

NEUROBIOLOGY OF ADHD IN CHILDREN AND ADOLESCENTS: A LITERATURE BASED REVIEW

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ABSTRACT

Attention Deficit Hyperactivity Disorder (ADHD) is a chronic neurobehavioral disorder whose clinical presentation alters across development. This review examines the direct relationship between underlying neurobiological mechanisms and age-related differences in ADHD manifestations between children and adolescents. Structurally, a narrative review with a systematic approach was conducted primarily using PubMed (supplemented by Google Scholar) for literature published between 2016 and 2026. Following a rigorous screening of 182 initial records, a qualitative thematic synthesis was applied to 30 peer-reviewed articles (28 for primary analysis and 2 for supplementary context) alongside 2 manually included academic textbooks. Findings reveal that delayed brain maturation and dysfunction of dopaminergic and noradrenergic neurotransmitters, combined with reduced neural flexibility between the Default Mode Network (DMN) and Executive Control Network (ECN), underpin these age-specific variations. These mechanisms directly drive the clinical shift from overt hyperactivity and impulsivity in childhood to predominant inattention, executive dysfunction, and emotional dysregulation in adolescence. In conclusion, understanding the developmental neurobiology of ADHD is clinically paramount for improving diagnostic accuracy and designing individualized management strategies. Clinicians must move beyond static treatments and adopt age-developmentally appropriate multimodal approaches that integrate targeted pharmacology and tailored psychosocial support to enhance patients' overall quality of life.

Keywords: ADHD, Children, Adolescents, Neurobiology, Brain Maturation

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INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common chronic neurobehavioral disorders in children and adolescents. It is characterized by three main symptoms: inattention (lack of attention), hyperactivity (restlessness), and impulsivity (acting without thinking) (Kliegman *et al.*, 2020). Because these symptoms often resemble developmentally appropriate behavior, it is crucial to assess whether they are excessive for the child's age. The diagnosis of ADHD is primarily clinical and based on a comprehensive history obtained from parents, teachers, and self-reports from the adolescent. According to the DSM-5-TR criteria, symptoms must appear before age 12, occur in at least two settings, cause significant impairment in functioning, and not be attributable to another medical or psychiatric disorder. A physical examination is necessary to rule out other conditions, while additional tests, such as thyroid function tests or brain imaging, are only performed if indicated by the clinical examination (Kliegman *et al.*, 2020).

The urgency of addressing ADHD lies in its profound impact on patients' daily lives; if left unmanaged, it significantly impairs academic performance, social interactions, emotional functioning, and overall quality of life (Jangmo *et al.*, 2019; De Rossi *et al.*, 2023; Katz *et al.*, 2025). Globally, the prevalence of ADHD in children and adolescents is estimated at around 8% (Ayano *et al.*, 2023). However, figures in Indonesia show quite striking variations. One 2021 study in Surakarta even reported a prevalence of up to 26.4% (Prasaja, Harumi and Fatmawati, 2022). This difference likely reflects the large number of cases that remain under-detected, as well as variations in assessment methods used. This suggests that ADHD is not only a clinical issue, but also relates to how it is recognized and diagnosed.

To understand the complexity of ADHD, it is crucial to examine the disorder through a developmental neurobiological lens. ADHD in children and adolescents must be discussed separately because distinct differences in brain development, prefrontal cortex maturation, and psychosocial needs contribute to vastly different clinical manifestations in these two age groups. Neurobiologically, ADHD is driven by an interaction between genetic factors and dysfunction in neurotransmitter systems primarily dopamine and norepinephrine alongside delayed brain maturation and alterations in neural networks (Aster *et al.*, 2024). In childhood, these neurobiological deficits typically manifest as overt hyperactivity. However, as children enter adolescence, the clinical picture changes. ADHD symptoms in adolescents are often underrecognized because hyperactivity tends to decrease and gradually shift into more subtle manifestations of inattention and emotional dysregulation (Rubia, 2018; De Rossi *et al.*, 2023). Unfortunately, this subtle shift is often mistaken for a lack of motivation, laziness, or common behavioral problems, causing many adolescents to go undiagnosed or receive appropriate treatment late.

