional impairment.

Although people with ADHD struggle to persist on tasks with temporally delayed consequences, they may be able to do so on tasks they find intrinsically interesting or immediately rewarding; [28][16] this is known as <u>hyperfocus</u> (more colloquially)[29] or perseverative responding. [30] This mental state is often hard to disengage from [31][32] and can be related to risks such as for internet addiction [33] and types of offending behaviour. [34]

ADHD represents the extreme lower end of the continuous dimensional trait (bell curve) of executive functioning and self-regulation, which is supported by twin, brain imaging and molecular genetic studies. [35][12][36][16][37][38][39][40]

The precise causes of ADHD are unknown in the majority of cases. [41][42] For most people with ADHD, many genetic and environmental risk factors accumulate to cause the disorder. [43] The environmental risks are biological and most often exert their effects in the prenatal period. [7] However, in rare cases ADHD may be caused by a single event such as traumatic brain injury, [44][45][46][47] exposure to biohazards during pregnancy, [7] a major genetic mutation [48] or extreme environmental deprivation

Attention deficit hyperactivity disorder



ADHD arises from maldevelopment in brain regions such as the prefrontal cortex, basal ganglia and anterior cingulate cortex, which regulate the executive functions necessary for human self-regulation.

	regulation:
Specialty	Psychiatry · pediatrics
Symptoms	Inattention • carelessness • hyperactivity • executive dysfunction • disinhibition • emotional dysregulation • impulsivity • impaired working memory
Usual onset	In most cases at least some ADHD symptoms and impairments onset prior to age 12.
Causes	Genetic (inherited, de novo) and to a lesser extent, environmental factors (exposure to biohazards during

very early in life. There is no biologically distinct adult-onset ADHD except for when ADHD occurs after traumatic brain injury. [49][45][7]

Signs and symptoms

Inattention, hyperactivity (restlessness in adults), disruptive behaviour, and impulsivity are common in ADHD. [50][51][52] Academic difficulties are frequent, as are problems with relationships. [51][52][53] The signs and symptoms can be difficult to define, as it is hard to draw a line at where normal levels of inattention, hyperactivity, and impulsivity end and significant levels requiring interventions begin. [54]

According to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) and its text revision (DSM-5-TR), symptoms must be present for six months or more to a degree that is much greater than others of the same age. [3][4] This requires at least six symptoms of either inattention or hyperactivity/impulsivity for those under 17 and at least five symptoms for those 17 years or older. [3][4] The symptoms must be present in at least two settings (e.g., social, school, work, or home), and must directly interfere with or reduce quality of functioning. [3] Additionally, several symptoms must have been present before age twelve. [4] The DSM-5 's required age of onset of symptoms is 12 years. [3][4][55] However, research indicates the age of onset should not be interpreted as a prerequisite for diagnosis given contextual exceptions. [49]

Presentations

ADHD is divided into three primary presentations: [4][54]

- predominantly inattentive (ADHD-PI or ADHD-I)
- predominantly hyperactive-impulsive (ADHD-PH or ADHD-HI)
- combined presentation (ADHD-C).

The table "Symptoms" lists the symptoms for ADHD-I and

ADHD-HI from two major classification systems. Symptoms which can be better explained by another psychiatric or medical condition which an individual has are not considered to be a symptom of ADHD for that person. In DSM-5, subtypes were discarded and reclassified as presentations of the disorder that change over time.

	pregnancy, <u>traumatic</u> brain injury)	
Diagnostic method	Based on impairing symptoms after other possible causes have been ruled out	
Differential	Individual without	
diagnosis	ADHD · symptomatic but unimpaired · bipolar disorder · cognitive disengagement syndrome · conduct disorder · major depressive disorder · autism spectrum disorder · oppositional defiant disorder · learning disorder · intellectual disability · anxiety disorder [1] · borderline personality disorder · fetal alcohol	
	spectrum disorder	
Treatment	Medication · psychotherapy	
Medication	CNS stimulants (methylphenidate, amphetamine) · non- stimulants (atomoxetine, viloxazine) · alpha-2a agonists (guanfacine XR, clonidine XR)	
Frequency	0.8–1.5% (2019, using DSM-IV-TR and ICD-10) ^[2]	

Symptoms

Presentations	DSM-5 and DSM-5-TR symptoms ^{[3][4]}	ICD-11 symptoms ^[5]
Inattention	Six or more of the following symptoms in children, and five or more in adults, excluding situations where these symptoms are better explained by another psychiatric or medical condition: Frequently overlooks details or makes careless mistakes Often has difficulty maintaining focus on one task or play activity Often appears not to be listening when spoken to, including when there is no obvious distraction Frequently does not finish following instructions, failing to complete tasks Often struggles to organise tasks and activities, to meet deadlines, and to keep belongings in order Is frequently reluctant to engage in tasks which require sustained attention Frequently loses items required for tasks and activities Is frequently easily distracted by extraneous stimuli, including thoughts in adults and older teenagers Often forgets daily activities, or is forgetful while completing them.	Multiple symptoms of inattention that directly negatively impact occupational, academic or social functioning. Symptoms may not be present when engaged in highly stimulating tasks with frequent rewards. Symptoms are generally from the following clusters: Struggles to maintain focus on tasks that aren't highly stimulating/rewarding or that require continuous effort; details are often missed, and careless mistakes are frequent in school and work tasks; tasks are often abandoned before they are completed. Easily distracted (including by own thoughts); may not listen when spoken to; frequently appears to be lost in thought Often loses things; is forgetful and disorganised in daily activities. The individual may also meet the criteria for hyperactivity-impulsivity, but the inattentive symptoms are predominant.

Hyperactivity- Impulsivity	Six or more of the following symptoms in children, and five or more in adults, excluding situations where these symptoms are better explained by another psychiatric or medical condition: Is often fidgeting or squirming in seat Frequently has trouble sitting still during dinner, class, in meetings, etc. Frequently runs around or climbs in inappropriate situations. In adults and teenagers, this may be present only as restlessness. Often cannot quietly engage in leisure activities or play Frequently seems to be "on the go" or appears uncomfortable when not in motion Often talks excessively Often answers a question before it is finished, or finishes people's sentences Often struggles to wait their turn, including waiting in lines Frequently interrupts or intrudes, including into others' conversations or activities, or by using people's things without asking.	Multiple symptoms of hyperactivity/impulsivity that directly negatively impact occupational, academic or social functioning. Typically, these tend to be most apparent in environments with structure or which require self-control. Symptoms are generally from the following clusters: Excessive motor activity; struggles to sit still, often leaving their seat; prefers to run about; in younger children, will fidget when attempting to sit still; in adolescents and adults, a sense of physical restlessness or discomfort with being quiet and still. Talks too much; struggles to quietly engage in activities. Blurts out answers or comments; struggles to wait their turn in conversation, games, or activities; will interrupt or intrude on conversations or games. A lack of forethought or consideration of consequences when making decisions or taking action, instead tending to act immediately (e.g., physically dangerous behaviours including reckless driving; impulsive decisions). The individual may also meet the criteria for inattention, but the hyperactive-impulsive symptoms are predominant.
Combined	Meet the criteria for both inattentive and hyperactive-impulsive ADHD.	Criteria are met for both inattentive and hyperactive-impulsive ADHD, with neither clearly predominating.

Girls and women with ADHD tend to display fewer hyperactivity and impulsivity symptoms but more symptoms of inattention and distractibility. [56]

Symptoms are expressed differently and more subtly as the individual ages. [57]: 6 Hyperactivity tends to become less overt with age and turns into inner restlessness, difficulty relaxing or remaining still, talkativeness or constant mental activity in teens and adults with ADHD. [57]: 6-7 Impulsivity in adulthood may appear as thoughtless behaviour, impatience, irresponsible spending and sensation-seeking behaviours, [57]: 6 while inattention may appear as becoming easily bored, difficulty with organization, remaining on task and making decisions, and sensitivity to stress. [57]: 6

Although not listed as an official symptom, emotional dysregulation or mood lability is generally understood to be a common symptom of ADHD. People with ADHD of all ages are more likely to have problems with social skills, such as social interaction and forming and maintaining friendships. This is true for all presentations. About half of children and adolescents with ADHD experience social rejection by their peers compared to 10–15% of non-ADHD children and adolescents. People with attention deficits are prone to having difficulty processing verbal and nonverbal language which can negatively affect social interaction. They may also drift off during conversations, miss social cues, and have trouble learning social skills.

