Inhibition in Attention Deficit Hyperactivity Disorder

Jared Bubb

Doctoral thesis

Department of Psychology

University of Essex

November 15, 2023

Acknowledgements

I am deeply grateful to my supervisor, Dr Geoff Cole, for all his help and advice with this doctoral thesis. I would also like to thank my wife and family because this would have been impossible without their support and patience. Lastly, I would like to thank the school, parents, and students for participating.

Abstract

Attention-deficit hyperactivity disorder (ADHD) is a developmental condition estimated to affect approximately 5.9 to 7.1 per cent of children and 5.29 per cent of adults worldwide (Willcutt, 2012). Whilst the aetiology of the disorder is unknown, ADHD is characterised by attention deficits, hyperactivity, and impulsivity. These symptoms often significantly impair an individual's functioning in the domains of school, employment, home life, and general social settings.

In this thesis, I investigated the effect of dysfunctional inhibition in individuals with ADHD. Specifically, bottom-up processes, such as motor inhibition, motor control, and attentional inertia, were investigated to see to what extent these processes are affected by inhibitory dysfunction. This essential process is impaired in individuals with ADHD. This was accomplished by a battery of tasks, including a motor inhibition task, two motor skills tasks, and an attentional inertia task. These tasks were indexed to a Stroop task to investigate the possibility of a general inhibitory dysfunction in both top-down and bottom-up processes. In addition, the motor inhibition and Stroop task were indexed to self-inventories commonly used to identify individuals with ADHD.

Across these eight experiments, adolescent ADHD individuals, age-matched controls, and adults undertook various tasks designed to index automatic bottom-up motor inhibition. Results showed that, compared with controls, ADHD individuals did not exhibit usual levels of inhibition. However, higher-level cognitive inhibition, as measured with

Stroop, was comparable with controls. Results also revealed a positive association, but not a statistically significant one, between the degree to which a person exhibits ADHD-like behaviour and the degree to which they lack automatic motor inhibition. I will later show that it is due to a specific diagnostic construct of ADHD that does not include motor difficulties. These data suggest that bottom-up inhibitory motor processes are an essential component of ADHD. I will argue that including dysfunctional motor inhibition complements current ADHD models, particularly those developed by Barkley (1997) and Nigg (2001).

Chapter 1

Introduction

Prior ADHD research has given rise to models attributing the dysfunction associated with ADHD to atypical executive functions. These models, which will be explored in more detail later, focus on the extent to which the executive functions of working memory, attention and inhibition contribute to the symptoms of ADHD. The executive functions listed above are considered top-down functions/high-ordered cognitive abilities (Cristofori et al., 2019).

This thesis primarily investigated bottom-up processes. The definition used in this thesis for bottom-up processes is any real-time or unconscious processes that interpret sensory information. Further, these processes do not require prior knowledge or experience. They are characterised as a process that involves the information travelling "up" from the stimulus generated by the senses to the brain, which interprets them passively (Gibson, 1966). Bottom-up processes begin with retrieving sensory information to build perceptions based on current sensory information input (Gibson, 1966).

Bottom-up processes can be contrasted with top-down processes. Gregory (1970) defined a top-down process as one that processes the world around us by drawing from what we already know to interpret new information. Gregory (1970) argued that a constant stimulus stream would overload the brain's processing power. Schemas are constructed from past experiences, prior knowledge, emotions, and expectations to use the data stream to hypothesise the new information. Processes that use schema are characterised as top-down in this thesis.

This research explored the use of novel measures of inhibition of bottom-up processes. This was done to investigate whether giving more weight to the inhibition of bottom-up processes was warranted in understanding ADHD. The intention is that this would complement existing models of ADHD that recognise dysfunctional inhibition as a driver of the disorder. One such model is Barkley (1997), who developed the Self-Regulation model, with behavioural inhibition playing a central role.

A Personal Motivation

Between 1999 and 2020, the present author was a special education teacher and then head teacher working with children in residential special schools. These settings were populated with children diagnosed with Attention Deficit Hyperactive Disorder (ADHD). Students often exhibited extremely challenging behaviour that seemed unprovoked and impulsive. I was physically attacked by pupils who got along with me and expressed a secure attachment towards me and the other staff. These attacks happened often, but one memorable case early on in my career stood out and created a particular interest in the inhibition topic. Whilst ending a lesson before lunch, I reached over to a light switch near a seated student to turn off a light. To my surprise, I was stabbed with a pencil about four inches deep into the side of my stomach, an action that required a considerable amount of force to accomplish. The student who stabbed me had ADHD and instantly broke into tears and began to apologise, as bewildered by their behaviour as I was.

It is clear to me now that the impulsive, violent behaviour I witnessed resulted from two separate brain systems, as described by Kahneman (2000). Kahneman (2000) argued for a slow-thinking cortical system that “knew” that this behaviour was socially

unacceptable but was too slow to regulate the fast-thinking motor system that was not being inhibited. This kind of fundamental dichotomy is a central feature of the brain. For example, there is a non-conscious emotional recognition system and a conscious one (Chien et al., 2016). Blindsight, in which the conscious visual experience is abolished but the motor system which acts is intact, would be another example (Tong, 2003). My experiences within the school setting suggested to me the possibility that complete top-down cognitive control, specifically using inhibition to regulate actions, may be an illusion. At the very least, I believe it is worthy of empirical investigation.

As a special education teacher with over 20 years of experience, I have encountered numerous children with ADHD. It always seemed interesting that two categorically different traits or pathological constructs, inattention and hyperactivity, were linked together in a single disorder. Initially, my interest in ADHD was motivated by the desire to help students who suffered from the devastating effects of ADHD that Barkley (1997) attributed to behavioural inhibitory dysregulation. Barkley (1997) stated that behavioural inhibitory dysregulation has a detrimental impact on educational and social outcomes. Barkley (2001) hypothesised that behavioural inhibitory dysregulation resulted from the dysfunctional management of attention and hyperactivity. The empirical experiments of this thesis intend to demonstrate that dysfunction across a broader range of tasks than Barkley (1997). This expansion will complement his inhibition model of ADHD by including more motor tasks, which I describe as bottom-up, and indexing them with known cognitive tasks (Stroop), which I define as top-down. By expanding the range of tasks and inhibitory processes not addressed directly by Barkley (2001) or Barkley (1997), I will argue that inhibitory dysfunction plays a more prominent causal role in the symptoms of ADHD

and will later argue for a broader diagnostic construct of ADHD that is weighted in such a way that reflects a more comprehensive range of symptoms.

At the beginning of my career as a special educator, I investigated ADHD, hoping to learn practical ways to help students. I quickly learned that there is an abundance of information available to front-line educators about ADHD that is not based on peer-reviewed science and that I would need to be critical in my approach.

With this PhD, I aimed to empirically explore some of my experiences under more controlled circumstances and rigorously challenge some assumptions I, or others, may have made regarding the role of inhibition in individuals with ADHD. I also intend to build upon the inhibitory model of ADHD described by Barkley (1997) to include a broader range of motor tasks which show a growing importance for bottom-up forms of inhibition in the symptoms of ADHD.

Attention Deficit Hyperactivity Disorder (ADHD)

The Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5; American Psychiatric Association, 2013) is a widely accepted diagnostic manual used by clinicians and researchers to classify mental disorders, including ADHD. What we now call ADHD has been known by many other names in the past: minimal brain dysfunction DSM-I (1952), hyperkinetic reaction of childhood DSM-II (1968), attention-deficit disorder with or without hyperactivity DSM-III (1980). The exact term ADHD was adopted in 1987 with the release of DSM-III-R. In 1994, with DSM-IV, ADHD was split into three subtypes: ADHD inattentive, ADHD hyperactive-impulsive, and ADHD combined. The subtypes continue to be found in DSM-5-TR (2022) following some revisions of the 5th edition.

ADHD is a neurodevelopment disorder characterised by high levels of hyperactive and impulsive behaviours coupled with inattention and trouble focusing. The DSM-5 and the ICD-11 from the World Health Organization (2022) use a similar diagnostic criterion that lists symptoms in three presentations that a person can possess to be diagnosed with the disorder. The presentations are a collection of similar symptoms that correspond to the three subtypes of ADHD, namely, Inattention, hyperactivity-impulsivity, and combined. In the DSM-5-R, children must have six symptoms from the inattention and hyperactivity lists, while adults need only five symptoms from each list. The symptoms of inattention include overlooking careless mistakes, difficulty maintaining focus on tasks, not following verbal instructions, not finishing tasks, difficulty with organisation, reluctance to engage in attention-heavy tasks, frequently losing items, often being distracted by extraneous stimuli, and forgetting daily activities. The symptoms of hyperactivity-impulsivity include fidgeting, trouble staying seated, running or climbing in inappropriate situations, inability to engage in leisure activities, seeming “on the go”, talking excessively, answering questions before the question is finished, struggles to wait their turn, and frequently interrupts. Children with this disorder find school difficult, have difficulty attending to tasks, are forgetful, easily distracted, and find it challenging to sit still, often interrupting others (DuPaul et al., 2011).

The prevalence of ADHD within the general population is estimated to be between three and seven per cent, with males showing a significantly higher ratio of prevalence than females at 3:1 (Szatmari, 1992). The average child is diagnosed at seven (Danielson et al., 2018). Sixty-four per cent of children with ADHD have at least one other disorder. For example, 52% have behaviour or conduct difficulties, 33% have anxiety, 17% have depression, 14% have autism spectrum disorder, and 1% have Tourettes’s syndrome

(Gnanaval et al., 2019; Danielson et al., 2018). This level of comorbidity may suggest a common aetiology of ADHD with other neurological disorders. This thesis excluded all students with known comorbidities from the study after examining school records.

Children with an ADHD diagnosis form significant comorbidity with children with additional special needs: 10% to 25% of children with a learning disability also have ADHD (Schnoes et al., 2006); 25% to 44% of children with an emotional disturbance also have ADHD (Schnoes et al., 2006); 43% to 93% of children with opposition defiant disorder have ADHD; with a further 13% to 51% of children with anxiety disorders, and mood disorders having ADHD (Bird et al., 1994; Jensen et al., 1997; Barkley, 1997).

The symptoms for diagnosis of ADHD fall into two broad categories -- inattention and hyperactivity/impulsivity (Kooij et al., 2010). In most countries, three descriptors are crucial to diagnosis - poor sustained attention, impulsivity, and hyperactivity (Kooij et al., 2010). As defined by The *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM–5*; American Psychiatric Association, 2013), current diagnoses must be made with the addition of the following factors: symptoms persist for more than six months, must be to a greater severity than what would be age-appropriate, and are coupled with significant difficulties in two settings, typically school and home. The DSM-5 further states that ADHD can be seen as a spectrum with two extremes: hyperactivity and inattentiveness, with individuals having symptoms of both being somewhere in between. This results in three main subtypes with emphasis on predominantly inattentiveness (ADHD-PI), predominately hyperactive (ADHD-PH), or a combination of the two (ADHD-C). ADHD-C is

estimated at around 70% for children and adults (Salvi et al., 2019). Children with a known subtype other than ADHD-C were screened from the study.

Neurological Profile of ADHD

Pievsky & McGrath (2018) conducted a systematic review and quantitative summary of those meta-analyses to determine the extent to which individuals with ADHD differ cognitively from typically developing controls. They collected 253 separate standardised mean differences from 34 meta-analyses. From that collection, 244 (96%) were positive, indicating better neurocognitive performance in the control group than in the ADHD group. The mean effect size was 0.45 (SD = 0.27). Unweighted means of standard mean differences for neurocognitive domains ranged from 0.35 (set shifting) to 0.54 (working memory).

Pievsky & McGrath (2018) then weighted the studies and aggregated them to find means from 0.35 (set shifting) to 0.66 (reaction time variability). Furthermore, they included some prominent findings from a variety of neurocognitive domains when weighted by the number of aggregated studies from the domains with mean effects over 0.50, This list included reaction time variability (0.66), intelligence/ achievement (0.60), vigilance (0.56), working memory (0.54), and response inhibition (0.52). Age moderated the relationship between ADHD diagnosis and neurocognitive functioning, with greater between-group differences among children and adults than among adolescents. The evidence suggests that ADHD is associated with substantial deficits across various neurocognitive domains.

These finding are of particular relevance to the experiments of this thesis. It should be expected that ADHD groups should have deficits in reaction times, and response inhibition.

History of ADHD

What we today call ADHD was first described by the Scottish physician Alexander Crichton in 1785, who referred to an “Incapacity of attending with a necessary degree of constancy to any one object” (Lange et al., 2010). German physician Heinrich Hoffman added the kinetic component with his descriptions of “Fidgety Phill”, who had symptoms of inattention. In his 1902 lectures, British paediatrician George Still developed our modern scientific understanding of ADHD with a more rigorous and less anecdotal definition. He still referred to it as “an abnormal defect of moral control in children” unrelated to cognitive ability (Still, 1902). The first medical treatment for ADHD occurred in 1937 when Charles Bradley gave the stimulant Benzedrine to children with headaches who also had behaviour problems. Employing the rationale that the headaches caused behaviour difficulties, Bradley (1937) found that the headaches remained, but the behaviour improved. He also reported that Benzedrine had a positive effect and improved the behaviour of half of the children via a reduction in motor activity. Bradley also said the paradoxical effect of a stimulant is potentially promoting the inhibition functions of the central nervous system, with the result being an improvement in voluntary control for the child. Unfortunately, these findings were ignored as the prevailing view at the time was that behaviour disorders had no biological basis (Cyr & Brown, 1998).

In 1954, the stimulant Methylphenidate was marketed as “Ritalin” by the Ciba-Geigy Pharmaceutical Company and, in 1955, was approved for sale in the United States for use with patients who had chronic fatigue, lethargy, depressive states, and narcolepsy (Lange et al., 2010). Renewed interest in using stimulants to treat mental health disorders

began in the late 1950s. For example, Laufer et al. (1957) showed that using stimuli, including the new Methylphenidate drug, reduced the symptoms of hyperkinetic impulse disorder (i.e., AHDH) in children. Successful treatment with stimulants would prove pivotal to the possible aetiology of ADHD, which will be reviewed later in greater detail. The use of Benzedrine became less frequently used during the 1960s due to its addictive properties, with Methylphenidate becoming the most common drug used for the treatment of the disorder (Sinzig et al., 2007).

Despite the finding of an effective treatment, the aetiology of ADHD is not well understood (Thapar et al., 2013). The neurobiological aspects associated with its cause have three pathological components: differences within the structures of the brain (Grace, 2001; Sagvolden et al., 2005; Volkow & Li, 2005), differences in how the brain functions (Lanciego et al., 2012), and the role of neurotransmitters within those systems (Volkow et al., 2007; Mehleir-Wex et al., 2006).

In the early 20th century, it was proposed that differences in brain structure, via brain damage, caused behavioural difficulties, and, specifically, minimal brain damage was considered the aetiology of ADHD in children (Kessler, 1980). Still and Tredgold (1908,1917) separately conducted research with children who suffered from encephalitis. Tredgold (1908) concluded that “gross lesion of the brain and a variety of acute diseases, conditions, and injuries that presumably resulted in brain damage” was the cause of the symptoms that are now recognised as ADHD (Ross & Ross, 1976). Research by Kramer and Pollnow (1932) established a causal connection between brain damage and deviant behaviour, demonstrating that children with brain lesions could not complete academic tasks or

continue concentrating on complex tasks when a distracting stimulus was present. This research helped to establish the diagnostic criteria of inattention that was later conflated into the diagnostic criterion of hyperactivity. Kramer and Pollnow (1932) contributed to the prevalent view that brain damage was the cause of hyperkinetic disorder (i.e., ADHD; Ross & Ross, 1976). A flurry of research in the 1930s and 1940s found links between other sources of brain damage --infections, lead toxicity, epilepsy, and frontal lobe ablation -- that caused the damage that would result in ADHD (Barkley, 2006). Knobloch and Pasamanick (1959) proposed a spectrum of damage, from severe to minimal, with corresponding disorders attached to levels of severity with minimal damage that is difficult or impossible to observe being responsible for ADHD (Barkley, 2006). This view became so prevalent that by the 1970s, it was assumed that some form of brain damage, though unseen, must exist following a diagnosis of ADHD.

Critics had begun to challenge brain damage as the sole cause of ADHD in the 1960s. The successful use of amphetamines undermined both the idea that brain damage caused ADHD as well as environmental explanations like social factors or non-biological explanations that prevailed with psychoanalysis at the time (Herbert, 1964; Rapin, 1964; Laufer & Denhoff, 1957; Clements & Peters, 1962). Amphetamines successfully treated children, regardless of lesions, backgrounds, or unknown causes. It was argued that some other unidentified process was being supported that alleviated the symptoms of ADHD (Herbert, 1964; Rapin, 1964; Laufer & Denhoff, 1957; Clements & Peters, 1962)

By the late 1960s, a growing consensus had been formed that led to the *Oxford International Study Group of Child Neurology* and *The National Institute of Neurological*

Diseases and Blindness, two prominent research groups that developed diagnostic guidelines at the time, to challenge brain damage as the cause of ADHD (Lange, 2010)

Hereditability of ADHD

Genetic causes of ADHD via brain damage were investigated in the 1990s. ADHD was found to be highly heritable (Neale, 2010; Faraone, Doyle, Mick, & Biederman, 2001; Gillis, 1992). Using bivariate genetic modelling, it was found that ADHD-PI was 88% heritable and ADHD-PH was 79% heritable (Mcloughlin et al., 2007). Mcloughlin et al. (2007) further suggested that multiple genes play a role in the disorder and its two main subtypes. However, the authors also found evidence that some additional individual genes play a role in only one of the two subtypes, opening the possibility that the two variants are genetically separate. Genetic studies have found the gene markers DAT1, DRD4, DRD5, 5HTT, HTR1B, SNAP25 are associated with ADHD (Gizer, Ficks, & Waldman, 2009).

Stevens et al. (2007) found evidence that the environment can cause the symptoms of inattention and overactivity in instances of early and severe deprivation. These symptoms were found in children who had suffered extreme levels of deprivation in early childhood before the age of 3½ years, thus suggesting that during critical periods of development, a 'pathway to impairment' may be possible. These findings came from a study of 52 children adopted into the UK from Romanian orphanages that suffered institutional deprivation for at least six months, leaving open the possibility that environment, particularly traumatic events, can have a lasting effect that resembles ADHD (Stevens et al., 2007).

Models of ADHD

Currently, five main models attempt to explain how ADHD functions: state regulation theories, executive dysfunction theory, dynamic developmental theory, self-regulation, and delay aversion/dual pathway theories. Inhibition plays a role in all five main models that explain the dysfunction associated with ADHD.

State Regulation Theory/Cognitive Energetic Model.

The state regulation theory was developed by Sanders using the cognitive, energetic model in the 1980s (Sanders, 1983). In this account, a well-regulated cognitive function is in a state of optimal energy management. Actions are completed in fundamental cognitive stages requiring mental energy. These stages are --stimulus encoding, memory search, binary decisions, and motor preparation (Sternberg, 1969). Arousal is the initial response to a stimulus, dissipating relatively quickly. After arousal, this information may become available for action preparation. If an individual uses mental effort, this information is used in preparation for action and can be retained over a long period.

Sanders (1983) stated that processes are allocated resources based on incoming stimuli. When a stimulus lacks the strength to maintain the individual's attention to a task, an evaluation mechanism "decides" if more mental effort is needed. The mental effort could increase activation levels or inhibit activation levels. This effort is the currency of the evaluation mechanism, which observes the present momentary state of the arousal and activation levels, spending effort as needed to regulate normal cognitive functions.

The state regulation theory explains the symptoms of ADHD as an impairment in the evaluative mechanism. Evidence to support state regulation theory came from using

Sternberg's additive factor method to collect reaction times under different speeds and cognitive loads (Sternberg, 1969; Sergeant, 2005). Performance on slowly administered tasks decreased performance in children with ADHD, resulting in higher error rates. Additionally, children with ADHD improved to a level comparable to children without ADHD when the time between tasks decreased (Sanders, 1983).

The cognitive, energetic model predicted that event rate influences motor activation levels. Event rate measures how often an exact task can be completed in succession in a set amount of time. Sanders (1983) stated that in typical individuals, as the event rate increases, less effort is required to complete the experimental task, measured as a response time. The pool of "attention" available for the typical individual should be adequate to have consistent response times. If ADHD is a dysfunction linked to a smaller pool of attention, tasks should have slower response times towards the end of the series of repeated tasks (Saunders, 1983). However, Saunders (1983) found that children with ADHD have little difficulty with task response times when the speed of activation levels increases. Sauders (1983) argued that this evidence showed that errors and inattention are not due to an inadequate pool of mental effort to draw from. Saunders (1983) also showed that as interval times between tasks slowed, the ADHD children had disorganised or deficient response organisation, making it difficult to access the static pool of mental resources. Sauders (1983) attributed these slower response times to an inability to inhibit extraneous stimulus when given more time between tasks. Typical participants with intact inhibition used less mental effort overall and used the larger interval between tasks for top-up functions that recharged the attentional pool.

Further studies have shown an inverted “U” function in the relationship between increases and decreases in preparation for action and performance in children with ADHD. Sonuga-Barke (2002) increased the interval between tasks and duplicated the results that showed that children with ADHD cannot inhibit extraneous stimuli. Still, if the time between tasks was shorter, these same children had errors in the tasks. Children with ADHD were found to have problems with response inhibition in fast and slow conditions but performed equally well as controls in medium conditions (Sonuga-Barke, 2002). Sonuga-Barke (2002) stated that these results meant that a capacity to inhibit remained; however, optimal inhibition of extraneous stimulus occurred without difficulty for children with ADHD when intervals for tasks were not in the medium term.

Model of self-regulation

According to Barkley’s model of self-regulation, the core deficits of ADHD are impulsiveness and inattention. The model argues from an evolutional perspective. Barkley (2001) views executive functions, including inhibition, as forms of behaviour-to-the-self that evolved from overt (public) to covert (private) responses as a means of self-regulation.

Barkley stated that executive functions, including inhibition, “is necessary given the interpersonal competition that arises within this group-living species”. Particularly in ADHD, impulsiveness is a failure to inhibit behaviour, and inattention is a failure to focus on relevant information and filter out task-irrelevant information. Suitable regulation of impulsiveness and attention are seen as evolutionary advantages, as well as most executive functions, and a result of the evolutionary pressures of a social organism. Barkley demonstrated that these core deficits are due to problems regulating behaviour, emotions, and cognition due to unidentified dysregulation within the brain's central executive. His research found that

children with ADHD have deficits in behavioural inhibition (Barkley, 1997; 1997b, 2001), working memory (Barkley, 1997; 1997b, 2001), and self-regulation of affect (Barkley, 1997; 1997b, 2001).

Barkley (1997) posits that ADHD is a disorder of impulsivity and self-control. Barkley further argues that the disorder results from deficits in the control of executive functions that typical people often use to regulate their emotions and actions. The regulation of emotions and actions is accomplished by what Barkley (1997) referred to as behavioural inhibition. Behavioural inhibition is a resource used to regulate prepotent responses actively, interrupt an ongoing response, and control interference at the start of a neurological chain of processes. The dysfunction associated with the symptoms of ADHD is derived from the reduced ability of an individual's inhibitory power over the next set of four main executive functions in the chain. Barkley (1997) names the four main executive functions: working memory, self-regulation of effect, internalised speech, and reconstruction/behavioural analysis and synthesis. Barkley (1997) defines working memory as a type of nonverbal operation memory that allows an individual to retain information whilst working on a task after the stimulus has disappeared (Barkley, 2001). Within this definition of working memory, Barkley (1997) nested the following sub-functions within working memory. The first is the manipulation of events and actions in the mind. When this manipulation is done with memories of past events, Barkley (1997) calls it hindsight or retrospective function. When this is done to anticipated actions in the future, it is called prospective function or forethought. Behavioural inhibition temporarily regulates the individual's self-awareness through the correct synthesis of hindsight and forethought.

Individuals find regulating working memory difficult due to inhibition and often “get it wrong” with their behaviour, particularly in social situations (Barkley, 1997).

In contrast to the non-verbal working memory, Barkley (1997) described internalised speech as a verbal working memory that allows an individual to use internalised speech to be descriptive and reflective on past behaviours. This is accomplished through verbal self-questioning, problem-solving, understanding rules and following instructions. Dysfunctional behavioural inhibition results in a lack of understanding of rules and adherence to instructions.

Self-regulation of affect, motivation and arousal is defined by Barkley (1997) as the ability of an individual to self-regulate their affect/overt behaviours whilst simultaneously being objective when taking the perspective of others. This function regulates motivation and arousal to stimulus, particularly when in the service of goal-directed actions. Barkley (2010) used meta-analysis to show that Individuals with ADHD have difficulty inhibiting their affect and often have hyperactive motor movement and emotional outbursts. Finally, arousal to stimulus is not regulated in a typical way, making it difficult for individuals to know what stimulus is essential for goal-directed tasks.

As defined by Barkley (1997), Reconstitution is the capacity of an individual to analyse and synthesise their behaviour from the interpretation of the observations of their own and others' behaviour. Barkley (1997) further argues that reconstitution allows individuals to acquire behavioural fluency by mentally simulated rules to predict a social outcome.

Behavioural inhibition successfully regulates neurological inputs into the four executive functions, contributing to the correct goal-directed outputs in motor control,

verbal fluency, and syntax. Barkley (1997) describes the output of these executive functions as the ability to inhibit task-irrelevant responses, execute goal-direct responses, execute novel or complex motor sequences, engage in behaviours that promote goal-direct persistence, re-engage after a disruption, and have sensitivity to motor responses.

I tested the ability of ADHD individuals to inhibit task-irrelevant responses with the Stroop Task. ADHD participants could execute novel motor sequences compared to their non-disabled peers when completing the motor inhibition and dice-rolling motor tasks. The attentional inertia task tested the behaviours that promote goal-directed persistence. The motor inhibition task is also a measure of the sensitivity of ADHD students to motor responses.

Executive Dysfunction Theory.

The executive dysfunction theory proposes that ADHD is a symptom of dysfunctional administration of executive control over higher-order cognitive functions caused by an impairment to the normal neurological functioning of the fronto-parietal and frontostriatal neural networks (Willcutt et al., 2005). This impairment results in deficits in higher-order cognitive processes – planning, sequencing, attention, working memory, and inhibition – that control low-level cognitive processes, such as language, perception, explicit memory, learning, and action. These impairments have been observed directly, using fMRI and EEG, and indirectly, via behavioural studies (Booth et al., 2002). Stimulants that act as agonists can reduce the symptoms of ADHD as they promote regular functioning in the dopaminergic and noradrenergic neurotransmitter dysfunction connected with the impairment in these specific neural circuits.

Posner's theory of attention proposed that separate cognitive functions are responsible for alerting, orienting, and executive control (Posner, 1990). There is evidence of dysfunction in the alerting and executive control networks in children with ADHD (Johnson et al., 2008). However, it should be noted that not all children with ADHD have signs of executive dysfunction, and executive dysfunction does not account for the hyperactivity associated with ADHD (Willcutt et al., 2005). Additionally, the root cause of poor performance on neuropsychological tasks might be a motivational or a state regulation deficit that causes a down-regulation of the neural circuits associated with executive functioning. Therefore, the executive dysfunction theory cannot be said to explain all the symptoms of ADHD.

The Delay Aversion Theory/Dual Pathway Theory.

A question most theories around ADHD struggle to answer is the finding that children with ADHD can wait but do not want to (Sonuga-Barke et al., 1992). It was assumed that children with ADHD could not wait, which was related to the trait of impulsivity. However, under different circumstances, Sonuga-Barke et al. (1992) found that ADHD children retained the capacity to wait but were not motivated to wait. This implies that the motivation of children with ADHD is to prioritise immediate rewards but only to reduce delays. This means that an ADHD child is motivated to avoid delay whilst retaining the ability to delay impulses. This motivational source of the impairment contrasts starkly with the cognitive impairment-based explanations. However, this theory has started incorporating aspects of the executive dysfunction theory. The lack of attention and

hyperactivity associated with the disorder are outwardly visible behaviours of the child trying to avert delay when they cannot (Sonuga-Barke et al., 1994).

In the 1990s and early 2000s, Sonuga-Barke et al. (1994) developed the delay aversion theory and proposed a dual pathway explanation for ADHD. The first pathway, citing evidence from brain imaging to develop the executive dysfunction theory, was seen as an essential neurological circuit whose impairment could account for inhibitory deficits linked to the mesocortical dopamine branch. The second pathway explained the delay aversion, which was linked to the mesolimbic dopamine branch.

Sergeant, Geurts, Huijbregts, and Scheres (2003) were critical of this explanation when they developed the cognitive-energetic model, pointing out that "a widely distributed neural network involving frontal, basal ganglia, limbic and cerebellar loci seem implicated in ADHD" meant that the dual pathway theory was too limited in scope. Sergeant et al. (2003) criticised the focus on only two parts of the brain when a growing body of evidence showed a deeper level of complexity and interrelated neural circuits that would need to be addressed. Critics of the theory's delay aversion pathway component pointed out that this pathway is an elaborate explanation that impulsivity is a lack of self-control (Marco et al., 2009). In later studies, Sonuga-Barke, Seargeant, Nigg, and Willcutt (2008) moved away from this pathway by reimagining delay aversion in situations where rewards are ambiguous and rebranding the idea as choice impulsivity.

The Dynamic Developmental Theory

Building on work started in the 1980s by Gray (1982), Gray, Feldon, Rawlins, Hemsley, and Smith (1991) proposed a comprehensive developmental theory of ADHD. This

theory was further developed following research by Sagvolden et al. (2005). The theory attempts to explain ADHD from the level of the individual neurons to macro social settings. One of the strengths of this theory is that it also attempts to explain all three symptoms associated with ADHD (Sagvolden et al., 2005). Due to its behaviourist roots, the theory argues that two behavioural mechanisms explain the symptoms of ADHD. The first is "altered reinforcement of novel behaviour", and the second is called "deficient extinction of inadequate behaviour". Sagvolden et al. (2005) argued that there is a smaller "window of opportunity" for children with ADHD due to lower dopamine levels between the delayed response to stimulus and the reinforcement. The effectiveness of the reinforcer needs to happen more quickly if it is to have any effect in shaping behaviour. This means that children with the condition cannot acquire socially appropriate behaviours through typical timeframes of reinforcement. All symptoms of ADHD are explained through this lens. The child will only experience an extinction of their current behaviours if the reinforcer of their current behaviours is not satiated and improvements are made to dopaminergic systems when new behaviours are reinforced.

This theory has adapted and incorporated findings from the executive dysfunction and delay aversion studies by conflating the attentional, behavioural organisation, motor coordination, nondeclarative habit learning deficits, and delay aversion into a larger dopaminergic, frontostriatal neurological model (Johnson et al., 2009). Also critical to this theory is incorporating behaviourist ideas around reinforcement and how these symptoms play out in a social setting. Reinforcement and extinction processes are thought to be impaired in children with ADHD due to abnormally low dopamine levels, thus providing a neurological and behaviourally linked explanation. Reduced dopamine levels constrain the

typical functioning of the anterior cingulate, dorsolateral prefrontal and motor circuits, acutely affecting behaviours associated with ADHD. The theory explains individual differences between children with ADHD as variability in the reinforcement and genetic predisposition to low dopaminergic levels that result in differences in the size of the window of opportunity for effective reinforcement.

According to Johnson et al. (2009), a strength of this theory is that its two main predictions are readily testable and relate to the three main symptoms associated with ADHD. Firstly, the delay of reinforcement is described as a gradient (i.e., the size of the window of opportunity), with typical children having the largest and ADHD children the smallest. To test this, children with ADHD would benefit from reinforcement that occurs quickly after any task to be effective. Children with ADHD would exhibit hyperactivity through a high number of fast responses, thus showing hyperactivity and impulsivity, and signs of these symptoms would be seen only if reinforcement occurred outside of the window of opportunity. Secondly, the theory predicts a short window of opportunity with a weakened association between reinforcement and response, seen as a deficit in attention. Studies in rats and children have provided strong evidence to support these predictions (Sagvolden et al., 1998; Wultz et al., 2013; Sagvolden et al., 2007; Aase & Sagvolden, 2006).

Theoretical Groundwork for the Aetiology of ADHD

These five models provide the theoretical groundwork for the aetiology of ADHD and share common explanations and evidence of deficiencies in the inhibition of top-down cognitive processes. All five models focus on the role of executive function. They differ on the weighting of the importance of specific executive functions or the order in which executive

functions regulate each other. However, the hyperactivity of individuals with ADHD was not often explained comprehensively. This is something that will be addressed experimentally in this thesis.

Barkley (1997) was cognisant of the lack of explanation for the hyperactivity in the models of ADHD and developed an explanation that motor movements were the result of goal-driven cognitive functions. For example, a child may be given praise in the past for good handwriting. This praise is retained in memory. When asked to write something up in the future, the child remembers the praise and the expectation of neat handwriting. This prior knowledge is synthesised, and then internal speech simulates some outcomes based on experience and predicts that good handwriting would result in a good outcome. This expectation would result in the motor action of neat handwriting. The teacher then praises the child for neat handwriting and reinforces the schema, increasing the likelihood of neat handwriting. In an ADHD child, Barkley (1997) would argue that in any place where a specific executive function was employed for that example, that executive function would either fail or be reduced to such an extent that the ADHD individual would not be able to complete the handwriting to a neat standard, would be frustrated by this, and not feel rewarded for their efforts. Their motor actions would have high levels of randomness that is characterised as an outcome of hyperactivity. When inhibition was absent, hyperactivity was the result, as these "sloppy" movements resulted from a lack of a goal. Barkley (1997) argued that dysfunctional inhibition of executive functions was the source of the symptoms of ADHD. The predictions made by Barkley's model of self-regulation are being tested in this thesis. The empirical work of this thesis aimed to show that inhibition is not entirely dysfunctional in the top-down mode; however, dysfunctional inhibition remains present in bottom-up processes. These dysfunctional bottom-up processes result in hyperactivity, which is not always present in the

diagnostic criteria, resulting in some inhibitory dysfunction associated with the symptoms of ADHD not being measured in the diagnostic process.

Motor Control

Rosenbaum (2010) states that in psychology, human motor control is focused on the functional control of movement and stability, which Rosenbaum refers to as the software of motor control. Motor control involves processes of sequencing and timing, learning new skills, memory systems that form mental representations used in motor control, and states of mind (Rosenbaum, 2010). Within the process of sequencing and timing is an idea called "response chaining". This occurs when a stimulus from a prior movement triggers another movement. Stimulus feedback from these movements shapes the next set of movements in a successive chain. However, successive movements often happen too quickly for some movements to result from feedback. Movements with the same output often have different movements following them on different occasions, and finally, when sensory feedback is interrupted, it does not always stop movements (Rosenbaum, 2010). In these circumstances, the "software" of motor control indicates some other processes coming into play. Not all movements are then under voluntary control.

Mason (2017) defined voluntary motor control as control that is derived from a neural hierarchy that consists of muscle, motor neurons, motor interneurons, central pattern generators, brainstem motor control centres, cortical motor control centres, and two modulator systems; the cerebellum, and basal ganglia. She went on to separate voluntary movement into two parts; one plans the action by associating a goal or meaning behind a movement, which is updated and modulated in real-time based on feedback, and the

second executes the movement. Movement plus meaning/purpose is defined as a voluntary motor action. In other words, a movement to achieve a goal is voluntary.

Central pattern generators are the output manifold that drives simultaneous integrated complex movements (Wojcik et al., 2014). The integration of complex movements becomes possible within the central pattern generators of the brain. Multiple motor interneurons receive input from the central pattern generators as they send instructions to their respective motor neurons, resulting in coordinated and integrated motor movements (Wojcik et al.,2014). The central pattern generators can execute complicated movements and are optimised in making and executing integrated complex movements from the various simple muscle movements. The generators are a repository for motor memories, potentially valuable and necessary for integrated and complex movements (Steuer & Guertin, 2019). After the central pattern generators are instructed to initiate a series of integrated complex motor movements, they send the manifold of actions down the respective motor interneuron lines. In crude terms, the top part of the hierarchy "wants to pick up a glass", whilst the central pattern generators make or select the specific motor memories from the suite of motor memories required to complete the motor movements of grasping a glass, as well as all its associated muscle movements (Steuer & Guertin, 2019; Mason, 2017).

Instruction must come from one of two motor control centres to initiate motor movements. The first is the brainstem motor control centre, and the second is the cortical motor control centre, which includes the somatic motor control centre (Mason, 2017). The distinction between the two types of motor control centres is based on the role that meaning plays in the movements (Ugawa et al., 2020). The resultant movements initiated

within the cortical motor control centre are derived from an activity imbued with meaning. This meets the definition of an action at this point. It follows that any specific action observable and driven by the cortical motor control centres results from a combination of meaning and movement (Mason, 2017). If meaning remains the same between participants, and the mechanisms that drive movement remain the same, then there should be consistency between different people regarding actions (Mason, 2017). For example, children have a consensus view on the colour red or green or if one line is longer than another. Although not a priori or hardwired into the brain, colour identification is possible due to shared experiences between the participants: the acquisition of language and the shared learning of the definition of green or red. Identification of longer lines would also be similar. After one learns the definition of a word like 'longer' or 'shorter', this definition takes on a shared meaning held in common between participants. When a participant sees a longer line, that should generate a consistent motor action with others who share the understanding of a longer line. In our empirical experiments, a visual stimulus would have a dimension of longer or shorter or a characteristic of colour. The participant is instructed to perform a specific motor action when presented with a stimulus. This stimulus would trigger a motor action in the form of a specific key press to match a correct key press to the presented stimulus successfully. At this point in the voluntary motor control hierarchy, the participant can be observed doing an intentional or meaningful motor action.

The cerebellum is responsible for organising different muscle groups so that movement actions are precise and can be accomplished in novel situations (Guertin, 2019). An infinite amount of muscle movement is possible, and the cerebellum can complete the predictive computational load required to drive novel and unique motor actions (Mason,

2017). This is also known as contextual variability (Kielhofner, 2000). To be successful, the cerebellum must be able to respond to changing conditions, which requires the cerebellum to have a learning component (Mason, 2017).

The second modulation system within the voluntary motor control hierarchy is a neurological loop running out of the basal ganglia, containing inhibitory circuits crucial for motor inhibition (Mink, 1996). The structure is also known to chain together complex actions into integrated behaviours (Mason, 2017). The experimental design of the motor inhibition experiment described in Chapter 2 reduces the variables to a simple choice that results in a single motor action driven by the result of choice. Within the voluntary motor control hierarchy, motor action selection is made within the basal ganglia (Kielhofner, 2009). Individual differences in basal ganglia functioning could lead to impairment of the consistent making of motor movements based on meaning (Garcia-Garcia et al., 2018). This is also the centre of inhibitory circuits within this system (Sesak & Grace, 2010). The role of motor inhibitory functions within the basal ganglia will be discussed in greater detail following the overview of the voluntary motor hierarchy.

There are four distinct movement types: passive movement, reflexive movement, stereotyped movement, and self-generated movement (Mason, 2017). The performance characteristics of each type can be analysed, as well as any movement pathology. Close examination of the pathological characteristics can indicate what part of the voluntary motor control could be damaged (Cole & Tufano, 2008).

Passive movement is resistance to outside pressure. If a person is pushed, the resistance to the force to stay upright is passive movement (Kielhofner, 2009). People with passive movement disabilities who are too weak in their resistance to outside movement

are generally linked to disorders centred in the cerebellum. In contrast, passive resistance that is too rigid is thought to derive from disorders derived from the basal ganglia (Mason, 2017).

A stereotyped movement denotes a movement reproduced in a standardised form; walking and standing are examples. However, stereotyped movement can also be pathological, such as repetitive behaviours observed in individuals with autism and ADHD. These movements are encoded at the central pattern generator level (Kielhofner, 2009) and are not considered actions as they are not initiated through the interaction between movement and meaning.

Inhibition plays a role in automatic motor control as well. Bottom-up processes focus on interpreting sensory information in real time (Gregory, 1968). Gregory (1968) found that bottom-up processes are triggered when sensory receptors receive updated sensory information. This information flow starts with a stimulus at the senses flowing through neural pathways to the brain, implying that prior knowledge is not used when generating a response.

Whilst researching eye movements, Jonides (1981) noted that there seemed to be automatic and voluntary control over the eye. This finding would influence the work of Posner and Cohen (1984) when they discovered inhibition of return. They found that responses to a target stimulus can be slower when it appears in the same, rather than a different location to a previous event. Posner and Cohen (1984) defined inhibition of return as "an inhibitory effect produced by a peripheral (or exogenous) cue or target."

Inhibition of return was considered an automatic visual motor process initially described by Posner, but others would find inhibition of return behaviour outside of the

visual domain. Welsh et al. (2005) explored a social aspect of automatic processing. They observed that when two people alternate responses to a target, one person's responses are slower when directed to the exact locations as their partner's previous response. This became known as "social inhibition of return". Inhibition of return is the motor inhibitory effect used in the empirical experiments below.

Review of Motor Skills and ADHD

Kaiser et al. (2015) systematically reviewed the motor skills problems of children with ADHD. They found that there had not been a review on this topic before and that it could provide insight into the relationship between motor skill deficits and attention, hyperactivity, and impulsivity. The review focused on studies examining the motor skills of unmedicated children with ADHD and the impact of medication on motor control. The article selection criteria included studies in English, with participants between six and sixteen years old, diagnosed with ADHD, objectively measured motor skills, included a control group of typically developing peers, and included unmedicated children with ADHD. The review found that over half of the children with ADHD had gross and fine motor skills difficulties. Additionally, children with the inattentive subtype of ADHD had more impairment with fine motor skills, slow reaction time, and online motor control during complex tasks.

