Investigating the Mechanism, Diagnosis, and Treatment of Attention Deficit/Hyperactivity Disorder

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Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is a highly prevalent and heritable disorder characterised by inattentiveness, impulsivity, and hyperactivity. The principal causes of ADHD are genetics, prenatal exposure to toxic substances, and brain damage. When left untreated, the symptoms can last until adulthood. The diagnosis of ADHD involves interviews, questionnaires/tests, and evaluations from parents or teachers to establish which of the three types of ADHD the patient has. ADHD is usually more common in males than females. The treatment of ADHD should consider the patient's comorbidities, age, and medical history when prescribing medication, therapy, or devices. This paper reviews the modifiable and non-modifiable causes of ADHD, tests, interviews conducted to diagnose ADHD, and the multiple forms of treatment for children and adults.

Index Terms— Attention deficit hyperactivity disorder, cause, diagnosis, predominantly hyperactive, predominantly inattentive, treatment.

1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a neurological disorder defined by a prolonged pattern of inattentiveness or impulsivity and hyperactivity that impedes the development or functioning of individuals. ADHD can be manifested differently across the lifespan. Children with ADHD may find it challenging to keep quiet and stand still, whereas teenagers and adults can experience more anxiety and depression, as well as appear forgetful, careless, and disorganised [1], [2].

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth edition (2013), there are three categories of ADHD: (a) predominantly inattentive, (b) predominantly hyperactive-impulsive, or (c) a combined presentation. Considered one of the most common neurodevelopment illnesses being diagnosed, ADHD affects up to 8.4% of children and 2.5% of adults [3], [4]. For up to half of the diagnosed cases, symptoms can persist into adulthood and continue to affect the functioning of daily activities [5]. ADHD can adversely impact the patient by undermining academic and professional performance and subjecting them to social discrimination; the patient's family would face increasing financial costs as well as emotional burnout.

There has been an evident increase in the diagnosis of ADHD worldwide, especially in the United States, where prevalence rose from 6.1% in 1997-1998 to 10.2% in 2015-2016 [6]. Reasons underlying this rapid growth could be due to parents' and educators' higher awareness of symptoms or as a consequence of recent technological developments. The popularisation of short videos and social media is believed to have shortened the attention span of users, leading to restlessness and worse performance that manifest in ADHD-like behaviour [7].

Due to a lack of pathognomonic markers, measuring ADHD symptoms remains challenging. Some clinicians argue that a number of people diagnosed with ADHD have their personality traits labelled as a symptom causing over-diagnosis. Others even go as far as suggesting that ADHD is not a disorder but solely a state of mind that is unconventional to societal standards [8].

On the contrary, the opinion against over-diagnosis states that there is a more significant problem of under-diagnosis, especially among females. ADHD symptoms can be milder and more internalised (inattentive rather than impulsive) in females compared to males as society exerts more pressure on females to perform well and behave according to the norms; hence females can mask and better cope with their symptoms [9]. The prevalence rate of ADHD in males can be 3-16 folds higher than that of females in European countries, revealing how under-represented female symptoms can be in the diagnosis of the disorder [10].

ADHD, like most neurobehavioral disorders, is not entirely curable, but having an early diagnosis following proper treatment and an education plan can highly alleviate the symptoms. ADHD patients can be treated with pharmacological and psychotherapeutic interventions depending on the type of ADHD and its severity. Whether it is the traditional use of stimulants or the prescriptions of noradrenaline reuptake inhibitors and α-2 adrenergic receptor agonists, medication is almost certainly needed if there is functional impairment in two or more domains, and it usually leads to favourable outcomes and better performance. However, there remains scepticism towards the long-term effectiveness of these drugs, as well as a growing worry surrounding their abuse.

This paper will take into account the genetic and environmental causes of ADHD, including modifiable and non-modifiable risk factors, and evaluate the various diagnostic measures and tests to propose better treatment of ADHD to improve the well-being of patients on the individual, familial and societal levels.

