Attention-Deficit/Hyperactivity Disorder

CHAPTER 19

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Definition and Classification

Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental condition characterised by persistent patterns of inattention and/or hyperactivity-impulsivity that interfere with functioning and are inappropriate for an individual's developmental stage.[1] Symptoms typically present before 12 years of age, though clinical identification may extend into adolescence or adulthood.

The two primary international diagnostic frameworks, the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5),[2] and the International Classification of Diseases, 11th Revision (ICD-11),[3] classify ADHD into three main presentations based on symptom profile:[1, 4]

- **Predominantly inattentive presentation:** Characterised by difficulties with focus, organisation, and sustaining attention on tasks lacking frequent stimulation or rewards.
- **Predominantly hyperactive-impulsive presentation:** Characterised by excessive motor movement, difficulty remaining still, and acting on immediate stimuli without evaluating risks or consequences.
- **Combined presentations:** Meeting diagnostic criteria for both inattentive and hyperactive-impulsive domains.

Core diagnostic criteria require symptom persistence for at least six months across multiple settings, such as home, academic, occupational, or social environments. Although ADHD can be identified at any age, its developmental onset occurs in childhood, requiring evidence of early symptoms when evaluating adolescents or adults.[1, 4] The major international frameworks operationalise these criteria differently: the DSM-5 enforces specific numerical thresholds, requiring at least six of nine symptoms in either the inattentive or the hyperactive-impulsive domain for children, and at least five for individuals aged 17 and older. By contrast, the ICD-11 includes fewer symptom threshold requirements allowing for more flexibility and clinical judgment.[4]

Aetiology

The aetiology of ADHD is complex, arising primarily from genetic influences combined with environmental risk factors and gene-environment interactions. In most individuals with ADHD, no single causal factor is identified (Figure 1); rather, multiple biological and environmental exposures cumulatively increase susceptibility to developing the disorder.[4, 5]

