FOCUSING ON THOSE WHO CANNOT FOCUS

by

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Abstract

The neurological differences present in the brains of people with ADHD have been extensively studied in the past 30 years using neuroimaging techniques such as functional magnetic resonance imaging (fMRI), among others. After going through the history of ADHD research, this paper reviews a wide range of research conducted using these two techniques and uses their findings to attempt to persuade skeptics that ADHD is not simply 'people being lazy'—it is a real, neurologically differentiated disorder involving functional deficiencies in multiple specific brain regions. Differences in neural activation rates in the ventral striatum and ventral anterior thalamus greatly hinder the reward-processing capabilities of individuals with ADHD, making it more difficult for them to make wise decisions. Lower frontal striatal network activation in ADHD brains hinders response-inhibition capabilities. Methodological concerns involving the differences between selective attention and response inhibition are also addressed. Activation differences in the left inferior prefrontal cortex, cerebellum, and left inferior parietal lobe adversely impact ADHD individuals' ability to accurately judge timing. Heightened activation in the precuneus and posterior cingulate cortex leads to issues with the brain's default mode network in ADHD populations. Lowered activation in the frontal parietal, lateral prefrontal, temporal, striatum, and posterior parietal regions of the brain are associated with decreased working memory capabilities, the possible source of ADHD people's characteristic scatterbrained tendencies. There is evidence to suggest that ADHD has a genetic component, specifically involving the DAT-1 genotype. In conclusion, people with ADHD are not simply 'being lazy'—they are often trying their best to cope with neurological differences that make life more difficult for them than for those without the disorder.

Acknowledgements

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Focusing on Those Who Cannot Focus – Literature Review of fMRI Research on ADHD Populations

There have always been people who cannot focus. People who cannot sit still. People who cannot spend prolonged periods of time doing things they are not interested in. And yes, people who cannot even spend prolonged periods of time doing things they *are* interested in. Treated as though they lacked the conviction to do these tasks, these people were simply deemed 'lazy.' As it turns out, some people have biological differences in their brains that affect their ability to concentrate, to regulate impulsivity, to sit still, and more. In 2020, a diagnosis that can be given to such people (provided they meet the necessary conditions) is 'Attention Deficit Hyperactivity Disorder,' or ADHD for short. In popular culture, there is somewhat of a stigma against this diagnosis. Some members of older generations feel the need to decry the designation. '*Back in my day, we didn't make excuses. We got stuff done and didn't complain.'* Some medical professionals believe that the disorder is over-diagnosed; some believe that it should not be diagnosed at all. In this thesis, I will aim to prove those people wrong. I will be conducting a rigorous review of literature on the physically observable differences between the brains of people with ADHD and the brains of 'neurotypical' individuals—if there is even such a thing. How can these differences be observed? Of course, behavior can be observed, but I wish to dive deeper. Using functional magnetic resonance imaging (fMRI), changes in blood oxygen concentration can be observed, allowing scientists to determine which parts of the brain are responsible for which tasks and how effective they are at doing so. Using this, ADHD and non-ADHD brains can be compared. What brain regions work differently? What do these differences affect? Throughout the course of this thesis, I will seek answers for these questions and explain what I find.

Review of Historical Literature

18th Century

In 1798, Sir Alexander Crichton, a Scottish physician, recorded the earliest known scientific observations of the disorder now known as ADHD in the second chapter of his book, "On Attention and its Diseases." He refers to "the morbid alteration to which attention is subject" as "the incapacity of attending with a necessary degree of constancy to any one object" (Crichton, 1798, reprint p. 203).

> "When born with a person it becomes evident at a very early period of life, and has a very bad effect, inasmuch as it renders him incapable of attending with constancy to any one object of education. But it seldom is in so great a degree as totally to impede all instruction; and what is very fortunate, it is generally diminished with age" (Crichton, 1798, reprint p. 203).

In this passage, Crichton makes a handful of observations that remain true to this day. He also treated the disorder with a certain degree of respect, a surprising mindset to have in the field of 18th century 'proto-psychology.'