2. Method

Publicly available databases such as PubMed, Scopus, and Web of Science were searched for peer-reviewed articles published in English without any time constraints, with search terms such as “attention deficit hyperactivity disorder,” “sleep disorders,” and “ADHD.” The title, abstract, and method of published papers were screened for suitability. Relevant papers were selected, and data were extracted for inclusion in the review. Publicly available search engines such as Google were searched with similar terms for additional resources.

3. Discussion

A. Causes

Similar to many other neurological disorders, ADHD has a complex aetiology. There is no single causal factor leading to its development; instead, multiple genetic markers, environmental factors, and the interplay between the two are responsible [11] (**Fig. 1**). It is worth noting that the presence of such risk factors does not necessarily lead to ADHD, as such factors can also be present in unaffected individuals.

The biological and external factors are interdependent and indirectly affect each other. Having specific genes may predispose affected individuals to environmental risk factors (gene-environment interaction) or increase the likelihood of someone coming into contact with certain environmental factors (gene-environment correlation) [12].

Structural Changes in the Brain

Not only is ADHD subject to genetic and environmental risk factors, but it also appears to be partially attributed to changes in brain structure and volume. People with ADHD have approximately 3-8% less cortical grey matter volume, smaller prefrontal, and anterior cingulate volumes, but higher white matter volume compared to control subjects [13], [14], [15] (**Fig. 1**). Moreover, there is significant cortical thinning across the whole brain, especially in the frontal and parietal lobes [16]. Such changes are nonpermanent as the reduction in cortical thinning is shown to disappear with age and eventually reach a similar average thickness as the controls [17], [18]. The rate of cortical thinning has an inverse relationship with the severity of ADHD symptoms [19]. More severe hyperactivity and impulsivity were seen in youths with a slower cortical thinning rate in prefrontal cortical regions, anterior cingulate, and orbitofrontal cortex. Thinning in the cerebral cortex can delay neurological development as cognitive networks underlying attention and executive functions are affected [15]. For children with ADHD, the median age of reaching peak thickness throughout the cerebrum is 10.5 years, much longer compared to 7.5 years of typically developing controls [20].

A study done on males aged 8-13 years old with ADHD using magnetic resonance imaging revealed a reduction of volume and change in the shape of the basal ganglia, especially in the right putamen/globus pallidus region [21] (**Fig.2**). Changes in right putamen can be a neuroanatomical marker for defectiveness in frontostriatal circuits modulating cognitive functions. 15 children out of 76 with no prior ADHD history showed ADHD symptoms after head injuries, and they showed more lesions localised in the right putamen than the remaining 61 [22].

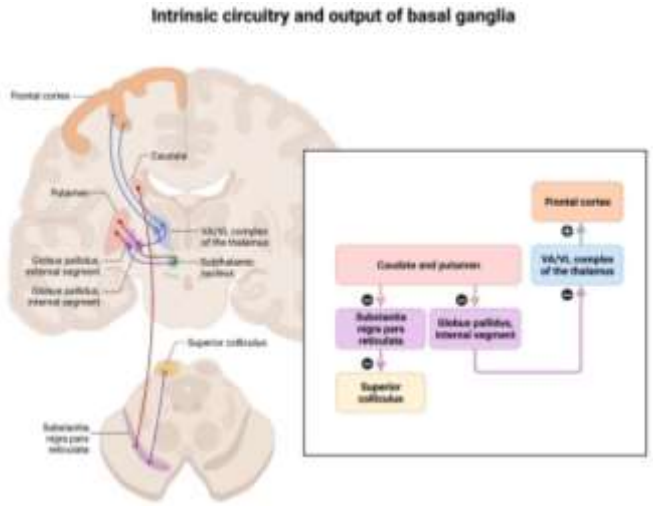

Figure 1. The diagram shows the neuronal circuits and neurotransmission mechanism of the direct, indirect and nigrostriatal pathways of the basal ganglia. The stimulatory and inhibitory nature of the neurotransmitters glutamate, GABA, dopamine as well as D1 and D2 receptors

allow the basal ganglia to carry out its core functions of starting, stopping and modulating actions.

