A cognitive neuroscience review of the aetiology of ADHD

Imagine having your MacBook open, you have 103 tabs open in Safari and another 79 open in Firefox, but you have no idea why you opened them, what's the importance of each page, then you become frustrated because your MacBook is running slow. Jeremy Kyle is on in the background and you're waiting to find out who stole that purse. Also, your phone is beside you with IGTV (Instagram television) playing video after video and you're keen to find out what happened in last night's Love Island. But then you have a new idea and you can't resist opening a new tab to google about it. This analogy can describe how people with attention deficit hyperactivity disorder (ADHD) feel, they have so many images, sounds and ideas running through their minds they don't know which to focus on first and which is the most important. It seems individuals with ADHD are living in a whirlwind of disorganised, frenzied situations jumping from one activity to the next without finishing any of them. In my career as a teacher, I have taught many pupils who have a diagnosis of ADHD, each pupil is different and have developed different coping strategies for their disorder. However, the majority of pupils are still tainted by a stigma that they are the disruptive pupil who lack focus and never sit still. As with all stigmas, understanding and education about the disorder is paramount to counteract ADHD and this should begin with understanding.

Introduction

ADHD is the most common neurodevelopment disorder of childhood and has a strong persistence throughout the individual's lifespan. ADHD is characterised by age-inappropriate behaviour is thought to affect between 3% and 5% of all school-aged children (Buitelaar, 2002) age-inappropriate behaviour typically characterises it.

ADHD is a lifelong disorder which can increase morbidity, including impaired academic results; unable to maintain a job successfully; relationship failures; increased rates of substance abuse; persistent neuropsychological impairments (Biederman, 2004; Vos et al., 2005). Therefore, it is clear how vital determining the neural substrate of ADHD is.

People with ADHD display a persistent pattern of inattention, impulsivity and hyperactivity that interferes with functioning or development (Buitelaar, 2002). Inattention in this instance refers to the inability to direct and maintain attention to relevant tasks. Impulsivity refers to acting without thought of the consequences, such as shouting out answers in class. Hyperactivity refers to excessive motor activity.

Affected areas of the brain in ADHD

Looking at data from neuroimaging, neuropsychological, and neurochemical studies, they have mostly indicated frontostriatal differences which contribute to ADHD (Bush et al., 2005; Durston et al., 2003; Vaidya and Stollstorff, 2008).

The frontostriatal is a neural pathway connecting the frontal lobe region to the basal ganglia; these areas are often impaired in people with ADHD (Cherkasova & Hechtman, 2009). Cognitive skills controlled by this area include decision making, memory and attention.

Neuroimaging studies can help to identify the pathophysiology of ADHD, as it searches for abnormalities of brain regions that are usually involved in the symptoms of ADHD; attention,

cognition, executive function, motor control, response inhibition, working memory, and motivation.

In a positron emission tomography (PET) study, a tracer (glucose) is emitted into the body, and then an imaging technique is used to observe metabolic processes in the body. Results found in adults with ADHD glucose metabolism was reduced significantly compared to the control groups values. In 30 out of 60 regions of the brain, the most significant reduction was found in the premotor cortex, which are associated with motor activity and controlling attention (Zametkin et al., 1990).

Imaging studies on people with ADHD show altered patterns of activity in the dorsal anterior midcingulate cortex (daMCC), dorsolateral prefrontal (DLPFC) and ventrolateral prefrontal cortices (VLPFC), parietal, and cerebellar regions. These areas have been identified as regions associated with attention and cognition (Nigg and Casey, 2005: Bush, 2011).

Volumetric abnormalities were found in the basal ganglia which comprise the caudate (Stark et al., 2011), primarily responsible for motor control. Results show a decrease in caudate volume using MRI in those with ADHD compared to the control group (Castellanos et al., 2008), in contrast, other research found no volume differences in people with ADHD (Hill et al., 2003). The striatum nuclei in the subcortical basal ganglia (that facilitates voluntary movement, Schultz, 2006), the dorsal striatum consists of the putamen, caudate and nucleus accumbens. It is frequently reported that the putamen and caudate are larger in people with ADHD compared to those without (Valera et al., 2007). If this area is linked to voluntary movement and if there is damage to this area, it's clear to see why people with ADHD have hyperactivity.