Consequently, treatment approaches that focus predominantly on general pediatric populations or solely on medication are often inadequate for adolescents, whose developmental and psychosocial demands require tailored care. Despite these recognized age-related challenges, a significant research gap remains: there are still few studies that comprehensively discuss the relationship between neurobiological changes, age-related shifts in clinical manifestations, and their implications for comprehensive ADHD management. Therefore, this paper aims to provide a critical analysis of the neurobiology of ADHD in children and adolescents to support clinical decision-making. To achieve this,

the article is structured as follows: first, the underlying neurobiology of ADHD is discussed; second, differences in clinical manifestations between children and adolescents are analyzed; and finally, the implications for diagnostic accuracy and comprehensive management strategies are evaluated.

RESEARCH METHODS

This study is a narrative review with a systematic approach. A literature search was conducted in PubMed (March–April 2026) using the Boolean search string: ("ADHD" OR "Attention-Deficit/Hyperactivity Disorder") AND ("neurobiology") AND ("children" OR "adolescents"). PubMed was selected as the primary database due to its broad coverage of biomedical and neuroscience literature. Eligibility criteria included peer-reviewed original articles, review articles, and clinically relevant translational neuroscience studies published in English or Indonesian between 2016 and 2026 discussing structural, functional, and developmental brain networks in ADHD, alongside relevant animal studies. Non-peer-reviewed articles, inaccessible full texts, and studies lacking translational clinical relevance were excluded. The initial search yielded 182 records. After title and abstract screening removed 120 irrelevant or duplicate records, 62 full-text articles were assessed for eligibility. Of these, 34 articles were excluded for failing to meet the predefined criteria, leaving 28 articles for primary analysis. To support the discussion and provide foundational concepts, two textbooks were manually included. Furthermore, two supplementary articles (including one identified through Google Scholar) were used exclusively for introductory context. Finally, the selected articles were analyzed using a qualitative thematic synthesis approach, categorizing findings into major neurobiological domains to address the study objectives.

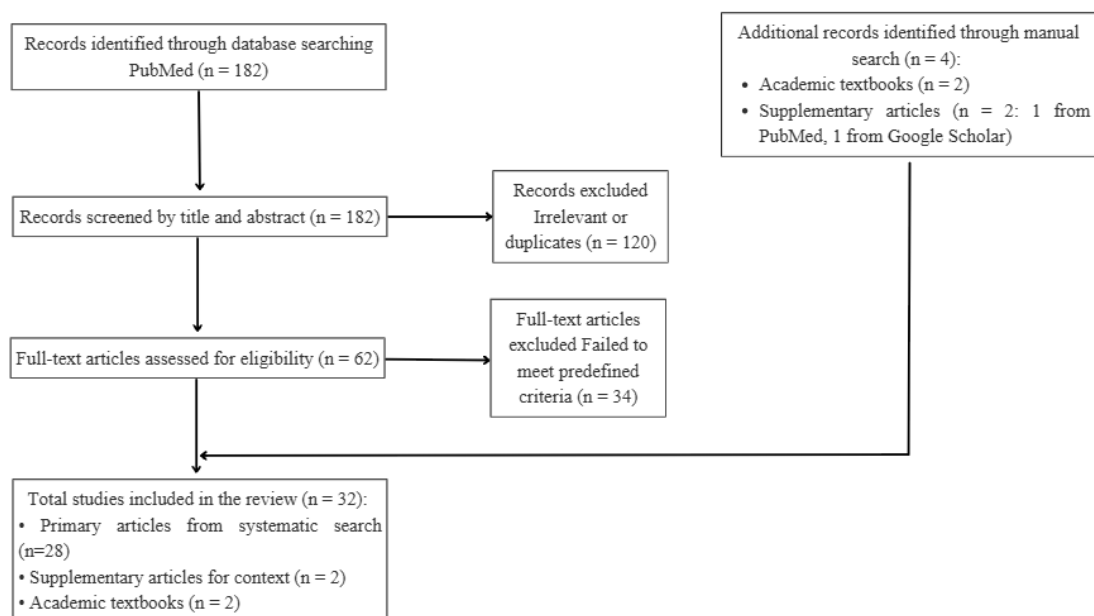


Figure 1. Flow diagram of the literature search and selection process.

RESULTS AND DISCUSSION

A systematic summary of the primary studies included in this review is provided in Table 1. This table highlights the underlying neurobiological mechanisms and how they translate into age-related clinical trajectories in children and adolescents with ADHD.

