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# What do ADHD Neuroimaging Studies reveal for Teachers, Teacher Educators and Inclusive Education?

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## Abstract

### *Background:*

*Ongoing debate about Attention Deficit Hyperactivity Disorder (ADHD) has not resolved ambivalent teacher beliefs about ADHD. This is an important matter since teachers' beliefs influence their pedagogy, classroom management, and their referral procedures for formal diagnoses of ADHD. They therefore must be provided with up-to-date professional learning about ADHD.*

### *Objective:*

*To synthesise neuroimaging studies, which examined differences in brain organisation and function in those with ADHD compared to matched unaffected controls. The overarching goal was to enhance teachers' understanding of ADHD by providing synthesised research findings around the neurological basis of ADHD.*

### *Methods:*

*The PRISMA method was used to search the Medline, PsycINFO, Web of Science and Scopus databases to complete a systematic review of peer-reviewed research that compared individuals with ADHD with matched controls published between 2010 and December 2015.*

### *Results:*

*The identification and analyses of 174 MRI and fMRI relevant studies across a sample of over 24,000 showed that there are significant differences in neural anatomy and processing in ADHD compared to unaffected matched controls.*

*Conclusions:*

*Compelling evidence shows ADHD is a neurodevelopmental disability, not a socially determined set of behaviours. Results point to an urgent need for teacher professional learning and systematic up-to-date preservice teacher education along with inclusive education policy reform.*

**Key words: ADHD, neuroimaging, preservice teacher education, MRI, fMRI, professional learning**

## **Introduction**

Attention deficit/hyperactivity disorder (ADHD) is one of the most prevalent neurodevelopmental disorders, with a worldwide prevalence of 7.2% (Thomas, Sanders, Doust, Beller, & Glaziou, 2015). Additionally 50–80% of identified cases persist into adulthood, resulting in an adult prevalence of ADHD of 3–4% (Fayyad et al. 2017; Frodl & Skokauskas, 2012). Rates of adult prevalence of ADHD also suggest that not all children with ADHD are identified while at school. Recent community cohort studies have suggested that there may be cases of ‘adult onset’ ADHD among individuals without a prior history of ADHD in childhood (Agnew-Blais et al. 2016; Caye et al. 2016). According to the Diagnostic and Statistical Manual of Mental Disorders 5th edition [DSM–V; American Psychiatric Association (APA), 2013], ADHD manifests as three possible subtypes: a combined attention deficit and hyperactivity type, a predominantly inattentive type, or a predominantly hyperactive-impulsive type with symptoms typical of either inattention or hyperactivity-impulsivity or a combination of both. In addition to

core symptoms of chronic inattention, hyperactivity, and impulsivity, ADHD is commonly associated with significant impairments in social, academic, and executive functioning, atypical motivational processes and depression, aggressiveness, and anxiety during adolescence. ADHD symptoms can change with age: the hyperactive and impulsive symptoms of childhood decrease, while inattentive symptoms persist across all subtypes (Biederman, Mick & Faraone, 2000).

That said, how ADHD is understood by teachers, teacher educators and policy makers is critical because it informs teacher education and practice, as well as inclusive educational policy, all of which influence the school support offered to children with ADHD. Currently, additional school supports by way of teacher aide time, an Individual Education Plan and accommodations may not be granted for children diagnosed with ADHD in Australia unless they have other impairments or additional disabilities such as Autism Spectrum Disorder (ASD) which often presents with ADHD (Taurines et al., 2012). In Australia, this is because ADHD is not classed as one of six disability categories requiring verification for an Education Adjustment Program and teacher aide support in public schools. These are ASD, hearing impairment, intellectual disability, physical impairment, speech–language impairment, or vision impairment. Non-state school sectors have processes for identifying students with disability in the categories of ASD, Intellectual Impairment and Speech-Language Impairment. Moreover, Australian inclusive educational policy has not changed as a result of the listing of ADHD under ‘Neurodevelopmental Disorders’ in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-V) (American Psychiatric Association, 2013), nor in light of Australian medication rates for ADHD which range from 13.0% to 28.6% per 100,000 youths (Pharmaceutical Benefits Scheme, 2015).

Notwithstanding, support for students with ADHD is critical because it is more prevalent in the context of poverty (Boat & Wu, 2015), increasing the risks for intergenerational family disadvantage since it predicts academic underachievement and societal costs (NICE, 2008) resulting from unemployment and/or problems with the law (Young et al., 2010). Despite these well-known correlates of ADHD, it continues to divide public and professional opinion. There is a widespread belief that ADHD diagnosis is subjective and misconceptions about ADHD persist, resulting in regular parental reports of stigmatizing experiences (Lebowitz, 2016; Moldavsky & Sayal, 2013). Australian parents in particular have reported that teachers have inadequate understanding of ADHD and are under-prepared to address their children's needs (Efron, Sciberras & Hassell, 2008).

Negative teacher views and limited knowledge of ADHD is widespread (Shroff, Hardikar-Sawant & Prabhudesai, 2017; Khademi, et al., 2016; Anderson, Watt, Noble & Shanley, 2012) with ambivalent beliefs, emotions and behaviours in Australia linked to teacher and preservice teacher knowledge of ADHD (Anderson, Watt & Shanley, 2017). A literature review of teachers' perceptions of ADHD showed they were inconsistent, founded on inaccurate knowledge and representing deep cultural values associated with family discipline and upbringing (Frigerio, Montali & Marzocchi, 2014). Preservice teachers also expressed these views, while their limited understanding of ADHD lead to anxiety and anger during practicums when dealing with the management of students with ADHD (Kikas & Timoštšuk, 2016).

ADHD is widely regarded as problematic social behaviour (Wright, 2012). This is because ADHD is considered to be ill-defined, not convincingly diagnosed, a condition better understood through a sociological lens, a view publicly endorsed, for example, by eminent psychiatrist Bruce Perry who declared ADHD was not "a real disease" (Boffey, 2014).

Prevailing societal and collegial opinions can influence teacher beliefs (Murik, Shaddock, Spinks, Zilber & Curry, 2005). In turn, beliefs affect teacher pedagogy of students with ADHD (Ojionuka, 2016), and their classroom management (Blotnicky-Gallant, Martin, McGonnell, & Corkum, 2015; Coles, Owens, Serrano, Slavec, & Evans, 2015) which impacts upon student outcomes (Jimerson & Haddock, 2015; Hattie, 2009). Teachers' management of classroom behaviours and their beliefs about acceptable interventions not only affect student performance they also influence the social acceptance of those with ADHD (Sherman, Rasmussen & Baydala, 2008).

