From Genes to Behavior: A Comprehensive Review of ADHD and Dyslexia and Their Triggers Across Multiple Factors

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Abstract**:** ADHD and dyslexia frequently co-occur, suggesting they may share some neurobiological similarities. These neurodevelopmental disorders are shaped not only by inherited genes but also by environmental factors experienced before birth. Traditionally, ADHD and dyslexia have been studied by focusing on either genetic or environmental factors alone. However, recent research highlights a more complex interaction, showing that both genetic inheritance and external conditions influence the development and progression of these disorders. Studies indicate that both ADHD and dyslexia involve atypical development in brain regions responsible for executive functioning, attention, and language processing, often linked to changes in dopamine signaling. This overlap showcases the common genetic and environmental influences that may impact cognitive control.

Objective: The objective of this paper is to explore the interplay between genetic and environmental factors in ADHD and dyslexia, and to investigate potential overlaps in their underlying mechanisms. This paper emphasizes that ADHD arises from a complex interplay of inherited traits and external conditions.

Thesis: This paper addresses the gap in understanding how genetic factors and environmental exposures interact to shape both ADHD and dyslexia over time. By integrating findings from neuroimaging techniques, genetic analyses, epigenetic research, and assessments of environmental influences, this study aims to provide a comprehensive view of the causes and interconnections between these two neurodevelopmental disorders.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder that affects millions of individuals worldwide (Salari et al.,, *2023).* ADHD is characterized by inattention, impulsivity, and hyperactivity, which research suggests are linked to structural differences in key brain regions (Faraone et al., 2024). These areas include the prefrontal cortex, basal ganglia, and cerebellum, which play important roles in managing executive function and motor control (Arnsten,2009). Alongside ADHD is another recurring neurodevelopmental disorder known as Dyslexia(McGrath et al.,2019). Dyslexia affects reading and phonological processing, with disruptions observed in left hemisphere regions, particularly the occipito-temporal regions, which are critical for reading (Kim, 2021). Although ADHD and dyslexia are different in terms of their symptoms and the brain regions affected, researchers are investigating their potential genetic connections.

Which is why the purpose of this paper is to explore the neurobiological underpinnings of ADHD and dyslexia, examining genetic, epigenetic, and environmental factors that may contribute to both disorders. Specifically, this paper will address how certain genes, such as the dopamine receptor D4 (DRD4) and dopamine transporter gene (DAT1), are implicated in ADHD and dyslexia. It will further explore how environmental factors, like stress and nutrition, interact with these genes through epigenetic mechanisms, potentially influencing the progression of each disorder.

While there is limited evidence to show a direct overlap in genetic causes between the two disorders, some studies suggest similarities in brain structures of people with adhd and people with dyslexia(Liloia et al.,2022). For instance, functional imaging techniques, like functional magnetic resonance imaging (fMRI) and single photon emission computed tomography (SPECT), have revealed that in ADHD there is decreased activation in the prefrontal cortex (PFC) during tasks that require focus and self-control. This explains the attention and impulse-control challenges. Similarly, in dyslexia during reading tasks, fMRI analysis highlighted lower activity linked to reading difficulties (Amen et al., 2021) (Peyrin et al., 2011).

ADHD also shows a strong genetic component, with heritability linked to genes like the dopamine receptor D4 (DRD4) and dopamine transporter gene (DAT1)(Hasler et al.,2015). These genes are known to play a role in the development of ADHD and dyslexia. In addition, environmental factors also affect ADHD by modifying gene expression through epigenetics—factors like stress and nutrition influence genes without changing the DNA itself(Cecil et al., 2022). For example, genes like BDNF (important for brain adaptability) and FoxP2 (related to language and motor skills) are sensitive to environmental changes(Murray et al.,2011) Interestingly, these same genes, including DAT1, FoxP2, and DRD4, have also been associated with dyslexia, showing that both genetic and environmental factors may shape the outcomes of both ADHD and dyslexia (Hongyao *et al.,2023*; *Sanchez-Moran et al., 2018)*