Many PET and fMRI (functional magnetic resonance imaging; measures brain activity by detecting changes associated with blood flow) studies have reported hypofunction in the daMCC in people with ADHD using a variety of cognitive tasks and techniques (Zametkin et al., 1990; Bush et al., 1999). Such dysfunction in these areas could lead to inattention as the target cannot be detected, it may lead to hyperactivity by not reducing motor activity that is not in line with the individual's target, or by failing to modify the behaviour by using reward and error feedback. Impulsivity could be as a result of damage to the area which encodes information insufficiently, resulting in displaying behaviour characterised by little or no forethought, reflection, or consideration of the consequences.

Moving out of the forebrain and frontal lobe of the brain, the parietal cortex that has key roles in processing sensory information and attention allocation (Culham, 2002) is enlarged in individuals with ADHD (Bush, 2009). Moving down to the hindbrain, (responsible for motor coordination) findings using magnetic resonance imaging (MRI) have reported smaller cerebellum volumes in people with ADHD (Bledsoe et al., 2009).

In this brief review, there has been a focus on imaging studies. There are many other methods to measure brain activity with ADHD such as neuropsychological, genetics, and neurochemical studies. It must be noted that neuroimaging studies are still controversial, they are expensive, so sample sizes tend to be small. Neuroimaging studies have been criticised for being oversimplified, highly unconstrained and atheoretical (Lee and Cohen, 2003). Nevertheless, brain imaging techniques are likely to be an important force in cognitive neuroscience for the foreseeable future.

The affected areas of the brain and the skills associated give an insight into the behaviour that is manifested by people with ADHD, and these skill impairments contribute to the three key ADHD symptoms. When cogitating the aetiology of the disorder, the intricacy of ADHD needs to be understood, and the complex interplay of different risk factors need to be considered.

Future studies should focus on a deeper understanding of these brain regions, and continuous technological advancements will improve imaging techniques to help with a more increased understanding.

Brain Chemistry

In addition to functional and structural abnormalities in the brain, neurotransmitters in the brain are considered to contribute to the symptoms of ADHD. The neurotransmitter, dopamine is responsible for feelings of pleasure and reward, it helps regulate emotional responses and takes actions to achieve specific rewards. Neurotransmitters surround the frontostriatal pathway, so changes in the levels of neurotransmitters explains why there is an effect on prefrontal function. Research has shown lower levels of dopamine are linked to symptoms of ADHD (Swanson et al., 2007).

Dopamine contributes to the functioning of the prefrontal cortex and basal ganglia. Therefore, minimal amounts of dopamine in these areas restrict inhibition movement; producing hyperactivity; reducing impulse control which will result in deficits in working memory (Spencer et al., 2005). Dopamine deficiency impairs the functioning of the basal ganglia, resulting in hyperactivity in people with ADHD (Curatolo et al., 2010). A recent study in adults with ADHD shows lower dopamine levels in the caudate, hippocampus and amygdala, which is associated with inattention (Volkow et al., 2009).

Neurons in the brain and nervous system have higher concentrations of proteins called dopamine transporters, these prevent dopamine from moving onto the next cell, reducing the effect of dopamine.

The concentration of these proteins is known as dopamine transporter density (DTD). There was a 70% increase in DTD in adults with ADHD compared to typical controls (Dougherty, 1999). This was supported Campo et al., (2011), that also found people with ADHD have an increased DTD in cortical areas meaning that dopamine levels were not at optimal levels. However, the researchers argue the use of participants who take no medication compared to participants using stimulants to control ADHD symptoms, make it difficult to establish cause and effect (del Campo et al. 2011).

Other research shows lower levels of dopamine transporters in the left brain in participants that had ADHD (Volkow et al., 2007). Results in this area are conflicting, it's difficult to establish if higher levels of DTD and lower levels of dopamine indicate ADHD. However, research is showing an association suggesting that dopamine could be a possible treatment for ADHD. Further studies need to investigate dopamine's role in ADHD before any firm conclusion can be made.

