ChatGPT

Neuropsychology of ADHD: Brain Structures and Neural Pathways in Children and Adults

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental condition characterized by persistent symptoms of inattention, hyperactivity, and impulsivity. Modern neuropsychology and neuroimaging research have linked these behavioral symptoms to differences in specific brain structures and neural circuits. Key regions implicated include the **prefrontal cortex** (involved in executive functions), the **basal ganglia** (involved in motor control and reward processing), and the **cerebellum** (involved in coordination and cognitive timing). Additionally, network-level disruptions such as in the **default mode network (DMN)** – a brain network active during rest and mind-wandering – have been found. Notably, ADHD affects both children and adults, and while many neural features are shared across development, there are important **developmental differences** in brain maturation and network function between pediatric and adult ADHD [1] [2]. Below, we provide an up-to-date overview of how these brain structures and pathways contribute to ADHD symptoms, highlighting recent findings (especially from the past five years) and comparing their roles in children versus adults.

Prefrontal Cortex and Executive Function

The prefrontal cortex (PFC) is critical for **attention regulation, impulse control,** and other executive functions. In individuals with ADHD, the PFC often shows atypical structure and activity. **Structural MRI** studies indicate that children and adults with ADHD may have a slightly smaller or delayed-developing prefrontal cortex. For example, a large meta-analysis published in 2024 found reduced gray matter volume in multiple frontal lobe regions of ADHD patients, including the bilateral orbitofrontal cortex (OFC), the inferior frontal gyrus, the superior frontal gyrus, and the anterior cingulate cortex (ACC) [3]. These frontal regions support functions like sustained attention, working memory, and inhibition of inappropriate responses. Functionally, ADHD is associated with underactivation of prefrontal regions during tasks that demand self-control and focus [4] [3]. Consistent with these findings, delayed maturation of the PFC has been documented in ADHD youth: a longitudinal study showed that ADHD children reach peak cortical thickness in the PFC several years later than typically developing children (a median of ~10.5 years vs. 7.5 years in controls) [5]. This cortical delay, most prominent in lateral prefrontal areas, likely contributes to the lag in executive function development observed in ADHD [5]. In practical terms, a less developed or less active PFC can lead to difficulties in **sustaining attention** (leading to distractibility) and **inhibiting impulses** (leading to impulsive behavior), core features of ADHD. Notably, many of these prefrontal differences persist into adulthood, although adults with ADHD often develop compensatory strategies and PFC differences may be subtler than in children [1].

Basal Ganglia and Reward/Inhibition Circuits

The basal ganglia – a group of subcortical structures including the caudate nucleus, putamen, and globus pallidus – form crucial neural pathways with the cortex (often called **frontostriatal circuits**). These circuits modulate **motor activity, motivation/reward, and inhibitory control**. Research has consistently found that the basal ganglia are structurally and functionally altered in ADHD. Children with ADHD, in particular, tend to have slightly smaller basal ganglia volumes compared to peers [6]. A large-

scale analysis reported significant volume reductions in the *striatum* of ADHD patients, including the caudate and putamen, as well as related regions like the nucleus accumbens [6]. These differences are linked to the dopamine-driven reward pathways, which may explain why individuals with ADHD often exhibit altered reward sensitivity and **impulsive decision-making**. Indeed, the basal ganglia are densely populated with dopamine neurons, and ADHD's well-known dopamine imbalances can disrupt the normal function of these circuits [7]. Functional studies (including fMRI) have shown reduced activation in fronto-striatal loops during tasks requiring response inhibition and motor control in ADHD, reflecting a **dysfunction of the inhibition circuit** that connects the **inferior prefrontal cortex** with the basal ganglia [4]. This can manifest as hyperactive behavior and difficulty suppressing impulses. Moreover, the basal ganglia also connect to the limbic system; abnormalities in these regions may not only underlie impulsivity but also contribute to emotional dysregulation sometimes seen in ADHD [8]. Recent research continues to support basal ganglia involvement: for instance, a 2023 study highlighted the importance of the *pallidum* (part of the basal ganglia) in ADHD, finding that structural differences in the right globus pallidus correlated with symptom severity and were linked into broader dysfunctional networks (fronto-striatal-cerebellar circuits) across children, adolescents, and adults [9] [10]. In summary, **basal ganglia abnormalities** disrupt the brain's ability to regulate movement and impulses and to modulate reward, thereby contributing to the hyperactivity and impulsivity characteristic of ADHD.