Difficulties managing anger are more common in children with ADHD^[60] as are delays in speech, language and motor development.^{[61][62]} Poorer handwriting is more common in children with ADHD.^[63] Poor handwriting can be a symptom of ADHD in itself due to decreased attentiveness. When this is a pervasive problem, it may also be attributable to dyslexia^{[64][65]} or dysgraphia. There is significant overlap in the symptomatologies of ADHD, dyslexia, and dysgraphia,^[66] and 3 in 10 people diagnosed with dyslexia experience co-occurring ADHD.^[67] Although it causes significant difficulty, many children with ADHD have an attention span equal to or greater than that of other children for tasks and subjects they find interesting.^[68]

IQ test performance

Certain studies have found that people with ADHD tend to have lower scores on <u>intelligence quotient</u> (IQ) tests. [69] The significance of this is controversial due to the differences between people with ADHD and the difficulty determining the influence of symptoms, such as distractibility, on lower scores rather than intellectual capacity. In studies of ADHD, higher IQs may be over-represented because many studies exclude individuals who have lower IQs despite those with ADHD scoring on average nine points lower on standardised intelligence measures. [70] However, other studies contradict this, saying that in individuals with high intelligence, there is an increased risk of a missed ADHD diagnosis, possibly because of compensatory strategies in said individuals. [71]

Studies of adults suggest that negative differences in intelligence are not meaningful and may be explained by associated health problems. [72]

Comorbidities

Psychiatric comorbidities

In children, ADHD occurs with other disorders about two-thirds of the time. [68]

Other neurodevelopmental conditions are common comorbidities. <u>Autism spectrum disorder</u> (ASD), cooccurring at a rate of 21% in those with ADHD, affects social skills, ability to communicate, behaviour, and interests. <u>[73][74]</u> <u>Learning disabilities</u> have been found to occur in about 20–30% of children with ADHD. Learning disabilities can include developmental speech and language disorders, and academic skills disorders. <u>[75]</u> ADHD, however, is not considered a learning disability, but it very frequently causes academic difficulties. <u>[75]</u> Intellectual disabilities and Tourette's syndrome.

ADHD is often comorbid with disruptive, impulse control, and conduct disorders. Oppositional defiant disorder (ODD) occurs in about 25% of children with an inattentive presentation and 50% of those with a combined presentation. It is characterised by angry or irritable mood, argumentative or defiant behaviour and vindictiveness which are age-inappropriate. Conduct disorder (CD) occurs in about 25% of adolescents with ADHD. It is characterised by aggression, destruction of property, deceitfulness, theft and violations of rules. Adolescents with ADHD who also have CD are more likely to develop antisocial personality disorder in adulthood. Brain imaging supports that CD and ADHD are separate conditions: conduct disorder was shown to reduce the size of one's temporal lobe and limbic system, and increase the size of one's orbitofrontal cortex, whereas ADHD was shown to reduce connections in the cerebellum and

prefrontal cortex more broadly. Conduct disorder involves more impairment in motivation control than ADHD. [78] Intermittent explosive disorder is characterised by sudden and disproportionate outbursts of anger and co-occurs in individuals with ADHD more frequently than in the general population.

Anxiety and mood disorders are frequent comorbidities. <u>Anxiety disorders</u> have been found to occur more commonly in the ADHD population, as have <u>mood disorders</u> (especially <u>bipolar disorder</u> and <u>major depressive disorder</u>). Boys diagnosed with the combined ADHD subtype are more likely to have a mood disorder. <u>Adults and children with ADHD sometimes also have bipolar disorder</u>, which requires careful assessment to accurately diagnose and treat both conditions. <u>[80][81]</u>

<u>Sleep disorders</u> and ADHD commonly co-exist. They can also occur as a side effect of medications used to treat ADHD. In children with ADHD, <u>insomnia</u> is the most common sleep disorder with behavioural therapy being the preferred treatment. Problems with sleep initiation are common among individuals with ADHD but often they will be deep sleepers and have significant difficulty getting up in the morning. Melatonin is sometimes used in children who have sleep onset insomnia. Restless legs syndrome has been found to be more common in those with ADHD and is often due to <u>iron deficiency anemia</u>. However, restless legs can simply be a part of ADHD and requires careful assessment to differentiate between the two disorders.

Individuals with ADHD are at increased risk of <u>substance use disorders</u>. This is most commonly seen with <u>alcohol</u> or <u>cannabis</u>. The reason for this may be an altered reward pathway in the brains of ADHD individuals, self-treatment and increased psychosocial risk factors. This makes the evaluation and treatment of ADHD more difficult, with serious substance misuse problems usually treated first due to their greater risks. Other psychiatric conditions include <u>reactive attachment disorder</u>, characterised by a severe inability to appropriately relate socially, and <u>cognitive disengagement syndrome</u>, a distinct attention disorder occurring in 30–50% of ADHD cases as a comorbidity, regardless of the presentation; a subset of cases diagnosed with ADHD-PIP have been found to have CDS instead. Individuals with ADHD are three times more likely to be diagnosed with an <u>eating disorder</u> compared to those without ADHD; conversely, individuals with eating disorders are two times more likely to have ADHD than those without eating disorders.

Trauma

ADHD, <u>trauma</u>, and <u>adverse childhood experiences</u> are also comorbid, which could in part be potentially explained by the similarity in presentation between different diagnoses. The symptoms of ADHD and <u>PTSD</u> can have significant behavioural overlap—in particular, motor restlessness, difficulty concentrating, distractibility, irritability/anger, emotional constriction or dysregulation, poor impulse control, and forgetfulness are common in both. This could result in trauma-related disorders or ADHD being mis-identified as the other. Additionally, traumatic events in childhood are a risk factor for ADHD; they can lead to structural brain changes and the development of ADHD behaviours. Finally, the behavioural consequences of ADHD symptoms cause a higher chance of the individual experiencing trauma (and therefore ADHD leads to a concrete diagnosis of a trauma-related disorder). [102][103]

Non-psychiatric

Some non-psychiatric conditions are also comorbidities of ADHD. This includes <u>epilepsy</u>, [74] a neurological condition characterised by recurrent seizures. There are well established associations between ADHD and obesity, <u>asthma</u> and sleep disorders, and an association with celiac disease. Children with ADHD have a higher risk for <u>migraine</u> headaches, but have no increased risk of tension-type headaches. Children with ADHD may also experience headaches as a result of medication.

A 2021 review reported that several neurometabolic disorders caused by <u>inborn errors of metabolism</u> converge on common neurochemical mechanisms that interfere with biological mechanisms also considered central in ADHD pathophysiology and treatment. This highlights the importance of close collaboration between health services to avoid clinical overshadowing. [111]

In June 2021, *Neuroscience & Biobehavioral Reviews* published a systematic review of 82 studies that all confirmed or implied elevated accident-proneness in ADHD patients and whose data suggested that the type of accidents or injuries and overall risk changes in ADHD patients over the lifespan. [112] In January 2014, *Accident Analysis & Prevention* published a meta-analysis of 16 studies examining the relative risk of traffic collisions for drivers with ADHD, finding an overall relative risk estimate of 1.36 without controlling for exposure, a relative risk estimate of 1.29 when controlling for publication bias, a relative risk estimate of 1.23 when controlling for exposure, and a relative risk estimate of 1.86 for ADHD drivers with oppositional defiant disorder and/or conduct disorder comorbidities. [113][114]

Problematic digital media use

In April 2018, the *International Journal of Environmental Research and Public Health* published a systematic review of 24 studies researching associations between internet gaming disorder (IGD) and various psychopathologies that found an 85% correlation between IGD and ADHD. [115] In October 2018, *PNAS USA* published a systematic review of four decades of research on the relationship between children and adolescents' screen media use and ADHD-related behaviours and concluded that a statistically small relationship between children's media use and ADHD-related behaviours exists. [116] In November 2018, *Cyberpsychology* published a systematic review and meta-analysis of 5 studies that found evidence for a relationship between problematic smartphone use and impulsivity traits. [117] In October 2020, the *Journal of Behavioral Addictions* published a systematic review and meta-analysis of 40 studies with 33,650 post-secondary student subjects that found a weak-to-moderate positive association between mobile phone addiction and impulsivity. [118] In January 2021, the *Journal of Psychiatric Research* published a systematic review of 29 studies including 56,650 subjects that found that ADHD symptoms were consistently associated with gaming disorder and more frequent associations between inattention and gaming disorder than other ADHD scales. [119]

