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Attention Deficit and Hyperactivity Disorder: A Review of Etiology, Pathology, Consequences and Treatment

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Abstract: Attention-deficit/hyperactivity disorder (ADHD) causes attention problems and hyperactive or impulsive behaviours that seriously affect the daily life of the person. As a common mental developmental disorder, the number of published studies on the subject is large and the relationship is complex. This article attempts to categorise the relevant studies by reviewing those related to ADHD. Forty-six studies are included in this paper, which are broadly categorised into three areas: etiology, pathology and consequences and treatment. These categories do not exist in isolation, but are interrelated and interact with each other. It concludes with an outlook on future research in relation to the limitations of the current studies.

Keywords: ADHD, etiology, pathology, treatment

1. Introduction

Attention deficit/hyperactivity disorder (ADHD), a group of behavioral patterns which can be present in a variety of environments, is characterised by pronounced difficulties focusing, short duration of attention, excessive activity, or impulsivity [1]. These issues obstruct the patient's daily social, educational, and occupational processes [1]. ADHD is not an emerging psychological disorder; the phenomenon of the 'uncontrolled and frantic' child has been documented in clinical diagnostic descriptions for over three centuries [2]. Still was the first to identify ADHD as a childhood disorder that primarily affected boys and was previously referred to as 'hyperactivity' or 'hyperkinetic disorder of children' [3]. Numerous studies have shown that the prevalence of ADHD, a common behavioural disorder in children, ranges from 2% to 7% worldwide, with an average of about 5% [4].

Over the past century, a great deal of research has been published on ADHD. These studies have explored topics such as the etiology, pathology, associated consequences and treatment of ADHD. Due to the number and range of studies currently available on ADHD and the relative complexity of the relationships between studies, this literature review attempts to reduce this confusion by categorising and analysing relevant studies. In the first part of this article, the causes of ADHD will be discussed from a genetic and environmental perspective. The second part will show the diagnostic criteria and epidemiological studies related to ADHD. In the third part, the consequences of ADHD and the interventions commonly used to treat ADHD, including pharmacological interventions and psychological interventions, are summarised. Finally, this paper concludes with a further discussion and summary of the three parts.

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2. Method

The literature search strategy and results used in this paper are as follows. Inclusion criteria were that studies: 1) describe the causes of the development of ADHD; 2) describe the diagnostic assessment of ADHD and the epidemiological studies; 3) assess the consequences arising from ADHD and the effectiveness of interventions. Studies were excluded if they: 1) did not contain empirical data. The search for this database was conducted on 1 December 2022.

This article was searched from Google Scholar, and Web of Science using the following combinations of search terms and their derivatives like: ['ADHD' OR 'attention-deficit/hyperactivity disorder'] and ['diagnostic criteria' OR 'etiology' OR 'assessment'] and ['consequence' OR 'treatment' OR 'intervention']. This study has obtained 46 articles about ADHD with its diagnostic criteria, causes, and interventions. The time of these articles was from 2000 to 2022.

3. Etiology/Risk

Many studies have focused on elucidating the specific etiology and risk factors for ADHD. Each of these risk factors will be discussed below.

3.1. Genetic Factors

Many studies based on families, twins and adoption provide evidence for a genetic component to ADHD. Chang et al. conducted a follow-up study of 1480 twin pairs with ADHD [5]. The study used the Attention Problems Scale to measure ADHD at the ages of 8 to 9, 13 to 14, 16 to 17 and 19 to 20 years. The results showed genetic stability of attention problems from childhood to early adulthood. Another international collaborative study included 894 children diagnosed with ADHD from 8 European countries and 1135 of their siblings. The results showed that the sibling relapse rate for ADHD increased ninefold to 12.7% compared to the control group, demonstrating a family association with ADHD symptoms [6]. Several studies focused on the association between the prevalence of ADHD in biological and adoptive parents and the prevalence of ADHD in their children. The results showed that the biological parents of children with ADHD were more likely to have ADHD than their adoptive parents or the parents of children without ADHD; and that children with parents with ADHD had more severe symptoms than children without parents with ADHD [7,8].