Besides the management of children with ADHD, teachers also influence the diagnostic rates of children with ADHD, because their interpretation of classroom behaviours can lead to medical referrals for a diagnosis for ADHD. It is clear then that negative or uninformed views of ADHD are problematic. Particularly so because ADHD is more prevalent among those living with poverty, perhaps not possessing the knowledge and capacity to be effective agents for their children's education (Houtrow & Okumura, 2011). Teachers must base their beliefs and expectations of students with ADHD on accurate knowledge, which teacher educators must facilitate (Anderson et al, 2017; Blotnicky-Gallant, et al., 2015).

In sum, the overarching goal of this review is to provide neuroimaging study findings in clear, non-technical language so teachers, teacher educators and policy makers can better understand ADHD. Consequently, this might lead to improved educational access and support for students with ADHD through changes in teachers' beliefs and pedagogy, and educational policy and funding.

## **Aims**

This review examined findings from recent neuroimaging studies on the biological basis of ADHD for the purpose of enhancing teacher educators', teachers' and policy makers'

knowledge and understanding of the condition. This is because educators worldwide are increasingly drawing on neuroscience to inform policy and pedagogy. In Australia that was evidenced by the new Science and Learning Centre at the University of Queensland which draws on neuroscience to understand learning processes; a number of other higher education institutions also include courses on neuroscience to inform teaching and learning.

## **Method**

### **Search strategy and inclusion criteria**

A systematic literature review was conducted during December 2015 using the Medline, PsycINFO, Web of Science, and Scopus databases because they were deemed to contain the most relevant neurological studies. The search terms aimed to identify studies using neuroimaging techniques to compare the characteristics of typically developing individuals with those with ADHD across all ages. Studies based on adult samples were included to clarify whether ADHD might result from developmental delay.

The search terms were: Neuroimaging OR MRI OR fMRI OR EEG OR ERP OR MEG OR PET OR SPECT OR NIRS OR MRS OR CT in the Title/ Abstract/ Keywords AND ADHD OR attention deficit OR hyperactivity in the Title/Abstract/keywords. The search included studies published since 2010 and yielded 3,465 unique citations. All studies were screened against the eligibility criteria by 2 independent reviewers, the author and a research fellow with a doctorate in neuroscience. Inclusion criteria stipulated the study must (i) use neuroimaging techniques; (ii) directly compare a group of ADHD individuals with matched controls or be a meta-analysis or review of such studies and (iii) be written in English. If the title and abstract information were insufficient to determine whether the relevance criteria were satisfied the article was included through the first filter, resulting in 504 articles. Of those, 429 articles were accessible via open

access or the university's journal subscription database. Further inspection excluded 51 studies which did not include a control group, leaving 417 studies. Of these 65 MRI (magnetic resonance) and 109 fMRI (functional magnetic resonance) imaging studies were sequestered for review. Details of the search process are in Figure 1.

The reasons for using MRI and fMRI studies are based on the type of information they provide. Neuroimaging techniques can image structure (structural techniques), or directly or indirectly, measure neural activity as a function of time (functional techniques). Structural techniques like MRI scans can identify trauma to the brain, or tumours, and they can be used to compare the size of different regions within the brain. Therefore, for example, patients with various kinds of dementia will often show atrophy in certain regions of the brain; at a group level people with various psychiatric conditions can have differences in brain anatomy compared to controls. Again, at a group level, these differences can be shown to be related to specific abilities; for example, musicians compared to others have differences in the motor, auditory, and visual-spatial regions of their brains.

Functional neuroimaging techniques can be divided into two groups. The first group has good temporal but poor spatial resolution. This group comprises EEGs (electroencephalograms), ERPs (event-related potentials), MEGs (magneto-encephalograms), and EROS (event-related optical signals). The second group has good spatial resolution but poor temporal resolution and includes fMRI. The distinction between techniques with poor temporal resolution and those with good temporal resolution relates to how directly they measure neural activity. Techniques, which measure neural activity more directly, have good temporal resolution, while techniques that measure neural activity indirectly by imaging cerebral blood flow or glucose metabolism have poor temporal resolution.



This review focuses on 65 MRI and 109 fMRI studies and their findings as they relate to differences between those with ADHD and unaffected control individuals.

**Compliance with *Ethical Standards* was met.** In the search strategies, analyses and retention of collected papers there was no conflict of interest; the research did not involve human participants and/or animals and therefore there was no requirement for informed consent to be obtained.

Insert Figure 1 here

## **Results**

MRI results are presented according to key brain regions studied; fMRI studies are summarized according to the function assessed and by the experimental design employed.

### **Structural MRI: Anatomical differences observed in individuals with ADHD compared to typically developing control individuals**

Structural MRI provides information to describe the shape, size, and health of grey and white matter structures in the brain. This is possible because different tissue types reflect and interact with the radio and magnetic waves uniquely and so images of body tissue can be obtained. A sample of over 8000 individuals participated in these studies (**Appendix A**).

#### **Basal Ganglia**

The basal ganglia have been consistently implicated in problems associated with ADHD because they are a relay centre that helps to integrate and coordinate motor and cognitive activities (Hall, 2016). They are highly interconnected loops, also connected to the higher brain centres (cortex) and thalamus during mental activities. They are involved in rewards, impulse control during mental tasks, and coordination of motor control, like eye movements, during mental processing and other executive functions (EF). EFs involve the working memory,

emotional control, sustained attention, planning and prioritization, time management of activities and self-regulation. Meta-analyses of a range of studies comparing typically developing children to those with ADHD reported reduced basal ganglia volumes in children with ADHD (Carrey et al., 2012; Frodl & Skokauskas, 2012; Igual et al., 2011; Mahone et al., 2011a; Nakao, Radua, Rubia, & Mataix-Cols, 2011; Seidman et al., 2011; Soliva et al. 2010; Semrud-Clikeman, Pliszka, Bledsoe, & Lancaster, 2014; Qiu et al., 2011), in adolescents (de Mello et al., 2013; Moreno et al., 2014) and in adult males (Onnink et al., 2014; Proal et al., 2011). In a longitudinal study of 278 children aged 4 followed to age 18.9 years, Shaw et al. (2014) found a progressive reduction in the volume of the basal ganglia from childhood to adolescence. Similar results in adults with ADHD showed increased symptom severity linked to smaller volumes (Montes et al., 2010). Basal ganglia findings in ADHD have consistently been correlated to signs of emotionality, inattention and hyperactivity as well as to difficulties in EF reasoning processes.