Table 1. Summary of Reviewed Studies

Author	Study Focus	Population	Key Findings
Neurotransmitter Dysfunction			
Aster et al. (2024)	Neurocomputational mechanisms of reward-based learning and flexibility.	N= 34 young adults (17 with ADHD, 17 controls).	Patients exhibited hyper-flexibility and blunted sensitivity to reinforcement, associated with diminished choice signals in the parietal cortex and ventral striatum.
Harris et al. (2022)	Neuropharmacological mechanisms of psychostimulants.	Animal model: DAT-KO and NET-KO mice.	The paradoxical calming effect of psychostimulants is driven specifically by dopamine elevation in the prefrontal cortex.
Volnova et al. (2023)	Effects of receptor agonist α 2A on spatial working memory and PPI.	Animal model: DAT-KO and WT rats.	Guanfacine (receptor agonist α 2A) significantly improved working memory in DAT-KO rats, highlighting the crucial role of the dopamine-norepinephrine balance
Stahl, S. M. (2013)	Neuroscientific basis of CSTC loops and catecholamine modulation.	General clinical framework.	Deficits in dopamine and norepinephrine disrupt cortico-striatal-thalamo-cortical loops, where prefrontal-striatum dysregulation leads to core ADHD symptoms.
Changes in Brain Structure			
Agoalikum et al. (2023)	Structural differences and Granger causality of the right pallidum.	N = 35 children, 40 adolescents, 39 adults.	Structural differences in various brain areas, particularly the right pallidum that influence disease severity, confirm that the frontal-striatal-cerebellar circuit plays an important role in the mechanism of ADHD.
Li, Chen, and Ide (2022)	Correlation of gray matter volume deficits with ADHD traits.	N = 11,502 emerging adolescents (aged 9–10 years) including monozygotic and dizygotic twins/siblings	Higher ADHD scores correlated with reduced gray matter volume in the basal ganglia, orbitofrontal, and occipital cortex, linked to diminished working memory.
Cundari et al. (2023)	Review of Cerebellar structural/functional differences in neuropsychiatric disorders.	Clinical populations with ADHD, ASD, and SCA3.	Cerebellar subregion abnormalities disrupt circuits, impairing task performance and neurocognitive symptom regulation.
Brain Network Abnormalities			
Blomberg et al. (2022)	Resting-state functional connectivity between default mode network (DMN), ventral attention (VA)/salience network (SN), and auditory networks.	N = 34 adults (17 ADHD, 17 controls).	Patients exhibited weaker anticorrelation between VA/SN and DMN, linking auditory distraction to dysfunctional network interactions.
Hong & Hwang (2022)	Resting-state DMN activation and signal variability.	N = 245 youth aged 7-17 years (105 ADHD, 140 controls).	Youth with ADHD showed increased variability in neural signaling and decreased resting-state connectivity within the DMN.
Hilger et al. (2020)	ADHD-related Event-Related Potential markers (P3 component).	N = 31 typically-developing school children (aged 9–11 years).	Higher ADHD symptoms correlated with smaller P3 amplitudes and lower temporal variability, highlighting ERPs as neurophysiological indicators.