With advances in genetic technology and global research collaborations, researchers have been able to analyse genetic variation in ADHD across the genome by studying large numbers of samples. A study using the Genome Scan Meta-Analysis (GSMA) method to analyse data from multiple published ADHD genome-wide association scan studies sought to find consistent gene sequences with genetic signatures across studies [9]. The results showed consistent genome-wide significant linkage on a genomic region between 64 and 83 Mb of chromosome 16 [9]. Another genome-wide association study on ADHD collected genetic sequences from more than 20,000 people with ADHD and more than 30,000 controls from regions including Europe, North America and China [10]. Meta-analysis of genome-wide associations showed that genetic variants at a total of 12 loci were of genome-wide significance [10]. It is clear that, in most cases, ADHD, like many mental disorders, is influenced by a combination of genes. However, the effect of each variation is very small [11].

3.2. Brain Structure and Neurological Function Factors

In the last decade or so, with the development and use of techniques such as EEG, event-related potentials (ERP) and functional magnetic resonance imaging (fMRI), the number of studies on brain structure and function and neuroimaging in ADHD has grown rapidly [12]. Many neurostructural and functional brain imaging studies have shown that ADHD is also a neurodevelopmental disorder, with

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significant differences in brain structure and neurological function in individuals with ADHD compared to individuals without ADHD. In early studies, based on the dopamine reward circuit hypothesis, researchers noted that disrupted transmission of the neurotransmitter dopamine was associated with ADHD [12]. Volkow et al. scanned dopamine receptors and dopamine transporters in ADHD patients and normal individuals and found that dopamine receptors were lower and transporter protein availability was lower in ADHD patients than in normal individuals [13]. Another meta-study combining multiple scans of dopamine transporter protein levels in ADHD patients found that patients with higher than normal striatal dopamine transporter protein levels were unable to dispense with prior interference from drug treatment [14].

Some studies focused on changes in the volume, area and thickness of the patient's brain structures. For example, Norman et al. conducted a meta-analysis of structural and neurological brain function studies with over 1,500 ADHD patients and over 1,500 controls [15]. The results show that patients with ADHD have reduced grey matter volumes throughout the brain compared to normal individuals and exhibit abnormal structural and functional abnormalities in the basal ganglia and insula [15]. Other studies have shown differences in cortical thickness across the brain in individuals with ADHD compared to normal individuals [12]. For instance, one study included MRI data from 70 individuals aged 9-17 years with ADHD and typically developing individuals, and the analysis found that total cortical volume, surface area and mean cortical thickness were smaller in people with ADHD.

3.3. Environmental Factors

In addition to possible genetic susceptibility and abnormalities in neurological brain function, environmental factors are also a major risk factor for ADHD. As with many neurodevelopmental disorders, ADHD is closely linked to prenatal environmental factors. The results of several studies confirmed this view. For example, Manzari et al. conducted a systematic review and meta-analysis to examine the relationship between maternal prenatal stress and children with neurodevelopmental disorders [16]. Prenatal stress is a major stressful life event for the mother during pregnancy, such as loss of a loved one, natural disasters and other traumatic events, with adverse consequences such as anxiety and depression [16]. The results suggested that, similar to autism spectrum disorders, a mother's prenatal stress exposure is associated with an increased risk of ADHD in her child [16].

In addition to prenatal maternal stress exposure, other early risk factors have been of interest to researchers, such as exposure to toxic substances. Lead is a toxin that can cause damage to many organ systems in the body, particularly the brain areas associated with ADHD (prefrontal cortex, basal ganglia, striatum, etc.) and the neurotransmitter system (dopamine pathway). More seriously, young children are more likely to absorb lead and are less resistant to the toxin than adults [17]. A meta-analysis included more than 30 studies over a 40-year period that examined the relationship between children and adolescents with ADHD and their exposure to lead toxins, including more than 10,000 subjects [17]. The results showed that lead toxin exposure was significantly associated with two major symptoms in patients: inattention and hyperactive or impulsive behaviour, although both effect sizes were in the moderate range, still suggesting that lead exposure is associated with ADHD [17]. Some other toxic exposures have also gained the attention of researchers, such as air pollution and parental smoking is associated with an increased risk of ADHD in children and adolescents, but these risk factors need to be considered in combination with genetic and socio-economic factors [18,19].

