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Review Paper

Attention-Deficit Hyperactivity Disorder: A Comprehensive Review

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ABSTRACT

Attention-Deficit/Hyperactivity Disorder (ADHD) is not merely a collection of symptoms but a deeply intricate neurodevelopmental condition that challenges conventional boundaries of cognition, behavior, and emotion. With its roots reaching back centuries, ADHD has evolved from moral judgments of "defective control" to a deeper understanding shaped by genetics, neurobiology, and environmental interplay. At its core, ADHD reflects disruptions in the brain's dopamine and norepinephrine systems, leading to altered reward processing, impaired attention, and diminished executive function. Despite these advancements, ADHD remains a diagnostic puzzle. Cultural biases, gender differences, and the variability of symptoms across contexts complicate assessments. Recent breakthroughs in neuroimaging and genetic research reveal the need for holistic interventions. Beyond symptoms, ADHD is a story of resilience, adaptability, and innovation in treatment approaches. This review reimagines ADHD not as a deficit, but as a lens through which to explore human diversity in cognition and behavior. By weaving historical, biological, and therapeutic narratives, this synthesis calls for a future where ADHD management transcends reductionist approaches and embraces individualized care.

Keywords: *ADHD, Neurodevelopment, Neurodivergence, Psychobiology*

The term ADHD has increasingly found its way into the vocabulary of today's milieu. The accuracy of this term's usage is debatable, especially with its popularity on social media which has a propensity to exaggerate and mislabel symptoms. Attention-deficit/hyperactivity disorder (ADHD) is a complex, multifaceted neurodevelopmental condition marked by persistent symptoms of inattention, hyperactivity, and impulsivity. Inattention and disorganization refer to difficulty focussing on a task, not listening, and forgetting materials at inappropriate levels based on age or development. Hyperactivity-impulsivity is characterized by excessive activity, fidgeting, difficulty sitting, interfering with others' activities, and unwillingness to wait (American Psychiatric Association, 2013). Aligning with the significant prevalence of such behaviors in formative years, the onset of ADHD is in childhood therefore there is a requirement for the symptoms

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to be present before the age of 12 years. Due to faulty memory of ADHD symptoms in childhood and later presenting inattentive signs, the age of onset for ADHD symptoms was raised from 7 to 12 years in the Diagnostic Statistical Manual of Mental Disorders (DSM-5). This expansion enables greater diagnosis in additional children with substantial impairments (Vande et al., 2014).

In India, around 7.1% of children and adolescents meet the criteria for ADHD (Joseph & Devu, 2019). Among adults, 5.48 to 25.7% of the population screened positive for ADHD (Mishra et al., 2024). For accurate diagnosis of this condition, the inattentive and/or hyperactive symptoms must manifest in two or more settings (home, work, school, etc.) and not be explained by any other psychotic, mood, anxiety, personality, substance, or other disorders. It is essential to consult ancillary sources to confirm symptoms across contexts. Given the variance in the presentation of the symptoms in different circumstances, the signs may be diminished when the individual is in a new setting, is receiving reinforcements for appropriate behavior, is engaged by some external source, or is communicating in an individual setting (American Psychiatric Association, 2013). However, there remain differences in diagnosis based on culture and gender. ADHD is observed more in males than in females, the ratio being almost 2:1 in childhood and 1.6:1 in adults (American Psychiatric Association, 2013). Females often go undiagnosed as they present more inattention symptoms than males who tend to display more hyperactive symptoms. Therefore, boys showing disruptive behavior in class are more likely to be referred to a clinician than girls for ADHD testing (Slobodin & Davidovitch, 2019). As for culture, diagnosis may differ due to multiple reasons. A genuine propensity towards ADHD, differences in parenting styles, and varying cultural tolerance for the symptoms of hyperactivity are some (Gomez et al., 2015). In the US, clinical identification rates are lower for African American and Latino people compared to Caucasian groups (American Psychiatric Association, 2013). Further, the presence of informants gives higher positive results for ADHD than rating scales, signifying the reason behind higher prevalence in childhood where informants are likelier, as compared to adulthood (Gomez et al., 2015).