Grey Matter

Studies of cortical or grey matter thickness using MRI have approximately 3-4% thinning of the cortex in all four lobes in people with ADHD (Castellanos et al. 2002). Shaw et al. (2007) conducted a study on the developmental trajectory of cortical maturation. The results showed children with ADHD had a three-year delay in attaining peak thickness in the cerebrum and regions controlling attention affected, (ADHD reached at 10.5 years, while control group reached peak cortical thickness at 7.5 years). The prefrontal cortex functions are associated with suppressed thoughts, executive control of attention, high order motor control and working memory. This suggests ADHD is characterised by a delay in cortical maturation rather than deviance. However, Vaidya and Stollstorff (2008) argue the increase in grey matter may be due to areas have to compensate for reduced prefrontal activity in cognitive tasks. Research has supported this, a meta-analysis of 55 fMRI studies concluded areas have to compensate for the functional deficiencies (Cortese et al., 2002). There is little other research that supports this theory of delay in cortical maturation in ADHD. However, trajectories of brain development based on neuroanatomic data is providing an understanding of ADHD, which may guide future research.

Genetics and the Environment

The abnormalities in the function and structure of the brain are complemented by genetic studies, which demonstrate the inheritability of ADHD's pathogenesis, focusing of gene abnormalities including the dopamine system (Poelmans et al., 2011). Research found 40% of children with ADHD have a parent with the disorder (Faraone et al., 2000; Chen et al., 2008). Also, twin studies show there is a high chance of inheriting ADHD (Faraone et al., 2005). Adoption studies show children have more similarities to their biological relatives than their adoptive relatives (Faraone et al., 2005).

The nature versus nurture debate is often studied when investigating the cause of ADHD. Research shows there is a complex interplay of genes and the environment. Faraone et al. (2000) argues that twin studies have shown genes cannot be the sole cause of ADHD, as the rate would be 100%, so environmental factors must be an attributable factor. But, there is little research conducted on causal environmental factors (Thapar, 2009).

Conclusion

People with ADHD have a debilitating disorder, that can lead to social, academic and mental health problems. The whole debate of ADHD is mired in adversity; from conceptualisations, treatment, assessment to comorbidities. Since ADHD is a heterogeneous condition, a simple neurobiological basis is not possible and research has been unable to identify a distinctive aetiology. The majority of research uses neuroimaging, but this technique has questionable probative value. Advancements in neuroimaging techniques will give an enriched understanding of regions of the brain associated with ADHD. The cardinal symptoms of ADHD are not unique to the disorder, and there is strong comorbidity with other mental health conditions. Although much research has been conducted, there is still a lack of clarity to the cause and the variation of symptoms. Behavioural manifestations of ADHD are thought to be produced by dysfunctions in brain circuits, which are attributed to cognitive functioning. The research discussed shows there are multiple abnormalities in circuitry, chemistry and structure. Dopamine levels has been identified as a crucial factor in the disorder, methodological issues in the research still cloud the findings of the studies. Research findings into ADHD being a delay rather than deviance gives a new understanding

of the cause of ADHD and improvements in imaging techniques are a stepping stone to getting closer to finding the aetiology of the disorder.

Looking Forward

A simple neurological explanation has yet to identify an aetiology and pathogenesis of the disorder. However, advancements in imaging techniques should help to give a more detailed understanding of the brain regions that are different to those without ADHD. It is paramount that further studies are carried out so that a clear cause of the disorder is established. It is only with understanding of the disorder can the stigma be counteracted and individuals with ADHD be free from judgement.

The management of ADHD requires multiple professionals working together. A multi-method approach is the most effective way for managing ADHD, including stimulants and behavioural and educational strategies.

It is vital that individuals with ADHD are given the help they deserve from an early age to help them achieve their potential and live an enriched life. Teachers, parents, family members, and healthcare professionals play an important role in supporting and meeting the needs of the individual affected.

Multiple services have failed to recognise the importance of ADHD. Government and local educational authorities must now implement successful inclusion strategies. Untreated and undiagnosed ADHD creates a childhood full of self-doubt and challenges. But, it can be treated effectively, and early diagnosis with successful management can present a positive future.

With constant advancements in technology there will always be more to learn about ADHD; however, this should not prevent existing knowledge being applied. This should help eliminate the misconceptions and misinformation that are generally associated with ADHD.

Currently, research shows teachers in the educational system are not provided with enough help and support to provide a successful inclusive classroom, with intervention programmes producing contrasting results. Future research should establish relevant intervention strategies which can be used to make a successful inclusive classroom, this will support inclusive well-being of everyone. If this is conducted efficiently, pupils with ADHD can have a positive outlook with a thriving future.

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