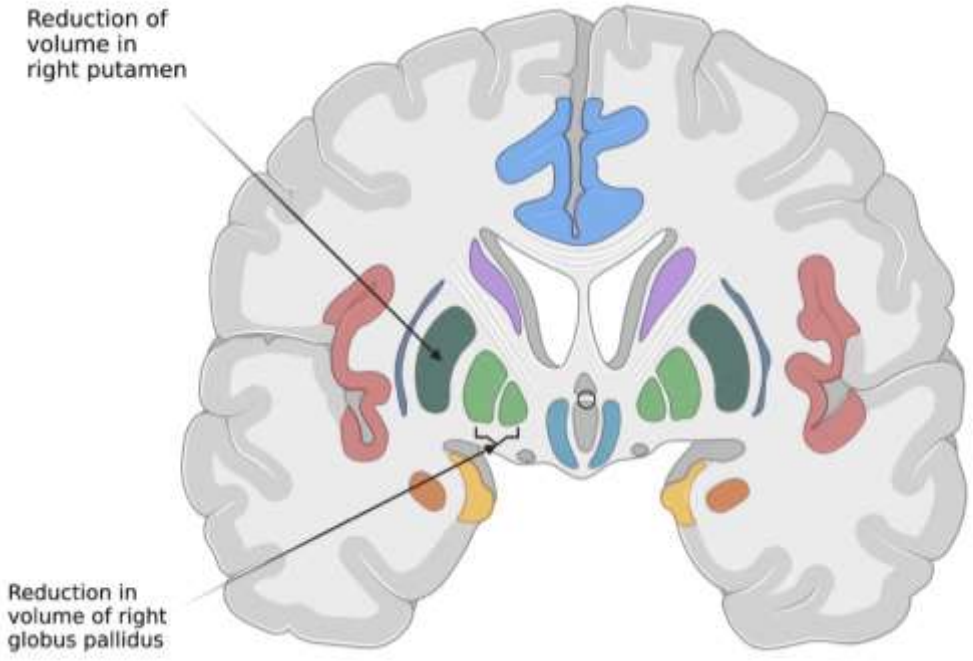

Figure 2. The reduction of volume and change in shape of the right putamen/globus pallidus in males aged 8-13 years old.

Genetically Inherited

Multiple studies indicated that ADHD has a genetic component. It is shown that the siblings of those diagnosed with ADHD were nine times more likely to be affected by the disorder compared to individuals without affected relatives [23]. Multiple genes, such as the tryptophan hydroxylase 2 (TPH2) gene on chromosome 12q21, encoding for the enzyme TPH2, or Cadherin 2 (CDH2) on chromosome 18q12, encoding for the Cadherin-2 protein have been suggested to play a role in ADHD [24], [25], [26].

The results of a twin study examining parents' and teachers' reports estimated the heritability of ADHD to be around 72% to 80% [27]. The results, however, are limited in depicting how much genetics contribute to the development of ADHD, as environmental factors are not excluded. Further research needs to be done to ensure the validity of the findings, as the basis of the twin studies is built upon the equal-environment assumption (EEA), which states that monozygotic and dizygotic twins are exposed to the same environmental factors to the same extent. There exist opposite opinions and evidence that support and argue whether the violation of EEA would undermine the credibility of the findings, for instance, various critical analyses published by Jay Joseph, or on the contrary, research showing that the EEA was not [28]. In turn, studies on adoptive families are more reliable in excluding external factors from prenatal ones. A study found the rate of ADHD in adoptive parents of adopted ADHD probands to be 8%, with 3% of the biological parents of the control proband having ADHD. This is compared to the 18% of the biological parents of non-adopted ADHD probands who had ADHD [29].

Meta-analysis of various genome-wide linkage scans is used to compare and identify whether any chromosomal region is shared more often between family members with ADHD. Using

Genome Scan Meta-Analysis, a study found genome-wide linkage on chromosome 16 between 64 and 83Mb [30]. However, follow-up research is required to further determine the increase in the probability of people having ADHD with the presence of this genetic region.