19th Century

For better or worse, the next recorded depiction of what we now know as ADHD came in the form of a 19th century children's storybook from Germany. In 1844, a German physician named Heinrich Hoffmann wrote and illustrated a collection of children's stories for his son. One of these stories was "Zappelphilipp" ("Fidgety Phil"), a tale of a young boy whose inability to sit still at the dinner table causes family conflict (Lange et al., 2010). Philipp engages in numerous behaviors characteristic of ADHD cases, such as inattention (not listening to his father), hyperactivity (squirming around in his chair excessively), and perhaps most importantly of all,

his behavior causes social conflict and interpersonal social impairment (his parents get very angry at him). In this setting, ADHD is not really treated as a disorder—rather, it is treated as an undesirable personality trait, rather than an organic disorder, it was regarded as a lack of will by the child or a parenting flaw of the parents.

20th Century

In the early 20th century, the disorder we now know of as ADHD was still regarded as a character flaw. In 1902, forty-six years after the death of Alexander Crichton, Sir George Frederic Still delivered the Goulstonian Lectures, widely considered to be the beginning of the scientific study of what we now know as ADHD (Lange et al., 2010). A British pediatrician, Still discussed "the particular psychical [sic.] conditions" that are "concerned with an abnormal defect of moral control in children" (Still, 1902, p.1008, cited by Lange et al., 2010). 'Moral control' was "the control of action in conformity with the good of all" (Still, 1902, p. 1008, cited by Lange et al., 2010). Interestingly, Still observed a set of 20 children with a 'defect of moral control as a morbid manifestation, without general impairment of intellect and without physical disease'' (Still, 1902, p. 1079, cited by Lange et al., 2010); 15 of these children were boys, and 5 of them were girls. This is consistent with modern observations of the disorder that commonly describe a 3:1 male to female ratio of ADHD occurrence (Barkley, 2006b, cited by Palmer & Finger, 2001, cited by Lange et al., 2010).

The first known occurrence of medicinal treatment for ADHD occurred in 1937, when Charles Bradley treated children hospitalized with "emotional problems," learning, and behavioral difficulties with Benzedrine, a stimulant, to reduce headaches caused by a different procedure (Gross, 1995, cited by Lange et al., 2010). This treatment led to marked increases in

school performance and subduing of hyperactive behavior (Gross, 1995). These findings went largely unnoticed in the scientific community for the next 25 years, however (Conners, 2000).

The penultimate major phase of historical understanding of ADHD involved a now-outdated diagnosis called Minimal Brain Dysfunction (Conners, 2000). Research that scientists like Bradley had conducted indicated that some sort of functional disturbance was at play, rather than overt brain damage (Conners, 2000). In 1962, Clements and Peters took this research and established a clinical approach for diagnosing 'Minimal Brain Dysfunction,' the first widely accepted medical diagnosis of the disorder now known as ADHD. It involved teachers and parents providing a holistic perspective of symptoms, empirical measurement of learning, perceptual-motor, and coordination deficits, observation, and longitudinal documentation of hyperkinesis, impulsivity, short attention span, and emotional liability (Clements & Peters, 1962, cited by Conners, 2000).

In the 1980s with the release of the DSM-III, the disorder was referred to as "Attention Deficit Disorder, divided into two different categories: With Hyperactivity and Without Hyperactivity. When the DSM-III-R came out, the 'ADD with hyperactivity' distinction was reworked into Attention Deficit Hyperactivity Disorder, and the 'ADD without hyperactivity' distinction was reworked into a residual category called 'undifferentiated ADD" (Lange 2010).

21st Century

As of the early 2010s, the classification of 'ADD' has been retired. Instead, there are three subtypes of ADHD: Inattentive type (formerly ADD without hyperactivity), Hyperactive/Impulsive type (formerly ADD with hyperactivity), and Combined type (has symptoms of both) (American Psychiatric Association, 2013). A 2010 study surveying the different subtypes of ADHD found that 62% of adults ADHD were of the combined type, 31%

were of the inattentive only type, and 7% were of the hyperactive/impulsive only subtype (Wilens et al., 2010). This shows us that there is much overlap between the two conditions, making a purely dichotomous classification impractical.

Review of Current Literature

Having discussed the history of ADHD 'research,' I will now move on to a discussion of current, empirical, and neuroimaging-based research on the disorder.

Table 1: fMRI Studies Covered.