Many studies have shown that acquired adverse environmental factors are also associated with the development and maintenance of ADHD symptoms in individuals. For example, studies have shown that parenting styles are associated with the development of ADHD. Ullsperger et al. conducted a study attempting to demonstrate the indirect effects of parenting on symptoms of childhood ADHD through child temperament characteristics [20]. The results showed that inconsistent parenting was

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associated with the severity and persistence of ADHD symptoms and could predict more hyperactive or impulsive behaviour. The results of another study on parenting styles and ADHD symptoms similarly showed that patients were subjected to less emotional care and excessive protection and control compared to normal individuals, and these were significantly associated with inattentive and hyperactive-impulsive behaviour [21].

Childhood adversity and socio-economic factors surrounding children are also important environmental risk factors of concern to scholars. Adverse childhood experiences (ACEs) refer to traumatic events surrounding children before the age of 18 that can cause physical or psychological harm or threat, such as low socio-economic status, bereavement, adoption, etc. [22]. A follow-up study that included more than half a million subjects was conducted in order to analyse the relationship between adverse childhood experiences and ADHD symptoms. The results showed that individuals who had this experience had a five-fold increased risk of developing ADHD compared to individuals who did not experience adverse childhood experiences, and that the higher the number of experiences, the higher the risk [23]. Another systematic review included over forty studies on the association between low socioeconomic status and ADHD. Socioeconomic status can refer to parental income, education, marital status, etc [24]. The results showed that individuals in low SES families were at higher risk of ADHD compared to individuals in high SES families.

4. Pathology

4.1. Diagnostic Criteria

The diagnostic system for mental disorders has undergone several major updates and developments in the last decade or so. Currently, the most widely used diagnostic criteria in diagnostic studies of mental disorders are the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) published by the American Psychiatric Association and the 11th revision of the International Classification of Diseases (ICD-11) approved by the World Health Assembly [25]. In particular, there have been some changes in the classification assessment and diagnostic criteria for ADHD. In the following section, the changes in the DSM-5 and ICD-11 regarding ADHD and their similarities and differences are described.

The biggest change in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) from the previous fourth edition is the removal of the multi-axial classification system and the addition of a new chapter on neurodevelopmental disorders, with ADHD being included under this category [26]. Another large change was to increase the age of onset from 7 to 12 years of age. The reason for this change is that research has shown that there is no significant difference between individuals who develop ADHD symptoms before the age of 7 and those who develop symptoms later, and that individuals have less difficulty recalling early onset experiences [26]. In addition, the previous 18 symptoms have been retained, but specific examples of the interpretation of each symptom have been added to better suit the diagnosis of adults.

In ICD-10, ADHD is classified under the category of behavioural and emotional disorders that usually occur in childhood and adolescence, and hyperactivity disorder is excluded as a separate category from ADHD [26]. In the newly updated ICD-11, ADHD has been moved to the category of neurodevelopmental disorders and replaces hyperactivity disorder in ICD-10, using the descriptions of inattention, hyperactivity-impulsivity or a combination of these types.

Overall, the diagnostic requirements for ADHD) are broadly similar between the two diagnostic criteria, both of which include ADHD in the category of neurodevelopmental disorders, but differ in details, such as the number of symptoms that need to be met to give a diagnosis [25].