Prenatal Exposure to Acetaminophen

Paracetamol, otherwise known as acetaminophen (APAP), has been shown to induce damage to foetal development during pregnancy and infancy if used for a prolonged period of time. APAP affects endocrine-disrupting compounds known to alter masculinisation by inhibiting testosterone level production in mice, causing direct neurotoxic damage to rat cortical neurones both in vitro and in vivo. [31], [32]. Significant differences in DNA methylation were correlated to prenatal exposure to APAP for more than 20 days in children diagnosed with ADHD compared to controls [33]. DNA methylation at birth was found to be associated with later ADHD symptoms development with genome-wide significance at nine loci, and cord blood methylation is suggested to be a marker for some ADHD risk factors before birth [34]. Exposure to APAP during pregnancy was found to lead to a 30% higher risk of ADHD, as well as an increased risk of developing other neurological disorders, such as a 20% higher risk of autism spectrum disorder [35].

Childhood Exposure to Lead

Childhood exposure to lead, even at very low levels, has been shown to cause ADHD in children. Blood Lead Level (BLL) of less than 10μg/dL and even less than 5μg/dL are associated with at least one type of ADHD, i.e., inattentive, hyperactivity/impulsivity, or combined [36]. In one study, children with a BLL of 2.3μg/dL were at a 2.5-fold higher risk of attaining ADHD than those who did not come into contact with it. The association between BLL and ADHD symptoms, especially hyperactivity/impulsivity, was significantly heightened in males by a gene mutation HFE C282Y, which is estimated to be found in 10% of the U.S. population [37], [38].

Extremes of Psychosocial Deprivation

Many forms of negligence from caretakers or the lack of resources such as books and toys may limit the cognitive development of children, resulting in long-lasting effects that would carry over into adulthood. Children exposed to early institutional deprivation have been shown to be defective in many neurological functioning, such as I.Q., prospective memory, proactive inhibition, quality of decision, and recognition of emotions, compared to non-deprived controls [39]. Golm's study also supported a positive correlation between childhood psychosocial deprivation and symptoms of ADHD and autism spectrum disorder.

The extent to which children are exposed to institutional deprivation is associated with the severity of ADHD symptoms experienced in adolescence and adulthood. At low deprivation of below 6 months, the rate of ADHD in adolescents and adults was similar to those in the general population at 5.6% and 3.8%; however, when children were exposed to deprivation of more than 6 months (high-deprivation), the rates can go as high as 19% and 29.3% respectively [40].

Low Birth Weight

Low birth weight is defined as weighing less than 2500g at birth and is further categorised into a very low birth weight of <1500g and an extremely low birth weight of <1000g [41]. Children

born with low birth weight were found to have more difficulties in executive functions, verbal fluency, or working memory, as well as an increased chance of psychiatric disorders such as anxiety disorders, Asperger's syndrome, and ADHD [42], [43]. Approximately 23% of children born with very low birth weight meet the clinical criteria of ADHD compared to only 6% of controls [44]. Low birth weight is shown to be an independent risk factor for ADHD [45].

Low birth weight can be due to maternal smoking, maternal age, premature birth, or poor prenatal nutrition, which have all been shown to be harmful to fetal neurological development, even leading to a higher probability of neurological disorders. [46], [47], [48], [49].

Brain Injury

Traumatic brain injury (TBI) can lead to the development of ADHD in patients long after the event. The increased diagnosis of secondary ADHD, defined as an elevated T-score on the DSM-Oriented Attention-Deficit/Hyperactivity Problems Scale of the parent-reported Child Behavior Checklist was observed after 7 years post-injury in 26.7% of the 187 children who participated [50]. Adults with lifetime TBI were also significantly likelier to score higher on the Adult ADHD Self-Report Scale [51].