Study	**Page**	**Topic in Question**	**Task Used**	**Findings**
Booth et al., 2005	p.14	Response Inhibition & Selective Attention: How ADHD diagnosis affects selective attention and/or response inhibition in children	Functional activation task (detailed below)	Compared to those with ADHD, children without ADHD displayed greater neural activation in many brain regions during a selective attention task, and much greater neural activation in many brain regions during a response inhibition task.
Braet et al., 2011	p.19	Genetic factors: Interaction between DAT-1 genotype and ADHD diagnosis and how it affects neural activation	SART	In many brain areas, there was a significant difference in neural activation between DAT-1 and non-DAT-1 genotypes ONLY when ADHD was also present. When ADHD was not present, there was not a significant difference in neural activation between DAT-1 and non-DAT-1 genotypes.
Hart et al., 2012	p.16	Timing-processing differences, impulsivity, affect regulation	Meta-analysis of studies using: Sensorimotor synchronization task, freely timed finger sequencing	ADHD patients had reduced activation in many brain areas typically involved in timing, such as the left parietal lobe, caudate, and anterior cingulate, corresponding to greater impulsivity and reduced affect regulation. However, ADHD

			task, delay tasks of second intervals	patients had consistently higher levels of activation in the precuneus and posterior cingulate cortex
Hwang et al., 2019	p.15	Methodological concerns regarding selective attention and response inhibition: Segregation of response inhibition and selective attention	Factorial go/no-go task	ADHD was significantly more positively correlated with error rate on the no-go (response inhibition) trials than on the go (selective attention) trials. In ADHD patients, response inhibition seems to be more severely impaired than selective attention. fMRI results found that different regions in the brain were involved in the response inhibition tasks and sustained attention. ADHD diagnosis showed a negative linear relationship with BOLD responses in the left interior insular complex, associated with diminished ability for response inhibition.
Ko et al., 2013	p.16	Working memory differences: Phonological and visuospatial differences	Phonological and visuospatial 2-back & 3-back tasks	With ADHD adults exhibiting higher brain activation in certain areas on the easier task but much lower activation in different areas on the harder task, they argue that ADHD adults spend more mental energy trying to focus on simpler phonological working memory tasks, while brain activation of the left frontal parietal network is impaired when it comes to more difficult working memory tasks.
Metin et al., 2018	p.13	Reward-processing differences: how cues signaling	Reward-giving spatial attention task	In the ventral anterior thalamus (VA), the controls exhibited greater activation in response to

		reward and feedback are processed in ADHD brain differently than in non-ADHD brains.		reward-predicting cues, as compared with no-reward cues, while the reverse pattern was observed in the ADHD group.
Plichta & Scheres, 2014	p.12	Reward-processing differences	Meta-analysis of studies using: Monetary incentive delay task	For ADHD populations, the more impulsive you are, the less responsive the ventral-striatal system in your brain is, as opposed to normal populations, where increased impulsivity is associated with increased ventral-striatal system responsiveness.
Silk et al., 2008	p.18	Visuospatial working memory differences in ADHD patients	Raven's Progressive Matrices Task	Found different neural activation rates in numerous parts of the brain, suggesting the existence of a widespread 'functional network' that may be fundamental for visuo-spatial information processing and relational reasoning, and that this network is not activated as broadly in ADHD brain than in non-ADHD ones.

Reward-Processing Differences

A meta-analysis of fMRI studies on reward anticipation found that for ADHD populations, the more impulsive you are, the less responsive the ventral-striatal system in your brain is, as opposed to normal populations, where increased impulsivity is associated with increased ventral-striatal system responsiveness (Plichta & Scheres, 2014). The ventral striatum is a part of the brain's dopaminergic reward system in a relatively centrally located part of the brain. It is responsible for reward processing. A defect in the brain's reward-processing center means that people with ADHD do not respond to rewards correctly. Tasks utilized in the studies