To assess gross motor skills and fine motor skills, in 13 studies, a standardised battery such as the (MABC; Henderson & Sugden, 2000), the Bruininks-Oseretsky Test of Motor Performance (BOTMP; Bruininks, 1978) or the Test of Gross Motor Development-2 (TGMD-2; Ulrich, 2000) was administered. Three studies used a neurodevelopmental

examination, such as the Zurich Neuromotor Assessment (Largo et al., 2002) or the Physical and Neurological Examination for Subtle Signs (PANESS, Denckla, 1985). Finally, the remaining studies assessed one motor component, such as balance or sequential opposition of thumb-fingers.

Reaction time and ADHD

The level of sustained attention and motor control can be determined by the time it takes to react. Kaiser et al. (2015) reviewed articles exploring the relationship between reaction time and motor control when participants had ADHD. One such article was Pedersen et al.'s (2004) study, which required subjects to move their dominant leg to the right, middle, or left depending on the stimulus. The reaction time was broken down into two parts: premotor time, which is the time from the stimulus to the muscle reaction, and movement time, which is the time from the muscle reaction to the start of the movement, using an EMG analysis. The study found that children with ADHD had slower movement preparation.

Kaiser et al. (2015) reviewed four studies on the upper limb, which are relevant to the pure motor inhibition task. In this research, simple tasks such as one-finger tapping, simple choice reaction time, or simple graphic task did not show any significant reaction time differences between the ADHD and control groups (Meyer & Terje, 2006; Rubia et al., 2003; Leung & Connolly, 1998; Schoemaker et al., 2005). However, in a sequential opposition thumb-to-finger task, the reaction time for the ADHD group was longer than that of the control group (Klotz et al., 2012). The pure motor inhibition task has similar experimental methodologies to the sequential opposition thumb-to-finger task due to the alternation of finger reaction times being the dependent being present in both the thesis experiment and the reviewed experiments.

Movement Timings

Kaiser et al. (2015) noted that Yan and Thomas (2002) also discovered that the timing of movements in the ADHD group was more variable than that of the control group children. In a tapping task, children with ADHD had slower and more significant variability between sequences (Rosch et al., 2013). However, when performing a synchronised tapping task, ADHD children were more variable than the control group (Rubia et al., 2003). On a tapping task that required strength, the results of children with ADHD did not differ from those of the TD group concerning peak force (Steger et al., 2001). On an aiming task, Yan and Thomas (2002) discovered that children with ADHD were slower than their counterparts when the movement required more complex motor coordination. This result was confirmed by Klotz et al. (2012), who found that the speed was slower for children with ADHD than for TD children on a sequential opposition of thumb to fingers task. The dice-rolling experiment, which combines an element of aim, correct timing, and the successful application of appropriate strength/force, will investigate the impact of ADHD on this task in this naturalistic dice-rolling experiment.

ADHD and handwriting

Specifically relevant to the experiments of this thesis was a review of articles that discussed the handwriting of ADHD students. Kaiser et al. (2015) found research that indicated that children with ADHD tend to have less legible handwriting than their peers without ADHD (Tucha & Lange, 2001). They tend to make more spelling errors, insertions, or deletions of letters and have more giant letters (Adi-Japha et al., 2007; Shen et al., 2012). However, the biggest concern is the variability in the production of letters, including variations in letter height, spacing between letters and words, and alignment with the baseline (Tucha & Lange, 2001; Adi-Japha et al., 2007). This variability increases with longer writing tasks,

making it more difficult for children with ADHD to produce consistent handwriting over time (Borella et al., 2011). This variation would be something that this thesis would investigate using a novel method to measure the variation of the intended line against the produced line.

Three studies (Langmaid et al., 2013; Schoemaker et al., 2005; Adi-Japha et al., 2007) have identified issues with handwriting accuracy. One study had children write a cursive "l" under 10 mm and 40 mm conditions (Langmaid et al., 2013). This study showed that children with ADHD were less accurate than the control group, missing the upper line more often in the 40 mm condition (Langmaid et al., 2013). The 10mm to 40mm cursive “l” writing task is experimentally the most similar to the novel method used in this thesis. Instead of a cursive “l” used by Langmaid et al. (2013), the novel task simplified the fine motor movement to a horizontal straight line. Developmentally, the straight line is the simplest handwritten form. Both were in the 40mm to 60mm scale. Another study gave a graphic task using very small 4 mm and 6 mm conditions. Langmaid et al. (2013) showed that children with ADHD were less accurate in the 60 mm condition, as ADHD participants did not even reach the margin lines when drawing forms (Schoemaker et al., 2005). Finally, in a third study, children with ADHD were faster but less accurate than the control group when drawing an ellipse (Adi-Japha et al., 2007).

ADHD and Academic Achievement

In a meta-analysis, Frazier et al. (2007) identified poor academic performance as the most prominent feature of attention-deficit/hyperactivity disorder. Individuals with ADHD are at risk for a range of academic complications, including a higher incidence of failing grades, elevated rates of grade retention (Ferguson & Horwood, 1995; Ferguson et al.,

1997) and lower scores on standardised tests achievement (Abikoff et al., 1996; Carlson & Tamm, 2000; Carteret al., 1995; Frankenberger & Cannon, 1999; Gaub & Carlson, 1997; Halperin et al., 1993; Hoza et al., 2002: Lahey et al., 1998; Purvis & Tannock, 1997, 2000; Seidman et al., 1997; Semrud-Clikeman et al.,2000; Tannock et al.,2000; Zametkin et al., 1993).

The meta-analytic results of Frazier et al. (2007) indicated a moderate to large discrepancy in academic achievement between individuals with ADHD and typical controls (weighted d= .71). This outcome substantiates the significant impact of ADHD symptoms on academic performance.

Automaticity and controlled processing

Broadbent (1958) argued that information in parallel channels was processed or attended to. However, there was a limit, and a filter would cut off all further processing, with all information being lost in the unattended channels. However, Treisman and Riley (1969) found that information left in the non-attended channel could be processed, and processing depended on the information type. This finding could not be reconciled with an all-or-none attention filter. Treisman and Riley (1969) proposed that the attention filter attenuates messages on non-attended channels but that information arriving on these channels is processed at least to some extent.

Schneider and Shiffrin (1977) defined selective attention as the control of information processing so that sensory input is perceived/remembered better in one situation than another. Schneider and Shiffrin (1977) justified this proposition with the idea that attention resources are limited, and, in some cases, new information must be given “special attention”.

Schneider and Shiffrin (1977) described two possible attentional bottlenecks. The first is when attention is divided over several inputs. For example, they are attempting to listen to multiple conversations simultaneously. The second attentional bottleneck occurs when the individual becomes distracted by an irrelevant stimulus when focused on a relevant stimulus.

Schneider and Shiffrin (1977) proposed two quantitatively and qualitatively distinct processes: controlled search and automatic detection. They proposed that controlled search is a serial process in which a matching decision occurs after comparing each item in the display to memory set items. It is modifiable, flexible, can be used in novel situations and uses short-term memory capacity. Schneider and Shiffrin (1977) proposed that automatic detection operates parallel and independently of attention. Automatic detection operates independently of the subject's control and independent of attention, not using up processing resources like short-term memory. Schneider and Shiffrin (1977) also argued that through consistent training, automatic detection can attract attention automatically.

Model of Action Selection

Norman and Shallice (1980) developed a model of attentional control of executive functioning. In this model, thoughts and actions form schemas or set scripts of actions to be used under specific circumstances. These scripts are triggered by specific perceptual stimuli or from prior triggered scripts. The scripts/schema are divided into high-level schemas for problem-solving and low-level for actions (Hommel et al., 2002).

The model further proposes two main processes that manage the functioning and storage of schemas. The first is contention scheduling, and the second is a supervisory attentional system. This process does not require conscious control and is triggered by

familiar stimuli. Contention scheduling regulates the schemata of familiar and automatic actions. Contention scheduling prioritises using specific schema by inhibiting competing schema from executing simultaneously (Shallice & Burgess, 1996). When thresholds are met, contention scheduling ensures the correct schema is activated. The more a schema is reused, the easier and more likely that schema is reactivated in the future.

The supervisory attentional system (SAS) is the second part of the Norman and Shallice model. Contention scheduling is subordinate to the SAS. The SAS manages deliberate and conscious actions in new situations where scripts are not readily available or when committing errors or forming habits is to be avoided (Hommel et al., 2002). This is accomplished by modifying the existing schema. In entirely new situations, a new schema can be created, assessed, and implemented (Badgaiyan, 1999). When comparing SAS to contention scheduling, SAS is slower, voluntary, but flexible, whilst contention scheduling is faster as it does not require conscious control.

Another key idea in the Norman and Shallice model is that SAS uses working memory to process information in schema development (Leach, 2005). The SAS makes independent behaviour possible by using memory to plan, make decisions, and solve problems. Hommel, Ridderinkhof and Theeuwes (2002) state that the SAS encompasses the main components of human attention, particularly the selection component of where attention should be focused. The SAS maintains attention over long periods, the ability to switch attention quickly from one task to another and prime attention in anticipation of a task. When the SAS is unsuccessful in suppressing irrelevant schema, attention is adversely influenced (Friedenberg & Silverman, 2010).

Bargh and Uleman (1989) defined the distinction between controlled and automatic cognitive processes as a function of the amount of attention used to complete the cognitive process. They went on to explain that processes that draw few processing resources and require no attention, awareness of initiation or cessation of the process are categorised as automatic processes. This idea fits into the paradigm of top-down and bottom-up driven processes. The distinguishing factors between controlled and automatic processes most relevant to the empirical experiments are to what extent the individual is aware or controls the actions and to what extent inhibition affects individuals with ADHD compared to their non-disabled peers.

Proactive and Reactive cognitive control

Braver (2012) proposed the dual cognitive control framework (DMC) mechanisms. His hypothesis of the DMC framework is that cognitive control operates via two distinct operating modes: 'proactive control' and 'reactive control'. The quality of time is responsible for which mode is utilised. The proactive control takes place in the lateral prefrontal cortex. It is a form of 'early selection'. Braver (2012) states that proactive control is a "top-down bias that facilitates the processing of expected events". The lateral PFC helps to hold and maintain goal-relevant information. Ideally, this happens before the occurrence of cognitively demanding events to optimally bias or focus attention, perception, and action systems toward those goals. A sign of this occurring is the involvement of the dopaminergic system to sustain inputs into the PCF actively. Without this, the dopaminergic signal only transient activation is possible (Braver, 2012)

Braver (2012) states that the transient activation of the lateral PFC is a sign of reactive control. He goes on to characterise reactive control as a bottom-up reactivation of task

goals that is mediated when interference is detected by conflict-monitoring regions of the brain, such as the anterior cingulate cortex (Braver, 2012)

Reactive control uses attention as a 'late correction' mechanism as needed. Braver (2012) states that attention is only used after a high interference, just in time after an event is detected. The main distinction between proactive and reactive control is that it relies on anticipating and preventing interference before it occurs, whilst reactive control utilises detecting and resolving interference after the fact.

Braver (2012) states that both systems are semi-independent and can be used simultaneously but suggests that there may be a bias towards one. This bias is often related to the task or even the individual. Braver (2012) argues that the DMC is a unifying framework that explains differences in cognitive control within individuals and between different people and potentially explains changes in cognitive control associated with the development of children, older people, and neuropsychiatric disorders.

Cognitive control, as conceptualised by Braver and applied to ADHD individuals, could explain the inability to flexibly adapt to changing circumstances by regulating behaviours so that inappropriate ones are suppressed and required ones are facilitated in response to environmental demands (Braver et al., 2003). Existing models of ADHD (Barkley, 1997; Nigg, 2005; Sergeant et al., 2003) and experimental evidence implicate abnormalities in cognitive control as a factor significantly contributing to ADHD symptomatology (Durston, 2003; Durston et al., 2009; Willcutt et al., 2005). Children and adults with ADHD have been measured to have poor cognitive control when completing tasks that measure cognitive control (Cepeda et al., 2000; Dibbets et al., 2010).

According to Sidlauskaite et al. (2020), studies on ADHD and cognitive control have primarily focused on reactive control mechanisms, which are related to target-based disruptions. However, recent studies involving ADHD participants have revealed promising evidence from event-related potential studies that suggest inferior task performance may be due to altered preparatory processing during the cue-target phase. This evidence could mean a problem with proactive control, resulting in reduced attentional orientation to cues and diminished contingent negative variation amplitudes, indicating less motor preparation or stimulus anticipation in ADHD. Studies by Albrecht et al. (2013), Banaschewski et al. (2008), Hauser et al. (2014), Kenemans et al. (2005), McLoughlin et al. (2010), and Valko et al. (2009) have also indicated less motor preparation or stimulus anticipation in ADHD. This means that in tasks that rely on button pressing, their reaction times may increase due to a lack of motor preparation, which is related to pro-active control depending on the type of cue preceding it.

Sidlauskaite et al. (2020) investigated neural correlates of proactive and reactive cognitive control in adults with ADHD versus healthy controls by employing various switching tasks. Sidlauskaite et al. (2020) would attenuate the cue's informativeness across a spectrum of informativeness during trials. This manipulation was recorded with an EEG. Sidlauskaite et al. (2020) investigated cognitive control processes in adults with ADHD from the perspective of Braver's (2012) dual model of cognitive control. The ADHD group was found to have slower reaction times overall; This was attributed to diminished preparatory proactive rather than reactive cognitive control difficulties. Sidlauskaite et al. (2012) pointed to reduced usage of cue information and potentially deviant preparatory switch-related activity, as evidenced in the ERP findings.

However, Sidlauskaite et al. (2020) found no differences in target-locked activity. These results suggest that adults with ADHD may have altered proactive control, characterised by reduced utilisation of cued advance information and abnormal preparatory processes for upcoming tasks rather than reactive control.

Using the Braver model and relating the findings of Sidlauskaite et al. (2020) to this thesis, it would seem that the Braver model would predict that ADHD participants would find it difficult to proactively prepare motor response resources to complete the Stroop, Pure Motor Inhibition Task, and Attentional Inertia Tasks as a result of poor proactive control. Braver would characterise proactive control as a top-down process.

Inhibition

Ubiquitous in cognitive psychology, inhibition is the ability to either fully or partially stop effortful or automatic neurological function. The definition of inhibition can be used in other ways to mean activity reduction below a baseline. In both cases, a separate active process is used to halt, slow, or reverse the progress of some other process. In this thesis, inhibition is analogous to a force vector actively working to change the direction of a current ongoing process to halt, slow or reverse its progress. It should be noted that inhibition is not the cessation or absence of a process. Inhibition is conceptualised in this paper as an active oppositional force that is directionally opposed to the ongoing initial processes.

Inhibition is a term used to refer to many aspects of processing, from the single neuron level through various collections of neural circuits and overt behaviour. Inhibitory control is a core executive function that will be investigated in this study. It is a dynamic process that makes choice and change possible. Inhibitory mechanisms are found in various domains, such as response control, memory, selective attention, theory of mind, emotional regulation, empathy, and intentional forgetting (see Anderson & Levy, 2008). Inhibition of attention is crucial to processing competing stimuli (Postel et al., 2002) and can also be important in resisting competing stimuli proactively. Indeed, working memory and inhibition can work closely, and the latter can alter or change how representations assist memory (Wright & Diamond, 2014). The development of inhibition of prepotent responses seems to mature with an individual (Davidson et al., 2006). Children ages 8-13 are particularly unable to resist prepotent representations and make errors in Go/No-Go tasks (Barry & De Blasio, 2015), theory of mind tasks (Carlson et al. (2004) and Day and Night/ Stroop tasks (Gandolfi et al., 2014).

When inhibitory mechanisms fail to function adequately, it can have severe consequences for the process that it is meant to inhibit, such as anger (Fetterman et al., 2012) and attention (Wodka et al, 2007; Vaurio et al., 2009; Van De Voorde, Roeyers, Verte & Wiersema 2011; Rodriquez-Jimenez et al., 2006; Pievsky & McGrath, 2018). At all levels of processing, some form of inhibition is usually said to be present, as Anderson and Spellman (1995) stated, “The existence of such inhibitory mechanisms in the functional architecture of cognition seems both plausible and necessary: plausible because the substrate on which that architecture operates – the brain – uses both excitatory and inhibitory processes to

perform neural computation, and necessary because computational analyses show that inhibitory mechanisms are critical for maintaining stability in neuronal networks".

The results of diminished inhibitory control can be far-reaching. Using confirmatory factor analysis, Friedman and Miyake (2004) showed that prepotent response inhibition and resistance to distractor interference were closely related and could explain a range of lifelong outcomes: academic performance, social competence, health, wealth, and criminality (Friedman & Miyake, 2004) Several authors (e.g., Carlson, Moses, & Breton, 2002; Dagenbach & Carr, 1994; Dempster & Brainerd, 1995; Sarason et al., 1996) have shown the importance of inhibitory processes within the broader context of executive function.

ADHD as a Problem of Inhibition Examined

Specifically important to this thesis is that prominent models of ADHD rely on inhibition to explain symptoms of ADHD. Experiments were selected and designed to complement the findings of Barkley, who based his inhibitory model of ADHD on the impairments he found in three behavioural inhibition processes: inhibition of prepotent responses (Stroop, pure motor inhibition task), the stopping of ongoing responses given feedback on errors (dice rolling, attentional inertia), and interference control (handwriting) (Barkley, 1997: 1997b, 2001).

Having impairments to inhibition has been linked to disorders such as ADHD (Barkley, 1997; Nigg, 2001), schizophrenia (Nestor & O'Donnell, 1998), autism (Ciesielski & Harris, 1997), and obsessive-compulsive disorder (Enright & Beech, 1993). Effective intervention

and treatment of these individuals, particularly in an educational environment, should be developed and managed based on a clearer understanding of all inhibitory functions.

The current scientific understanding of inhibition has been greatly influenced by three factors: the growth and use of cognitive neuroscience, particularly with brain imaging work; more sophisticated cognitive modelling; and the discovery of new cognitive phenomena, such as negative priming (Tipper, 1985) and inhibition of return (Posner & Cohen, 1984). Interest in cognitive inhibition, inhibitory processes at the categorical level above the neuron, has grown with the increased prevalence and use of technology in neuroscience, and researchers have attempted to establish the importance of inhibition in our understanding of how the mind, cognition, and executive function work collaboratively.

This thesis ascribes to the idea that the nervous system can process information in 'two directions'. The first is top-down, where processing begins in higher-level brain structures and constructs representations using contextual information, memory, and experiences. These representations are cognitive constructs that accomplish intentions, such as performing a task or preparing for action. The second is a bottom-up process that directly builds representations from the sensory data. Inhibition is essential in either process or direction and potentially uses the same neural circuits (Wiecki & Frank, 2013; Wigman et al., 2015). Bottom-up processing is associated with perception and data within the sensory data stream and is inhibited within the basal ganglia (Kaji, 2001). Processing models have linked ADHD through neural task interactions with the three core characteristics of ADHD. The three core characteristics of ADHD linked to inhibitory dysfunction are a reduction in attention, an essential component in perception,

hyperactivity, and impulsivity linked with preparation for motor action (Barkley, 1997: 1997b, 2001).

Barkley based his inhibitory model of ADHD on the impairment he found in three behavioural inhibition processes: inhibition of prepotent responses, stopping ongoing responses given feedback on errors, and interference control. Further, Barkley showed correlations (Barkley, 1997: 1997b, 2001). In the empirical studies of this thesis, I expanded the possible forms of inhibition that could be dysfunctional beyond those three original processes with the addition of bottom-up processes, such as motor control. I attempted to show that inhibition in individuals with ADHD may be another essential factor to consider.

Albin, Young, and Penney, (1989). (see also Alexander, DeLong, & Strick, 1986; Kemp & Powell, 1971) have provided evidence that the basal ganglia regulate access to the prefrontal cortex. These access points within the basal ganglia, referred to as the direct and indirect route, are a set of interconnected subcortical nuclei manifolds that receive inputs through converging neurons from the entire cortex, including the prefrontal cortex, the traditional seat of executive function. After inhibitory processing, a single output from the basal ganglia is routed only to the frontal lobes. The basal ganglia ‘‘funnels” cortical signals, processing them through a series of inhibitory gates so that they “compete for access” to the prefrontal cortex (Stocco et al., 2017). This evidence suggests that the basal ganglia act as an intake manifold for neural signals, with the output being selected by the basal ganglia using yet unknown selection criteria among competing signals that need to be outputted (through the thalamus) to the prefrontal cortex (Gurney et al., 2001; Redgrave et al., 1999; Stephenson-Jones et al., 2011).

Diamond (2013) evaluated the commonalities and differences between different variants of inhibition. For example, Nigg (2000) proposed that most forms of inhibition are separate functions. However, Diamond pointed out that there is evidence that interference inhibition and inhibition of action share a common neural resource (Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Congdon, Mumford, Cohen, Galvon, Canli, & Poldrack, 2012). Diamond (2013) went further by stating that distinctions between other forms of cognitive inhibition, such as inhibitory control of attention and inhibitory control action, share a substantial neural base, which was also found by Engelhardt et al. (2008) and Freidman & Miyake, (2004). Friedman and Miyake (2004) did extensive work using factor analysis to establish a strong correlation between inhibition of attention, sometimes known as “resisting distractor interference”, and inhibition of action, which Friedman and Miyake (2004) refer to as “inhibiting a proponent response” to establish that both are the result of a single factor. Further evidence that many forms of inhibition may share a common resource comes from research completed by Muraven (2010) and Muraven and Baumeister (2000). This research shows that when two very different tasks are completed close to one another, an individual will have diminished inhibition in the second task, even in a very different domain. This may show that a common resource may be shared across all inhibitory functions even without a standard neural network. Diamond (2013) would not subscribe to the hypothesis that bottom-up inhibition, such as attentional blink and negative priming, share a common neurological circuit with top-down forms of inhibition. Carr, Nigg, & Henderson (2006) and Nigg, Butler, Huang-Pollock, & Henderson (2002) also distinguished between effortful and other forms of cognitive inhibition by not conflating the two.

There needs to be more research associating different categorical forms of inhibition with locations in the neural circuits. However, there is growing evidence that executive functions (e.g., working memory) can be modelled, and these models have been used to predict captured patterns of electrophysiology of humans and animals engaging in cognitive control tasks. For example, Wiecki and Frank (2013) mapped neural circuits, resulting in a model that predicted neuron activation data recorded experimentally.

Nigg (2000) has argued that there should be a distinction between effortful inhibition and motor inhibition. He further broke down effortful inhibition into four subtypes: interference control, which manages competing stimuli; cognitive inhibition, which manages irrelevant thoughts; behavioural inhibition, which helps to regulate prepotent emotional outbursts in social situations; and oculomotor, which stops the saccade reflex of the eyes. Nigg intended to classify and describe observed forms of inhibition, but latent variable analysis was completed by Friedman and Miyake (2004), which established three main types of inhibition: prepotent response inhibitions, resistance to distractor interference, and resistance to pro-active resistance. These classifications can be mapped onto Nigg's subtypes, providing a framework for understanding inhibitory responses.

Prepotent response inhibition is a conceptionally important idea that may provide a neurological explanation for the impairment of ADHD. Prepotent response inhibition is the intentional management of prepotent representations, either suppressing, filtering, or resisting the distraction of any number of prepotent representations whilst retaining the long-term representation beneficial to the goal at hand. Prepotent response inhibition is thought to have the ability to resist automatic responses such as inhibition of return

(Carlson & Moses, 2001) and saccade eye movements (Munoz et al., 2004). Distractor interference is thought to manage new external distractions, whilst proactive interference resists returning to stimuli deriving from prior tasks (Nigg, 2002).

Other researchers have proposed dividing inhibitory control along different lines. Diamond (2013), for example, considers inhibition as cognitive inhibition, executive attention, and response inhibition. Cognitive inhibition regulates unwanted thoughts and memories that could coalesce to form a distraction. Executive attention is the cognitive mechanism by which representations are managed at the level of attention. Response inhibition helps to regulate behaviour by suppressing prepotent emotional responses. Cognitive inhibition and executive control work in tandem to manage interference from prepotent representations by suppressing them. This allows the re-engagement or continued engagement with the representation in pursuit of an intention or goal. Diamond (2013) agreed with the definition of a 'bottom-up stimulus' proposed by Posner and DiGirolamo (1998). She made it synonymous with 'automatic', 'involuntary attention' or 'driven by the stimulus's properties. Voluntary mental effort towards a goal would override these bottom-up processes towards the goal.

Bunge et al. (2002) distinguished between inhibition as a filtering or suppressing function—inhibitory functions, like response inhibition, suppress prepotent representations completely. The filtering of external stimuli at the prepotent level via interference monitoring and suppression. Evidence for this came from brain imaging work using Flanker and Stroop tasks (Bunge et al., 2002). Research also shows that the process is associated

with activity in the basal ganglia and frontal lobe and is often atypical for individuals with ADHD (Kaji, 2001; Alexander & Crutcher, 1990; Hazy et al., 2007).

Inhibition has also been linked with working memory. For example, Fuster (1989) argued that inhibition is crucial in retaining information for analysis so that it can be compared with the past and be used to plan towards a goal or process in the future. The absence of inhibitory functions would mean that an individual would be in constant flux, reacting to every stimulus without regard to importance, unable to reach any goal or complete a function. Without working memory and inhibitory functions, a person could not stop and think to engage in higher-level processes. At the same time, a person without inhibitory control would constantly be locked into bottom-up processes, drawn to the most recent salient stimulus without regard to other things in the environment and unable to orient to things of importance (Koch & Fuster, 1989).

One must note that apart from sharing the term inhibition, not all researchers agree that the different forms of inhibition can be linked in any meaningful way. In a general and broad criticism of the inhibition notion, Goerfein and MacLeod (2007) argued that the widespread use of the word inhibition in psychology is not beneficial and that conflating the diverse forms of inhibition is contradictory. The authors stated that a connection between neural and cognitive inhibition might not exist. They warned that forcing the multiple levels of analysis of inhibitory function together is not helpful because it is unlikely to help our understanding of the different inhibition categories. Furthermore, Goerfein and MacLeod warned that the relationship between the different types of inhibition is ambiguous and that the conflation of inhibitory processes is not helpful. However, they do

not rule out a possible tangential relationship between different types of inhibition, insomuch as they are connected as inhibitory processes at the neuronal level, which is the building block of cognitive circuits.

Goerfein and MacLeod thus suggest that there is only a tenuous relationship between the different forms of inhibition and are reticent to draw a direct or clear connecting line between neural and cognitive inhibition. They further argue that it is unlikely that cognitive inhibition, or any form of inhibition, is a single function. The observed types or categories of inhibition are a collection of neural circuits working similarly to inhibit specific processes. However, evidence suggests that individually identified cognitive inhibitory functions, e.g., top-down inhibitory functions, result from a single factor and not multiple related specialised factors (Miyake et al., 2000). One does have to note, however, that although MacLeod's broad criticism of the inhibition notion raises many important issues, it is not one that psychologists have widely accepted.

Inhibition and ADHD

Research suggests deficient inhibition is a primary symptom of ADHD (Barkly, 2007; Nigg, 2001; Adams et al., 2007). The main models of inhibitory control are the *Independent Race Model* (Logan & Cowan, 1984; Logan et al., 2014), *The Interactive Race Model* (Boucher et al., 2007), and *The Brain Circuitry Model* (Frank, 2006; Wiecki & Frank, 2013), which broadly fall into one of three possible explanations for the neurological circuits that complete the inhibitory computations. The *Independent Race Model* involves two systems without connection that perform the computations necessary for top-down and bottom-up inhibition utterly independent of one another (Pinto et al., 2013). The second possible

explanation, *The Interactive Race Model*, proposes that two separate systems are interdependent but still use separate circuits that share information (Boucher et al., 2007). The final possibility, the *Brain Circuitry Model*, argues that both top-down and bottom-up inhibition result from a single neurological circuit (Frank, 2006).

People with ADHD have pre-potent response difficulties, as the threshold for resisting these pre-potent representations is weakened due to their impairment. Barkley (1997) found that children with ADHD required more mental effort than a typical person to filter out an unwanted stimulus concerning a range of tasks, including motor action, behaviour, and impulsivity, by comparing 34 preschool boys with attention deficit hyperactivity disorder (ADHD) to 30 preschool boys without behavioural problems on a battery of neuropsychological and academic achievement tests along with behavioural observations. In some cases, this threshold cannot be reinforced with any amount of mental effort and is beyond the individual's ability to control.

ADHD and Stroop Interference

This thesis uses a simple Stroop test, an original Stroop 1935 test adaptation. The test version used in this thesis reduces the colours from 3 to 2 and further adapts it for online use. Golden (1978) named the Stroop test one of the most often used measures of response inhibition. Barkley (1997) argued that people with ADHD have difficulty inhibiting prepotent responses, also known as response inhibition (Miyake et al., 2000). Schartz and Verhaeghen (2008) completed a meta-analysis to investigate the presumed response inhibition problem associated with ADHD, an issue not examined in meta-analyses on inhibition and ADHD (Boonstra et al., 2005).

The Stroop test assesses a person's capability to suppress a dominant response, such as reading, and instead focus on completing a given task, like naming colours. According to Cohen et al. (1990), two processes are involved in this test: an automatic response that demands minimal attention and a controlled response that requires attention and is voluntarily controlled. According to Stroop's study in 1935, individuals are typically quicker at reading words than identifying colours. This is because reading is an automatic process that occurs faster than the controlled process of colour identification. During the third trial of the Stroop test, also known as the incongruent trial, people tend to read the word instead of identifying the colour of the ink. This leads to a competition between word reading and colour identification, resulting in poorer performance (Cohen et al., 1990). The theory suggests that those with ADHD may struggle more with focusing on the controlled process than individuals of the same age.

A meta-analysis conducted in 2008 by Schartz and Verhaeghen investigated the effects of ADHD on attention and response inhibition. They analysed 25 studies that concentrated on the Stroop colour word test, which included children and adults with ADHD and control groups of the same age. The analysis used a hierarchical approach to measure the strength of the Stroop effect and its variation based on age. The studies also explored differences in maturation rates based on the reaction time of colour and colour-word conditions.

The researchers found a relationship between colour-word and colour response time and characterised the relationship as the slope of this function being identical across age groups and ADHD status. They also found that, although ADHD individuals were, on

average, 1.14 times slower than age-matched controls in both the colour and the colour-word condition, the maturation rate was identical for both ADHD and control groups.

The results indicate that the Stroop interference effect is not more prominent in ADHD individuals than in age-matched controls, and there is no evidence for differential maturation rates for persons with ADHD and control groups. These findings suggest that ADHD children have similar response characteristics in inhibition for the Stroop task compared to their control group peers. It also predicts a similar or non-significant difference between ADHD and control groups when the Stroop task is used in this thesis. If this is shown to be the case, when deficiencies in inhibition are found, they can be attributed to different processes or sources.

The present work

Chapter 2 examines motor inhibition in three populations: a control group, an ADHD group, and a developmentally younger group. The paradigm used indexes inhibitory processes that are purely motor in origin and do not involve higher mechanisms. Responses were then compared with results from a Stroop task, i.e., a paradigm that does involve higher cognitive processes. The first experiment tested the online versions of the tasks to ensure that inhibitory effects in both tasks were replicable. In the second experiment, ADHD children and a control group completed a motor inhibition and Stroop task. It was predicted that children with ADHD should have significant difficulties with inhibition, resulting in an absence of the motor inhibition effect. In contrast, however, their Stroop performance was predicted to be intact. In the third experiment, children aged 10-11 completed the motor inhibition and Stroop tasks. The rationale is that this population, like

ADHD individuals, have an under-developed inhibition system (possibly due to a non-fully functioning basal ganglia).

Chapter 3 investigates to what extent inhibition regulates a gross motor skill task in participants with ADHD and a control group. Participants were asked to roll a dice multiple times and keep the dice on the table. This requires participants to inhibit gross motor actions to accomplish the task. It was predicted that children with ADHD would have difficulty inhibiting such actions, resulting in the dice going off the table significantly more than a control group.

Chapter 4 examines the role of inhibition in fine motor control. The novel use of ImageJ software measured straight lines drawn by a control group and an ADHD group. This process measures the deviation of a written line compared to a presumed intention. Students with ADHD were predicted to have a more significant deviation in the line due to less inhibitory control than the comparison group.

Chapter 5 examines the degree to which ADHD children are subject to "Attentional Inertia". Attentional inertia, thought to be regulated by inhibition, is the phenomenon in which old irrelevant rules and stimuli fail to be discarded in favour of new relevant information. It was predicted that children with ADHD would be particularly subject to this effect compared with controls. Participants were asked to complete ten mathematical problems starting with a run of multiplications. On the 9^{th} question, the mathematical operation sign changed to addition. The experiment aimed to measure the extent to which participants can inhibit the now-irrelevant multiplication rule in favour of the new addition rule.

Chapter 6 examines whether one of the findings shown in the ADHD participants (in Chapter 2) generalises to the broader population who exhibit ADHD-like behaviour. It was predicted that there would be an association between the degree to which a person shows motor-induced motor inhibition and the degree to which they exhibit ADHD, as measured by a commonly used inventory.

Chapter 2

Motor-induced motor inhibition

Barkley (1997) argued that hyperactivity in children with ADHD results from inhibitory dysfunction. Dysregulation in motor inhibitory control should be measurable in ADHD participants using a motor-induced motor inhibition task. Any motor inhibition task selected for this thesis must manage extraneous variables to ensure the measured effect is due to motor inhibition. ADHD can throw up many variables, including intelligence, motivation, and attentional difficulties. The novel paradigm developed by Cole and Skarratt (2022) arguably produces a motor-induced motor inhibition effect that has some advantages to being used due to its reliability to produce an effect and its adaptability to use online whilst also being a fast-paced task, requires one only to have the ability to judge the length of lines, and the ability to press buttons. Due to the constraints of COVID-19, in-person access to children was impossible, which put further considerations onto the task selection. Consequently, the Cole and Skarratt (2022) motor inhibition task was adapted for distribution over the internet and provided a pure motor inhibition effect.

Cole and Skarratt (2022) addressed concerns regarding using their novel paradigm as a valid measure of motor inhibition. They argue that many experimental paradigms require working memory to perform a typical task (e.g., red horizontal target = left response; blue vertical target = correct response), but the relatively high-level processing required can make participants fully aware of the critical conditions of interest to the experimenter. Take, for example, the classic Go/No-go task in which participants must

make a speeded response when T1 appears (e.g., a green square) but withhold the response when T2 appears (e.g., a red square). A participant is often fully aware of mistakenly responding on No-Go trials and can be frustrated when they fail to inhibit a response. This is similar to the Stroop task in which participants respond to ink colour while distracted by word identity. Again, one can feel the mental effort of being slowed by an incongruent pairing of word and colour. This applies to many so-called response inhibition paradigms (e.g., the Stop-Signal task). Indeed, Verbruggen and Logan (2008) remarked that response inhibition is a "hallmark of executive control". In other words, such paradigms do not solely index processes that are exclusively unconscious and stimulus-driven

Of course, there are many inhibition tasks in which participants are unaware of the different conditions and the effect being induced. (Note that when the present author refers to participants knowing the different conditions, he is not making a demand characteristics point. He illustrates that these inhibition tasks involve higher-level executive functioning). For example, in the negative priming paradigm (Tipper, 1985), an inhibited stimulus response on one trial becomes the target on the following trial. Participants must be aware of this manipulation and the resultant reaction time effect. However, this effect can still be relatively high-level in that it is very much due to processes that occur in the cortex (e.g., left temporal lobe, inferior parietal lobe, prefrontal cortex; see Steel et al., 2001).

To index inhibitory processes with limited cortical involvement (outside of the pre-motor cortex), a task is needed to measure motor processes solely. Such a candidate is the motor-induced motor-inhibition paradigm.

The Motor Induced Motor Inhibition effect.

Cole and Skarratt (2023; Cole & Skarratt, 2022) described a series of experiments where participants performed two simple tasks on each trial (see Figure 1). In Task A, they discriminate a letter and respond by pressing one of two buttons: left index finger for one letter and right index finger for the other. This first task is designed solely to induce a motor response. In other words, the response time for the letter discrimination is not of primary interest. In Task B, two stimuli appear, and participants must make a speeded comparative judgement between the two. In the basic experiment, they are asked to decide which of the two lines is the longer. The same two buttons are used to make the judgement for Task A. Results show that participants are less likely to choose the line that corresponds to the response just made on the letter discrimination task. For example, if a participant made the correct response on Task A, they are less likely to indicate in Task B that the line on the right is longer. To put this another way, pressing one button reduces the likelihood of performing the same action immediately after. Instead, it switches to another response (i.e., action). Note that the two lines are the same length on most of the trials of interest. This is to induce uncertainty; certainty, in terms of a participant being confident as to which line is longer when there is an actual size difference, is likely to reduce any motor inhibition effect.

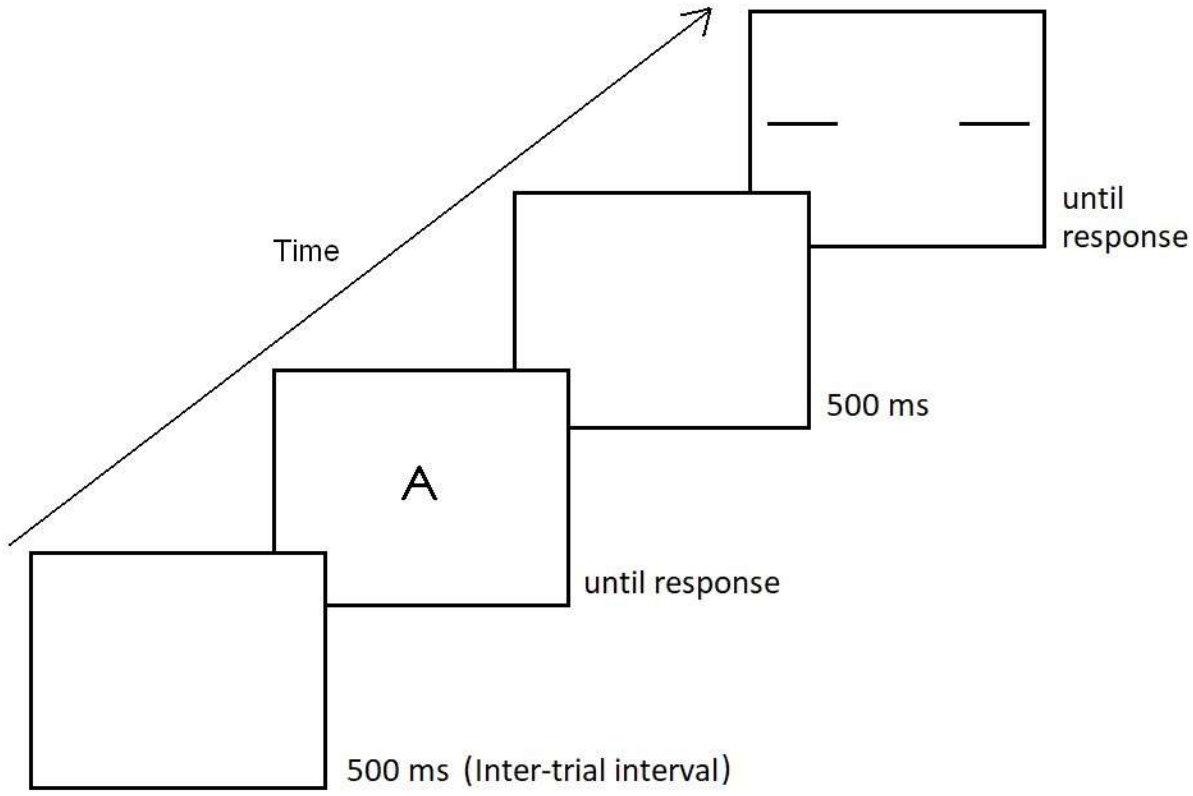

Figure 1. Trial sequence of the motor-induced motor-inhibition effect, as used in Experiment 1. Participants discriminate the letter by pressing one of two buttons. They then indicate, using the same two buttons, which of the two lines they think is the longer. In most trials, the lines are the same length, with the others being foils. Typical results show that participants avoid making repeated button presses. Thus, if they have just pressed a lefthand button because the letter was "A", they are less likely to indicate that the left line is longer.

Cole and Skarratt did not argue that the *perception* of the two lines had changed (although they did not assess this). They argued that a motor response had been inhibited, an effect purely induced by the participant's motor response. This finding is important because other behavioural paradigms that index Inhibition invariably use reaction times and error rates as the dependent measure. In contrast, the Cole and Skarratt effect is one in which a motor response influences a *decision*, especially a decision under uncertainty.

Significant to the argument that this phenomenon is purely due to motor processes is the fact that Cole and Skarratt eliminated the possibility that the effect is due to an attentional shift, specifically inhibition of return (Posner & Cohen, 1984; Posner et al., 1985; see Klein, 2000, and Lupiáñez et al., 2006, for reviews). As noted in Chapter 1, Inhibition of return, or IOR, is when responses to stimuli located at a recently attended position are relatively slowed. For example, if we read a book in a library and our attention is directed to a door where a person has just entered, we will look back at the book a moment later. We will now be slower to process any stimulus at the door location, such as a second person entering. Our attention (and motor processes) have been inhibited from returning to the door. In the standard IOR experiment, an attention-capturing cue appears in the periphery, quickly replacing another cue presented at fixation designed to bring attention back to the centre of the display. A target appears either at the previously cued location or, typically, on the other side of fixation. The standard IOR effect shows that responses are relatively slow when the target appears at the cued location (and more than 300 ms after the initial cue appeared; see Klein, 2000).