### **Thalamus**

The thalamus relays sensory and motor signals to the cerebral cortex, a higher order, grey matter layer where cognitive functions are processed (Hall, 2016). A nerve impulse travels from the body surface towards the thalamus which receives it as a sensation. This sensation then passes onto the cerebral cortex for interpretation as touch, visual information, pain or temperature. The thalamus helps regulate consciousness, sleep and alertness; it is involved in memory formation and emotional expression. Inattention ADHD symptoms are linked to reduced thalamic volumes (de Mello et al., 2013; Ivanov et al., 2010; Proal et al., 2011; Xia et al., 2012), with significantly reduced iron levels found in the thalamus of children with ADHD (Cortese et al., 2012) thought to cause some inattention symptoms.

### **Cerebellum**

The cerebellum controls movement-related, cognitive functions and emotional regulation (Hall 2016). Motor commands from higher brain centres are modified in the cerebellum to make movements more accurate. It plays a major role in adapting and fine-tuning movements through a trial-and-error process to help a child, for example, to learn to colour-in within a specific area or to hit a ball at a target. Also it is involved in language processing. Lower cerebellar volumes were confirmed longitudinally in a study following children for 33 years; most affected regions were those involved in control of attention through the management of sensory information, and regulation of emotion and motivation (Proal et al., 2011; Montes et al., 2011; Bledsoe, Semrud-Clikeman & Pliszka, 2011; Lim et al., 2015).

### **Cerebral Cortex – Grey Matter**

The cerebral cortex is the outer covering of grey matter over the two brain hemispheres. Its regions receive sense (vision, hearing, and physical) information or initiate the production of body movements. Other regions are involved in more complex mental functions. The frontal cortex, temporal and parietal cortex manage memory, language, abstraction, creativity, judgment, emotion and attention (Hall, 2016).

Grey matter contains the cell bodies, dendrites and axon terminals of neurons; it is where all synapses terminate, that is where nerves make connections with other nerves. Grey matter typically follows a U-shaped developmental course, with males achieving the peak of frontal lobe grey matter volume around 12, females around 11; parietal lobes reach peak volumes at 10 for females and 12 for males and temporal lobes at 17 years for both sexes (Poletti, 2009). One area, the dorsolateral prefrontal cortex, is the last region to reach its final mature volume, a process that can continue into the third decade of life. The reduction of grey matter results from pruning of redundant nerve connections (synapses) and is thought to increase efficiency of mental integration across regions of the brain. It occurs in tandem with an improved

efficiency of nerve conduction, the result of the myelination of neuronal axons; this is observed as an increase in white matter which takes place at a linear rate through adolescence and into one's second decade of life (Steinberg, 2008). Therefore, as a child develops it gains speed and accuracy in mental processes, and better self-regulation over emotions.

Grey matter abnormalities are thought to be related to memory, attention, and EF deficits. Those with ADHD have reduced cortical thickness overall (Amico, Stauber, Koutsouleris, & Frodl, 2011; Bledsoe, Semrud-Clikeman, & Pliszka, 2013; Frodl & Skokauskas, 2012; Greven et al., 2015; Hoekzema et al., 2012; Lopez-Larson, King, Terry, McGlade, & Yurgelun-Todd, 2012; Makris et al., 2010; Proal et al., 2011; Almeida et al., 2010; Batty et al., 2010; Ahrendts et al., 2011; Fernández-Jaén et al., 2014). This cortical thinning is correlated with symptom severity (Shaw et al., 2013). A common finding in ADHD, confirmed in longitudinal studies (Shaw et al., 2011), is a delay of the cortical thinning that normally occurs around 12- 14 years, particularly in the prefrontal cortex. This delay, superimposed on an overall thinner prefrontal cortex (Shaw et al., 2012; Schweren et al., 2015), might reflect the difficulties in mental organisation and timing found in ADHD. By contrast, regions responsible for motor and sensory functions are thicker in adults with ADHD (Duerden, Tannock, & Dockstader, 2012) reflecting persistent hyperactivity. These variations, found across all ages, indicate abnormal development rather than developmental delay (Almeida Montes et al., 2013; Mahone et al., 2011b). This is because increased volumes in the anterior cingulate cortex (ACC<sup>1</sup>), frontal and prefrontal cortex have been observed in children with combined inattention-hyperactivity ADHD subtype (Semrud-Clikeman et al., 2014). Possible mediating factors for these results are proposed. For example,

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<sup>1</sup> The anterior cingulate cortex (ACC) has connections to both the "emotional" limbic system and the "cognitive" prefrontal cortex (PFC). It has an important role in integration of neuronal circuitry for emotional regulation, for example the ability to control and manage uncomfortable emotions. Avoidance of painful emotions is often the motivating force in negative behaviours such as substance abuse, binge eating, and suicide. (Stevens, Hurley, & Taber, 2011).

the pattern of cortical abnormalities found in children with ADHD and high IQ differ substantially from those with lower IQ (de Zeeuw et al., 2012). Grey matter volume was positively correlated with IQ for controls, but not in ADHD. De Zeeuw et al. (2012) concluded that the typical relationship between IQ and neural developmental structure is altered with ADHD.

### **Cerebral Cortex – White Matter**

White matter comprises of the white, myelinated connecting fibres between neurons. White matter reductions in ADHD tend to be seen in tracts connecting regions which show reductions in volume or other abnormalities (Cortese et al., 2013). This includes areas used for memory and EF. These white matter changes persist into adulthood, even in those with remitted ADHD (Cortese et al., 2013). Such connectivity disturbances were found in ADHD in a wide range of tracts connecting various brain regions (Adisetiyo et al., 2014; Cao et al., 2013; Chuang, Wu, Huang, Weng, & Yang, 2013; Hong et al., 2014; Konrad et al., 2010; Konrad et al., 2012; Qiu et al., 2011; Van Ewijk et al., 2014; Wu, Gau, Lo, & Tseng, 2014; Xia et al., 2012). They do not appear to be the result of myelination of axons (De Zeeuw et al., 2012; Helpern et al., 2011).

