

Attention-Deficit/Hyperactivity Disorder: A Review of Neurocognitive Mechanisms, Diagnosis, and Treatment

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Abstract. Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder that involves long-term issues with attention regulation, impulse control and activity regulation which may disrupt various core functions like learning, socializing and daily functioning. The epidemiological data of ADHD worldwide show that the prevalence of this disorder is very high in females throughout their lives. Over fifty percent of child patients carry their core symptoms into adulthood and most of them also have high comorbidity with anxiety and depression. This review explains the two most widely used diagnostic systems in clinical practice and outlines the differences in the age-specific diagnostic criteria. It also takes into account the shortcomings of using behavioural symptoms as the sole means of diagnosis. Existing evidence indicates that ADHD has been associated with a number of interacting neurocognitive processes including executive control deficits, abnormal reward processing, dysregulated dynamic attention and imbalanced large-scale brain networks. These mechanisms have been supported by behavioural studies and multimodal neuroimaging. ADHD is caused by the interplay between genetic predisposition and environmental factors. Polymorphisms in genes controlling dopamine and serotonin signaling could affect maturation of frontostriatal and frontoparietal neural pathways, thus making one susceptible to the disorder. A variety of behavioral and cognitive psychological interventions are currently used along with pharmacologic treatments, both stimulant and nonstimulant. Although these approaches can enhance symptoms and functionality in real life, they do not directly target the neurobiological abnormalities that cause the disorder. Response to treatment is also highly individualized. In an effort to offer combined theoretical references to maximize clinical diagnosis and intervention of ADHD, the paper summarizes the gaps in the current literature and suggests some promising future research directions such as early developmental risk intervention, personalized neuromodulation, and etiology-targeted precise therapy.

Keywords: ADHD, neurocognitive mechanism, diagnosis, gene-environment interaction, clinical intervention

1. Introduction

Many neurodevelopmental diseases are centered on attentional disruption mechanisms. One of the most noticeable characteristics of Attention-Deficit/Hyperactivity Disorder (ADHD) is difficulty

controlling attention. Persistent issues with attention, impulse control, and activity regulation are common in people with this illness. According to research, the cognitive and behavioral issues seen in people with ADHD are mostly caused by deficiencies in sustained attention, executive control, and attentional regulation. Weikard first characterized ADHD in 1775, and it was further defined in the early 20th century [1]. The processes of ADHD and the best methods for diagnosis and treatment are still being studied, even though it has been well documented historically.

The prevalence rate of ADHD in the children and adolescent population worldwide is 5.29%, and regional differences are not significant [2]. The prevalence of persistent adult ADHD was 2.58 and symptomatic adult ADHD was 6.76 [3]. In addition, 60% of the ADHD sample showed continuity of symptoms, with 41% meeting symptom and impairment criteria as adults [4]. Adults with ADHD who responded to a survey report much lower health-related quality of life, greater impairment in daily activities, and substantial productivity losses at work compared to population norms [5]. Also, the multiple linear regression analyses revealed that symptoms of depression and anxiety were significantly positively correlated with symptoms of ADHD, particularly among women [6]. This frequent co-occurrence also highlights the complexity of this barrier and the interaction between cognitive and social functions [7]. This comorbid characteristic also explains why a purely symptomatic diagnosis cannot fully capture the nature of ADHD. Although ADHD is defined behaviourally in symptom-based classification systems, these classifications describe behaviours and not processes. There are many clinical variations: people with ADHD differ in their cognitive profile, pattern of comorbidity, course of development and response to treatment. Symptom clusters provide little insight into the causes and persistence of these impairments. To overcome this limitation, more recent research has taken a mechanistic approach, focusing on the neurocognitive processes that contribute to behavioural dysregulation.

The diagnosis of ADHD is currently based on the criteria of either the Diagnostic and Statistical Manual of the American Psychiatric Association, 5th edition (DSM-5-TR) or the International Classification of Diseases, 11th Revision (ICD-11). Besides fulfilling the core symptom criteria, a complete evaluation involving self-report measures, structured clinical interviews, behavioral observations, and neuropsychological testing is typically needed to make the diagnosis. Nevertheless, these sophisticated measures do not clarify the differences in symptoms by age, gender, and comorbidity yet.

ADHD is a multifactorial neurodevelopmental disorder that has both genetic and environmental influences. Furthermore, it is essential to continue to explore its underlying causal and neurobiological mechanisms for prevention and intervention. Moreover, the long-term clinical use of various treatments offers insights into safe and effective individualised treatment strategies.