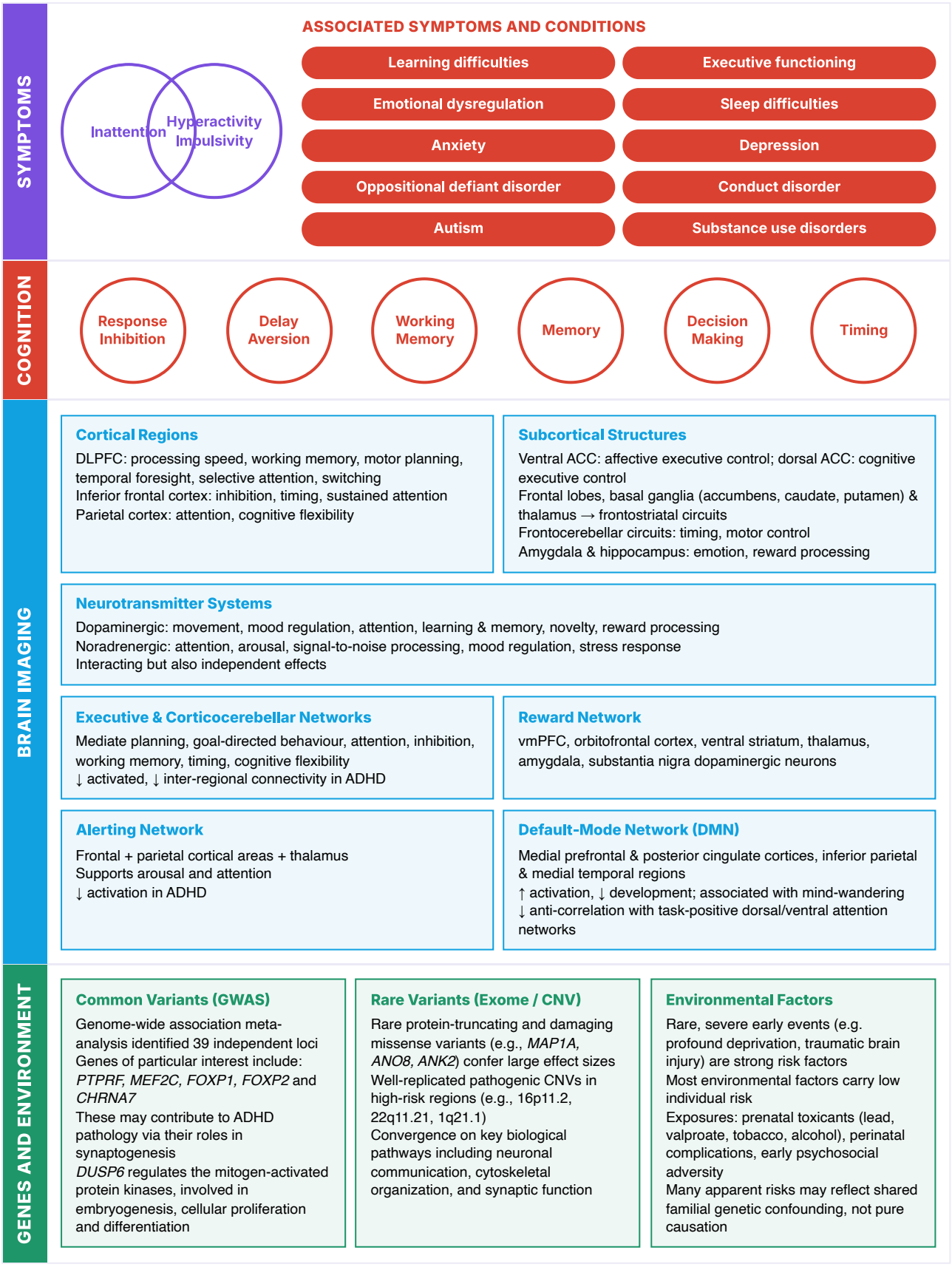


Figure 1 - Aetiological Framework for ADHD: ADHD has a complex and heterogeneous aetiology that is not possible to fully capture in one schematic. For this reason, while this figure highlights some key features across the different levels of analysis, it should not be interpreted as definitive or comprehensive. Timing – includes time perception and reproduction; ACC - Anterior Cingulate Cortex; DLPFC – Dorsolateral Prefrontal Cortex; vmPFC – Venteromedial Prefrontal Cortex; CNV – Copy Number Variant; GWAS – Genome Wide Association Study.

Meta-analyses of twin and family studies consistently estimate heritability around 70-80% across sexes, age groups, and symptom domains.[6-10] Genetic factors influence both clinical ADHD diagnoses and trait variations within the general population, supporting a continuous liability model whereby the genetic influences impacting on attention, activity and impulsivity are distributed continuously across the general population with those meeting criteria for ADHD falling at the extreme end of this continuum.[5, 11, 12] Furthermore, widespread pleiotropy exists between ADHD and broad phenotypic domains, including educational attainment, cognitive performance, cardiometabolic traits (e.g., obesity), and psychiatric comorbidities (e.g., major depression, autism spectrum disorder, conduct disorder, and substance use disorder).[5, 9, 13-15] This shared genetic architecture helps explains multifinality, where individuals with similar underlying genetic risk, such as biological siblings, may present with distinct clinical outcomes.

Molecular genetic studies confirm a polygenic architecture comprising common single-nucleotide polymorphisms (SNPs) and rare variants. Common SNPs account for 11-20% of phenotypic variance,[6, 16] with a recent genome-wide association meta-analysis identifying 39 independent genomic risk loci and prioritising 22 potential effector genes, identifying eight novel gene targets (e.g., *EMCN*, *STK32C*, *PCDH17*, *TCF12*, *PEAK1*, *IGF1R*, *CTNNA2*, and *ABCA12*) alongside established neurodevelopmental genes (e.g., *FOXP1*, *FOXP2*, *MEF2C*).[17] In parallel, exome sequencing and copy number variation (CNV) studies reveal an increased burden of rare coding mutations (e.g., *MAP1A*, *ANK2*) and structural variants (e.g., 15q11-13, 22q11.21).[18-21] These rare variants overlap with genetic liabilities for autism spectrum disorder and schizophrenia, implicating pathways in nicotinic signalling (*CHRNA7*) and dopamine regulation (*PRKN*).

Major clinical guidelines from the American Academy of Pediatrics (AAP) and the National Institute for Health and Care Excellence (NICE), alongside the World Federation of ADHD International Consensus Statement, identify key early environmental correlates for ADHD.[9, 22, 23] While rare, severe, early-life adverse events, such as profound deprivation or early traumatic brain injury act as strong aetiological risk factors,[24, 25] most of the environmental factors associated with ADHD carry low individual risk ratios.[9, 10, 26] Implicated exposures include prenatal toxicants and substances (e.g., lead, valproate, tobacco, and alcohol), perinatal complications (e.g., prematurity and low birth weight), and early psychosocial adversity.[9, 10] However, genetically informative studies and major consensus reviews have highlighted that many apparent environmental risks seem to reflect shared familial genetic confounding rather than pure causation.[27] For instance, epidemiological links like maternal prenatal smoking substantially attenuate or disappear in sibling-control and oocyte-donation studies when parental genetic liability is accounted for.[28, 29] Overall, these non-specific exposures contribute broadly to general neurodevelopmental vulnerability.

Neuroimaging and neurocognitive studies have identified clear group level differences in brain structure and function, and neurocognitive performance between individuals with ADHD and their typically developing peers.[30-32] However, as with the genetic data, there is also evidence for both multifinality and equifinality across these levels of analysis. The data highlight significant both within disorder heterogeneity across all these domains, and considerable overlap between the findings for ADHD and those for other mental health conditions.[33, 34] At the neurocognitive level there is also evidence for a dissociation between cognition and core ADHD symptoms.[35] Taken together these findings attest to the complexity of the biological underpinnings and suggest that we will need a greater degree of granularity to unravel the causal pathways leading to the clinical syndrome we recognise as ADHD.