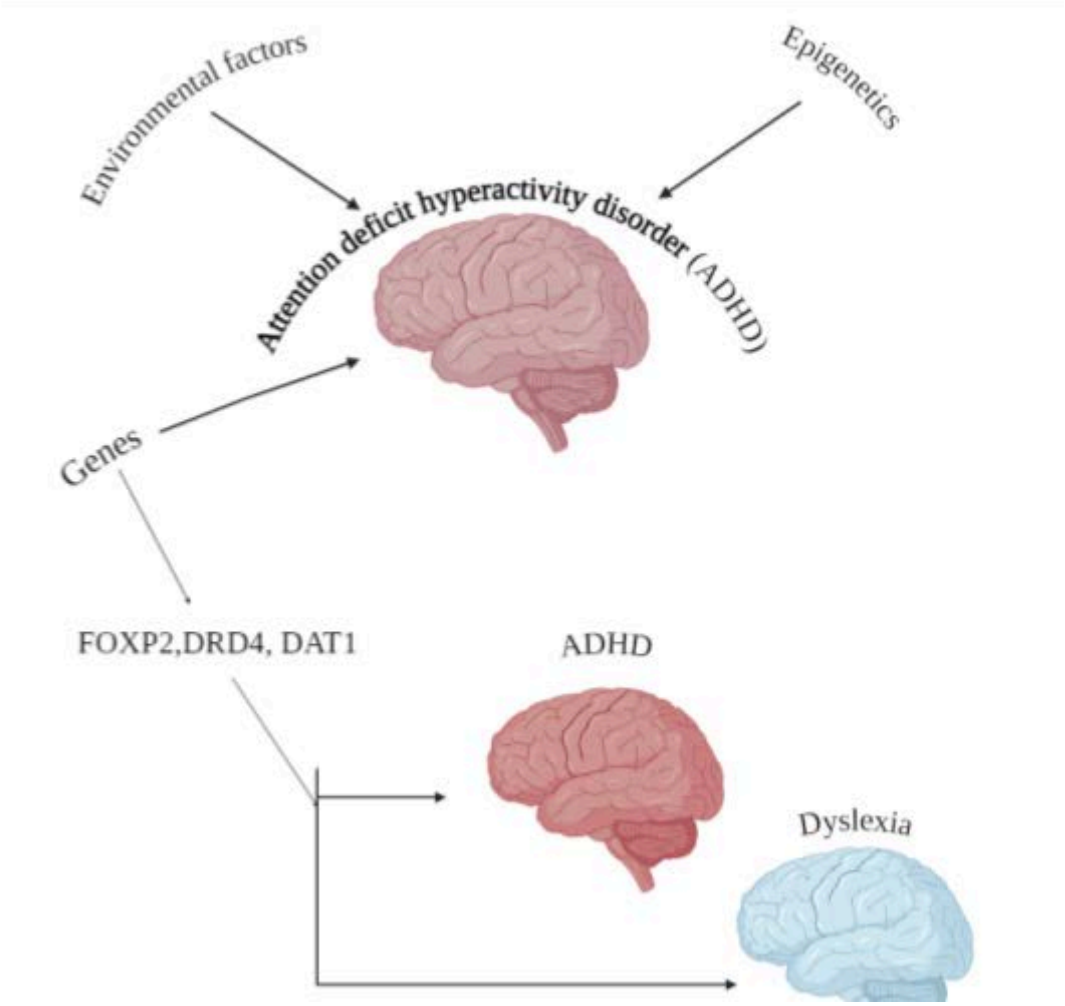

Figure 1: Factors Influencing Attention Deficit Hyperactivity Disorder (ADHD) and its Genetic Link To Dyslexia:

This diagram illustrates how environmental factors, epigenetics, and genes contribute to the development of ADHD. It also highlights a connection between ADHD and dyslexia, suggesting a shared genetic similarities.

Brain Regions Implicated in ADHD and Dyslexia

Although the human brain operates as an integrated whole, different regions within the two cerebral hemispheres specialize in processing specific cognitive information (Waldie et al., 2000). Disorders like ADHD and dyslexia are linked to brain regions, which contribute to the challenges associated with each condition. However, it is important to acknowledge that despite the interconnectedness of the brain, no strong evidence exists that suggests significant overlap

between the two conditions. This only leaves researchers open to speculate on possible areas of commonality (McGrath et al.,2019).

In both ADHD and dyslexia, abnormalities in brain morphology have been observed, although the affected regions differ in their functional roles. For ADHD, brain regions that are implicated in structural changes are found in key areas such as the basal ganglia, cerebellum, parietal cortex, prefrontal-cingulate cortex and parieto-temporal regions(Cubillo et al.,2011; Liloia et al.,2022). These regions are key to managing tasks such as motor control, and attention regulation. Accordingly, changes in these regions also align with symptoms like impulsivity, hyperactivity, and inattention. (Arnsten, 2006; Shaw et al.,2014; Stoodley, 2016; Bush 2011). Conversely, in dyslexia, structural abnormalities are noticed in areas such as the occipito-temporal regions, crucial for visual and phonological processing involved in reading. (Liloiaet al.,2022; Moore et al., 1999). Disruptions in these areas affect the ability to decode written language and process auditory-visual information, contributing to the reading difficulties that characterize dyslexia (Kim, 2021).

The Role of the Prefrontal Cortex in ADHD

One of the most crucial brain regions that affects ADHD is the prefrontal cortex (PFC). The prefrontal cortex is responsible for a set of cognitive abilities collectively referred to as executive functions. These functions include planning, decision-making, working memory, and impulse control (Jobson et al., 2021). Research suggests that dysfunction of the prefrontal cortex is widely regarded as the key factor in ADHD (Arnsten, 2010;Sullivan et al., 2023). By utilizing figures from (Amen et al., 2021) that employ fMRI, particularly SPECT scans—which visualize brain activity and blood flow—we gain valuable insights into the neural activity associated with ADHD.