Cerebellum's Role in ADHD

Historically regarded for its role in coordination and balance, the **cerebellum** is now recognized as a key player in cognition and attention as well. In ADHD, the cerebellum often shows structural and connectivity differences that parallel the disorder's symptoms. Neuroimaging studies have consistently found that individuals with ADHD have reduced cerebellar volume or specific cerebellar regions that are smaller than in controls [11]. Notably, the cerebellar vermis (the midline region) has been found to be significantly smaller in some ADHD samples, which has been associated with symptoms of inattention and hyperactivity in children [11]. The cerebellum is interconnected with frontal regions (forming **fronto-cerebellar circuits**), and it contributes to timing, prediction, and fine-tuning of both motor and cognitive processes. Thus, cerebellar differences in ADHD can result in deficits in **motor coordination** (many children with ADHD show clumsiness or poor motor skills) as well as difficulties with **timing and rhythm** that underlie pacing of attention and behavior [12] [13]. Recent findings reinforce the cerebellum's involvement: for example, an adult ADHD study in 2024 used advanced diffusion tractography and found microstructural alterations in the cerebellar peduncles (the major white-matter tracts connecting the cerebellum to the rest of the brain) [14] [15]. Specifically, adult ADHD patients showed reduced integrity of the middle cerebellar peduncle – a pathway linking the cerebellum with the cerebral cortex – which was significantly associated with hyperactivity symptoms [16] [17]. These results suggest that disrupted cerebellar connectivity contributes to ADHD pathophysiology even in adulthood [18]. Furthermore, stimulant medications like methylphenidate (a common ADHD treatment) are known to engage cerebellar circuitry (via cerebellar dopamine systems), highlighting that improving cerebellar function may be one mechanism by which symptoms are alleviated [11]. In summary, the **cerebellum supports not just motor control but also cognitive and emotional regulation**, and its atypical development or connectivity in ADHD likely underlies some of the disorder's motor clumsiness, poor timing, and even aspects of inattention.

Default Mode Network (DMN) and Mind-Wandering in ADHD

Beyond discrete brain structures, ADHD is importantly a disorder of brain **network dynamics**. The **default mode network (DMN)** is a set of interconnected brain regions (including the medial prefrontal cortex, posterior cingulate cortex/precuneus, and angular gyrus, among others) that becomes active

when the mind is at rest or wandering – essentially, our "idle mode." In tasks that require focus, the DMN typically deactivates in favor of task-positive networks (like the dorsal attention network and executive control networks). In ADHD, however, there is evidence of **DMN dysfunction**: the DMN may not properly deactivate, and it can intrude on tasks, leading to lapses of attention or daydreaming. For example, one recent neuroimaging study of adults with ADHD found abnormal functional connectivity patterns consistent with the "default mode interference" hypothesis [19] [20]. Adults with ADHD showed decreased internal connectivity within the DMN itself and within attention networks, coupled with *increased* connectivity *between* the DMN and attention-related regions [21]. In essence, the boundaries between the DMN (mind-wandering network) and task-focused networks are blurred in ADHD, causing interference. These findings support that an overactive or improperly timed DMN can derail attention – a phenomenon linked to excessive mind-wandering in ADHD [22] [23]. In children with ADHD, similar issues are observed: studies have reported that the DMN's activity is less suppressed during cognitive tasks, correlating with inattention. In fact, **resting-state fMRI** meta-analyses show ADHD-related hypoactivity in key DMN hubs like the posterior cingulate cortex (PCC) and ACC [24], alongside hyperactivity in regions like the OFC and parahippocampal gyrus which may reflect internally directed thought or emotional processing [25]. Moreover, both children and adults with ADHD demonstrate anomalies in how the DMN interacts with attention systems. A 2020 study identified that *shared* across child and adult ADHD are connectivity deficits **within the DMN and between the DMN and the brain's ventral attention network** (which is involved in reorienting attention) [2]. These shared DMN-attention network disruptions were positively correlated with the severity of ADHD symptoms [2]. This means that in both age groups, when the brain should be focusing, the DMN either remains too active or poorly coordinated with attention networks, contributing to symptoms of **inattention and distractibility**. Interestingly, there may also be developmental shifts: the same study noted that children with ADHD had unique connectivity problems between motor (somatomotor) networks and the dorsal attention network (likely relating to hyperactive behavior), whereas adults had more unique disruption between the DMN and limbic network (relating to mood and internalizing symptoms) [26]. In summary, **an inability to properly regulate the default mode network** – essentially turning off the "background chatter" of the brain when trying to concentrate – appears to be a core neuropsychological feature of ADHD, explaining the frequent *mind-wandering, difficulty sustaining attention,* and even some impulsive errors seen in patients [20].