Epidemiology

ADHD is the most prevalent neurodevelopmental disorder in children and adolescents globally.[36-38] Global epidemiological meta-analyses estimate the worldwide community prevalence of ADHD at approximately 5% to 8% in children and adolescents, and roughly 2.5% in adults.[9] Meta-analytic reviews of the epidemiological data suggest that, notwithstanding a significant increase in rates of diagnosis (administrative prevalence)[39] this almost certainly reflects increased awareness and changes in clinical practice as there is no evidence for an increase in epidemiological prevalence.[40]

While molecular genetic studies show that the underlying heritability and genetic architecture of ADHD are largely equivalent across sexes, the clinical presentation, diagnostic rates, and referral patterns exhibit notable sex and gender differences across the lifespan. In paediatric community samples, the male-to-female ratio ranges from 2:1 to roughly 3:1, but climbs to 4:1 or higher in clinical referral settings.[5, 41, 42] This discrepancy is driven largely by phenotypic expression and ascertainment bias, where males frequently manifest overt, disruptive hyperactivity that prompts early clinical evaluation, whereas females often present with inattentive traits, internalising symptoms, and compensatory masking, frequently resulting in delayed identification or misdiagnosis.[43] As individuals transition into adulthood, the diagnosed gender ratio converges toward near-parity, approaching 1.1:1 in population surveys.[44] This convergence reflects a shift toward self-referral, greater healthcare utilisation among adult women, and diagnostic criteria that place less emphasis on overt motor hyperactivity, better capturing the more subtle adult female presentation.

Presentation and Course of Illness

The clinical presentation of ADHD evolves across the lifespan, adapting to changing developmental demands. In early childhood, the presentation is frequently dominated by hyperactive and impulsive behaviours, manifesting as motor restlessness, constant motion, and difficulty remaining seated.[45, 46]

As individuals enter formal schooling, overt physical hyperactivity typically diminishes, whereas cognitive demands of the classroom expose marked deficits in sustained attention, working memory, organisation, and task completion. In adolescence, physical hyperactivity often transforms into a subjective internal restlessness, while executive dysfunction and emotional dysregulation become more prominent, frequently contributing to academic underachievement, peer conflict, and risk-taking behaviours.[2, 46, 47]

In adulthood, core symptoms frequently internalise, manifesting as subtle fidgeting, inner tension, or an inability to relax.[48] Adults with ADHD commonly report severe difficulties with time management, task prioritisation, and sustained executive control. As diagnostic criteria are historically anchored in childhood behavioural markers, adult presentations can be more subtle, often identified only when secondary functional impairments emerge in academic, occupational, or interpersonal domains.[48]

The longitudinal course of ADHD is characterised by high rates of psychiatric comorbidity across the lifespan.[49] While early prospective longitudinal cohorts demonstrate that at least 70% individuals diagnosed in childhood continue to experience symptoms into adulthood, highlighting ADHD as a chronic, lifelong condition,[50] more recent data from the Multimodal Treatment Study of ADHD (MTA) suggests that the persistence of ADHD across development is rarely a simple, fixed path. They found that around 90% of individuals continue to experience residual or recurring symptoms into young adulthood, with symptoms typically fluctuating over time[51]. This trajectory is frequently compounded by high rates of neurodevelopmental and psychiatric comorbidities, with approximately 50% meeting criteria for at least one additional comorbid condition,[52, 53] such as autism spectrum disorder, anxiety, depression, conduct disorder, and substance use disorder,[54]which further complicate the long-term clinical course.

Socio-economic Implications and Disease Burden

Beyond its direct clinical manifestations, ADHD carries significant, and frequently unrecognised, socioeconomic consequences for individuals, families, healthcare systems, and society as a whole. According to the Global Burden of Disease (GBD) analysis, ADHD resulted in 3.73 million global disability-adjusted life years (DALYs) in 2023, accounting for 2.2% of the total global mental disorder DALYs.[55] However, these figures likely underestimate the true burden due to conservative estimates of prevalence and disability weights used in the calculations. Systematic reviews and cost-of-illness studies consistently demonstrate that healthcare utilisation and health system costs per individual are substantially higher in children with ADHD than in their unaffected counterparts.[56-59] From a broader societal perspective, high rates of grade retention, special education allocation, disciplinary suspensions, and school dropouts place heavy demands on educational budgets. Individual and caregiver presenteeism and absenteeism at work, missed opportunities, and unemployment translate into substantial productivity losses and indirect costs to society. Furthermore, epidemiological evidence indicates that individuals with ADHD experience higher rates of involvement with the criminal justice system, including increased police proceedings, court charges or convictions, and higher rates of incarcerations compared to the general population.[57] Health economic evaluations highlight the multisectoral burden of ADHD.[56, 57] A systematic review estimated the annual per-person societal cost of ADHD to range from $US831 to $US20,538, with national estimates spanning from $US356 million in Denmark to $US20.27 billion in the United States in 2019 values.[56]