Sometimes increases in white matter have been found in tracts connecting the temporal lobe which receives and relays sensory information from the ears with other regions (Peterson et al., 2011), and fronto-striatal pathways which connect frontal lobe regions with the basal ganglia that mediate motor, cognitive, and behavioural functions (Tamm, Barnea-Goraly, & Reiss, 2012) explaining the attention difficulties in ADHD. Other disturbances in white matter tract connections correlate with the variability of symptoms found in ADHD (Lin et al., 2014).

### **Corpus Callosum**

The corpus callosum consists of about 300,000 white matter nerve fibres whose function is to connect the two brain hemispheres together; to relay and integrate motor, sensory, and cognitive messages between the two hemispheres. Abnormalities of growth found in the corpus callosum may reflect or perhaps even cause the delayed cortical thinning in grey matter in the prefrontal cortex in ADHD (Gilliam et al., 2011; Langevin, Macmaster, Crawford, Lebel, & Dewey, 2014; Cao et al., 2010; Dramsdahl, Westerhausen, Haavik, Hugdahl, & Plessen, 2012; Schnoebelen, Semrud-Clikeman, & Pliszka, 2010). Some differences found in ADHD have been thought to partly reflect differences in the study population as ADHD often coexists with undiagnosed Autism Spectrum Disorder (ASD) (O'Dwyer et al., 2014), conduct disorder (Sasayama et al., 2010), and epilepsy (Saute et al., 2014).

### **Summary of MRI findings**

The above studies provide statistically significant evidence that there are anatomical brain differences between those with ADHD and controls in regions of the brain which govern the integration of physical movement, the processing of mental tasks and the regulation of emotion and attention. Significant differences in the basal ganglia, cerebellum, corpus callosum, thalamus, and the white matter and grey matter of the cerebral cortex echo mental processing, attention, motivational and movement regulation, and the behavioural characteristics routinely observed in ADHD. These differences are able to distinguish those with ADHD from others with neurological disorders (Bansal et al., 2012). Moreover, grey matter differences identified in longitudinal studies point to abnormal development rather than developmental delay.

## **Functional MRI Findings in ADHD: Neural Circuitry Differences Observed In Those with ADHD Compared To Control Individuals**

Functional MRIs (fMRIs) provide a snapshot of the communication between different areas of the brain during a mental activity. In other words, they outline the neural circuits that are active during the processing of a task. fMRIs do this by measuring changes in blood flow to different parts of the brain and so it is possible to observe brain tissue activity, important when investigating the way different brain areas are connected to respond to stimuli. Findings from fMRIs depend upon the task, or experimental design, which the subjects undertake while the data is acquired. If the subjects are not required to undertake any task, fMRIs reflects resting brain activity. However, if subjects were required to perform some mental activity, then the fMRI data is analysed to extract the task related brain activity from the background “noise” unrelated to task performance. In all cases, images depict changes in blood flow to a given region of the brain, at a slightly delayed time from the actual neural activity in that region. Findings from the fMRI studies in ADHD below are clustered for the purpose of discussion by the function they were designed to assess and by the experimental design employed. The fMRI studies identified comprise studies with a total sample of 15123 participants (**Appendix B**).

## **Emotional Regulation and Reward Processing**

### **Resting state fMRI studies**

Resting fMRI studies of those with ADHD confirm that they have atypical neural circuit activations. Resting state studies yield information about the attention levels of an individual. In resting state fMRI studies subjects either have their eyes closed or open, or typically, data is recorded under both these conditions and compared. The brain networks engaged in resting state studies are known as the default network. The functions of the default network are related to EF and reward processing.

Resting state fMRI studies have shown a higher interconnectivity of the default network in children (Di Martino et al., 2013), in youth (7-16 years) (Fair et al., 2010), and adults (Mattfeld

et al., 2014) especially in the basal ganglia which participate in reward experiences and in focusing attention. Abnormal connectivity between the basal ganglia, the prefrontal cortex (Costa Dias et al., 2013) and other regions (McLeod, Langevin, Goodyear, & Dewey, 2014; Mennes et al., 2011; Mills et al., 2012; Sun et al., 2012; De Celis Alonso et al., 2014; Qiu et al., 2011), reflects the erratic emotional profiles of children with ADHD and their low persistence with tasks.

Other widespread abnormalities found in adults (An et al., 2013a; Cocchi et al., 2012; Hoekzema et al., 2014; Sato, Hoexter, Castellanos, & Rohde, 2012; Sokunbi et al., 2013), and in children (Ray et al., 2014) have been interpreted as representing a maturational lag (Sripada, Kessler, & Angstadt, 2014). In studies showing reduced connectivity in the default network, multiple hyperactive neural circuits were noted, including those involved in EF and emotional regulation in children (Posner et al., 2013), verifying the source of hyperactivity and inattention. Paralleling the abnormal increased activation in a range of circuits, decreased prefrontal activity was confirmed in adults (Cannon, Kerson, & Hampshire, 2011).

Abnormal patterns of connectivity between the amygdala, part of the emotion sensing system of the brain, and other regions in children, correlated with measures of emotional instability (Hulvershorn et al., 2014). A range of resting state fMRI abnormalities in boys (An et al., 2013a; Yang et al., 2011) were normalized with acute doses of methylphenidate (An et al., 2013b) which exerts its effects by acting on the dopamine and norepinephrine<sup>2</sup> transporters in various regions of the brain, including the frontal lobe, to upregulate those regions (Kowalczyk et al., 2019).

One resting fMRI study showed that the severity of different aspects of the symptom profiles in ADHD related to different regional brain abnormalities, which might account for the

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<sup>2</sup> Norepinephrine has multiple roles but essentially norepinephrine mediates arousal.



subtypes of ADHD and their expression (Chabernaud et al., 2012). A large longitudinal follow-up study demonstrated that a decrease in hyperactive/impulsive ADHD symptoms was linked to stronger connectivity in regions of the executive control network (Francx et al., 2015). Francx et al. (2015) also showed that those with remitting ADHD symptoms had higher frontal functional connectivity than controls. The brain's plasticity and compensatory mechanisms used by individuals in remitting cases, or genetically determined maturation aspects, are likely responsible for decreases in ADHD symptoms. These results and the brain's plasticity point to the need for support and early interventions for those diagnosed with ADHD.