IOR explanation

An IOR explanation of the Cole and Skarratt effect suggests that a participant's response acts as a visual/auditory cue that shifts attention to the side of the display in which it occurred via within-hemisphere attention "spreading" (i.e., Hughes & Zimba, 1985). Furthermore, tactile cues induce within-hemifield facilitation for visual stimuli (Kennett & Driver, 2014). The potential consequence of these processes is that inhibition may occur for responses on the side of the display where the initial response to the letter occurred. Cole and Skarratt eliminated the attention/IOR account by showing that the phenomenon is

abolished when participants use different but adjacent fingers to perform the letter discrimination and line judgment tasks. The attention/IOR explanation of the basic effect suggests that if a participant made, for example, a left-finger button press when discriminating the letter, a subsequent left response would be inhibited. This account, therefore, predicts that responding with a left index finger will induce subsequent inhibition for a left *middle* finger response (or any left response). However, Cole and Skarratt found that the basic effect was abolished when the letter discrimination and line judgements tasks were required to be performed by different but adjacent fingers. In another experiment, the authors showed that the inhibition effect was significantly larger when participants responded to a large peripheral cue in Task A instead of when they were instructed not to respond to this cue in (a separate block). This suggests that although the inhibition effect can be induced when attention *does* shift to the side (by the peripheral cue), the additive effect of responding on Task A must be due to the motor component aspect of the task. The button press of a control group participant with the same finger in the same place puts an unconscious bias to inhibit further button presses by the same finger in the same place when presented with ambiguous stimulus in the form of equal-length lines. This results in the control group participants using their other hand to press a different button. I hypothesised that the ADHD group would not have the same level of inhibitory effect in the ambiguous state and fail to inhibit button presses in the same area.

In the present chapter, and as outlined in each experiment, the author employed the Cole and Skarratt motor-induced motor inhibition effect to index purely motor mechanisms and processing. The degree of inhibition induced was then compared with the

degree of inhibition induced by a different inhibition task, drawing upon higher cortical processing (i.e., the Stroop task).

It is worth noting how robust the motor inhibition finding typically is. The Cole and Skarratt (under review, 2022) paper includes eight experiments, each with several factors, assessing the phenomenon differently. For example, the authors showed that the effect occurs not only for the line judgement task but also for a variant of the basic procedure in which, rather than two lines being judged for length, participants were required to decide which of two faces they thought were the most attractive. Here, participants were less likely to choose a face if that choice required them to repeat a button press (made on the letter discrimination task). The same effect occurs when participants indicate which of two products they prefer (Skarratt & Cole, in preparation). Furthermore, Cole, Bubb, and Skarratt (in preparation) also found that response inhibition can influence recognition memory for both words and faces. In this further variant of the basic motor inhibition paradigm, participants, during the recognition stage of the task, either chose a face on the left or right of the display. Immediately before this decision, they were required to perform the letter discrimination. Again, results showed that a response (on the discrimination task) influenced subsequent responses (i.e., recognition memory). Therefore, the motor-induced motor inhibition effect is highly robust, observed under an extensive range of scenarios.

Node Structure Theory and Motor Inhibition: A Possible Alternative Explanation

One possible mediating mechanism that could account for the motor-induced motor inhibition effect is what Mackay (1987) called “self-inhibition”. The importance of “self-inhibition” in connectionist processing models is well known (Feldman & Ballard, 1982). The argument is that some form of self-inhibition is necessary to prevent general

convulsions occurring as a result of neural activity that persists after activation. Nodes, representing collections of neurons, separately represent the production of muscle movement and action—however, some nodes code for movement and sensory input. Higher-level cognitive nodes prime these so-called subordinate nodes through top-down connections that generate action (Omrani et al., 2017). Immediately after the cognitive nodes activate subordinate nodes, bottom-up connections between these nodes (that enable perception) prime the cognitive nodes. Mackay argued that reverberatory effects on higher nodes would occur throughout the system without a self-inhibition process. Theories of motor control often incorporate inhibitory processes following activation to prevent convulsions. Node structure theory, proposed by Mackay (1987), views self-inhibition as an essential component due to reverberation potential. Mackay (1987) states that nodes are basic units representing perception and action production separately. Some nodes represent both sensory experience and muscle movement. Higher-level mental nodes prime subordinate nodes via top-down connections that enable action. The bottom-up connections between these nodes prime mental nodes immediately after the mental nodes activate subordinate nodes. The potential for reverberatory effects on higher nodes can occur at all levels in the system, hence the need for self-inhibition.

Mackay (1987) also provides evidence for self-inhibition, which goes through a complete "recovery cycle." Following maximal inhibition, the system returns to its resting state gradually and linearly over a period of time. This gradual and linear return is supported by empirical evidence from various paradigms concerned with cognitive processes. For instance, letter omission errors during writing show a particular pattern in dyslexic and non-dyslexic individuals (Mackay, 1969. The likelihood of dropping a letter is high if it occurs near

the beginning of the word. For instance, “elderly” may be written as “eldery”. Here, the second occurrence of the letter “L” has been dropped. Furthermore, the likelihood of making this error decreases linearly as the distance between the dropped letter and its earlier duplicate increases. The present Experiment 2 is also suggestive of this linear waning effect. Future work that more precisely measures the time course of the present effect will determine whether the developing function seen in Figure 2 is the beginning of what Mackay referred to as “hyper excitability.” This is a facilitation or “rebound” period following the complete cessation of inhibition.

Event Files

There may, however, be other non-inhibition-based alternatives to explain the basic effect. For example, Hommel (1998) showed that visual attention plays a crucial role in perception and action, particularly in integrating all the information related to an object. Hommel (1998) described this integration as an "object file," a hypothetical memory structure that encodes the episodic combinations of stimulus features. However, action-oriented approaches to attention suggest that perceptually derived object files may need to be completed. Hommel (1998) argued that object files may include action-related information if attention subserves action control. This means that feature binding may not be limited to stimulus features but can also include features of the responses made to the respective stimulus. Hommel then demonstrated this idea in three experiments. Participants performed simply prepared left- or right-key responses (R1) to the mere presence of "Go" signals (S1) that varied randomly in form, colour, and location. Shortly after the prepared response, a binary choice reaction (R2) was made to the form or colour of a second stimulus (S2). The results indicate that benefits due to stimulus-feature

repetitions (S1-S2) interact. Form repetition only enhances performance if colour is repeated and repeating the relevant stimulus feature (form or colour) only improves performance if the stimulus location is repeated. This provides evidence for object-file formation. However, Hommel (1998) found evidence of bindings between stimulus and response features. Repetition benefits associated with the relevant stimulus feature and stimulus location depended on response repetition. This suggests that object files represent only one component of more complex "event files" that link information about stimulus and response aspects of an experienced episode.

The Stroop Task

The task is broadly used to assess the extent to which an individual can inhibit cognitive interference from the stimulus of one event whilst situationally processing a second competing event with similar characteristics. In the basic paradigm, participants are required to discriminate the ink colour of a written word. Crucially, the word identity and ink colour are congruent (e.g., the word RED written in red ink) or incongruent (e.g., RED written in blue ink). In the original version of the experiment, Stroop administered the test by asking participants to read colour words as quickly as possible from cards or identify the colour of coloured squares as a control. As the cards were shown to participants, reaction times were recorded. The paradigm has been used in various research contexts (e.g., MacLeod & Dunbar, 1988; Ivnik et al., 1996).

The classic finding is that the incongruent condition's response time and error rates are greater. The Stroop effect is thus observed when two processes occur simultaneously but with unequal strength or bias. In this case, the near-automatic reading process is

stronger than the competing process of colour identification. This paradigm effectively forces the participant to inhibit the initial inclination or bias towards responding to the identity of the written word and instead respond to the ink colour.

One model to explain the Stroop effect comes from Cohen et al. (1990) using principles of parallel distributed processing (PDP). Cohen et al. (1990) described the Stroop effect as " providing a clear illustration of our capacity for selective attention and the ability of some stimulus to escape attentional control". Cohen et al. (1990) used PDP to argue that Stroop affects two components, speed of processing and interference effect, and are related to a common underlying variable called "strength of processing". Furthermore, Cohen et al. (1990) argued that this model will show three things. The first point is that the model will show that processing strength is a continuous function of practice. The second point is that the strength of two competing processes is relative, which drives the interference effect. The third point is that attention is given to processes with the greatest strength.

The Stroop effect consists of two processing pathways. One pathway processes the colour, and the other processes the reading of the word. These pathways ultimately come together to drive a response. The illustration below shows that Cohen et al. (1990) divided the processing path into three tiers: input units, immediate units, and output units. The input units are on the bottom and converge through connections to the intermediate units and then through connections to the output units. The output units are located at the top of the figure. The input units for ink colour and word are connected to all intermediate units in their respective ink colour and word pathways. Each input unit has a constant bias term and is added to the net input. The intermediate units from both pathways converge

on all the output units. Cohen et al. (1990) state that the processing is feedforward, with stimulus-activating units starting at the input level. As units get activated, their relative strengths build up and propagate through to the intermediate units and finally to the output unit. When a threshold is exceeded, a response is triggered. The reaction time is the sum of time it takes to exceed the threshold. In the centre of the diagram are two task demand units or attention units. One is for colour, and the other is for word. This attention unit is connected to all intermediate units. These attention units can only be given to one intermediate unit. When a task demand is triggered, it “sensitises” that pathway.

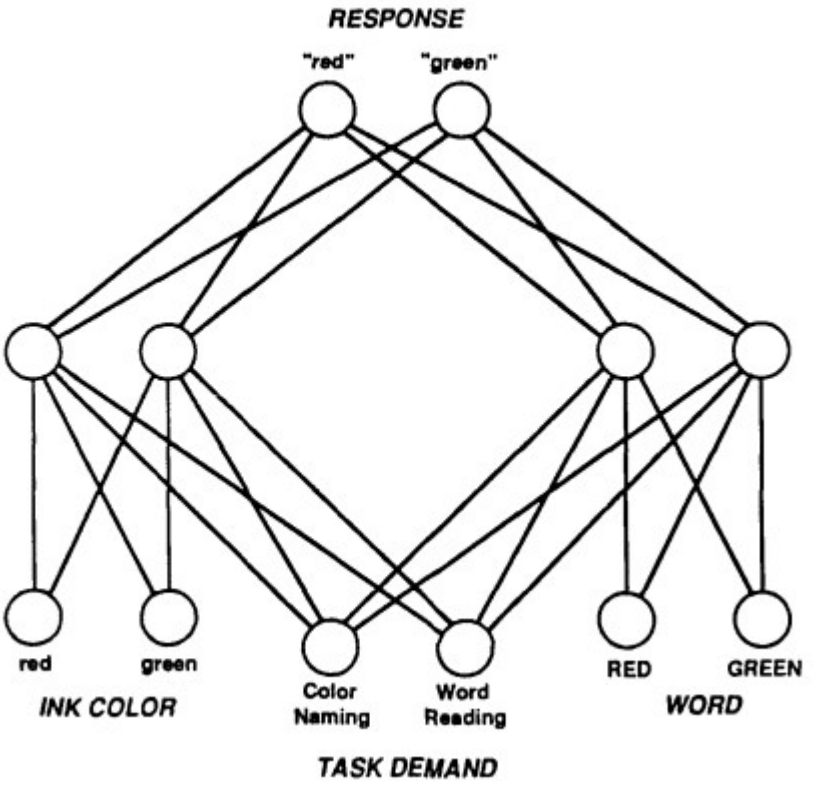

Figure 2. Cohen et al. (1990) stated that stimuli in this model are discrete representations. Colour is a single input, whilst word is a single input. Only a single output is possible.

Attention is the process by which one of the competing processes is selected based on the task demand. This happens when one of the task demands is provided. Cohen et al. (1990) argue that attention influences processing by interacting with the strength of processing to determine the pattern of the effects that result in the Stroop Effect. Cohen et al. (1990) state that attention could activate task-appropriate units, desensitise inappropriate units, or do both. The use of constructs and attention at the input level would indicate input from prior cognitive processes. Cohen et al. (1990) address this when they argue that this model would treat any input as information to be processed and that the model is flexible enough to be expanded to include several parallel processing paths.

The phenomenon is undisputedly associated with higher-level mechanisms, and thus, the task is often called indexing “cognitive inhibition” (Bjorklund, 2002). In summary, the rationale for using the Cole and Skarratt motor inhibition paradigm was, as described above, to use a task that indexes pure motor inhibition (with minimal higher-level processes being involved). The rationale for using the Stroop paradigm is that it is also thought to reflect response/motor-based inhibition but crucially involves higher-level executive functioning.

Experiment 1

The principle aim of the first experiment was to assess whether the motor-induced motor-inhibition paradigm reliably occurs in older children and in the particular way the present author ran the task. The experiment will also assess whether the particular variant of the Stroop paradigm reliably shows interference in the cohort.[1]

Participants performed the basic motor-induced motor-inhibition paradigm and a keyboard response-based 2-alternative-forced-choice version of the basic Stroop task. This extremely stripped-down version of the Stroop paradigm was designed for simplicity; children aged ten years (and upward) needed to perform that task at home. Without the experimenter's instructions and guidance. The author is also unaware of any previous 2-alternative-forced-choice version of the Stroop, another reason why it needed to be tested. On each trial of the Stroop task, a single word ("RED" or "GREEN") appeared in the centre of the display. The word would be written in either green or red ink, and participants were asked to indicate the colour by pressing one of two buttons. The motor-induced motor-inhibition paradigm is shown in Figure 1.

[1] The present PhD began in October 2019. By the end of February 2020, ethical approval had been given (see Methods section), the literature review completed and largely written-up, and the two tasks to be used in the present chapter ready to be undertaken by the students at the school where the author worked as a maths teacher. The pandemic then occurred, and all university testing was ceased. By the time it became clear that this would be prolonged the summer holidays had begun meaning that testing could not begin until September 2020 at the earliest. At this time the author decided on a different experimental testing approach. This involved learning to program the experiments using the PsychToolKit platform so that pupils could undertake the testing remotely on their own device at home. Although this is not ideal, the data reveal that remote testing is not of concern.

Method

Participants

There were 18 participants, ten male and eight female, all of whom attended a local comprehensive school in Sudbury, Suffolk. They were aged between 15 and 18 and were recruited from the middle academic sets, which means that they are of average intelligence as measured by a standardised scaled score that was received after taking their SATs, standard attainment tests, also known as the national curriculum assessment, that was administered to them at age 10-11. SATs are national curriculum assessments with validity and reliability monitored by the Standards and Testing Agency (STA), an executive agency within the Department for Education (DfE). The validity and reliability of these examples are guaranteed by law, with a report required by parliament to be produced yearly to evaluate the prior year's results. At the end of Key Stage 2, the scaled score 100 on the national curriculum tests represents the “expected standard” defined by the Department for Education (DfE). This score predicts a pass of 4 on eight different GCSE tests at the end of year 11. Standardised scores compare a pupil’s performance to a nationally representative sample of pupils from the relevant year group, who will have all taken the same test at the same time of year. The normal distribution of scores means that about two-thirds of pupils will have standardised scores between 85 and 115. Almost all pupils fall within 70 to 140, so scores outside this range can be considered exceptional. All students selected for this study had a scaled score between 100-110, firmly in the average. For 2022 in England, 65% of all students scored 100 or above on their SATs. The average score was 105. All children, regardless of ability or disability, must take the SATs. (*Key Stage 2 Attainment: National Headlines, Methodology*, 2022)

Psychometric validity data is available in Appendix B of each year's validity framework report provided by the Standards and Testing Agency online at https://assets.publishing.service.gov.uk/. Each subject area has its report.

Stimuli and Procedure

The Stroop task: Two possible words could appear: RED or GREEN. These were either written in red ink (RGB colour values = 237, 28, and 36, respectively) or green ink (34, 177, and 76), which generated four possible trial types. The font was Arial Bold, and on a 32 cm x 20 cm display, the letters were 15 mm in height and 10 mm in width.

The motor-induced motor inhibition task: Participants were presented with the letter “A” or “B” in the centre of the display. This was Arial font, 0.7° in height and 0.6° in width, and black (RGB = 0, 0, 0). No fixation cross was present (the letter effectively acted as a fixation stimulus). There were two kinds of display where the two lines occurred. The lines either differed in length or were the same. When they were the same, they could be three different sizes (1.3°, 2.5°, or 3.4°). On “foils”, in which the lines were different sizes, the difference in length would either be relatively significant (0.6°), medium (0.3°) or small (0.2°). This made the discrimination relatively easy, medium, or hard. The lines were positioned so that their centre was 10° (on a 32 cm x 20 cm monitor) to the left or right from the centre of the display.

Design and procedure

The Stroop task: Participants were instructed to rest their index fingers on the “R” and “G” buttons. They were further instructed to identify the colour of the printed word, ignoring the written word’s meaning. If the participant saw red ink, they were instructed to

press “R”; if they saw green ink, they were instructed to press “G”. In each trial, a word appeared in the centre of the display until a response was made. The inter-trial interval was 1500 ms. The dependent variable was the time to indicate colour ink via button press for each word presentation. A single block was presented where one hundred and ninety-two trials occurred (96 congruent and 96 incongruent). The four trial types were presented in a pseudo-random order. The experiment was run via the PsychToolKit (Stoet, 2010, 2017) platform, a well-established experiment-generating system that supports millisecond timing. The task occurred “remotely”; participants accessed the experiment on their device via a web link. This directed them to an introduction message that provided general background about the study (e.g., ethics information and approval number) and stated that a keyboard was required.

The motor-induced motor inhibition task: The trial sequence is represented in Figure 1. Participants were instructed to discriminate the central letter via a button press on their keyboard (“A” for the letter “A” and “B” for the letter “B”). When a response was made, the letter disappeared, and 500 ms later, the two lines appeared, and participants were asked to decide which of the two was longer. Ninety-six trials were presented in total. Seventy-two were the trials of central interest, and 24 were the foils. Foils were presented to reduce the likelihood that participants would eventually realise that the lines were the same size and, as stated above, to induce uncertainty. All the trial types were presented in a pseudorandom order.

The University approved the experiments described in the present thesis of

Essex Department of Psychology ethics committee or the University of Essex faculty of

Science and Health Ethics Committee (Ethics numbers: ETH1920-0503, ETH2122-1943, ETH2223-0218, ETH2223-0216, & ETH2122-0164).

Results

The Motor Inhibition task

The mean error rate for the letter discrimination task was 14.0% (SD = 9). A one-sample t-test found that participants were less likely to make a repeat button press (39%) compared with the chance value of 50%, $t(17) = 4.3$, $p = 0.001$, $d = 1.0$ (see Figure 2). In other words, when a person has just made their response in discriminating the letter, they are then less likely to make the same response on the following line length judgement task. For example, if they just pressed the right-hand button (because the letter "B" appeared), they tended to indicate that the left line was longer; they had inhibited a correct response. Although repeat responses (726 ms) were 34 ms slower than "switch" responses (692 ms), these were not significantly different, $t(17) = 1.3$, $p = 0.22$, $d = 13$.

The Stroop task

One participant's data was not used for the Stroop task because they did not achieve 75% correct ink colour response accuracy[2]. With this participant removed, the mean error rate was 4.0% (SD = 6.5). A within-participant t-test revealed that it was incongruent.

[2] In Experiments 1-3, all participants are included in the analysis for the motor inhibition task, even one who generated 34% errors on the letter discrimination task. This is because the analysis for that task concerns whether the participant repeated a response or switched responses (irrespective of how they responded on the discrimination).

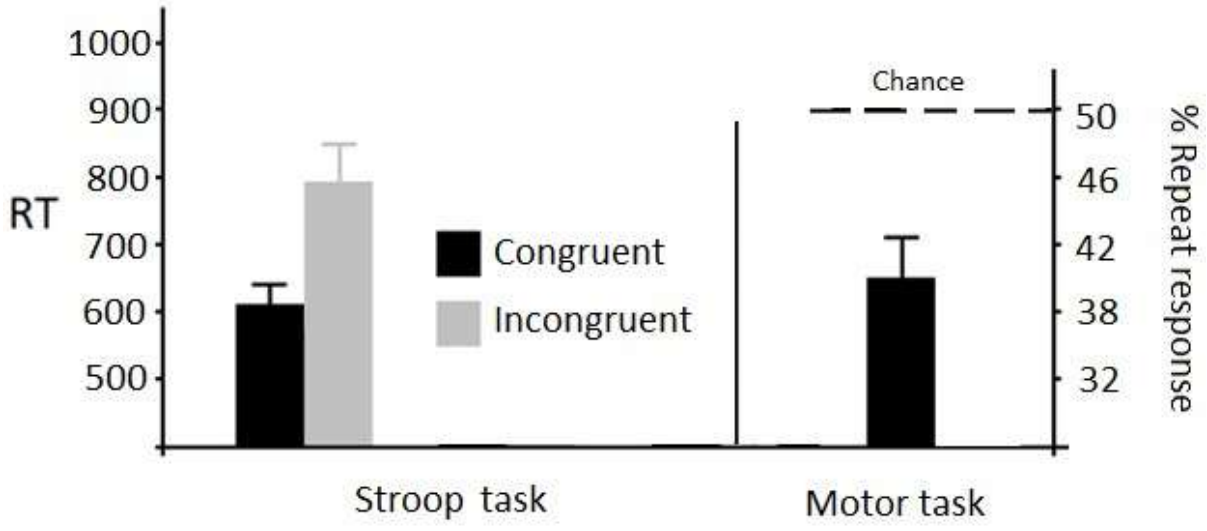

Figure 2 Results from Experiment 1. The bars show means with standard errors.

reaction times were significantly slower than congruent, t(16) = 3.9, p = 0.001, d = 0.72. Although there was a medium effect size (d = 0.66), error rates were not significantly different, t(16) = 1.9 p = 0.072, (congruent errors = 1.4% (SD = 2.0); incongruent errors = 6.0%, (SD = 9.2).

The results from Experiment 1 have shown that the motor-induced motor response effect is reliable. The data closely replicate those of Cole and Skarratt (under review, 2022). The results also show that the effect is robust enough to occur under the conditions in which it was carried out remotely. The same is true for the Stroop effect. This showed a significant 195 ms effect. Both findings give the present author confidence that the paradigms can be used for the present research.

Experiment 2

The present experiment

In Experiment 2, the two paradigms used in Experiment 1 were again employed. Indeed, the present experiment can be seen as a direct replication of the first study, except that the two tasks were each undertaken by a group of ADHD children and a control group. This experiment will explore the possibility that the attention deficit symptoms of ADHD are due to a failure of inhibitory regulation of lower-level or bottom-up inhibitory functions. By comparing the inhibition of a cognitive task with a motor task, we can categorise the failure of inhibition predicted to occur with the ADHD group into one of 3 cases: solely a cognitive inhibitory failure, solely a motor inhibitory, or some combination of both types if the ADHD was found to have difficulty with the motor task, and not with the Stroop task that would indicate a specific type of inhibitory control difficulty. Furthermore, and equally significant, this absence of inhibition should *not* occur on another response inhibition task but one that *does* involve higher-level cortical processes (i.e., the Stroop task). Such an overall effect would show that there is not an absence of inhibition *per se* but an absence that results from non-cortically based mechanisms.

Method

All aspects of the method were described in Experiment 1 with the following exceptions: 41 participants aged 14 to 18 years. The control group consisted of Twenty-five students, ten male and 15 female. The ADHD group consisted of 16 students, nine males and seven females. The two groups had comparable cognitive ability and age. The control group were selected from the academic middle sets of the school and can be

considered cognitively average, as measured by the standardised SATS test administered to all students. The ADHD group consisted of children with no identified comorbidities and the ADHD-C subtype. Their medication status could not be reliably obtained. They were also of average cognitive ability and had a formal diagnosis of the disorder on record at the school.

Concerning design, the Stroop experiment was a 2 x 2 mixed design with Group. (ADHD, control) and congruency (congruent, incongruent) as the two factors. As in Experiment 1, the motor-inhibition task compares the frequency with which a participant repeats a button press (after the letter discrimination task) against the chance value (50%) for both groups.

Results

The Motor Inhibition task

For the control group, the mean error rate for the letter discrimination task was 17.0% (SD = 14). A one-sample t-test found that participants were less likely to make a repeat button press (again compared with the chance value of 50%), $t(24) = 5.574$, $p = 0.00001$, $d = 1.1$. As Figure 3 shows, participants made a repeat button press in 38% of trials. In terms of reaction times to make repeat and switch presses, the difference

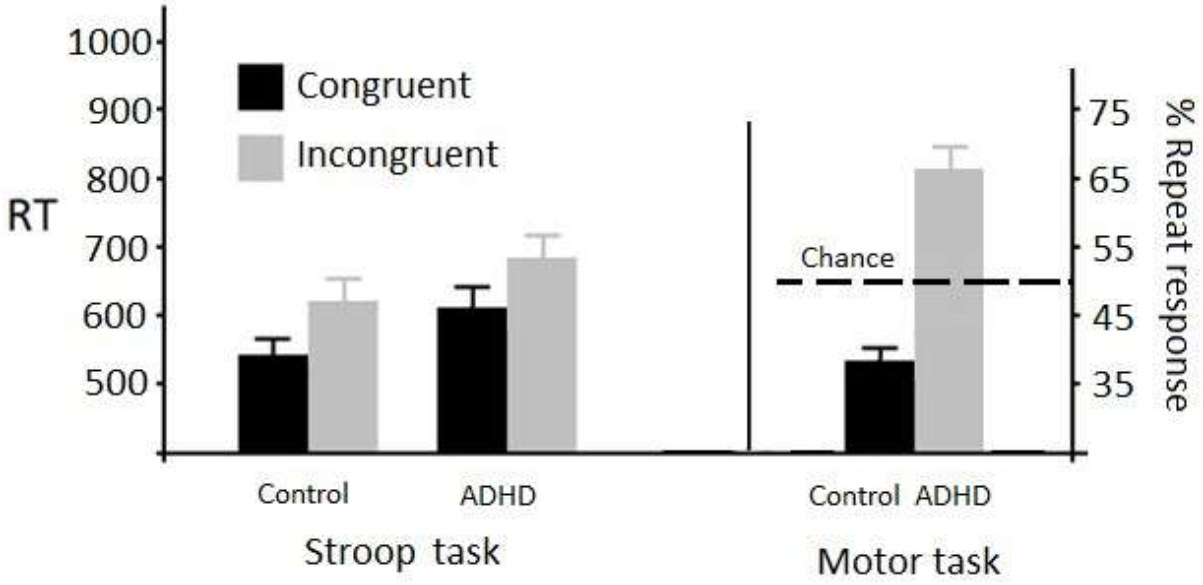

Figure 3. Results from Experiment 2.

was not signifiant, t(24) = 2.01, p = 0.06, d = 0.23. As with Experiment 1, repeat responses (743 ms) were slower than switch responses (720 ms).

For the ADHD group, the mean error rate for the letter discrimination task was 19.0% (SD = 11). A one-sample t-test found that participants were *more* likely (66%) to make a repeat button press than chance, t(15) = 3.4, p = 0.004, d = 0.85. RT had no effect on repeat and switch responses, t(15) = .256, p = 0.8, d = 0.04, (repeat response RTs = 719; switch response RTs =728).

The Stroop task

One participant's data was not used because they did not achieve 75% correct ink colour response accuracy. The means for the congruent and incongruent conditions for both groups are shown in Figure 3. A 2 x 2 mixed ANOVA revealed a main effect of

congruency, F(1, 39) = 7.6, p = 0.01, d = 0.6, and a main effect of group F(1, 39) = 5.858, p = 0.02, d = 0.63. The interaction was not however significant, F(1, 39) = .32, p = 0.57, d = 0.04.

Discussion

Experiment 2 has revealed that a group of typically developed adolescents show motor-induced motor inhibition. As with Experiment 1 and Cole and Skarratt (under review, 2022), they inhibited a motor response they had just performed, which manifested itself on the line judgment task. However, this effect did not occur in the ADHD group. Indeed, these individuals showed the *opposite* effect; they tended to repeat their responses rather than inhibit them. Furthermore, the Stroop effect in the ADHD group was no different to that of the control group.

This shows that lower-level motor inhibition was absent in the ADHD group, but inhibition that includes a range of high-level processes (e.g., Stroop) was intact. This suggests that the difficulty for the ADHD children concerned (motor) inhibition that does not involve higher processes.

Experiment 3

Experiment 3 was designed to test further the theory that the underdevelopment of structures within children can adversely affect motor inhibition. The same two paradigms were again used (i.e., the motor inhibition effect and Stroop), this time on a group of 10- and 11-year-old children. Since structures within children's brains are not yet fully formed at these ages, it is predicted that their inhibitory function is also not fully formed. These participants should fail to show a typical motor inhibition effect whilst retaining a typical Stroop effect. In effect, the present experiment assumes that, in terms of brain structural and developmental functioning, children aged 10-11 would have similar performance characteristics as Older ADHD children who have yet to develop. Young children can, therefore, be used as a proxy for ADHD; both have an underdeveloped motor inhibition system.

Method

All aspects of this were as described in Experiment 1 with the following exceptions:

There were 35 participants, all aged 10 or 11 years. They were selected from the same school as previously described.

Results

The Motor-Inhibition task

The mean error rate for the letter discrimination task was 23% (SD = 12). A one-sample t-test found that participants were *not* less likely to make a repeat button press (47%) compared with the chance value of 50%, $t(34) = 1.1$, $p = 0.3$, $d = 0.18$. The means are

shown in Figure 4. There was no difference in reaction times between repeat and switch responses, t(34) = 0.153, p = 0.88, d = 0.01, (repeat response RTs = 756; switch response RTs = 753).

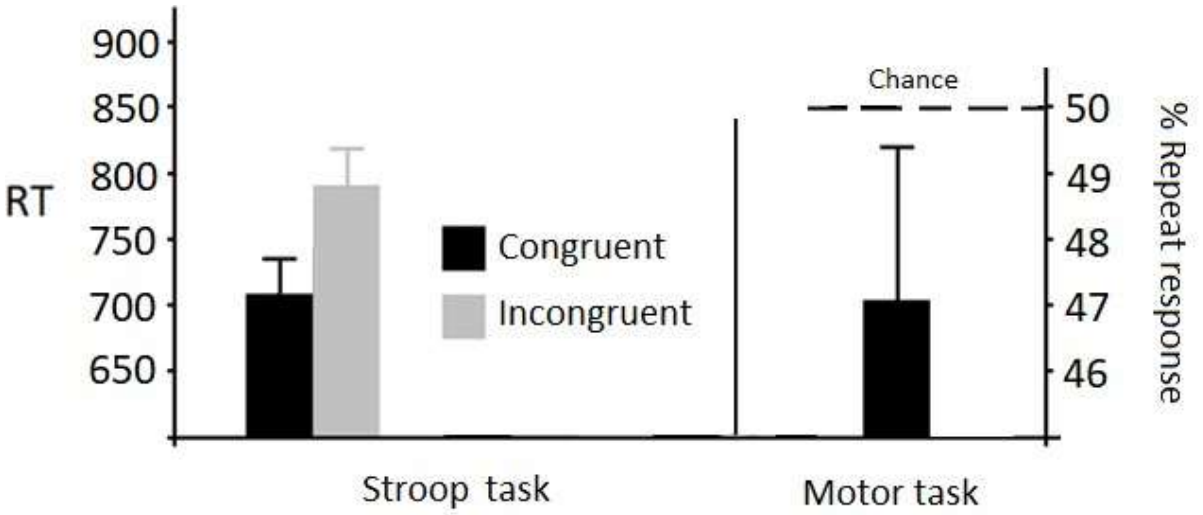

Figure 4. Results from Experiment 3.

The Stroop task

Three participant's data were not used because they did not achieve the 75% threshold for accuracy. A within-participant t-test revealed that incongruent reaction times were significantly slower than congruent, t(31) = 3.0, p = 0.005, d = 0.41. (See figure 4). There was also a significant effect of error rates, t(31) =7.1, p = 0.001, d = 1.0, (congruent errors = 6.2%; incongruent errors = 11.3%).

Discussion

Overall, the present experiment has found an absence of the motor inhibition effect but an intact Stroop effect. These results support that children aged 10-11 have

underdeveloped brain regions responsible for motor inhibition. These similarities to older children with ADHD warrant further investigation if one narrows down a specific area of the brain as a candidate for a centre of motor inhibitory control that is underdeveloped in children and people with ADHD. By themselves, these results are not strong and unambiguous support of the basal ganglia motor inhibition hypotheses; indeed, many other variables remain that could explain motor inhibition differences that are related to the brain's overall development with age, not exclusively differences sourced within the basal ganglia. However, it is interesting to note that the observed differences in inhibitory control only occurred between the two groups for the motor task, implying that overall brain development for cognitive inhibition was similar between groups except for the brain structures regulating motor inhibition. The children aged 10-11 in this experiment were also shown to have difficulty with the motor-induced motor inhibition task, as observed in the ADHD groups from Experiment 2. In contrast, all groups retained an inhibition effect that inhibits high cortical areas (i.e., Stroop). The fact that motor inhibition was absent for the participants in the present experiment suggests that children aged 10 and 11 show an absolute absence of the motor inhibition effect, as opposed to a false negative.

Chapter Discussion

ADHD is characterised, in part, by a failure to inhibit behaviour. The present chapter has examined the hypothesis that this failure is partly due to the structures in the brain that manage the regulation of motor inhibition either not fully functioning (ADHD group) or are not fully developed (age 10-11 group). Results have shown that when a task/effect requires cortically modulated cognitive inhibition (e.g., Stroop), the phenomenon is observed in both

ADHD and non-ADHD individuals, including children aged 10 and 11. In contrast, an inhibition effect that relies on “pure” motor processes with limited cortical involvement is this sound reasoning inhibition was not present in ADHD individuals, nor the young children. This suggests that the inhibition problems associated with ADHD may be due to compromised structures in the brain that regulate motor inhibition and not a generalised inhibitory dysfunction.

In sum, the present chapter has provided evidence that typical children aged 15-18 has a significant cognitive inhibitory effect, as expected in the Stroop task, and a significant inhibitory effect in the novel motor task. When this experiment was repeated with a control group and an ADHD group, the control group continued to have the same inhibitory effect. However, the ADHD group had mixed results.

The ADHD group showed a cognitive inhibitory effect in the Stroop task whilst not having a significant motor inhibitory effect. Finally, the younger students retain the Stroop effect but do not have motor inhibition.

Chapter 3

Motor Control

Motor control is an organism's ability to regulate movement using the nervous system to initiate, direct, and regulate purposeful movement. It is often considered to have driven the development of the brain (Wolpert, 2011). Movement is accomplished through integrating and managing sensory information to produce muscle movement to achieve an intention or goal. This action plan is coordinated with the central nervous system and is executed through the motor neurons in the brain stem and spinal cord to the muscles. As reviewed by Wolpert and Flanagan (2001), sensory feedback is supplied to the central nervous system by the movement in a feedback loop, which helps to regulate the movement. This regulation can take the form of modification, ending, or continuation of the movement until a decision that the movement has met the intended goal. The final step is to store the performance of the movement for future use when circumstances call for this motor movement.

Motor control in humans is a broad category that includes reflexes and directed movements. Voluntary movements can be further broken into two types: gross motor control (the subject of the present chapter) and fine motor control (the subject of the next chapter). When the nervous system is degraded in some way, as with ADHD, the impaired individuals may find it challenging to produce the functional motor movements necessary to accomplish their intentions and complete relevant tasks. In an educational setting, controlling motor movements is essential for tasks such as writing. In addition to being

linked to achievement, control of motor movements is seen as “good behaviour” in most educational settings.

Unlike fine motor control (e.g., picking up a small item with the index finger and thumb or writing), gross motor control coordinates large general movements such as throwing a ball or waving a hand. This set of skills is acquired during childhood and is a central part of the motor learning maturation process. The World Health Organization published a study (Onis, 2007) of gross motor skill developmental milestones that states that the ability to move using large muscles begins at birth, if not before, and is learned throughout childhood as the various skills develop (Onis, 2007). Children typically develop gross motor skills along a predictable timeline, starting with the ability to control the head and ending with controlling and manipulating the toes (Onis, 2007).

Theories of Motor Control

Motor control theories commonly desire to explain the production of reflexive, automatic, adaptive, and voluntary movements. These theories have considered the nervous system's information processing, including the sensory input, motor output, and central processing of multiple processing loops, all working simultaneously at multiple levels within the system. The main theoretical accounts are *Reflex Theory* (Sherrington, 1906), *Dynamic Systems Theory* (Bernstein, 1966; Turvey, 1977; Kelso & Tuller, (1984). Thelen, 1987), *Hierarchical Theories* (Adams, 1971), *Ecological Theory* (Gibson & Pick, 2000) and the *Systems Model* (Shumway-Cook, 2007).

The role of inhibition as a means of managing motor control was explored in this experiment. In this particular case, the timing of actions and the management of strength

require a level of control to complete the dice-rolling task successfully. Suppose a person cannot utilise inhibitory functions, even with the exertion of mental effort, a characteristic of ADHD. In that case, there should be a significant difference in performance between AHDH participants and their typical peers.

Reflex Theory (Sherrington, 1906)

Sherrington's influential book, *Integrative Action of the Nervous System,* contained the idea later called the *Reflex Theory of Motor Control*. He coined the term synapse and established the idea of inhibition in neuronal function (Elsevier, 1965). As noted by Levine (2006), his text *The Integrative Action of the Nervous System* also provided the conceptual framework for a century of research on the mechanisms of synaptic transmission and the neuronal discharges associated with perception and action. Reflex Theory sought to demonstrate how the reflex is the most straightforward unit of nervous system integration. Sherrington was the first to propose that the synapse was where the reflexes interact. Another key idea is that a synaptic nervous system was a key evolutionary innovation that would later provide the neurological building block for further evolutionary developments within a broad central nervous system, such as the cerebrum and cerebellum. Inhibition at the neuronal level would later be expanded to inhibitory neural pathways throughout the brain and inhibitory functions that manage data movements throughout the brain and central nervous system, including motor inhibition.

Hierarchical Theory (Adams, 1971)

Hierarchical Theory proposes that movement control is entirely top-down, i.e., it never results from a bottom-up stimulus-driven process. As noted by Broadbent (1977),

Hughlings Jackson emphasised the hierarchical nature of the brain by pointing out that higher, middle, and lower levels of control exist that correspond to the motor cortex and spinal areas of motor function. This theory was developed in response to the Reflex theory of Magnus (1925). Magnus examined the functional reflexes of individuals at different ages and found that certain reflexes were only present when cortical damage was present. This implied that reflexes were part of a motor control hierarchy, with higher centres inhibiting lower reflex centres. Continuing this line of reasoning, Schaltenbrand (1928) noticed that motor control seems to develop with the appearance and disappearance of complex reflexes. These reflexes were organised around a progression of hierarchically organised reflexes that emerged and replaced primitive collections of reflexes. He also noticed that damage or pathology in the brain may freeze development at the point of injury. In 1938, Weisz proposed that reflex actions were organised hierarchically. He collected data on children's developmental ability to sit, stand, and walk. These observations and experiments coalesced into a theory of motor control that proposes reflexes as being nested within the central nervous systems' hierarchical control (Weisz, 1938). Gesell and Armatruda (1941) provided more detailed descriptions of the reflexes seen in infants as they matured. As the infants' movements developed, this was attributed to the maturation of the central nervous system. During this development, layers of hierarchy developed, giving better control over the nested lower-level reflexes.

The theory proposes that higher centres of the central nervous system control and regulate subordinate parts of the nervous system. This theory rejects the monosynaptic stretch reflex and other bottom-up reflex movements as not entirely outside top-down control. At the core of the theory is the nervous system's organisational style, which

comprises a hierarchy of descending commands built into the nervous system, which includes a series of nested reflexes within the hierarchical system's ultimate control. Meaningful or volitional movements are started spontaneously and then regulated by sensory feedback nested within the various reflexes.

In Experiment 4 (see below), the dice-rolling task requires a specific amount of force from the gross motor action that engages the arm. To succeed in this task, the hand and arm must complete a roll that is inhibited enough to keep the dice on the workspace. Keeping a die on a table becomes a salient sensory feedback reference as feedback from prior rolls is processed with inhibitory adjustments being made on the fly. As the individual rolls the dice, they will discover that after the initial spontaneous action of the first attempt, they either met their goal or did not. If it is the case that they rolled the dice off the table, a typical child can inhibit multiple levels of force by dialling back the strength of the roll. Many levels of force need to be managed because all the nested muscle systems are employed to roll a die. This task is similar to other motor tasks that require a specific level of inhibitory control over the lower nested motor reflexes. In other similar tasks, Bobath (1948) and Brunnstrom (1956) found that damage to the brain contributed to symptoms that included difficulty inhibiting motor actions to complete motor tasks successfully.

Bobath (1948) and Brunnstrom (1956) proposed that when damage to higher centres of control occurs, lower centres of control manage movement, resulting in less intentional control. Their work led to the development of physical therapy to assist stroke victims and people living with cerebral palsy. Strict adherence to hierarchical control has been modified to accommodate known instances of bottom-up control (Shumway-Cook & Woollacott, 2007); however, it has been recognised that each level of the central nervous

system can control subordinate levels and are task-dependent. This modification to a strict top-down hierarchy allows for the flexibility of a system to complete motor movements that can be matched with observation. It should also be noted that reflexes are no longer considered the sole determinant of motor control. Reflexes are now seen as one of many tools available to the central nervous system that can be deployed to generate and control motor movements (Shumway-Cook & Woollacott, 2007).