In individuals with ADHD, shortages in executive functioning are prevalent, and many of these deficits can be traced to abnormal activity within the prefrontal cortex (Arnsten et al., 2012;Tabiee, et al.,2023;Schreiber et al.,2015). For example, reduced activation in this region has been associated with difficulties in sustaining attention and regulating impulsive behaviors, which are also symptoms of ADHD (See Figure 2 and Figure 3). They reveal significant differences in brain activity between individuals. Figure 2, demonstrates that a healthy brain shows increased activity in the prefrontal cortex (PFC) during tasks requiring concentration. In contrast, Figure 3, exhibits decreased PFC activity. This difference is attributed to lower levels of oxyhemoglobin concentration in the PFC among people with ADHD (Wu et al.,2023). This lower level of blood suggests that norepinephrine (NE) and dopamine (DA) may not function properly. When these neurotransmitters are imbalanced, reduced PFC activity can lead to struggles such as distractibility, disorganization, hyperactivity, and even procrastination. (Arnsten et al.,2023) and (Zhang et al., 2023).

Healthy Brain SPECT Scan

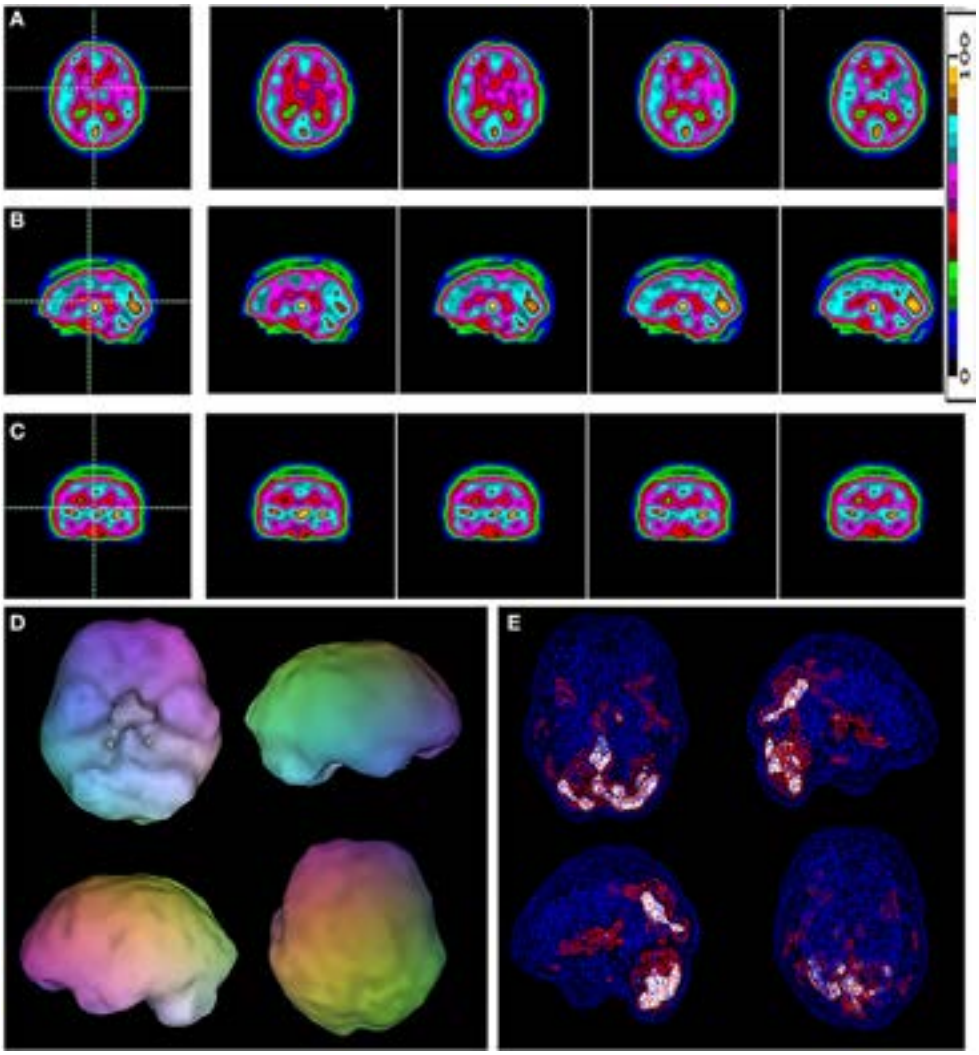

Figure 2: Normal SPECT Brain Scans of Control Group Without ADHD

Pictures(A), (B), and (C) display images of a healthy brain from various angles, with colours indicating different levels of brain activity. The colour shows how active each part of the brain is. Panel (D) provides a 3D view where areas with lower activity appear as dips or holes which essentially means that the brain has symmetrical blood flow/activity. Finally, picture(E) presents a wireframe of the brain, highlighting regions with high activity in red and very high activity in white (Amen et al., 2021). [Figure can be viewed at https://www.frontiersin.org/journals/psychiatry/articles/10.3389/fpsyt.2021.725788/full#F1]