### **Timing, Delay and Reward Studies**

One form of timing studies requires the participant to engage in paced and un-paced finger tapping. Results show that adults with ADHD have reduced activity in regions associated with sensorimotor timing which is needed for accurate responses to these tasks (Valera, Spencer, et al., 2010). Boys with ADHD aged 10-17 years have a reduction in activation in areas required for the processing of a response and the inhibition of irrelevant stimuli (Hart et al., 2014) as do younger children with ADHD (Vloet et al., 2010). Another timing study showed abnormal reward processing in youths with ADHD (von Rhein et al., 2015) reflecting the lack of motivation found in those with ADHD.

In a range of related tasks, adolescents with ADHD showed significantly increased brain activation in the amygdala, which processes emotions (Lemiere et al., 2012; Christakou et al., 2013). Monetary reward-delay tasks which present a cue indicating the possibility to gain money, lose money, or keep the same amount, showed adults (Carmona et al., 2012; Furukawa et al., 2014; Edel et al., 2013; Stoy et al., 2011; Wilbertz et al., 2013) and adolescents (Paloyelis, Mehta, Faraone, Asherson, & Kuntsi, 2012; Mizuno et al., 2013; Chantiluke et al., 2014) had abnormal activity in the basal ganglia, which are usually active during reward anticipation, and

had differences in a region involved in processing risks (Wilbertz et al., 2012). A meta-analysis of timing fMRI studies confirmed that differences were a result of reduction in activity in areas related to tasks involving timing (Hart, Radua, Mataix-Cols, & Rubia, 2012). Another meta-analysis of reward anticipation studies established the main finding seen above: the suppression of areas controlling impulsivity related to reward anticipation and emotional reaction was not evident in ADHD (Plichta & Scheres, 2014).

### **Emotional and Neutral Faces, Affective Images**

These experimental designs involve viewing and rating emotional and neutral faces. Youths with ADHD exhibit hyperactivity in the left amygdala, involved in the processing of facial emotions, while rating the fearfulness of neutral faces (Brotman et al., 2010) while adults with persistent ADHD show decreased activation to emotional stimuli in the brain's reward centres (Schlochtermeyer et al., 2011).

A failure of emotional inhibition in ADHD has been demonstrated through various studies; these show abnormal activity in the amygdala, and greater connectivity between the amygdala and the prefrontal cortex in adolescents (Posner et al., 2011b) and in adults (Schulz et al., 2014; Depue et al., 2010; Maier et al., 2014).

### **Go/no-go Tasks and Continuous Performance Tasks**

Motivation abnormalities have been demonstrated in those with ADHD with prevailing theories suggesting that inattention, impulsivity and hyperactivity may result from unregulated reward-seeking tendencies, including oversensitivity to rewards and an abnormal preference for immediate over delayed rewards ("delay aversion"). To examine the reward circuitry in ADHD studies have used a "go/no-go" experiment with no reward, monetary reward, or social reward conditions.

Findings showed disturbed information processing of the frontal EF areas resulting in disturbed decision-making processes in males aged 9-18 years old (Kohls et al., 2014) and adults (Dillo et al., 2010; Karch et al., 2010; Karch et al., 2014; Sebastian et al., 2012). Other studies have failed to find reduced frontal activity in these experiments pointing to response variability in ADHD (Carmona et al., 2012). A study of 11 sibling male pairs aged 8-20 years only one of whom had ADHD, compared with male controls, suggested genetic and/or environmental effects because connectivity between particular areas (the anterior cingulate gyrus and cerebellum) was reduced in both ADHD subjects and in their unaffected siblings compared to controls (Mulder, Van Belle, Van Engeland, & Durston, 2011). Other studies showed children (Spinelli et al., 2011; Ma et al., 2012) adolescents (Braet et al., 2011), and adults (Mulligan et al., 2011; Kooistra et al., 2010) had difficulties in refocusing their attention on task-relevant information after an error occurred.

## **Attention Regulation**

### **Interference tasks**

These tasks employ some form of interference to the required mental task by the use of distractor stimuli. Overall, they show atypical connectivity between various salient regions of the brain in ADHD and variability across studies. In the Stroop colour-word task, for instance, the subject is required to press a button corresponding to the ink colour of a word. Stroop task studies have shown that adults with ADHD combined subtype have a reduced activation in areas of the prefrontal cortex which correlated with measures of working memory (Burgess et al., 2010). Young adults (Depue, Burgess, Willcutt, Ruzic, & Banich, 2010) and adolescents (Passarotti, Sweeney, & Pavuluri, 2010a; Posner, et al., 2011a) have widespread abnormalities in regions implicated in EF, reward and motivation, and stimulus representation and timing.

Children and adolescents had more activation in areas involved in risk aversion and less activation in the areas processing visual and orientation data from the hand (Fan, Gau & Chou, 2014).

In a visual picture matching task in which distractor faces were shown to test working memory and distractibility, it was found that medication (methylphenidate) did not normalize the reduced caudate nucleus activity<sup>3</sup> in adolescents (Prehn-Kristensen et al., 2011). However, this caudate nucleus abnormality was partially normalised in some children through single doses of the medication methylphenidate (MPH), suggesting brain plasticity effects in some individuals where compensatory function effects are taken up by other regions (Rubia, Cubillo, Woolley, Brammer, & Smith, 2011a; Rubia et al., 2011b). MPH, the first-line treatment in ADHD, increases prefrontal dopamine and noradrenaline as well as subcortical dopamine availability by blocking the dopamine transporter (Prehn-Kristensen et al., 2011). Nonetheless, the contribution of MPH to working memory distractibility tested in the above studies and the precise mechanism of action of MPH in ADHD is not fully understood. Used in therapeutic doses, MPH inhibits over half of dopamine<sup>4</sup> transporters in the striatum and over three quarters of noradrenaline transporters in prefrontal regions leading to increased striatal dopamine levels and catecholamine levels in frontal and other cortical regions, and resulting in normalisation and upregulation of brain networks involved in sustained attention in ADHD (Kowalczyk et al., 2019). The mechanism of MPH's action is beside the point though. The issue here is that it is required to normalise the frontal lobe's responses in ADHD. It is worth noting also that although the use of MPH improves performance outcomes in some individuals, for some tasks, the latest systematic review and meta-analysis aimed to quantify MPH effects on

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<sup>3</sup> Caudate nucleus activity is essential for learning to take place.