History

While the proper terminology and diagnosis may have entered the medical dictionary recently, this condition spans the ages. One of the earliest mentions of such a disorder in medical literature could be traced back to Scottish physician Sir Alexander Crichton in 1798. His interest in mental illnesses gave rise to three books, one with a special chapter on attention, highlighting a condition similar to present-day ADHD (Palmer & Finger, 2001). In scientific history, however, credit for the earliest mention of ADHD popularly goes to British pediatrician Sir George Frederic Still. In his Goulstonian Lectures, which he delivered before the Royal College of Physicians of London in 1902, Still gave an account of "defect of moral control as a morbid manifestation, without general impairment of intellect and without physical disease" (Still 1902, p. 1079). His work formed a significant foundation upon which the future of ADHD research was built.

Post-encephalitic behavior observed during the encephalitic epidemic in the 1920s showed distracted and hyperactive symptoms similar to Still's prior account (Ebaugh, 1923; Hohmann, 1922). Although not all those affected would qualify for a diagnosis of ADHD, these findings gave momentum to exploring the connection between brain damage and hyperactivity. Further, it medicalized the concept of abnormal behavior in children and many of the symptoms have found their way into the present-day description of ADHD (Rafalovich, 2001). In 1932, German physicians Franz Kramer and Hans Pollnow were the

first to introduce hyperkinetic disease. Features included significant motor activity, easy distraction by novel stimuli, impulsivity, and an inability to focus on difficult tasks, which align with those in the DSM-5. Kramer and Pollnow also delineated the impairments in social, academic, or occupational functioning, which forms one of the present criteria of ADHD.

In 1937, Charles Bradley's pioneering finding implicated benzedrine as a way to curb the symptoms of ADHD. It is paradoxical that a stimulant like benzedrine can produce such effects but much of the central nervous system has an inhibitory function, which on stimulation can generate subdued behavior (Bradley, 1937). It also highlighted a physiological basis for a psychological condition. This paved the way for the connection between brain damage and hyperactivity. However, there were arguments against this causal relationship as children who had not experienced any trauma to the brain also showed hyperactive symptoms (Laufer et al., 1957). This resulted in a semantic change from minimal brain damage to minimal brain dysfunction (DSM-I) by the Oxford International Study Group of Child Neurology in the 1970s, catering to functional rather than structural problems in the brain (Ross & Ross, 1976). The focus was on neurological factors instead of environmental factors. Yet, this too came under scrutiny when it was realized that not all brain dysfunctions gave rise to hyperactivity and that minimal brain dysfunction was too general of a term (Birch 1964, cited by Conners 2000). Hyperactivity syndrome was thus produced which found a definition in DSM-II as Hyperkinetic Reaction of Childhood. The 1970s saw a shift in focus from hyperactivity to inattention as the main feature. The DSM-III published in 1980 renamed it Attention Deficit Disorder (with or without hyperactivity) (Barkley, 2006a). For empirical validity, in the revised edition of the DSM-III, it was renamed to Attention Deficit Hyperactivity Disorder. However, the fourth edition once again separated the counterparts by providing three sub-types i.e. a predominantly inattentive type, a predominantly hyperactive-impulsive type, and a combined type with symptoms of both dimensions (American Psychiatric Association, 1994). It also added that ADHD was found in adulthood and gave examples of workspace problems highlighting the same. The current version of DSM-5 consists of the same description except for a few more examples and a decrease in the minimum number of symptoms required for diagnosis.