Systems Model (Shumway-Cook & Woolacott, 2007).

Systems theory proposes that movement results from the interaction of three central factors: the task, the individual, and the environment. These three factors are developed within themselves as a resulting constraint created by the interaction of the constituent parts of the significant factor. Tasks that require movement are thus broken down into three attributes. The resultant functional movement is partially affected by the result of the individual to manage interactions between stability, mobility, and manipulation. Stability is the ability to regulate posture during the commission of a task. Mobility is the gross motor movements of the body within the larger environment. Manipulation is the ability of the individual to engage objects in the environment, although this tends to be associated with fine motor skills.

The individual's general factors contributing to functional motor movements are cognition, perception, and action. As the name suggests, the latter is an individual's motor capabilities. Body mechanics constrains functional movements as they relate to the intended action. The peripheral nervous system, cerebellum, basal ganglia, and motor cortex also apply the constraint on functional action. Perception constrains motor movement by detecting relevant sensory information that regulates a motor task. This is

accomplished through feedback during the motor action. It can be visual, auditory, vestibular, or haptic feedback. Cognition plays a role in motor movement by considering the individual's ability to manage and access attention, motivation, emotion, learning, and memory whilst completing a functional motor movement. With this model, it is essential to recognise that motor impairments can be related to an individual's memory, attention, or motivation impairments, leading to atypical motor movements or an inability to perform them. The symptoms of ADHD include difficulty with attention and hyperactivity, a type of atypical motor movement. This theory of motor movement is ideally suited to explaining the symptoms of ADHD.

The final factor comprises regulatory and non-regulatory constraints derived from the environment (Gordon, 1987). Regulatory constraints are features of the environment that constrain the form/organisation and function of the motor movement. For example, when someone is walking on ice, one cannot walk the same way one could on a dry pavement. The individual must thus change many functional movements to compensate for the constraints of the environmental conditions. The environment's regulatory characteristics constrain how the body can act when completing a motor movement. Nonregulatory features are constraints that can affect motor movement, but movements are not forced to conform. A person may stop crossing a street due to the sign indicating that they should not, or they may stop talking in a library due to customs. Consequently, this theory has top-down control as a central feature.

Central to the theory is the interaction of motor actions within a dynamic environment. Each cell is thought to play a role in the movement, each having an almost infinite number of possible outputs or degrees of freedom. To manage this near-infinite

degree of freedom, the researchers who contributed to the model attempted to find the organising factor of the movement for each cell involved in the movement and how the organising principle was regulated and directed in a coordinated way that was also responsive to the environment. Kugler, Kelso, and Turvey (1980) stated that the constituent parts of the motor movements that each cell is responsible for are unified by a self-organising principle. These individual parts come together and behave collectively without needing a higher organising centre.

In sum, the central claim of this model is that movement is an emergent property resulting from the interaction of multiple elements. The behaviour results from self-organising principles based on the properties of those elements and modulated by environmental or neurological constraints. As these constraints change, so do the self-organising principles associated with them. However, neural structure does play a less critical role in this model as the constraints apply the self-organising principle separate from neurological control derived from top-down processing.

Gross Motor Control in ADHD

In a meta-analysis of motor skills, Kaiser et al. (2015) found that motor skills are often impaired in ADHD across a spectrum of impairment, deficits in both fine and motor skills are prevalent, and more than half of ADHD can improve their motor skills with medication. Finally, motor skills deficits can be attributed to a comorbidity of inattention and lack of inhibition. These findings predict that ADHD children should have difficulty with motor skills tasks such as the die rolling task.

Harvey et al. (2009) reported that children with ADHD had difficulty combining movements required to play sports and performing individual movements by assessing

gross motor skills in activities such as ball catching (Ho et al., 1996). Harvey and Reid (1997) also found that children with ADHD scored lower on the performance criteria of locomotor skills than control groups. The skills measured included the ability to run, gallop, hop, leap, horizontal jump, skip, and slide. Also measured were object control skills like stationary bounce with a ball, catch, kick, and overhand throw. Later evidence from Harvey and Reid (2005) further demonstrated that many children with ADHD have difficulties performing locomotor and object control skills.

Hung et al. (2013) examined the relationship between motor ability and inhibitory control in children with ADHD using a neuroelectric approach. The authors asked 32 children (mean age of 8.9 years) to complete a battery of tests included in the BMAT (Basic Motor Ability Tests revised). The BMAT includes seven subtests, with each subtest assessing different motor abilities. For example, a basketball throw for distance was used to test arm and shoulder explosive strength, bead stringing to test bilateral eye–hand coordination and dexterity, target throwing to test eye–hand coordination associated with throwing, marble transfer to test finger dexterity, back and hamstring stretch to test the flexibility of the back and hamstring muscles, a standing long jump to test the strength and power in the thigh and lower leg muscles, and ball striking to test coordination associated with striking. In addition to the BMAT, a Go/No-Go task was also administered to these same participants. Analysing event-related potentials (ERPs), Hung et al. (2013) found that ADHD children with better motor abilities exhibit better inhibitory control than ADHD children with worse motor abilities, suggesting a mitigating effect of inhibition on behaviour.

Chen et al. (2011) asked children with ADHD aged 10 to bounce a ball higher than the shoulder but not higher than the head. The “bounciness” of the ball was varied

between a soft ball, a medium ball, and a hard ball by deflating them. When throwing the hard ball, the ADHD group showed more variable force when striking the initial bounce area than the control group, resulting in more varied heights. Chen et al. (2011) stated that "children with ADHD have a poor ability to maintain stable force control performance when throwing balls and that ADHD participants showed poor performance with low overall success rate when throwing balls."

As used in the present experiment, the gross motor skill of dice rolling requires whole-arm use and, as with throwing, a locomotor action in the wrist to control downward movement. Similar to the target throwing task in the BMAT and the ball throwing experiment used by Chen et al. (2011), movement is limited by the particular goal, in the present case, limiting the strength of a throw to prevent dice from rolling off a table. In a pilot study, Chen et al. (2011) showed that motor tasks are difficult for ADHD children to regulate. They also noted that it is problematic for such children to regulate motor actions when the objects react energetically (i.e., a hard ball, marble, or a dice). Hung et al. (2013) measured the participant's inhibitory control on a spectrum using event-related potentials. Students with higher levels of inhibitory control were found to have higher levels of motor control.

Finally, Barkley (1997) stated that children with ADHD have self-regulation difficulties and deficits in the ability to proceduralize or perform known motor actions. Barkley argues that their "declarative knowledge", or the gross motor actions that a child with ADHD describes, often differs from the gross motor action an outside observer sees. When the ADHD person describes their intended action, it is often the correct action, which Barkley referred to as a correctly proceduralized action. The difference between the

"declarative knowledge" and the actual action is attributed to that particular individual's inhibitory control. This suggests that top-down processes remain intact; however, the bottom-up execution of gross motor actions, specifically those nested actions required, for instance, to roll a die, cannot be "proceduralized" due to dysfunctional inhibitory control (Hung et al., 2013). Barkley (1997) argued that "ADHD is more a problem of doing what one knows rather than knowing what to do". In other words, Barkley (1997) argued that children with ADHD had access to the schema of the dice-rolling action. However, the motor actions to deliver the scheme suffered from diminished behavioural inhibition of the executive functions that regulate the implementation of the specific action, specifically the ability to respond to feedback from prior rolls. In the present dice rolling experiment, children know how to roll the dice, but they find it difficult to dial in the strength required to keep the dice on the table in a new or novel circumstance, even with the schema of dice rolling action intact. They are aware of the ability to inhibit the strength of the motor action of the roll, as indicated by attempts to change their roll strength. In the experiment, a reward was offered to those who could achieve the goal to encourage and help motivate. I argue that in the experiment, the ADHD participants, compared to controls, do not have enough inhibitory control over this specific nested motor function located lower down the motor control hierarchy, as proposed by Bobath (1948) and Brunnstrom (1956). As the inhibitory control to regulate the strength of the throw is outside of top-down control, the action is dysfunctional due to diminished inhibitory functionality. A ball skills test conducted by Piek et al.(1999) found a significant deficit in gross motor ability for the ADHD-C group compared with the comparison group. Pitcher et al. (2003) used different ball skills and a Purdue pegboard task measuring finger and hand dexterity. They found that the outcomes

for ball skills (gross motor) and pegboard skills (fine motor) supported the prediction of a greater performance deficit for the ADHD-PI and ADHD-C groups compared to the comparison group.

ADHD and Spatial Cognition

Feldman and Huang-Pollock (2020) investigated the spatial cognition performance of 99 ADHD children aged eight to twelve years by comparing their performance with 207 non-disabled peers using a mental rotation task. They found that the performance of children with ADHD was less accurate and variable than non-ADHD controls, but there were no group differences in mean response time to trials in the task.

Children were asked to pick a rotated stick figure with two salient features: a yellow dot on the right hand and a black dot on the left foot. The stick figure was rotated in quarter turns randomly around a circle, with some stick figures being congruent to the original upright one, whilst others being incongruent with their salient features. ADHD children did not take longer to make their choices but made fewer correct choices.

Feldman and Huang-Pollock (2020) found that the poorer performance on the mental rotation task was due to the slow rate of evidence accumulation and relative inflexibility in adjusting boundary separation, but not to impaired visuospatial processing specifically.

These findings are relevant to the die-rolling experiment. The die-rolling experiment was a natural experiment with high ecological validity, meaning that any detected effect will not be a subtle one thought at the expense of possible extraneous variables. Without a set time between rolls of the dice, participants would have as much time as needed to

accumulate the evidence required to prepare for the roll. This may control for spatial working memory deficits prevalent in ADHD participants. Meta-analytic effect sizes for spatial working memory deficits in ADHD are among the largest across all neurocognitive domains (.63–1.06) and are more significant than effect sizes for verbal working memory (.43–.55) (Martinussen et al.,2005; Hayden et al., 2005; Willcutt et al., 2005), and have been shown to persist even after controlling for IQ, spatial memory span, and other rapid processing tasks (Lin et al., 2014).

Visual-spatial cognition in ADHD is not significantly different from their non-disabled peers (Feldman & Huang-Pollock, 2020; Silk et al., 2005; Vance et al., 2007; Williams et al., 2013); however, error rates persist in visual rotation tasks. This further strengthens the argument that spatial working memory is the primary driver of errors in spatial cognition when movement is triggered due to more preparation times being required to rotate an image that uses spatial working memory, which is known to be less efficient in individuals with ADHD.

The Social Context of Motor Movement for Those With ADHD

Goulardins et al. (2015) state that psychosocial problems, including ADHD, are hypothesised to be secondary consequences of motor problems (Cairney et al., 2013) and tend to appear once a child faces social and peer demands. This is apparent in early school years (Piek et al., 2008). Longitudinal studies show that motor skill difficulties earlier in childhood are related to later psychosocial problems (Lingam et al., 2012; Losse et al., 1991). Participation in activities like sports and play also promotes the development of supportive interpersonal relationships and social ties in children (McHale et al., 2001). This creates a problem for those who experience difficulties with movement skills, which are

symptomatic of individuals with ADHD. Research has shown that children with motor problems are less likely to participate in organised (e.g., sports teams) and unorganised (free play) activities (Cairney et al., 2005), further alienating them from their peers (Boyer et al., 2014). Children with motor problems appear aware of these difficulties as they have reported lower levels of perceived social acceptance and support

(Boyer et al., 2014; Skinner & Piek, 2001).

Researchers have found a connection between motor difficulties and ADHD, which is thought to be around 50% (Fliers et al., 2008; Pitcher et al., 2003). Numerous studies also link ADHD symptoms with problems in social functioning as a result of motor difficulties (Frederick & Olmi, 1994; Marques et al., 2013; Nijmeijer et al., 2008; Schlack et al., 2014; Wehmeier et al., 2010).

The main body of evidence suggests that social context is not the driver of poor motor performance of ADHD participants. However, ADHD can drive poor motor performance, which in turn drives behaviour difficulties in school which has been established by results of Goulardins et al. (2015) that showed that ADHD and social problems are related, which is also consistent with the previous findings of (Antrop et al., 2002; Coates et al., 2014; Fagan & Fantuzzo, 1999).

Experiment 4 was conducted in a setting familiar to the participants. The motor action was a naturalistic one. The children could have declined the activity, like any other school activity. However, none chose to, implying enough confidence in their motor skills to attempt the task of dice rolling 60 times without behaviour dysregulation in the classroom.

In this instance, students seemed to enjoy the activity but did not indicate that social context affected performance.

Experiment 4

Method

Participants

There were 178 participants, all students at the same secondary state school previously described. They were members of the middle sets, meaning they were cognitively average as measured by their scores on the SAT taken in Year 6, which is used to place children into the middle set at the school. Students from Year 7 to Year 10 were present in the study. The age range of the students was, therefore, 11-14. Eighteen students had been diagnosed with ADHD, as recorded in school data.

Stimulus and apparatus

Students were asked to roll a standard 6-sided dice 60 times during a Maths lesson. All students were given the same type of dice. All students were seated behind the same desk that measures 180 cm in length and 150 cm in width.

Design and procedure

Student were told to roll their dice 60 times. A partner recorded the outcome of their rolls in their exercise book (i.e., the face-up number). From the student's point of view, the exercise was to calculate the average number of the 60 rolls. However, one of the instructions was to keep the dice on the desk. Students who successfully kept their dice off the floor were rewarded with achievement points that are part of the school's more

extensive rewards system. The author recorded all occurrences in which the dice rolled onto the floor. Typically, each session (i.e., each class) comprised 20-30 students.

Results

The mean number of times that the ADHD group rolled the dice off the table was 4.7 (SD = 3.5) times. For the control group, this figure was 0.69 (SD = 1.21). These values were significantly different, $t(176) = 9.48$, $p < 0.001$.

Discussion

Results from the present experiment have shown that children with ADHD are not as able to regulate the strength of their dice rolls to keep them on the table. This is consistent with the ball-bouncing experiment of Chen et al. (2011). These results also concur with the findings of Hung et al. (2013), who reported that children with poor inhibitory control, as measured on the Go/No Go task, had poor motor control as measured by the BMAT. Returning to Barkley's notion that "ADHD is more a problem of doing what one knows rather than of knowing what to do", the dice-rolling task is something a student knows how to do; they have conceptual cognitive control over the action and can, therefore describe the "performance of the action". It is safe to assume that children have rolled dice many times before being asked to perform the task in a Maths class. However, in many cases, children have not been asked to perform a novel action, such as rolling a die within a specific parameter that requires a level of inhibitory control to regulate the strength of the throw. In future experiment iterations, confounding variables like spatial judgements that direct the participant's dice throw could be controlled for, mainly where to start the throw.

Other considerations could be to what extent the size of the table could affect the outcomes of ADHD children when compared to the control group.

In Chen et al. (2011), children were asked to keep a ball bounce to a specific height. All the children, both the control and ADHD groups, could bounce the ball. They knew what was required of the "performance". Some of the actions required were present. For example, they could change the height of the ball between bounces, but they could not "dial it in" to bounce just between the shoulders and top of the head.

The findings of the present experiment also support the findings of Bobath (1948) and Brunnstrom (1956), who reported that by working with stroke victims to practice actions, they could reconnect damaged neural networks with the lower nested function to perform old actions again. It should be noted that Harvey et al. (2009) found that no amount of practice for boys with ADHD improved their ability to regulate motor control in sports. The authors demonstrated that stimulants could not improve the motor skills of children with ADHD. Other research suggests that the movements children with ADHD generate are quantitatively different from those of control groups (Beyer 1999; Christiansen 2000; Doyle et al. 1995; Harvey & Reid 1997; Kaplan et al. 1998; Miyahara et al. 1995; Miyahara, Piek, & Barrett 2006; Pelham et al. 1990; Piek, Pitcher, & Hay 1999; Pitcher, Piek, & Hay 2003; Wade 1976), meaning that, unlike stroke patients, no amount of practice will help individuals with ADHD gains inhibitory control over lower nested motor functions. The aetiology of ADHD must be something that cannot be repaired with practice. No amount of practice in dice rolling or any other gross motor skill should improve an individual's ability to regulate motor actions. This could affect how students with ADHD are managed, as teachers often think that ADHD children are "hyperactive" and need to participate in sports

to put that movement “to good use”. However, Harvey et al. (2009) found that for those with ADHD who participate in sports, the experience is not often positive socially, with slight improvement in their competitive ability.

Results from Chen et al. (2011) and Hung et al. (2013) are good predictors of motor control. ADHD individuals are known to have poor motor control, which has manifested in dice rolling. It should also be noted that the dysfunctional aspects found in the dice rolling control are linked to bottom-up nested motor actions. As Andersson and Grillner (1983) reported, these actions can be separated from top-down control in a threshold/step-like manner. For ADHD children, these nested motor actions are not being regulated by the basal ganglia through the feedback loops that would inhibit these nested functions. Without these inhibitory circuits, these nested motor actions lack refinement of action from sensory feedback, resulting in a lack of control that cannot be improved by practice.

Chapter 4

Fine motor control

Writing is a ubiquitous form of visual communication comprehended through reading. Handwriting is successful when an individual masters the necessary motor movements to produce the written conventions of letters, symbols, printing or connected script. Idiosyncratic production of handwriting is often mitigated through standardised education, emphasising “neat” handwriting and an educator’s anecdotal judgement of the individual’s mastery of the standard form of the intended letters or shapes. The production of this set of handwritten conventions is often used as a starting point for young people to begin their development of written communication, and triumphant mastery is used to judge academic success. Lines that are straight, vertical, horizontal, and diagonal, as well as more complicated objects like circles and triangles, are often shapes that individuals begin to produce in a standardised form. They then move on to letter production, word production, and sentence writing. Sassoon (1990) stated that “People present themselves to the world through their handwriting and are inevitably judged by it. From our earliest days, success and failure are often measured in terms of neat handwriting” (Sassoon, 1990). It has been a long time since Sassoon’s statement. It could be argued that handwriting has been given less emphasis because other forms of written communication, such as typing or texting, have become more prevalent in our information age of mobile phones and computers. However, handwriting remains an essential aspect of student progress in education for cultural reasons and as a measure of academic attainment (Santangelo & Graham, 2016).

Motor control is significant at the beginning of a child's developmental journey, in which writing needs to be efficient. I argue that deficits in motor control are apparent early in the development of motor movements. This includes the movement required to form the letters, shapes, and symbols largely disconnected from words and more cognitively complex collections of written language. Difficulties in motor control at this beginning point will have an important effect. The research reviewed below argues that individuals focus on motor movements when learning to write and move on to more sophisticated writing when the skills have been mastered. However, if one cannot completely master the motor movements, constant negative interactions regarding poor handwriting could cause attainment delays over a lifetime.

Centres of control within the brain for handwriting

Before neural imaging, identifying brain areas responsible for handwriting was accomplished by studying dysgraphia and agraphia, usually following a lesion or disease. Research assessing the specific parts of the brain that control handwriting started with sufferers of dysorthographias, with this work tending to examine lexical and phonological processes (Nicolson & Fawcett, 2011). Brain imaging work (Roeltgen, 2003) identified the brain regions responsible for the retention and preservation of letter formation for individuals with apraxic agraphia. This disorder prevents the participant from tracing learned shapes and letters that were known before acquiring their injury. This research strongly implied a separation of linguistic and motor function due to the difference in lesion location. Work with lesion-based dysorthographians also established brain areas responsible for subordinate parts of the functional behaviour of handwriting (Chung et al., 2020).

As technology improved, other methods would be developed that further helped to locate brain regions responsible for handwriting. Using Positron Emission Tomography, Petrides (1995) observed brain activity whilst participants wrote dictated words. Nakamura et al. (2000) used functional Magnetic Resonance Imaging to examine the neural substrate underlying the mechanisms of Kanji and Kana writing, which has a linguistic feature unique to the Japanese language (see also: Katanoda, Yoshikawa, & Sugishita, 2001; Nakamura et al., 2012; Omura et al., 2004; Sugihara et al., 2006). Harrington et al. (2007) showed that drawing an object helped people with aphasia, although, intriguingly, writing words diminished the accurate naming of the same object. Harrington et al. (2007) also provided evidence that drawing activates the brain's right hemispheric and left perilesional regions. The undamaged right hemisphere could 'support' the damaged left hemisphere in aphasic individuals. Roux et al. (2015) argued that the support for semantic recall in the left hemisphere could be connected to stimulation derived from the drawing activity mediated by the right side. This also explains why writing could not support an individual with object recognition difficulties owing to the location of the lesion and the centre of linguistic recall being within the left hemisphere.

Handwriting and motor control

Handwriting analysis of neurological disorders has shown that individuals with neurological impairments are more likely to have difficulties with fine motor control, resulting in poor handwriting. These disorders include Alzheimer's (Cilia et al., 2019), Tourette's (Mitchell et al., 2020), Parkinson's (Cilia et al., 2019), ADHD (Capodieci, Lachina, & Cornoldi, 2018), And mild cognitive impairment (Chai et al., 2022). An explanation for the connection between poor handwriting and motor-related neurological disorders comes

from an analysis of the requirements of the central nervous system to produce handwriting. Handwriting has two components. The first is a linguistic component, and the other is a motor control component. One could imagine a spectrum of graphomotor control as a function of these two components mapped onto two axes. The horizontal axis is the level of neurological resources reserved for linguistic cognition, and the vertical axis is the level of neurological resources devoted to motor control. As a child develops automaticity in motor control, these neurological resources can be diverted to linguistics. Graphomotor behaviours that require the least linguistic resources are called drawing; those that require substantial linguistic resources are typically called handwriting. The identified differences in the quality of the handwriting are attributed to the overall amount of neurological resources available to an individual to complete the required graphomotor handwriting behaviours. They are dependent on an individual's stage of development.

It is known that children who find it challenging to master the motor components of handwriting do not have good academic outcomes (Feder & Majnemer, 2007; Graham & Perin, 2007; Bangert-Drowns et al., 2004; Greenwald et al., 1999). An exciting implication resulting from the identification of brain areas responsible for linguistic and motor function of handwriting seems to be a conflation or assumption, mainly from lawmakers and teachers, that when a person can adequately construct words following the mastery of the motor skill component of handwriting, improvements in the fluency of writing will follow. In other words, written expressive language will improve or develop only after the individual has mastered the functional motor skills that result in writing. As noted, good handwriting includes accurate formation of shapes and letters, orientations, and size, and finally, the production speed of handwriting if we only focus on the output, not the writing content.

However, word writing encompasses processes that include sensory inputs, both auditory and visual, as well as an assumed shared orthographic representation of words (Folk et al., 2002) and a graphemic memory buffer (Cloutman et al., 2009). A further issue is the idiosyncratic way each forms letters through a unique series of motor programming of the muscles (Van Galen, 1991). It is along these lines that Planton et al. (2013) stated, "Neuroimaging studies should be able to disentangle writing/spelling process (i.e., conceived as the preparation of a message and its conversation into the graphic form) from the unrelated input of linguistic processing, and from the non-specific motor movements."

Being able to grasp a pen to draw or write typically requires the coordination of the index finger and thumb. This ability is seen as a milestone in infant development and signifies that the brain and muscles are beginning to work together to execute motor movements with volition. A typical child will acquire this ability at 9-10 years. The successful completion of a pincer movement is at the end of a series of developmental building blocks, beginning with the so-called palmer grasp. This occurs when the infant can curl their fingers around an object against the palm; the movement needed to grasp onto the handlebars of a bicycle. The raking grasp typically develops next and consists of a motion that brings items toward the infant by curling the fingers over the object and often dragging the object closer. The inferior pincer grasp uses the thumb and index finger to hold objects.

Biotteau et al. (2019) attempted to isolate the kinematic and dynamic variables contributing to handwriting movements. These idiosyncratic variables include hand posture, pen grip force, and pen tilt -- all of which are mitigated to a certain extent through teaching. For example, students are taught to hold a pen "properly", which tends to be

between the thumb and index finger. Another point made by Biotteau et al. (2019) concerns fluctuations in the mean speed, resulting in abnormal handwriting fluctuations. This is particularly noticeable in individuals with developmental coordination disorders, dyslexia, and individuals with ADHD (Biotteau et al., 2019).

A standard developmental test to assess writing readiness before entering reception is the Beery-Buktenica Developmental Test of Visual-Motor Integration (VMI). As the name suggests, the (fine) motor aspect of writing is emphasised. This tool has become one of the most commonly used by paediatric occupational therapists (Bagatell et al., 2013; Kennedy et al., 2012; Burtner et al., 2002; Dunford et al., 2013; Feder et al., 2000; Watling et al., 1999). The Beery VMI is a standardised norm-referenced assessment requiring the examinee to imitate and copy a series of progressively more complex forms. The tool gives nine shapes in chronological order, starting at age two and ending at five years three months. The Beery VMI battery consists of two additional supplemental tests: the Developmental Test of Visual Perception and the Developmental Test of Motor Coordination. When all three tests are administered together, the examiner can compare the examinee's visual and motor abilities and subsequently target one or more of these performance skills during the intervention. Notably, using a dichotomous classification of assessments as either top-down or bottom-up, where bottom-up assessments are defined as examining small components of a child's skills and top-down assessments focus on activities and participation (Brown & Chien, 2010), the Beery VMI is categorised as a bottom-up assessment.

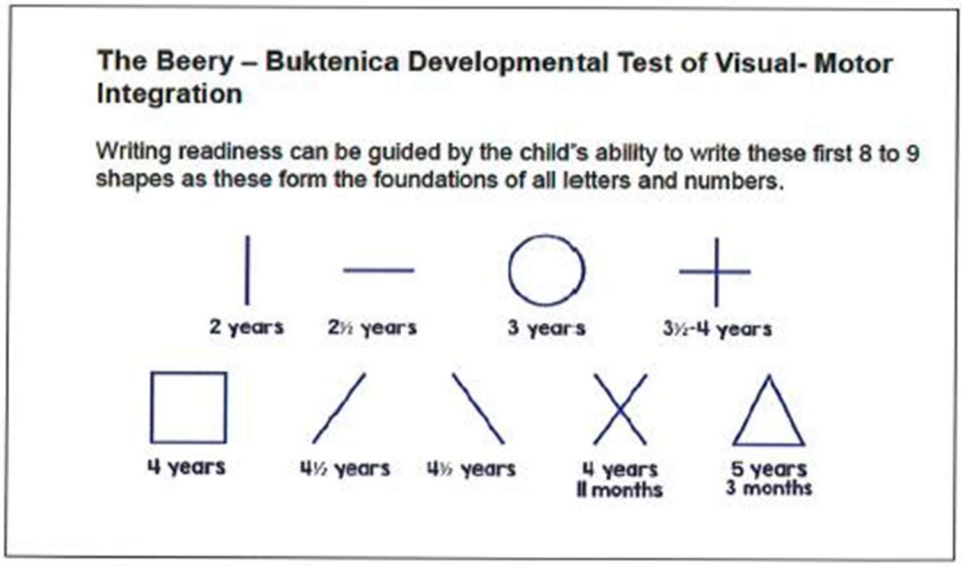

Figure 5. From VMI Administration, Scoring and Teaching Manual. 6th Edition (Beery, 2010).

Early research into drawing development consisted of analysis of children who were asked to copy drawings (Bernbaum et al., 1974; Nino & Leiblich, 1976; Simner, 1981; Smits-Engelsman et al., 1998). Specifically, these researchers assessed the importance of automating children's graphomotor skills to free up cognitive resources for the linguistic requirements of handwriting (Plamondon et al., 2013). Further research into the diagnosis of writing disorders described a funnelling effect of a child's expressive language in writing due to the constraints of the graphomotor component of handwriting (Zesigner,1995; Hamstra-Bletz & Blote, 1990).

Inhibition

As with other motor skills, handwriting relies on inhibitory processes. Hu et al. (2016) identified the middle frontal gyrus as a region that connects orthography and motor programs specific to handwriting. It also showed that it plays a role in inhibitory control.

The authors also showed that the nondominant (right) superior frontal gyrus is involved in impulse control and that its activation modulates inhibitory control and motor urgency, an essential aspect of control when handwriting (Hu et al., 2016). This region is associated more broadly with literacy and numeracy, with the dominant (left) middle frontal gyrus playing a vital role in the development of literacy and the nondominant (right) middle frontal gyrus being responsible for numeracy (Koyama et al., 2017).

Using Activation Likelihood meta-analytical methods, Planton et al. (2013) reviewed 18 neuroimaging studies to identify brain regions used in handwriting, regions that also play a role in inhibition. Tasks from the study were placed in two categories: non-specific motor/output control and linguistic control. The imaging revealed extensive use of the left hemisphere and sub-cortical areas. The authors concluded that the primary region used for writing occurred in the left superior frontal sulcus/middle frontal gyrus area, left intraparietal sulcus/superior parietal area, and right cerebellum. The primary motor and sensorimotor cortex, supplementary motor area, thalamus, and putamen manage the motor movements used in the creation of handwriting. The linguistic components used in handwriting activated the ventral premotor cortex posterior/inferior temporal cortex. These findings supported a tripartite division of handwriting, with writing, motor, and linguistic divisions working together (Planton et al., 2013).

Objective Measurements of Handwriting and Drawing

Standardised objective measures of handwriting generally come in the form of evaluation scales. These scales are divided into global readability and analytically based evaluations. Global readability is a qualitative judgement of the subject's handwriting. When evaluating global readability, the individual rating the handwriting will often use

evaluation scales to form an overall judgment of a written sample by comparing the readability of the subject writing to a group of standard handwriting samples previously graded from “readable” to “unreadable.” Thorndike (1910) developed the first known scale of global readability, which he called the “general merit” scale of handwriting, where he provided exemplars of “good merit” handwriting to teachers so they could compare student handwriting. Aryes developed a global readability scale in 1912 (Andersen, 1965) derived from the average time it took ten judges to read a subject’s handwriting, with faster average times judged as better handwriting.

The Wisconsin scale, developed by Erlbacher and Herrick (1961, 1963), represented an attempt to improve accuracy by providing 200 samples of handwriting graded according to letter size, tilt, and readability for each of the three school grades. This scale signalled a desire to move away from the subjective measures of global readability into evaluative tools that used objective measures. Measures like the Wisconsin Scale were an early attempt to provide a commercial product to teachers and psychologists that was more analytical. However, these products still retained a component of subjectivity. Individual teachers used their judgement when comparing samples to the scales, which proved too cumbersome and time-intensive for teachers to use as a classroom tool (Tseng & Murray, 1994).

Whilst evaluating available handwriting assessments, Harris (1960) found that the preferred measures of handwriting used by teachers and researchers had started to change from global-subjective scales to specific-objective scales and that most handwriting evaluation products were starting to use norm-referencing when evaluating the criteria of the parts of good handwriting. These components were letterform, size, slant, spacing, and

line straightness (Bruinsma & Nieusenhuis, 1991). Teachers would compare their student samples against examples indexed to a scale. The scale would allow the teacher to rate the tilt, letter formation, roundness, etc. These scores would then be added together to create a combined score. This score could be used to norm-reference the sample for age or grade level, and this type of evaluation became standard for most handwriting assessments in the late 1960s and early 1970s (Phelps et al., 1985).

An early attempt to reduce the subjectivity inherent in using scales was using transparent overlays (Collins et al., 1980; Helwing et al., 1976; Jones et al., 1971). Evaluators would score a student's handwriting based on a set criterion applied to handwriting that protruded from under a predetermined sample printed on a transparent overlay. A transparent overlay would help make specified performance standards salient to the evaluator.

Another innovation involved the use of questionnaires. Rubin and Henderson (1982) and Alston (1983) developed scales that relied on questionnaires completed by parents, educators, and other professionals. These scales would ask questions such as, "Are the letters that are supposed to be round indeed rounded?" (Alston, 1983). Ziviani and Elkins (1984) combine all the elements, including transparent overlays, questionnaires, scales, timed writing, and readability scales, to provide a measure for teachers. This scale would include the old norms for reading handwriting samples developed by Aryes (1912).

From the 1980s onward into the mid-1990s, until digital photography became widely available, the following scales were developed with varying emphasis on criteria or process, without any real innovation in objective measures: the Children Handwriting Evaluation Scale (Phelps et al., 1985), the Concise Evaluation Scale for Children's

Handwriting (HamstraBletz et al., 1987), the Diagnosis and Remediation of Handwriting Problems (Stott et al., 1985), and the Minnesota Handwriting Test (Reisman, 1993).

Technology advancements made electronic pad capture of handwriting possible in the late 1990s. Also crucial to objective measures was the widespread use and development of digital photography and digital image manipulation software. Taking advantage of these technological advances, Remi et al. (1997) noted the lack of objective measures for detecting handwriting difficulties and developed a set of objective measures. These included the spatiotemporal and kinematic characteristics for identifying handwriting disorders they called 'descriptors of automation of graphic activity' by having individuals write directly onto a digitiser tablet. This digitised image would then be objectively analysed using an experimental protocol that contained writing exercises, including copying figures and writing sentences, all under circumstances requiring increasing complexity and mastery of graphomotor skills. The digitiser was able to record the production of the handwriting in real time to identify an objectively temporal component of handwriting. Remi et al. (1997) were interested in documenting developmental changes in letter formation and other graphomotor skills without addressing this spectrum's drawing side.

Clark (2010) stated that drawing, a fine motor skill, is the first developmental step toward the more sophisticated fine motor skill of handwriting. Mastery of drawing, specifically lines, circles, and simple shapes, would lead to an individual developing their skill with handwriting (Clark, 2010). Letter formation, tilt, size, and writing speed are measurable along a developmental continuum that starts with drawing and ends with sophisticated writing. It would follow that an objective measure of an individual's ability to draw the constituent parts of handwriting, free from the subjectivity of prior scales, would

be superior to previous methods that may have been technically difficult prior to technological advances. By taking advantage of technological advances in digital photography and image manipulation software, one can potentially remove the subjectivity of analyses and replace it with empirical data derived from a computer's analysis of digital images. This is something that assessments of handwriting using scales have been attempting to do since the 1960s.

Motor control analysis with *ImageJ*

As stated above, whatever the orthographic system, handwriting or typing, the writing process requires several cognitive and motor functions. As the present results show, these functions have an inhibitory component that can be measured objectively and compared across different populations. In the present experiment, the author removed all linguistic components and focused on the simplest of drawings: a single short, straight line. The author realised the potential of the *ImageJ* software for this use and believes that the technique has yet to be employed previously to analyse handwriting and, by proxy, fine motor control and inhibition. Lines were analysed for deviation from the straight path by the software. Drawing a line has little, if any, linguistic need and does not rely on any graphemic memory buffer, leaving only the relatively bottom-up or "pure" motor functions left for evaluation.

School exercise books record the notes and work completed throughout the year. One of the routines students must follow is producing a book with an excellent presentation of their handwritten work. Marking policies are often created in schools to accomplish this. In the school of the present author, the students must comply with the Mathematics department marking policy, which is physically glued to the inside of the front

cover of their exercise books. All students must underline the title, date, learning objective for each lesson (at a minimum) and any other critical subject headings. This produces many examples of lines that one can assume were intended to be of a certain length, usually to match the text above it. Students are also asked to make these lines as neat as possible during their presentation assessment. They often opt to use a ruler, but in many cases, students do not comply with the policy and attempt a line by hand. Again, one can assume that this action intends to draw a straight line. The present author went through each student's exercise book until a line was found that was drawn by hand. This line was then digitally photographed and analysed using *ImageJ*—details of this process are found in the methods section.

ImageJ is an open-source image processing software developed by the National.

Institute of Health and the Laboratory for Optical Instrumentation at the University of Wisconsin. It was developed in the public domain and is free to use with an open architecture, allowing easy program expansion via Java plugins. The use of this tool lends itself to high levels of credibility due to its transparency and inherent ability for method sharing, including the use of macros (Swedlow, 2009). Method sharing within bio-image research communities has streamlined experimentation with further streamlining accomplished by integrating R, Python, and MATLAB programs. User-written plugins for *ImageJ* have been used to analyse an extensive range of objects and images, including cells (Reuden, 2005), mould growth (Hundhausen et al., 2013), radiological processing (Barboriak et al., 2005), and automated haematology (Gering & Atkinson, 2004). Using it to make measurements of drawings or handwriting is a novel method of analysing characteristics of handwritten objects of any type. (Indeed, when the present author first came across

ImageJ, he assumed using the software was commonplace in experimental psychology. This is not the case).

ImageJ capability was employed to calculate area and pixel value statistics of user-defined selections by manipulating intensity-thresholded objects in the students' hand-drawn lines and the fractal box count plug-in (see later). This use of the software solves the challenge of making measurements of highly complex, non-regular shapes. *ImageJ* (amongst many other functions) can take an image of a line and treat it like a non-straight piece of string. The straight-line distance from one end of the line written to the other can be assumed to be the distance the child is meant to traverse (although this assumption is not critical). This distance is compared with the length of the line when it is stretched out (as with string). This ratio indexes how much the line deviates from a straight path. In other words, it is a measure of fine motor control. This analysis was performed on lines generated by ADHD and non-ADHD children. It was hypothesised that children with poor inhibitory motor control (i.e., ADHD individuals) would have more variation in their line. In other words, the length of the drawn line, compared with the intended line, will be more significant for ADHD pupils.

Experiment 5

Method

Participants

There were 178 participants, all students at the same secondary state school previously described. Their age range was between 11-14. Eighteen of those students had been diagnosed with ADHD.

Stimulus and Apparatus

A Nikon d610 digital camera on a tripod was used to photograph the lines from the exercise books. The format used was the raw image format with a resolution of 24.3 million pixels. The camera was focused so that the drawn line's ends were as close to the left and right edges as possible in the frame of the camera's shot. ImageJ then processed this image. *ImageJ* was used to measure the line length and the line's perimeter in pixels. This numerical data was used to derive the fractal dimension of the line.

Procedure: Line Drawing analysis

Since we are using a ratio for analysis, line length and differences in camera focal point are controlled. Students can take home their used exercise books at the end of each school year. They often choose not to, and the books are disposed of. Many hundreds of exercise books are therefore collected to be recycled. The abandoned books are typically anonymised by tearing off the front cover containing the student's name. A digital photograph of the first line drawn in each book was taken. The selection criteria depended on the unambiguous signs of freehand drawing. Lines drawn with rulers tended to be exactly on the light green preprinted graphs paper lines and only deviated above the

horizontal, indicating a physical barrier below. Freehanded lines generally start randomly on the line but quickly wander off that line by going above and below the starting point, indicating a ruler not being present, as going below the starting point would mean going through the ruler. Only hand-drawn lines that wandered above and below the starting point were used for analysis.

There are two measurements taken of the lines that compare what was intended and what was produced. The lines were between 40 to 50 mm in length. Most lines were drawn with a standard medium black or blue ballpoint pen, leaving a line approximately 0.5 to 0.7 mm thick. Pencils were not used. The first measurement, i.e., deviation from straight, is possible due to the line being a 2-D rectangle under magnification. As noted, a rectangle (i.e., a magnified line) with some "wobble" can be mathematically straightened. The second measurement is the fractal dimension. Cartographers use This exact measurement to measure the length of borders or coastlines on maps. This was used to ensure the validity of measurements of curved shapes and provides some control over the resolution or detail that should be considered. This process enables the exclusion of straight-line deviations attributable to such factors as imperfections in the ballpoint pen or artefacts in the digital image. Combining these two types of measures strengthens the reliability of the objective measure of the wobble as not too fine a measure and not too gross a measure of drawing.

The image of the line was first magnified digitally. Due to the nature of digital photography, this invariably resulted in a rectangular-shaped polygon of pixels. Generally, this rectangle is the width of a standard ball in a ballpoint pen but does vary with the force or pressure used when drawing the line. The ImageJ software computed the perimeter of the line and the area of that rectangle by selecting the line as instructed by the software

using a mouse. If there are no complications, the line will be highlighted for the user to verify that the correct selection has been made. After selection, the desired line measurements are displayed for the evaluator to record. The specific measurements taken of the line are then used for data analysis (see below).

If there were any artefacts or connecting text that 'cluttered' the line, some editing of the image became necessary. This was accomplished by first cropping the image to include only the line as much as possible and converting the colour image to black and white. The image was then converted to binary, a process in which the program changes each pixel to black if the image is 50% or greater black or white if the pixel is less than 50% black. This leaves an irregular polygon made up of 1 by 1-pixel black squares. Any visual artefacts outside the main image were removed if needed, leaving only the line in the image.

ImageJ was then used to determine the perimeter and the area of the line. These measurements are made in pixels and are the program's standard analysis feature. This information was then used to find the length of the straightened version of the line. Specifically, *ImageJ* finds the *p* (perimeter), which we know equals two lengths plus two widths. The formula for the perimeter of a rectangle is P=2L+2W. *ImageJ* also returns the area A=LW. A mathematical property of quadratics is that only one combination of widths and lengths can produce these results in a rectangle, which is found through substitution. Let's determine the width for the first equation: $w = \frac{P}{2-L}$.

Then, we can substitute for w into the second equation. A=LW becomes $A = L \times \frac{P}{2-L}$.

Then rearrange this so that it is in quadratic form $L^2 - \frac{P}{2}(L) + A = 0$.

Using the quadratic formula $x = \frac{-b \pm \sqrt{b^2-4ac}}{2a}$ we can find the 2 solutions.

$Length = \frac{-\left(-\frac{perimeter}{2}\right)+\sqrt{(-\frac{perimeter}{2})^2-4(1)(\text{area})}}{2(area)}$, and

$Width = \frac{-\left(-\frac{perimeter}{2}\right)-\sqrt{(-\frac{perimeter}{2})^2-4(1)(\text{area})}}{2(area)}$.