Developmental Differences Between Children and Adults

ADHD is a lifespan condition, but the brain differences associated with ADHD can change from childhood to adulthood. **Developmental neuroimaging** reveals both continuity and change in ADHD's neural correlates. On one hand, many core abnormalities (in frontal-striatal-cerebellar circuits and DMN connectivity) are present in childhood ADHD and persist into adult ADHD [1] [2]. For instance, structural brain anomalies commonly seen in children (smaller prefrontal and striatal volumes) are also detected in adults with ADHD, though often with smaller effect sizes [27]. A machine-learning analysis of MRI data confirmed that the anatomical features distinguishing ADHD from controls in childhood significantly overlap with those in adults, supporting a fundamental continuity of ADHD's neurobiology across ages [1]. On the other hand, **maturational differences** are evident. As noted earlier, children with ADHD show a marked delay in cortical maturation, especially in the prefrontal cortex [5]. During adolescence and young adulthood, some of these structural differences become less pronounced as the brain continues to develop. For example, the volume differences in subcortical structures (caudate, putamen) tend to be more significant in children than in adults [28] [29]. This aligns with the observation that overt hyperactivity often declines with age – possibly as frontal circuits catch up in development and exert better top-down control over motor outputs. In contrast, symptoms of **inattention** and executive dysfunction may persist or even become more apparent in adulthood (when life demands increase), and these may relate to enduring functional connectivity differences (like those in the DMN or

frontal networks) that do not simply “normalize” with age [2]. Additionally, adults with ADHD often exhibit differences in neural systems related to emotional regulation and motivation, consistent with the higher rates of mood and anxiety comorbidities in adult ADHD [30]. The 2020 connectivity study, for example, found an adult-specific disruption in connectivity between the DMN and limbic network [31], which could underlie the greater emotional dysregulation or internal restlessness seen in adults. Another recent study focusing on adults reinforced that **ADHD-related network alterations remain clinically relevant in later life**: it showed persistent DMN-attention network interference in adults, tied to genetic risk factors and even influencing treatment response [20]. In summary, while children and adults with ADHD share a common core of brain circuit dysfunctions (notably in frontal, striatal, and default-mode networks), the **expression of these neural differences evolves with brain development**. Childhood ADHD is marked by developmental lags (especially in cortical maturation and subcortical growth) and prominent dysregulation of motor/attention circuits (aligning with hyperactivity), whereas adult ADHD features more subtle structural differences but ongoing functional network dysregulation (particularly involving attention and emotion circuits). Understanding these developmental neuropsychological differences is crucial, as it highlights that ADHD is not “outgrown” in a simple way – rather, the disorder’s neural profile shifts, and symptoms may morph from externalizing (hyperactive) in youth to more internal and cognitive (inattentive, executive dysfunction) in adulthood [32] [2].

Conclusion

In conclusion, the neuropsychology of ADHD involves a distributed network of brain abnormalities that together produce the symptoms of inattention, hyperactivity, and impulsivity. **Structural differences** in the prefrontal cortex, basal ganglia, and cerebellum – and the **disruption of neural pathways** connecting these regions – are central to the disorder. These differences undermine the brain’s ability to sustain attention, suppress inappropriate actions, regulate motor activity, and maintain cognitive control. In parallel, **functional network disturbances** such as aberrant default mode network activity interfere with the engagement of attention networks, leading to characteristic lapses in focus and executive function. Importantly, decades of research, now bolstered by recent findings from advanced imaging and large meta-analyses, indicate that these neurobiological features are present in both children and adults with ADHD. While the young ADHD brain is marked by delays in maturation and pronounced hyperactive circuit dysfunction, the adult ADHD brain continues to show many of the same circuit-level anomalies (albeit often moderated or compensated) and retains the core attentional network issues that can impair daily functioning [1] [2]. This evolving understanding of ADHD’s neural underpinnings – spanning cortical development, frontostriatal and cerebellar loops, and default-mode interference – not only explains why the disorder presents the way it does, but also guides ongoing efforts in treatment (for example, targeting dopamine networks with medication or training patients to modulate their default mode activity via neurofeedback). Overall, ADHD emerges from a convergence of brain structure and network dysfunctions, making it a prime example of how intricate brain pathways govern behavior across the lifespan [7] [3].