Author	Study Focus	Population	Key Findings
Luo et al. (2023)	Dynamic functional connectivity (dFC) patterns.	N = 122 children (63 diagnosed with ADHD and 59 controls).	Inattention correlated with increased dFC within the DMN and decreased dFC between the DMN and sensorimotor network.
Michelini et al. (2021)	Etiological pathways of cognitive-neurophysiological impairments.	N = 356 adolescents and young adults (aged 11–27 years) from ADHD and control sibling pairs	Identified three separable pathways affecting IQ/reaction time, working memory, and inhibition/error processing.
Yin et al. (2022)	Evaluation of neural flexibility via multilayer community detection.	N = 360 children in total: 180 with ADHD and 180 typically developing controls (TDC).	ADHD children had significantly decreased neural flexibility, which was normalized to control levels following medication.
Rubia (2018)	Cognitive neuroscience of ADHD via fMRI meta-analysis.	Systematic review	Identified an abnormal interrelationship between poorly deactivated DMNs and hypo-engaged task-positive networks.
Sutclubasi et al. (2020)	Meta-analysis of resting-state functional connectivity across core networks.	Meta-analysis of 20 studies involving 944 patients with ADHD and 1,121 healthy controls	Pediatric ADHD is a default mode network dysconnectivity disorder characterized by reduced connectivity within the core DMN and with other networks.
Developmental Changes			
Wang et al. (2024)	Trajectories of ADHD symptoms (GMV and PRS).	N = 487 children and adolescents aged 6–15 years	Identified three ADHD symptom trajectories (ascending-high, stable-low, descending-medium) neurally based on variations in gray matter volume in the brain's attention centers, where the ascending-high symptom group had the highest polygenic genetic risk score (PRS).
Kurth et al. (2022)	Brain maturation (BrainAGE index) using machine learning.	N = 144 children (mean age ~11.86 years): 89 with ADHD, 34 asymptomatic siblings, and 21 healthy controls.	Brains of ADHD children were estimated to be significantly younger (-0.85 years); greater severity correlated with younger-appearing brains.
Asherson & Agnew-Blais (2019)	Trajectories of late-onset ADHD.	Longitudinal population and control samples (children to young adults).	Late-onset ADHD exists (onset typically ages 12-16); impairing ADHD can emerge beyond age 12 despite subthreshold childhood symptoms.
Ha et al. (2020)	Topological characteristics of maturational delay.	Animal Model (SHR vs Wistar Kyoto rats).	Confirmed a neurodevelopmental maturational delay (lag in strengthening limbic-cortical connections) resulting in abnormal pubertal connectivity.
Apter (2018)	Debate on childhood vs adult onset ADHD.	General ADHD population (Editorial review of longitudinal cohorts).	Adult structural brain anomalies reflect childhood ADHD diagnosis more accurately than current adult symptoms.
Posner, Polanczyk and Sonuga-Barke (2020)	Review of diagnosis and structural maturation.	Comprehensive literature review.	Emerging findings challenge current conceptions of ADHD causes, suggesting a need to re-evaluate models of maturation into adolescence.
Clinical Implications and Management			
De Rossi et al. (2023)	Age-related clinical characteristics and functional impairment.	N = 750 children (<11 years) and adolescents (≥11 years)	Adolescents had lower core symptom severity but significantly worse global functioning, adaptive skills, and emotional dysregulation than children.

Author	Study Focus	Population	Key Findings
Jangmo et al. (2019)	School performance and pharmacological treatment.	N = 657,720 Swedish students.	ADHD linked to lower academic performance; however, just 3 months of medication significantly improved school grades.
Katz et al. (2025)	Impact on teen health behaviors and well-being.	N = 1172 adolescents aged 12 to 17 years in the United States	Teens with ADHD faced more bullying, irregular sleep, and excessive screen time, but surprisingly less lack of peer support.
Song et al. (2021)	Family resilience and behavioral/mental health.	N = 4,169 U.S. adolescents with ADHD.	High family resilience serves as a protective factor, mitigating depression and conduct issues even amidst adverse childhood experiences.
Stern et al. (2020)	Longitudinal associations between ADHD and emotional problems.	N = 2,232 British twins tracked from childhood to adulthood.	ADHD symptoms consistently predicted later emotional problems (anxiety/depression), explained heavily by shared genetic factors.
Wolraich et al. (2019)	Pediatric guidelines and systemic barriers.	Pediatricians, children, and adolescents.	Emphasized treating comorbidities and highlighted that systemic barriers hamper guideline adoption.
Janzen, Chang and Farrelly (2024)	A review of the neurobiological and behavioral characteristics, and unique challenges in managing the patient's transition to adulthood.	Adolescents and Young Adults (AYAs) with ADHD	The transition to adulthood in patients with ADHD is complicated by significant cognitive, emotional, and neurobiological changes. This review highlights the need for tailored treatment strategies and innovative solutions to guide clinicians unfamiliar with managing and treating this demographic.
Kliegman et al. (2020)	Pediatric clinical guidelines and diagnostic criteria.	General pediatric and adolescent population.	Outlined DSM-5 criteria, emphasizing that symptoms must appear before age 12 and cause significant functional impairment.
Ogundele and Ayyash (2023)	Non-pharmacological management strategies.	Children and adolescents (Review).	School-based interventions improve social/academic skills, while neurocognitive training improve executive functions.