Clinical Journey

Recognition and identification

The clinical journey begins with recognising difficulties that may benefit from additional supports. These are commonly first noticed by parents, teachers, or the individual themselves, often after recognising persistent challenges at home, school, or work. Alternatively, ADHD may be suspected when an individual seeks help from a healthcare professional for unrelated difficulties and it is recognised that the history and presentation may be consistent with ADHD.[5] Recognition within a community setting will generally prompt an

initial consultation with a General Practitioner or other healthcare professional, who will determine whether referral for a formal ADHD assessment is appropriate.[60]

Public awareness of ADHD has increased substantially in recent years, particularly through the growing availability of information online and across social media, however it is increasingly recognised that the information available through popular social media channels is often inaccurate and misleading. While globally ADHD is still very much under-recognised and under treated there is now a tension in some communities between continued missed diagnosis and increasing rates of self-diagnosis and referral based on online screeners and advertising. It is also the case that many of the professionals who are relied on to first recognise an individual's difficulties, namely education staff, allied health, and GPs have received limited training in ADHD.[5, 60]

Cultural factors also influence the recognition of ADHD. Individuals from minority groups and culturally diverse communities may be less likely to have their difficulties recognised and subsequently be referred for assessment.[61] Cultural beliefs and understanding of mental illness, as well as experiences of stigma, can also influence whether difficulties are recognised as problematic, whether they might be attributed to ADHD, and whether help is sought.[61]

A separate but no less important aspect of identification has been highlighted through systematic reviews of both traditional questionnaire-based screening and the more recent focus on the use of computerised continuous performance tasks (CPT) as potential screeners for ADHD. [62, 63] These have identified that while both approaches have reasonable sensitivity, around 0.8, their specificity, also around 0.8, is lower than required and results in many false positives. In practice this would mean that, if assume a prevalence of 5% and screen 100 individuals, while 4 out of the 5 with ADHD would screen positive, there would be 19 false positives. Modern evidence-based guidelines therefore recommend against using these approaches as standalone screeners. If you are going to use either approach in a clinical setting it is important to augment with clinical questioning otherwise there is a high possibility that assessment services will be inundated with referrals of individuals who do not have ADHD and there will be a significant risk that some of these people will receive a diagnosis and be offered treatment. There is some early indication that augmented approaches to screening will improve accuracy, but these are still in development and further studies are required.[64]

Improving the identification of ADHD will require not only more accurate approaches to screening but also greater public and professional understanding of the condition, including its symptoms and diverse presentations. Accurate education is particularly important for parents, education staff, and healthcare professionals who are the gatekeepers for assessment. Culturally appropriate efforts to improve recognition in under-represented and culturally diverse communities is also needed.

The ADHD Clinical Journey

1 Recognition and Identification

- Difficulties first noticed by parents, teachers, the individual themselves, or during clinical care for unrelated concerns
- A GP or other healthcare professional decides whether to refer for formal assessment
- Recognition can be missed or delayed due to limited clinician training, inattentive presentations overlooked (especially in girls), and cultural or stigma barriers
- Improved screening approaches and greater public and professional understanding are needed to support accurate identification

2 Assessment

- A comprehensive process conducted by trained specialists (psychiatrists, paediatricians, psychologists or trained GPs)
- Core component: a structured clinical interview with the client/family covering medical and developmental history, symptoms, and functioning
- Questionnaires and cognitive tests support the interview but should never replace it

3 Clinical Formulation and Diagnosis

- Synthesises assessment findings into a holistic understanding and not just a diagnostic label
- Considers differential diagnoses and biopsychosocial contributing factors (predisposing, precipitating, perpetuating, protective)

4 Treatment Planning

- A collaborative, individually tailored plan agreed with the client/family
- Treatment goals are prioritised, with symptom reduction usually first

5 ADHD Management

- **Psychoeducation:** a foundation for everyone diagnosed. Both to explain ADHD and corrects misinformation
- **Pharmacological treatment:** extended-release stimulants are usually first-line, with individualised selection and titration
- **Non-pharmacological support:** CBT, parent training, and other approaches to improve broader functioning
- A multimodal combination of these approaches is generally recommended

6 Continuing Care and Ongoing Support

- ADHD is often lifelong, needing regular monitoring and review
- Purpose is to track treatment response, adherence, and adverse effects over time
- Measurement-based care helps guide ongoing adjustments

Figure 2 - The clinical journey for ADHD.