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4.2. Epidemiology

A search of the database reveals a large number of studies on the prevalence of ADHD from all regions of the world in recent decades, but with varying results [27]. Polanczyk et al. conducted a systematic review and meta-analysis study of the global prevalence of ADHD, including more than 170,000 subjects from all regions of the world [28]. The results showed that the global pooled prevalence of ADHD was approximately 5.29%. Another study reviewed the epidemiology of ADHD in three time periods between 1990 and 2010. The results found no significant differences in global prevalence between the three time periods and a global prevalence of approximately 2.2% [29]. For a long time, ADHD as a common mental disorder in the child and adolescent population, has caused adults to be neglected in epidemiological studies. In recent years, many researchers have begun to focus on the prevalence of ADHD in adultsA global systematic review and meta-analysis of the prevalence of ADHD in adults found that although symptoms resolved with age, the prevalence persisted from childhood-onset into adulthood at 2.58% and first onset in adulthood at 6.76% [30].

Researchers in epidemiological studies have also found the presence of some variability. A meta-analysis of ADHD prevalence showed that males were more likely to meet the diagnostic criteria for ADHD than females [31]. The results of another meta-analysis showed that the sex ratio for ADHD in childhood was ten to one, with males outnumbering females, and that although the ratio decreased somewhat to 2. 73 to 1 in adulthood, males still outnumbered females [32]. In addition to gender differences, some studies have noted differences in prevalence across ethnic groups. The results of a meta-study that included over 150,000 Black American participants showed that the prevalence of ADHD in Black people was 14.54%, a higher risk than in the general US population [33].

5. Ramifications

5.1. Consequences

The adverse outcomes associated with individuals with ADHD are also a hot topic of interest for researchers. A number of studies have shown that individuals with ADHD are at increased risk for other mental disorders as well as physical health risks. For example, Chen et al. conducted a cross-sectional study of the relationship between ADHD and other psychiatric disorders and common physical disorders, covering more than half a million adult subjects [34]. The results showed that adults with ADHD had a nine-fold higher risk of having bipolar disorder, depression and anxiety compared to normal individuals. In addition, these patients had an increased risk of co-occurring diabetes and hypertension. Another aspect that is closely related to ADHD is the patient's academic performance. For example, a longitudinal study investigating the impact of ADHD symptoms on academic performance found that early symptoms had a negative long-term impact on children's academic performance [35]. The same tendency has been found in adulthood. Results from a study exploring the academic performance of individuals with ADHD in a college student population showed that patients reported lower academic performance compared to normal individuals [36]. To make matters worse, some studies have found that people with ADHD are associated with suicidal thoughts and suicidal behaviour. For example, a follow-up study examined the association between early ADHD symptoms and suicidal ideation and attempts during adolescence. The results suggest that ADHD is a significant risk factor for suicide in adolescence [37].

Other studies have shown that ADHD symptoms can affect not only the person themselves, but also their family environment and even society. The results of a longitudinal study investigating the relationship between raising a child with ADHD and family burden showed that families with ADHD bear more than five times the financial burden compared to families raising normal children. Not only that, but parents of ADHD families report more stress, being laid off from work, and being less

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productive [38]. Several other studies have similarly shown that living with a child with ADHD is associated with significantly reduced life satisfaction and impaired emotional and social functioning for the mother [39,40]. To make matters worse, some researchers are concerned about the presence of crime in people with ADHD. The results of a longitudinal study investigating the long-term effects of early-onset ADHD on crime showed that individuals with ADHD were more likely to report criminal behaviour and were at an exponentially increased risk of engaging in criminal activity compared to normal individuals [41]. Another meta-analysis of 11 published studies on the relationship between ADHD and risk of the crime showed that people with early ADHD were significantly associated with arrests, convictions and incarceration during adolescence and adulthood and developed antisocial behaviour at a younger age compared to controls [42].

5.2. Treatment

Because ADHD is a common neurodevelopmental disorder with significant attendant effects and risks, the prevention and treatment of ADHD have also been a hot topic of interest for researchers. Psychostimulants (such as methylphenidate and amphetamines) and non-psychostimulant drugs (such as atomoxetine and 2-agonists) are currently the main pharmacological treatments for ADHD. For example, a meta-analysis of the effectiveness of more than one hundred treatments with methylphenidate for ADHD showed that methylphenidate significantly improved ADHD symptoms and was also associated with improved parent-reported quality of life [43]. Another study reviewed studies on the use of atomoxetine for the treatment of ADHD and showed that atomoxetine was effective in relieving ADHD symptoms compared to the placebo group, but the effect was weaker than the effect of long-acting psychostimulants [44].