This review will offer a detailed picture of ADHD through the combination of neurobiological and clinical evidence. In particular, it examines existing diagnostic models and evaluation methods, discusses pathophysiological processes underlying ADHD, assesses available treatment options, and describes the state of clinical practice.

2. Diagnosis

Attention-Deficit/Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder and has been traditionally thought of as an executive dysfunction disorder. The medical field has diagnostic systems or frameworks. One is Diagnostic and Statistical Manual of Mental Disorders, 5th Edition, Text Revision (DSM-5-TR) which operationalises ADHD diagnosis through symptom counts and explicit thresholds to ensure maximum reliability, while ICD-11 emphasises

developmental appropriateness and functional impairment, with more flexibility for clinical judgement in practice [8, 9].

Both of these models view ADHD as a neurodevelopmental disorder with deficits in attention control, and some people also having problems with excessive activity levels or impulsive behaviours. These symptoms last at least six months of a person's life with ADHD and cause functional impairment (e.g., failure in school, work, safety problems, poor socialization). But the common thing is to have two or more diagnoses because people with ADHD usually have comorbidities with other psychiatric disorders.

There are some diagnostic differences between children and adults. The current DSM-5-TR and ICD-11 criteria require 6 symptoms in children but 5 in individuals aged 17 years or older. Symptoms must be persistent (i.e., present for at least six months), occur in more than one setting, cause clinically significant impairment, and have an onset prior to age 12 years [10]. For adults, evidence of childhood symptoms is still required. Importantly, the symptoms must be beyond developmental expectations and not better explained by other conditions.

Moreover, although not a formal part of the diagnostic criteria, emotional dysregulation is common and may be indicative of more general deficits in cognitive control processes [11]. This is in line with evidence that executive dysfunction, especially impairments in inhibitory control, is a core cognitive characteristic of ADHD [12]. Together, these findings support the notion that ADHD can be more coherently conceptualised within a cognitive control framework that includes both core behavioural symptoms and associated emotional difficulties.

3. Mechanism

Current models consider ADHD to be a disorder of interacting neurocognitive systems, not a unitary deficit. Recent models have gone beyond a simplistic view of ADHD as an attentional deficit from a cognitive perspective. Rather, the disorder has been increasingly described in terms of the interactions between executive regulation, motivational systems, and the dynamic allocation of attention. Understanding these mechanisms is important to elucidate how core symptoms originate and are maintained.

Advances in behavioural paradigms and neuroimaging technologies, such as MRI, diffusion tensor imaging (DTI), fMRI, electroencephalography (EEG), event-related potential (ERP), and positron emission tomography (PET), have led to the development of explanatory frameworks at different levels of analysis. Whereas the functional models of executive and motivational deficits in ADHD are derived from the behavioural paradigms, the neuroimaging studies have allowed mapping of such impairments to the neural circuits and large-scale brain networks, providing a more mechanistic account of attentional and behavioural dysfunction.

3.1. Executive control deficits: core impairments in top-down regulation

Of these domains, deficits in executive control are the most consistent cognitive deficits reported in ADHD [13]. The executive function deficits in ADHD as the basis of top-down cognitive control have been validated at multi-dimensional levels, beyond the previous limitation of isolated executive function deficits. People with ADHD tend to have impaired inhibitory control, working memory capacity, and cognitive flexibility [12, 14].

During response inhibition tasks such as the Stop-Signal Task, people with ADHD demonstrate much longer stop-signal reaction times (SSRT), a deficit that is age-independent and largely sample-characteristic independent [13], which reflects underregulation of prefrontal-striatal circuits [15].

Under greater load conditions, deficits are more pronounced. Adults with ADHD are less accurate and have slower reaction times in visual-spatial and phonological working memory tests [16]. In about 62% to 85% of children with ADHD, there are impairments in working memory, and classic paradigms like the Corsi Block Tapping Test and Wechsler Digit-Letter Sequencing always show poorer performance compared to healthy controls [17]. Individuals with ADHD also tend to have higher switch costs and perseverative errors in task switching paradigms [12]. To a certain extent, these impairments are discovered to persist after the working memory and inhibitory control demands are controlled, which suggests a possible cognitive flexibility deficit [18]. It is important to note that these deficits are not universal but selective: there are people who are impaired in one subcomponent and only a small percentage of them show no exceptional executive dysfunction, which is in line with the phenotypic heterogeneity of ADHD [19].