References (Key Sources)

- Faraone, S.V. *et al.* (2024). *Nature Reviews Disease Primers*, 10(11): Comprehensive review of ADHD including neurobiology.
- Chen, C. *et al.* (2025). Multimodal meta-analysis of ADHD neuroimaging (structural & functional): highlights OFC, ACC, basal ganglia differences [24] [3].
- Agoalikum, E. *et al.* (2023). *Frontiers in Hum. Neurosci.*: Structural differences in children vs adults with ADHD; emphasizes pallidum and fronto-striatal-cerebellar circuit [9] [10].

- Cao, Q. *et al.* (2020). *Translational Psychiatry*, 10: Shared and distinct functional connectivity patterns in child vs adult ADHD, especially involving DMN and attention networks [2].
- Parkkinen, S. *et al.* (2024). *J. Psychiatry Neurosci.*, 49(4): Cerebellar peduncle alterations in adult ADHD, linking cerebellar network to hyperactivity [18].
- Shaw, P. *et al.* (2007). *PNAS*, 104(49): Landmark study showing 3-year cortical maturation delay (especially in prefrontal cortex) in ADHD youth [5].
- Additional references from Frontiers in Neuroscience (2025) reviewing cerebellar dysfunction in ADHD [11], and other peer-reviewed sources as cited above.

---

[1] [27] [29] **Evidence for similar structural brain anomalies in youth and adult attention-deficit/hyperactivity disorder: a machine learning analysis | Translational Psychiatry**
https://www.nature.com/articles/s41398-021-01201-4

[2] [26] [30] [31] [32] **Shared and distinct resting functional connectivity in children and adults with attention-deficit/hyperactivity disorder | Translational Psychiatry**
https://www.nature.com/articles/s41398-020-0740-y

[3] [24] [25] **A multimodal neuroimaging meta-analysis of functional and structural brain abnormalities in attention-deficit/hyperactivity disorder - PubMed**
https://pubmed.ncbi.nlm.nih.gov/39615871/

[4] [6] [7] [8] [28] **Frontiers | Structural differences among children, adolescents, and adults with attention-deficit/hyperactivity disorder and abnormal Granger causality of the right pallidum and whole-brain**
https://doi.org/10.3389/fnhum.2023.1076873

[5] **Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation - PubMed**
https://pubmed.ncbi.nlm.nih.gov/18024590/

[9] [10] **Structural differences among children, adolescents, and adults with attention-deficit/hyperactivity disorder and abnormal Granger causality of the right pallidum and whole-brain - New Jersey Institute of Technology**
https://researchwith.njit.edu/en/publications/structural-differences-among-children-adolescents-and-adults-with

[11] [12] [13] **Frontiers | Can attention-deficit/hyperactivity disorder be considered a form of cerebellar dysfunction?**
https://www.frontiersin.org/journals/neuroscience/articles/10.3389/fnins.2025.1453025/full

[14] [15] [16] [17] [18] **Cerebellar network alterations in adult attention-deficit/hyperactivity disorder - PubMed**
https://pubmed.ncbi.nlm.nih.gov/38960626/

[19] [20] [21] **Interference of default mode on attention networks in adults with attention-deficit/hyperactivity disorder and its association with genetic variants and treatment outcomes - PubMed**
https://pubmed.ncbi.nlm.nih.gov/39145420/

[22] **Mind wandering perspective on attention-deficit/hyperactivity disorder**
https://pmc.ncbi.nlm.nih.gov/articles/PMC6525148/

[23] **How the Default Mode Network Contributes to Inattentive ADHD ...**
https://www.petrahoggarth.co.nz/post/how-the-default-mode-network-contributes-to-inattentive-adhd-symptoms