In July 2021, <u>Frontiers in Psychiatry</u> published a meta-analysis reviewing 40 <u>voxel-based morphometry</u> studies and 59 <u>functional magnetic resonance imaging</u> studies comparing subjects with IGD or ADHD to control groups that found that IGD and ADHD subjects had disorder-differentiating structural <u>neuroimage</u> alterations in the <u>putamen</u> and <u>orbitofrontal cortex</u> (OFC) respectively, and functional alterations in the <u>precuneus</u> for IGD subjects and in the <u>rewards circuit</u> (including the OFC, the <u>anterior cingulate cortex</u>, and <u>striatum</u>) for both IGD and ADHD subjects. [120] In March 2022, <u>JAMA Psychiatry</u> published a systematic review and meta-analysis of 87 studies with 159,425 subjects 12 years of age or younger that found a small but statistically significant correlation between screen time and ADHD symptoms in children. [121] In April 2022, <u>Developmental Neuropsychology</u> published a systematic review of 11 studies where the data from all but one study suggested that heightened screen time for children is associated with attention problems. [122]

In July 2022, the *Journal of Behavioral Addictions* published a meta-analysis of 14 studies comprising 2,488 subjects aged 6 to 18 years that found significantly more severe problematic internet use in subjects diagnosed with ADHD to control groups. [123]

In December 2022, *European Child & Adolescent Psychiatry* published a systematic literature review of 28 longitudinal studies published from 2011 through 2021 of associations between digital media use by children and adolescents and later ADHD symptoms and found reciprocal associations between digital media use and ADHD symptoms (i.e. that subjects with ADHD symptoms were more likely to develop problematic digital media use and that increased digital media use was associated with increased subsequent severity of ADHD symptoms). [124] In May 2023, *Reviews on Environmental Health* published a meta-analysis of 9 studies with 81,234 child subjects that found a positive correlation between screen time and ADHD risk in children and that higher amounts of screen time in childhood may significantly contribute to the development of ADHD. [125] In December 2023, the *Journal of Psychiatric Research* published a meta-analysis of 24 studies with 18,859 subjects with a mean age of 18.4 years that found significant associations between ADHD and problematic internet use, [126] while *Clinical Psychology Review* published a systematic review and meta-analysis of 48 studies examining associations between ADHD and gaming disorder that found a statistically significant association between the disorders. [127]

Suicide risk

Systematic reviews in 2017 and 2020 found strong evidence that ADHD is associated with increased suicide risk across all age groups, as well as growing evidence that an ADHD diagnosis in childhood or adolescence represents a significant future suicidal risk factor. Potential causes include ADHD's association with functional impairment, negative social, educational and occupational outcomes, and financial distress. A 2019 meta-analysis indicated a significant association between ADHD and suicidal spectrum behaviours (suicidal attempts, ideations, plans, and completed suicides); across the studies examined, the prevalence of suicide attempts in individuals with ADHD was 18.9%, compared to 9.3% in individuals without ADHD, and the findings were substantially replicated among studies which adjusted for other variables. However, the relationship between ADHD and suicidal spectrum behaviours remains unclear due to mixed findings across individual studies and the complicating impact of comorbid psychiatric disorders. There is no clear data on whether there is a direct relationship between ADHD and suicidality, or whether ADHD increases suicide risk through comorbidities.

Causes

ADHD arises from brain maldevelopment especially in the prefrontal executive networks that can arise either from genetic factors (different gene variants and mutations for building and regulating such networks) or from acquired disruptions to the development of these networks and regions; involved in executive functioning and self-regulation. Their reduced size, functional connectivity, and activation contribute to the pathophysiology of ADHD, as well as imbalances in the noradrenergic and dopaminergic systems that mediate these brain regions. [7][132]

Genetic factors play an important role; ADHD has a heritability rate of 70-80%. The remaining 20-30% of variance is mediated by de-novo mutations and non-shared environmental factors that provide for or produce brain injuries; there is no significant contribution of the rearing family and social environment. [139] Very rarely, ADHD can also be the result of abnormalities in the chromosomes. [140]

Genetics

In November 1999, *Biological Psychiatry* published a <u>literature review</u> by psychiatrists <u>Joseph Biederman</u> and Thomas Spencer found the average <u>heritability</u> estimate of ADHD from <u>twin studies</u> to be 0.8, while a subsequent <u>family</u>, twin, and <u>adoption studies</u> literature review published in <u>Molecular Psychiatry</u> in April 2019 by psychologists <u>Stephen Faraone</u> and Henrik Larsson that found an average heritability estimate of 0.74. Additionally, <u>evolutionary psychiatrist</u> <u>Randolph M. Nesse</u> has argued that the 5:1 male-to-female sex ratio in the <u>epidemiology</u> of <u>ADHD</u> suggests that ADHD may be the <u>end of a continuum where males</u> are overrepresented at the tails, citing clinical psychologist <u>Simon Baron-Cohen's</u> suggestion for the sex ratio in the epidemiology of autism as an analogue. [143][144][145]

<u>Natural selection</u> has been acting against the genetic variants for ADHD over the course of at least 45,000 years, indicating that it was not an adaptative trait in ancient times. [146] The disorder may remain at a stable rate by the balance of genetic mutations and removal rate (natural selection) across generations; over thousands of years, these genetic variants become more stable, decreasing disorder prevalence. [147] Throughout human evolution, the EFs involved in ADHD likely provide the capacity to bind contingencies across time thereby directing behaviour toward future over immediate events so as to maximise future social consequences for humans. [148]

ADHD has a high <u>heritability</u> of 74%, meaning that 74% of the presence of ADHD in the population is due to genetic factors. There are multiple gene variants which each slightly increase the likelihood of a person having ADHD; it is <u>polygenic</u> and thus arises through the accumulation of many genetic risks each having a very small effect. [7][149] The siblings of children with ADHD are three to four times more likely to develop the disorder than siblings of children without the disorder. [150]

The association of maternal smoking observed in large population studies disappears after adjusting for family history of ADHD, which indicates that the association between maternal smoking during pregnancy and ADHD is due to familial or genetic factors that increase the risk for the confluence of smoking and ADHD. [151][152]

ADHD presents with reduced size, functional connectivity and activation^[7] as well as low noradrenergic and dopaminergic functioning^{[153][154]} in brain regions and networks crucial for executive functioning and self-regulation.^{[7][39][16]} Typically, a number of genes are involved, many of which directly affect brain functioning and neurotransmission.^[7] Those involved with dopamine include DAT, DRD4, DRD5, TAAR1, MAOA, COMT, and DBH.^{[155][156][157]} Other genes associated with ADHD include SERT, HTR1B, SNAP25, GRIN2A, ADRA2A, TPH2, and BDNF.^[158] A common variant of a gene called latrophilin 3 is estimated to be responsible for about 9% of cases and when this variant is present, people are particularly responsive to stimulant medication.^[159] The 7 repeat variant of dopamine receptor D4 (DRD4–7R) causes increased inhibitory effects induced by dopamine and is associated with ADHD. The DRD4 receptor is a G protein-coupled receptor that inhibits adenylyl cyclase. The DRD4–7R mutation results in a wide range of behavioural phenotypes, including ADHD symptoms reflecting split attention.^[160] The DRD4 gene is both linked to novelty seeking and ADHD. The genes GFOD1 and CDH13 show strong

genetic associations with ADHD. CDH13's association with ASD, <u>schizophrenia</u>, bipolar disorder, and <u>depression</u> make it an interesting candidate causative gene. [137] Another candidate causative gene that has been identified is <u>ADGRL3</u>. In <u>zebrafish</u>, knockout of this gene causes a loss of dopaminergic function in the ventral diencephalon and the fish display a hyperactive/impulsive phenotype. [137]

For genetic variation to be used as a tool for diagnosis, more validating studies need to be performed. However, smaller studies have shown that genetic polymorphisms in genes related to catecholaminergic neurotransmission or the SNARE complex of the synapse can reliably predict a person's response to stimulant medication. Rare genetic variants show more relevant clinical significance as their penetrance (the chance of developing the disorder) tends to be much higher. However their usefulness as tools for diagnosis is limited as no single gene predicts ADHD. ASD shows genetic overlap with ADHD at both common and rare levels of genetic variation.