this meta-analysis covers predominantly included the monetary incentive delay (MID) task (Scheres et al., 2007; Knutson et al., 2000; 2001b), a task that includes three distinctive phases: an anticipation period (a delay), a target, and feedback (Plichta & Scheres, 2014). The MID task is helpful for studying two important features of neural reward processing—reward anticipation, occurring between cue and target presentation, and feedback processing, which occurs during the presentation of the performance feedback. Metin & colleagues (2018) wanted to see how reward processing differs between ADHD and non-ADHD populations. They used a spatial attention task in which the participants had to decide if the upper or lower gap in a circle (target) was larger while only observing them using their covert attention. With two circles appearing on each side of a computer screen, the participants looked at the middle and were directed to attend specifically to a particular side of the screen by an arrow that would sometimes appear in the middle of the screen. Correctly answering would cause a congratulatory message to appear on the screen; this was designed to involve the reward-processing parts of the brain (Metin et al., 2018). They used BOLD fMRI analysis to support their arguments (Metin et al., 2018). They found that, in the ventral anterior thalamus (VA), an area not typically associated with reward processing; rather, with motor preparation for pressing a button after observing a cue (Metzger et al., 2010; cited by Metin et al., 2018), the controls exhibited greater activation in response to reward-predicting cues, as compared with no-reward cues, while the reverse pattern was observed in the ADHD group (Metin et al., 2018). This suggests that children with ADHD seem to have a different preparatory response to rewards during the orienting phase of attention, possibly associated with a deficit in guiding attentional resources according to reward contingencies, leading to impaired information processing.

Response Inhibition & Selective Attention Differences

Response inhibition refers to the brain's ability to filter out actions that are no longer appropriate to the goal at hand (Verbruggen & Logan, 2008). On the other hand, selective attention refers to the ability to or process of selecting a certain stimulus out of many occurring simultaneously. A 2005 study by Booth & colleagues wanted to analyze response inhibition and selective attention differences between ADHD and non-ADHD children using fMRI. Two tasks were used during this experiment. All participants performed selective attention tasks followed by response inhibition tasks, both of which were functional activation tasks in which the participant is rapidly presented with red triangles, blue triangles, or red trapezoids. The selective attention task alternated between blocks of one and nine targets on the screen at a time. The participant would press a button with their index finger if a red triangle was not shown and would press a button with their middle finger if a red triangle was being shown. The idea here is to test a participant's ability to selectively attend to red triangles present on the screen. In the response inhibition task, the participants would go a few minutes pressing a button every time something appeared on the screen, regardless of whether it was a red triangle. Then they would go a few minutes only pressing the button if a red triangle was not on the screen. Here, the ability to inhibit a response to a target is tested. They used fMRI while this task was being performed to assess neural activation in various brain regions to see if ADHD had any effect—and it did. They argue that while there are significant differences in both areas between the ADHD and non-ADHD groups, the differences are more pronounced when it comes to response inhibition. Children without ADHD showed [barely] significantly greater anterior cingulate and thalamic activation than children with ADHD during

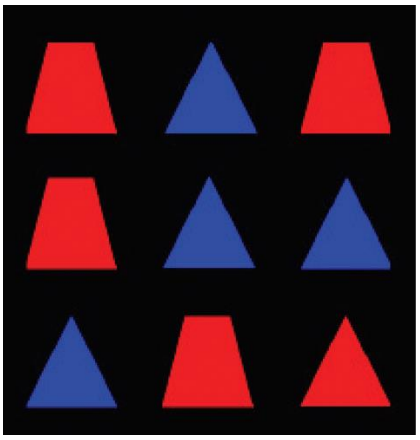

Figure 1: Selective attention task used in Booth et al., 2005.

the selective attention task. Regarding response inhibition, the effect was greater; children without ADHD showed considerably greater frontal striatal network activation than those with ADHD on the response inhibition task (Booth et al., 2005). A methodological question present in the field of neuroimaging ADHD research is the ambiguity between differences attributed to response inhibition and those attributed to selective attention. The ability to selectively attend to necessary information and the ability to inhibit response to unnecessary information seem somewhat intertwined, and so a 2019 study by Hwang & colleagues wanted to use a factorial no/go design to segregate response inhibition from selective attention when testing ADHD differences using fMRI (Hwang et al., 2019). They used a simple no/go task in which the participants would press a button whenever they saw Spider-Man appear on the screen, and not press the button whenever they saw the Green Goblin show up on the screen. The idea was that accurately recognizing and pressing the button when Spider-Man appeared on the screen would test their selective attention capabilities, and their ability to correctly not press the button when Green Goblin was on the screen would test their response-inhibition capabilities (Hwang et al., 2019). Their behavioral results indicated that ADHD was significantly more positively correlated with error rate on the no-go (response inhibition) trials than on the go (selective attention) trials. This trend echoes what Booth & colleagues (2005) found; in ADHD patients, response inhibition seems to be more severely impaired than selective attention. Their fMRI results found that different regions in the brain were involved in the response inhibition tasks (esp. the anterior insula) and sustained attention