<sup>4</sup> Dopamine is involved in both executive functioning neural pathways and the reward and motivation neural pathways.

academic productivity and accuracy for math, reading and spelling found no evidence that MPH leads to demonstrable gains in academic outcomes (Kortekaas-Rijlaarsdam, Luman, Sonuga-Barke, & Oosterlaan, 2019).

Longitudinal studies with adults followed up since childhood, showed reduced activation across a range of regions connected with EF (Cubillo, Halari, Giampietro, Taylor, & Rubia, 2011; Sebastian et al., 2012). In a short term memory test adolescents showed decreased prefrontal activation, involved in EF, and increased activation in the cerebellar vermis, involved in movement (Poissant, Mendrek, & Senhadji, 2014; Vloet et al., 2010), highlighting a source of hyperactivity in ADHD.

### **Cued Reaction Time Tasks**

In a cued reaction time task, letters are displayed in random order, with one letter being a target for response. In half of the cases, the target letter is preceded by a cue letter, in the other half no cue precedes the target. As would be expected reaction times to non-cued targets were significantly longer. Adults with persistent and remitted ADHD were followed longitudinally from childhood. Their results showed deficits in sustained attention and target detection, leading Clerkin et al. (2013) to conclude that ADHD is a neurodevelopmental disorder associated with lasting abnormalities in activation of areas responsible for working memory and consciousness, and during response preparation, even in cases where significant remittance of symptoms has occurred.

## **Hyperactivity and Sensory - Motor regulation**

### **Stop Tasks and Switch Tasks**

A stop task requires the suppression of an already triggered motor response, and is designed to assess hyperactivity. Areas involved in coordinating motor inhibition were under-

activated in boys (Cubillo et al., 2014; Sebastian et al., 2012), children, (Rubia, Cubillo, et al., 2010; Passarotti, Sweeney, & Pavuluri, 2010a; Passarotti et al., 2010c), and in adults (Cubillo et al., 2010; Congdon et al., 2014).

Switch tasks require subjects to switch between processing in two different spatial dimensions; sometimes they must indicate whether an arrow points up or down, on other occasions whether it points left or right. Deficits in frontal and prefrontal regions as in a stop task, plus additional deficits in activation of the basal ganglia and cingulate gyri (Rubia et al., 2010a), and prefrontal activity (Rubia et al., 2010b) were found in children, and also adults who engage different brain areas during task switching compared with controls (Dibbets, Evers, Hurks, Bakker & Jolles, 2010).

### **Go/no-go tasks and Continuous Performance Tasks**

Experimental designs which are used to assess motivation and emotional regulation, are also employed to assess impulsivity/hyperactivity as they require an individual to control and suppress eye movements, that is, to look away from an input stimulus and suppress the automatic urge to look. Adults with ADHD had reduced activation in preparation for the suppression of eye movements in the responsible brain areas (Hakvoort Schwerdtfeger et al., 2013). Continuous performance tasks (CPTs) similar to the “go/no-go” experiments require participants to remember a three-digit sequence and press one button if a subsequent presentation matched the sequence or another button if it did not. Results of these studies showed decreased connectivity in the visual attention network compared to controls in youths aged 9-15 years (Xia, Foxe, Sroubek, Branch, & Li, 2014), and adults (Schneider et al., 2010).

Further evidence of abnormal sensory regulation was obtained from a similarly designed auditory attention task. Adolescents with ADHD inattentive subtype showed deficits in the temporal lobe which is responsible for processing hearing (Orinstein & Stevens, 2014).

## **Judgment tasks**

Much research has identified abnormal visual information processing in ADHD.

Hyperactivity interferes with the attention focus required in judgement tasks; to test this children with ADHD had to indicate whether a coloured letter in a word was to the left or right of a word, or if the test letter was an “A” or not. Results showed lower activation in the visual cortex and other relevant EF networks because the authors noted that this characteristic reflects greater engagement with task-extraneous content, indicative of less efficient top-down task-directed control over visual processing (Sigi Hale et al., 2014).

## **Executive function - Working Memory**

EF refers to a number of mental functions used to control and process information and responses and integrate new stimuli to update responses. EF is a core component of self-control or self-regulation ability so it is involved in emotional regulation, as well as many other functions. One model of EF is the three-factor model of EF that includes inhibition, updating (working memory), and shifting (Miyake, et al., 2000). Inhibition is the ability to delay or dismiss a dominant response. An example might be giving into an impulse to yell out in class. Updating is the ability to monitor and process incoming information in the working memory to adapt responses as a result. Shifting reflects the cognitive flexibility to switch between different tasks or mental states in light of changing circumstances or demands. There are however, difficulties in assessing EF and extracting the three components noted above because of task-impurity found in experimental designs. The studies on EF identified below are centred on the dimension of updating (working – memory).

## **Working memory tasks**

It has been speculated that working memory deficits may underlie the difficulties with attention and EF in ADHD (Burgess et al., 2010). To test this, a working memory task was used, whereby a letter previously presented is presented again and requires the person to identify it. To test for attention as well as short-term memory the letter 'X' is presented at the same time. Male adults showed significant reductions in activation of widespread brain regions during these tasks, unlike female adults (Valera et al., 2009).

In another working memory task adults showed raised activity in some regions (the ACC, parts of the frontal lobe, hippocampus, motor areas), but reduced activation in the area involved in language (Ko et al., 2013; Li et al., 2014). In similar tasks children also showed increased activation in some areas (dorsolateral prefrontal) and decreases in others (posterior parietal and occipital) (Malisza et al., 2012; Fassbender et al., 2011; Massat et al., 2012; Kobel et al., 2010). Results were consistent in youths (Bédard et al., 2014). Investigations of the genetics of ADHD have shown that working memory abnormalities observed through fMRIs were linked to a gene (the DAT1) (Brown et al., 2011) which might explain the familial prevalence of ADHD.