Neuroimaging data assist in understanding the neural underpinnings of executive dysfunction. Executive dysfunction in ADHD has been linked to hypoactivation of frontostriatal pathways, i. e. , the dorsolateral prefrontal cortex and anterior cingulate cortex when inhibiting control tasks [20]. Structural MRI reveals that there is a decrease in gray matter volume in the prefrontal cortex, striatum and cerebellum with the most consistent decreases being in the right globus pallidus, putamen, caudate and cerebellum [21]. Delayed cortical maturation is also a feature of these areas, and the dorsolateral prefrontal cortex matures about 3 years later than normal maturation [22].

Functional MRI shows hypoactivation of prefrontal-striatal and prefrontal-parietal networks during executive functioning, and reduced activation in the right inferior frontal gyrus in medication-free boys, which is associated with self-reported impulsivity [23]. Diffusion tensor imaging (DTI) studies show decreased fractional anisotropy (FA) in white matter tracts linking the prefrontal cortex and striatum, such as the anterior limb of the internal capsule, with greater reductions in FA correlated with longer SSRT [24]. This structure–function–behaviour model implies that deficits in executive control in ADHD are related to impaired development of prefrontal guided neural networks, rather than focal brain regions [25]. Dysfunction in these systems may compromise top-down control over behaviour, leading to impulsivity, distractibility and problems with goal-directed persistence.

3.2. Reward processing abnormalities: cognitive biases in motivation and decision-making

Another important dimension of cognitive mechanisms of ADHD is abnormal reward processing, which involves certain alterations in temporal discounting, encoding of expectations and sensitivity to rewards, but not simply a deficiency of motivation. Behavioural experiments have discovered that ADHD subjects show a strong preference towards immediate rewards- in delay discounting tasks they are more inclined to take small immediate rewards as opposed to large delayed ones. Luman [26] demonstrated that they are 2.3 times more sensitive to the delay of rewards than controls. ADHD patients have problems with adapting behavioural strategies to previous feedback in probabilistic reward tasks, imprecise encoding of reward expectations, and low decision-making efficiency [27]. Also, individuals with ADHD demonstrate biases in how they perceive the magnitude of rewards, overestimating the value of proximal rewards and underestimating long-term benefits of distal rewards. This bias is especially high in ADHD persons who have salient impulsive symptoms [28].

Neuroimaging studies offer a circuit-level account of abnormal reward processing. Functional MRI reveals that in the reward expectation phase, ADHD subjects have much lower activation of the ventral striatum (in particular, the nucleus accumbens). In a meta-analysis, Plichta and Scheres [29] indicated that the delay discounting rate is correlated with the extent of activation of this region. The

ventral striatum, particularly the nucleus accumbens, is critically dependent on reward anticipation and value coding in dopaminergic corticostriatal circuits. During the reward feedback phase, the functional connectivity abnormalities of prefrontal-ventral striatal pathways disrupt the integration of feedback information and behavioural adaptation. Furthermore, default mode network (DMN) and the reward network also negatively correlate less in ADHD individuals. This imbalance was shown to be indicative of impaired brain resource allocation during rest in a resting-state fMRI experiment by Tomasi and Volkow [30], which makes it difficult to switch between internal thought and external reward-oriented processing flexibly.

3.3. Attention regulation disorders: from "deficits in selective attention" to "dysfunctions in dynamic regulation"

In traditional models, the disorder in ADHD was reduced to "insufficient selective attention". Recent research has shown that the core problem is in dynamic regulation of attention. There are synchronized deficits in three subsystems: maintenance of vigilance, selective attention and attention shifting. Behavioral experiments have shown that in sustained attention tasks (e.g., Continuous Performance Test), the error rate of ADHD individuals increases significantly with time, indicating a decreased ability to sustain vigilance. Furthermore, a large-scale meta-analysis of 55 fMRI studies demonstrated a widespread pattern of hypoactivation in frontoparietal and attentional control networks in ADHD, consistent with deficits in sustained attention [31]. Individuals with ADHD have difficulties filtering interference from irrelevant stimuli in selective attention tasks unbalanced allocation of attention resources and insufficient depth of processing for target stimuli. In attention-shifting tasks, reaction times are slowed down with a switch from one target to another and more switching errors are committed, indicating regulatory deficits of the parietal-prefrontal attention network [25].