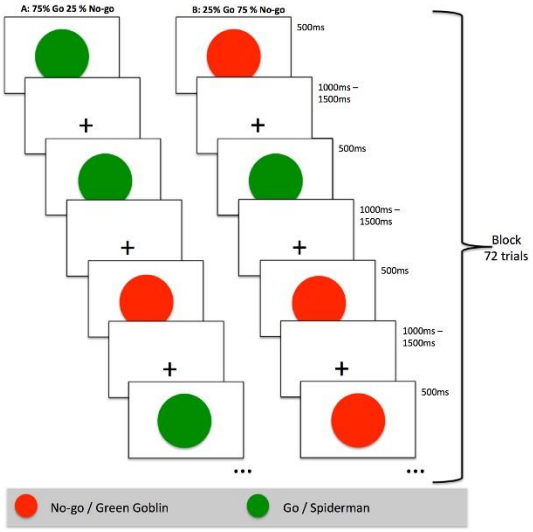

Figure 2: No/go task used by Hwang et al., 2019.

tasks (esp. the superior frontal cortex) (Hwang et al., 2019). They also found that ADHD diagnosis showed a negative linear relationship with BOLD responses in the left interior insular complex, associated with diminished ability for response inhibition. When it came to selective attention-involved brain regions, the results were somewhat mixed between groups; the relationship here was not so convincing. Based on these findings, the researchers assert that they found sufficient evidence to establish that there are two separate forms of dysfunction impairing ADHD patients (Hwang et al., 2019).

Timing Differences, Impulsiveness, & Affect Regulation

Having impaired timing functions is closely associated with impulsiveness, a poorly timed, impatient response pattern in which an individual weighs the passing of time as being worse than the consequences for doing so, showing poor temporal foresight (Rubia et al., 2009a; cited by Hart et al., 2012). A meta-analysis of fMRI studies on timing deficits in ADHD found that, relative to control (non-ADHD) populations, ADHD patients had reduced activation in many brain areas typically involved in timing (Hart et al., 2012). In one such study covered in this meta-analysis, a freely timed visual signal sequencing task was used alongside fMRI to find that ADHD children had lower parietal activation levels than those without ADHD (Mostovsky et al., 2006). Two studies using delay tasks of second intervals measuring supra-second sensorimotor anticipation showed reduced activation in the caudate, which is involved in sensorimotor anticipation (Wiener et al., 2010a; cited by Hart et al., 2012), and in the anterior cingulate cortex (Rubia et al., 1999; cited by Hart et al., 2012) in the brains of children with ADHD compared to those without ADHD. Reduced activation of the anterior cingulate cortex, a brain region responsible for mediating cognitive influences on emotion (Stevens et al., 2011), could point towards ADHD individuals having reduced ability to regulate affect, also possible

leading to increased impulsiveness. Hart et al., 2012 also found that ADHD patients had consistently higher levels of activation in the precuneus and posterior cingulate cortex (Hart et al., 2012). The posterior cingulate cortex is involved in the brain's default mode network (DMN), and the precuneus is involved with a great many things, among them self-referential memory and thought. ADHD patients having higher activation in these areas could mean that the ADHD brain easily slips into a state of getting "caught up in" (Brewer et al., 2013) one's own mental state or memories, and has a more difficult time deactivating these kinds of thoughts (Hart et al., 2012).

