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# Stress and ADHD: Neurotransmitters in Intervention

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## Abstract

Attention Deficit/Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder often associated with increased stress, emotional dysfunction, and executive function impairments. In recent years, increasing attention has been given to the neurobiological mechanisms underlying the relationship between stress and ADHD, particularly the role of neurotransmitter systems. The aim of this study is to examine the relationship between stress and ADHD, with particular emphasis on the contribution of neurotransmitters to symptom expression and intervention strategies. For the purposes of this research, a literature review was conducted based on studies published between 2000 and 2025. The findings indicate that the dysfunction of dopamine, norepinephrine, serotonin, GABA, and glutamate are closely associated with the regulation of attention, emotional regulation, arousal, and reactivity to stress in individuals with ADHD. Furthermore, both pharmacological and non- pharmacological approaches targeting mechanisms related to neurotransmitters appear to contribute to symptom management and stress reduction. In conclusion, the literature supports the view that neurotransmitter systems play an important role in understanding and supporting individuals with ADHD in educational and clinical settings.

**Keywords:** ADHD; Stress; Neurotransmitters; Dopamine; Norepinephrine; Emotional Regulation; Intervention

## 1. Introduction

Attention Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders and is characterized by persistent patterns of inattention, hyperactivity, and impulsivity that affect individuals' daily functioning and development. Beyond these symptoms, ADHD has, in recent years, been associated with deficits in executive function, emotional regulation, and adaptive coping. These difficulties often make children, adolescents, and adults with ADHD more vulnerable to stress and less able to effectively manage demanding situations.

In recent years, the relationship between stress and ADHD has attracted more scientific interest. Individuals with ADHD often experience elevated levels of stress, which can exacerbate their difficulties with attention, emotional instability, and behavioral dysfunction. Anxiety is therefore not merely a secondary difficulty for individuals with ADHD, but a significant factor that can influence both the severity of symptoms and the course of the disorder over time.

At the same time, research has increasingly focused on the neurobiological basis of ADHD, particularly on the role of neurotransmitter systems. Dopamine and norepinephrine were considered by earlier studies to be key neurotransmitters for the disorder, but more recent findings also highlight the contribution of serotonin, GABA, and glutamate to attention, emotional regulation, arousal, and stress response. These systems appear to play a significant role, not only in the core symptoms of ADHD, but also in how individuals respond to internal and external stressors.

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The aim of this study is to examine the relationship between stress and ADHD, with particular emphasis on the role of neurotransmitters in the expression and modulation of symptoms. Understanding these neurobiological mechanisms may contribute to a more comprehensive approach to ADHD, which would combine psychological, educational, and neurobiological perspectives.

We now need to emphasize how useful and productive digital technologies are in the realm of education. To strengthen the field of ADHD and STRESS education, we must encourage the use of digital technology in all areas of education. Mobile devices (35), various ICTs (36-38), AI & STEM ROBOTICS (39-44), and other technologies and paradigms that enable and enhance educational processes like evaluation, intervention, and instruction might all be embraced and used in ADHD education. Additionally, educational practices and outcomes are accelerated and improved when ICTs are used in conjunction with theories and models of metacognition, mindfulness, and emotional intelligence cultivation [45-48]. This is especially true for ADHD and STRESS challenges.

## 2. Theoretical Foundations of Stress, ADHD and Midndfulness

### 2.1. Stress and Neurobiological Vulnerability in ADHD

Stress is a normal adaptive response to environmental demands or perceived threats. However, when stress becomes chronic or excessive, especially during development, it can disrupt cognitive, emotional, and neurobiological functioning (Lupien et al., 2009). In the case of ADHD, this issue takes on particular significance, as the disorder is already associated with deficits in executive control, emotional regulation, and adaptive functioning. For this reason, individuals with ADHD appear to be more vulnerable not only to the psychological effects of stress but also to its neurobiological consequences.

Studies have shown that children with ADHD often experience anxiety more frequently and more intensely than their typically developing peers (Seymour et al., 2012). This increased vulnerability is partly related to the difficulties they face in regulating attention, inhibitory control, and self-monitoring, which make it harder to manage daily academic, social, and family demands. As a result, even ordinary situations may be experienced as disproportionately stressful, increasing both emotional burden and physiological reactivity.