CAUSES AND RISK FACTORS

Genetic factors

ADHD's genetic basis is robustly supported by various studies, indicating that approximately 79% of the variability in ADHD can be attributed to genetic factors. Family studies have consistently shown that ADHD is more common among first- degree relatives of individuals with this disorder, implicating a significant hereditary component. Twin studies further corroborate this with monozygotic twins displaying higher consistency for ADHD than fraternal twins. This genetic link is prevalent in adoption studies too, with higher rates of ADHD among biological parents of affected children than their adoptive parents. In the arguments made for genetic links of ADHD, the key genes implied include those involved in the dopaminergic system, particularly the DRD4 receptor gene and the DRD5 receptor gene. Besides, genes like DAT1 (dopamine transporter) and catechol-O-methyltransferase (COMT) have been studied extensively, with DAT1 showing more consistent associations. These genetic findings suggest a substantial hereditary influence on ADHD. Acting jointly, various rare genetic variants, including chromosomal anomalies and genetic syndromes too contribute to ADHD. Chromosomal anomalies involve abnormalities in chromosome number, such as sex chromosome aneuploidies, and structural abnormalities. Specific gene disorders such as Fragile X syndrome, tuberous sclerosis and certain

microdeletion syndromes such as Smith-Magenis syndrome and Velocardiofacial syndrome (VCFS) are linked to elevated ADHD rates, particularly the inattentive type. These genetic disorders are frequently associated with other neurodevelopmental and psychiatric conditions, including autism spectrum disorder (ASD) and psychosis in the case of VCFS. Despite these associations, routine genetic screening for these anomalies in ADHD patients is not standard practice due to the rarity and specificity of these conditions.

Environmental factors

ADHD is a complex disorder influenced by a mix of genetic and environmental factors. While inherited factors are significant, numerous environmental risk factors are also associated with ADHD. However, distinguishing which of these factors are causal is challenging due to the potential for reverse causation. Prenatal and perinatal factors such as complications during pregnancy or birth, maternal health issues, maternal stress, bleeding and protracted or complicated delivery all appear to be the primary environmental risks linked to ADHD. Additionally, substance exposure during pregnancy is another significant risk factor. Maternal smoking, alcohol consumption, and drug use have been consistently associated with an increased risk of ADHD. While heavy maternal alcohol use can cause fetal alcohol syndrome, which includes ADHD-like symptoms, the evidence linking less extreme alcohol use and ADHD is inconsistent. Similarly, the association between prenatal illicit drug use and ADHD remains inconclusive. Prematurity and low birth weight are also important factors. The association appears particularly strong for extreme prematurity and very low birth weight, which are related to inattention symptoms and the inattentive subtype of ADHD. Besides, intrauterine growth restriction has been preliminarily linked to ADHD, although it is unclear whether these factors are direct causes or simply risk markers.

Dietary factors

Dietary influences on ADHD have been comprehensively studied, though their impact is considered secondary to genetic and environmental factors. The hypothesis that sugar intake exacerbates ADHD symptoms has been largely debunked by research, which shows no consistent evidence supporting a direct link. However, some studies suggest that artificial food colorings and preservatives might aggravate symptoms in susceptible individuals. Restricted elimination diets, which remove certain food additives, have shown some promise in reducing symptoms for a subset of children with ADHD. Additionally, deficiencies in micronutrients like zinc, iron, magnesium, and omega-3 fatty acids have been examined. While supplementation of these nutrients can sometimes alleviate symptoms, particularly in children with existing deficiencies, diet alone is not seen as a primary cause of ADHD. These findings suggest that dietary adjustments might be beneficial in managing symptoms but are not a core factor in the disorder's etiology.

Psychosocial factors

Psychosocial elements significantly influence the expression and severity of ADHD symptoms. Adverse family environments, including low parental education, poverty, negative parenting practices, and high levels of family conflict, are associated with an increased risk of ADHD. These factors can exacerbate symptoms in children who are genetically predisposed to the disorder, indicating that the environment can significantly impact how ADHD manifests and progresses. Findings suggest that while psychosocial factors do not directly cause ADHD, they play a critical role in its development and can significantly affect its severity and management. Supportive and structured environments are crucial in mitigating the impact of ADHD symptoms

These risks have remained relatively consistent over time, suggesting a complex interplay of genetic and environmental factors. Gene-environment interactions and epigenetic mechanisms might further complicate this relationship. Despite significant associations between various environmental factors and ADHD, establishing clear causal links requires more rigorous research.