ADHD Brain SPECT Scan

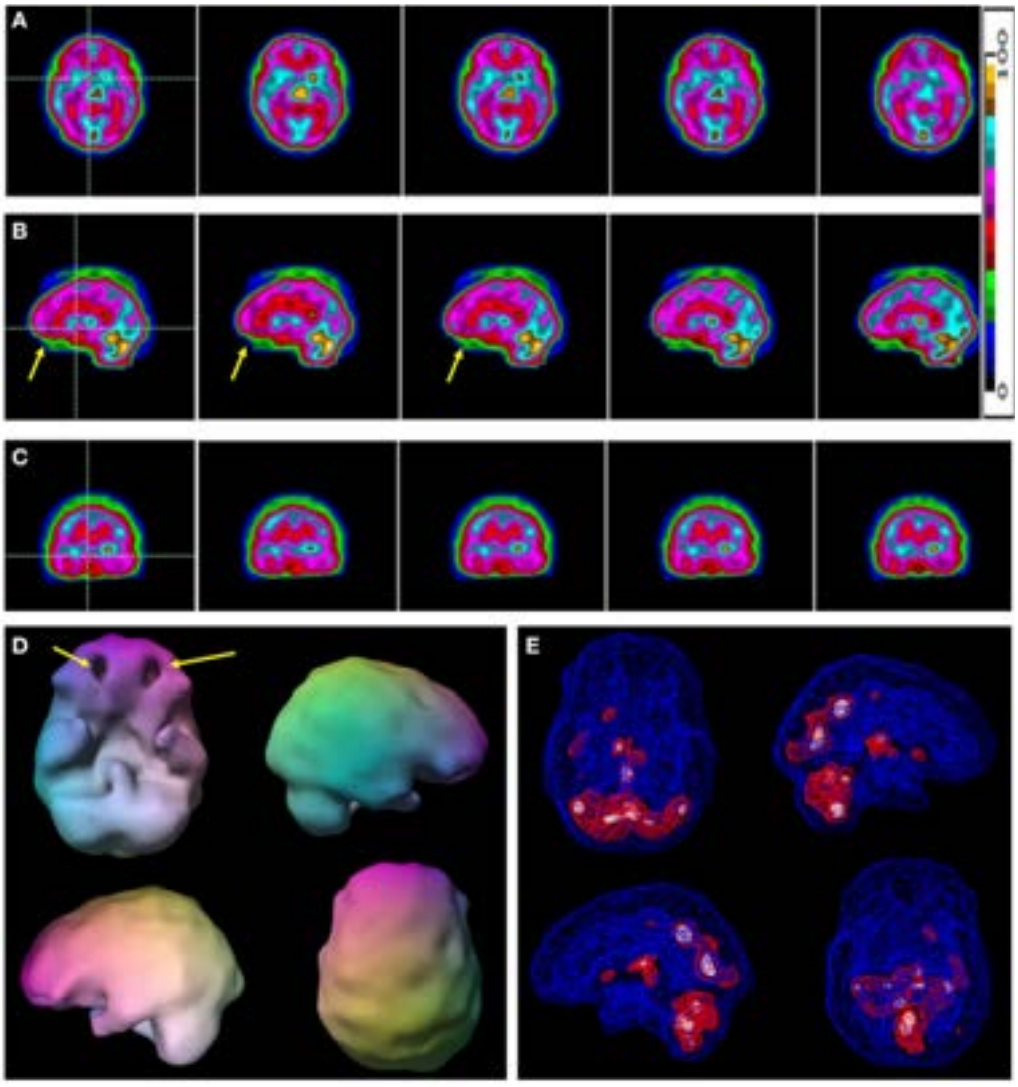

Figure 3: SPECT Brain Scans of Individuals with ADHD

Pictures(A), (B), and (C) present images of a brain from an individual with ADHD, using the same colour scale as in Figure 2. Yellow arrows are used to highlight regions in the front part of the brain that exhibit lower activity levels. Panel (D) provides a 3D view of the brain, where these yellow arrows indicate areas of reduced activity in the frontal region which once again means blood flow/activity in the prefrontal cortex. In the image here, the holes indicate areas of low blood flow. Panel (E) shows a wireframe of the brain, similar to Figure 2, but does not provide additional details about the specific activity levels (Amen et al., 2021). [Figure can be viewed at https://www.frontiersin.org/journals/psychiatry/articles/10.3389/fpsyt.2021.725788/full#F1]

Dyslexia and the Left Hemisphere

Recent studies, including those by (Munzer et al.,2020) and (Nora et al.,2021) highlighted the role of the left hemisphere in language processing and its functions in areas like phonological processing, word recognition, and reading. While dyslexia affects a variety of cognitive functions, disruptions in the left hemisphere have emerged as key contributors to the condition (Habib, 2021) and (Raschle et al., 2012). Technological advancements, including the use of fMRI and PET scans, have further allowed researchers to observe differences in brain functioning between people with dyslexia and those without dyslexia while processing written information (Habib, 2021). Findings from imaging studies by (Peyrin et al., 2011) reveal that, during reading tasks, non-dyslexic individuals show activation in left hemisphere regions involved in language, whereas dyslexic individuals exhibit reduced activation in these areas.