This sensitivity to stress has also been linked to altered biological stress response mechanisms. Studies using cortisol measurements have indicated atypical functioning of the hypothalamic-pituitary-adrenal (HPA) axis in children with ADHD, suggesting a dysfunctional stress response system rather than a typical adaptive response (Isaksson et al., 2012). Some individuals appear to be overreactive, while others exhibit blunted or reduced stress responses; in both cases, however, the pattern is maladaptive and can interfere with emotional stability, sleep, concentration, and behavioral regulation.

From a neurobiological perspective, chronic exposure to stress can further exacerbate vulnerabilities already present in ADHD. Prolonged activation of stress-related systems has been associated with functional and structural changes in brain regions such as the prefrontal cortex and hippocampus, which are critical for attention, working memory, and self-regulation (Liston et al., 2009). Given that these functions are already impaired in ADHD, the cumulative effect of stress can exacerbate executive dysfunction and reduce an individual's ability to effectively cope with internal and external demands.

For this reason, stress in ADHD should not be viewed solely as an emotional or environmental challenge, but also as a neurobiological risk factor that can influence the course and severity of the disorder. As Zografou and Drigas (2022) note, stress is closely linked to self-regulation difficulties in ADHD and can significantly exacerbate both cognitive and behavioral symptoms. This interaction underscores the importance of examining the biological pathways through which anxiety affects individuals with ADHD, particularly the neurotransmitter systems that regulate attention, arousal, emotions, and adaptive response.

### 2.2. Neurotransmitter Systems in ADHD

Given the above, it is clear that ADHD is no longer merely a behavioral or cognitive disorder, but also a neurobiological disorder involving complex alterations in the structure, function, and neurochemistry of the brain. Among the most critical components of this biological basis are neurotransmitters—the chemical messengers that regulate communication between neurons and influence various cognitive, emotional, and behavioral processes. In ADHD, the dysfunction of specific neurotransmitter systems has been linked to the core symptoms of the disorder: inattention, hyperactivity, and impulsivity (Faraone & Radonjić, 2024).

*2.2.1. Dopamine (DA)*

Dopamine is perhaps the most extensively studied neurotransmitter associated with ADHD. It is a substance that plays a very important role in motivation, stimulus processing, attention, and executive functions—that is, in the areas where individuals with ADHD typically struggle (Volkow et al., 2009). Functional brain imaging studies have revealed reduced availability of the dopamine transporter (DAT) in the striatum and reduced density of dopamine receptors in the prefrontal cortex and basal ganglia in children and adults with ADHD. These brain regions are particularly important for inhibitory control and working memory (Del Campo et al., 2011).

Furthermore, genetic studies have identified polymorphisms in genes related to dopamine function, such as DRD4, DAT1, and COMT, which are more common in individuals with ADHD (Franke et al., 2012). These findings suggest that altered dopaminergic signaling contributes significantly to the neurodevelopmental etiology of ADHD, particularly in relation to a lack of response to praise, delayed gratification, and reduced persistence in task performance.

Pharmacological treatments, such as methylphenidate and amphetamine-based stimulants, increase dopamine availability in the synaptic cleft, primarily by blocking dopamine reuptake, thereby improving attention and reducing hyperactivity (Arnsten, 2006). This reinforces dopamine's central role in the pathophysiology of ADHD.

*2.2.2. Norepinephrine (NE)*

Closely linked to dopamine is norepinephrine (NE), another catecholamine neurotransmitter, which is associated with the regulation of arousal, alertness, attention shifting, and response inhibition. This substance is particularly important for the function of the locus coeruleus–prefrontal cortex circuit, which is essential for regulating attention. Dysfunctions in norepinephrine transmission have been associated with reduced cognitive control and increased distractibility in children with ADHD (Berridge & Waterhouse, 2003).