Neurotransmitter Functions and Pathophysiology

The pathophysiology of Attention-Deficit/Hyperactivity Disorder (ADHD) is neurobiologically rooted in an imbalance and suboptimal interaction between the dopaminergic and noradrenergic systems, which subsequently disrupts the cortico-striatal-thalamo-cortical (CSTC) circuit (Stahl, 2013). Dopamine plays a central role in reward processing and the regulation of decision-making. Dysfunction within these pathways, such as a reduced sensitivity to reinforcement, systematically lowers the signal-to-noise ratio in the parietal cortex and ventral striatum, making it inherently difficult for patients to filter out irrelevant external stimuli (Aster *et al.*, 2024). Within the prefrontal cortex (PFC), maintaining optimal dopamine levels serves as a key mechanism for reducing hyperactivity and impulsivity, highlighting its dominant role in behavioral control (Harris *et al.*, 2022). Concurrently, norepinephrine plays a crucial role in enhancing attention, alertness, and response stability by activating $\alpha 2A$ receptors in the PFC. This activation strengthens synaptic connectivity, thereby bolstering working memory and inhibitory control (Volnova *et al.*, 2023)

Structural Alterations and Brain Network Abnormalities

Structural abnormalities in ADHD do not occur in isolation; rather, they involve widespread dysfunction across complex neural networks. Higher ADHD clinical scores significantly correlate with reduced gray matter volume (GMV) in the basal ganglia, as well as the orbitofrontal and occipital cortices. These structural deficits directly underlie the diminished working memory capacity frequently observed in affected individuals (Li, Chen and Ide, 2022). Furthermore, these disruptions extend deeply into the cerebellum. Structural abnormalities in specific cerebellar subregions disrupt critical circuits, impairing both task performance and broader neurocognitive regulation (Cundari *et al.*, 2023).

Importantly, these structural alterations persist across the lifespan and strongly influence clinical outcomes. Significant structural variations within the pallidum and thalamus are evident across children, adolescents, and adults with ADHD. Specifically, alterations in the right pallidum positively correlate with symptom severity. This reinforces the premise that the frontal-striatal-cerebellar circuit, along with its connectivity, is central to the neurobiological profile of the disorder (Agoalikum *et al.*, 2023)

From a macro-scale brain network perspective, cognitive impairments in ADHD are fundamentally driven by an imbalance and reduced flexibility between two key systems: the Default Mode Network (DMN, the brain's resting state) and the Executive Control Network (ECN, the task-positive state) (Sutubasi *et al.*, 2020; Hong and Hwang, 2022). These cognitive deficits stem from distinct etiological pathways that independently affect working memory, inhibition, and reaction time (Michellini *et al.*, 2021). Individuals with ADHD exhibit decreased neural flexibility, which severely hinders the brain's ability to adaptively shift from a DMN-dominant resting state to an ECN-dominant task state. This shifting deficit directly contributes to the core symptoms of inattention and poor inhibitory control (Yin *et al.*, 2022; Luo *et al.*, 2023). Neurophysiologically, higher ADHD symptoms also correlate with smaller P3 amplitudes and lower temporal variability, further reflecting these cognitive processing challenges (Hilger *et al.*, 2020)

Neurodevelopmental Trajectories and Clinical Implications

From a developmental perspective, brain maturation in individuals with ADHD is characterized by profound structural delays. Evidence from machine learning algorithms suggests that the overall brain age of children with ADHD lags behind their chronological age by an average of 0.85 years (Kurth *et al.*, 2022), alongside specific impairments in the strengthening of limbic-cortical connections during pubertal transitions (Ha *et al.*, 2020). These complex insights continually challenge older, static conceptions of ADHD etiology, highlighting the critical need to re-evaluate our models of brain maturation from childhood into adolescence (Posner, Polanczyk and Sonuga-barke, 2020). Crucially, this neurodevelopmental delay provides a direct biological basis for the shifting clinical manifestations observed during adolescence. For instance, the failure to properly synchronize neural networks and shut down the DMN during task performance manifests behaviorally as severe concentration problems and uncontrollable daydreaming (Rubia, 2018). Interestingly, alternative research suggests that this dysconnectivity might not represent a pure deficit, but rather a form of heightened sensitivity to external environmental stimuli; this is evidenced by auditory distractions linked to weaker anticorrelations between the DMN and the Ventral Attention/Salience Network (VA/SN) (Blomberg *et al.*, 2022). Nevertheless, pharmacological interventions have proven highly

effective in normalizing the flexibility of these neural networks, altering connectivity patterns to closely resemble those of typically developing peers (Yin *et al.*, 2022).