Signs and Symptoms

This neurodevelopmental disorder is oftentimes characterized by persistent patterns of inattention, hyperactivity, and impulsivity that significantly impact an individual's daily functioning, relationships, and work performance. Although commonly diagnosed in childhood, ADHD symptoms often persist into adulthood, presenting differently as individuals age with higher rates among males.

Inattention in ADHD is characterized by a persistent and pervasive difficulty in maintaining focus and attention on tasks. Individuals with ADHD often experience significant challenges in sustaining concentration, particularly on activities that demand prolonged mental effort. These individuals may initiate tasks but frequently fail to complete them, transitioning from one activity to another without finishing any. Forgetfulness is a common symptom, manifesting as a frequent loss of items, failure to complete chores or homework, and difficulty recalling details of conversations or plans. This inattention frequently leads to numerous errors in tasks that require meticulous attention to detail. Additionally, individuals with ADHD are easily distracted by extraneous stimuli. Background noise, concurrent activities, or even their own thoughts can significantly sidetrack their focus, further exacerbating the challenges associated with inattention. Driving impairments are common due to difficulties in attention, maneuvering, and planning along with increased sensation-seeking behavior in those with ADHD which can lead to more road accidents (Goodman, 2007).

While inattention is a significant aspect of ADHD, the hyperactivity symptoms, although more noticeable in children, can also persist into adulthood. In children, hyperactivity might manifest as constant fidgeting, an inability to stay seated, running or climbing in inappropriate situations, and excessive talking. Adults, on the other hand, may experience hyperactivity more as an internal sense of restlessness or agitation. They might feel a constant urge to be in motion, have difficulty relaxing, or engage in excessive talking. This restlessness can affect their ability to stay focused, maintain steady work on tasks, or enjoy sedentary activities. The internal hyperactivity in adults can lead to a feeling of being constantly on edge or unable to sit still, which can be socially and professionally disruptive.
Impulsivity in individuals with ADHD is marked by a tendency for hasty actions without considering consequences. This often appears as interruptive speech, difficulty waiting for one's turn which impacts social, academic, and professional settings. Lower academic achievement, poor job performance, attendance issues, isolation, and unemployment may manifest (American Psychiatric Association, 2013; Usami, 2016). Impulsivity significantly affects professional and social environments. Socially, the inability to regulate impulsive responses can disrupt interactions and be perceived as disrespectful, complicating relationships. Individuals with ADHD may engage in antisocial behavior and experience higher incarceration (Goodman, 2007). ADHD-related impulsivity is a consistent pattern that interferes with effective functioning across various life areas.

Pathophysiology

ADHD is frequently associated with cognitive and motivational challenges, alongside abnormalities in resting-state brain activity, indicating impaired functioning in distinct neuronal networks. Cognitive deficits in ADHD may result from dysfunctions in fronto-striatal or meso-cortical brain networks, while issues with reward processing might be linked to dysfunctions in the mesolimbic dopaminergic system. However, these deficits can also manifest in the resting brain. A fundamental neuronal network approach suggests that, in ADHD, activity in the Default Mode Network (DMN), typically active during rest, may disrupt the activity of neuronal networks involved in task processing, leading to difficulties in state regulation and periodic lapses in attention. The precise cause of ADHD remains unclear, but early hypotheses about diminished brain function were founded on observations of reduced volume or functionality in gray and white matter, associated with deficits in cognitive processing, attention, motor planning, response speed, and other behavioral issues characteristic of ADHD. More recently, key areas demonstrating abnormalities in individuals with ADHD have been identified as the prefrontal cortex, caudate nucleus, and cerebellum. These regions are interconnected by neuronal networks that collectively manage attention, thoughts, emotions, behavior, and actions. Studies have shown that patients with ADHD often exhibit slower maturation of the prefrontal cortex or reduced volume and activity in the prefrontal cortex, caudate, and cerebellum. The network activity among these areas is highly sensitive to the neurochemical environment and is regulated by neurotransmitters, specifically dopamine and norepinephrine, which interact through multiple pre- and postsynaptic receptors.