Atomoxetine is a non-stimulant medication for ADHD that selectively inhibits the norepinephrine transporter (NET), increasing extracellular NE levels, particularly in the dorsolateral prefrontal cortex, and is effective in treating ADHD symptoms in both children and adults. The efficacy of these medications supports the hypothesis that noradrenergic dysfunction is an integral part of ADHD (Ravishankar et al., 2016).

*2.2.3. Serotonin (5-HT)*

Although it is not as central a neurotransmitter as the previous two, serotonin (5-HT) plays a regulatory role in ADHD, particularly in relation to mood regulation, impulse control, and emotional processing. Abnormal serotonin levels have been associated with the co-occurrence of anxiety, aggression, and mood disorders, which are frequently observed in children with ADHD (Oades, 2008).

Serotonin likely interacts with dopamine systems as well, influencing the reward circuitry and contributing to the emotional instability observed in many individuals with ADHD. Some studies suggest that low serotonergic activity, particularly in the prefrontal cortex, may be the cause of poor decision-making and increased impulsivity (Rubia, 2018).

*2.2.4. Gamma-Aminobutyric Acid (GABA)*

GABA is the brain's primary inhibitory neurotransmitter and is responsible for reducing neuronal excitability and promoting calmness and concentration. Evidence suggests that dysfunction of this neurotransmitter may negatively affect ADHD symptoms, particularly in the hyperactive/impulsive subtype (Endres et al., 2015). Specifically, GABA plays a significant role in regulating executive functions and emotional balance (Sideraki & Drigas, 2024). Magnetic spectroscopy studies have shown lower GABA concentrations in the anterior cingulate cortex and motor areas in children with ADHD, suggesting reduced inhibitory tone in brain regions associated with impulse control and motor regulation (Edden et al., 2012).

Although current treatments for ADHD do not directly target the GABA system, new research on GABA agonists and modulators highlights potential therapeutic directions for reducing hyperactivity and increasing inhibitory control (Biederman et al., 2019).

*2.2.5. Glutamic acid (GLU)*

The glutamatergic system is the brain's primary excitatory system and has been linked to the pathophysiology of ADHD. An imbalance between excitatory (glutamate) and inhibitory (GABA) signals may contribute to cortical hyperexcitability and the difficulty in sustaining attention that characterizes the disorder under study (Dickstein et al., 2021). In some

children with ADHD, elevated levels of glutamate have been observed in the striatum and anterior cingulate cortex, which likely reflects excessive neural activity in the circuits governing attention and motor behavior (Naaijen et al., 2017).

These findings support the view that ADHD involves not only deficits but also dysfunction in multiple neurotransmitter systems, and that therapeutic strategies could benefit from broader neurochemical targets in the future.

*2.2.6. Neuropsychological Correlates*

Neurochemical abnormalities in ADHD are reflected in distinct neuropsychological deficits, such as impaired working memory, delayed processing speed, reduced inhibitory control, and poor error monitoring (Willcutt et al., 2005). These cognitive functions are closely linked to the frontostriatal network, where dopamine and norepinephrine play key regulatory roles. Children with ADHD often show reduced activation in the posterior lateral prefrontal cortex during tasks requiring sustained attention and careful planning (Bush et al., 2005).

On an emotional level, children with ADHD exhibit increased sensitivity to negative stimuli and difficulty regulating frustration, a phenomenon that may also stem from neurotransmitter imbalances in the limbic-prefrontal pathways (Shaw et al., 2016). These biological vulnerabilities interact with environmental stressors and the psychosocial context, contributing to the heterogeneity and persistence of the disorder's symptoms.

*2.2.7. Neurochemical Integration*

Although early models of ADHD focused narrowly on dopamine dysfunction, current research supports a multisystemic model that includes dopaminergic, noradrenergic, serotonergic, GABAergic, and glutamatergic contributions. These systems interact in complex ways to regulate attention, emotions, behavior, and stress response, thus making ADHD a disorder of neurotransmitter network dysfunction rather than an isolated deficiency. A comprehensive understanding of these neurochemical foundations is not only useful for pharmacological treatment but also for non-pharmacological interventions, such as mindfulness training, exercise, and diet, which have been shown to influence neurotransmitter levels and support neural plasticity (Tang et al., 2015).