Finally, these dimensions return the drawn straightened length of the rectangle, which is compared to a measure of the intended straight line. The measured feature in *ImageJ* was also used to determine the length of the intended straight line, measured from the bottom left corner to the top right corner of the line drawn by the student. One can find the proportion difference between the two by comparing the length of the intended line to the drawn line. This difference would be negligible for individuals with greater control over their fine motor skills. The reasoning was that the hand was more precise due to greater inhibitory control. In other words, less wobble in the line means motor inhibition guiding an individual's drawing skill to make as straight a line as possible for that individual.

Like a coastline, the measured length of the handwritten object depends on the method used to measure it; when objects on a map are changed due to the map maker's choice of scale, the cartographer needs to consider the degree of cartographic generalisation. In the past, the scale of generalisation had been limited to the practicality of physical measurements. In our case, we are only limited by the camera's resolution. The cartographic generalisation process includes all changes in a map that are made to make a smaller-scale map from a larger-scale map, in the present case, a zoomed-in picture from a life-size picture. The process that *ImageJ* uses to perform this is called the box-counting technique. This is accomplished by superimposing different box sizes over the image. The

software then records the number of boxes the image will pass through at each size. More detail is picked up as the grids become smaller. ImageJ uses the equation $N=cs^d$ where *N* represents the number of boxes, c is the proportionality constant, *s* is the scale factor, and *d* is the fractal dimension.

This equation form is rearranged into slope form so that the gradient is the fractal dimension.

$\log N = \log cs^{d,}$ which can be made split due to log rules

$\log N = \log c + \text{Log } s^d$

log N = Log c + d log s rearranges to slope form y=mx+c7854|aASW

log N = D log s + log c

y= log N , m= D, x = log s, c = log c

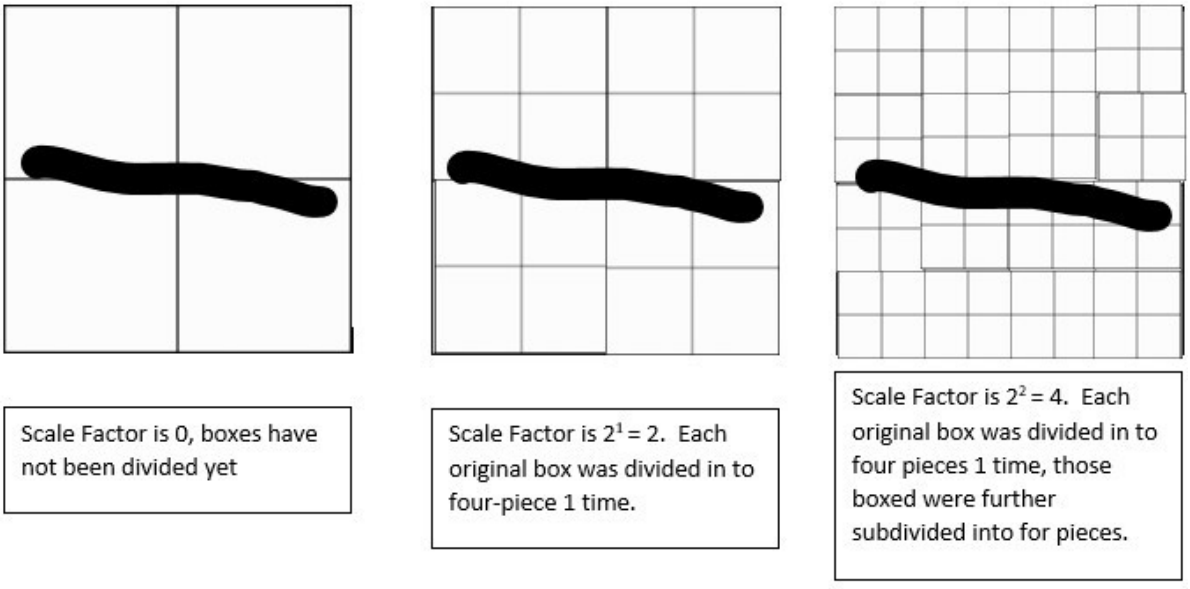

Figure 6 Line measurement scale factor process.

If we performed another division, it would be $2^3 = 8$. *ImageJ* automatically counts the number of boxes in which the line enters at different levels of scale. In our example, the following is what *ImageJ* is performing.

Scale Factor (S)	0	2	4	8
Log S	0	0.30103	0.60206	0.90309
Number of boxes (N)	4	7	15	30
Log N	0.60205999	0.84509804	1.176091	1.477121

Figure 7 Example of fractal data.

Thus, the slope of the line becomes the fractal dimension. This is a measurement of the "complexity" of the shape. A slope close to 1 is closer to a straight line with little complexity. A slope closer to two would be a very curvy line.

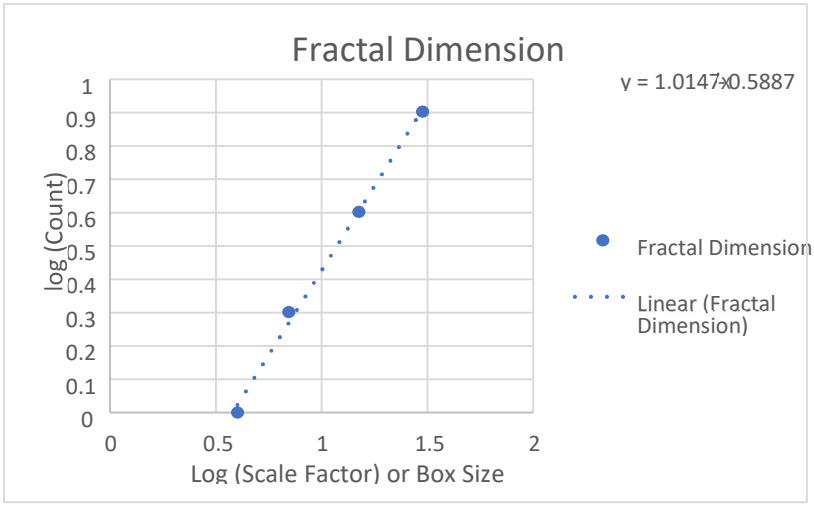

Figure 8 Example of fractal dimension taken from ImageJ

Using the process proposed by Mandelbrot (1967), it is irrelevant if the object is a landmass, mould growth, or a handwritten object; they can be treated the same mathematically. Thus, We can obtain a difference between the intended straight and deviated lines.

In conjunction with the ImageJ tool, this process allows for objective measurements of handwritten objects with features at various scale factors. We are only limited to the smallest feature desirable by *ImageJ* under magnification, which, as noted, is limited by the resolution of the camera and the size of a single pixel, as there is no apparent size of the smallest feature that should be taken into consideration when making measurements of handwritten objects, a process needed to be developed to find characteristics at the same scale factor such as the perimeter of the handwritten object, to ensure inter-relatable comparisons. Using a fractal dimension of 64, an objective approximation can be made about the minimum feature size to be considered. However, a scale factor of four could be

enough as this is a novel technique; the quadradic method correlated to the fractal method to ensure that both methods measured the same thing.

Results

An Independent Samples T-test was conducted for the fractal and quadratic analysis methods. The fractal method of the analysis showed that when asked to draw a straight line, the 16-member ADHD group (M = 1.42, SD = 0.08) when compared with 162 member control group (M = 1.32, SD = 0.1) demonstrated more deviation in their handwriting $t(176)=4.186$, $p = 0.001$

The quadratic method of analysis showed that when asked to draw a straight line, the ADHD group (M = 0.08, SD = 0.05), when compared with the control group (M = 0.04, SD = 0.02), demonstrated significantly less motor inhibition resulting in more deviation in their handwriting $t(176)=7.491$, $p = 0.001$

There was also a strong correlation between the two measures ($r(176) = 0.71$, $p =0.001$), suggesting that the two measures index a related phenomenon.

Discussion

In quadratic and fractal measures, individuals with ADHD have more deviation from their presumed intentions. This implies they have less control over their fine motor skills when drawing a straight line. This can be attributed to insufficient inhibitory strength to control the motor actions of the individual to complete the intended goal of creating a straight line.

The present results complement previous findings that ADHD children have less precise and less stable movements during a tracking task (Slaats-Willemse et al., 2005) and during a pursuit task (Rommelse et al., 2007). For example, Yan and Thomas (2002) observed that more corrections were needed by children with ADHD to keep their finger on a line when tracing and found more "jerky" movements. These findings are similar to the results of the "wobble" effect in the present lines. ADHD children had significantly more deviation than the control group. Tucha and Lange (2001) also found that children with ADHD have less legible handwriting and that individual letters tend to be larger (AdiJapha et al., 2007; Shen et al., 2012). The present author found a similar effect that could be expanded to shape formation ability.

The finding that lines drawn by ADHD individuals tend to be longer and have much higher levels of variation is also consistent with the findings of prior handwriting experiments involving children with ADHD. For example, Langmaid et al. (2011) asked children with ADHD to write a series of letters L's. The researchers used vertical movement, speed, and pen pressure measurements to scrutinise the handwriting. The authors used this "L" task when working with ADHD children as an adaptation of a previous experiment developed to investigate motor abnormalities in individuals diagnosed with Parkinson's disease and other cerebellar deficits. The task was explicitly developed to limit any cognitive influence on handwriting (Langmaid et al., 2011). Phillips et al. (1991) stated that the task reduced or eliminated any attentional demands, thus maximising the use of motor rather than higher-level cognitive processes often associated with handwriting. Having a similar concern and specifically wishing to index inhibitory control, the present author used a horizontal line to reduce cognitive load in forming simple shapes.

In Langmaid et al. (2014) "Ls" experiment, writing was recorded on a graphics tablet for analysis using MovAlyzeR software. MovAlyzeR is a commercially available movement analysis software that uses a pen tablet to measure and interpret movements with a pen, mouse or finger. Langmaid et al. (2014) found that children with ADHD had more variable handwriting size than a control group and short, fast, choppy movements. This concurs with the present author's findings that children with ADHD have more variation or wobble in their lines. The fractal dimension measurement found a similar effect. Langmaid et al. (2014) called letter formation, variability of letter height, and speed of letter production the "ballistic trajectory". This is partially similar to the fractal dimensional measure of the variation in the lines drawn. Similarly, the present ADHD students were found to have a higher dimension of variability when compared to the control group, similar to how ballistic trajectory was higher for ADHD individuals in Langmaid et al. (2014).

Chen and Cherng (2013) also used a digital pad to measure the handwriting of Chinese ADHD students. The ADHD group showed poorer handwriting performance compared to the control group. Specifically, the ADHD group scored significantly lower on scales that evaluate the construction, sequencing, accuracy, and directionality factors of handwriting. This study was a hybrid between technology use and scales, specifically the Tseng Handwriting Problem Checklist. The authors' results were limited to the construction dimension of the Chinese symbols, but in both those results and the present data, the ADHD students were less precise in their letter construction.

Line drawing and the lack of inhibitory control

ADHD, a disorder of inhibitory control, has been correlated with reduced motor function, reduced movement accuracy, and increased movement variability (Kalff et al.,

2005; Kalff et al., 2003; Meyer & Sagvolden, 2006). Guan and Wessel (2022) state that adaptive behaviour requires the ability to react to action errors. They suggest that humans implement a higher degree of caution when repeating an action during which they just committed a mistake. The authors use the term “post-error inhibition of response latencies” to describe this observation. Using transcranial magnetic stimulation whilst completing a Simon task, they showed that after error commission, the human cortico-motor system is momentarily inhibited immediately after an error and during the preparation of the following action. Especially relevant to the present author’s hypothesis, Guan and Wessel (2022) found that motor inhibition directly influences post-error slowing, affecting adaptive behaviour. Guan and Wessel (2022) do not directly address ADHD children in their work. However, it is reasonable to conclude that ADHD individuals lack the “caution” as described by Guan and Wessel (2022) when it comes to the adaptive behaviour needed to produce an intentional motor movement due to reduced inhibitory control (Barkley, 1997).

In conclusion, the experiment in the present chapter has shown that individuals with ADHD are less able to draw a straight than non- ADHD individuals. This is likely due to a lack of inhibitory motor control. This concurs with the results from Chapter 2.

Chapter 5

Attentional Inertia

Attentional Inertia is when humans have difficulty redirecting attention once it is focused on a feature or property of a stimulus or task; in effect, they become focused on the stimulus at hand and find it more difficult to shift attention to new relevant information. Consequently, the individual fails to inhibit old irrelevant impulses in favour of pertinent new stimuli. This is relevant to the current study because it allows one to measure another type of inhibitory control and look for differences between controls and AHDH individuals.

It was first described by Anderson, Alwitt, Lorch, and Levin (1979), who reported that the longer a child watched television, the greater the probability they would continue watching. The attentional inertia effect was mathematically predictive of future behaviour. Several researchers had previously noticed similar phenomena before formal Attentional Inertia research in the late 1970s. For example, James (1890) observed that the probability that a person will continue to talk about a topic seems to increase over time. People become noticeably locked into topical frameworks, meaning the longer they talk about a specific topic, the greater the probability they stay on it. Later, Hebb (1949) coined the idea of an "attitude", an aeronautical term for the orientation of an aeroplane in three-dimensional space, as a broad attentional resource that is focused on the specific task at hand, which then needs to be shifted to subsequent studies. Hochberg and Brooks (1978) linked Attentional Inertia to an unknown process or form of attention that keeps awareness "locked" onto a specific topic, task, or visual stimulus, even when the task demand requires switching.

The early evidence for Attentional Inertia was based on so-called “conditional survival probability” (CSP), also known as survivor function/survival time analysis in biology or a “reliability function” in engineering. The survival time analysis is a group of statistical methods in which the variable studied is the time until an event occurs (Klienbaum et al., 2012). Anderson et al. (1979) used this function to analyse the duration, or “survival”, of television viewing as a probability that the behaviour would continue over time as a function of time already spent watching television. Anderson et al. (1979) graphed the probability that children would watch different shows for three hours. The function showed how the longer children watched television, the higher the probability they would continue watching.

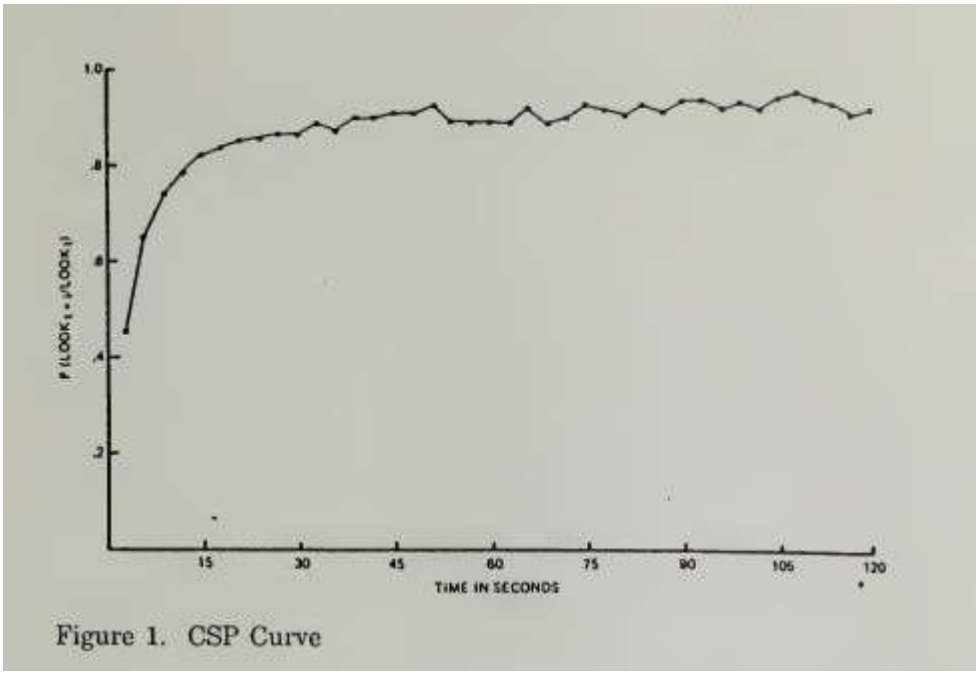

Figure 9. The figure presented by Anderson et al. (1979) shows that as time increases, the chance that a child continues to look at the television increases logarithmically to a peak of 85% at 15 sec, where it remains roughly constant.

Several authors assessed whether the type of content presented (using the TV viewing paradigm) had any effect on the degree of Attentional Inertia or if it was a general arousal phenomenon. For example, Alwitt, Anderson, Lorch, and Levin (1980) hypothesised that content changes should reset the curve. In other words, the Attentional Inertia would reduce to baseline when the content changed. The authors suggested two possible explanations concerning the nature of Attentional Inertia. If it was content-specific, then changes in content should have a strong terminating effect on the inertia. The participants in the experiment should notice the terminating effect regardless of how long they had been viewing the television show. In essence, if the content is changed, the experiment should be able to record the participant looking away from the television, indicating a reset in the Attentional Inertia effect regardless of time spent watching television overall. Alternatively, if Attentional Inertia concerns a more general, non-specific attentional arousal phenomenon, then any interruption will have the same terminating effect on Attentional Inertia. Data observed by Alwitt et al. supported the general arousal hypothesis.

Anderson, Choi, and Lorch (1987) examined the extent to which Attentional Inertia may reflect an increasing concentration of attentional resources. The authors argued that if Attentional Inertia increases attentional engagement, distractibility should decrease as inertia builds, simply because less capacity is available for processing external stimulation. The authors examined the effects of an external distractor as a function of television viewing length. They chose preschool children because “they have generally been characterised as relatively distractible”. Each child was asked to watch a one-hour episode of *Sesame Street*. A set of slides were projected onto a screen placed immediately adjacent to the television to act as the distractor. Anderson et al. recorded the location of the

children's gaze and used the same analysis as before (i.e., CSP curve). Results showed that after 15 seconds of viewing, the likelihood that the children would be distracted decreased and that turning a head action to orient to the distractor took longer. However, if the distractor happened before 15 seconds, the head turned more quickly, and the distraction was more likely to occur. Anderson et al. argued that these results support the hypothesis that Attentional Inertia reflects an increase in attentional engagement.

Richards and Gibson (1997) developed an Attentional Inertia model in which they argued that the CSP curve (see Figure 9) could be used to predict the outcome of the inertia. The authors would formalise a mathematical model of Attentional Inertia first proposed by Burns and Anderson (1993). This model would provide a solid empirical foundation to what was hypothesised by Burns and Anderson (1993), who observed Attentional Inertia as a function initiated at the start of, for instance, a television show when attention is unengaged. They further argued that the probability of being distracted or looking away as time passes would decrease, and attention resources become more engaged whilst also cumulatively increasing the probability of longer viewing of the television show as time passes. This will appear in the data as a positively skewed lognormal distribution, first observed by Anderson et al. (1979) and further elaborated by Burns and Anderson (1993).

Attentional Inertia as a Generalisable Effect

After the early television-watching paradigms, Attentional Inertia became seen as a more generalised attentional effect. For example, Choi and Anderson (1991) recorded children aged 5 playing with toys. The authors found that the longer a child played with a toy, the longer they were likely to continue to do so. The authors would also show that this

probability had the same characteristic logarithmic shape as the television-watching experiments conducted by Anderson et al. (1979), Burns and Anderson (1993), and Anderson et al. (1987).

Attentional Inertia has thus been used to explain children's difficulty switching between several tasks. For example, Kirkham et al. (2003) investigated children's ability to switch between the sub-components of the Zelanzo card sorting task. The Kirkham et al. (2003) variant consisted of four conditions designed to investigate the role of inhibitory control in performance by increasing or decreasing the inhibitory demands, with the standard condition serving as a baseline.

In the "standard" condition, the participants sorted blue and red star cards under a blue-star or red-truck model card. In the first iteration, they were given a rule that blue cards should be placed under blue cards and red cards under red cards. They were then asked to change the rule to match cards by shape, i.e., stars should be placed with stars and trucks with trucks. Kirkham, Cruess, and Diamond (2003) found that 3-year-olds sorted correctly until the rule changed. In this case, they continued to sort by the initial rule. Kirkham et al. (2003) attributed this result to children having difficulty inhibiting and continuing to use the prior rule. In another condition, called the "label condition", the children were instructed to label each card verbally before sorting. The test giver would ask the child, "What kind of card is this?" before allowing the child to sort each card. Kirkham et al. (2003) reasoned that this verbal labelling would aid the inhibition of Attentional Inertia that was focused on the initial sorting rule by promoting the re-focusing of attention on the current salient sorting condition, thus resetting Attentional Inertia. Kirkham et al. (2003) found that verbal labelling helped most participants successfully inhibit the initial sorting

rule and allowed children to resort to the cards successfully. The third condition, the "sleeve" condition, had participants look at the card and then put the card into a sleeve, thus hiding the salient sorting feature of the card. Children found it more difficult to inhibit the initial sorting rule without a salient visual reminder of the card's features and reverted to it. Kirkham et al. (2003) stated that this condition promoted the Attentional Inertia effect. The final "face-up condition" was similar to the label condition, where the experimenter promoted inhibition visually. When cards were drawn, they were left face up so that children could refer back to them for as long as required before sorting the cards after the rule change. Kirkham et al. (2003) stated that because sorted cards were left face-up, this promoted the inhibition of the initial sorting rules by causing the participant to retain focus on the cards' sorting feature. Kirkham et al. had thus manipulated the degree to which I

Attentional Inertia affected children during the tasks by aiding or confounding inhibition.

The role of inhibition in Attentional Inertia

Kirkham and Diamond (2003) demonstrated inhibition's critical role as a regulatory process on Attentional Inertia. A lack of inhibitory control explains why the children made card-sorting errors in the abovementioned experiments. Kirkham et al. (2003) stated that "when children are presented with a conflicting stimulus (the cards dual salient features/dimensions), children are pulled to focus on the previously relevant dimension and its rules ('Attentional Inertia'; overcoming that tendency requires inhibition." Evidence for the relationship between inhibition and Attentional Inertia comes from manipulating inhibition of the four conditions in the Zelanzo card sorting task (Kirkham et al., 2003). The manipulated inhibition load demonstrated that when inhibition is promoted, the

Attentional Inertia is reduced, and when inhibition is disinhibited, Attentional Inertia is more powerful (Kirkham et al., 2003).

Kirkham and Diamond (2003) argued that inhibition makes possible all prerequisite behaviours previously described as refocusing, switching, and disengaging in other Attentional Inertia experiments. They state unequivocally that children cannot refocus, switch or disengage until that child first inhibits conflicting stimuli in favour of the new stimulus. When a new behaviour is observed, it occurs only after a prior one is inhibited. Failure to inhibit results in Attentional Inertia, which requires more inhibitory effort over time to overcome (Anderson et al., 1979; Anderson & Lorch, 1983; Richards & Cronise, 2000; Towse et al., 2000).

Towse, Redbond, Houston-Price, and Cook (2000) found that the experimenter could promote inhibition by asking the child to verbally state the salient feature of a card after a failed attempt. A greater success rate of sorting would result in a continued success rate during future sorting. Towse et al. (2000) attributed the increased success rate to the manipulation of inhibition by drawing attention to the salient feature of the failed attempt. The promotion of inhibition would carry over into the next instance of a card being drawn, contributing to successfully applying the new sorting rule.

Any manipulation that promotes inhibition, what is inhibition here a mechanism, a behavioural outcome, be it verbal, visual, or even as feedback on a failed attempt, when administering the Zelanzo card sorting task to children increases the success rate of the second sorting rule. The various researchers seem to provide a “scaffold” that helps children switch between conflicting stimuli (Towse et al., 2000; Kirkham et al., 2003).

Kirkham et al. (2003) state that this scaffold gives children time, clarity, or incentive to inhibit the mental set that is no longer correct and thus refocus their attention.

Attentional Inertia as an automatic bottom-up process

Anderson and Lorch (1983) argued that the phenomenon is not under voluntary, strategic control. Instead, the authors suggested that attentional 'drift' is essentially controlled by bottom-up or automatic mechanisms. In support of this, Richards and Cronise (2000) found that Attentional Inertia effects were also observed for infants between 6 and 24 months.

Introzzi et al. (2015) aimed to tease out the contribution of categorically different inhibitory processes: perceptual, cognitive and behavioural inhibition and find a relationship between these inhibitory processes and the switching cost effect associated with alternating cognitive tasks. Introzzi et al. (2015) used a correlational design. Several experimental paradigms (e.g., Stop signal, visual search, Stenberg's experimental and Simon's paradigm) were adapted and included in a computerised program called TAC (Introzzi & Canet Juric, 2014) to assess the different cognitive processes and associated switching costs. Introzzi et al. (2015) found that perceptual and behavioural inhibition showed moderate and low correlations with attentional cost. Cognitive inhibition showed no relation with flexibility, and only perceptual inhibition predicts switching cost effects, suggesting that different inhibitory processes contribute differently to switching costs. This could be interpreted as evidence of the Attentional Inertia Theory's central argument that postulates that inhibition plays an essential role in the ability to switch between tasks and/or representations flexibly.

Attentional inertia as a solely top-down-driven effect predicts that ADHD students who previously were able to exert response inhibitory control in a Stroop task (as in Chapter

2; see also Schwarts & Verhaeghen, 2008) should be able to exert top-down response inhibitory control in the Attentional Inertia task. The Stroop results confirm the broader findings that ADHD children do not have difficulty with cognitive inhibition/response inhibition. In a meta-analysis assessing degrees of response inhibition in ADHD individuals, Schwarts and Verhaeghen (2008) found that Stroop interference is no more extensive in ADHD individuals than in age-matched controls. If top-down mechanisms mediate both Stroop and Attentional Inertia, there should be similar results between ADHD and control groups. However, suppose Attentional Inertia has a different effect on the ADHD group when compared to controls. In that case, the present author argues that this is not a top-down effect but a bottom-up phenomenon possibly explained by a divergence between attention and expectation. (Summerfield & Enger, 2009). It was, therefore, predicted that attentional Inertia will cause students with ADHD to miss changes to a stimulus rule due to the separation of attention and expectation.

In the present experiment, children aged 11-14 were shown a series of number sentences they were instructed to complete; a number sentence was traditionally referred to as “sums” (e.g., 12 x 4, 6 - 2). The first eight sentences were all multiplications. The ninth critical trial was an addition number sentence. Therefore, the child needed to switch from a frequently presented stimulus (i.e., a multiplication sign) to a new one (i.e., an addition). The ability of ADHD children to correctly switch was compared with controls. In effect, the child was required to inhibit the multiplication on the critical trial.

Task Switching Performance of ADHD Individuals

Few studies of ADHD samples have investigated two central cognitive control processes: interference control and task-set coordination. King et al. (2007) noted that neuropsychological models of ADHD implicate impaired cognitive control as contributing to disorder-characteristic behavioural deficiencies and excesses and further stated that the traditional view of ADHD postulates a core deficiency in cognitive control processes. King et al. (2007) used chronometric Stroop and task-switching paradigms to investigate the efficiency of interference control and task-set coordination processes in ADHD participants. An ADHD (n=22) and a control group (n=22) performed a manual trial-by-trial Stroop colour-word test and a blocked explicitly cued task-switching paradigm.

Attentional inertial and task switching seem connected to a temporal component for ADHD participants. In both cases, time before critical trials predicts success when the researcher manipulates task preparation processes. Further investigation would need to be conducted to find conclusive evidence of the distinction between difficulties in performing tasks that require an ADHD individual to change tasks midstream. King et al. (2007) measured transient task-set updating, sustained task-set maintenance, preparatory mechanisms and interference control during the task-switching paradigm. Control analyses tested for the specificity of group × condition interactions. Abnormal processing of task-irrelevant stimulus features was evident in the ADHD group's performance when task switching. ADHD group interference effects on the task-switching paradigm were found to be dependent on the time allotted to prepare for an upcoming task. Group differences in sustained task-set maintenance and transient task-set updating were also found to be dependent on experimental manipulation of task preparation processes. Except for Stroop

task error rates, all analyses revealed generally slower and less accurate ADHD group response patterns.

There is not enough evidence to conclusively rule out difficulties in task switching in ADHD populations as a cognitive explanation for the results of experiment 6. Task-switching difficulties derive from top-down cognitive processes and have a measurable effect, whilst attentional inertia is considered a bottom-up process. In either event, the outcome is the same as the performance of ADHD participants in this experiment is significantly different than their non-disabled peers. Further investigation would help find a distinction between the two possible explanations.

Perseveration: An Alternative Explanation

Gillen et al. (2016) defined perseveration as the inability to shift from one concept to another or to change or cease a behaviour pattern once it has started. Furthermore, it's an idea that describes a person “stuck in a set” (Gillen et al., 2016) and leaves the person unable to discard previous behaviours and activate new ones in a new situation. The person stuck in a set attempts to solve another problem with information relevant to a previous problem. Bringing perseveration to a conscious level and training the patient to inhibit the perseverative behaviour has been successful (Gillen et al., 2016). Testing for perseveration is done by asking a person to copy alternating sequences by hand. If the pattern cannot be reproduced due to a participant's inability to switch components of the pattern, it is thought that that person suffers from perseveration. Perseveration is thought to be associated with lesions to the frontal lobes, which is thought to degrade executive functions (Mateer and Sira, 2003)

Although the description of perseveration is apt for the experiment results, it is unlikely for two reasons. Instances of perseveration are linked to lesions in the frontal lobe. Individuals with ADHD are not known to have lesions or damage to their frontal lobes, and there is no association between frontal lobe damage and ADHD. Also, in experiment 4, there were many instances of practice. ADHD students did not improve with practice even when the sign change was known. It was the point of the exercise to practice looking for changes over a half term and improve performance. The symptoms of perseveration could be mitigated or removed with practice, but ADHD students showed no sign of improving with practice.

Experiment 6

Method

Participants

There were 178 participants aged between 11-14, and all were at the same secondary state school previously described. Eighteen had been diagnosed with ADHD. Students had a known success rate for addition and multiplication before the experiment, as measured by a collection of addition and multiplication assessments used to assess achievement. The control groups had a known multiplication success rate of 99% and an addition success rate of 100%. The ADHD group had a success rate for multiplication of 99% and an addition success rate of 99%.

Stimulus and Apparatus

Students were presented with ten random number sentences projected onto a 200 (w) x 200cm (h) screen. The first eight number sentences were multiplication, the 9th was an addition, and the 10th was multiplication. Answers were written in an A4 exercise book daily as a starter before the Maths lessons over seven weeks. Students were given one minute to complete the task before each lesson to practice looking for signs that change in math questions as a form of GCSE test preparation. Teachers would then mark the answers and record the results of each question on an Excel spreadsheet.

Procedure

Over a half term, students were tasked with answering the ten-number sentence questions on fifteen occasions. The ninth question was changed to an addition number sentence, following eight prior multiplication questions, with the 10th and final questions returning to multiplication. The average rate of correctly answering the 9th question was calculated using the results of the 15 answers to the critical 9th question. The answers were taken from marked exercise books. Rates for correctly answering questions in the non-critical trials were collected. This process was completed for all participants. Results for all responses were recorded on an Excel spreadsheet. A “1” was used for correct responses, and a “0” was used for incorrect responses when recording the answers to questions 1 through 10. This process was repeated for each participant. The rates of correct responses for the 9th question were compared between a control group and an ADHD group.

Results

An independent-sample t-test was conducted to determine whether there is a difference in the rate of ADHD students answering the 9th number sentence correctly compared to the control group, where the rule had changed and required inhibition of the previous rule and a reset of Attentional Inertia. There was a significant difference between the ADHD group (M = 76.9%, SD = 18.9%) and the control group (M = 98.0%, SD = 4.68%), t(176) = 11.2, p < 0 .001]. This means that ADHD students inhibited the multiplication rule on the 9th question in favour of the new stimulus significantly worse than the control group. ADHD students answered the noncritical multiplication questions correctly 98% of the time. They also answered the final questions correctly if they were still multiplication questions at the same 98% rate. This implies that they continued to apply the multiplication rule to the questions with no reduction in accuracy. This further supports that they could not inhibit the prior rule in favour of the new stimulus in the critical questions to change the rule to addition.

Discussion

The number sentence task has a measurable effect consistent with what Attentional Inertia predicts would happen. Additional evidence to show a solid attentional inertia effect could be done in future to correlate different numbers on non-critical trials to strengthen or reduce the probability of the attentional inertia, as shown by the CPS curve. The ADHD children in this experiment have shown that they did not have the inhibitory resources to stop the multiplication process in favour of the addition process. An alternative explanation could be difficulties with task switching; please see above.

The finding suggests that the strength of non-critical trials biased the answer to such an extent that the participants with ADHD continued to successfully apply the old rule even when the new stimulus was present in the form of the addition sign. This indicates that attention was focused on the numbers, not the visual feature containing the salient information, the mathematic operation sign. Results of the experiment further indicate that ADHD participants assumed that they knew what to do with the numbers due to consistency in the non-critical trials and continued with assumption throughout the critical trials. ADHD children were more likely than their peers to continue with the assumptions. These findings provide evidence that inhibitory control in ADHD children is not strong enough, when compared to their peers, to switch the rule from multiplication to addition in the critical trials.

Similar to the findings of the card sorting task and the letter/digit task, the number sentence task created a situation where a prior rule (i.e., multiplying) would need to be inhibited in place of a new second rule (i.e., addition). The number sentence task had similar results as the television watching and playing tasks described above. The participants in these experiments included a control group able to successfully inhibit the prior rule set, meaning they overcame Attentional Inertia, whilst the ADHD group could not. The ADHD group provided evidence of applying the initial mathematical operation when they continued to answer the changed rule of the 9th question as if they were multiplying, which means they were not “paying attention” as the educator intended and had indeed failed to “learn” a (i.e., a top-down process) how to inhibit the salient feature essential to the first eight number sentences in favour of the new salient feature in the ninth question.

As with card sorting paradigms, an initial rule was given, in the present case, multiplication. By the 8th question, students would have passed what Anderson et al. (1987) called the 15-second threshold, the time at which maximum Attentional Inertia occurs. In the present experiment, this would have been around the time of the critical 9th question. These data matched the curve Anderson et al. (1979) and Anderson et al. (1987) described: students typically take two seconds to answer each question.

Also, similar to the card sorting task, the students demonstrated that they could complete both tasks in isolation. All children had demonstrated prior knowledge and mastery of the arithmetic skills required for the task. Students who participated were known to be able to find the answer to their times tables up to 12 and were able to add single-digit numbers in isolation. In line with the Attentional Inertia model, when the children began to write their answers, they focused on the salient feature of the number sentence, the changing numbers and, after a few instances of multiplication, the Attentional Inertia of multiplication set in. However, as that sign remained the same, it became less salient than the changing numbers, meaning that the Attentional Inertial continued to build for the operational sign. When the mathematical operation was changed from multiplication to addition, Attentional Inertia needed to be overcome using inhibitory control. Typical children, as in the "label condition" of the card sorting task, could exert inhibitory control and change from multiplication to addition, whilst their ADHD peers were not.

Diamond and Kirkham (2001) and Kirkland et al. (2003) continued using the Zelano card sorting task to show that manipulation of inhibition load in different sorting conditions better explained Attentional Inertia. These results would challenge the Cognitive

Complexity and Control Theory (the CCC Theory), which proposed a two-level hierarchical system. The top level of the hierarchy is a top-down representational mechanism, which is under conscious control. This mechanism controls the lower-level response-based system. This lower-level system is bottom-up, unconscious, and automatic (Zelano & Frye, 1997). However, Kirkham et al. (2003) would show that inhibition pre-empts any top-down mechanism.

In Chapter 7, the present author will discuss some general teaching and intervention issues given the findings of this research. However, It is worth noting that although well-meaning, the Maths faculty within the school where the experiments were conducted states that students need to practice “paying attention” to improve GCSE performance. However, ADHD students did not improve their inhibition rate after 15 weeks of practice. They often knew and were conscious of the strategy in which the task, similar to the “label” condition in the card sorting task, always included a switched rule. Refocusing strategies can help overcome Attentional Inertia, but, crucially, less so, if at all, with ADHD students. ADHD students did not have the inhibitory resources to overcome Attentional Inertia and notice changes to the operation signs in simple number sentences. This lack of inhibitory control is more often than not anecdotally attributed by teaching staff as a simple case of carelessness, and, in some cases, this is used as evidence for the need to teach “paying attention”. However, such efforts would not likely produce the desired results.

Chapter 6

Inhibition in the typically developed population.

It is essential to know whether the effects found in the experiments generalise to the broader ADHD population (or ADHD-like population). The central aim of the final empirical chapter is to explore and examine the generalisability of this research using one of the paradigms previously employed. Specifically, the degree to which a person shows motor-induced motor inhibition (as researched in Chapter 2) is measured as a function of the degree to which they exhibit ADHD-like behaviour.

Spectrum Disorders and ADHD

Maser and Akiskal (2002) noted that the term "spectrum" is an analogy that is useful for the diagnostician to use when grouping conditions "that are qualitatively distinct in appearance but believed to be related from an underlying pathogenic point of view". The authors also noted the many spectrum disorders recognised in clinical practice, including anxiety, stress, obsessions and compulsions, general developmental disorders, psychosis, Schizoaffective disorders, Schizophrenia-like personality disorders, mood, and substance use, amongst many others. Murphy, Rosenthal, and Kety (1968) used the term "spectrum" in psychiatry to bring together the various types of schizoid personalities under the unified umbrella of schizophrenia.

There is emerging evidence that ADHD can also be considered a spectrum disorder (Shaw et al., 2014; Posner et al., 2011; Sonuga-Barke et al., 2022). Because there are a range of linked symptoms that are different in appearance and severity but are thought to

be caused by a single underlying mechanism, Sonuga-Barke et al., 2022 also argued that ADHD can be conceived of as a matter of degree. Heidbreder (2015) noted, “The symptoms associated with ADHD can be viewed as dimensional markers that point to a spectrum of related disorders”.

Efstratopoulou, Jansses, and Simmons (2012) pointed out that ASD and ADHD are examples of two syndromes with high levels of overlap in their diagnostic criteria. The comorbidity of ADHD in children with ASD is high, at 50 to 70 per cent (Gadow et al., 2004; Gadow et al., 2005). ASD and ADHD children also share higher rates and greater severity of aggression, anxiety, and depression (Wood & Gadow, 2006). Children with ASD display ADHD symptoms that correlate with ADHD subtypes, suggesting a continuum within ADHD shared with oppositional defiant disorder, conduct disorders, learning disabilities, and other psychiatric conditions, such as anxiety disorders and depression (Klassen et al., 2004).

The ASRS and BIS

The Adult ADHD self-report scale (ASRS) has 18 criteria and is commonly employed with adults as a screening tool as part of a clinical interview. The scale has high internal consistency (Cronbach’s alpha = 0.88) and concurrent validity (r = 0.84; Adler et al., 2006). Empirical work by Stanton, Forbes, and Zimmerman (2018) suggested that the ASRS has three principal factors: the Inattentive subscale, the Motor Hyperactive/Impulsive subscale, and the Verbal Hyperactive/Impulsive subscale. These factors are used to compute subscale scores. It should be noted that these scores are not usually taken to reflect the extent to which a person has ADHD but predict that a person who self-reports will go on to receive a diagnosis of ADHD. For example, if the respondent scores four or more in the first section of

the scale (i.e., on Part A), then the symptom profile of the individual is considered to be highly consistent with an ADHD diagnosis in adults (Adler et al., 2006; Kessler et al., 2007). The ASRS does not provide a quantitative measure of ADHD but is helpful when validating a task to ensure that it is measuring a specific aspect of the disorder. This measure is closely linked to the diagnostic criteria of the DSM-V, although the DSM-V does not make a distinction between verbal and motor hyperactive subtypes. However, the ASRS has the advantage of being able to make the distinction between these two subtypes.

The Barratt Impulsiveness Scale (BIS-11) is a 30-item self-reporting tool designed to assess an adult's impulsiveness. Fossati, Di Ceglia, Acquarini, and Barratt (2001) reported that the questionnaire has adequate construct validity compared to a similar measure, i.e., the Wender Utah Rating Scale (WIRS). Stanford et al. (2009) also state that the scale has excellent test/retest reliability (r=0.83) and internal consistency (Cronbach's alpha = 0.82). The BIS-11's scales were validated by Patton et al. (1995). It is norm-referenced into three categories: attention impulsivity, motor impulsivity, and non-planning impulsivity (Malloy-Diniz et al., 2015). The first of these was defined as a lack of focus on an ongoing task and was further divided into two first-order factors: attention and cognitive instability. The second factor is motor impulsiveness, defined as an action without inhibiting prepotent or ongoing responses and includes two first-order factors, motor and perseverance. This motor impulsiveness factor will be particularly important to the present chapter as the paradigm used (i.e., motor-induced motor inhibition) can be seen as a measure of this, particularly a lack of motor inhibition. The third factor is non-planning impulsiveness, defined as an orientation towards the present rather than the future (Patton et al., 1995).

Both the ASRS and BIS scales were employed in the present chapter.

Experiment 7

Recall from Chapter 2 that ADHD children, unlike controls, did not show motor-induced motor inhibition. One can, therefore, assume that the more ADHD-like a person is, the less inhibition they will show in this paradigm. In Experiment 7, seventy adults undertook the basic paradigm used in Chapter 2 and then completed the ASRS-v1.1. It was predicted that there would be a positive correlation between the ASRS score and the proportion Repeat value. As discussed in Chapter 2 (and Cole & Skarratt, under review), a relatively large motor inhibition proportion value (i.e., > 50%) is taken as evidence for 'poor' motor inhibition. In other words, the individual tends to repeat a response just performed rather than inhibit that action.