These findings showcase how disruption impacts the brain's ability to process the sounds of language, making it difficult for individuals with dyslexia to learn and to read fluently. Figures 4 and 5 illustrate these differences in brain activation within regions of the left hemisphere, providing a visual representation.

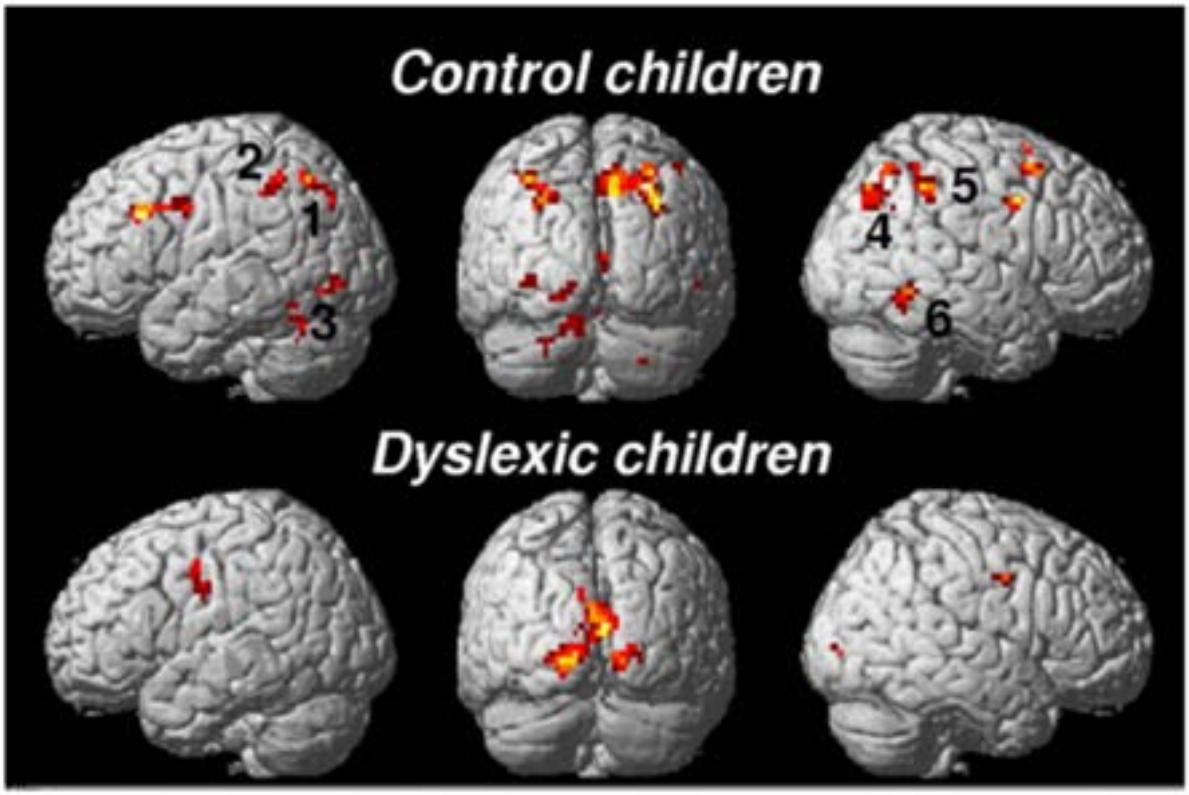

Figure 4: **fMRI brain scans of individuals with dyslexia and non-dyslexic children to highlight which brain regions are active during reading**

Labels 1-6 showcase brain activation patterns, with control children demonstrating stronger activation compared to those with dyslexia. Particularly images 1-3 illustrate significant activation in the right superior parietal lobule, and images 5-6 show strong activation in the left Visual Word Form Area (VWFA), both essential for word recognition. In contrast, dyslexic children exhibit reduced activation in these areas, indicating a disruption in connecting attention and visual processing during reading. This underactivation in the VWFA and superior parietal lobule suggests challenges in word recognition and processing efficiency for individuals with dyslexia (Peyrin et al., 2011). [Figure can be viewed at https://www.sciencedirect.com/science/article/pii/S0093934X10001264]

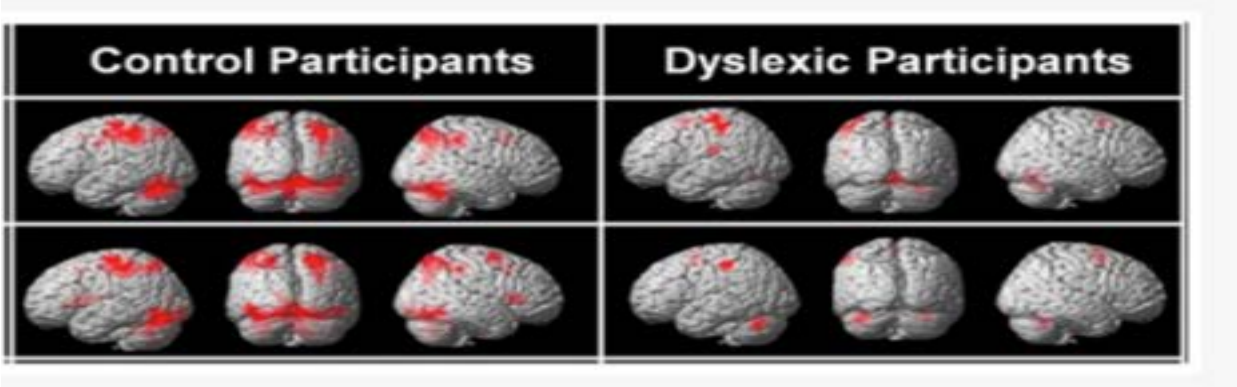

Figure 5: **Brain scans of individuals with dyslexia and non-dyslexic children to highlight which brain regions are active during reading**