In ADHD, dopaminergic activity is notably dysregulated, particularly in the prefrontal cortex and striatum, crucial for attention, executive function, and reward processing. The prefrontal cortex relies on optimal dopamine levels for tasks requiring attention and working memory. Reduced dopamine transmission in the prefrontal cortex leads to deficits in these cognitive functions, manifesting as inattention and poor executive control. Additionally, the striatum, part of the brain's reward circuitry, exhibits altered dopamine signaling in ADHD. This results in an impaired reward system, where individuals have difficulty with delayed gratification and exhibit increased impulsivity and hyperactivity. The mesolimbic pathway, connecting the ventral tegmental area to the nucleus accumbens, also shows reduced dopamine activity, contributing to motivation deficits seen in ADHD. Genetically, ADHD is associated with variations in genes responsible for dopamine transport (e.g., DAT1) and receptors (e.g., DRD4). These genetic differences lead to increased dopamine transporter density, causing rapid dopamine reuptake and reduced synaptic dopamine availability. This synaptic imbalance disrupts normal dopamine signaling, exacerbating the core symptoms of ADHD. Additionally, norepinephrine produced in the locus coeruleus, is essential for modulating attention, arousal, and executive functions. In the prefrontal cortex, norepinephrine enhances focus and cognitive control. Individuals with ADHD exhibit reduced norepinephrine activity in the prefrontal cortex, leading to difficulties in sustaining attention, organizing tasks, and inhibiting impulses, core symptoms of ADHD. Additionally, norepinephrine dysregulation impacts the brain's attention networks, including the dorsal attention network and the ventral attention network. This results in impaired sustained attention and difficulty shifting focus appropriately. ADHD is also associated with hypoarousal, where decreased norepinephrine levels result in problems maintaining alertness and responsiveness to stimuli, exacerbating inattentive symptoms. Genetically, variations in the norepinephrine transporter and adrenergic receptors affect norepinephrine reuptake and receptor function, further contributing to the dysregulation seen in ADHD. These genetic factors lead to altered norepinephrine availability and signaling, impairing cognitive and

behavioral regulation. Medications used to treat ADHD aim to restore the balance in neurotransmitters, but accurate diagnosis is essential to avoid unnecessary drug exposure in unaffected individuals.

Prophylaxis

Due to its genetic and neurodevelopmental etiology, ADHD cannot be prevented. However, the risk for ADHD can be reduced by maintaining good brain health. Pregnant women must not engage in drinking alcohol and smoking and exposure to environmental toxins must be restricted. Maternal stress and diabetes have also been linked to ADHD in the child (Halperin, 2012) so being mindful about maternal health is crucial. It is also essential for them to maintain higher high-density lipoprotein levels that benefit fetal brain development (Y et al., 2017). Maintaining a low-sugar diet with necessary probiotic intake during pregnancy may also be a preventative measure (Jain, 2023). A study has shown that maternal phone usage during pregnancy results in children who have hyperactivity or inattention problems (Birks et al., 2017) thus lifestyle changes may be advantageous. There are also ways the effects of ADHD can be reduced if discovered early enough. Genes and environment interact to manifest the symptoms of ADHD epigenetically. Although not much research shows secondary preventative measures, targeting the risk factors may help. Exercise can greatly benefit brain development, especially as it targets the activation of the prefrontal cortex and enhances executive functioning which is germane to ADHD. Attention and cognitive-related exercises directed toward school children have shown better attention management and improvements in impulse inhibition, working memory, and executive functioning (Klinberg; Kerns; Shalev). These effects stretch to preschool children as well (Diamond, 2007; Thorell, 2009). Directive parenting, which involves guiding the child with verbal and physical cues without being controlling, can foster the child's self-regulatory abilities and help prevent the escalation of ADHD symptoms (Nicole et al., 2023). By intervening at a young age, the brain’s neuroplasticity allows for longer-lasting change. This can help reduce the complications and comorbidities that follow ADHD.