Environment

In addition to genetics, some environmental factors might play a role in causing ADHD. [162][163] Alcohol intake during pregnancy can cause fetal alcohol spectrum disorders which can include ADHD or symptoms like it. [164] Children exposed to certain toxic substances, such as lead or polychlorinated biphenyls, may develop problems which resemble ADHD. [41][165] Exposure to the organophosphate insecticides chlorpyrifos and dialkyl phosphate is associated with an increased risk; however, the evidence is not conclusive. [166] Exposure to tobacco smoke during pregnancy can cause problems with central nervous system development and can increase the risk of ADHD. [41][167] Nicotine exposure during pregnancy may be an environmental risk. [168]

Extreme premature birth, very <u>low birth weight</u>, and extreme neglect, abuse, or social deprivation also increase the $risk^{[169][41][170]}$ as do certain infections during pregnancy, at birth, and in early childhood. These infections include, among others, various viruses (<u>measles</u>, <u>varicella zoster encephalitis</u>, <u>rubella</u>, <u>enterovirus 71). At least 30% of children with a <u>traumatic brain injury</u> later develop ADHD and about 5% of cases are due to brain damage. 173</u>

Some studies suggest that in a small number of children, artificial <u>food dyes</u> or <u>preservatives</u> may be associated with an increased prevalence of ADHD or ADHD-like symptoms, $\frac{[41][174]}{[175]}$ but the evidence is weak and may apply to only children with <u>food sensitivities</u>, $\frac{[162][174][175]}{[174][175]}$ The <u>European Union</u> has put in place regulatory measures based on these concerns. $\frac{[176]}{[174]}$ In a minority of children, <u>intolerances</u> or <u>allergies</u> to certain foods may worsen ADHD symptoms.

Individuals with <u>hypokalemic sensory overstimulation</u> are sometimes diagnosed as having ADHD, raising the possibility that a subtype of ADHD has a cause that can be understood mechanistically and treated in a novel way. The sensory overload is treatable with oral potassium gluconate.

Research does not support popular beliefs that ADHD is caused by eating too much refined sugar, watching too much television, bad parenting, poverty or family chaos; however, they might worsen ADHD symptoms in certain people. [50]

The youngest children in a class have been found to be more likely to be diagnosed as having ADHD, possibly due to their being developmentally behind their older classmates. [178][179] One study showed that the youngest children in fifth and eight grade was nearly twice as likely to use stimulant medication than their older peers. [180]

In some cases, an inappropriate diagnosis of ADHD may reflect a <u>dysfunctional family</u> or a poor <u>educational system</u>, rather than any true presence of ADHD in the individual. In other cases, it may be explained by increasing academic expectations, with a diagnosis being a method for parents in some countries to get extra financial and educational support for their child. Behaviours typical of ADHD occur more commonly in children who have experienced violence and emotional abuse.

Pathophysiology

Current models of ADHD suggest that it is associated with functional impairments in some of the brain's neurotransmitter systems, particularly those involving dopamine and norepinephrine. The dopamine and norepinephrine pathways that originate in the ventral tegmental area and locus coeruleus project to diverse regions of the brain and govern a variety of cognitive processes. The dopamine pathways and norepinephrine pathways which project to the prefrontal cortex and striatum are directly responsible for modulating executive function (cognitive control of behaviour), motivation, reward perception, and motor function; these pathways are known to play a central role in the pathophysiology of ADHD. [184][14][185][186] Larger models of ADHD with additional pathways have been proposed. [185][186]

Brain structure

In children with ADHD, there is a general reduction of volume in certain brain structures, with a proportionally greater decrease in the volume in the left-sided prefrontal cortex. The posterior parietal cortex also shows thinning in individuals with ADHD compared to controls. Other brain structures in the prefrontal-striatal-cerebellar and prefrontal-striatal-thalamic circuits have also been found to differ between people with and without ADHD. [183][185][186]



The left prefrontal cortex, shown here in blue, is often affected in ADHD

The subcortical volumes of the <u>accumbens</u>, <u>amygdala</u>, caudate, hippocampus, and putamen appears smaller in

individuals with ADHD compared with controls. [188] Structural MRI studies have also revealed differences in white matter, with marked differences in inter-hemispheric asymmetry between ADHD and typically developing youths. [189]

<u>Functional MRI</u> (fMRI) studies have revealed a number of differences between ADHD and control brains. Mirroring what is known from structural findings, fMRI studies have showed evidence for a higher connectivity between subcortical and cortical regions, such as between the caudate and prefrontal cortex. The degree of hyperconnectivity between these regions correlated with the severity of inattention or hyperactivity [190] Hemispheric lateralization processes have also been postulated as being implicated in ADHD, but empiric results showed contrasting evidence on the topic.[191][192]

Neurotransmitter pathways

Previously, it had been suggested that the elevated number of <u>dopamine transporters</u> in people with ADHD was part of the pathophysiology, but it appears the elevated numbers may be due to adaptation following exposure to stimulant medication. Current models involve the <u>mesocorticolimbic dopamine pathway</u> and the <u>locus coeruleus-noradrenergic system</u>. ADHD psychostimulants possess treatment efficacy because they increase neurotransmitter activity in these systems. ADHD There may additionally be abnormalities in <u>serotonergic</u>, glutamatergic, or <u>cholinergic</u> pathways.

Executive function and motivation

ADHD arises from a core deficit in executive functions (e.g., attentional control, inhibitory control, and working memory), which are a set of cognitive processes that are required to successfully select and monitor behaviours that facilitate the attainment of one's chosen goals. [14][15] The executive function impairments that occur in ADHD individuals result in problems with staying organised, time keeping, excessive procrastination, maintaining concentration, paying attention, ignoring distractions, regulating emotions, and remembering details. [13][183][14] People with ADHD appear to have unimpaired long-term memory, and deficits in long-term recall appear to be attributed to impairments in working memory. [197] Due to the rates of brain maturation and the increasing demands for executive control as a person gets older, ADHD impairments may not fully manifest themselves until adolescence or even early adulthood. [13] Conversely, brain maturation trajectories, potentially exhibiting diverging longitudinal trends in ADHD, may support a later improvement in executive functions after reaching adulthood. [191]

ADHD has also been associated with motivational deficits in children. Children with ADHD often find it difficult to focus on long-term over short-term rewards, and exhibit impulsive behaviour for short-term rewards. [198]

Paradoxical reaction to neuroactive substances

Another sign of the structurally altered signal processing in the central nervous system in this group of people is the conspicuously common paradoxical reaction (c. 10–20% of patients). These are unexpected reactions in the opposite direction as with a normal effect, or otherwise significant different reactions. These are reactions to neuroactive substances such as <u>local anesthetic</u> at the dentist, <u>sedative</u>, <u>caffeine</u>, <u>antihistamine</u>, weak <u>neuroleptics</u> and central and peripheral <u>painkillers</u>. Since the causes of *paradoxical reactions* are at least partly genetic, it may be useful in critical situations, for example before operations, to ask whether such abnormalities may also exist in family members. [199][200]

Diagnosis

ADHD is diagnosed by an assessment of a person's behavioural and mental development, including ruling out the effects of drugs, medications, and other medical or psychiatric problems as explanations for the symptoms. [90] ADHD diagnosis often takes into account feedback from parents and teachers [201] with most diagnoses begun after a teacher raises concerns. [173] While many tools exist to aide in the diagnosis of ADHD, their validity varies in different populations, and a reliable and valid diagnosis requires confirmation by a clinician while supplemented by standardized rating scales and input from multiple informants across various settings. [202]

The most commonly used rating scales for diagnosing ADHD are the Achenbach System of Empirically Based Assessment (ASEBA) and include the Child Behavior Checklist (CBCL) used for parents to rate their child's behaviour, the Youth Self Report Form (YSR) used for children to rate their own behaviour, and the Teacher Report Form (TRF) used for teachers to rate their pupil's behaviour. Additional rating scales that have been used alone or in combination with other measures to diagnose ADHD include the Behavior Assessment System for Children (BASC), Behavior Rating Inventory of Executive Function - Second Edition (BRIEF2), Revised Conners Rating Scale (CRS-R), Conduct-Hyperactive-Attention Problem-Oppositional Symptom scale (CHAOS), Developmental Behavior Checklist Hyperactivity Index (DBC-HI), Parent Disruptive Behavior Disorder Ratings Scale (DBDRS), Diagnostic Infant and Preschool Assessment (DIPA-L), Pediatric Symptom Checklist (PSC), Social Communication Questionnaire (SCQ), Social Responsiveness Scale (SRS), Strengths and Weaknesses of ADHD Symptoms and Normal Behavior Rating Scale (SWAN), and the Vanderbilt ADHD diagnostic rating scale.