Figure 5 further supports Figure 4, in which was used fMRI brain scans of both dyslexic and non-dyslexic children to highlight the brain regions that are active during reading– It showed that dyslexics exhibit lower activation in regions associated with reading, particularly in areas responsible for processing both alphanumeric and non-alphanumeric characters (Peyrin et al., 2011) . [Figure can be viewed at https://www.sciencedirect.com/science/article/pii/S0093934X10001264]

Summary of Figures 4 and 5

Figures 4 and 5 show clear differences in brain activity when dyslexic and non-dyslexic people read. In dyslexic individuals, certain areas of the brain that are linked to attention, particularly on

the right side, are less active. In contrast, non-dyslexic individuals show greater activity in areas on the left side of the brain that are responsible for recognizing written words. The left side of the brain is crucial for language and acts as the brain's language center. This suggests that dyslexic individuals experience a disconnect between the areas responsible for attention and those involved in visual processing (Peyrin et al., 2011) . These findings highlight the importance of the left hemisphere in reading and language processing, especially in tasks like phonological processing and word recognition. Research by (Richlan et al., 2009) corroborates these findings, demonstrating that underactivation in these areas hinders dyslexic individuals' ability to efficiently read and comprehend written text.

Moreover vast amount of neuroimaging studies have concluded that dyslexia is associated with underactivation in the left hemisphere's which are critical for skilled reading and visual word processing (Démonet, 2004; Grigorenko, 2001; Habib, 2000; Heim and Keil, 2004).

Genetic Inheritance of ADHD

ADHD is a complex condition that can significantly impact a person's life. One of the key ways researchers explore the causes of ADHD is through twin studies, which helps understand the role of genetics and environment in its development. According to (Grimm et al., 2020), one primary method for assessing the heritability of ADHD is through twin studies due to the possibility of providing insights into the genetic basis of it.

One twin study carried out by(Faraone et al., 2019) and (Tatay et al.,2019) compared monozygotic (MZ) twins, who share 100% of their genes, with dizygotic (DZ) twins, who share about 50%. This comparison allowed them to estimate how much ADHD is influenced by genetic factors compared to environmental factors. Their meta-analysis found that the heritability of ADHD ranges from 77% to 88%, indicating a strong genetic component. Similar results were seen in studies of full and half-siblings, with heritability estimates around 80%, further supporting the idea that genetic factors play a significant role in developing ADHD. This consistency in findings suggests that ADHD tends to run in families.

it is imperative to understand the difference between the terms “genetic” and “heritability.” While hereditary refers to conditions passed down from parents, genetics encompasses broader mutations or changes in genes that may not be inherited. For instance, cancer involves gene mutation but isn’t always inherited. Which is why ADHD is classified as both a genetic and hereditary condition because it involves inherited gene mutations. Both inherited and non-inherited factors contribute to the disorder, and their effects are interdependent (Thapar et al., 2012). So while ADHD shows a strong genetic link, this doesn’t automatically mean that a child will inherit ADHD.
In addition to genetics and herbility, other factors, such as a person’s environment, can also have some influence on how likely they are to develop ADHD (Livingstone et al, 2016). Research by (Thapar et al., 2012) and (Faraone et al., 2019) suggests that while each of these

factors has a relatively small individual impact, collectively, they contribute to the overall risk of inheriting ADHD. Environmental factors contributing to ADHD will be further discussed later in this paper.

Genetic Inheritance of Dyslexia

Studies show that dyslexia often runs in families, with children of dyslexic parents having a 40-60% higher risk of developing the condition themselves (Schumacher et al.,2007). Twin studies provide significant insights, particularly by comparing identical twins, who share nearly 100% of their DNA, with fraternal twins, who share about 50%. These studies reveal a high heritability for dyslexia, often estimated between 60% and 70% (Doust et al., 2020), indicating that genetic factors play a major role in its development.