Method

Participants

Seventy participants, all over 18, self-identified as residing in the US or UK. Thirty-five were identified as male and 35 as female. They were paid £1.20, or the US dollar equivalent.

Stimuli, design and procedure.

All aspects of the motor inhibition task were described in Experiment 1 (shown again in Figure 10. Participants discriminated between the letter "A" or "B" before judging which of the two lines was longer. After the motor task, they completed the ASRS v1.1. The two tasks were completed remotely via the *Prolific* platform, and the experiment was programmed using PsychToolKit. A desktop or laptop computer was needed to run the experiment.

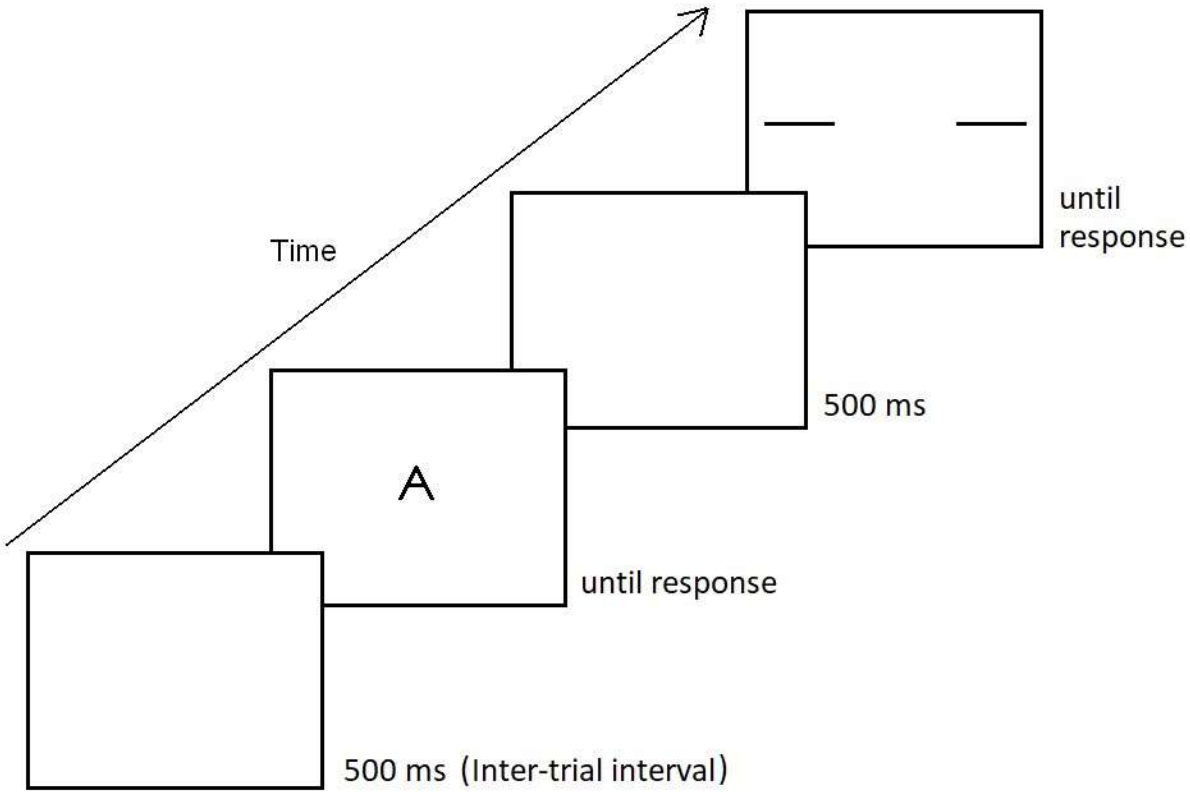

Figure 10. Trial sequence in Experiment 7.

Results and Discussion

Four participants did not reach the 80% accuracy threshold on the letter discrimination task. The final sample size was, therefore, 66. The mean letter discrimination performance for the remaining 66 was 95.1% (SD = 4.2), and the mean RT was 512 ms (SD = 82).

The mean repeat response rate was 48.2% (SD = 7.0). This value significantly differed from the chance value of 50%, $t(65) = 2.2$ $p = 0.042$. In other words, the sample showed an overall motor-induced motor inhibition effect. This effect was also apparent in the RTs; Repeat responses (M = 737, SD = 105) were significantly slower than Switch responses (M = 724, SD = 96), $t(65) = 3.62$ $p = 0.02$). Concerning the ASRS-v1.1, the mean

score was 47.4 (SD = 9.4). The central analysis concerned the nature of the correlation between motor inhibition and ASRS values. A small to medium effect was observed that did not reach conventional statistical significance, r = 0.18, p = 0.073 (see Figure 11)

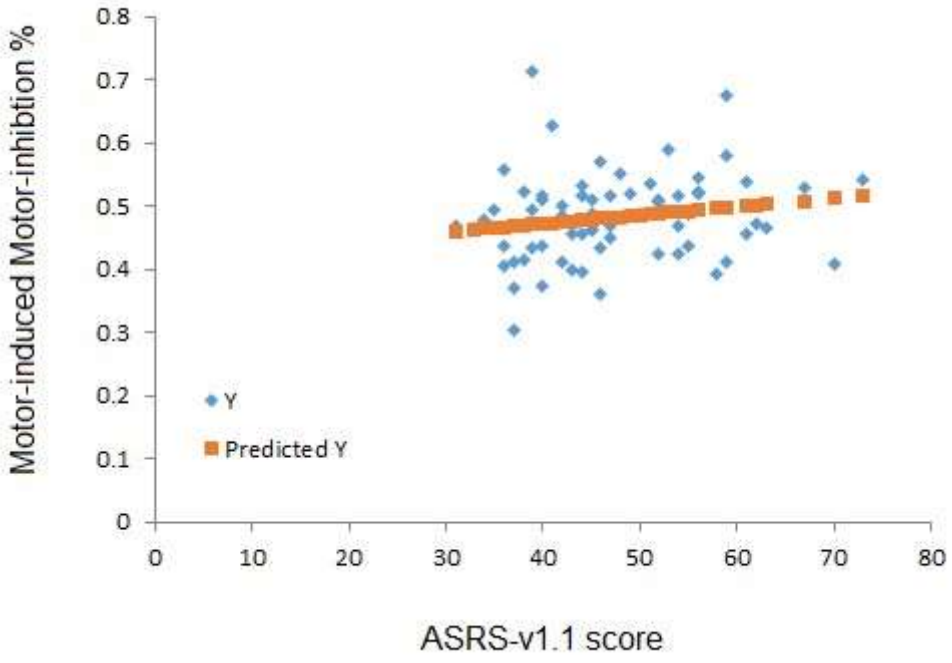

Figure 11. Results from Experiment 7. The Line Fit Plot shows the positive relationship between motor inhibition and ASRS. Note that a more excellent value on the Y axis reveals the lack of inhibition.

The present results have revealed a small to medium relationship between the degree to which a person can be said to have ADHD and the degree to which they lack motor inhibition. As they become more ADHD-like, they tend to repeat a simple action (i.e., button press) that they have just performed. Put another way, less ADHD-like people are likelier to inhibit a simple motor response. This concurs with the findings from the present Experiment 2 (Chapter 2); ADHD children have less motor inhibition than controls. Indeed, motor inhibition was utterly absent in that group. Although the effect in the present experiment is

not large, one can be reasonably confident that there is a relationship between ADHD and motor inhibition in the general population. The phenomenon described in Experiment 2 thus appears to generalise beyond the sample employed in that experiment. The central aim of Experiment 8 was to examine this generalisability issue further.

Experiment 8

One of the central characteristics of ADHD is impulsivity. As stated before, impulsivity is a multidimensional construct in which an individual acts quickly without adequate thought or conscious judgment to achieve some goal and without consideration of future consequences (Moeller et al., 2001). As Vanden-Bos (2007) stated, impulsivity is a "tendency to act with little forethought, reflection, or consideration for the consequences". Impulsivity has been argued to comprise two independent components: acting without thought (Daruna & Barnes,1993) and choosing short-term gains over long-term gains (Rachlin, 2000).

Differences in the level of impulsivity observed between individuals have been recognised as different aspects of inhibitory control comorbid with multiple psychological disorders, including ADHD (Vasconcelos et al., 2012). Using factor analyses of commonly used impulsivity measures, Barkley (1997) and Nigg (2000) found that the single construct inhibitory control was correlated with ADHD ratings (see also Avila et al., 2004). There have been further studies comparing different measures of impulsivity using laboratory tasks and correlating them to rating scales (Carrillo de la Pena, Otero, &
Romero, 1993; Gerbing, Ahadi, & Patton, 1987; Luengo, Carrillo de la Pena, & Otero, 1991).

As well as in ADHD individuals, impulsivity has also been the subject of much research in the typically developed population. Some of this work also shows that it is associated with an inability to inhibit a response (Logan et al., 1997). There is additional evidence that commonly used behavioural measures of inhibitory control correlate with standard self-report measures of impulsivity in normal adults. For example, Enticott, Ogloff, and Bradshaw (2006) found an association between scores on the BIS and responses on several commonly used behavioural paradigms of inhibitory control (e.g., Stop Signal, Stroop, and Negative Priming). Thus, there is clear evidence to suggest that impulsivity is associated with some specific measures that indicate a lack of inhibitory control.

The rationale for Experiment 8 was identical to that of Experiment 7, except that responses on the motor-induced motor inhibition task were correlated with the BIS.

Method

Participants

There were 76 participants. As with Experiment 7, they were all over 18 and self-identified as residing in the US or UK. Thirty-eight were identified as male and 38 female. They were paid £1.20, or the US equivalent.

Stimuli, design and procedure.

All aspects of the motor inhibition task were as described previously. After this task was finished, participants completed the BIS. The two tasks were again completed remotely via the *Prolific* platform and programmed with PsychToolKit. A desktop or laptop computer was also needed to run the experiment. When the work for the present thesis began in

October 2019, pre-registering studies were in their infancy, and none of the previous experiments were pre-registered. The registration for the present experiment can be found at https://osf.io/f6b9v.

Results and Discussion

Although 76 participants undertook the study, the pre-registration stipulated that the sample size analysed would be 64. The extra participants were run to generate enough replacements for those whose error rate (on the letter discrimination task) was above the 20% threshold for inclusion. However, none of these were needed because the first 64 who completed the task had an error rate of less than 20%. Mean letter discrimination performance was 95.1% (SD = 4.2; note that these values are identical to those of Experiment 7), and the mean RT was 532 ms (SD = 95).

The mean Repeat response rate was 47.2% (SD = 7.2). This value was significantly different to the chance value of 50%, $t(63) = 2.3$ $p = 0.042$. As with Experiment 7, the sample thus showed an overall motor-induced motor inhibition effect. This effect was also revealed in the RTs. Repeat responses (M = 783, SD = 96) were significantly slower than Switch responses (M = 763, SD = 107), $t(63) = 3.6$ $p = 0.02$). The mean BIS score was 68.0 (SD = 9.9). The central analysis again concerned the correlation between the two variables of interest,

i.e., motor inhibition and BIS. However, There was no meaningful association between the two, $r = 0.05$, $p = 0.68$ (see Figure 12).

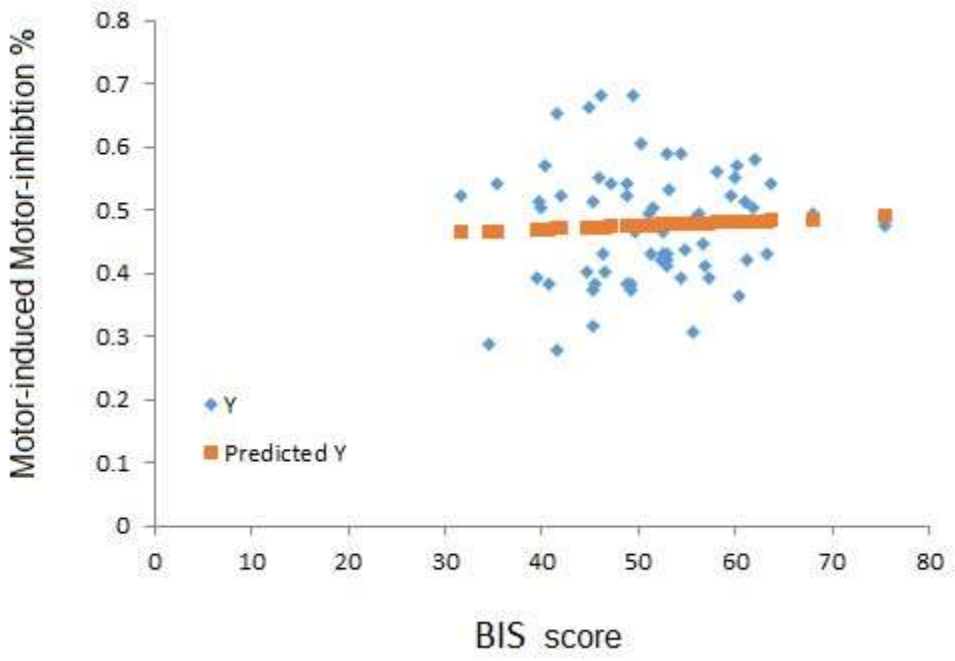

Figure 12 Results from Experiment 8. The Line Fit Plot shows the null relationship between motor inhibition and the BIS.

Unplanned analysis

As described above, Patton et al. (1995) showed that the BIS has three central components. The authors referred to these as Attentional Impulsiveness, Motor Impulsiveness, and Non-planning Impulsiveness. The second of these is of central concern to the present thesis. However, the motor component did not meaningfully correlate with motor inhibition, r = -0.03.

Overall, the present experiment has not found any association between impulsivity, as measured by the BIS and motor inhibition.

Chapter Discussion

Experiments 7 and 8 successfully replicated the motor-induced motor inhibition effect previously reported. Furthermore, and most importantly, motor inhibition was associated with scores on the ASRS (Experiment 7). However, this correlation did not occur for the BIS (Experiment 8). A small to medium effect was observed with the former measure. Although tentative, given the results of Experiment 8, this does suggest that motor inhibition is related to ADHD in the wider population.

Convergence of validity between specific lab-based tasks and self-reporting instruments has been problematic in ADHD work. Barnhart and Buelow (2017) observed similar results to Experiment 8 when working with undergraduate student participants who completed several self-report measures of impulsivity (i.e., the BIS, the Impulsive Sensation Seeking subscale, Conner's Adult ADHD Rating Scale, and Frontal Systems Behavior Rating. Scale) and three behavioural measures of impulsivity (Balloon Analogue Risk Task, Delay Discounting Task, Stroop). Their analysis failed to detect any relationships between self-report and behavioural measures, consistent with previous research (Bayard, Raffard, & Gely-Nargeot, (2011). Meda et al., 2009; Simonoff et al., 2013; Rubia, Smith, & Taylor, 2007). Barnhart and Buelow (2017) concluded that the failure to observe convergent validity resulted from how the self-reporting tools define impulsivity. As noted, impulsivity is a multidimensional construct comprising three components: attentional, reward sensitivity, and behavioural and motor impulsivity. Further analysis of how each self-reporting tool builds the various constructs, like impulsivity and the weighting of the component factors, could shed light on the lack of convergence.

The paradigm employed in Experiments 7 and 8 indexes motor inhibition, whilst the self-reporting scales take a broader view of impulsivity as a factor of ADHD. It remains to be seen to what quantitative extent motor inhibition, as measured by the paradigm, can affect scores on either self-reporting instrument. It remains possible that the "weight" of motor inhibition is higher for the ASRS score compared to the BIS.

The primary effect shown in Experiment 7 concurs with results from Albajara et al. (2020) who used a Stop-Signal-Delay task to distinguish between Autism and ADHD. Furthermore, Albajara et al. (2020) found convergent validity with the ADHD Rating Scale-IV developed by DePaul et al. (1998). In terms of Experiment 8, the BIS-11 is a broad measure with three factors, all of which rely on higher-level processing, indicating a poor possibility of overlap with the task. If we examine those previously mentioned factors - attentional impulsiveness, motor impulsiveness, and non-planning impulsiveness -- one should also look at the corresponding subfactors to get a sense of the weighting towards top-down regulation of impulsivity, which would bias the self-reporting tool away from convergent validity. Further examination of the factors contributing to the construct of impulsivity as a predominately top-down process within the BIS-11 would show that convergent validity between a purely motor inhibition task and a broad top-down definition of impulsivity is impossible. Further analysis of how self-reporting tools measure broad constructs could also contribute to how to incorporate specific tasks into the diagnostic process of spectrum disorders. The present author argues that lab-based tasks are superior when making the problematic diagnostic distinctions between spectrum disorders such as ADHD and ASD.

In sum, if not particularly strong, this final empirical chapter has found evidence that a measure of motor inhibition correlates with the degree to which a person shows ADHD-

like behaviour. This, in turn suggests that ADHD is related to automatic motor behaviour in the general population.

Chapter 7

General Discussion

ADHD is a condition in which individuals experience a combination of persistent deficits in attention, hyperactivity, and impulsive behaviour. Whilst it affects an estimated 5.7% of children worldwide, the cause of ADHD is unknown (Polanczyk et al., 2007). Academic difficulties are a common problem in those who have ADHD, and this is often the reason for a child's initial referral for clinical evaluation (Loe & Feldman, 2007).

The present thesis has put forward and examined the notion that individuals with ADHD have difficulty inhibiting automatic bottom-up motor behaviour. In Chapter 2, ADHD children, non-ADHD children aged 10 and 11, and controls undertook a task that indexes 'pure' motor inhibition. Results revealed that controls showed a form of inhibition absent in ADHD children. Indeed, the latter showed a facilitation effect. Furthermore, the youngest children also showed a significantly reduced inhibition effect. The possibility was proposed that the basal ganglia, underdeveloped in both ADHD and young children, is the locus of this lack of inhibition.

In Chapter 3, inhibitory control over a gross motor skill was assessed for children with ADHD aged 11 to 14 and a similarly aged control group. These children completed a gross motor task - rolling dice with the explicit goal of keeping it on a table - to measure levels of inhibitory control of a relatively large motor action. Children with ADHD found it significantly more difficult to inhibit motor action enough to keep the dice within the workspace consistently. These results indicate that reduced inhibition significantly impairs gross motor control in individuals with ADHD when completing the dice-rolling task.

Chapter 4 considered the role of inhibition whilst undertaking a fine motor skill (i.e., handwriting). Two groups of children aged 11 to 14, an ADHD group and a control group, were asked to draw a straight line measured by computer software. Any deviation from the straight path was compared to the line the child intended to draw. The ADHD group was found to have significantly more variation than the control group. Once again, this was attributed to reduced levels of inhibitory control when engaged in fine-motor actions.

The phenomenon in which humans have difficulty redirecting attention once it is focused on a feature or property of a stimulus or task was examined in Chapter 5. The phenomenon of Attentional Inertia is thought to be regulated by inhibition. An ADHD and a control group were asked to complete 10 number sentences that began as multiplication and continued until the 9th sentence wherein the sign changed to addition. Children with ADHD were found to fixate on the prior multiplication rule. This was taken as a failure to inhibit the initial multiplication rule in favour of the new addition rule.

Chapter 6 investigated the convergent validity between motor-induced motor inhibition (as used in Chapter 2) and two commonly employed ADHD behaviour scales, as well as the generalisability of the previous findings. Adults were asked to complete the Adult ADHD self-report scale (ASRS) and the Barrett Impulsivity Scale (BIS-11) after undertaking the motor-induced motor inhibition paradigm used in Experiment 2. Whereas scores on the ASRS were associated with the motor task, scores on the BIS-11 were not. Despite the lack of convergence in the BIS-11, these results still suggest that the failure to inhibit a response on a task that indexes automatic motor inhibition is related to ADHD-like behaviour in the general population.

ADHD is a problem with inhibition

Problems with inhibition are the central cognitive abnormality in ADHD and are related to its underlying neuropathology (Barkley, 2001; Polanczk, Willcutt, Salum, Kieling, & Rohde, 2014; Doyle et al., 2005 Nigg, 2001; Willcutt, Doyle, Nigg, Farone, & Pennington, 2005). For example, imaging studies of individuals with ADHD have shown that similar regions of the brain are activated when completing both motor control and executive function tasks related to inhibition (Schoemaker et al., 2005; Hyde et al., 2021; Paloyelis et al., 2007; Rubia et al., 2011; Rubia, Smith, Taylor, & Brammer, 2007). Furthermore, Kumar, Arya, and Agarwal (2022) attributed inhibitory dysfunction to faulty connections between different brain areas. The tasks employed in this thesis, often novel paradigms (e.g., motor-induced motor inhibition, straight-line drawing, dice rolling), intended to trigger various inhibitory effects that could be measured and compared between ADHD and control groups. If problems with automatic inhibitory control are a central component of ADHD, it was expected that children with the condition would perform the inhibitory tasks relatively poorly. This was indeed the case. Significantly, when ADHD individuals undertook a task that is known to involve higher-level cortical processes (i.e., Stroop; Chapter 1), there was no difference between the ADHD group and the control (Experiment 2). This illustrates that ADHD individuals do not necessarily have problems inhibiting *per se*; the issue is inhibiting relatively automatic motor patterns. This suggests that the inhibition of automatic. 'bottom-up' motor processes are an essential component of ADHD.

The present results support several previous empirical findings concerning ADHD and inhibition. For example, Schachar et al. (2007) found a deficit in the ability to cancel and restrain a speeded motor response using a variation of the Stop Signal task. Furthermore,

approximately 50% of individuals with ADHD show dysfunction on a range of paradigms and processes associated with inhibition. These include fine motor precision, manual dexterity, bilateral coordination, balance, postural control, running speed, agility, and limb coordination (Farran, 2020). Glover (2004) attributed deficiencies in motor movements to disinhibited motor planning or deficiencies in the "mechanisms of inhibition". Indeed, he argued that inhibition was the driver of motor planning. Dahan (2017) also identified a lack of inhibition as responsible for the poor motor control seen by individuals with ADHD. Inhibitory processes are employed when selecting an action and managing the timings of motor movements (e.g., reaction times, movement times, and acceleration/velocity parameters).

The Inhibition Model of Barkley (1997) attributes all deficits in ADHD children to a failure of inhibitory control. Based on a comprehensive literature review, Barkley (1997) also suggested that this deficit modulates performance in executive functions. He envisaged a broader definition of inhibition as the mechanism used for regulating bottom-up processes such as arousal and other sensory processes. The model predicts that children with ADHD would perform a variety of top-down and bottom-up lab-based tasks of inhibition poorly compared to their non-disabled peers. Indeed, this was seen in the tasks designed and used in the present research.

Objective lab-based assessments of ADHD

The paradigms used in the present thesis can also be viewed as objective 'lab-based' measures of ADHD, except for the dice rolling and line drawing tasks. Many authors have attempted to uncover and develop such measures for several years. This can be seen

as a ‘holy grail’ of ADHD research. According to Nichols and Waschbusch (2004), the current diagnostic processes involving clinical interviews and behaviour rating scales are costly and prone to bias. This inherent bias is derived from the specific construct used to develop the behaviour rating scale. A scale’s scores reflect the power of the behaviour rating scale to predict a successful diagnosis of ADHD. The effectiveness of subjective versus objective measures in diagnosing ADHD was investigated by Emser et al. (2018). The authors found that different rating scales measure different constructs of ADHD. In other words, the various tools may not measure the same phenomena. Emser et al. (2018) found that approximately 80% of individuals, measured solely by lab-based objective measures, would go on to be diagnosed with ADHD. In combination with traditional subjective measures, successful diagnosis rates approach 90%. The present author concurs with these researchers, who argue that lab-based tasks are superior to the current processes and could be used more effectively.

One of the most commonly used objective measures of ADHD is The Quantified Behavioural Test (QbTest). This diagnostic assessment includes computerised neuropsychological tests that index the three core symptoms using a continuous performance test (CPT). Participants are shown several symbols on a computer screen in the basic procedure. Participants are tasked with pushing the responder button each time a symbol with the same shape and colour is repeated on the screen. An infrared camera, a headband with a reflective marker, measures movement and eye gaze to derive an impulsivity measure. Although promising, Hult et al. (2015) reported limited convergent and discriminant validity compared to the older, more established Conners’ CPT III (CPT-III, 2014). The QbTest may be limited, particularly when trying to distinguish between

symptoms of Autism and ADHD. Hult et al. also note that the test cannot differentiate between impulsivity and hyperactivity. It is 73.8% accurate in predicting that a child will go on to receive an ADHD diagnosis and 86% effective for adults. However, this sensitivity drops to 36% when distinguishing between ADHD and other disorders (Edebol, Helldin, & Norlander, 2012).

Therefore, choosing what to measure and the prominence that a symptom plays in the construct of ADHD seems central to a successful lab-based task. Many CPT tests measure an individual's sustained attention. When an individual has low sustained attention, they are, almost by definition, considered distractible, a primary symptom of ADHD. Common CPTs used in the diagnosis of ADHD include the Integrated Visual and Auditory CPT (IVA-2) Test of Variables of Attention (TOVA) and the Conners Continuous Performance Test (Conners CPT-III). These CPTs are heavily weighted in favour of a person's executive functioning, whilst the QbTest includes tasks that measure executive functioning and impulsivity.

The present work suggests that automatic motor inhibition is central to ADHD in children. One approach to assessment would be for a child to undertake a battery of lab-based tasks that measure inhibition rather than executive function. The author would not want to overstate the case, but the present paradigms are promising in developing lab-based objective measures of ADHD. Motor-induced motor inhibition, dice throwing, straight-line drawing, and the number sentence sign change paradigms have all enabled ADHD individuals to be distinguished from controls. Furthermore, although the effect was only small to medium, the first of these paradigms was also associated with scores on the ASRS.

However, this does present us with a further complication concerning the diagnostic process and the DSM-5. The criteria for ADHD described in the DSM-5 does not mention inhibition. As far as diagnosis is concerned, any underlying “cause or causes” that produce these symptoms is as good as another. It is implied that there may be a root cause of ADHD, but the clinician is only concerned with the observable symptoms. To a certain extent, this makes sense; the symptoms of ADHD are descriptors derived from an arbitrary construction of parts of ADHD. There is no known cause of ADHD or reason for the observed symptoms. However, the fact remains that ADHD exists; the symptoms are tangible, observable, and, more importantly, measurable. I would argue that lab-based objective measures are possible with the right combination of tasks, including the issue of motor inhibition in ADHD.

To use lab-based tasks effectively in diagnosing ADHD, these tasks need to measure the specific trait they intend to measure. Inhibition is theorised to be the main difficulty of individuals with ADHD, but inhibition itself is a construct with developmental, cognitive, and motor dimensions. One central distinction within the inhibition construct is between automatic bottom-up processes and cognitive top-down inhibitory control (Nigg, 2000; Derryberry & Rothbart, 1997). Nigg (2001) argued that including inhibition in the diagnostic construct of AHDH was not considered when developing clinical rating scales. This could partly explain why the results from experiment 8 in this thesis showed that impulsivity was not consistently associated with a self-reporting tool (i.e., the BIS). For example, children with ADHD are, by clinical definition, inattentive, hyperactive, and impulsive. To meet the criteria of dysfunction in impulsivity, children need to exhibit six or more symptoms from a list of nine in the DSM-5. These symptoms could be derived from several inhibitory variants,

including motor inhibition and response inhibition. Consequently, the descriptors are not objective measures and are prone to bias. For example, “Is on the go” is a possible symptom of impulsivity, taken from the DSM-5. Behavioural impulsivity (e.g., variously described as fast, careless, acting without regard in context that are unsafe) as measured by clinical rating scales does not reveal the core psychological process of dysfunction, which could be in arousal, activation, effort, attention, and strength of impulse motivation or inhibition (Milich et al. 1994; Sergeant et al. 1999).

Neuroanatomy of ADHD

In the mid-1960s, Clements (1966) developed a working definition of minimal brain dysfunction. His work separated three main axes with measurable spectra of control: attention, impulse, and motor control. These three spectra measured symptoms and became the diagnostic criteria of minimal brain dysfunction, which would later influence the defining diagnostic characteristics of ADHD as inattention, impulsivity, and hyperactivity (Connor, 2002). It also separated children with ADHD from “the brain-damaged mentally subnormal groups” (Clemens, 1966). This allowed children with average intelligence to be diagnosed with ADHD without assuming brain damage or cognitive impairment (Clemens, 1966).

In the late 1960s, following the criticism that minimum brain dysfunction was too broad and did not differentiate between different groups of children with ADHD, work was undertaken to refine the disorder's definition, culminating with Douglas and Ditto's influential research (1972). They defined hyperactivity as a biological syndrome without environmental cause (Douglas, 1972; Barkley, 2006). The diagnostic criteria for attention

deficit became more defined by the third edition of the DSM, following the research of Douglas (1972), who argued that attention and impulse control were more significant features of the disorder than hyperactivity. Douglas argued that attention and impulse control respond best to stimulants and deemphasised the importance of hyperactivity. This research led the American Psychiatric Association, in the 1980 edition of the DSM, to change the name of the disorder from “hyperkinetic reaction of childhood” to “ADD” or “attention deficit disorder with hyperactivity or without hyperactivity”.

Neural imaging became important to the study of ADHD in the 1990s. Imaging sought to identify correlates of motivational factors, such as deficits in reinforcement, and assessing past ideas regarding the brain's structure or damage to specific parts as a potential cause of ADHD (Barkley, 2006). Neural imaging indeed showed differences in brain structure between people with ADHD and the neurotypical person. Imaging work also showed that the pre-frontal-striated network was smaller in children with ADHD and that supporting neural circuitry throughout the brain does not appear typical (Kondrad et al., 2010; Nagal et al., 2011). ADHD also became associated with alterations in cortical development that seemed to be pre-natal (Shaw et al., 2006) and changes made to the frontal-striatal-thalamic circuitry (Bush, 2011).

Imaging research made it possible to target specific functions within the brain that were not possible earlier, allowing researchers to focus on what Joel Nigg (2012) called “task-related brain activations”. By looking for evidence of where brain functions are taking place, it was thought imaging would clarify how alterations to areas and neural circuits would influence task-related brain function (Bush, 2011). At the start of the use of this technology, researchers typically ignored the neural background activity that occurred

between experimental task conditions, referred to as “spontaneous activations” (Nigg, 2012). Neuroscientists came to see patterns within the spontaneous activations and began mapping these to what is called “synchronised neural oscillations” that occur throughout the brain (Castellanos et al., 2008; Fair et al., 2009; Uddin et al., 2008). When looking at the maps of typical task-related brain functions and the neural circuits associated as tools of comparison between typical function and altered function, this type of research would benefit the understanding of ADHD and other mental disorders (Nigg, 2012).

Role of the Basal Ganglia: connecting Inhibition with ADHD

Albin, Young, and Penney, 1989 (see also Alexander, DeLong, & Strick, 1986; Kemp & Powell, 1971) have provided evidence that the basal ganglia regulate access to the prefrontal cortex. These access points within the basal ganglia, referred to as the direct and indirect route, are a set of interconnected subcortical nuclei manifolds that receive inputs through converging neurons from the entire cortex, including the prefrontal cortex, the traditional seat of executive function. After inhibitory processing, a single output from the basal ganglia is routed only to the frontal lobes. The basal ganglia ‘‘funnels” cortical signals, processing them through a series of inhibitory gates so that they “compete for access” to the prefrontal cortex (Stocco et al., 2017). This evidence suggests that the basal ganglia act as an intake manifold for neural signals, with the output being selected by the basal ganglia using as-yet-unknown selection criteria among competing signals that need to be outputted (through the thalamus) to the prefrontal cortex (Gurney, Prescott, & Redgrave, 2001;

Redgrave, Prescott, & Gurney, 1999; Stephenson-Jones, Samuelsson, Ericsson, Robertson, & Grillner, 2011).

A possible location linking ADHD and inhibition is the basal ganglia, specifically within the inhibitory neural circuitry found within the direct and indirect pathways. The indirect pathway has an inhibitory effect on the thalamus. It has already been established that the indirect pathway in the basal ganglia is responsible for inhibiting the thalamus. When the indirect pathway is active, it inhibits the thalamus, which inhibits the motor cortex, resulting in decreased movement. The location of the thalamus puts it at the beginning of many neural pathways. The thalamus is a relay station for incoming sensory information and is in the brain's centre, just above the brainstem. The thalamus is responsible for sending incoming sensory information to the appropriate area of the brain for processing. The inhibitory effect from this pathway becomes more prominent as cognitive performance becomes more dependent on discarding irrelevant task features (Gerfen & Wilson, 1996). In other words, as the cognitive load becomes heavier due to the "gating" of irrelevant tasks, this inhibitory feature within the neural circuit has more switching power.

The basal ganglia regulate voluntary motor movements, procedural learning, habit learning, eye movements, and general cognition (Stocco, Lebiere, & Anderson, 2010). A growing body of evidence suggests that instead of explicitly being used for motor selection, it is also associated with cognitive task selection (Helie, Chakravarthy, & Moustafa, 2013). For example, damage to the basal ganglia limbic sector has been linked to dysfunction in the reward learning system (Seger & Spiering, 2011). Interestingly, the dynamic learning

theory of ADHD proposes that ADHD results from the duration window between the reward dopamine hit and actions being too short for learning, leading to impulsive behaviours.

Wiecki and Frank (2013) created a computational model of the basal ganglia that simulates how the basal ganglia operates as the critical inhibitory circuit. Wiecki and Frank (2013) state that “planning and executing volitional actions in the face of conflicting habitual responses is a critical aspect of human behaviour”. In the model, interactions between planning and volitional actions are moderated by an “overriding” mechanism, which they identified as inhibition. Their model further showed that inhibition can suppress the habitual selection process, leading to the promotion of executive control. Their model was constructed from behavioural and electrophysiological data on various response inhibition paradigms. This model extends a previous model of action selection they developed of the basal ganglia by including a frontal executive control network that integrates information about sensory input. Another innovation and extension to the model is the inclusion of task rules, which facilitate decision-making via the oculomotor system. They demonstrated the model's competency by simulating the anti-saccade, Simon, and saccade override tasks. Further simulations of the model showed how the conflict between a prepotent and controlled response could be resolved via projections to the subthalamic nucleus. The model also reproduced key behavioural and electrophysiological patterns that simulate key qualitative patterns of global response inhibition demands as required in the Stop-Signal task.

The authors also argued that inhibitory actions of the basal ganglia on the anterior anatomical circuits act as a gate that modulates when to update prefrontal working

memory representations. When acting as a gate, the basal ganglia use inhibitory functions. Specifically, GABAergic connects directly with the thalamus and indirectly through the thalamus using glutamatergic excitatory pathways to the cortex to regulate working memory specifically (Mehler-Wex, Riederer & Gerlach, 2006). Hazey, Frank, and O'Reilly (2007) also proposed a computational model demonstrating the basal ganglia's gating function. This gating is assumed to be inhibitory, undermining the need for a distinction between top-down and bottom-up forms of inhibition, which become less relevant or valuable, effectively kicking the homunculus out of the prefrontal cortex (Hazey et al. 2007).

The neural circuits of the basal ganglia are also known to shape encoded excitatory impulses into more manageable chunks via inhibitory processes (Eisinger, Cernera, Gittis, Gunduz, & Okum, 2019; Hazey et al., 2007). Encoded sensory information travels through the basal ganglia using a pathway called the cortico-basal ganglia-thalamo-cortical loop, or CBGTC loop. This loop has an output that connects with neural pathways as an output that feeds the frontal cortex, with some information looping back into the basal ganglia. There is also an output to the thalamus—this CBGTC loop additionally outputs encoded information back to the cortex. The loop has been associated with dysfunction in ADHD (Sobel et al., 2010). It also contains excitatory and inhibitory neural circuits connecting parts of the loop (Utter & Basso, 2008). It has within it two pathways referred to as the "direct" and "indirect pathways" (Purves et al., 2001).

These developmental differences within the basal ganglia are known to occur as early as seven years old for children with ADHD and are thought to affect behaviour in three ways (Shaw et al., 2014). Firstly, dysfunction in the circuit spanning the ventral striatum

(nucleus accumbens, ventral caudate, and putamen) and limbic cortex has been linked with the abnormal processing of rewards found in ADHD (Coghill, Nigg, Rothenburger, Sonuga-Barke, & Tannock, 2005). Secondly, problems with executive functions, such as cognitive control and working memory, have been tied to anomalies in the circuit linking the lateral prefrontal cortex, specifically the head of caudate and anterior putamen (Willcutt et al., 2005; Barkley, 1997; Hart et al., 2013). Finally, problems in motor planning and control, a key characteristic of ADHD, result from disruptions between the links of the posterior caudal regions of the basal ganglia and sensorimotor cortex. (Cortese et al., 2013; Mostofsky et al., 2006; Kadesjo & Gillberg, 2003). These studies all found functional deficits connected with structural differences. Specifically, it was found that in children with ADHD, the basal ganglia had reduced total striatal volumes specific to the putamen and head of caudate (Valera et al., 2007; Nakao et al., 2011). Two further studies have mapped these particular changes in the surface morphology of the striatum. It was found that there were highly localised surface deformities undetectable by traditional volumetric techniques (Shaw,2014). In both studies, surface area contractions were found in the tail of the caudate, the mid-body of the putamen, and medial, anterior globus pallidus, with less prominent expansion in the posterior putamen (Qiu et al., 2009) and head of the caudate (Sobel & Bansal, 2010). Shaw et al. (2014) mapped basal ganglia development from childhood into late adolescence, using novel methods derived from advances in MRI scanning technology that allowed for the study of the surface morphology of the basal ganglia with previously unavailable levels of spatial resolution. Using this method, Shaw et al. (2014) were able to map the developmental trajectories of children with ADHD aged 4 through 19 across approximately 7,500 surface vertices in the striatum and globus pallidus.

This process showed that developmental difference for children with ADHD was linked to differences in the surface area of ventral striatal surfaces. The ADHD group also showed significant fixed surface area reductions in dorsal striatal regions. These differences were detected in early childhood when participants began the study and were again found in adolescence. Shaw et al. (2014) also found that "progressive, atypical contraction of the ventral striatal surfaces characterises ADHD". This basal ganglia area has been identified with reward processing, confirming findings by Coghill et al. (2005).

Age and development of the Basal Ganglia

The basal ganglia structures take shape within the embryonic central nervous system when two types of cells, neurons and glia, differentiate into specialised structures. This usually occurs at around four weeks prenatal. The basal ganglia establish a regulatory neural network with two other proto-structures: the neural progenitor cells and the neo-cortex (Gohlke et al., 2008). During this phase (week five to around birth), all regions of the cerebral cortex project and establish a directional neural pathway to the basal ganglia, with the output being directed to the frontal lobe along two pathways. During this time, the premotor and supplementary motor cortex connect with the basal ganglia. These connections will become used for attention, working memory, and executive functioning (Grillner & Robertson, 2016; Pyrgaki et al., 2010; Leisman et al., 2014).

The brain's total volume, including the basal ganglia, fluctuates between 7-24 for all individuals. The grey matter volume follows an inverted U-shaped development curve that peaks at age 10-12 years and declines with adolescence while increasing in density (Lenroot et al., 2007). The total mass of grey matter decreases slightly from five to 23 years of age, suggesting that an increase in density may partly counter a decrease in volume (Gennatas

et al., 2017). These changes are reflected in the basal ganglia during this period (Shaw et al., 2014). Gennatas et al. (2017) found that MRI scans indicated a decline in grey matter volume during adolescence and attributed it to a combination of "synaptic pruning of exuberant connections". It is also known that the book of Grey Matter follows the same inverted U-shape trend during that timeframe (Sowell et al. 2001; Gogtay et al. 2004). As grey matter volume declines, white matter grows linearly, increasing throughout childhood into adolescence (Pfefferbaum et al., 1994; Giedd et al., 1999).

Specific studies assessing the development of the basal ganglia after birth are relatively rare. List (2016) reports that "a variety of developmental analyses have been carried out to understand the progressive and regressive neuroanatomical changes, no study has investigated the cortical, subcortical, and cerebellar anatomy in a single large cohort with equal numbers of male and female children and adolescents." In the years since this statement, some research has occurred on basal ganglia development in children and adolescents.

The specific changes to volume and surface area of the basal ganglia have been technically challenging to measure for children ages 7-24. Wierenga et al. (2014) observed "substantial diversity in the developmental trajectories", meaning that the various parts of the basal ganglia develop independently, some linearly, whilst some parts of the basal ganglia follow the inverted U-shape development paths. In a longitudinal study of participants aged 7-24, Wierenga et al. measured the different subcortical grey matter structures of the basal ganglia by using multiple MRI scans over more extended time frames to determine the changes in volume and surface area of the caudate, putamen and nucleus accumbens. They found that as children age, there is a marked decrease in volume. In

contrast to those findings, they also found that the hippocampus, amygdala, pallidum and cerebellum volume showed an inverted U-shaped developmental trajectory similar to whole brain development (Wierenga et al., 2014). Less dramatically, the thalamus showed an initial small increase in volume followed by a slight decrease. These findings support the conclusion that subcortical structures appear not yet fully developed in childhood, similar to the cerebral cortex, and continue to show maturational changes into adolescence.

The possible explanation for these findings could be linked to malformation or damage to a specific area in the basal ganglia. The divergent inputs into the basal ganglia could explain the absence of motor inhibition but retained cognitive inhibition. One path is the putamen, which deals primarily with motor control, whereas the caudate appears to be involved in controlling eye movements and specific cognitive functions (Litvan et al., 1998; Vitk & Giroux, 2002).