TREATMENT METHODS

Although ADHD has no cure, there are ways to manage and reduce the symptoms experienced. Medication, therapy, education, training, or a combination of treatments are used to treat ADHD.

Medications

Stimulants have proved to be quite effective in the management of ADHD symptoms. They are not a permanent remedy but they can help an individual to focus, control excessive moments or impulses, and allow the individual to function in their daily lives. They stimulate the central nervous system (CNS) to increase dopamine and norepinephrine secretions in the prefrontal cortex. However, like most pharmaceuticals, they have side effects that could affect one’s health and thus need to be monitored carefully. Decreased appetite, sleep problems, mood swings, headaches, and stomach aches are often reported as side effects. They tend to elevate heart rates and blood pressure which can cause cardiovascular issues (Olfson). The risk of psychosis cannot be ignored; medications tend to induce psychosis and tics but they usually subside on discontinuation. The presence of ADHD itself could be a risk factor for psychosis (Reiersen et al., 2018). Physical dependence and addiction are additional potential side effects of stimulant medications that must be seriously considered. Non-stimulants are also prescribed, although the Food and Drug Administration (FDA) has not approved them for the sole purpose of treating ADHD (*Attention-Deficit/Hyperactivity Disorder - National Institute of Mental Health (NIMH)*,

n.d.). They may reduce hyperactivity and impulses. Individuals may have to take several medicines to gauge what works best for them.

Methylphenidate (e.g., Ritalin) and amphetamines (e.g., Adderall) remain first-line treatments for ADHD, effectively improving attention and reducing impulsivity. These medications are widely prescribed and accessible in India. Atomoxetine (Strattera) offers an alternative for those who do not respond to stimulants. It is available in India but may be less commonly prescribed due to cost considerations (Ledbetter, 2006).

Psychotherapy and psychosocial interventions

To reach a child with ADHD, the remedy needs to pass through the parents. The American Academy of Pediatrics recommends behavioral management through parental training for children below 6 years of age. It is only post 6 years that medicines are introduced, along with continued behavioral management training (Centre for Disease Control and Prevention, n.d.). Young & Myanthe gave a comprehensive recommendation on the best approaches to non-pharmacological treatment from pre-school to adulthood. For parents of younger children, education about the disorder and its symptoms is essential along with arming them with strategies to deal with it. They must be taught behavioral techniques such as rewarding children with praise, positive attention, and materials such as stars or stickers that can be accumulated to redeem a desired reward. Unwanted behaviors can be reduced by implementing strategies like strategic ignoring, time-outs for disallowed actions, clear directives, and other comparable non-physical disciplinary techniques. Parents must look out for their mental health through counseling and support groups to efficiently rear their children. The authors also suggest roles for the educational sector to deal with ADHD children. Along with appropriate psychoeducation and written material on ADHD, they must be informed of behavioral interventions that can be used to reinforce or deter children from specific actions. Tokens may be used along with targets and goals in the form of Daily Report Cards to direct the child's behaviour. They must recognise the triggers and consequences of the children's actions and manipulate their environment to decrease disturbance, like seating them away from the window. Parents and teachers must have meetings to discuss the child's behavior. Middle or high school children might need remedial or revision lessons and individual support.