Research has identified several genes associated with dyslexia, notably DCDC2, KIAA0319 (Marino et al.,2012; Gostic et al., 2019; Paniagua et al.,2022;Charish et al., 2023).These genes are involved in critical aspects of brain development and function, particularly in areas tied to language and reading. For instance, DCDC2 and KIAA0319 are linked to the development of the left temporoparietal cortex, which is essential for phonological processing—an area that tends to be less active in individuals with dyslexia (Darki et al.,2014). Additionally, researchers have identified genes associated with ADHD that are also linked to dyslexia, such as DAT1 and DRD4 (Hongyao *et al.,2023*; *Sanchez-Moran et al., 2018)*

DAT1 genes are linked to attention-related issues, often found in individuals with dyslexia, potentially impacting their focus on reading and language processing (Cornish, 2011). These deficits may intensify the reading challenges associated with dyslexia. Similarly, the *DRD4* gene, involved in dopamine signalling for attention and executive functioning, is connected to an increased risk of both dyslexia and ADHD(Kegel, 2012). Variants in *DRD4* may affect learning and memory retention, which are essential for word recognition and decoding—key difficulties in dyslexia(Scerri & Schulte-Körne, 2009).

However, dyslexia is not caused by a single gene but rather involves multiple genetic and environmental factors(Almahrag, 2021). Environmental influences, such as early literacy exposure can shape the extent of reading difficulties in individuals (Haughbrook, 2016).

For example, intensive reading intervention programs have shown that children with dyslexic traits can improve their reading skills significantly, highlighting the role of the environment in mitigating genetic risks.

Understanding dyslexia as both genetic and environmental is crucial. While genetics establish a foundation for dyslexia, environmental factors help determine its expression and severity.

Genetic Contributions to the Development of ADHD: A Look into the DRD4 and DAT1 Alleles.

The DRD4 and DAT1 genes play critical roles in the development of ADHD and dyslexia, two neurodevelopmental disorders often marked by overlapping symptoms such as attention

deficits. While ADHD and dyslexia share some genetic links, this section of the paper will focuses on ADHD, highlighting the DRD4 and DAT1 alleles—particularly the DRD4 7-repeat allele and the DAT1 10-repeat allele—and their impact on dopamine regulation in the brain(Austeja et al.,2024)

The 7-repeat allele in DRD4 has been associated with a greater risk of ADHD (Gilsbach et al., 2012) because dopamine receptors with the 7-R allele are less responsive to dopamine, contributing to the behavioral characteristics commonly seen in ADHD (Swanson, et al., 2000).

Similarly, individuals that possess the 10-repeat allele of DAT1 tend to have more dopamine transporter proteins present in their brains compared to those without this allele (Volkow, 2007). This increased availability can result in impulsivity and difficulty concentrating, symptoms usually observed in ADHD (Kim et al.,2006).

Genetics of ADHD: DRD4 and 7 Repeat Allele

One of the most extensively studied genes in relation to ADHD is the DRD4 gene, particularly its 7R allele. This allele plays a significant role in the manifestation of ADHD symptoms due to its location in PFC(Chen et al.,2021) (Swanson, 2000). Because of this, individuals with the 7R allele often experience reduced dopamine efficiency in the PFC, contributing to ADHD symptoms like impulsivity, inattention, and difficulty in focus.

Essentially, the presence of the 7R allele can alter receptor expression leading to a dysfunctional receptor that fails to respond quickly to dopamine (Ptáček et al.,2010), (Slifstein et al.,2015), (Rao et al., 2019). Furthermore, disrupted dopamine transmission can hinder the formation of neural connections within the PFC, this may delay the maturation of the PFC, further contributing to ADHD symptoms (Kolk et al., ,2021).

Genetics of ADHD: DAT 1 and 10 Repeat Allele

While much genetic evidence links DAT1 to ADHD, the strength of this association is not as strong as the DRD4 gene, largely due to inconsistencies in research. For instance, some studies by(Turic et al., 2010) and (Johansson, 2008) suggest that more investigation is needed to clarify the role of DAT1 in this disorder. However, other research (Pinto et al.,2018) (Luo et al., 2019) indicates a more consistent connection between DAT1 and ADHD.

Despite these inconsistencies, the DAT1 gene, particularly the 10-R allele, contributes to ADHD by affecting dopamine regulation in the brain. This allele leads to a less efficient dopamine transporter, resulting in altered dopamine levels in the PFC which is crucial for executive functions.

Moreover genetic association studies(Cornish et al., 2005), (Kirley et al., 2002) have shown a higher prevalence of the 10-R allele in individuals with ADHD compared to those who don't have, which demonstrates that genetic variations within individuals can contribute to the development of ADHD symptoms.

Epigenetics and ADHD

In addition to genetic factors, epigenetic mechanisms are increasingly recognized as important contributors to the development of ADHD (Schuch et al., 2015; Mirkovic et al., 2020). Epigenetics refers to changes in how genes are expressed without altering the actual DNA sequence. These changes can be influenced by environmental factors (Berger et al., 2009).

ADHD has both genetic and environmental influences that play critical roles in its development (Turic et al., 2010; Waldie et al., 2021). Much of the research has focused primarily on genetic factors, especially genes like DRD4 and DAT1, which are linked to dopamine regulation. However, focusing only on these genes does not capture the complexity of how ADHD develops. Recent studies have begun to explore other factors, such as FoxP2 (Forkhead Box Protein P2) and BDNF (Brain-Derived Neurotrophic Factor) . While FoxP2 is primarily associated with language development and is linked to dyslexia, it also influences cognitive and motor functions relevant to ADHD by affecting the growth of brain circuits involved in learning and behaviour (Chen et al., 2016).