### 2.3. Neurotransmitters and Stress Regulation

As mentioned earlier, among all neurotransmitters, dopamine (DA) has received the most attention in research on ADHD. Dopamine plays a critical role in motivation, reward processing, and executive function, which are typically impaired in individuals with ADHD. Neuroimaging studies have shown that children with ADHD exhibit reduced availability of dopamine transporters and hypereactivity in dopaminergic circuits, particularly in the prefrontal cortex and striatal regions (Volkow et al., 2019). This dopaminergic hypofunction is thought to contribute to inattention, difficulty sustaining effort, and reduced sensitivity to praise.

Closely related is the role of norepinephrine (NE), another catecholamine involved in arousal, alertness, and response inhibition. NE pathways project from the locus coeruleus to the prefrontal cortex, where they contribute to the regulation of cognitive flexibility and working memory. Disorders in this system are associated with poor impulse control and inattention, which are common symptoms in individuals with ADHD (Arnsten & Rubia, 2012). Because both dopamine and norepinephrine are involved, treatments often aim to simultaneously enhance the synaptic availability of these neurotransmitters.

Although dopamine and norepinephrine dominate research on ADHD, it has also been demonstrated that serotonin (5-HT) plays a prominent role, particularly in the emotional and behavioral dimensions of the disorder. Specifically, serotonin contributes to mood regulation, social behavior, and the control of aggression. Low serotonin activity has been associated with impulsivity, emotional instability, and oppositional behaviors in children with ADHD (Oades, 2008) .

Some researchers argue that serotonergic dysfunction may underlie the high comorbidity between ADHD and mood or anxiety disorders, highlighting the need for holistic therapeutic approaches (Michelini et al., 2016). While serotonergic agents are not typically used as first-line treatments for ADHD, they may be prescribed for complex clinical presentations that include depression or generalized anxiety.

Beyond monoamines, research in recent years highlights the importance of glutamic acid and GABA in the pathophysiology of ADHD. The balance between these systems is essential for optimal cognitive and behavioral functioning (Sideraki, Moraiti, Gavriilidou & Drigas, 2022).

Magnetic resonance spectroscopy studies have shown that children with ADHD often exhibit elevated levels of glutamate in the anterior cingulate cortex and reduced concentrations of GABA in regions associated with attention and impulse control impulse control. These imbalances may lead to neuronal hyperexcitability, resulting in inattention, impulsivity, and poor cognitive filtering (Endres et al., 2015; Edden et al., 2012).

Although there are currently no medications that directly target glutamate or GABA for ADHD, this area appears to have significant potential for future development. Preliminary studies have investigated the modulation of GABAergic function through neurofeedback, mindfulness meditation, and dietary supplements, with early evidence suggesting positive effects on attention and emotional balance (Naaijen et al., 2017).

### 2.4. Neurotransmitter-related interventions

The most widely used pharmacological treatments for ADHD are stimulants, such as methylphenidate and amphetamine-based medications. These drugs primarily work by inhibiting the reuptake of dopamine and norepinephrine, increasing their availability in the synaptic clefts. Numerous studies have demonstrated that stimulants improve attention, inhibition, academic performance, and even emotional regulation in children with ADHD (Faraone et al., 2021).

In recent years, non-stimulant medications have also gained ground in pharmacological interventions for ADHD. Atomoxetine, a selective norepinephrine reuptake inhibitor (NRI), works by increasing norepinephrine levels in the prefrontal cortex without directly affecting dopamine levels. It is particularly useful for children with comorbid anxiety disorders, for whom stimulants may exacerbate symptoms (Khoodoruth et al., 2022). Similarly, guanfacine and clonidine, both α-2 adrenergic agonists, act on the noradrenergic system to reduce hyperactivity and improve behavioral regulation.

Of course, the use of these medications also has certain limitations. Side effects such as appetite suppression, sleep disturbances, or emotional blunting are common, and long-term effects remain a cause for concern. Furthermore, although these medications appear to improve symptoms in the short term, they do not “cure” ADHD, nor do they directly address social or emotional skills, making it necessary to include behavioral or educational interventions to achieve long-term outcomes (Faraone et al., 2021; Khoodoruth et al., 2022).