The ASEBA, BASC, CHAOS, CRS, and Vanderbilt diagnostic rating scales allow for both parents and teachers as raters in the diagnosis of childhood and adolescent ADHD. Adolescents may also self report their symptoms using self report scales from the ASEBA, SWAN, and the Dominic Interactive for Adolescents-Revised (DIA-R). [203]

Based on a 2024 systematic literature review and meta analysis commissioned by the Patient-Centered Outcomes Research Institute (PCORI), rating scales based on parent report, teacher report, or self-assessment from the adolescent have high internal consistency as a diagnostic tool meaning that the items within the scale are highly interrelated. The reliability of the scales between raters (i.e. their degree of agreement) however is poor to moderate making it important to include information from multiple raters to best inform a diagnosis. [203]

Imaging studies of the brain do not give consistent results between individuals; thus, they are only used for research purposes and not a diagnosis. [204] A 2024 systematic review concluded that the use of biomarkers such as blood or urine samples, electroencephalogram (EEG) markers, and neuroimaging such as MRIs, in diagnosis for ADHD remains unclear; studies showed great variability, did not assess test-retest reliability, and were not independently replicable. [205]

In North America and Australia, DSM-5 criteria are used for diagnosis, while European countries usually use the ICD-10. The DSM-IV criteria for diagnosis of ADHD is 3–4 times more likely to diagnose ADHD than is the ICD-10 criteria. [206] ADHD is alternately classified as neurodevelopmental disorder [207] or a disruptive behaviour disorder along with ODD, CD, and antisocial personality disorder. [208] A diagnosis does not imply a neurological disorder. [182]

Associated conditions that should be screened for include anxiety, depression, ODD, CD, and learning and language disorders. Other conditions that should be considered are other neurodevelopmental disorders, tics, and sleep apnea. [209]

Self-rating scales, such as the <u>ADHD</u> rating scale and the <u>Vanderbilt ADHD</u> diagnostic rating scale, are used in the screening and evaluation of ADHD. [210] Electroencephalography is not accurate enough to make an ADHD diagnosis. [211][212][213]

Very few studies have been conducted on diagnosis of ADHD on children younger than 7 years of age, and those that have were found in a 2024 systematic review to be of low or insufficient strength of evidence. [214]

Classification

Diagnostic and Statistical Manual

As with many other psychiatric disorders, a formal diagnosis should be made by a qualified professional based on a set number of criteria. In the United States, these criteria are defined by the <u>American Psychiatric Association</u> in the <u>DSM</u>. Based on the DSM-5 criteria published in 2013 and the DSM-5-TR criteria published in 2022, there are three presentations of ADHD:

- 1. ADHD, predominantly inattentive presentation, presents with symptoms including being easily distracted, forgetful, daydreaming, disorganization, poor sustained attention, and difficulty completing tasks.
- 2. ADHD, predominantly hyperactive-impulsive presentation, presents with excessive fidgeting and restlessness, hyperactivity, and difficulty waiting and remaining seated.
- 3. ADHD, combined presentation, is a combination of the first two presentations.

This subdivision is based on presence of at least six (in children) or five (in older teenagers and adults) $^{[215]}$ out of nine long-term (lasting at least six months) symptoms of inattention, hyperactivity—impulsivity, or both. $^{[3][4]}$ To be considered, several symptoms must have appeared by the age of six to twelve and occur in more than one environment (e.g. at home and at school or work). The symptoms must be inappropriate for a child of that age $^{[216]}$ and there must be clear evidence that they are causing impairment in multiple domains of life. $^{[217]}$

The DSM-5 and the DSM-5-TR also provide two diagnoses for individuals who have symptoms of ADHD but do not entirely meet the requirements. *Other Specified ADHD* allows the clinician to describe why the individual does not meet the criteria, whereas $Unspecified\ ADHD$ is used where the clinician chooses not to describe the reason. [3][4]

International Classification of Diseases

In the eleventh revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-11) by the World Health Organization, the disorder is classified as Attention deficit hyperactivity disorder (code 6A05). The defined subtypes are *predominantly inattentive presentation* (6A05.0); *predominantly hyperactive-impulsive presentation* (6A05.1); and *combined presentation* (6A05.2). However, the ICD-11 includes two residual categories for individuals who do not entirely match any of the defined subtypes: *other specified presentation* (6A05.Y) where the clinician includes detail on the individual's presentation; and *presentation unspecified* (6A05.Z) where the clinician does not provide detail. [5]

In the tenth revision (ICD-10), the symptoms of *hyperkinetic disorder* were analogous to ADHD in the ICD-11. When a <u>conduct disorder</u> (as defined by ICD-10) $^{[61]}$ is present, the condition was referred to as *hyperkinetic conduct disorder*. Otherwise, the disorder was classified as *disturbance of activity and attention*, *other hyperkinetic disorders* or *hyperkinetic disorders*, *unspecified*. The latter was sometimes referred to as *hyperkinetic syndrome*. $^{[61]}$

Social construct theory

The <u>social construct theory of ADHD</u> suggests that, because the boundaries between normal and abnormal behaviour are socially constructed (i.e. jointly created and validated by all members of society, and in particular by <u>physicians</u>, parents, teachers, and others), it then follows that subjective valuations and judgements determine which diagnostic criteria are used and thus, the number of people affected. Thomas Szasz, a supporter of this theory, has argued that ADHD was "invented and then given a name". [219]

Adults

Adults with ADHD are diagnosed under the same criteria, including that their signs must have been present by the age of six to twelve. The individual is the best source for information in diagnosis, however others may provide useful information about the individual's symptoms currently and in childhood; a family history of ADHD also adds weight to a diagnosis. [57]:7,9 While the core symptoms of ADHD are similar in children and adults, they often present differently in adults than in children: for example, excessive physical activity seen in children may present as feelings of restlessness and constant mental activity in adults. [57]:6

Worldwide, it is estimated that 2.58% of adults have persistent ADHD (where the individual currently meets the criteria and there is evidence of childhood onset), and 6.76% of adults have symptomatic ADHD (meaning that they currently meet the criteria for ADHD, regardless of childhood onset). [220] In 2020, this was 139.84 million and 366.33 million affected adults respectively. [220] Around 15% of children with ADHD continue to meet full DSM-IV-TR criteria at 25 years of age, and 50% still experience some symptoms. As of 2010, most adults remain untreated. [221] Many adults with ADHD without diagnosis and treatment have a disorganised life, and some use non-prescribed drugs or alcohol as a coping mechanism. Other problems may include relationship and job difficulties, and an increased risk of criminal activities. Associated mental health problems include depression, anxiety disorders, and learning disabilities.

Some ADHD symptoms in adults differ from those seen in children. While children with ADHD may climb and run about excessively, adults may experience an inability to relax, or may talk excessively in social situations. Adults with ADHD may start relationships impulsively, display sensation-seeking behaviour, and be short-tempered. Addictive behaviour such as substance abuse and gambling are common. This led to those who presented differently as they aged having outgrown the DSM-IV criteria. The DSM-5 criteria does specifically deal with adults unlike that of DSM-IV, which does not fully take into account the differences in impairments seen in adulthood compared to childhood.

For diagnosis in an adult, having symptoms since childhood is required. Nevertheless, a proportion of adults who meet the criteria for ADHD in adulthood would not have been diagnosed with ADHD as children. Most cases of late-onset ADHD develop the disorder between the ages of 12–16 and may therefore be considered early adult or adolescent-onset ADHD. [224]

Differential diagnosis

Symptoms related to other disorders^[225]

Depression disorder	Anxiety disorder	Bipolar disorder
 feelings of hopelessness, low self-esteem or unhappiness loss of interest in hobbies or regular activities fatigue sleep problems difficulty maintaining attention change in appetite irritability or hostility low tolerance for stress thoughts of death unexplained pain 	 persistent feeling of anxiety irritability occasional feelings of panic or fear being hyperalert inability to pay attention tire easily low tolerance for stress difficulty maintaining attention 	in manic state - excessive happiness - hyperactivity - racing thoughts - aggression - excessive talking - grandiose delusions - decreased need for sleep - inappropriate social behaviour - difficulty maintaining attention in depressive state - same symptoms as in depression section

The DSM provides <u>differential diagnoses</u> – potential alternate explanations for specific symptoms. Assessment and investigation of clinical history determines which is the most appropriate diagnosis. The DSM-5 suggests ODD, intermittent explosive disorder, and other neurodevelopmental disorders (such as stereotypic movement disorder and Tourette's disorder), in addition to specific learning disorder, intellectual developmental disorder, ASD, reactive attachment disorder, anxiety disorders, depressive disorders, bipolar disorder, disruptive mood dysregulation disorder, substance use disorder, personality disorders, psychotic disorders, medication-induced symptoms, and neurocognitive disorders. Many but not all of these are also common comorbidities of ADHD. The DSM-5-TR also suggests post-traumatic stress disorder.