Assessment

Assessment for ADHD should be seen as one part of a comprehensive and systematic clinical process. Those assessing for ADHD require specialised skills and training. The purpose of the assessment is not simply to determine whether an individual does or does not meet the criteria for a categorical diagnosis of ADHD based on the DSM or ICD criteria. Rather, it should be broad enough to develop a meaningful holistic understanding of an individual, be able to identify strengths, determine whether any co-occurring conditions are present and, where relevant, whether these occur in addition to ADHD or whether they are a better explanation for the ADHD presentation.

Depending on the healthcare system and an individual's age, assessments are typically conducted by psychiatrists, developmental paediatricians, clinical psychologists, or neuropsychologists. In some countries GPs who pursue specialised training are also permitted to independently assess, diagnose, and initiate and monitor treatment for ADHD.

Clinical interview

The clinical interview is the core of an ADHD assessment. It is best when conducted as a structured or semi-structured interview with the parent and child, or for adults the client and a collateral informant. It should include both general and ADHD-specific elements. General information to obtain includes medical and developmental history, family and upbringing, and the different domains of their life (e.g., work, school, home life, relationships etc). To ensure comprehensiveness, the interview should not only seek to understand the negative aspects of these details but also the positive aspects, including an individual's strengths and any environmental factors that may support their functioning.[4] The ADHD-component of the interview primarily focuses on the nature and course of symptoms; that is, the type of symptoms, their onset, whether they have changed over time, and in what settings they occur (e.g., work, home, school, etc).[4, 23] Equally important is to establish whether these symptoms cause functional impairments and their impact on daily life.

While parent-administered interviews are typically the norm for child and adolescent assessments, the young person should also be given an opportunity to participate in the assessment process where appropriate [4, 23, 65]. For instance, a clinician may conduct a second more developmentally appropriate interview with the young person to allow them to describe their difficulties in their own words. This can be valuable because it can provide additional insights that parents may be unaware of about their child, while also providing an opportunity for the clinician to conduct a behavioural assessment on the young person's behaviour and presentation.

Concerns have been voiced recently about for-profit telehealth services offering assessments for ADHD that do not meet the current standards of care. Many of these are reported to rely on brief assessments that rely heavily on questionnaire data, do not include collateral information and, typically, make treatment recommendations, but do not offer ongoing care.

Questionnaires and rating scales

Clinicians often incorporate rating scales to supplement the interview. These can be used to assess core ADHD symptoms and severity, associated impairment, and broader psychopathology.[5] While adult clients typically complete these as self-reports, parent and teacher reports should be relied on for child and adolescent assessments. However, it is important to emphasise that because of the issues with specificity described above, questionnaire tools should never replace a clinical interview or be used in isolation to establish an ADHD diagnosis.[63] Not only can they produce high false-positive rates, but they do not adequately capture the complexities of an individual's presentation or determine whether another condition better explain the presenting issue.[66] As such, scores from these tools should always be interpreted alongside information gathered from the clinical interview and other relevant components of the assessment.

Cognitive tests

Some clinicians incorporate measures of cognitive and intellectual functioning into ADHD assessments to inform diagnosis decisions. However, this can be problematic given there is limited evidence to suggest that cognitive functioning is a good indicator of ADHD core symptoms .[67] Cognitive/intellectual assessments should therefore not be used as a diagnostic tool to determine whether an individual has ADHD.[4] However, information about cognitive and intellectual functioning can contribute to a more detailed understanding of an individual's strengths and weaknesses, which, at times, can help explain difficulties in educational or occupational contexts. This information can also inform learning accommodations and tailored support in these environments.

Clinical formulation and diagnosis

After gathering the assessment information, the next step is to develop a clinical formulation and reach a diagnostic conclusion. The formulation process involves synthesising and interpreting the assessment findings to develop an understanding of the individual's difficulties within the broader context of their life. Its purpose extends beyond merely determining whether the individual has ADHD, by considering how and why their difficulties may have developed and are being maintained. It also provides the opportunity for clinicians to consider differential diagnoses and alternative explanations for their presentation.

Applying a biopsychosocial approach can be useful during the formulation process by considering biological, psychological, and social influences. This may involve considering predisposing factors that increase vulnerability (e.g., genetic or familial factors), precipitating factors that contribute to the emergence or worsening of difficulties (e.g., significant life changes or increased demands), perpetuating factors that maintain them (e.g., sleep problems or stress), and protective factors that support positive functioning (e.g., supportive relationships or structured environments). Broader contextual factors such as trauma, family difficulties, financial hardship, or relationship problems should also be considered in understanding the individual's presentation.

Treatment planning

It is then important to develop a shared treatment plan. This is a collaborative process in which the clinician and client/family work together to establish treatment goals and consider appropriate treatment options. To be effective the treatment plan should be realistic, feasible, and tailored to the individuals' circumstances, considering factors such as age, clinical factors, co-occurring conditions, and relevant environmental or cultural factors.[5] The treatment plan must be endorsed by the client and/or family. This can be achieved through shared-decision making and encouraging clients to express their preferences, goals, or concerns.