Working memory tests with happy, angry, or neutral faces also showed abnormal activation of various parts of the brain in adolescents (Passarotti, Sweeney, & Pavuluri, 2010b).

## **Fluid reasoning tasks**

Fluid reasoning tasks test the ability to solve problems with no prior knowledge by concentrating on a task using the working memory, which has been found to be problematic in ADHD. Examples of these tests are found in Raven's Progressive Matrices tests (Raven & Court, 1998). In one such task children select one of two coloured shapes by pressing a button to



complete a sequence of coloured shapes. Children with ADHD were faster but less accurate in selecting shapes, confirming classroom observations, and showed lower activation of the relevant brain areas during the processing of the task (Tamm & Juranek, 2012).

An fMRI meta-analysis of 39 studies of children and 16 studies of adults found that systems involved in EF and attention showed low activation, while systems involving the motor networks showed hyper-activation (Cortese et al., 2012b). Another meta-analysis of studies employing go/no-go, stop, and working memory tasks found decreased frontal lobe activity (responsible for EF) across all tasks (McCarthy, Skokauskas, & Frodl, 2014). Such findings correlate with observations of inattention and physical hyperactivity in ADHD. In sum, the difficulties observed in mental processing tasks in ADHD probably originate in abnormal working memory capacity, a problem that is likely genetic.

### **Summary of fMRI findings**

Results from the various experimental designs employed to investigate the processing characteristics of those with ADHD show they process information and respond to tasks differently from controls due to disordered fronto-striato-cerebellar brain circuitry which underpins their executive function difficulties at the core of ADHD. These differences reflect and corroborate observations that have been documented about the characteristics of ADHD. Differences in the resting brain across the default network which shows levels of alertness and attentiveness, through to differences in accuracy with task processing, reward anticipation and self-monitoring and regulation are all reflected in the way neural circuits are activated and employed in ADHD.

### **Discussion**

This systematic review has outlined findings emerging from MRI and fMRI neuroimaging studies of individuals with ADHD compared to matched control participants. It indicates that

individuals with ADHD have distinct anatomical brain differences compared to controls and important functional differences in mental processing. These differences have been correlated to behavioural and cognitive differences with diagnostic neuroimaging techniques corroborating findings (e.g., Bohland, Saperstein, Pereira, Rapin, & Grady, 2012; Colby et al., 2012; Dey, Rao, & Shah, 2012; 2014; Eloyan et al., 2012; Fair et al., 2012; Peng, Lin, Zhang, & Wang, 2013). The reviewed studies also show ADHD can persist into adulthood, which is reflected in statistical determinations of the adult prevalence of ADHD (Fayyad et al. 2017; Caye et al. 2016). Findings in this review are reinforced by research published in *Lancet Psychiatry* (Hoogman et al., 2017) which documents an MRI study across a sample of 1713 individuals with ADHD and 1529 controls aged 4-63 years, and concludes that those with ADHD have altered brain anatomy.

Understanding differences in brain morphology in ADHD is important for teachers, teacher educators and inclusive educational policy. These differences support the view that ADHD has biological correlates and antecedents, and is not a manifestation of sociological factors, poor behavioural control or parenting as Hinshaw (2005) cautioned. Even if teachers and inclusive educational policymakers believe that ADHD results from discipline patterns or another socially constructed aetiology, an explanation which is possible given the brain's plasticity although it has not been verified by longitudinal studies, school students with ADHD need access to targeted funding under the Disabilities Act, like others with disabilities such as hearing impairment or ASD. This will ensure that they get the tailored pedagogies that they require to flourish in school.

The critical matter of the brain's plasticity is an important consideration here. It could be argued that the anatomical and functional differences found in ADHD are simply due to the brain's unique ability to adapt to experience, its inherent plasticity. Those who do not believe

that ADHD is a neurobiological condition might argue that the lack of appropriate discipline and socialisation is responsible for the development of the behavioural profile of ADHD. It is indeed the case that neuroplasticity could result in anatomical differences between those with ADHD and others, just as it is possible that differences found in those who have, say, musical ability or fine drawing skills, might have arisen as a result of practice, rather than from a biological, genetic predisposition for musical or artistic talent. Nonetheless, this argument is not a valid one for the purposes of teacher education or inclusive educational policy to support students with ADHD in schools. After all, a student whose visual impairment resulted from an accident would not be denied access to inclusive educational provisions based on the argument that the visual impairment was accidentally caused as a result of poor parental supervision. No disability should hold precedence over another in relation to government funding for schools.

Inclusive educational provision through the public education system under the Disabilities Act, is desirable because research consistently shows that cognitive and behavioural approaches are successful in addressing academic outcomes (Purdie, Hattie, & Carroll, 2002) and symptoms of ADHD (Hoekzema et al., 2011). For example, many of the grey matter structural abnormalities responsible for EF found in ADHD with MRI, have been shown to be improved or eliminated entirely with educational training (Hoekzema et al., 2011). The processes targeted by the training program are working memory, cognitive flexibility, attention, planning, and problem solving. Some examples of tasks used in the training are labyrinths (planning), word list recall (memory), detecting the missing numbers from numerical lists (attention), creating lists of objects sharing certain characteristics (cognitive flexibility), and code deciphering (problem solving). Hoekzema et al. (2011) state their “findings illustrate

the capacity of the nervous system for rapid morphological adjustments in response to environmental triggers” (Hoekzema et al., 2011, p. 1741).

### **Implications for Practice**

Teacher education, professional learning and inclusive educational policy changes to fund school support for those with ADHD must be addressed in the near future. This follows when considering that the prefrontal cortex responsible for EFs like planning, attention, cognitive flexibility and attention setting, is one of the last cortical regions to reach its mature thickness in adolescence (Poletti, 2009) making it highly amenable to training. Studies with adolescents who have ADHD demonstrate that different symptoms are related to different EF deficits: the dysfunction of the orbital portion of the prefrontal cortex leads to delay aversion and is related to hyperactivity/impulsivity symptoms and motivational, reward related impairment, while dysfunction of the dorsolateral prefrontal cortex demonstrated in poor inhibitory control and poor spatial working memory, is related to inattentive symptoms. Since ADHD impairments are related to specific EFs it is possible to plan educational support with success. Preliminary results of educational training of those EFs in ADHD have shown promise, in particular for the working memory of children aged 7-12 years old (Klingberg et al., 2002; 2005). These studies found that a brief, intensive training of the working memory not only improved working memory performances but also improved performances of other non-trained EFs (planning, inhibition) and had beneficial effects at a behavioural level, that is, a reduction in the number of head movements.