Additionally, BDNF is a protein crucial for the growth, development, and survival of neurons. It supports neuroplasticity, which is the brain's ability to reorganize and form new connections—an essential process for learning and memory (Bathina & Das, 2015). BDNF also regulates communication between neurons, impacting cognitive processes and emotional regulation, which can be influenced by early life experiences (Marzola et al., 2023; Rauti et al., 2020).

Although there are many contributions to ADHD research suggests that altered BDNF levels may be linked to ADHD. Low levels of BDNF are associated with reduced neuroplasticity, which can impair learning, attention, and emotional regulation—key difficulties for individuals with ADHD. Since BDNF supports the development of neural circuits involved in attention and impulse control, disruptions in its expression could worsen ADHD symptoms (Corominas-Roso et al., 2013).

Similarly, dysregulation of FoxP2 may contribute to language delays, poor motor coordination, and difficulties with attention and impulse control, which are often observed in ADHD (Haghighatfard et al., 2022; Dark et al., 2015).

Epigenetic Modifications and ADHD

One primary way that gene activity is regulated is through a process called DNA methylation. This involves adding methyl groups to DNA, which turns down the activity of certain genes (Moore et al., 2013). In the context of ADHD, epigenetic modifications can affect how genes related to brain development and neurotransmitter regulation are expressed.

Various factors, such as stress, environment, and diet, can change DNA methylation patterns, which might influence the expression of genes involved in dopamine signaling(Allison et al., 2021). Dopamine is an important neurotransmitter for regulating attention and behavior. For example, research has shown that children who were exposed to high levels of stress from their mothers during pregnancy may have changes in their DNA methylation (Alexandra et al.,2021) These changes can affect genes involved in dopamine pathways, possibly leading to altered

dopamine levels and impacting attention, impulse control, and other behaviors associated with ADHD (Allison et al., 2021).

Further research (Neumann et al., 2020) has shown that DNA methylation patterns established at birth can affect brain development pathways related to ADHD. For instance, changes in methylation at specific genes linked to brain connectivity and dopamine function may result from environmental influences experienced during pregnancy or early life, such as exposure to toxins. These early epigenetic changes could make children more likely to develop ADHD by impacting how their brains develop and function.

Environmental Factors and ADHD

While a lot of research on ADHD focuses on genetic predisposition, environmental factors also play a role in the development of ADHD. Particularly through epigenetic mechanisms that influence gene expression without altering the DNA sequence—a process known as *epigenetics* (Walton et al., 2016). For example, factors such as exposure to toxins during pregnancy, high stress in mothers, and poor nutrition can create epigenetic changes that may increase the risk of ADHD (Doi et al., 2022). Remarkably, some of these epigenetic changes may even be passed down to future generations (Stenz et al., 2018).

One example is how maternal stress can alter DNA in ways that affect dopamine—a key chemical in the brain linked to attention and impulse control, both of which are crucial in ADHD symptoms (Allison et al., 2021). Additionally, maternal smoking during pregnancy has been linked to DNA changes in the child, potentially increasing their risk of ADHD (Ke et al., 2021). Some studies suggest that children with certain genetic variants, like DAT1 or DRD4, are more affected by these environmental factors, such as maternal smoking, and may have a higher risk of ADHD as a result (Neuman et al., 2007). Overall, these findings show that both genetics and environment work together in complex ways to influence ADHD, making it difficult to pinpoint a single cause of the disorder.

Conclusion

Altogether, these findings highlight that ADHD is not caused by genetics alone but by a complex mix of inherited traits and environmental factors. Genes like DRD4 and DAT1, which influence how dopamine is processed in the brain, play a major role in attention control and impulse regulation—both of which are key aspects of ADHD.DRD4 affects dopamine receptors in the prefrontal cortex, which is critical for focus, while DAT1 helps control dopamine reabsorption into neurons. However, these genetic effects can be shaped by environmental factors—such as prenatal stress, maternal behaviors, and nutrition—which alter gene expression and impact ADHD symptoms. Studies of the brain also show that people with ADHD tend to have lower activity in the prefrontal cortex, which is essential for attention and self-control.

While ADHD and dyslexia are distinct conditions—dyslexia primarily impacts language processing in the left side of the brain—they share common genetic and environmental

influences. For example, FoxP2, a gene associated with language development, has been linked to dyslexia and is also implicated in cognitive processes that overlap with ADHD, such as attention and impulse control. This connection illustrates the importance of understanding how these genes interact in the context of both disorders.

Epigenetics, or changes in gene expression due to environmental influences, plays a significant role in ADHD. For instance, prenatal stress can lead to modifications in DNA that affect dopamine pathways essential for attention and impulse control.

Overall, dyslexia has genetic roots that interact with genes such as DRD4, DAT1, and FoxP2, which also contribute to ADHD. Despite the distinct ways in which ADHD and dyslexia manifest in the brain, further research could explore shared cognitive processes, such as attention and memory, which impact both disorders.

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