Neuroimaging has narrowed down the circuit mechanisms of disorders of attention control. Functional MRI experiments have demonstrated that individuals with ADHD do not activate the brainstem-thalamus-parietal vigilance network adequately when performing vigilance tasks, which leads to a diminished capacity to respond quickly to changes in the environment [32]. Selective attention tasks over-activate the ventral attention network (which is responsible in exogenous attention redirection) and under-activates the dorsal attention network (which is responsible in endogenous attention maintenance) creating an imbalance state of exogenous attention dominance and endogenous attention weakness [25]. Resting-state MRI studies have discovered abnormal functional connectivity in the attention network as well as between the attention network and other cognitive networks (including the executive control network). In particular, the negative correlation between the DMN and the attention network decreases, causing an imbalance in attention resources between task-related processing and task-unrelated processing [33]. The evidence provided by electroencephalography (EEG) and event-related potential (ERP) studies is complementary on the temporal dimension: ADHD subjects exhibit decreased amplitude and longer latency of the P300 component, which reflects deficits in the ability to allocate attention resources and stimulus evaluation processes; abnormalities in the N2 component reflect a deficit in conflict monitoring ability, which is related to interference inhibition during attention selection [34].

3.4. Network interaction model of ADHD

ADHD is a network dysregulation disorder, and this can be thought of as a balance between top-down control and reward processing. Recent evidence suggests that ADHD may not be defined by

impairments in specific executive control, reward or attention networks, but rather by dysfunction across interacting networks. Top-down modulation of subcortical reward circuits by prefrontal executive control networks and the suppression of the default mode network during effective task performance are examples of network interactions. Decreased prefrontal control, altered reward sensitivity and unstable large-scale network dynamics may all play a role in behavioural variability in ADHD. This network perspective can explain conflicting results and supports the view of ADHD as a disorder of dynamic brain control, rather than a primary deficit.

4. Gene and environment

There is increasing evidence that ADHD is caused by the interplay between genetic susceptibility and environmental factors rather than being caused by a single factor [35]. Twin and family studies clearly show that ADHD has a high heritability, with estimates as high as 74% [36]. Recent genome-wide association studies (GWAS) have also shown that ADHD is highly polygenic. The risk of ADHD seems to be determined not by a single gene but by the cumulative influence of multiple genetic variants, each of which makes a small contribution to the overall risk. International GWAS meta-analyses have revealed several genetic loci linked to ADHD, indicating that ADHD is caused by complex genetic mechanisms affecting brain development and function and identifies variations with genome-wide significance in 12 different locations, providing crucial new insights into the biology of ADHD [37]. These results confirm the hypothesis that ADHD is highly genetic and that many genetic variants contribute to the biological processes underlying ADHD. Molecular genetics have also identified several genetic variants associated with ADHD: Genetic variants such as dopamine D4 receptor (DRD4) and dopamine transporter (SLC6A3) are associated with impaired reward processing, impacting the efficiency of dopamine transmission in the striatum and prefrontal cortex, resulting in abnormal reward signal amplification and regulation [38]. Changes in these genes may affect dopamine signaling in corticosteroid circuits, especially in the prefrontal cortex and striatum, brain systems that play a key role in cognitive control and motivation [21, 36].

The genetic influences are big but environmental influences also play a significant role in determining the developmental outcomes. Poorer behavioral control and greater risk and persistence of ADHD have been linked to adverse family environments, socioeconomic disadvantage and chronic stress [39, 40]. Furthermore, genetic predispositions can modify the sensitivity of individuals to environmental influences, and environmental exposures can modify the expression of genetic risk. For example, individuals with dopaminergic genetic variants may be more sensitive to environmental stressors that target motivational systems or attentional control. Such dynamic interactions between biological susceptibility and environmental experiences can influence neural development and contribute to the emergence of ADHD symptoms throughout childhood and adolescence [41]. Moreover, the polymorphism of the serotonin transporter gene 5-HTTLPR has been found to moderate the effect of environmental stress on ADHD symptoms, especially on hyperactivity and impulsivity [42].

Developmentally, it is thought that genetic and environmental influences converge on neural circuits involved in cognitive control and reward processing. These influences affect the development of frontostriatal and frontoparietal networks that control attention, behavioral inhibition and goal-directed behavior [25]. Disruptions in the development of these neural systems may ultimately result in the cognitive impairments described in previous sections, including executive dysfunction, altered reward sensitivity and instability in attentional regulation [12]. Thus, ADHD can be conceptualized as the outcome of complex developmental pathways linking genetic susceptibility, environmental exposures and neural network dysregulation.

Knowledge of these gene-environment interactions gives more insight into the aetiology of ADHD and also highlights the need to take both biological and contextual factors into account in prevention and intervention efforts.

5. Treatment and future direction

The most common treatment of ADHD is a combination of medication and psychological and behavioural interventions that aim at alleviating the symptoms and enhancing day-to-day functioning [43, 44]. Stimulant drugs (e. g. methylphenidate, amphetamine-based drugs) are the most widely used and effective medications. It is believed that these drugs increase dopaminergic and noradrenergic transmission in frontostriatal pathways, which results in better executive control, attention regulation, and behavioral inhibition [20]. Other non-stimulant drugs that are commonly used include atomoxetine and guanfacine to those who do not respond well to stimulants or have side effects. Meta-analytic data suggest that both stimulant and non-stimulant treatments can be effective in alleviating ADHD symptoms, but there remains a significant variability in response among individuals [45].