In contrast, another study in adults showed no correlation between head injury or trauma with ADHD. The prevalence of ADHD in adults with head trauma was 4.9% compared to 5.1% in non-traumatic adults, which did not show a significant statistical difference [52]. These discrepancies can be due to the differences in the participants being studied, differences in study design, or experimental variations. In Ilie's study, TBI was precisely defined as a trauma to the head that resulted in at least five minutes of unconsciousness or overnight hospitalisation, whereas Amiri did not specify the type or duration of trauma. Other factors such as the sample size and type, amount of care and support received after TBI, other diseases in participants, and participants' genetic differences could also be the reason for such inconsistencies. Hence, further research is needed to distinguish between the effect of TBI during childhood and adulthood on the likelihood and severity of ADHD post-injury to better inform and aid patients suffering from TBI.

B. Diagnosis

The three categories of ADHD (primarily inattentive, primarily hyperactive-impulsive, or combined) manifest different symptoms accordingly (**Table 1**). Predominantly inattentive ADHD, previously known as attention deficit disorder, is now a category of ADHD in which patients are distractible but not hyperactive. It has been shown that predominantly inattentive ADHD is the most common type of ADHD amongst the population, even though those with combined ADHD are more likely to be referred to clinical services [53]. According to DSM-5, the common symptoms in children can include making careless mistakes, having difficulty sustaining attention, having poor organisational skills, failing to follow instructions or rules, losing personal items, or being forgetful.

The second type of ADHD is predominantly hyperactive ADHD. People with hyperactive ADHD were diagnosed with attention deficit disorder with hyperactivity before the American Psychiatric Association's revision in 1987, which combined it with attention deficit disorder with hyperactivity to form one condition called ADHD [54]. Symptoms of predominantly hyperactive

ADHD include fidgeting, squirming, leaving seats when asked not to, restlessness, interrupting others, or talking excessively.

The last type of ADHD is combined ADHD. Children would experience symptoms of both inattentiveness and hyperactivity. One study showed that 62% of adults with ADHD had combined ADHD, of which 31% are predominantly inattentive and 7% are predominantly hyperactive [55]. This may be due to the fact that teachers and parents more easily recognise the combined presentation as the symptoms align more closely with what most people understand of ADHD.

Table 1. The table illustrates the 3 categories of ADHD, and their symptoms according to DSM 5 Criteria. To be diagnosed with ADHD, at least five or more of these symptoms should be experienced for more than 6 months.

Type of ADHD	**Symptoms**
Predominantly Inattentive	1. Makes careless mistakes/lacks attention to detail 2. Difficulty sustaining attention 3. Does not seem to listen when spoken to directly 4. Fails to follow through on tasks and instructions 5. Exhibits poor organisation 6. Avoids/dislikes tasks requiring sustained mental effort 7. Loses things necessary for tasks/activities 8. Easily distracted (including unrelated thoughts) 9. Is forgetful in daily activities
Predominantly Hyperactive	1. Fidgets with or taps hands or feet, squirms in seat 2. Leaves seat in situations when remaining seated is expected 3. Experiences feelings of restlessness 4. Has difficulty engaging in quiet, leisurely activities 5. Is “on-the-go” or acts as if “driven by a motor” 6. Talks excessively 7. Blurts out answers 8. Has difficulty waiting their turn 9. Interrupts or intrudes on others
Combined	May include symptoms from both predominantly inattentive ADHD and predominantly hyperactive ADHD

ADHD has a much higher prevalence rate in males than females, with a male-to-female ratio of 4:1 in community samples [56], [57]. It is also argued that females have developed better coping strategies to mask their underperformance by working harder [9].

Depression, anxiety, bipolar, and personality disorders have a significantly higher prevalence in women with ADHD than men, who showed a more considerable prevalence in schizophrenia and substance use disorder [58]. This could lead to the misdiagnosis of these disorders without ADHD being considered. Females with predominantly inattentive ADHD also tend to report lower arousal, and may be falsely treated for dysthymia rather than ADHD, whereas those exhibiting combined or predominantly hyperactivity could be considered bipolar [9]. Future research is required to further examine the gender differences to address the under-diagnosis and under-representation of females in ADHD. The limitations of current literature should be considered, including referral bias, potential confounding variables, methods of collecting data, sample size, and diagnostic criteria.