Cognitive behavioural techniques effectively help a child traverse through their daily life. Cognitive restructuring can help combat negative thoughts and beliefs. They can be taught how to socialize appropriately, think constructively to solve problems, and learn to independently monitor and reinforce themselves. These can be achieved through role-playing, making children think out loud, asking them to rate their attention, and acknowledging their gains. Cognitive training may also be utilized wherein the working memory is strengthened through computerized training programs like Cogmed Working Memory Training (CWMT) and Braingame Brian (Shreshtha et al., 2020). Multimodal treatment is increasingly used which involves a combination of medications and therapies and is found to have significantly higher treatment efficacy (91.84%) than pharmacotherapy alone (75.51%) (Lv et al., 2023).

Alternative therapies

Neurofeedback is becoming increasingly popular in the treatment of ADHD. It involves the measurement of one's brain activity which is related to one thus facilitating self-regulation and changing the underlying neural connections directing one's cognitive and behavioural outputs (Enriquez-Geppert et al., 2019). There are several standard protocols used in

neurofeedback. Theta/beta ratio (TBR) neurofeedback aims to reduce theta waves and/or increase beta waves in central and frontal areas. This is important because children and adults with ADHD often have high theta power and low beta power. It has been found to reduce ADHD symptoms and improve academic performance as effectively as the medication methylphenidate. Sensorimotor rhythm (SMR) neurofeedback focuses on improving sleep quality thus improving attention. Slow cortical potential training (SCM) helps the individual control cortical activation and inhibition, thus improving attention, reaction time, and stimulus detection. Neurofeedback and behavioural modification most commonly show improvements in ADHD (Hodgson et al., 2012).

Integrated therapies have also been employed in the treatment of ADHD. Herbal medicines, dietary supplements, and mind-body connection practices like mindfulness and yoga are often used to deal with symptoms. Ayurveda, homoeopathy, and Chinese traditional medicines are also in use in several places around the world. Research has shown improvements in the reaction time of ADHD children when given ayurvedic products along with medicines (Singhal et al., 2010). Tai chi, which involves slow, gentle movements with a focus on deep breathing, has been effective in reducing hyperactivity and aggressiveness (Wen, 2009). Clinical hypnotherapy encourages a mind-body connection in a state of altered consciousness and has been found to reduce ADHD symptoms (Hiltunene et al., 2004; Anderson et al., 2000). The addition of Vitamin D and Omega-3 fatty acids in the diet can also benefit those with ADHD (Patrick & Ames, 2015).

It is essential to use a combination of therapies to determine which is best suited for an individual with ADHD. Behavioural interventions are crucial and must be used alongside medicines. Integrated techniques have become popular but more research is required to estimate the extent of their benefits.

Current Research

The aetiology of ADHD is multifaceted, involving genetic, environmental, and neurobiological factors. Recent research has increasingly focused on the genetic components of ADHD, with particular attention to polymorphisms in genes associated with dopamine regulation. A notable study identified a significant link between the SLC6A3 gene, which encodes the dopamine transporter, and ADHD. Consistent with the previous research, the study found that the 5R allele and the homozygous 5R/5R genotype have a higher frequency in individuals with ADHD compared to the control group. The 5R allele has been associated with enhanced expression of the SLC6A3 gene, which could lead to altered dopamine transporter function. This is consistent with the existing hypothesis that dysregulation of dopamine transport and signalling is a core feature of ADHD pathology (Maitra et al., 2014).

The polymorphism rs3836790 also appears to influence the response to methylphenidate, a common stimulant medication used to treat ADHD. The study found that individuals with the 5R genotype have weaker therapeutic responses to methylphenidate compared to those with the 6R allele. This suggests that genetic testing for this polymorphism could potentially guide more personalized ADHD treatment strategies, and optimize therapeutic outcomes based on an individual's genetic profile. An intriguing aspect of the study is the identification of a recessive inheritance pattern for the 5R allele in ADHD. The data indicate that individuals with the 5R allele at both loci have more than a 2.5 times higher likelihood of exhibiting ADHD symptoms (Seymari et al., 2023).