Symptoms of ADHD, such as low mood and poor self-image, mood swings, and irritability, can be confused with dysthymia, cyclothymia or bipolar disorder as well as with borderline personality disorder. Some symptoms that are due to anxiety disorders, personality disorder, developmental disabilities or intellectual disability or the effects of substance abuse such as intoxication and withdrawal can overlap with ADHD. These disorders can also sometimes occur along with ADHD. Medical conditions which can cause ADHD-type symptoms include: hyperthyroidism, seizure disorder, lead toxicity, hearing deficits, hepatic disease, sleep apnea, drug interactions, untreated celiac disease, and head injury. [226][222]

Primary sleep disorders may affect attention and behaviour and the symptoms of ADHD may affect sleep. [227] It is thus recommended that children with ADHD be regularly assessed for sleep problems. [228] Sleepiness in children may result in symptoms ranging from the classic ones of yawning and rubbing the eyes, to hyperactivity and inattentiveness. Obstructive sleep apnea can also cause ADHD-type symptoms. [229]

Management

The management of ADHD typically involves counseling or medications, either alone or in combination. While there are various options of treatment to improve ADHD symptoms, medication therapies substantially improves long-term outcomes, and while completely eliminating some elevated risks such as obesity, [7] they do come with some risks of adverse events. [230] Medications used include stimulants, atomoxetine, alpha-2 adrenergic receptor agonists, and sometimes antidepressants. [79][194] In those who have trouble focusing on long-term rewards, a large amount of positive reinforcement improves task performance. [198] Medications are the most effective treatment, [7][231] and any side effects are typically mild and easy to resolve [7] although any improvements will be reverted if medication is ceased. [232] ADHD stimulants also improve persistence and task performance in children with ADHD. [183][198] To quote one systematic review, "recent evidence from observational and registry studies indicates that pharmacological treatment of ADHD is associated with increased achievement and decreased absenteeism at school, a reduced risk of trauma-related emergency hospital visits, reduced risks of suicide and attempted suicide, and decreased rates of substance abuse and criminality". [233] Data also suggest that combining medication with CBT is a good idea: although CBT is substantially less effective, it can help address problems that reside after medication has been optimised. [7]

Behavioural therapies

There is good evidence for the use of <u>behavioural therapies</u> in ADHD. They are the recommended first-line treatment in those who have mild symptoms or who are preschool-aged. [234][235] Psychological therapies used include: psychoeducational input, behavior therapy, <u>cognitive behavioral therapy</u>, [236] interpersonal psychotherapy, family therapy, school-based interventions, social skills training, behavioural peer intervention, organization training, [237] and parent management training. [182] Neurofeedback has greater treatment effects than non-active controls for up to 6 months and possibly a year following treatment, and may have treatment effects comparable to active controls (controls proven to have a clinical effect) over that time period. [238] Despite efficacy in research, there is insufficient regulation of neurofeedback practice, leading to ineffective applications and false claims regarding innovations. [239] Parent training may improve a number of behavioural problems including oppositional and non-compliant behaviours. [240]

There is little high-quality research on the effectiveness of family therapy for ADHD—but the existing evidence shows that it is similar to community care, and better than placebo. [241] ADHD-specific support groups can provide information and may help families cope with ADHD. [242]

Social skills training, behavioural modification, and medication may have some limited beneficial effects in peer relationships. Stable, high-quality friendships with <u>non-deviant</u> peers protect against later psychological problems. [243]

Digital interventions

Several clinical trials have investigated the efficacy of digital therapeutics, particularly Akili Interactive Labs's video game-based digital therapeutic AKL-T01, marketed as EndeavourRx. The pediatric STARS-ADHD randomized, double-blind, parallel-group, controlled trial demonstrated that AKL-T01 significantly improved performance on the Test of Variables of Attention, an objective measure of attention and inhibitory control, compared to a control group after four weeks of at-home use. [244] A subsequent pediatric open-label study, STARS-Adjunct, published in Nature Portfolio's npj Digital Medicine evaluated AKL-T01 as an adjunctive treatment for children with ADHD who were either on stimulant medication or not on stimulant pharmacotherapy. Results showed improvements in ADHD-related impairment (measured by the Impairment Rating Scale) and ADHD symptoms after 4 weeks of treatment, with effects persisting during a 4-week pause and further improving with an additional treatment period. [245] Notably, the magnitude of the measured improvement was similar for children both on and off stimulants. [245] In 2020, AKL-T01 received marketing authorization for pediatric ADHD from the FDA, becoming "the first game-based therapeutic granted marketing authorization by the FDA for any type of condition."

In addition to pediatric populations, a 2023 study in the *Journal of the American Academy of Child & Adolescent Psychiatry* investigated the efficacy and safety of AKL-T01 in adults with ADHD. After six weeks of at-home treatment with AKL-T01, participants showed significant improvements in objective measures of attention (TOVA - Attention Comparison Score), reported ADHD symptoms (ADHD-RS-IV inattention subscale and total score), and reported quality of life (AAQoL). The magnitude of improvement in attention was nearly seven times greater than that reported in pediatric trials. The treatment was well-tolerated, with high compliance and no serious adverse events.

Medication

The medications for ADHD appear to alleviate symptoms via their effects on the pre-frontal executive, striatal and related regions and networks in the brain; usually by increasing neurotransmission of norepinephrine and dopamine. [248][249][250]

Stimulants

Methylphenidate and amphetamine or its derivatives are often first-line treatments for ADHD. About 70 per cent respond to the first stimulant tried and as few as 10 per cent respond to neither amphetamines nor methylphenidate. Stimulants may also reduce the risk of unintentional injuries in children with ADHD. Magnetic resonance imaging studies suggest that long-term treatment with amphetamine or methylphenidate decreases abnormalities in brain structure and function found in subjects with ADHD. A 2018 review found the greatest short-term benefit with methylphenidate in children, and amphetamines in adults. Studies and meta-analyses show that amphetamine is slightly-to-modestly more effective than methylphenidate at reducing symptoms, and they are more effective pharmacotherapy for ADHD than α2-agonists but methylphenidate has comparable efficacy to non-stimulants such as atomoxetine.

Safety and efficacy data have been reviewed extensively by medical regulators (e.g., the US Food and Drug Administration and the European Medicines Agency), the developers of evidence-based international guidelines (e.g., the UK National Institute for Health and Care Excellence and the American Academy of Pediatrics), and government agencies who have endorsed these guidelines (e.g., the Australian National Health and Medical Research Council). These professional groups unanimously conclude, based on the scientific evidence, that methylphenidate is safe and effective and should be considered as a first-line treatment for ADHD. [261]

The likelihood of developing <u>insomnia</u> for ADHD patients taking stimulants has been measured at between 11 and 45 per cent for different medications, [262] and may be a main reason for discontinuation. Other side effects, such as <u>tics</u>, decreased appetite and weight loss, or <u>emotional lability</u>, may also lead to discontinuation. Stimulant psychosis and <u>mania</u> are rare at therapeutic doses, appearing to occur in approximately 0.1% of individuals, within the first several weeks after starting amphetamine therapy. The safety of these medications in pregnancy is unclear. Symptom improvement is not sustained if medication is ceased. [267][232][268]

The long-term effects of ADHD medication have yet to be fully determined, $\frac{[269][270]}{}$ although stimulants are generally beneficial and safe for up to two years for children and adolescents. $\frac{[271]}{}$ A 2022 meta-analysis found no statistically significant association between ADHD medications and the risk of <u>cardiovascular disease</u> (CVD) across age groups, although the study suggests further investigation is warranted for patients with preexisting CVD as well as long-term medication use. $\frac{[272]}{}$ Regular monitoring has been recommended in those on long-term treatment. $\frac{[273]}{}$ There are indications suggesting that stimulant therapy for children and adolescents should be stopped periodically to assess continuing need for medication, decrease possible growth delay, and reduce tolerance. $\frac{[274][275]}{}$ Although potentially addictive at high doses, $\frac{[276][277]}{}$ stimulants used to treat ADHD have low potential for abuse. $\frac{[251]}{}$ Treatment with stimulants is either protective against substance abuse or has no effect. $\frac{[57]}{}$ 12[269][276]

The majority of studies on <u>nicotine</u> and other <u>nicotinic</u> agonists as treatments for ADHD have shown favorable results; however, no nicotinic drug has been approved for ADHD treatment. <u>[278]</u> <u>Caffeine</u> was formerly used as a second-line treatment for ADHD but research indicates it has no significant effects in reducing ADHD symptoms. Caffeine appears to help with alertness, arousal and reaction time but not the type of inattention implicated in ADHD (sustained attention/persistence). <u>[279]</u> <u>Pseudoephedrine</u> and ephedrine do not affect ADHD symptoms.

<u>Modafinil</u> has shown some efficacy in reducing the severity of ADHD in children and adolescents. [280] It may be prescribed off-label to treat ADHD.

Non-stimulants

Two non-stimulant medications, <u>atomoxetine</u> and <u>viloxazine</u>, are approved by the FDA and in other countries for the treatment of ADHD.