Studies of teacher beliefs show that beliefs direct teacher pedagogy, classroom management and referral procedures. Teachers are instrumental in identifying ADHD and thus can influence the diagnosis of ADHD. It is therefore imperative that teacher educators, education departments and local jurisdictions provide up-to-date education and professional

learning to ensure teacher knowledge is current so they provide supportive pedagogy for students with ADHD. This could improve their trajectory and reduce their symptoms and stigmatisation. Furthermore, cognitive behavioural interventions carry no adverse health effects in relation to ADHD. In sum, funding to support those with ADHD needs to be more readily available. The current exclusion of ADHD from eligible categories for support under the Education Adjustment Program operating in Queensland, Australia

(<https://education.qld.gov.au/students/students-with-disability/education-adjustment-program>) which offers funding for individuals with ASD, hearing impairment, intellectual disability, physical impairment, speech–language impairment, or vision impairment, is a case of inequity. Funding will have a significant educational impact upon students with ADHD, and the teachers who currently feel unable to support their classroom needs (Anderson et al., 2012; 2017).

Meanwhile, teachers working towards inclusive practice should ensure they implement pedagogies that foster the learning of those students with ADHD. A promising approach to inclusive practice and instruction for students with ADHD is based on the principles of universal design for learning (UDL), an educational framework that guides the development of flexible learning environments intended to be inclusive, to help all learners succeed. Underpinned by Vygotsky's social constructivism theory and his emphasis on the 'zone of proximal development' (Mitchell, 2014) UDL is a multi-component approach that covers all aspects of education from curriculum, assessment and pedagogy, to classroom and school design, as it has its origins in architecture and engineering. UDL is premised on the view that educational practice must be flexible. That is, the way that information is presented, the ways students are required to demonstrate their learning and the ways that students engage in the classroom should not be fixed. Therefore, UDL is designed to reduce barriers in instruction, provide

appropriate accommodations and supports for students, while maintaining high achievement expectations for all students including those with disabilities or learning difficulties (National Center on Universal Design for Learning, 2014).

The principles of UDL require clarity of instruction and teachers need professional learning in order to understand and apply this framework in their classroom contexts for students with ADHD. In brief, UDL stipulates that curriculum is delivered through multiple means of representation to give learners various ways of acquiring new information and knowledge. Assessment must be derived through multiple means of expression to provide all learners with alternatives for demonstrating what they know. Moreover, multiple means of engagement must be employed to engage learners' interest, challenge them, and motivate them to learn (National Center on Universal Design for Learning, 2014). These principles support learning processes in students with ADHD, by engaging three primary brain networks: affective networks responsible for motivation and setting the priorities; recognition (auditory and visual) networks responsible for gathering information; and strategic, executive, networks responsible for planning and executing actions (Dell, Dell, & Blackwell, 2015; Meyer et al., 2014; Rao et al., 2014). Classroom procedures and structure are also integral to UDL. The classroom must have clear rules and expectations, a structured environment and routines. Students with ADHD should be seated at the front of the class near the teacher and near peers who are good behavioural role models (Mitchell, 2014). To be engaging learning activities should be stimulating, meaningful, and involve practical projects rather than just writing or lectures. Attention to routines to ensure variations in energy levels is important for students with ADHD so a range of low and high-energy activities should be offered during the school day to minimise fatigue, hyperactivity and inattention (Zelenka, 2017). Underscoring all this is the caveat that individual students with ADHD need support and accommodations tailored to meet

their specific needs. Therefore, a primarily inattentive student will need accommodations that are distinct from those tailored for a student who is primarily hyperactive.

### **Future directions**

Neuroimaging studies cannot not provide all the answers to salient questions about ADHD. Even longitudinal studies do not describe life-course experiences that might influence changes observed in brain structure due to neuroplasticity. We do not know why some ADHD cases persist into adulthood and others do not, in other words, whether some resolve because of school support or other experiences, such as entering university or undergoing some particular training.

While it might be commonly believed that a biological origin of a mental disorder means that the disorder is 'real', in considering future explorations of ADHD it has to be noted that discussions around mental disorders question the diagnostic approaches taken thus far (Borsboom, Cramer & Kalis, 2019; Schmittmann et al., 2013). Scholarly debates propose that currently used approaches to conceptualise mental disorders, based on either locating the source of the disorder in a particular neurological, biochemical or anatomical abnormality, described as a reflective model, or in defining a mental disorder by the observable symptoms, the formative model, have not fully elucidated the scope and causality of a range of mental disorders. This is particularly evident in the case of ADHD. An alternative way to explain mental disorders has been proposed (Borsboom, Cramer & Kalis, 2019; Schmittmann et al., 2013) which is based on a neurological network perspective of the interaction of symptoms, which does not imply that they are socially constructed, although they might be context dependent (Borsboom et al., 2019). This network approach to understanding ADHD would look at how distinct aspects of the condition's expression predispose or influence other aspects of ADHD.

For example, how hyperactivity might lead via neural pathways to inattention and so on, separate from an underlying brain structure causing this to happen. This network approach to examining ADHD supposes that symptoms do not co-occur because they are symptoms of a common underlying disorder, but because they directly influence each other. The details of these considerations are beyond the scope of this paper however, they do illustrate possible ways to understand ADHD not yet been investigated, which nonetheless do not imply that ADHD is socially constructed or any less 'real'.

Even so, qualitative studies need to be conducted with adults with persisting and remitting ADHD to understand their experiences to possibly disentangle the influences that might have affected their condition. In addition, longitudinal research must be conducted to disentangle environmental effects, such as school interventions or other ecological factors, upon children's maturation and developmental trajectories.

Research with teachers and other school personnel should be undertaken after they have engaged in professional learning to understand the underlying neurological basis of ADHD to see if their views about students with ADHD are modified. Finally, teacher educators need to be better informed about ADHD so that they can prepare adequate training for pre-service teachers to enter the inclusive classrooms that are now the norm rather than the exception.

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