Moreover, the study explored the association between various genotypes of the rs3836790 polymorphism and comorbid disorders. The frequency of the heterozygous 6R/5R genotype was significantly higher in ADHD patients with at least one comorbid disorder compared to those without comorbidities. This result contrasts with Maitra's study, which reported a higher frequency of the 5R/5R genotype in ADHD patients with comorbidities. The discrepancy may be attributed to differences in the types of comorbidities assessed and genetic variations among different populations (Maitra et al., 2014; Seymari et al., 2023). Additionally, some studies failed to find an independent association between the 6R allele and ADHD, suggesting that the relationship may be contingent on other genetic factors, such as the simultaneous presence of the 9R allele of the 40-bp 3′-UTR polymorphism.

Recent research has turned a spotlight on the genetic factors that might predispose individuals to experiencing Intimate Partner Violence (IPV), particularly focusing on the genetic underpinnings of mental health conditions like ADHD. A comprehensive study involving 11,344 women has provided critical insights into how genetic predispositions to ADHD and other mental illnesses correlate with IPV experiences. The study revealed that Polygenic Risk Scores (PRS) for ADHD, Major Depressive Disorder (MDD), neuroticism, schizophrenia, and combined mental illness were consistently associated with the experience of IPV. These associations spanned multiple forms of IPV, including physical, emotional, and sexual abuse. Notably, women with higher PRS for these conditions also reported a greater number of IPV types and more chronic experiences of IPV. Beyond the direct experience of IPV, PRS for neuroticism, schizophrenia, and overall genetic risk were linked to intimate partner intimidation and control. Furthermore, PRS for ADHD, Autism Spectrum Disorder (ASD), MDD, schizophrenia, and combined mental illness showed significant associations with harassment from a current or former partner (Ratanatharathorn et al., 2024). These findings are significant as they build upon previous research indicating that depression and ADHD are linked to a higher likelihood of subsequently experiencing IPV. While prior studies on neuroticism and ASD as predictors of IPV have yielded mixed results, the current research utilized genetic data, which is less susceptible to environmental confounders and reverse causation, providing a more robust analysis of these associations.
Researchers from Oregon Health & Science University (OHSU) and the University of Minnesota Masonic Institute for the Developing Brain have confirmed the brain-wide effects of Attention-Deficit Hyperactivity Disorder (ADHD). Using neuroimaging data from the Adolescent Brain Cognitive Development (ABCD) study, researchers developed a polyneuro risk score (PNRS). The association between the PNRS and ADHD symptoms was tested in a subset of the ABCD study group and further validated in the independent Oregon-ADHD-1000 case-control study group. In both groups, the findings demonstrated a robust association between brain-wide connectivity and ADHD symptoms. This is remarkable because much of the prior research has focused on individual regions of the brain, but this study shows that it isn’t the case across the board. The study's results suggest that signals from all areas of the brain contribute to the risk of ADHD, underscoring the need for a whole-brain approach in ADHD research, diagnosis, and treatment. Looking forward, researchers plan to explore whether these brain connectivity findings are consistent across different ages and throughout a child's lifespan. The ultimate goal is to refine the PNRS method to a point where it could be used in healthcare settings, providing predictive assessments for ADHD risk, thereby improving early identification and intervention strategies (Mooney et al., 2024; Norman et al., 2024).

CONCLUSION

Our comprehension of Attention-deficit Hyperactivity Disorder has reached sophisticated levels only after several authors have attempted to explain its intricacies. With a ubiquitous prevalence, ADHD has found its way into the lives of millions. Understanding a disorder is the first step towards its treatment. Although a cure for this condition remains elusive, effective coping methods can help individuals lead much more functional lives. The masses must therefore be acquainted with the clinical picture of ADHD in order to accurately identify and treat the disorder without over-medicalising or underestimating the symptoms. While medications and behavioral interventions have shown considerable results thus far, research continues to explore genetic and biological correlates, environmental influences as well as consequences, and treatments within and beyond the conventional forms. An in-depth appreciation of ADHD will allow clinicians, afflicted individuals, and laypeople to develop better strategies for attentional deficits and hyperactive manifestations.

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