Atomoxetine, due to its lack of addiction liability, may be preferred in those who are at risk of recreational or compulsive stimulant use, although evidence is lacking to support its use over stimulants for this reason. [57]:13 Atomoxetine alleviates ADHD symptoms through norepinephrine reuptake and by indirectly increasing dopamine in the pre-frontal cortex, [250] sharing 70-80% of the brain regions with stimulants in their produced effects. [249] Atomoxetine has been shown to significantly improve academic

performance. [281][282] Meta-analyses and systematic reviews have found that atomoxetine has comparable efficacy, equal tolerability and response rate (75%) to methylphenidate in children and adolescents. In adults, efficacy and discontinuation rates are equivalent. [283][284][285][286]

Analyses of clinical trial data suggests that <u>viloxazine</u> is about as effective as atomoxetine and methylphenidate but with fewer side effects. [287]

<u>Amantadine</u> was shown to induce similar improvements in children treated with <u>methylphenidate</u>, with less frequent side effects. [288] A 2021 retrospective study showed showed that amantadine may serve as an effective adjunct to stimulants for ADHD–related symptoms and appears to be a safer alternative to second-or third-generation antipsychotics. [289]

 $\underline{\underline{Bupropion}}$ is also used off-label by some clinicians due to research findings. It is effective, but modestly less than atomoxetine and methylphenidate. [290]

There is little evidence on the effects of medication on social behaviours. [291] Antipsychotics may also be used to treat aggression in ADHD. [292]

Alpha-2a agonists

Two <u>alpha-2a agonists</u>, extended-release formulations of <u>guanfacine</u> and <u>clonidine</u>, are approved by the FDA and in other countries for the treatment of ADHD (effective in children and adolescents but effectiveness has still not been shown for adults). [293][294] They appear to be modestly less effective than the stimulants (amphetamine and methylphenidate) and non-stimulants (atomoxetine and viloxazine) at reducing symptoms, [295][296] but can be useful alternatives or used in conjunction with a stimulant. These medications act by adjusting the alpha-2a ports on the outside of noradrenergic nerve cells in the pre-frontal executive networks, so the information (electrical signal) is less confounded by noise. [297]

Guidelines

Guidelines on when to use medications vary by country. The United Kingdom's National Institute for Health and Care Excellence recommends use for children only in severe cases, though for adults medication is a first-line treatment. Conversely, most United States guidelines recommend medications in most age groups. Medications are especially not recommended for preschool children. Underdosing of stimulants can occur, and can result in a lack of response or later loss of effectiveness. This is particularly common in adolescents and adults as approved dosing is based on school-aged children, causing some practitioners to use weight-based or benefit-based off-label dosing instead.

Exercise

Exercise does not reduce the symptoms of ADHD. The conclusion by the International Consensus Statement is based on two meta-analyses: one of 10 studies with 300 children and the other of 15 studies and 668 participants, which showed that exercise yields no statistically significant reductions on ADHD symptoms. A 2024 systematic review and meta analysis commissioned by the Patient-Centered Outcomes Research Institute (PCORI) identified seven studies on the effectiveness of physical exercise for treating ADHD symptoms. The type and amount of exercise varied widely across studies from martial arts

interventions to treadmill training, to table tennis or aerobic exercise. Effects reported were not replicated, causing the authors to conclude that there is insufficient evidence that exercise intervention is an effective form of treatment for ADHD symptoms. [203]

Diet

Dietary modifications are not recommended as of 2019 by the American Academy of Pediatrics, the National Institute for Health and Care Excellence, or the Agency for Healthcare Research and Quality due to insufficient evidence. [304][298] A 2013 meta-analysis found less than a third of children with ADHD see some improvement in symptoms with free fatty acid supplementation or decreased consumption of artificial food colouring. [162] These benefits may be limited to children with food sensitivities or those who are simultaneously being treated with ADHD medications. [162] This review also found that evidence does not support removing other foods from the diet to treat ADHD. [162] A 2014 review found that an elimination diet results in a small overall benefit in a minority of children, such as those with allergies. [177] A 2016 review stated that the use of a gluten-free diet as standard ADHD treatment is not advised. [226] A 2017 review showed that a few-foods elimination diet may help children too young to be medicated or not responding to medication, while free fatty acid supplementation or decreased eating of artificial food colouring as standard ADHD treatment is not advised. [305] Chronic deficiencies of iron, magnesium and iodine may have a negative impact on ADHD symptoms. [306] There is a small amount of evidence that lower tissue zinc levels may be associated with ADHD.[307] In the absence of a demonstrated zinc deficiency (which is rare outside of developing countries), zinc supplementation is not recommended as treatment for ADHD. [308] However, zinc supplementation may reduce the minimum effective dose of amphetamine when it is used with amphetamine for the treatment of ADHD. [309]

Prognosis

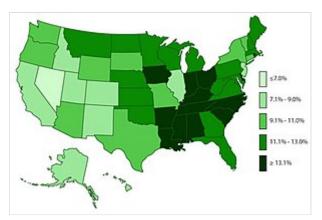
ADHD persists into adulthood in about 30–50% of cases. [310] Those affected are likely to develop coping mechanisms as they mature, thus compensating to some extent for their previous symptoms. [222] Children with ADHD have a higher risk of unintentional injuries. [253] Effects of medication on functional impairment and quality of life (e.g. reduced risk of accidents) have been found across multiple domains. [311] Rates of smoking among those with ADHD are higher than in the general population at about 40%. [312]

About 30–50% of people diagnosed in childhood continue to have <u>ADHD</u> in adulthood, with 2.58% of adults estimated to have ADHD which began in childhood. [220][313] In adults, hyperactivity is usually replaced by inner <u>restlessness</u>, and adults often develop <u>coping</u> skills to compensate for their impairments. The condition can be difficult to tell apart from other conditions, as well as from high levels of activity within the range of normal behaviour. ADHD has a negative impact on patient health-related quality of life that may be further exacerbated by, or may increase the risk of, other psychiatric conditions such as anxiety and depression. [233] Individuals with ADHD may also face misconceptions and stigma. [7]

Individuals with ADHD are significantly overrepresented in prison populations. Although there is no generally accepted estimate of ADHD prevalence among inmates, a 2015 meta-analysis estimated a prevalence of 25.5%, and a larger 2018 meta-analysis estimated the frequency to be 26.2%. [314]

Epidemiology

ADHD is estimated to affect about 6–7% of people aged 18 and under when diagnosed via the DSM-IV criteria. [316] When diagnosed via the ICD-10 criteria, rates in this age group are estimated around 1–2%. [317] Rates are similar between countries and differences in rates depend mostly on how it is diagnosed. [318] Children in North America appear to have a higher rate of ADHD than children in Africa and the Middle East; this is believed to be due to differing methods of diagnosis rather than a difference in underlying frequency. (The same publication which describes this difference also notes that the difference may be rooted in the available studies from these respective regions, as far more studies were from North America than



Percentage of people 4–17 ever diagnosed in the US as of $2011^{\boxed{[315]}}$

from Africa and the Middle East.)^[319] As of 2019, it was estimated to affect 84.7 million people globally.^[2]

ADHD is diagnosed approximately twice as often in boys as in girls, [4][316] and 1.6 times more often in men than in women, although the disorder is overlooked in girls or diagnosed in later life because their symptoms sometimes differ from diagnostic criteria. [323][324]

Studies from multiple countries have reported that children born closer to the start of the school year are more frequently diagnosed with and medicated for ADHD than their older classmates. Boys who were born in December where the school age cut-off was 31 December were shown to be 30% more likely to be diagnosed and 41% more likely to be treated than those born in January. Girls born in December had a diagnosis and treatment percentage increase of 70% and 77% respectively compared to those born in January. Children who were born at the last three days of a calendar year were reported to have significantly higher levels of diagnosis and treatment for ADHD than children born at the first three days of a calendar year. The studies suggest that ADHD diagnosis is prone to subjective analysis. [326]

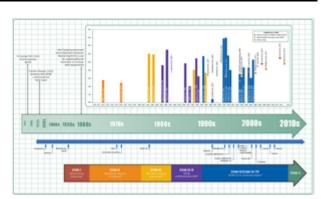
Rates of diagnosis and treatment have increased in both the United Kingdom and the United States since the 1970s. Prior to 1970, it was rare for children to be diagnosed with ADHD, while in the 1970s rates were about $1\%.^{[327]}$ This is believed to be primarily due to changes in how the condition is diagnosed and how readily people are willing to treat it with medications rather than a true change in incidence. [317]

Despite showing a higher frequency of symptoms associated with ADHD, <u>non-White</u> children in the US are less likely than <u>White</u> children to be diagnosed or treated for ADHD, a finding that is often explained by bias among health professionals, as well as parents who may be reluctant to acknowledge that their child has ADHD. Crosscultural differences in diagnosis of ADHD can also be attributed to the long-lasting effects of harmful, racially targeted medical practices. Medical pseudosciences, particularly those that targeted Black populations during the period of slavery in the US, lead to a distrust of medical practices

within certain communities. The combination of ADHD symptoms often being regarded as misbehaviour rather than as a psychiatric condition, and the use of drugs to regulate ADHD, result in a hesitancy to trust a diagnosis of ADHD. Cases of misdiagnosis in ADHD can also occur due to stereotyping of people of color. Due to ADHD's subjectively determined symptoms, medical professionals may diagnose individuals based on stereotyped behaviour or misdiagnose due to cultural differences in symptom presentation. [330]

History

Sir <u>Alexander Crichton</u> describes "mental restlessness" in his book *An inquiry into the nature and origin of mental derangement* written in 1798. [331][332] He made observations about children showing signs of being inattentive and having the "fidgets". The first clear description of ADHD is credited to <u>George Still</u> in 1902 during a series of lectures he gave to the Royal College of Physicians of London. [333][328] He noted both nature and nurture could be influencing this disorder.