Working Memory Differences

Working memory, formerly known as short-term memory, is the stage of memory occurring between sensory perception and long-term memory. According to the Multicomponent Working Memory Model, the overarching umbrella of 'working memory' is comprised of four subcomponents: the phonological loop, the visuospatial sketchpad, the central executive, and the episodic buffer (Chai et al., 2018). With ADHD greatly hindering central executive capabilities, it makes sense that working memory capacity would be lower for those with the diagnosis. However, in one 2013 study by Ko and colleagues, the researchers wanted to see how phonological and visuospatial working memory differs between ADHD and non-ADHD populations (Ko et al., 2013). To activate the brain regions responsible for these processes, they used an *N*-back task, a widely used short-term recollection task used to assess working memory. The *N*-back task involves the serial presentation of a stimulus such as a shape spaced several seconds apart. The participant must decide whether the stimulus being shown matches the one displayed *n* trials ago, where *n* is a variable number (Au et al., 2015). To trigger the phonological loop, participants were asked to mentally read all shown numbers in the task. The act of verbally

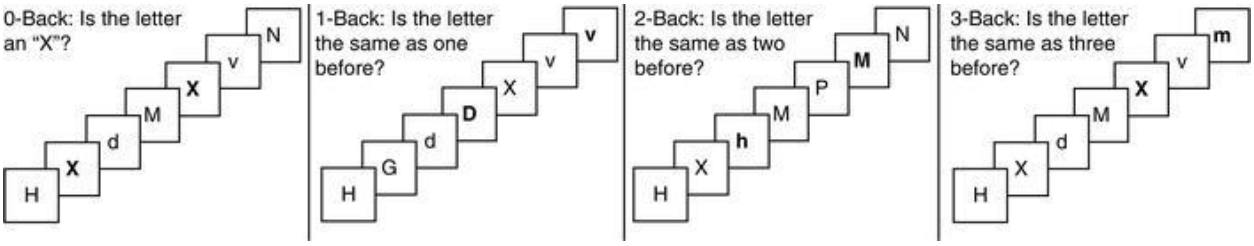

Figure 3: Depiction of the typical N-back task (Sweet, 2011).

imagining the name of the number being shown is thought to activate the phonological loop. To activate the visuospatial sketchpad, participants were shown several stimuli located on the vertices of a hexagon; the ability to remember which vertex a target appeared on was integral to performing well on the task. Between this and the fact that the targets themselves were difficult to distinguish, the participants' visual working memory capabilities were being put to the test. Using fMRI, they demonstrated that both groups exhibited similar brain activation in areas responsible for both phonological and visuospatial working memory (areas including the bilateral precuneus, inferior parietal lobe, DLPFC, SMS/pre-SMA, caudate, and inferior frontal lobe/insula) during the easier 2-back task, with the ADHD group having higher activation intensity (Ko et al., 2013). When it came to the harder 3-back task, however, the control group exhibited significantly increased activation over the bilateral DLPFC, and right SMA, insula, and anterior cingulate, while the ADHD group exhibited increased brain activation significantly over the right DLPFC, but reduced activation for the left precuneus, SMA, and insula/inferior frontal lobe (Ko et al., 2013). They interpreted this to mean that ADHD adults spend more mental energy trying to focus on simpler phonological working memory tasks, while brain activation of the left frontal parietal network is impaired when it comes to more difficult working memory tasks (Ko et al., 2013). A similar study was already run by Silk & colleagues in 2008, in which working memory deficits in ADHD were assessed using Raven's progressive matrices (RPM) task (Raven, 1989) and functional fMRI. Raven's progressive matrices task was selected because

it heavily involves visuospatial processing and problem solving and engages the prefrontal cortex, as demonstrated by some recent neuroimaging studies (Christoff et al. 2001; Prabhakaran et al. 1997; cited by Silk et al., 2008). Trials involved a diagrammatic puzzle displaying changes in the horizontal and vertical directions. As shown in Figure 4, participants would have to use trends in the diagram above and select 'what's missing' from options below. In addition to the dysfunctions in the frontal parietal lobe that would later be found in the Ko 2013 study, they found lowered activation in the lateral prefrontal, temporal, striatum, and posterior parietal regions of the brain (Silk et al., 2008). According to the authors, their findings support the existence of a widespread 'functional network' that may be fundamental for visuo-spatial information processing and relational reasoning, and that this network is not activated as broadly in ADHD brain than in non-ADHD ones (Silk et al., 2008).