Behavioral and psychosocial interventions are significant adjuncts to drug therapy. Strategies are behavioral therapy, parent training, school-based, and cognitive-behavioral therapy, to enhance self-control, organizational abilities, and adaptive functioning. Intensive, multimodal treatment regimens that target core symptoms and functional impairments especially in children and adolescents are particularly effective when applied over the long term [46]. These interventions also matter in enhancing long-term outcomes like academic performance, social functioning, and emotional well-being.

Moreover, psychological and counselling interventions are also critical in the management of ADHD, especially to address functional impairments that may not be addressed by pharmacological interventions. Cognitive-behavioral therapy (CBT) can enhance self-control, organisational abilities and attention control, particularly in adults with chronic symptoms [47]. Beyond cognitive restructuring, these approaches may also focus on emotional regulation, as people with ADHD often struggle with regulating emotions such as frustration and stress, which can contribute to impulsive and unstable attention. Skills training and coaching also offer practical strategies for time and activity management, goal setting, and planning, enabling individuals to apply their cognitive skills in practice [48]. Moreover, psychoeducation is essential for enhancing treatment adherence by educating patients and families about ADHD and its trajectory [49]. Overall, these strategies emphasise the need to combine psychological and behavioural interventions with medication to attain more holistic and long-term symptom and functional outcomes.

Although the existing treatments have proven to be effective, there are several limitations. The high heterogeneity of ADHD is one of its primary difficulties, as people vary in their manifestation of symptoms, cognitive profiles and treatment responsiveness [50]. A high percentage of patients with ADHD exhibit partial or incomplete response to pharmacological treatment and others exhibit adverse side effects [51-53]. Moreover, most of the existing therapies focus on alleviating the symptoms instead of the neurocognitive processes of the disorder. ADHD is a disorder of disruption across multiple interacting systems (e.g. executive control, reward processing, attention regulation), and existing interventions may not capture the complexity of the disorder. For example, the transcranial direct current electrical stimulation (tDCS) should be considered, and its combination with R-fMRI will provide a suitable therapy for ADHD individuals [54].

Current treatment strategies, while successful in decreasing core symptoms, are mainly used after diagnosis and concentrate on symptom control rather than the developmental and genetic risk factors

underlying the emergence of ADHD. Future research should thus take a more developmental perspective that aims at early risk factors [55]. In particular, increasing attention has been given to early intervention approaches that aim at modifying developmental trajectories by addressing environmental stress and gene–environment interactions [56]. Such interventions can potentially prevent or slow the progression of symptoms before impairments are completely developed, which highlights the importance of early detection and intervention in vulnerable groups.

6. Conclusion

Attention-Deficit/Hyperactivity Disorder (ADHD) is a complicated neurodevelopmental disorder, and it cannot be described by symptom diagnostic criteria only. The existing DSM-5-TR and ICD-11 are useful clinical diagnosis models, but they concentrate on describing the external behavior and provide little information about the neurobiological processes behind them.

This review has outlined recent research on the neurocognitive underpinnings of ADHD, including deficits in executive control, reward processing, attention and interactions between large-scale brain networks. These four inferences cover psychological studies, brain network, multimodal neuroimaging studies, and consistently point to these cognitive abnormalities being due to abnormal development of frontostriatal and frontoparietal circuits rather than a primary deficit in any one cognitive process. In addition, growing evidence suggests that ADHD is the result of the interplay between genetic vulnerability and environmental factors. Both of them emphasising the need to take a developmental approach to its aetiology.

Existing interventions today are mainly aimed at managing the symptoms and not altering the developmental and neurobiological processes. There are two approaches to treatment strategies that are predominantly pharmacological and psychological interventions. They have shown significant improvements in alleviating symptoms and enhancing daily activities. Nevertheless, there is a significant heterogeneity of symptom presentation, cognitive profiles, and response to treatment, which is a significant clinical challenge.

Future studies should therefore combine the recent developments in genetics, neuroimaging and cognitive neuroscience to help identify individuals at risk earlier and to inform more personalised interventions. Improved knowledge of gene-environment interactions and large-scale brain network dysfunction could help with precision diagnosis and treatment based on underlying mechanisms, leading to better long-term prognosis and quality of life for people with ADHD.

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