As individuals with ADHD commonly experience difficulties across multiple domains, it is important to prioritise and order treatment goals when they cannot all be targeted at once. Priorities may be determined by the severity and impact of each difficulty, their actual and perceived importance, and the availability of treatment options. While improved functioning and quality of life are the ultimate goal, given that the core ADHD symptoms can often contribute to other associated difficulties, symptom reduction is generally the first treatment priority.

ADHD Management

Following diagnosis, all individuals will generally require some form of intervention to manage their ADHD. The delivery of ADHD care varies considerably within and across countries,[68] and recommendations regarding the sequencing and combination of treatments also differ across clinical guidelines. Nevertheless, there is broad support for a multimodal approach to ADHD management, incorporating both pharmacological and non-pharmacological interventions.[4, 69, 70]

Psychoeducation

Psychoeducation is the first stage of treatment and should be provided early in the process to everyone given a diagnosis, as well as to families, teachers, or significant others involved in care. Clinicians should explain what ADHD is, including its symptoms, causes, impacts, and available treatment approaches, while also addressing common misconceptions about the condition. With the increasing availability of ADHD-related information on social media, individuals have often encountered misinformation and assumptions about ADHD.[5] Clinicians should therefore help distinguish accurate information from misinformation and direct them towards evidence-based resources designed for lay-reading. Clinicians may also offer information about local ADHD support groups and community organisations where relevant. Overall, while psychoeducation is an important foundation for ADHD management, it is generally insufficient as a standalone intervention to reduce symptoms and improve functioning.

Pharmacological intervention

Medication is the only treatment approach for ADHD with a strong evidence base for reducing core symptoms, and all evidence-based guidelines support the use medication is as part of the treatment plan. When medication is included as part of an individual's treatment plan, the next step is

selecting which medication is most appropriate. While medication availability and access vastly differ across countries, many high-income western countries provide both stimulant (e.g., methylphenidate and amphetamines) and non-stimulant (e.g., atomoxetine, clonidine and guanfacine) options.[5] International clinical guidelines generally recommend stimulant medications as a first-line pharmacological options because of their established efficacy and relatively rapid onset of effectiveness.[5, 70] However, medication selection should always be individualised, with consideration given to a client's clinical presentation, co-occurring conditions, potential adverse effects, and preferences. For example, certain conditions require additional caution when considering stimulant medications such as bipolar disorder, psychosis, glaucoma, hypertension, substance use disorders, known or suspected cardiovascular risks, and family history of congenital arrhythmia.[4, 23] Evidence based guidelines and expert consensus supports the use of stimulant medications for ADHD in the presence of co-occurring anxiety, Tourette syndrome, sleep difficulties, mood problems or substance abuse issues, although caution should be applied in these situations and increased monitoring for adverse effects is advised. [5]

When using stimulant medication, a further consideration is the choice between immediate-release and extended-release formulations. Extended-release medications last longer throughout the day, are associated with stronger adherence, and typically require less frequent administering than immediate-release options. They are therefore often considered the preferred starting option for clinicians. Specific stimulant recommendations do however vary somewhat between clinical guidelines. For example, NICE recommends methylphenidate as the preferred first medication to try in children and adolescents, followed by lisdexamfetamine or other amphetamine-based medications. In adults, they propose either lisdexamfetamine or methylphenidate[23]. The Australian and Canadian guidelines provide greater flexibility, recommending methylphenidate, dexamphetamine, or lisdexamfetamine as equivalent first-line options across children, adolescents, and adults [4, 71].

When using medications to treat ADHD, treatment optimisation (defined as maximum response at minimum dose), rather than simply accepting clinical response, is an important, but often overlooked, consideration[72, 73]. Evidence from the MTA, supported by observational data highlights the importance of optimising core symptom outcomes using structured approaches to titration based on the principles of measurement-based care.[74, 75]

Table 1: Commonly Prescribed ADHD Medications[1]

Medication	Formulation	Usual recommended dose range	Dose schedule	Duration of effect	Main adverse effects
Stimulants					
Methylphenidate	Immediate Release (IR)	5 – 60mg	Up to three times a day	4 hrs	Appetite suppression and associated weight loss and growth retardation Insomnia Increased heart rate and blood pressure
	Long acting (LA)[2]	10 – 80mg	Once a day in morning (often with IR at 4pm)	8 hrs	
	Extended[2] release	18 – 72 mg	Once daily in morning	12 hrs	