However, growing interest in non-pharmacological interventions for ADHD has led to the exploration of various approaches that influence neurotransmitter systems without medication. These include:

- Neurofeedback, which uses real-time EEG data to train individuals to self-regulate brainwave activity. Neurofeedback protocols have been associated with increased dopamine release and improved executive function (Arns et al., 2014).
- Physical activity, particularly aerobic exercise, has been shown to increase dopamine and norepinephrine levels, improve mood, and enhance executive control in children with ADHD (Vysniauske et al., 2020) .
- Mindfulness-based interventions, as discussed in the previous chapter, have been linked to increased GABA availability and reduced amygdala reactivity, supporting an individual’s emotional self-regulation (Drigas & Mitsea, 2020; Tang et al., 2015).
- Dietary interventions, such as omega-3 fatty acid supplementation, have also been proposed to support neurotransmitter synthesis and neural membrane stability, although the findings are mixed (Fotoglou et al., 2022; Richardson, 2006).

These approaches can serve as a supplement to medication or as alternatives for children who cannot tolerate pharmaceutical products, offering a more holistic and sustainable method for managing symptoms.

Neurotransmitters play a central role in the development and expression of ADHD symptoms, influencing attention, emotion, motivation, and behavior. Interventions targeting these systems, whether pharmacological or behavioral, have the potential to modulate brain function and improve outcomes for children with ADHD. While stimulant medications remain the most effective and widely used treatments in these cases, non-pharmacological strategies that support neurotransmitter balance are increasingly supported by contemporary research and offer complementary and novel avenues for intervention. Future advances in neuroimaging and personalized medicine may allow for even more precise targeting of neurotransmitter systems, paving the way for personalized, brain-based care for ADHD.

## 3. Conclusions

This review examined the association between stress and ADHD from a neurobiological perspective, emphasizing the role played by neurotransmitter systems. The current literature suggests that ADHD is not limited to behavioral symptoms but also includes impairments in attention, emotional regulation, arousal, and stress response. Within this context, stress appears to exacerbate existing vulnerabilities and further impact cognitive, emotional, and behavioral functioning in individuals with ADHD.

The findings make it clear that dopamine and norepinephrine remain central to the neurobiology of ADHD, especially in relation to executive function, attention control, and behavioral regulation. At the same time, serotonin appears to contribute to the regulation of mood and emotions, while GABA and glutamate are involved in the balance between inhibition and excitation that supports cognitive stability. These neurotransmitter systems therefore influence not only the core symptoms of ADHD but also the individual's response to stress.

Another important point to note is that stress in ADHD should be understood not only as a psychosocial burden but also as a biological challenge. Altered cortisol responses and atypical functioning of the HPA axis suggest that increased stress may further exacerbate already disrupted self-regulatory mechanisms. This perspective supports a more comprehensive understanding of ADHD, in which neurobiological and environmental factors are considered to interact rather than exist in isolation.

Furthermore, this review also highlights the importance of interventions targeting neurotransmitter-related mechanisms. Pharmacological treatments remain the most established approach, particularly through the modulation of dopaminergic and noradrenergic systems. However, non-pharmacological strategies, such as neurofeedback, physical activity, mindfulness, and nutritional support, are increasingly recognized as useful complementary approaches that can support emotional and cognitive regulation.

However, the literature review also revealed certain limitations. Differences in methodology across studies, in the characteristics of each study's sample, and in the design of the interventions make direct comparison between them difficult. Furthermore, although the evidence regarding dopamine and norepinephrine is strong, the role of serotonin, GABA, and glutamate requires further investigation.

In conclusion, the literature supports the view that neurotransmitter systems play an important role in both the symptomatology of ADHD and stress regulation. A better understanding of these mechanisms may contribute to the design of more personalized and comprehensive interventions. Future research should continue to explore the interaction between neurochemical, psychological, and environmental factors in ADHD, with a particular emphasis on long-term outcomes and practical applications in clinical and educational settings.

## Compliance with ethical standards

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The Authors proclaim no conflict of interest.

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