This complex neurodevelopmental trajectory alters the phenotypic shape of symptoms over time. The phenomenon of "late-onset" ADHD demonstrates that impairing symptoms can emerge and escalate specifically between the ages of 12 and 16, despite individuals exhibiting only subthreshold symptoms during childhood (Asherson and Agnew-blais, 2019). During this developmental window, symptom progression is non-uniform; longitudinal tracking reveals distinct trajectories—such as ascending-high, stable-low, or descending-medium—that are neurally rooted in gray matter volume variations within core attention centers (including the ACC/mPFC, IPL, and MFG/PCG), heavily driven by polygenic genetic risk scores (PRS) (Wang *et al.*, 2024). Ultimately, these early structural abnormalities leave a permanent imprint, with adult brain structure more closely reflecting the childhood ADHD diagnosis itself rather than the severity of current adult symptoms (Apter, 2018).

As these structural and network shifts unfold across developmental stages, striking real-world clinical differences emerge. While core childhood symptoms consistently predict later emotional problems (such as anxiety and depression) due to shared genetic vulnerabilities (Stern *et al.*, 2020), the overt presentation transforms significantly. Standard pediatric guidelines and diagnostic criteria require that symptoms manifest before age 12 and cause significant functional impairment (Kliegman *et al.*, 2020). However, as the brain's structural development lags behind chronological age, the resulting deficits become highly pronounced when environmental and social demands amplify during the teenage years. Consequently, adolescents often exhibit lower core symptom severity (such as overt hyperactivity) than children, yet they experience significantly poorer global functioning, compromised adaptive skills, and heightened emotional dysregulation (De Rossi *et al.*, 2023). Furthermore, adolescents face an increased risk of bullying, irregular sleep, and excessive screen time, although they are surprisingly not found to lack peer support (Katz *et al.*, 2025). Academically, while ADHD is deeply associated with lower school performance, targeted pharmacological treatment is highly effective, with research showing that just three months of consistent medication can significantly improve school grades (Jangmo *et al.*, 2019).

Systemic Management and Future Intervention

Given this intricate interplay between neurobiological delays and clinical complexity, managing ADHD demands a comprehensive, systemic approach. The transition to adulthood is uniquely complex due to the massive confluence of cognitive, emotional, and neurobiological changes, highlighting an urgent need for tailored treatment strategies and innovative clinical solutions (Janzen, Chang and Farrelly, 2024). Effective management necessitates a multimodal variety of interventions. For example, school-based interventions are highly proven to improve social and academic skills, while neurocognitive training and physical exercise can markedly enhance executive functions, directly compensating for the underlying maturational and network delays (Ogundele and Ayyash, 2023). Furthermore, clinical guidelines strongly emphasize the importance of identifying and treating psychiatric comorbidities, although persistent systemic barriers often hinder the widespread implementation of these guidelines (Wolraich *et al.*, 2019). Within this broader psychosocial framework, high family resilience serves as a crucial,

ultimate protective factor, proven to successfully mitigate depression and conduct problems in adolescents even amidst adverse or traumatic childhood experiences (Song *et al.*, 2021)

CONCLUSIONS

This review establishes that the shifting clinical manifestations of Attention Deficit Hyperactivity Disorder from childhood into adolescence are fundamentally driven by underlying neurobiological delays and network inflexibilities. Specifically, dysregulation of the dopamine and norepinephrine systems, combined with delayed structural maturation and reduced neural flexibility between the Default Mode Network (DMN) and the Executive Control Network (ECN), constitutes the primary biological basis for symptom evolution. Consequently, while childhood ADHD is predominantly characterized by overt hyperactivity, the widening gap between chronological age and delayed brain maturation manifests during adolescence as severe inattention, executive dysfunction, and emotional dysregulation.

These findings further highlight the importance of age- and developmentally appropriate diagnostic and therapeutic approaches. Developmentally tailored multimodal interventions, including pharmacological treatment, neurocognitive training, and family-based support, may improve treatment precision, reduce functional impairment, and enhance quality of life among affected adolescents. Although this review bridges neurobiological changes with clinical progression, the existing literature remains fragmented. Future research should prioritize long-term evaluations of developmentally tailored combined therapies and explore digital technologies that support executive functioning, particularly through validating these strategies within the Indonesian population.

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