Medication	Formulation	Usual recommended dose range	Dose schedule	Duration of effect	Main adverse effects
Dexamfetamine	Immediate Release (IR)	2.5 – 30mg	Up to three times a day	4-5 hrs	Appetite suppression and associated weight loss and growth retardation Insomnia Increased heart rate and blood pressure
Lisdexamfetamine	Prodrug	20 – 70mg	Once daily in morning	12 – 13 hrs	Appetite suppression and associated weight loss and growth retardation Insomnia Increased heart rate and blood pressure Palpitations
Non-Stimulants					
Medication	**Titration schedule**	**Usual recommended dose range**	**Dose schedule**	**Duration of effect**	**Main adverse effects**
Atomoxetine	Children and Adolescents up to 70 kg: 0.5 mg/kg for one week then 1.2 mg/kg (up to 80mg	1.2 – 1.6 mg/kg	Once daily either morning or evening or split doses am and pm	Not defined but has some effect across the day	Nausea (often reduces after first few weeks) Dry mouth and dyspepsia Increased heart rate and blood pressure Palpitations Reduced libido
	Children and adolescents over 70 kg and adults; Initial dose 40mg day (or lower) increased after one week to 80mg	80 – 100mg	Once daily either morning or evening or split doses am and pm		
Guanfacine extended release	1mg for one week, 2mg for one week then increase to 4mg	Children: 3 – 4 mg Adolescents: 3-7mg Not licensed for adults	Once daily, usually in evenings	24 hours	Somnolence and sedation Hypotension and bradycardia Elevated blood pressure and heart rate if stopped suddenly

Medication	Formulation	Usual recommended dose range	Dose schedule	Duration of effect	Main adverse effects

[1] Where are several other medications licensed for the treatment of ADHD, particularly in North America, we have not included them in this table as they are not widely licensed or available in other jurisdictions.

[2] Several different formulations of LA and XR methylphenidate are available. Many of these have look equivalent but have different pharmacokinetic and pharmacodynamic profiles. Clinicians are strongly advised to develop an understanding of the different profiles of medications available to them and take these into account both when initiating treatment and, perhaps most importantly, when switching between different formulations.

Non-pharmacological intervention

Non-pharmacological interventions are complementary to ADHD medications and can provide an alternative for individuals who do wish not take medication or are unable to access ADHD medications. Compared with pharmacological treatment, they are less effective at reducing core ADHD symptoms and are therefore often used to address the functional and broader difficulties associated with ADHD, including psychological wellbeing; self-esteem; behavioural, educational, and occupational functioning; family and peer relationships; and coping strategies.

A range of non-pharmacological interventions have been demonstrated to be helpful, with varying levels of evidence supporting their effectiveness. Behavioural interventions and cognitive-behavioural therapy (CBT) are among the most established psychosocial approaches with evidence supporting their efficacy in adolescents and adults. These typically aim to modify unhelpful behavioural and psychological patterns and address environmental factors that contribute to difficulties in everyday life. Depending on the intervention and individual's needs, they may involve developing organisational and time-management skills, cognitive restructuring, problem-solving, coping strategies, and reinforcement techniques. Interventions may also target behaviour such as defiance, impulsivity, and emotional dysregulation.

Parent and family training interventions are also commonly recommended in the management of ADHD in children and adolescents. These aim to equip parents with strategies to better support their child and manage their ADHD-related difficulties. Evidence suggests that they can improve child and family functioning, reduce symptom severity, and support parental mental health.[65, 76] Although trials are still underway there is an emerging evidence base to support parent training as an effective intervention for those waiting for an assessment.

Other non-pharmacological approaches include cognitive training, neurofeedback, exercise and lifestyle changes, ADHD coaching, and organisational or school-based interventions. Cognitive training typically involves structured, often computerised training programs that target cognitive processes such as attention and memory. Despite considerable effort there is little evidence for efficacy on core ADHD symptoms. There is however some support for effects on cognition [77, 78]. Neurofeedback uses operant conditioning principles to provide individuals with real-time feedback about aspects of their brain activity, with the aim of helping them learn to regulate these patterns. As with cognitive training there is yet little evidence for efficacy in reducing ADHD symptoms.[79] Lifestyle interventions may involve strategies to support sleep, physical activity, diet, and daily routines. Exercise has received some support for efficacy. A recent umbrella review identified multiple clinical trials and systematic reviews. [80] While the included meta-analysis reported large effect sizes (hedges g of 0.8 - 0.9) it is important to note than many trials were not blinded. Clinicians should however consider adding exercise as a component of a multimodal treatment approach to

ADHD. ADHD coaching typically focuses on practical strategies for organisation, time management, goal setting, and implementing behavioural changes while feedback is positive there are no clinical trials to demonstrate efficacy. Organisational and school-based interventions can similarly provide environmental structure and practical supports to reduce the impact of ADHD-related difficulties in educational or organisational settings.

Continuing care and ongoing support

ADHD is often a life-long condition, so ongoing care and support is an important and ongoing, aspect of the clinical journey. Once treatment has commenced, it is essential to recognise that circumstances, needs, and treatment goals change over time and that individuals require continued monitoring, outcome measurement, treatment adjustments based on these measures, and support. Once treatment has started, clinicians should regularly review the individual's response to treatment, including whether it is working as expected, whether they are experiencing any adverse effects, and whether changes are needed to the treatment plan. Where medication is prescribed, ongoing review should also consider tolerability, adherence, and whether dose titration, or other medication changes are required. The goal with medication is to maximise benefit at minimum dose with the least adverse effects.