Everyone can experience ADHD symptoms, but the symptoms interfere with regular activities and cause dysfunction in everyday life of ADHD patients. According to DSM 5, 5 criteria must be met for someone to be diagnosed with ADHD: 1) five or more symptoms of inattention and/or five or more symptoms of hyperactivity must have persisted for longer than six months to a level that is inconsistent with developmental level and negatively impacts social and academic activities; 2) several symptoms were present before the age of 12 years; 3) several symptoms must be present in more than two different settings; 4) there should be clear evidence that the symptoms interfere with or reduce the quality of social, academic, or occupational functioning; 5) symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder, and are not better explained by another mental disorder.

The diagnosis of ADHD comprises a variety of scales and interviews. The medical professional will start by checking for symptoms using the DSM-5 criteria to ensure that the patient does match the criteria for ADHD. Normed rating scales are often used as a form of self-report to gather initial information about the severity and frequency of symptoms, as they are primarily questionnaires and checklists. Educators' and parents'/guardians' reports can also be used as additional evidence to understand the patient holistically. It is most often used to diagnose children with ADHD, as they may have difficulty explaining their own symptoms. There are various rating scales, such as the Barkley scale, Behaviour Assessment System for Children, Brown, Conners, or the Vanderbilt Assessment Scales. Then, in-depth clinical interviews with patients and one or two of their relatives and friends would enable the doctor or therapist to investigate the emotions experienced by the patient and understand the reason behind such behaviour. The interview can involve discussions and questions about the patient's problems, self-image, family history, as well as their life in school or at work.

Occasionally, ADHD can also cause physical symptoms. The prevalence of thyroid abnormalities has been shown to be higher in children with ADHD than in the average population [59]. Another study analysing over 4 million individuals in Sweden found that alcohol-related liver disease, chronic obstructive pulmonary disease, epilepsy, and obesity are the strongest correlated diseases among the 34 different physical conditions that have an increased diagnosis in ADHD patients compared to controls. However, the causality of such symptoms requires to be established in future large-scale studies [60].

C. Treatment

Therapy for young children mainly focuses on psychoeducation and training for parents. Parents and guardians learn and understand the behaviour and mechanism of ADHD, allowing them to be patient and guide their children through ADHD. A study showed that children of parents who received 3 months of well-structured psychoeducation involving expert lectures, group sessions, and access to an online community had a significantly higher medication adherence rate and lower ADHD rating scale scores than those who only had general clinical counselling [61].

The training of teachers can also support children outside of the home. Through encouraging positive behaviours and a constructive reward system, this teacher-led approach can likely influence students' behaviour and increase academic engagement. Meta-analysis of studies showed that teacher training interventions significantly improved teachers' knowledge of ADHD. However, further research needs to be done to determine the efficacy of such training in reducing children's ADHD symptoms.

The external trigeminal nerve stimulation has recently been approved by the Food and Drug Administration (FDA) for children between 7-12 years of age who were not taking prescription medicine. The device produces low levels of electrical stimulation to the brain's trigeminal nerve, significantly reducing symptoms of ADHD with no meaningful adverse events [62], [63]. Similarly, transcranial magnetic stimulation was shown to be effective in improving ADHD symptoms in adults [64]. However, there was one instance where repetitive TMS was associated with inducing a seizure in an adolescent patient [65].

In June 2020, the FDA approved EndeavorRx, a video game doctors can prescribe to treat ADHD in children. A study funded by the game provider has shown that the game improves focus and attention in children while presenting minimal adverse impacts. Future studies would need to be done to further confirm the effect [66].