Another major direction in the treatment of ADHD is the use of psychotherapy. Cairncross, & Miller conducted a systematic review and meta-analysis of 11 studies using Mindfulness-based therapies for ADHD and found that Mindfulness-based therapies significantly reduced symptoms of inattention as well as hyperactivity and impulsivity [45]. Another meta-study included 32 previously published studies using non-pharmacological treatments for ADHD and showed that different forms of cognitive behavioural therapy, such as group and individual, were associated with a reduction in core ADHD symptoms [46]. This is also supported by another meta-study, which suggests that age-specific cognitive behavioural therapy for people with ADHD can address functional deficits that cannot be achieved through drug therapy alone [47].

In addition to traditional medication and psychotherapy, researchers have explored a number of innovative treatment approaches. A systematic review of 52 studies that included dietary changes to improve ADHD symptoms suggested that eating small amounts of food and supplementing with fish oil supplements may have a beneficial effect on reducing ADHD symptoms [48]. The results of another meta-study on whether physical activity can reduce ADHD symptoms showed a positive effect on motor function deficits in people with ADHD through longer periods of physical activity [49]. Another innovative therapeutic approach is the use of reality-enhancing technology to implement a personalised treatment process, using games to immerse people with ADHD in the learning and adjustment problems of their life situations [50].

6. Conclusion

This paper attempts to categorise relevant research by reviewing studies related to ADHD in three areas, the three areas being etiology, pathology and consequences and treatment. Each aspect can be subdivided into subcategories, but these categories and subcategories do not exist in isolation, but are interrelated and interact with each other. Epidemiological research, for example, helps researchers to understand the development of ADHD and to adapt diagnostic methods or treatments accordingly.

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Another example is that the contribution of biogenetic and environmental factors to ADHD is inseparable and the combined effects of gene-environment interactions need to be considered [51].

Research on genetic risk factors has evolved from twin studies to single-gene studies to genome-wide studies. There is no doubt that genetic susceptibility influences the risk of ADHD, but it is unclear from current research how much of a role genetics plays [11]. On the one hand, The structural and functional abnormalities found in neuroimaging studies concerning ADHD need to be considered in relation to differences in results confounded by other factors, such as task type, sample characteristics, etc [12]. On the other hand, studies of brain structure, function and neuroimaging should take into account the dynamic development of the individual. Differences in the volume of brain structures as individuals develop may tend to decrease or cease to be significant in adulthood [52]. It should be noted that studies on the relationship between environmental risk factors and the risk of developing ADHD are more descriptive and rely on the reliability of questionnaires. Furthermore, because in many cases, researchers are unable to manipulate environmental variables, environmental risk factor studies make extensive use of correlational designs and therefore are unable to describe causal trajectories with disease. These areas should be of concern in future studies.

Regarding the diagnosis and epidemiology of ADHD, studies have shown that African-Americans and Latinos are less likely to be diagnosed with ADHD compared to Caucasians, which may be due to under-diagnosis and will affect prevalence studies and subsequent treatment [53]. Explanations for the inconsistent findings of ADHD prevalence studies, some suggesting that geographical location and year of study are not related to variability in ADHD prevalence estimates, should take into account differences in the methods and procedures of the studies. Some studies have shown an increase in the global prevalence of ADHD, but others have shown insignificant differences in prevalence over recent decades, and this issue needs to be further explored in future studies [27,29].

Studies have shown that research on both pharmacological and psychological therapies for ADHD has limitations. For example, the use of medication for ADHD can cause adverse effects such as anorexia and fatigue and insomnia, as well as the possibility of developing resistance and, in severe cases, substance abuse and dependence [54]. The difficulty in comparing and generalising the effectiveness of different studies using psychotherapy for ADHD is due in large part to the small sample size of such studies, the large variation between samples and the specific process of treatment used. A treatment that combines both approaches may be a good direction for future research.

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