There is currently no single standardised approach to the delivery of ADHD continuing care, and the frequency and nature of follow-up appointments tend to vary across clinicians and clinical settings. Measurement-based care provides one approach to supporting ongoing monitoring and is recommended by the Dundee ADHD Clinical Pathway.[75] Measurement-based care involves the systematic and routine assessment of treatment outcomes, including both benefits and adverse effects, with these measures used to inform treatment adjustments and optimised outcomes. The International Consortium for Health Outcomes Measurement (ICHOM) neurodevelopmental disorders outcome set does however give strong suggestions for both measures and timelines for assessment.[81]

Table 2: ICHOM recommended outcome measures for ADHD

Outcome	Recommended measure	Age	Burden	Assessment timeline[1]
Core symptoms				
ADHD (inattention, hyperactivity, impulsivity)	SNAP-IV, ADHD-RS-5, VADRS	5-17	5 minutes	Baseline; every 6 months thereafter
Impact, functioning, and QOL				
Caregiver burden	FSI	6-18	2 minutes	Baseline; every 6 months thereafter
Overall functioning	DD-CGAS		Clinician-rated measure	Baseline; every 6 months thereafter
Quality of life	KIDSCREEN-10	8-18	2-5 minutes	Baseline; every 6 months thereafter

Outcome	Recommended measure	Age	Burden	Assessment timeline[1]
Common co-occurring problems				
Depression and anxiety	RCADS-25	8-18	5-10 minutes	Baseline; every 6 months thereafter
Sleep problems	CSHQ	4-13	5-10 minutes	Baseline; every 6 months thereafter
Aggression or irritability	SNAP-IV	6-17	2-3 minutes	Baseline; every 6 months thereafter
Emotional lability or reactivity	ARI	6-58	2 minutes	Baseline; every 6 months thereafter

[1]It is recommended that for those taking ADHD medication, outcomes are additionally measured before medication initiation or change, and end of titration.

ADHD Rating Scale 5 (ADHD-RS-5); Vanderbilt ADHD Diagnostic Rating Scale (VADRS); Quality of Life (QOL); Family Strain Index (FSI); Developmental-Disability Children's Global Assessment Scale (DD-CGAS); Revised Child Anxiety and Depression Scales 25 (RCADS-25); Children's Sleep Habits Questionnaire (CSHQ); Swanson, Nolan, and Pelham Rating Scale IV (SNAP-IV); Affective Reactivity Index (AFI).

In practice, both the Dundee measurement-based care model and ICHOM recommend using standardised rating scales at each appointment to provide objective measures of treatment outcomes and to track changes over time.[75, 81] To enable reliable comparisons, it is important to collect baseline measures before treatment begins so that subsequent changes can be assessed accurately. Overall, regularly reviewing symptom and functional outcomes provides an opportunity for clinicians and clients to jointly evaluate what is and is not working, consider whether treatment goals are being achieved, and making informed decisions about ongoing care.

Chapter Summary

Worldwide, ADHD impacts between 5% to 8% of children and adolescents, and roughly 2.5% of adults. While ADHD typically starts early in life it often continues into adulthood and is now acknowledged as a life course diagnosis. ADHD is associated with significant lifetime burden and is associated with substantial costs both to the individual and society.

While there are still many unanswered questions about the causes of ADHD there is high heritability with strong support for genetic influences playing a primary role. This includes both common SNPs and rare variants. The extent to which these are combined with environmental risk factors and gene-environment interactions is still to be determined. While there is clear support for a range of structural and functional brain changes in ADHD there is clear heterogeneity and it is not yet possible to use brain imaging techniques to diagnose ADHD. Similarly, ADHD is heterogeneous at the neurocognitive level, and while cognitive assessments can help identify strengths and weaknesses, they are not determinative diagnostically.

While ADHD continues to be under recognised, under diagnosed and under treated in many parts of the world, recent increases in visibility, primarily through social media, and in access to assessment through commercial telehealth clinics, has meant there may also increasing cases of misdiagnosis and overtreatment.

Assessment for ADHD should be integrated within a comprehensive evaluation of an individual's strengths and difficulties. Clinical interview remains the core of the assessment process and should not be replaced by questionnaire or cognitive testing. Once a diagnosis is made treatment planning should be a collaborative process. Psychoeducation provides the platform for all other treatment. In most cases, and where possible, it is appropriate to offer medication treatment as this is the only approach with strong evidence for improving core ADHD symptoms. The approach to medication will depend on local circumstances. Non-pharmacological approaches including parent training, CBT, exercise, educational accommodations, and ADHD coaching should also be considered as these can provide important support and reduce the day to day and long-term impacts of ADHD symptoms. Whichever approaches are used it is important to aim for optimal outcomes both for symptoms and functioning. This requires high-quality ongoing clinical care.

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