Shaw et al. (2014) and (Wierenga et al., 2014) found that the mid-body/tail of the caudate and posterior–inferior regions of the putamen showed surface area reduction in individuals with ADHD. It is possible that motor inhibitory processing first travels through the basal ganglia via the putamen, whilst other cognitive functions travel into the basal ganglia via the caudate. Malformation of this basal ganglia part could be overcome with a stimulant. Ritalin increases the action of neurotransmitters dopamine and norepinephrine by blocking their reabsorption into the neuron. Shaw et al. (2014) demonstrated that for ADHD, malformation of this part of the basal ganglia and other comorbid deficiencies within the basal ganglia contribute to motor inhibitory difficulties.

Ordaz et al. (2013) used fMRI to follow the development of inhibitory control of three neural circuits - the motor response circuit, the executive control circuit, and the error processing circuit - and determined through these scans of the brain that inhibition in all three circuits continues to mature through adolescence into adulthood.

The first circuit to be examined is the motor response control. The motor response control is a circuit that prepares timed, goal-directed responses. The neuropathway of this circuit includes the supplementary motor area, posterior parietal cortex, and putamen (Everling et al., 1999; Rubia et al., 2003). If your putamen remains intact, motor inhibition should remain intact. If your putamen were damaged or immature, it would be expected that motor inhibition would be impaired. The youngest participants in the experiment were found not to inhibit the motor task. This would make sense in context to the natural maturation of the putamen, which is not fully developed by age 10-11. Participants with ADHD also have similar results, which could be linked to the malformation of the putamen.

The motor task has an ambiguous goal during the equal lines phase. When presented with two equal lines, the participant is forced to pick a longer line quickly. Ell et al. (2006) demonstrate that rule-based tasks assume that participants can learn the category structures through an explicit reasoning process, which is impossible in our motor task due to situations where lines are equal in length, but the participant is forced to choose. Ell et al. (2006) also state that in information-integration tasks, optimal performance requires integrating information from two or more stimulus components, often unaware or implicit relationships. Using lines, Ell et al. (2006) found that participants with lesions only on the putamen and not the caudate impaired the ability to complete tasks that followed simple rules whilst retaining information-gathering capability. Their

findings are interesting because they align with the motor task findings for populations with impaired basal ganglia (ADHD) or immature basal ganglia (children aged 10-11).

The second control circuit known to mature with age is the executive control circuit, part of the cortico-basal ganglia thalamic loop (Yager et al., 2015). The executive control circuit coordinates and plans adaptive, goal-directed behaviour (Aron et al., 2004). fMRI analysis of this circuit by Ordaz et al. (2013) was localised to the dorsolateral prefrontal cortex and the ventrolateral prefrontal cortex, which also demonstrates its maturation process over time. This cortico-basal ganglia thamalic loop is regulated by the inhibitory circuits within the basal ganglia. If these circuits are not mature, then information fed forward into the loop would be hypoactive on inhibitory circuits, meaning that there would not be inhibitory activation. Additionally, we would see reduced or absent inhibitory effects on goal-directed tasks. Though not explicitly explored in this experiment, we will explore this circuit in the next chapter.

Ordaz et al. (2013) completed fMRI analysis of the third and final circuit, the error-processing circuit. This circuit consists of the dorsal anterior cingulate, which monitors performance through error detection. When an error is detected, it signals the executive control circuit to adjust activation, leading to improved performance through a feedback loop (Carter et al., 1998; Ridderinkhof et al., 2004; Kerns, 2006). To identify or rule out this particular circuit as a confounding variable, the Stroop and motor tasks were modified to eliminate participants' feedback on potential error detection. Ordaz et al., 2013 found that whole-brain studies suggest that brain function in motor response control regions may be mature by childhood, while brain function associated with error processing may continue to mature throughout adolescence (Rubia et al., 2007; Velanova et al., 2008).

The inverted U-shaped curve in volume and surface area is shared by both ADHD children and their typical peers (Shaw, 2014). However, ADHD children consistently have about 5% less volume than typical peers and 2.5 % less surface area to work with (Shaw et al., 2014). For both groups, the volume and the surface area of the Globus Pallidus reduced slightly by about 50 mm^3. However, the ADHD group starts with 45 mm^3 less than their typical peers (Shaw et al., 2014).

Furthermore, Shaw et al. (2014) found that the mid-body/tail of the caudate and posterior–inferior regions of the putamen showed surface area reduction in individuals with ADHD, confirming findings made by (Wierenga et al., 2014), who used a different method of finding the volume and area of these regions. These basal ganglia regions receive inputs from motor and premotor areas and parietal somatosensory areas. Hypoactivation in this circuit was confirmed in a meta-analysis of fMRI studies of motor inhibitory control (McNab, Leroux, Strand, Thorell, Bergman, & Klingberg, 2008). Fronto-striate-thalamic circuits interact and are known to interact with one another, allowing flexible and adaptive behaviour (Shaw et al., 2014; Haber, 2003). The results of motor experiments seem to confirm and align with prior research findings. In particular, participants with ADHD and young children have been found to have reduced levels of motor inhibitory control. In both populations, the basal ganglia has not fully developed. It should be noted that cognitive connections grow linearly, whilst motor connections seem to grow in the inverted U-developmental curve.

Understanding and tracking the outputs of these pathways will show the importance that inhibition plays in regulating the data streams processed by these pathways. The production of the direct pathway loop is excitatory, with efferent neurons in the cortex

sending an excitatory signal to the striatum. Signals are processed in the striatum, with an inhibitory output sent to the globus pallidus. The globus pallidus then processes the signal, sending another inhibitory signal to the thalamus. The thalamus will end the first round of the processing loop by sending an excitatory signal back to the cortex (Purves, Beau, Williams, Nundy, & Yang, 2001).

The indirect route has a more significant number of inhibitory circuits. Starting with the cortex, an excitatory signal is sent to the striatum for processing and, like the direct route, sends an inhibitory signal onto the globus pallidus exterior. The globus pallidus exterior then processes the signal and sends an inhibitory signal to the subthalamus. The subthalamus processes this signal, sends an excitatory signal to the globus pallidus interior, and processes the signal with an inhibitory output sent to the thalamus.

The thalamus then processes the signal differently than the direct route and sends an inhibitory signal to the cortex (Purves et al. 2001).

Signals from the cortex that use these two pathways within the basal ganglia send both inhibitory and excitatory signals to the cortex. It is important to remember that both pathways are regulated by dopaminergic neurons from the substantia nigra, which excite the excitatory direct path, and inhibit the inhibitory indirect path (Purves et al. 2001). Damage to the substantia nigra has also been linked to ADHD (Acros-Burgos et a., 2010; Krauel et al., 2010).

The inhibitory neural circuits found within the basal ganglia are critical to a prefrontal basal ganglion working model, which is a computational model that explains both the top-down and bottom-up atypical inhibitory symptoms of ADHD and fits within a

general computational model that removes the need for a central executive (Hazey et al., 2007). Further examination of this pathway will show other potential points of failure within this system. Information enters the CBGTC loop from two sources (Parent & Hazrati, 1995). The first input occurs via the striatum, and malformation of this part of the brain has been associated with ADHD (Oldehinkel et al., 2016). The second connection is the subthalamic nucleus, considered part of the basal ganglia system (Fujiyama, 2009) and has two kinds of neurons. The first is connective glutamatergic neurons between the subthalamic nucleus and the basal ganglia system (Levesque & Parent, 2005). The second type of neuron is the GABAergic interneurons, which comprise approximately 7.5% of the neurons found with the subthalamic nucleus neural circuits (Levesque & Parent, 2005). These GABAergic interneurons are thought to function entirely in a "closed" system (Surmeier, Mercer, & Chan, 2005) contained within the subthalamic neural circuit due to a lack of connections between the dendritic arbours of subthalamic neurons that do not connect directly to neurons outside the subthalamic nucleus (Levesque &Parent, 2005). Furthermore, information flows into the subthalamic core from the external globus pallidus (Canteras et al., 1990) via afferent GABAergic neurons of the external globus pallidus and inhibiting connective neurons within the subthalamic nucleus (Galvin et al., 2005). Reduced GABAergic functionality within this system has been linked to ADHD by magnetic resonance spectroscopy (Edden et al. 2012).

Inhibitory Functions at the Synaptic level in the basal ganglia.

At the synapse, two important proteins collect at the pre- and postsynaptic sides of the cell membrane of neurons that form the synapse. Running through the membrane,

these two proteins work together to regulate the distance of the gap between synapses. They are also responsible for the creation and maintenance of synapses. Neurexin is the protein located in the cell membrane of the presynaptic neuron, and neuroligin is the protein found in the cell membrane on the post-synaptic side. When these two proteins work correctly, the connection between neurons is maintained. Malformation of these proteins could contribute to the symptoms of ADHD. Bay et al. (2023) found that Neuroligins are essential in mediating trans-synaptic signalling and shaping the synapse, circuits, and neural network functioning. The mRNA levels of the Neuroligin gene family (NLGN1, NLGN2, NLGN3, and NLGN4X) were studied by Bay et al. (2023) in the peripheral blood of 450 unrelated ASD patients, 450 unrelated ADHD patients, and the normal group included 490, unrelated non-psychiatric children. In ADHD, a significant reduction of NLGN2 and NLGN3 was detected compared to normal children. The Neuroligin family gene may play an essential role in the aetiology of ADHD.

Neural networks rely upon the specific functions that are regulated by neuroligins. Neuroligins come in two varieties. Neuroligin (NLGN) is the presynaptic protein that helps to connect the neurons at the synapse by bridging through the neuron cell membrane at the synapse (Pizzarelli & Cherubini, 2011). It also helps to regulate the space between synapses by linking with a post-synaptic protein called b-neurexin (Scheiffele, Fan, Choih, Fetter, & Serafini, 2000). Three genes, NRXN1, NRXN2 and NRXN3, are responsible for the expression of this protein and play a role in synaptogenesis, or the creation of synapses (Dean & Dresbach 2006). When not expressed correctly within the basal ganglia, these genes could explain a weakened inhibitory effect in individuals with ADHD. They could further explain why ADHD has both a motor and attention component.

Fuster (2017, 2015) argues that the prefrontal cortex makes the brain a pre-adaptive system and further develops the perception action cycle model. In this model, the organism has two worlds. The first is the outer world, where sensory information is generated, and the organism can act out a goal-directed behaviour. The second world is the inner world. In its simplest form, the pre-adaptive cycle enters the brain via some receptor called a mark organ. Within the mark organ, a generalised term for all sense organs, sensory information is encoded and routed to the brain. The organism reacts to this information and acts upon the environment. This is a reactive model, and Fuster (2017) argues that this model can explain observed behaviour for simple organisms. However, for more complex organisms – including humans, Fuster (2017, 2015) discusses that a feedback loop within the cognitive circuits of two places in the brain, the prefrontal cortex and the posterior associated cortex, has evolved specifically to help the organism.

In sum, as several authors have suggested (Mehleir-Wex, Riederer, & Gerlach, 2006; Alexander & Crutcher, 1990; Alexander, Delong, Strick 1986; Seger, 2008; Olivia, Girolamo, di Girolamo, Malandrone, Iaia, Biasi & Maina, 2020), typical inhibitory processes that occur within basal ganglia neural circuits provide at least a mediating mechanism, if not partial explanation, of ADHD.

Basal Ganglia roles in cognitive and motor inhibition

Stocco et al. (2017) stated, "The theory that interference is resolved through top-down representations provides an elegant solution to the problem of cognitive control and has been largely confirmed, but it poses a second problem: How are these representations selected? “

It is currently known that the basal ganglia function this way to help motor movements and actions (Redgrave, Prescott, & Gurney, 1999). Neurocomputational models have been used to show that this exact neurological mechanism can be generalised to cognitive processes (Frank, Samanta, Moustafa, & Sherman, 2007; Booth, Wood, Lu, Houk, & Bitan, 2007; Humphries, Stewart, & Gurney, 2006; O'Reilly & Frank, 2006; Stewart, Choo, & Eliasmith, 2010; Stocco & Lebiere, 2014; Stocco, Lebiere, & Anderson, 2010). O'Reilly and Frank (2006) have shown that the basal ganglia "learn", through adaption, to use the prefrontal cortex as a working memory buffer. This action controls which stimuli are worth memorising through a process called ''gating" (O'Reilly & Frank, 2006). This process allows reinforcement of some processes whilst inhibiting less desirable processes, which can be discarded by the "gating" or selection process that uses competition between sensory signals as they go through the direct and indirect pathways (Albin, Young, & Penney, 1989; DeLong, 1990). It is known that the opposition between the two pathways is mediated by dopamine-driven reinforcement-learning mechanisms (Niv, Duff, & Dayan, 2005; Schultz, 1998; Schultz, Apicella, & Ljungberg, 1993; Schultz, Dayan, & Montague, 1997). Stocco et al. (2017) further developed the idea that the basal ganglia learn through this dopamine structure to optimise the selection of the most relevant signals to send to the prefrontal cortex based on their expected utility (O'Reilly & Frank, 2006; Stocco et al., 2010). It is also known that individuals with ADHD benefit from using Methylphenidate, which acts as a norepinephrine–dopamine reuptake inhibitor (Volkow & Swanson, 2003). As suggested by models of ADHD (Barkley, 1997, 1997b, 2001), the disorder has been associated with functional impairments in some of the brain's neurotransmitter systems (Robison et al., 2017). These functional impairments are particularly apparent in those

functions that involve dopamine in the mesocortical and mesolimbic pathways and deficiencies in norepinephrine in the prefrontal cortex and locus coeruleus (Busardò et al., 2016). Using stimulants, Methylphenidate and amphetamine, to increase stimulation of mesolimbic pathways offers effective treatment of ADHD (Molina et al., 2009; Dodson, 2005). There is also evidence that unintentional accidents, “clumsiness”, can be mitigated in individuals with ADHD with stimulants (Ruiz-Goikoetxea et al., 2018). The combination of effective treatment of attention and motor control with stimulants suggests a possible single point of failure.

Using the N-back task, Gerfen et al. (1990) showed that an increased expression of dopamine D2 receptors explicitly found in the indirect pathway was associated with better performance (Gerfen et al., 1990; Zhang et al., 2007). Because this task requires participants to maintain a moving window of the last N items in a stream of stimuli, it places severe demands on controlling which information is added to the contents of working memory (Stocco et al., 2017). McNab and Klingberg (2008) found that basal ganglia activity, most likely associated with the indirect pathway (because it is localised in the external globus pallidus), correlates with better performance in spatial working memory tasks in which participants are required to memorise the location of certain stimuli (e.g., red dots) while ignoring distractors (e.g., yellow dots). Conversely, reduced expression of D2 receptors has been associated with reduced visuospatial skills (Berman & Noble, 1995) and impaired performance on the Wisconsin Card Sorting Task (Han et al., 2008). One study reported that administration of a D2-agonist cabergoline resulted in improved performance in response inhibition and executive function tasks (Messer, 2011).

ADHD interventions

Overall, the present thesis has presented the possibility that ADHD can, in part, be characterised as a failure to inhibit automatic motor responses, i.e., behaviour not associated with higher-level brain mechanisms. This could have implications for behavioural interventions when treating the condition. Current approaches to managing and diagnosing children with ADHD, excluding medication, assume a *cognitive-heavy* aetiology of the disorder. The present author suggests that this reliance on top-down processing needs to be addressed. ADHD is a problem of inhibition generally; ADHD is not purely an inhibition problem of cognition but also includes inhibitory deficits associated with motor control. This has far-reaching consequences for young people because the inclusion of bottom-up processes is not widely considered by schools, leading to wasted energy and resources on ineffective interventions, policies, training, and frustration for students and teachers.

The present author would further argue that the DSM-V, used by clinicians to complete a formal diagnosis, has contributed to an oversimplification of the disorder by using only two predominant subtypes: inattentive and hyperactive/impulsive. The present author, in line with others (e.g., Sonuga-Barke et al., 2022), suggests that the DSM-V fails to describe the nuances of ADHD. Children have a spectrum of behavioural responses that are difficult to quantify when too narrowly defined. For example, variability within the symptoms of ADHD is hard to quantify with current diagnostic tools. Further complicating matters is the observation made by Sonuga-Barke et al. (2022) that children with ADHD exhibit different profiles of emotional regulation and attention problems. What is not known is how these profiles affect the typical patterns of maturation of brain networks, particularly the rewards centre. By only focusing on the symptoms of ADHD, front-line

professionals, teachers, and parents have not always appreciated that symptoms are the result of actual differences within the brain instead of a failure to behave, pay attention, or sit still. The ADHD brain, as shown by structural MRI scans, is quite different compared to typically developed children of the same age (Sutcubasi et al., 2020; Kumar, Arya, & Agarwak, 2021). Sonuga-Barke et al. (2022) discussed the possibility that people with ADHD have a much slower development of white matter and potentially don't fully mature until they are aged 30.

Future research

The use of medication, particularly dextroamphetamine, has been shown to improve performance on tasks that require inhibition (Sulzer, Sonders & Poulsen, 2005; Howland, 2008; Faaone et al., 2006; Malenka and Nestlet, 2009). There seems to be a trend of non-medical workers favouring cognitive behaviour therapies or even computer-based programs to improve the symptoms of ADHD (Rapport et al., 2013). However, Rapport et al. (2013) demonstrated that "training attention" or executive functions did not significantly improve those skills. The authors concluded, "Collectively, meta-analytic results indicate that claims regarding the academic, behavioural, and cognitive benefits associated with extant cognitive training programs are unsupported in ADHD". Despite these findings, cognitive-based interventions prevail in many teacher-development courses, websites, and other forms of media. With so many commercial interventions available, a discerning parent or professional would find it difficult to know what should be reasonable to expect from a child with ADHD. Though these cognitive training programs seem to fit into a "high expectations" environment of schools, these interventions achieve little, are costly, and

contribute to the stress and anxiety of those often forced into participating. Further study examining the prevalence of these ideas is warranted. Anecdotal observations made by the present author over a long special educational needs career suggest that erroneous ideas about ADHD continue to be spread among teachers, parents, and professionals. A review of the professional training of SENCOs and other frontline education officers who implement ADHD interventions may reveal the true extent of the problem.

Motor-induced motor inhibition.

It is also worth noting that results from the present thesis also extend knowledge about the basic motor-induced motor inhibition phenomenon. As well as reporting the basic phenomenon, a central aspect of Cole and Skarratt (2023; under review) was to eliminate attention as the cause of the effect, precisely, inhibition of return. Experiments 1 and 2 showed that motor-induced motor inhibition is robust enough to occur in children aged 10 and 11 (Experiment 3). Experiments 7 and 8 also revealed the effect when adults performed the task remotely (i.e. outside the standard laboratory set-up). Furthermore, other work by the present author and colleagues (not included in the present thesis) has shown that the basic phenomenon extends beyond decisions about size perception (of two short lines). In a variant of a standard word recognition memory paradigm, Cole, Bubb, and Skarratt (in preparation) showed that participants are less likely to indicate that they recall seeing an item if, to indicate that response, they are required to perform an action just made. For example, if the “old” item (i.e., the item presented previously during the ‘study’ phase of the procedure) is shown on the left of the display and a “new” item (i.e., not seen before) is presented on the right, participants are more likely to indicate that the new item

was shown previously if they have just pressed a left button. The same effect also occurred in a face recognition paradigm.

Summary

This thesis has examined the relationship between ADHD and inhibitory control in various tasks that index predominantly bottom-up processing. Compared with controls, ADHD children failed to inhibit motor behaviour on a motor-induced motor inhibition task, a gross motor task, a delicate motor task, and an attentional inertia task. They did not, however, inhibit responses to any greater extent than controls on the Stroop task. Convergent validity was also found between the degree to which motor-induced motor inhibition occurs and the degree of ADHD-like behaviour as measured by the ASRS (this was not, however, the case for the BIS-11). Overall, the present work suggests that automatic motor inhibition, motor behaviour with little cognitive involvement, is compromised in ADHD individuals.

References

Aase, H., & Sagvolden, T. (2006). Infrequent, but not frequent, reinforcers produce more variable responding and deficient sustained attention in young children with attention-deficit/hyperactivity disorder (ADHD) Journal *of Child Psychology and Psychiatry*, 47(5), 457471.

Adams, J. A. (1971). A closed-loop theory of motor learning. *Journal of motor behavior*, 3(2), 111-150.

Adams, Z. W., Derefinko, K. J., Milich, R., & Fillmore, M. T. (2008). Inhibitory functioning across ADHD subtypes: Recent findings, clinical implications, and future directions*. Developmental Disabilities Research Reviews*, 14(4), 268-275.

Adi-Japha, E., Landau, Y. E., Frenkel, L., Teicher, M., Gross-Tsur, V., & Shalev, R. S. (2007). ADHD and dysgraphia: underlying mechanisms. *Cortex*, 43(6), 700-709.

Adler, L. A., Spencer, T., Faraone, S. V., Kessler, R. C., Howes, M. J., Biederman, J., & Secnik, K. (2006).

Validity of pilot Adult ADHD Self-Report Scale (ASRS) to rate adult ADHD symptoms. *Annals of Clinical Psychiatry*, 18(3), 145-148.

Albajara Sáenz, A., Septier, M., Van Schuerbeek, P., Baijot, S., Deconinck, N., Defresne, P., ... & Massat, I. (2020). ADHD and ASD: distinct brain patterns of inhibition-related activation?. *Translational psychiatry*, 10(1), 1-10.

Albin, R. L., Young, A. B., & Penney, J. B. (1989). The functional anatomy of basal ganglia disorders. *Trends in neurosciences*, 12(10), 366-375.

Alexander, G. E., & Crutcher, M. D. (1990). Functional architecture of basal ganglia circuits: neural substrates of parallel processing. *Trends in neurosciences*, 13(7), 266-271.

Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual review of neuroscience*, 9(1), 357-381.

Alloway, T. P., Gathercole, S. E., Kirkwood, H., & Elliott, J. (2009). The cognitive and behavioral characteristics of children with low working memory. *Child development*, 80(2), 606-621.

Alston, J. (1983). A legibility index: Can handwriting be measured?. *Educational Review*, 35(3), 237-242.

Alwitt, L. F., Anderson, D. R., Lorch, E. P., & Levin, S. R. (1980). Preschool children's visual attention to attributes of television. *Human Communication Research*, 7(1), 52-67.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Washington, DC: Author.

Andersen, D. W. (1965). Handwriting research: Movement and quality. *Elementary English*, 42(1), 45-53.

Anderson, D. R., & Lorch, E. P. (1983). Looking at television: Action or reaction. Children's understanding of television: Research on attention and comprehension, 1-33.

Anderson, D. R., Alwitt, L. F., Lorch, E. P., & Levin, S. R. (1979). Watching children watch television. In *Attention and cognitive development* (pp. 331-361). Springer, Boston, MA.

Anderson, D. R., Choi, H. P., & Lorch, E. P. (1987). Attentional inertia reduces distractibility during

young children's TV viewing. *Child Development*, 798-806.

Anderson, M. C., & Spellman, B. A. (1995). On the status of inhibitory mechanisms in cognition: memory retrieval as a model case. *Psychological review*, 102(1), 68.

Andersson, O., & Grillner, S. (1983). Peripheral control of the cat's step cycle: II. Entrainment of the central pattern generators for locomotion by sinusoidal hip movements during "fictive locomotion". *Acta Physiologica Scandinavica*, 118(3), 229-239.

Arcia, E., Frank, R., Sanchez-LaCay, A., & Fernáindez, M. C. (2000). Teacher understanding of ADHD as reflected in attributions and classroom strategies. *Journal of attention disorders*, 4(2), 91-101.

Arcos-Burgos, M., Jain, M., Acosta, M. T., Shively, S., Stanescu, H., Wallis, D., ... & Muenke, M. (2010). A common variant of the latrophilin 3 gene, LPHN3, confers susceptibility to ADHD and predicts effectiveness of stimulant medication. *Molecular psychiatry*, 15(11), 1053-1066.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in cognitive sciences*, 8(4), 170-177.

Atlan, H. (1977). Sources of information in biological systems. *IFAC Proceedings Volumes*, 10(12), 177-184.

Augustinova, M., & Ferrand, L. (2014). Social priming of dyslexia and reduction of the Stroop effect: What component of the Stroop effect is actually reduced?. *Cognition*, 130(3), 442-454.

Baddeley, A. D., & Hitch, G. (1974). Working memory. In *Psychology of learning and motivation* (Vol.

8, pp. 47-89). Academic press.

Bagatell, N., Lawrence, J., Schwartz, M., & Vuernick, W. (2013). Occupational therapy student experiences and transformations during fieldwork in mental health settings. *Occupational Therapy in Mental Health*, 29(2), 181-196.

Bangert-Drowns, R. L., Hurley, M. M., & Wilkinson, B. (2004). The effects of school-based writing-to-learn interventions on academic achievement: A meta-analysis. *Review of educational research*, 74(1), 29-58.

Barboriak, D. P., Padua, A. O., York, G. E., & MacFall, J. R. (2005). Creation of DICOM—aware applications using ImageJ. *Journal of Digital Imaging*, 18(2), 91-99.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological bulletin*, 121(1), 65.

Barkley, R. A. (1997). ADHD and the nature of self-control. Guilford press.

Barkley, R. A. (2001). The inattentive type of ADHD as a distinct disorder: What remains to be done. *Clinical Psychology: Science and Practice*, 8(4), 489-493.

Barnhart, W. R., & Buelow, M. T. (2017). Assessing impulsivity: Relationships between behavioral and self-report measures in individuals with and without self-reported ADHD. *Personality and Individual Differences*, 106, 41-45.

Barry, R. J., & De Blasio, F. M. (2015). Performance and ERP components in the equiprobable go/no-go task: Inhibition in children. *Psychophysiolog*y, 52(9), 1228-1237.

Bass-Haugen, J., & Mathiowetz, V. (2008). Optimizing motor behavior using the occupational therapy

task-oriented approach. In *Occupational therapy for physical dysfunction* (pp. 598-617). Lippincott Williams & Wilkins.

Bayard, S., Raffard, S., & Gely-Nargeot, M. C. (2011). Do facets of self-reported impulsivity predict decision-making under ambiguity and risk? Evidence from a community sample. *Psychiatry Research*, 190(2-3), 322-326.

Berger, E. M., Fehr, E., Hermes, H., Schunk, D., & Winkel, K. (2020). The impact of working memory training on children's cognitive and noncognitive skills. *NHH Dept. of Economics Discussion Paper*, (09).

Berman, S. M., & Noble, E. P. (1995). Reduced visuospatial performance in children with the D2 dopamine receptor A1 allele. *Behavior genetics*, 25(1), 45-58.

Bernbaum, M., Goodnow, J., & Lehman, E. (1974). Relationships among perceptual-motor tasks: Tracing and copying. *Journal of Educational Psychology*, 66(5), 731.

Bernstein, N. (1966). The co-ordination and regulation of movements. *The co-ordination and regulation of movements*.

Beyer, R. (1999). Motor proficiency of boys with attention deficit hyperactivity disorder and boys with learning disabilities. *Adapted Physical Activity Quarterly*, 16(4), 403-414.

Biotteau, M., Danna, J., Baudou, É., Puyjarinet, F., Velay, J. L., Albaret, J. M., & Chaix, Y. (2019). Developmental coordination disorder and dysgraphia: signs and symptoms, diagnosis, and rehabilitation. *Neuropsychiatric disease and treatment*, 15, 1873.

Bird, H. R., Gould, M. S., & Staghezza-Jaramillo, B. M. (1994). The comorbidity of ADHD in a community sample of children aged 6 through 16 years. *Journal of child and family studies*, 3(4), 365-378.

Bjorklund, D. F., & Kipp, K. (2002). Social cognition, inhibition, and theory of mind: The evolution of human intelligence.

Bobath, B. (1948). A new treatment of lesions of the upper motor neurone. *British Journal of Physical Medicine*, 2(1), 26-29.

Booth, J. R., Burman, D. D., Meyer, J. R., Gitelman, D. R., Parrish, T. B., & Mesulam, M. M. (2002). Functional anatomy of intra-and cross-modal lexical tasks. *Neuroimage*, 16(1), 7-22.

Booth, J. R., Wood, L., Lu, D., Houk, J. C., & Bitan, T. (2007). The role of the basal ganglia and cerebellum in language processing. *Brain research*, 1133, 136-144.

Boucher, L., Palmeri, T. J., Logan, G. D., & Schall, J. D. (2007). Inhibitory control in mind and brain: an interactive race model of countermanding saccades. *Psychological review*, 114(2), 376.

Bradley, C. (1937). The behavior of children receiving benzedrine. *American journal of Psychiatry*, 94(3), 577-585.

Breese, B. B. (1899). On inhibition. Psychological Monographs: General and Applied, 3(1).

Broadbent, D. E. (1977). Levels, hierarchies, and the locus of control. *Quarterly Journal of Experimental Psychology*, 29(2), 181-201.

Brown, T., & Chien, C. W. (2010). Top-down or bottom-up occupational therapy assessment: which way do we go?. *British Journal of Occupational Therapy*, 73(3), 95-95.

Bruinsma, C., & Nieuwenhuis, C. (1991). A new method for the evaluation of handwriting material. *Development of graphic skills*, 41-49.

Brunnstrom, S. (1956). Associated reactions of the upper extremity in adult patients with hemiplegia: an approach, to training. *Physical Therapy*, 36(4), 225-236.

Bunge, S. A., Dudukovic, N. M., Thomason, M. E., Vaidya, C. J., & Gabrieli, J. D. (2002). Immature frontal lobe contributions to cognitive control in children: evidence from fMRI. *Neuron*, 33(2), 301-311.

Burns, J. J., & Anderson, D. R. (1993). Attentional inertia and recognition memory in adult television viewing. *Communication Research*, 20(6), 777-799.

Burtner, P. A., McMain, M. P., & Crowe, T. K. (2002). Survey of occupational therapy practitioners in southwestern schools: Assessments used and preparation of students for school-based practice. *Physical & Occupational Therapy in Pediatrics*, 22(1), 25-39.

Bush, G. (2011). Cingulate, frontal, and parietal cortical dysfunction in attention-deficit/hyperactivity disorder. *Biological psychiatry*, 69(12), 1160-1167.

Canteras, N. S., Shammah-Lagnado, S. J., Silva, B. A., & Ricardo, J. A. (1990). Afferent connections of the subthalamic nucleus: a combined retrograde and anterograde horseradish peroxidase study in the rat. *Brain research*, 513(1), 43-59.

Capodieci, A., Lachina, S., & Cornoldi, C. (2018). Handwriting difficulties in children with attention deficit hyperactivity disorder (ADHD). *Research in developmental disabilities*, 74, 41-49.

Carlson, S. M., Moses, L. J., & Breton, C. (2002). How specific is the relation between executive function and theory of mind? Contributions of inhibitory control and working memory. *Infant and Child Development: An International Journal of Research and Practice*, 11(2), 73-92.

Carlson, S. M., Moses, L. J., & Claxton, L. J. (2004). Individual differences in executive functioning and theory of mind: An investigation of inhibitory control and planning ability. *Journal of experimental child psychology*, 87(4), 299-319.

Carr, L. A., Nigg, J. T., & Henderson, J. M. (2006). Attentional versus motor inhibition in adults with attention-deficit/hyperactivity disorder. *Neuropsychology*, 20(4), 430.

Carrillo-De-La-Peña, M. T., Otero, J. M., & Romero, E. (1993). Comparison among various methods of assessment of impulsiveness. *Perceptual and motor skills*, 77(2), 567-575.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, 280(5364), 747-749.

Castellanos, F. X., Margulies, D. S., Kelly, C., Uddin, L. Q., Ghaffari, M., Kirsch, A., ... & Milham, M. P. (2008). Cingulate-precuneus interactions: a new locus of dysfunction in adult attentiondeficit/hyperactivity disorder. *Biological psychiatry*, 63(3), 332-337.

Chai, J., Wu, R., Li, A., Xue, C., Qiang, Y., Zhao, J., ... & Yang, Q. (2022). Classification of mild cognitive impairment based on handwriting dynamics and qEEG. *Computers in Biology and Medicine*, 106418.

Chen, J. Y., & Cherng, R. J. (2013). The proximate unit in Chinese handwritten character production. *Frontiers in psychology*, 4, 517.

Chen, Y. Y., Liaw, L. J., Liang, J. M., Hung, W. T., Wu, J. H., & Wu, W. L. (2011). A pilot study: Force control on ball throwing in children with attention deficit hyperactivity disorder. *Procedia Engineering*, 13, 328-333.

Choi, H. P., & Anderson, D. R. (1991). A temporal analysis of free toy play and distractibility in young children. *Journal of Experimental Child Psychology*, 52(1), 41-69.

Christiansen, A. S. (2000). Persisting motor control problems in 11-to 12-year-old boys previously diagnosed with deficits in attention, motor control and perception (DAMP). *Developmental Medicine and Child Neurology*, 42(1), 4-7.

Chung, P. J., Patel, D. R., & Nizami, I. (2020). Disorder of written expression and dysgraphia: definition, diagnosis, and management. *Translational pediatrics*, 9(Suppl 1), S46.

Ciesielski, K. T., & Harris, R. J. (1997). Factors related to performance failure on executive tasks in autism. *Child Neuropsychology*, 3(1), 1-12.

Cilia, N. D., Stefano, C. D., Fontanella, F., Molinara, M., & Scotto Di Freca, A. (2019, September). Handwriting analysis to support alzheimer's disease diagnosis: a preliminary study. In *International Conference on Computer Analysis of Images and Patterns* (pp. 143-151). Springer, Cham.

Clark, G. J. (2010). The relationship between handwriting, reading, fine motor and visual-motor skills in kindergarteners. Iowa State University.

Clements, S. D. (1966). *Minimal brain dysfunction in children: Terminology and identification: Phase one of a three phase project* (Vol. 55). US Department of Health, Education and Welfare.

Clements, S. D., & Peters, J. E. (1962). Minimal brain dysfunctions in the school-age child: diagnosis and treatment. *Archives of General Psychiatry*, 6(3), 185-197.

Cloutman, L., Gottesman, R., Chaudhry, P., Davis, C., Kleinman, J. T., Pawlak, M., ... & Hillis, A. E. (2009). Where (in the brain) do semantic errors come from?. *Cortex*, 45(5), 641-649.

Coghill, D., Nigg, J., Rothenberger, A., Sonuga-Barke, E., & Tannock, R. (2005). Whither causal models in the neuroscience of ADHD?. *Developmental Science*, 8(2), 105-114.

Cole, G. G. & Skarratt, P. A. (under review). Motor Induced Motor Inhibition.

Cole, G. G. & Skarratt, P. A. (2022). Motor Induced Motor Inhibition. *Paper presented at the Psychonomic Society conference Boston, Mass.*

Cole, G. G., Welsh, T. N., & Skarratt, P. A. (2019). The role of transients in action observation. *Attention, Perception, & Psychophysics*, 81(7), 2177-2191.

Cole, M. B., & Tufano, R. (2008). Applied theories in occupational therapy. A practical approach. Thorofare, NJ: Slack Inc.

Collins, F.L., Baer, G.R., Walls, N., & Jackson, M.S. (1980). The development of a behavioral assessment technique of evaluating gradual change in handwriting performance. *Behavioral Assessment*, 2(4), 369—387.

Congdon, E., Mumford, J. A., Cohen, J. R., Galvan, A., Canli, T., & Poldrack, R. A. (2012). Measurement and reliability of response inhibition. *Frontiers in psychology*, 3, 37.

Connor, D. F. (2002). Preschool attention deficit hyperactivity disorder: a review of prevalence, diagnosis, neurobiology, and stimulant treatment. *Journal of Developmental & Behavioral Pediatrics*, 23, S1-S9.

Cortese, S., Holtmann, M., Banaschewski, T., Buitelaar, J., Coghill, D., Danckaerts, M., ... & European ADHD Guidelines Group. (2013). Practitioner review: current best practice in the management of adverse events during treatment with ADHD medications in children and adolescents. *Journal of Child Psychology and Psychiatry*, 54(3), 227-246.

Cyr, M., & Brown, C. S. (1998). Current drug therapy recommendations for the treatment of attention deficit hyperactivity disorder. *Drugs*, 56(2), 215-223.

Dagenbach, D., & Carr, T. H. (Eds.). (1994). *Inhibitory processes in attention, memory, and language* (pp. 189-239). San Diego: Academic Press.

Dahan, A., & Reiner, M. (2017). Evidence for deficient motor planning in ADHD. *Scientific reports*, 7(1), 1-10.

Daneman, M., & Carpenter, P. A. (1980). Individual differences in working memory and reading. *Journal of verbal learning and verbal behavior*, 19(4), 450-466.

Daneman, M., & Merikle, P. M. (1996). Working memory and language comprehension: A meta-analysis. *Psychonomic bulletin & review*, 3(4), 422-433.

Danielson, M. L., Bitsko, R. H., Ghandour, R. M., Holbrook, J. R., Kogan, M. D., & Blumberg, S. J. (2018). Prevalence of parent-reported ADHD diagnosis and associated treatment among US children and adolescents, 2016. *Journal of Clinical Child & Adolescent Psychology*, 47(2), 199212.

Danielson, M. L., Visser, S. N., Chronis-Tuscano, A., & DuPaul, G. J. (2018). A national description of treatment among United States children and adolescents with attentiondeficit/hyperactivity disorder. *The Journal of pediatrics*, 192, 240-246.

Daruna, J. H., & Barnes, P. A. (1993). A neurodevelopmental view of impulsivity.

D'Ausilio, A., Pulvermüller, F., Salmas, P., Bufalari, I., Begliomini, C., & Fadiga, L. (2009). The motor somatotopy of speech perception. *Current Biology*, 19(5), 381-385.

Davidson, M. C., Amso, D., Anderson, L. C., & Diamond, A. (2006). Development of cognitive control and executive functions from 4 to 13 years: Evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia,* 44(11), 2037-2078.

Dean, C., & Dresbach, T. (2006). Neuroligins and neurexins: linking cell adhesion, synapse formation and cognitive function. *Trends in neurosciences*, 29(1), 21-29.

dela Peña, I. C., Yoon, S. Y., Kim, Y., Park, H., Kim, K. M., Ryu, J. H., ... & Cheong, J. H. (2013). 5, 7-Dihydroxy-6-methoxy-4'-phenoxyflavone, a derivative of oroxylin A improves attentiondeficit/hyperactivity disorder (ADHD)-like behaviors in spontaneously hypertensive rats.
European journal of pharmacology, 715(1-3), 337-344.

DeLong, M. R. (1990). Primate models of movement disorders of basal ganglia origin. *Trends in neurosciences*, 13(7), 281-285.

Dempster, F. N., & Brainerd, C. J. (1995). New perspectives on interference and inhibition in cognition: Final comments. In Interference and inhibition in cognition (pp. 401-407). *Academic Press*.

Derrickson, B., & Tortora, G. J. (2014). Principles of anatomy & physiology. *Wiley*.

Derryberry, D., & Rothbart, M. K. (1997). Reactive and effortful processes in the organization of temperament. *Development and psychopathology*, 9(4), 633-652.

Diamond, A., & Kirkham, N. (2001, October). Card sorting by children of 3 and 4 years and task switching by older children: inhibition needed to overcome attentional inertia. In *Cognitive Development Society Annual Meeting*, Virginia Beach, VA, October.

DiCarlo, J. J., Zoccolan, D., & Rust, N. C. (2012). How does the brain solve visual object recognition?. *Neuron*, 73(3), 415-434.

Dodson, W. W. (2005). Pharmacotherapy of adult ADHD. *Journal of clinical psychology*, 61(5), 589-606.

Doyle, A. E., Faraone, S. V., Seidman, L. J., Willcutt, E. G., Nigg, J. T., Waldman, I. D., ... & Biederman, J. (2005). Are endophenotypes based on measures of executive functions useful for molecular genetic studies of ADHD?. *Journal of Child Psychology and Psychiatry*, 46(7), 774803.

Doyle, S., Wallen, M., & Whitmont, S. (1995). Motor skills in Australian children with attention deficit hyperactivity disorder. *Occupational Therapy International*, 2(4), 229-240.

Dunford, C., Bannigan, K., & Wales, L. (2013). Measuring activity and participation outcomes for children and youth with acquired brain injury: An occupational therapy perspective. *British Journal of Occupational Therapy*, 76(2), 67-76.

DuPaul, G. J., Power, T. J., Anastopoulos, A. D., & Reid, R. (1998). *ADHD Rating Scale—IV: Checklists, norms, and clinical interpretation*. Guilford press.

DuPaul, G. J., Weyandt, L. L., & Janusis, G. M. (2011). ADHD in the classroom: Effective intervention strategies. *Theory into practice*, 50(1), 35-42.

Edden, R. A., Crocetti, D., Zhu, H., Gilbert, D. L., & Mostofsky, S. H. (2012). Reduced GABA concentration in attention-deficit/hyperactivity disorder. *Archives of general psychiatry*, 69(7), 750-753.

Edebol, H., Helldin, L., & Norlander, T. (2012). Objective measures of behavior manifestations in adult ADHD and differentiation from participants with bipolar II disorder, borderline personality disorder, participants with disconfirmed ADHD as well as normative participants. *Clinical practice and epidemiology in mental health*: *CP & EMH, 8*, 134.

Efstratopoulou, M., Janssen, R., & Simons, J. (2012). Differentiating children with attention-deficit/hyperactivity disorder, conduct disorder, learning disabilities and autistic spectrum disorders by means of their motor behaviour characteristics. *Research in developmental disabilities*, 33(1), 196-204.