Cognitive behavioural therapy (CBT) is one of the most effective forms of therapy in reducing ADHD symptoms in adults. It helps lessen the life impairments of people with ADHD, such as problems with organisation or procrastination. CBT changes how patients view themselves, allowing them to adopt a more positive thought process of reducing self-criticism or comparison to others. The therapist would work with the patient to improve self-regulation skills that often orient around 1) organisation, 2) distractibility, 3) cognitive restructuring, and optionally modules tackling procrastination and communication [67]. Throughout the course of many practices, the patient developed coping strategies and observed changes in their mindset, which would alter their choices and behaviours as their distorted thoughts and emotions diminished. Multiple studies showed that CBT, whether prescribed alone or combined with medication, can significantly improve core ADHD symptoms, emotional well-being, executive function, and social functional outcomes; the benefits of the treatment can last for 5-7 months after active treatment concluded [68], [69].

One of the core components of CBT is psychoeducation, where therapists explain and inform patients about ADHD, including its symptoms, how it can affect patients' thoughts, emotions, and behaviour and break down the process of therapy. A recent meta-analysis of the effectiveness of psychoeducational interventions shows that they significantly improved social skills in young people with ADHD [70].

The prescription of medication should consider the patient's age, coexisting conditions, and severity of existing symptoms. Therapy is advised for children below 6 years old, and some medications, such as methylphenidate, are not approved by the Food and Drug Administration to be used on young children. The prescription must be monitored and moderated by checking the patient's response to different medications.

Treatment for ADHD can involve either therapy or medication, but usually, a combination of both is used to achieve the best results. Medications prescribed for ADHD patients can be divided into stimulants and non-stimulants. Adderall, Focalin, Ritalin, Dexedrine, and Concerta are some of the most common stimulants prescribed for treating ADHD symptoms. Methylphenidate, one of the two molecules present in ADHD stimulants, has a responsiveness rate of 70%-80% [71], which functions by increasing the dopamine level.

Neural pathways may be defective in patients with ADHD, as neurotransmitters are prematurely reabsorbed by the pre-synaptic neuron, resulting in lower tonic dopamine levels in people with ADHD than those without ADHD, therefore requiring more extensive and frequent external stimuli to increase their phasic dopamine levels, and manifesting as hyperactivity or inattentiveness. Stimulants increase the dopamine level in the brain, resulting in more effective dopamine signaling in synapses [72]. Functional MRI data showed significantly enhanced activation in the bilateral inferior frontal cortex/insula, which are key areas of cognitive control, under the use of methylphenidate [73].

Common side effects of using stimulants involve loss of appetite, sleep disturbances, increased heart rate, and blood pressure, or even the development of tics in some rare cases [74], [75]. Patients with intolerable side effects have severe anxiety or addiction problems and may be prescribed non-stimulants instead. Large doses of stimulants can cause dependency or even substance use disorder, as they can quickly improve physical and mental performance. An estimated 20% of college students abuse prescription stimulants, mainly to increase concentration, alertness or to get a euphoric rush [76], [77].

Various types of non-stimulants/anti-depressants include selective noradrenaline reuptake inhibitors such as Strattera; norepinephrine-dopamine reuptake inhibitors, such as Wellbutrin, or tricyclic anti-depressants such as Pamelor. A 42% reduction in ADHD symptoms in adults was shown after 6 weeks of taking Wellbutrin [78]. Anti-depressants and anxiolytic medication are also effective in treating anxiety disorders and depression, which would be helpful for patients with comorbid conditions. However, high-dose of benzodiazepines and Z-drugs were shown to be associated with adult ADHD and a lower quality of life. Hence, it is crucial to notice signs of addiction [79].

4. Conclusion

Due to its high prevalence and heritability, ADHD is one of the most studied neurological illnesses. The symptoms of ADHD can range from having difficulty focusing, controlling impulsive behaviour, to organisational problems depending on the type of ADHD the person has. The aetiology of ADHD can be traced back to genetics, prenatal environment, and mental or brain trauma. ADHD can be treated medication, therapy, or more recently, devices and technology. More in-depth future research is needed in the field of ADHD, especially regarding the safety and

efficacy of pharmacological treatments in children with ADHD. More novel methods of treatment, such as the use of virtual reality and video games in improving symptoms, can also be a great area of research due to the large target population.

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