Timeline of ADHD diagnostic criteria, prevalence, and treatment

ADHD was officially known as **attention deficit disorder** (**ADD**) from 1980 to 1987; prior to the

1980s, it was known as **hyperkinetic reaction of childhood**. Symptoms similar to those of ADHD have been described in medical literature dating back to the 18th century.

<u>Alfred Tredgold</u> proposed an association between brain damage and behavioural or learning problems which was able to be validated by the encephalitis lethargica epidemic from 1917 through 1928. [334][335][336]

The terminology used to describe the condition has changed over time and has included: *minimal brain dysfunction* in the DSM-I (1952), *hyperkinetic reaction of childhood* in the DSM-II (1968), and *attention-deficit disorder with or without hyperactivity* in the DSM-III (1980). In 1987, this was changed to ADHD in the DSM-III-R, and in 1994 the DSM-IV in split the diagnosis into three subtypes: ADHD inattentive type, ADHD hyperactive-impulsive type, and ADHD combined type. These terms were kept in the DSM-5 in 2013 and in the DSM-5-TR in 2022. Prior to the DSM, terms included *minimal brain damage* in the 1930s.

In 1934, Benzedrine became the first amphetamine medication approved for use in the United States. [339] Methylphenidate was introduced in the 1950s, and enantiopure dextroamphetamine in the 1970s. [328] The use of stimulants to treat ADHD was first described in 1937. [340] Charles Bradley gave the children with behavioural disorders Benzedrine and found it improved academic performance and behaviour. [341][342]

Once neuroimaging studies were possible, studies in the 1990s provided support for the pre-existing theory that neurological differences (particularly in the <u>frontal lobes</u>) were involved in ADHD. A genetic component was identified and ADHD was acknowledged to be a persistent, long-term disorder which lasted from childhood into adulthood. [343][344]

ADHD was split into the current three sub-types because of a field trial completed by Lahey and colleagues and published in 1994. In 2021, global teams of scientists curated the International Consensus Statement compiling evidence-based findings about the disorder.

Controversy

ADHD, its diagnosis, and its treatment have been controversial since the 1970s. [232][6] The controversies involve clinicians, teachers, policymakers, parents, and the media. Positions range from the view that ADHD is within the normal range of behaviour [90][346] to the hypothesis that ADHD is a genetic condition. [347] Other areas of controversy include the use of stimulant medications in children, [232] the method of diagnosis, and the possibility of overdiagnosis. [348] In 2009, the National Institute for Health and Care Excellence, while acknowledging the controversy, states that the current treatments and methods of diagnosis are based on the dominant view of the academic literature. [349] In 2014, Keith Conners, one of the early advocates for recognition of the disorder, spoke out against overdiagnosis in a New York Times article. [350] In contrast, a 2014 peer-reviewed medical literature review indicated that ADHD is underdiagnosed in adults. [313]

The diagnosis of ADHD has been criticised as being subjective because it is not based on a biological test. The International Consensus Statement on ADHD concluded that this criticism is unfounded, on the basis that ADHD meets standard criteria for validity of a mental disorder established by Robins and Guze. They attest that the disorder is considered valid because: 1) well-trained professionals in a variety of settings and cultures agree on its presence or absence using well-defined criteria and 2) the diagnosis is useful for predicting a) additional problems the patient may have (e.g., difficulties learning in school); b) future patient outcomes (e.g., risk for future drug abuse); c) response to treatment (e.g., medications and psychological treatments); and d) features that indicate a consistent set of causes for the disorder (e.g., findings from genetics or brain imaging), and that professional associations have endorsed and published guidelines for diagnosing ADHD. [7]

With widely differing rates of diagnosis across countries, states within countries, races, and ethnicities, some suspect factors other than symptoms of ADHD are playing a role in diagnosis, such as cultural norms. [351][326] Some sociologists consider ADHD to be an example of the <u>medicalization</u> of deviant behaviour, that is, the turning of the previously non-medical issue of school performance into a medical one. [352] Among healthcare providers, debate mainly centres on diagnosis and treatment in the much greater number of people with mild symptoms and/or non-existent impairment. [173][353][354]

The nature and range of desirable endpoints of ADHD treatment vary among diagnostic standards for ADHD. [355] In most studies, the efficacy of treatment is determined by reductions in symptoms. [356] However, some studies have included subjective ratings from teachers and parents as part of their assessment of treatment efficacies. [257] By contrast, the subjective ratings of children undergoing ADHD treatment are seldom included in studies evaluating the efficacy of ADHD treatments.

Methylphenidate

In a Cochrane clinical synopsis, Dr Storebø and colleagues [357] summarised their meta-review [358] on methylphenidate for ADHD in children and adolescents. The meta-analysis raised substantial doubts about the drug's efficacy relative to a placebo, perpetuating controversy among clinicians and in media reports.

This led to a strong critical reaction from the European ADHD Guidelines Group and individuals in the scientific community; they identified a number of flaws in the review, including its use of idiosyncratic methods to assess the quality of evidence, unethical misinterpretations of the evidence in relation to adverse effects, and errors in the extraction of data, thereby resulting in misplaced conclusions. [359][360][361][362][363][364] Since at least September 2021, there is a unanimous and global scientific consensus that methylphenidate is safe and highly effective for treating ADHD. [7][365]

The same journal released a subsequent review (2022) of extended-release methylphenidate for adults, concluding similar doubts about the certainty of evidence. Other recent systematic reviews and meta-analyses, however, find certainty in the safety and high efficacy of methylphenidate for reducing ADHD symptoms for substantially reducing the adverse consequences of untreated ADHD with continuous treatment. Clinical guidelines internationally are also consistent in approving the safety and efficacy of methylphenidate and recommending it as a first-line treatment for the disorder.

Research directions

Possible positive traits

Possible positive traits of ADHD are a new avenue of research, and therefore limited.

A 2020 review found that creativity <u>may be associated</u> with ADHD symptoms, particularly <u>divergent thinking</u> and quantity of creative achievements, but not with the disorder of ADHD itself – i.e. it has not been found to be increased in people diagnosed with the disorder, only in people with subclinical symptoms or those that possess traits associated with the disorder. Divergent thinking is the ability to produce creative solutions which differ significantly from each other and consider the issue from multiple perspectives. Those with ADHD symptoms could be advantaged in this form of creativity as they tend to have diffuse attention, allowing rapid switching between aspects of the task under consideration; flexible <u>associative memory</u>, allowing them to remember and use more distantly-related ideas which is associated with creativity; and impulsivity, allowing them to consider ideas which others may not have. [372]

Possible biomarkers for diagnosis

Reviews of ADHD <u>biomarkers</u> have noted that platelet <u>monoamine oxidase</u> expression, urinary <u>norepinephrine</u>, urinary <u>MHPG</u>, and urinary <u>phenethylamine</u> levels consistently differ between ADHD individuals and non-ADHD controls. These measurements could serve as diagnostic biomarkers for ADHD, but more research is needed to establish their diagnostic utility. Urinary and <u>blood plasma</u> phenethylamine concentrations are lower in ADHD individuals relative to controls. The two most

commonly prescribed drugs for ADHD, <u>amphetamine</u> and <u>methylphenidate</u>, increase phenethylamine <u>biosynthesis</u> in treatment-responsive individuals with ADHD. Lower urinary phenethylamine concentrations are associated with symptoms of inattentiveness in ADHD individuals.

See also

- Directed attention fatigue a temporary state sharing many of the symptoms of ADHD
- Self-medication

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