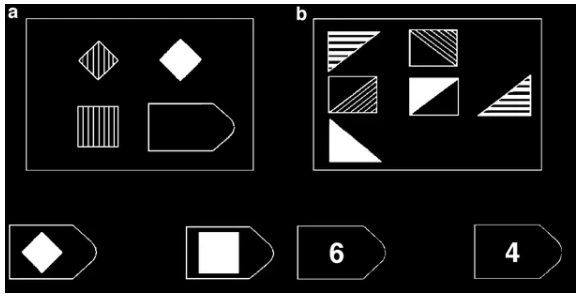

Figure 4: Raven's Progressive Matrices task used in Silk et al., 2008.

Genetic Factors

A 2011 study by Braet & colleagues wanted to investigate how dopaminergic genetic variation affects executive functioning. Executive functioning or control refers to the capacity of the brain, specifically the prefrontal cortex, to exert top-down control on the operations necessary to do what needs to be done. The researchers in this study argue that the DAT1 genotype has a significant statistical interaction with ADHD diagnosis. They used a SART (Sustained Attention to Response Task) in this study, a task designed to measure inhibitory function and sustained attention, two factors thought to play a role in executive functioning.

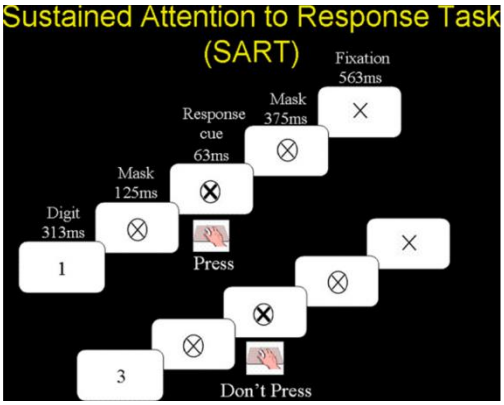

Figure 5: SART task used in Braet et al., 2011.

While the participants were taking the SART, their brains were being analyzed by fMRI equipment to determine which parts of the brain were being used in taking the SART. They found that in the left superior frontal gyrus, the anterior cingulate cortex (ACC), left cingulate gyrus, left caudate, right supramarginal gyrus, right precuneus, right cuneus, and right middle occipital gyrus, genotype interacted with diagnosis (Braet et al., 2011). In these areas of the brain, individuals with ADHD had significant differences in neural activation between the DAT1 and non-DAT1 genotype groups, while individuals without ADHD did not have significant activation differences between DAT1 and non-DAT1 groups. They support this argument by displaying fMRI results and the various tests they ran on these scans. While we do not have a complete understanding of why this interaction occurs, their data supports the growing body of evidence suggesting that ADHD is affected by significant genetic factors.

Discussion

What makes ADHD brains different from non-ADHD brains? As we have reviewed, many things do. The ventral striatum activates differently in ADHD brains, which hinders reward-processing ability. This contributes to the characteristic impulsiveness of people with ADHD, as well as to the tendency to get distracted easily. If you are supposed to be working on a major assignment that will greatly impact your grade in a class, but your friend invites you to play video games with him, your brain should ideally do a quick reward-calculus: Do you work on your assignment and ensure that you do not fail the class, or play video games and have fun for a few hours? One of these clearly has a much greater reward than the other. However, to the

ADHD mind, this becomes very unclear. Consider the immediate hedonistic value of each option: do work (not fun) or play video games (fun). In the mind of someone who is hardly able to take long-term rewards into consideration, suddenly the second option becomes much more appealing than the first. This phenomenon is referred to as 'temporal reward discounting', in which the subjective value of a reward decreases the longer one must wait for it (Critchfield & Kollins, 2001, cited by Scheres et al., 2008). Humans are constantly trying to make decisions like this, and ADHD people are constantly having a harder time picking the correct option. This does not mean that they are incapable of doing so, but the extra difficulty associated with making decisions like this have been shown to adversely affect the educational outcomes of young people with ADHD. A 2013 study comparing the educational outcomes of young adults with and without ADHD found that individuals with ADHD are 3 times less likely to hold undergraduate degrees and 90 times less likely to hold graduate degrees than non-ADHD individuals (Kuriyan et al., 2013). This almost certainly happens because schools fail to engage these highly distractible people enough to make them want to pursue higher levels of education. The reason why this is a problem seems obvious: If ADHD people are dramatically underrepresented in higher education, they are earning less money on average than non-ADHD people, all because processes in their brain do not work as well as they do, something entirely out of their control. As mentioned before, they can still choose to focus on school, but it will be more difficult for them.

On another note, the findings of Hart & colleagues (2012) regarding affect impulsiveness, affect regulation, and the default mode network subjectively make a lot of sense to me as someone with ADHD. As discussed earlier, the higher levels of activation in the precuneus and posterior cingulate cortex could possibly indicate problems with the brain's default mode

network in ADHD brains, problems involving impulsively slipping into a prolonged period of self-reflection that is in no way relevant to the task at hand and is usually detrimental to one's long-term goals. Until I read these articles (Hart et al., 2012; Brewer et al., 2013), I had no idea how precisely the scientific findings on ADHD could explain problems that I struggle with every single day. Take this scenario: I have been preparing for a crucial exam that is only hours away, an exam that will determine the grade I get in a class, a grade that could very well impact my chances of getting into college, or getting into grad school, or winning a scholarship, or any number of things. In this scenario, a song in my music rotation could start playing, a song that takes me back to a certain part of my life, and immediately I am struck with the desire to compile a playlist of my favorite songs from that period in my life. This impulse wipes away all urgency towards the impending deadline, and suddenly I am spending hours on end engaging in a self-reflective activity that is nowhere near as important as the thing I *should* be doing. My brain is not doing a very good job of regulating the emotional affect of these songs, it fails to prioritize the more essential task looming ahead of me (it is farther away than the task of compiling a playlist of songs to bring me back to days long gone), and in doing so, thanks to my hyperactive default mode network, I get trapped in a what feels like a 'prison in my own mind,' too caught up in self-referential thoughts to do anything productive at all. This is the type of everyday struggle that compelled me to write this thesis in the first place. I would imagine that other people with ADHD experience these kinds of struggles as well. To the reader still unsure about whether attention deficit hyperactivity disorder is simply 'kids being lazy:' I implore you to consider all the research I have compiled and synthesized in this thesis and realize that this is most certainly not the case.

Limitations & Strengths of This Study

The greatest strength of this thesis is its unique synthesis of the ways ADHD was viewed throughout history and a systematic meta-review of the recent and current research in the field. While this study does not provide new primary data to the field of ADHD research, it does provide a historical look at the ways the disorder now known as ADHD has been treated since it was first approached scientifically. It also looks at how modern neuroimaging and genetic techniques have provided numerous neurological and biological bases to a disorder previously thought to be rooted in character or will. Furthermore, unlike most ADHD studies, this one was written by someone with an intimate understanding of what it is like to experience the struggles associated with the disorder. This makes it easier to connect the dots between various mental processes impacted by neurological deficiencies.

Conclusion

ADHD is not a matter of character. It is not a defect of moral quality, nor is it rooted in personality. Every so often, a talking head gets on TV; publishes a book asserting that ADHD is a myth, and far too many people believe what they people say. A 2019 survey found that 70% of Americans believe that ADHD is over-diagnosed, with 78% believing that ADHD is more common today that it was twenty years ago (in 1999) (MerckManuals.com, 2019). As discussed earlier in this paper, it has not yet been twenty years since the scientific view of ADD was fully replaced by ADHD and its different subtypes, so of course it is more common than it was twenty years ago. Furthermore, 58% of Americans believe that this is caused by increased use of technology, with this percentage increasing among older generations (MerckManuals.com, 2019). This argument is most likely rooted in ignorance of the topic, choosing to employ 1840s ‘Zappelphilipp’-era character judgement to a disorder that has been empirically measured to involve neural activation differences in myriad brain regions.

Understanding the historical context of ADHD research is important because it helps people realize that it is not something that was invented in the 1990s to justify kids wanting to play video games. In 1798, Crichton knew that there was a biological basis for this disorder. What we now know of as ADHD has been known to the scientific world for over three hundred years, and even though researchers in the past might have been further from the truth than we are now, it grounds the disorder in a legacy of scientific development. To this end, I have composed a unique synthesis of the history of the understanding of the diagnosis and the modern-day neuroimaging work being done to discover more about the neurological underpinnings of ADHD.

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