Eisinger, R. S., Cernera, S., Gittis, A., Gunduz, A., & Okun, M. S. (2019). A review of basal ganglia circuits and physiology: application to deep brain stimulation. *Parkinsonism & related disorders*, 59, 9-20.

Eliceiri, K. W., & Rueden, C. (2005). Tools for visualizing multidimensional images from living specimens. *Photochemistry and photobiology*, 81(5), 1116-1122.

Ell, S. W., Marchant, N. L., & Ivry, R. B. (2006). Focal putamen lesions impair learning in rule-based, but not information-integration categorization tasks. *Neuropsychologia*, 44(10), 1737-1751.

Emser, T. S., Johnston, B. A., Steele, J. D., Kooij, S., Thorell, L., & Christiansen, H. (2018). Assessing ADHD symptoms in children and adults: evaluating the role of objective measures. *Behavioral and Brain Functions*, 14(1), 1-14.

Engelhardt, P. E., Nigg, J. T., Carr, L. A., & Ferreira, F. (2008). Cognitive inhibition and working memory in attention-deficit/hyperactivity disorder. *Journal of abnormal psychology*, 117(3), 591.

Enright, S. J., & Beech, A. R. (1993). Reduced cognitive inhibition in obsessive—compulsive disorder. *British Journal of Clinical Psychology*, 32(1), 67-74.

Enticott, P. G., Ogloff, J. R., & Bradshaw, J. L. (2006). Associations between laboratory measures of executive inhibitory control and self-reported impulsivity. *Personality and Individual Differences*, 41(2), 285-294.

Erlebacher, A., & Herrick, V. E. (1961). Quality of handwriting today and yesterday. *The Elementary School Journal*, 62(2), 89-93.

Everling, S., Dorris, M. C., Klein, R. M., & Munoz, D. P. (1999). Role of primate superior colliculus in preparation and execution of anti-saccades and pro-saccades. *Journal of Neuroscience*, 19(7), 2740-2754.

Fair, D. A., Cohen, A. L., Power, J. D., Dosenbach, N. U., Church, J. A., Miezin, F. M., ... & Petersen, S. E. (2009). Functional brain networks develop from a "local to distributed" organization. *PLoS computational biology*, 5(5), e1000381.

Faraone, S. V., Doyle, A. E., Mick, E., & Biederman, J. (2001). Meta-analysis of the association between the 7-repeat allele of the dopamine D4 receptor gene and attention deficit hyperactivity disorder. *American Journal of Psychiatry*, 158(7), 1052-1057.

Farran, B., Montenegro, R. C., Kasa, P., Pavitra, E., Huh, Y. S., Han, Y. K., ... & Raju, G. S. R. (2020). Folate-conjugated nanovehicles: Strategies for cancer therapy. *Materials Science and*

Engineering: C, 107, 110341.

Feder, K. P., & Majnemer, A. (2007). Handwriting development, competency, and intervention. *Developmental Medicine & Child Neurology*, 49(4), 312-317.

Feder, K., Majnemer, A., & Synnes, A. (2000). Handwriting: Current trends in occupational therapy practice. *Canadian Journal of Occupational Therapy*, 67(3), 197-204.

Feldman, J. A., & Ballard, D. H. (1982). Connectionist models and their properties. *Cognitive science*, 6(3), 205-254.

Fentress, J. C. (1973). Development of grooming in mice with amputated forelimbs. *Science*, 179(4074), 704-705.

Fetterman, A. K., Robinson, M. D., & Meier, B. P. (2012). Anger as "seeing red": Evidence for a perceptual association. *Cognition & emotion*, 26(8), 1445-1458.

Folk, J. R., Rapp, B., & Goldrick, M. (2002). The interaction of lexical and sublexical information in spelling: What's the point?. *Cognitive Neuropsychology*, 19(7), 653-671.

Fossati, A., Di Ceglie, A., Acquarini, E., & Barratt, E. S. (2001). Psychometric properties of an Italian version of the Barratt Impulsiveness Scale-11 (BIS-11) in nonclinical subjects. *Journal of clinical psychology*, 57(6), 815-828.

Frank, M. J. (2006). Hold your horses: a dynamic computational role for the subthalamic nucleus in decision making. *Neural networks*, 19(8), 1120-1136.

Frank, M. J., Samanta, J., Moustafa, A. A., & Sherman, S. J. (2007). Hold your horses: impulsivity, deep brain stimulation, and medication in parkinsonism. *science*, 318(5854), 1309-1312.

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and interference control functions: a latent-variable analysis. *Journal of experimental psychology*: General, 133(1), 101.

Fujiyama, F. (2009). Anatomical connections of the basal ganglia. *Brain and Nerve= Shinkei Kenkyu no Shinpo, 61*(4), 341-349.

Fuster, J. (2015). The prefrontal cortex. Academic press.

Fuster, J. M. (2017). Prefrontal cortex in decision-making: The perception–action cycle. In *Decision neuroscience* (pp. 95-105). Academic Press.

Gadow, K. D., DeVincent, C. J., & Pomeroy, J. (2006). ADHD symptom subtypes in children with pervasive developmental disorder. *Journal of autism and developmental disorders*, 36(2), 271-283.

Gadow, K. D., Devincent, C. J., Pomeroy, J., & Azizian, A. (2005). Comparison of DSM-IV symptoms in elementary school-age children with PDD versus clinic and community samples. Autism, 9(4), 392-415.

Galvan, A., Villalba, R. M., West, S. M., Maidment, N. T., Ackerson, L. C., Smith, Y., & Wichmann, T. (2005). GABAergic modulation of the activity of globus pallidus neurons in primates: in vivo analysis of the functions of GABA receptors and GABA transporters. *Journal of neurophysiology*, 94(2), 990-1000.

Gandolfi, E., Viterbori, P., Traverso, L., & Usai, M. C. (2014). Inhibitory processes in toddlers: a latent-variable approach. *Frontiers in psychology*, 5, 381.

Garcia-Garcia, M., Nikolaidis, A., Bellec, P., Craddock, R. C., Cheung, B., Castellanos, F. X., & Milham, M. P. (2018). Detecting stable individual differences in the functional organization of the human basal ganglia. *NeuroImage*, 170, 68-82.

Gennatas, E. D., Avants, B. B., Wolf, D. H., Satterthwaite, T. D., Ruparel, K., Ciric, R., ... & Gur, R. C. (2017). Age-related effects and sex differences in gray matter density, volume, mass, and cortical thickness from childhood to young adulthood. *Journal of Neuroscience*, 37(20), 50655073.

Gerbing, D. W., Ahadi, S. A., & Patton, J. H. (1987). Toward a conceptualization of impulsivity: Components across the behavioral and self-report domains. *Multivariate behavioral research*, 22(3), 357-379.

Gerfen, C. R., & Wilson, C. J. (1996). Chapter II The basal ganglia. In *Handbook of chemical neuroanatomy* (Vol. 12, pp. 371-468). Elsevier.

Gerfen, C. R., Engber, T. M., Mahan, L. C., Susel, Z. V. I., Chase, T. N., Monsma Jr, F. J., & Sibley, D. R. (1990). D1 and D2 dopamine receptor-regulated gene expression of striatonigral and striatopallidal neurons. *Science*, 250(4986), 1429-1432.

Gering, E., & Atkinson, C. T. (2004). A rapid method for counting nucleated erythrocytes on stained blood smears by digital image analysis. *Journal of Parasitology*, 90(4), 879-881.

Gesell, A., & Amatruda, C. S. (1941). Developmental diagnosis; normal and abnormal child development.

Gibson, E. J., & Pick, A. D. (2000). *An ecological approach to perceptual learning and development.* Oxford University Press, USA.

Gibson, J. J., & Carmichael, L. (1966). *The senses considered as perceptual systems* (Vol. 2, No. 1, pp. 44-73). Boston: Houghton Mifflin.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., ... & Rapoport, J. L. (1999). Brain development during childhood and adolescence: a longitudinal MRI study. *Nature neuroscience*, 2(10), 861-863.

Gillberg, C., & Kadesjö, B. (2003). Why bother about clumsiness? The implications of having developmental coordination disorder (DCD). *Neural plasticity*, 10(1-2), 59-68.

Gizer, I. R., Ficks, C., & Waldman, I. D. (2009). Candidate gene studies of ADHD: a meta-analytic review. *Human genetics*, 126(1), 51-90.

Glover, S. (2004). Separate visual representations in the planning and control of action. *Behavioral and brain sciences*, 27(1), 3-24.

Gnanavel, S., Sharma, P., Kaushal, P., & Hussain, S. (2019). Attention deficit hyperactivity disorder and comorbidity: A review of literature. *World journal of clinical cases*, 7(17), 2420.

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., ... & Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences*, 101(21), 8174-8179.

Gohlke, J. M., Armant, O., Parham, F. M., Smith, M. V., Zimmer, C., Castro, D. S., ... & Guillemot, F. (2008). Characterization of the proneural gene regulatory network during mouse telencephalon development. *BMC biology*, 6(1), 1-18.

Gordon, A. D. (1987). A review of hierarchical classification. *Journal of the Royal Statistical Society: Series A (General)*, 150(2), 119-137.

Gorfein, D. S., & MacLeod, C. M. (2007). *Inhibition in cognition* (pp. xvii-337). American Psychological Association.

Grace, A. A. (2001). Psychostimulant actions on dopamine and limbic system function: Relevance to the pathophysiology and treatment of ADHD.

Graham, S., & Perin, D. (2007). A meta-analysis of writing instruction for adolescent students. *Journal of educational psychology*, 99(3), 445.

Gray, J. A., Feldon, J., Rawlins, J. N. P., Hemsley, D. R., & Smith, A. D. (1991). The neuropsychology of schizophrenia. *Behavioral and Brain Sciences*, 14(1), 1-20.

Gray, P. (1982). "Developmental lag" in the evolution of technique for psychoanalysis of neurotic conflict. *Journal of the American Psychoanalytic Association*, 30(3), 621-655.

Greenwald, E. A., Persky, H. R., Campbell, J. R., & Mazzeo, J. (1999). The NAEP 1998 Writing Report Card for the Nation and the States (NCES 1999-462). US Department of Education. *National Center for Education Statistics. Washington, DC: US Government Printing Office*.

Gregory, R. L. (1968). Perceptual illusions and brain models. Proceedings of the Royal Society of London. Series B. *Biological Sciences*, 171(1024), 279-296.

Gregory, R. L. (1970). The intelligent eye.

Grillner, S., & Robertson, B. (2016). The basal ganglia over 500 million years. *Current Biology*, 26(20), R1088-R1100.

Grudin, J. T. (1983). Error patterns in novice and skilled transcription typing. In *Cognitive aspects of skilled typewriting* (pp. 121-143). Springer, New York, NY.

Guan, Y., & Wessel, J. R. (2022). Two types of motor inhibition after action errors in humans. *Journal of neuroscience*, 42(38), 7267-7275.

Gurney, K., Prescott, T. J., & Redgrave, P. (2001). A computational model of action selection in the basal ganglia. I. A new functional anatomy. *Biological cybernetics*, 84(6), 401-410.

Haber, S. N. (2003). The primate basal ganglia: parallel and integrative networks. *Journal of chemical neuroanatomy*, 26(4), 317-330.

Hamstra-Bletz, L., & Blöte, A. W. (1990). Development of handwriting in primary school: A longitudinal study. *Perceptual and motor skills*, 70(3), 759-770.

Hamstra-Bletz, L., DeBie, J., & Den Brinker, B. P. L. M. (1987). Concise evaluation scale for children's handwriting. *Lisse: Swets*, 1, 623-662.

Harrington, G. S., Farias, D., Davis, C. H., & Buonocore, M. H. (2007). Comparison of the neural basis for imagined writing and drawing. *Human Brain Mapping*, 28(5), 450-459.

Harris, T. L. (1960). Handwriting. *Encyclopedia of educational research*, 616-24.

Hart, H., Radua, J., Nakao, T., Mataix-Cols, D., & Rubia, K. (2013). Meta-analysis of functional magnetic resonance imaging studies of inhibition and attention in attentiondeficit/hyperactivity disorder: exploring task-specific, stimulant medication, and age effects.
JAMA psychiatry, 70(2), 185-198.

Harvey, E. A., Youngwirth, S. D., Thakar, D. A., & Errazuriz, P. A. (2009). Predicting attention-deficit/hyperactivity disorder and oppositional defiant disorder from preschool diagnostic assessments. *Journal of Consulting and Clinical Psychology*, 77(2), 349.

Harvey, W. J., & Reid, G. (1997). Motor performance of children with attention-deficit hyperactivity disorder: A preliminary investigation. Adapted physical activity quarterly, 14(3), 189-202.

Harvey, W. J., & Reid, G. (2005). Attention-Deficit/Hyperactivity Disorder: APA Research Challenges. *Adapted Physical Activity Quarterly*, 22(1).

Hazy, T. E., Frank, M. J., & O'reilly, R. C. (2007). Towards an executive without a homunculus: computational models of the prefrontal cortex/basal ganglia system. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 362(1485), 1601-1613.

Hebb, D. O. (1949). The first stage of perception: growth of the assembly. *The Organization of Behavior*, 4, 60-78.

Heidbreder, R. (2015). ADHD symptomatology is best conceptualized as a spectrum: a dimensional versus unitary approach to diagnosis. *ADHD Attention Deficit and Hyperactivity Disorders*, 7(4), 249-269.

Helie, S., Chakravarthy, S., & Moustafa, A. A. (2013). Exploring the cognitive and motor functions of the basal ganglia: an integrative review of computational cognitive neuroscience models. *Frontiers in computational neuroscience*, 7, 174.

Helwig, J. J., Johns, J. C., Norman, J. E., & Cooper, J. O. (1976). The measurement of manuscript letter strokes. *Journal of Applied Behavior Analysis*, 9(2), 231.

Herbert, M. (1964). The concept and testing of brain-damage in children: a review. *Journal of Child Psychology and Psychiatry*, 5(3-4), 197-216.

Herrick, V. E. (1963). New Horizons for Research in Handwriting. Report of the Invitational

Conference on Research in Handwriting (University of Wisconsin, October 1961).

Ho, T. P., Leung, P. W., Luk, E. S., Taylor, E., Bacon-Shone, J., & Mak, F. L. (1996). Establishing the constructs of childhood behavioral disturbances in a Chinese population: A questionnaire study. *Journal of Abnormal Child Psychology*, 24(4), 417-431.

Hochberg, J., & Brooks, V. (1978). The Perception of Motion Pictures. In (EC Carterette and MP Friedman, eds.) Perceptual Ecology: Handbook of Perception, vol. X.

Hu, S., Ide, J. S., Zhang, S., & Chiang-shan, R. L. (2016). The right superior frontal gyrus and individual variation in proactive control of impulsive response. *Journal of Neuroscience*, 36(50), 1268812696.

Hughes, H. C., & Zimba, L. D. (1985). Spatial maps of directed visual attention. *Journal of Experimental Psychology: Human Perception and Performance*, 11(4), 409.

Hult, N., Kadesjö, J., Kadesjö, B., Gillberg, C., & Billstedt, E. (2018). ADHD and the QbTest: diagnostic validity of QbTest. *Journal of attention disorders*, 22(11), 1074-1080.

Humphries, M. D., Stewart, R. D., & Gurney, K. N. (2006). A physiologically plausible model of action selection and oscillatory activity in the basal ganglia. *Journal of Neuroscience*, 26(50), 1292112942.

Hung, C. L., Chang, Y. K., Chan, Y. S., Shih, C. H., Huang, C. J., & Hung, T. M. (2013). Motor ability and inhibitory processes in children with ADHD: A neuroelectric study. *Journal of Sport and Exercise Psychology*, 35(3), 322-328.

Hyde, C., Fuelscher, I., Sciberras, E., Efron, D., Anderson, V. A., & Silk, T. (2021). Understanding motor

difficulties in children with ADHD: A fixel-based analysis of the corticospinal tract. *Progress in*

Neuro-Psychopharmacology and Biological Psychiatry, 105, 110125.

Iaboni, F., Douglas, V. I., & Ditto, B. (1997). Psychophysiological response of ADHD children to reward

and extinction. *Psychophysiology*, 34(1), 116-123.

Ivnik, R. J., Malec, J. F., Smith, G. E., Tangalos, E. G., & Petersen, R. C. (1996). Neuropsychological

tests' norms above age 55: COWAT, BNT, MAE token, WRAT-R reading, AMNART, STROOP, TMT, and JLO. *The Clinical Neuropsychologist*, 10(3), 262-278.

James, W. (1890). The perception of reality. Principles of psychology, 2, 283-324.

Jensen, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in ADHD: Implications for research,

practice, and DSM-V. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(8), 1065-1079.

Johnson, K. A., Robertson, I. H., Barry, E., Mulligan, A., Dáibhis, A., Daly, M., ... & Bellgrove, M. A.

(2008). Impaired conflict resolution and alerting in children with ADHD: evidence from the Attention Network Task (ANT). *Journal of Child Psychology and Psychiatry*, 49(12), 13391347.

Johnson, K. A., Wiersema, J. R., & Kuntsi, J. (2009). What would Karl Popper say? Are current

psychological theories of ADHD falsifiable?. *Behavioral and Brain Functions*, 5(1), 1-11.

Jones, H. E., & Seashore, R. H. (1944). The development of fine motor and mechanical abilities.

Teachers College Record, 45(9), 123-145.

Jonides, J. (1981). Voluntary versus automatic control over the mind's eye's movement. *Attention*

and performance, 187-203.

Joormann, J., Yoon, K. L., & Zetsche, U. (2007). Cognitive inhibition in depression. *Applied and preventive psychology*, 12(3), 128-139.

Kahneman, D. (1973). *Attention and effort* (Vol. 1063, pp. 218-226). Englewood Cliffs, NJ: Prentice-Hall.

Kaji, R. (2001). Basal ganglia as a sensory gating devise for motor control. *Journal of Medical Investigation*, 48(3/4), 142-146.

Kalff, A. C., De Sonneville, L. M., Hurks, P. P., Hendriksen, J. G., Kroes, M., Feron, F. J., ... & Jolles, J. (2003). Low-and high-level controlled processing in executive motor control tasks in 5–6year-old children at risk of ADHD. *Journal of Child Psychology and Psychiatry*, 44(7), 10491057.

Kalff, A. C., De Sonneville, L. M., Hurks, P. P., Hendriksen, J. G., Kroes, M., Feron, F. J., ... & Jolles, J. (2005). Speed, speed variability, and accuracy of information processing in 5 to 6-year-old children at risk of ADHD. *Journal of the International Neuropsychological Society*, 11(2), 173183.

Kaplan, B. J., Wilson, B. N., Dewey, D., & Crawford, S. G. (1998). DCD may not be a discrete disorder. *Human movement science*, 17(4-5), 471-490.

Katanoda, K., Yoshikawa, K., & Sugishita, M. (2001). A functional MRI study on the neural substrates for writing. *Human brain mapping*, 13(1), 34-42.

Keele, S. W. (1968). Movement control in skilled motor performance. *Psychological bulletin*, 70(6p1), 387.

Kelso, J. S., Tuller, B., Vatikiotis-Bateson, E., & Fowler, C. A. (1984). Functionally specific articulatory cooperation following jaw perturbations during speech: evidence for coordinative structures. *Journal of Experimental Psychology: Human Perception and Performance*, 10(6), 812.

Kemp, J. M., & Powell, T. P. S. (1971). The termination of fibres from the cerebral cortex and thalamus upon dendritic spines in the caudate nucleus: a study with the Golgi method. *Philosophical Transactions of the Royal Society of London. B, Biological Sciences,* 262(845), 429-439.

Kennedy, J., Brown, T., & Chien, C. W. (2012). Motor skill assessment of children: is there an association between performance-based, child-report, and parent-report measures of children's motor skills?. *Physical & Occupational Therapy in Pediatrics*, 32(2), 196-209.

Kennett, S., & Driver, J. (2014). Within-hemifield posture changes affect tactile–visual exogenous spatial cueing without spatial precision, especially in the dark. *Attention, Perception, & Psychophysics*, 76(4), 1121-1135.

Kerns, J. G. (2006). Anterior cingulate and prefrontal cortex activity in an FMRI study of trial-to-trial adjustments on the Simon task. *Neuroimage*, 33(1), 399-405.

Kessler, J. W. (1980). History of minimal brain dysfunctions. Handbook of minimal brain dysfunctions: A critical view, 18-52.

Key stage 2 attainment: National headlines, Methodology. (2022, July 5). https://explore-education-statistics.service.gov.uk/methodology/key-stage-2-attainment-national-headlines

Kielhofner, G. (2009). *Model of human occupation* (Vol. 1009). Baltimore.

Kirkham, N. Z., & Diamond, A. (2003). Sorting between theories of perseveration: performance in conflict tasks requires memory, attention and inhibition. *Developmental Science*, 6(5), 474476.

Kirkham, N. Z., Cruess, L., & Diamond, A. (2003). Helping children apply their knowledge to their behavior on a dimension-switching task. *Developmental science*, 6(5), 449-467.

Klassen, A. F., Miller, A., & Fine, S. (2004). Health-related quality of life in children and adolescents who have a diagnosis of attention-deficit/hyperactivity disorder. *Pediatrics*, 114(5), e541e547.

Klein, R. M. (2000). Inhibition of return. *Trends in cognitive sciences*, 4(4), 138-147.

Kleinbaum, D. G., & Klein, M. (2012). Survival analysis: a self-learning text (Vol. 3). New York: Springer.

Knobloch, H., & Pasamanick, B. (1959). Syndrome of minimal cerebral damage in infancy. Journal of the American Medical Association, 170(12), 1384-1387.

Koch, K. W., & Fuster, J. M. (1989). Unit activity in monkey parietal cortex related to haptic perception and temporary memory. Experimental Brain Research, 76(2), 292-306.

Konrad, A., Dielentheis, T. F., El Masri, D., Bayerl, M., Fehr, C., Gesierich, T., ... & Winterer, G. (2010).

Disturbed structural connectivity is related to inattention and impulsivity in adult attention deficit hyperactivity disorder. *European Journal of Neuroscience*, 31(5), 912-919.

Kooij, S. J., Bejerot, S., Blackwell, A., Caci, H., Casas-Brugué, M., Carpentier, P. J., ... & Asherson, P. (2010). European consensus statement on diagnosis and treatment of adult ADHD: The European Network Adult ADHD. *BMC psychiatry*, 10(1), 1-24.

Koyama, M. S., O'Connor, D., Shehzad, Z., & Milham, M. P. (2017). Differential contributions of the middle frontal gyrus functional connectivity to literacy and numeracy. *Scientific reports*, 7(1), 1-13.

Krauel, K., Feldhaus, H. C., Simon, A., Rehe, C., Glaser, M., Flechtner, H. H., ... & Niehaus, L. (2010). Increased echogenicity of the substantia nigra in children and adolescents with attentiondeficit/hyperactivity disorder. *Biological psychiatry*, 68(4), 352-358.

Kugler, P. N., Kelso, J. S., & Turvey, M. T. (1980). 1 on the concept of coordinative structures as dissipative structures: I. theoretical lines of convergence. In *Advances in psychology* (Vol. 1, pp. 3-47). North-Holland.

Kumar, U., Arya, A., & Agarwal, V. (2021). Neural network connectivity in ADHD children: an independent component and functional connectivity analysis of resting state fMRI data. *Brain imaging and behavior*, 15(1), 157-165.

Kumar, U., Arya, A., & Agarwal, V. (2022). Altered functional connectivity in children with ADHD while performing cognitive control task. *Psychiatry Research: Neuroimaging*, 326, 111531.

Lanciego, J. L., Luquin, N., & Obeso, J. A. (2012). Functional neuroanatomy of the basal ganglia. *Cold Spring Harbor perspectives in medicine*, 2(12), a009621.

Landy, D., Allen, C., & Zednik, C. (2014). A perceptual account of symbolic reasoning. *Frontiers in psychology*, 5, 275.

Lange, K. W., Reichl, S., Lange, K. M., Tucha, L., & Tucha, O. (2010). The history of attention deficit hyperactivity disorder. *ADHD Attention Deficit and Hyperactivity Disorders*, 2(4), 241-255.

Langmaid, R. A., Papadopoulos, N., Johnson, B. P., Phillips, J. G., & Rinehart, N. J. (2014). Handwriting in children with ADHD. *Journal of attention disorders*, 18(6), 504-510.

Laufer, M. W., & Denhoff, E. (1957). Hyperkinetic behavior syndrome in children. *The Journal of pediatrics*, 50(4), 463-474.

Leisman, G., Braun-Benjamin, O., & Melillo, R. (2014). Cognitive-motor interactions of the basal ganglia in development. *Frontiers in systems neuroscience*, 8, 16.

Lenroot, R. K., Gogtay, N., Greenstein, D. K., Wells, E. M., Wallace, G. L., Clasen, L. S., ... & Giedd, J. N. (2007). Sexual dimorphism of brain developmental trajectories during childhood and adolescence. *Neuroimage*, 36(4), 1065-1073.

Lévesque, J. C., & Parent, A. (2005). GABAergic interneurons in human subthalamic nucleus. *Movement disorders: official journal of the Movement Disorder Society*, 20(5), 574-584.

Levy, B. J., & Anderson, M. C. (2008). Individual differences in the suppression of unwanted memories: The executive deficit hypothesis. *Acta psychologica*, 127(3), 623-635.

Litvan, I., Paulsen, J. S., Mega, M. S., & Cummings, J. L. (1998). Neuropsychiatric assessment of patients with hyperkinetic and hypokinetic movement disorders. *Archives of Neurology*, 55(10), 1313-1319.

Loe, I. M., & Feldman, H. M. (2007). Academic and educational outcomes of children with ADHD.

Journal of pediatric psychology, 32(6), 643-654.

Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological review*, 91(3), 295.

Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological science*, 8(1), 60-64.

Logan, G. D., Van Zandt, T., Verbruggen, F., & Wagenmakers, E. J. (2014). On the ability to inhibit thought and action: general and special theories of an act of control. *Psychological review*, 121(1), 66.

Luengo, M. A., Carrillo-De-La-Pena, M. T., & Otero, J. M. (1991). The components of impulsiveness: A comparison of the I. 7 Impulsiveness Questionnaire and the Barratt Impulsiveness Scale. *Personality and Individual Differences*, 12(7), 657-667.

Lupiáñez, J., Klein, R. M., & Bartolomeo, P. (2006). Inhibition of return: Twenty years after. *Cognitive neuropsychology*, 23(7), 1003-1014.

MacKay, D. G. (1987). Self-Inhibition and the Recovery Cycle. In *The Organization of Perception and Action* (pp. 141-164). Springer, New York, NY.

MacLeod, C. M., & Dunbar, K. (1988). Training and Stroop-like interference: evidence for a continuum of automaticity. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 14(1), 126.

Magnus, R. (1925). Croonian Lecture. —Animal posture. *Proceedings of the Royal Society of London. Series B, Containing Papers of a Biological Character, 98*(690), 339-353.

Makeig, S., Gramann, K., Jung, T. P., Sejnowski, T. J., & Poizner, H. (2009). Linking brain, mind and behavior. *International Journal of Psychophysiology*, 73(2), 95-100.

Maljkovic, V., & Nakayama, K. (2000). Priming of popout: III. A short-term implicit memory system beneficial for rapid target selection. *Visual cognition*, 7(5), 571-595.

Maljkovic, V., & Nakayama, K. E. N. (1996). Priming of pop-out: II. The role of position. *Perception & psychophysics*, 58(7), 977-991.

Malloy-Diniz, L. F., Paula, J. J. D., Vasconcelos, A. G., Almondes, K. M. D., Pessoa, R., Faria, L., ... & Mattos, P. (2015). Normative data of the Barratt Impulsiveness Scale 11 (BIS-11) for Brazilian adults. *Brazilian Journal of Psychiatry*, 37, 245-248.

Mandelbrot, B. (1967). How long is the coast of Britain? Statistical self-similarity and fractional dimension. *science*, 156(3775), 636-638.

Marco, R., Miranda, A., Schlotz, W., Melia, A., Mulligan, A., Müller, U., ... & Sonuga-Barke, E. J. (2009). Delay and reward choice in ADHD: an experimental test of the role of delay aversion. *Neuropsychology*, 23(3), 367.

Maser, J. D., & Akiskal, H. S. (2002). Spectrum concepts in major mental disorders. *Psychiatric Clinics*, 25(4), xi-xiii.

Mason, P. (2017). Medical neurobiology. Oxford University Press.

McBride, J., Boy, F., Husain, M., & Sumner, P. (2012). Automatic motor activation in the executive control of action. Frontiers in human neuroscience, 6, 82.

McCulloch, W. S., & Pitts, W. (1943). A logical calculus of the ideas immanent in nervous activity. *The*

bulletin of mathematical biophysics, 5(4), 115-133.

McLoughlin, G., Ronald, A., Kuntsi, J., Asherson, P., & Plomin, R. (2007). Genetic support for the dual nature of attention deficit hyperactivity disorder: Substantial genetic overlap between the inattentive and hyperactive–impulsive components. *Journal of abnormal child psychology*, 35(6), 999-1008.

McNab, F., & Klingberg, T. (2008). Prefrontal cortex and basal ganglia control access to working memory. *Nature neuroscience*, 11(1), 103-107.

McNab, F., Leroux, G., Strand, F., Thorell, L., Bergman, S., & Klingberg, T. (2008). Common and unique components of inhibition and working memory: an fMRI, within-subjects investigation. *Neuropsychologia*, 46(11), 2668-2682.

Meda, S. A., Stevens, M. C., Potenza, M. N., Pittman, B., Gueorguieva, R., Andrews, M. M., ... & Pearlson, G. D. (2009). Investigating the behavioral and self-report constructs of impulsivity domains using principal component analysis. *Behavioural pharmacology*, 20(5-6), 390.

Mehler-Wex, C., Riederer, P., & Gerlach, M. (2006). Dopaminergic dysbalance in distinct basal ganglia neurocircuits: implications for the pathophysiology of Parkinson's disease, schizophrenia and attention deficit hyperactivity disorder. *Neurotoxicity research*, 10(3), 167-179.

Messer, C. (2011). Examining the influence of the dopamine D2 agonist, cabergoline, on measures of response inhibition and error awareness.

Meyer, A., & Sagvolden, T. (2006). Fine motor skills in South African children with symptoms of ADHD: influence of subtype, gender, age, and hand dominance. *Behavioral and Brain Functions*, 2(1), 1-13.

Mitchell, K., Simpson, H., & Yuen, H. (2020). Handwriting Deficits and Tourette's Syndrome (TS): Is There a Connection?. *AJOT: American Journal of Occupational Therapy*, 74(S1), NA-NA.

Miyahara, M., & Möbs, I. (1995). Developmental dyspraxia and developmental coordination disorder. *Neuropsychology review*, 5(4), 245-268.

Miyahara, M., Piek, J., & Barrett, N. (2006). Accuracy of drawing in a dual-task and resistance-to-distraction study: Motor or attention deficit?. *Human Movement Science*, 25(1), 100-109.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive psychology*, 41(1), 49-100.

Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American journal of psychiatry*, 158(11), 1783-1793.

Molina, B. S., Hinshaw, S. P., Swanson, J. M., Arnold, L. E., Vitiello, B., Jensen, P. S., ... & MTA Cooperative Group. (2009). The MTA at 8 years: prospective follow-up of children treated for combined-type ADHD in a multisite study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(5), 484-500.

Monsell, S., & Driver, J. (Eds.). (2000). *Control of cognitive processes: Attention and performance XVIII* (Vol. 18). MIT Press.

Mostofsky, S. H., Dubey, P., Jerath, V. K., Jansiewicz, E. M., Goldberg, M. C., & Denckla, M. B. (2006).

Developmental dyspraxia is not limited to imitation in children with autism spectrum disorders. *Journal of the International Neuropsychological Society*, 12(3), 314-326.

Munoz, D. P., & Everling, S. (2004). Look away: the anti-saccade task and the voluntary control of eye

movement. *Nature Reviews Neuroscience*, 5(3), 218-228.

Muraven, M. (2010). Building self-control strength: Practicing self-control leads to improved self-

control performance. *Journal of experimental social psychology*, 46(2), 465-468.

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does s

elf-control resemble a muscle?. *Psychological bulletin*, 126(2), 247.

Murphy, H. B. M., Rosenthal, D., & Kety, S. S. (1968). Cultural factors in the genesis of schizophrenia.

Nagel, B. J., Bathula, D., Herting, M., Schmitt, C., Kroenke, C. D., Fair, D., & Nigg, J. T. (2011). Altered

white matter microstructure in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 50(3), 283-292.

Nakamura, K., Honda, M., Okada, T., Hanakawa, T., Toma, K., Fukuyama, H., ... & Shibasaki, H.

(2000). Participation of the left posterior inferior temporal cortex in writing and mental recall of kanji orthography: A functional MRI study. *Brain*, 123(5), 954-967.

Nakamura, K., Kuo, W. J., Pegado, F., Cohen, L., Tzeng, O. J., & Dehaene, S. (2012). Universal brain

systems for recognizing word shapes and handwriting gestures during reading. *Proceedings of the National Academy of Sciences, 109*(50), 20762-20767.

Nakao, T., Radua, J., Rubia, K., & Mataix-Cols, D. (2011). Gray matter volume abnormalities in ADHD:

voxel-based meta-analysis exploring the effects of age and stimulant medication. American *Journal of Psychiatry*, 168(11), 1154-1163.

Nestor, P. G., Akdag, S. J., O'Donnell, B. F., Niznikiewicz, M., Law, S., Shenton, M. E., & McCarley, R. W. (1998). Word recall in schizophrenia: a connectionist model. *American Journal of Psychiatry, 155*(12), 1685-1690.

Nichols, S. L., & Waschbusch, D. A. (2004). A review of the validity of laboratory cognitive tasks used to assess symptoms of ADHD. *Child Psychiatry and Human Development*, 34(4), 297-315.

Nicolson, R. I., & Fawcett, A. J. (2011). Dyslexia, dysgraphia, procedural learning and the cerebellum. *Cortex*, 47(1), 117-127.

Nietzel, M. T., Bernstein, D. A., & Milich, R. (1994). Introduction to clinical psychology. Prentice-Hall, Inc.

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological bulletin*, 126(2), 220.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder?. *Psychological bulletin, 127*(5), 571.

Nigg, J. T. (2012). Future directions in ADHD etiology research. *Journal of clinical child & adolescent psychology, 41*(4), 524-533.

Nigg, J. T., Butler, K. M., Huang-Pollock, C. L., & Henderson, J. M. (2002). Inhibitory processes in adults with persistent childhood onset ADHD. *Journal of consulting and clinical psychology, 70*(1), 153.

Ninio, A., & Lieblich, A. (1976). The grammar of action:" phrase structure" in children's copying. *Child Development*, 846-850.

Niv, Y., Duff, M. O., & Dayan, P. (2005). Dopamine, uncertainty and TD learning. *Behavioral and brain*

Functions, 1(1), 1-9.

Noreen, S., & MacLeod, M. D. (2015). What do we really know about cognitive inhibition? Task

demands and inhibitory effects across a range of memory and behavioural tasks. *PLoS One, 10*(8), e0134951.

Norman, D. A., & Bobrow, D. G. (1975). On data-limited and resource-limited processes. *Cognitive*

psychology, 7(1), 44-64.

Oldehinkel, M., Beckmann, C. F., Pruim, R. H., Van Oort, E. S., Franke, B., Hartman, C. A., ... &

Mennes, M. (2016). Attention-deficit/hyperactivity disorder symptoms coincide with altered striatal connectivity. *Biological psychiatry: cognitive neuroscience and neuroimaging, 1*(4), 353-363.

Oliva, F., di Girolamo, G., Malandrone, F., Iaia, N., Biasi, F., & Maina, G. (2020). Early childhood

infections, antistreptococcal and basal ganglia antibodies in adult ADHD: a preliminary study.

BMC psychiatry, 20(1), 1-6.

Omrani, M., Kaufman, M. T., Hatsopoulos, N. G., & Cheney, P. D. (2017). Perspectives on classical

controversies about the motor cortex. *Journal of neurophysiology*, 118(3), 1828-1848.

Omura, K., Tsukamoto, T., Kotani, Y., Ohgami, Y., & Yoshikawa, K. (2004). Neural correlates of

phoneme-to-grapheme conversion. *Neuroreport, 15*(6), 949-953.

Ordaz, S. J., Foran, W., Velanova, K., & Luna, B. (2013). Longitudinal growth curves of brain function underlying inhibitory control through adolescence. *Journal of Neuroscience, 33*(46), 1810918124.

O'Reilly, R. C., & Frank, M. J. (2006). Making working memory work: a computational model of learning in the prefrontal cortex and basal ganglia. *Neural computation, 18*(2), 283-328.

Ozonoff, S., Young, G. S., Goldring, S., Greiss-Hess, L., Herrera, A. M., Steele, J., ... & Rogers, S. J. (2008). Gross motor development, movement abnormalities, and early identification of autism. *Journal of autism and developmental disorders, 38*(4), 644-656.

Paloyelis, Y., Mehta, M. A., Kuntsi, J., & Asherson, P. (2007). Functional MRI in ADHD: a systematic literature review. *Expert review of neurotherapeutics, 7(*10), 1337-1356.

Paolo Busardò, F., Kyriakou, C., Cipolloni, L., Zaami, S., & Frati, P. (2016). From clinical application to cognitive enhancement: the example of methylphenidate. *Current neuropharmacology*, 14(1), 17-27.

Parent, A., & Hazrati, L. N. (1995). Functional anatomy of the basal ganglia. I. The cortico-basal ganglia-thalamo-cortical loop. *Brain research reviews*, *20*(1), 91-127.

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. *Journal of clinical psychology*, *51*(6), 768-774.

Pelham, W. E., McBurnett, K., Harper, G. W., Milich, R., Murphy, D. A., Clinton, J., & Thiele, C. (1990). Methylphenidate and baseball playing in ADHD children: who's on first?. *Journal of consulting and clinical psychology, 58*(1), 130.

Perry, N. E. (1998). Young children's self-regulated learning and contexts that support it. *Journal of*

educational psychology, 90(4), 715.

Petrides, M. (1995). Impairments on nonspatial self-ordered and externally ordered working memory tasks after lesions of the mid-dorsal part of the lateral frontal cortex in the monkey. *Journal of Neuroscience, 15*(1), 359-375.

Pfefferbaum, A., Mathalon, D. H., Sullivan, E. V., Rawles, J. M., Zipursky, R. B., & Lim, K. O. (1994). A quantitative magnetic resonance imaging study of changes in brain morphology from infancy to late adulthood. *Archives of neurology, 51*(9), 874-887.

Phelps, J., Stempel, L., & Speck, G. (1985). The children's handwriting scale: A new diagnostic tool. *The Journal of Educational Research, 79*(1), 46-50.

Phillips, J. G., Stelmach, G. E., & Teasdale, N. (1991). What can indices of handwriting quality tell us about Parkinsonian handwriting?. *Human Movement Science, 10*(2-3), 301-314.

Piek, J. P., Pitcher, T. M., & Hay, D. A. (1999). Motor coordination and kinaesthesis in boys with attention deficit–hyperactivity disorder. *Developmental Medicine and Child Neurology, 41*(3), 159-165.

Pierce, G. R., Sarason, B. R., & Sarason, I. G. (Eds.). (1996). *Handbook of social support and the family*. Springer Science & Business Media.

Pievsky, M. A., & McGrath, R. E. (2018). The neurocognitive profile of attention-deficit/hyperactivity disorder: A review of meta-analyses. *Archives of Clinical Neuropsychology*, 33(2), 143-157.

Pillsbury, W. B. (1908). On meaning: A symposium before the Western Philosophical Association, December, 1907. *Psychological review*, 15(3), 150.

Pinto, Y., van der Leij, A. R., Sligte, I. G., Lamme, V. A., & Scholte, H. S. (2013). Bottom-up and top-

down attention are independent. *Journal of vision, 13*(3), 16-16.

Pitcher, T. M., Piek, J. P., & Hay, D. A. (2003). Fine and gross motor ability in males with ADHD. *Developmental medicine and child neurology, 45*(8), 525-535.

Pizzarelli, R., & Cherubini, E. (2011). Alterations of GABAergic signaling in autism spectrum disorders. *Neural plasticity*, 2011.

Plamondon, R., O'Reilly, C., Rémi, C., & Duval, T. (2013). The lognormal handwriter: learning, performing, and declining. *Frontiers in psychology*, 4, 945.

Planton, S., Jucla, M., Roux, F. E., & Démonet, J. F. (2013). The "handwriting brain": a meta-analysis of neuroimaging studies of motor versus orthographic processes. *Cortex, 49*(10), 2772-2787.

Polanczyk, G. V., Willcutt, E. G., Salum, G. A., Kieling, C., & Rohde, L. A. (2014). ADHD prevalence estimates across three decades: an updated systematic review and meta-regression analysis. *International journal of epidemiology, 43*(2), 434-442.

Polanczyk, G., De Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: a systematic review and metaregression analysis. *American journal of psychiatry, 164*(6), 942-948.

Popper, K. (1934). Scientific method. *Popper selections*, *133*, 142.

Posner, J., Maia, T. V., Fair, D., Peterson, B. S., Sonuga-Barke, E. J., & Nagel, B. J. (2011). The attenuation of dysfunctional emotional processing with stimulant medication: an fMRI study of adolescents with ADHD. *Psychiatry Research: Neuroimaging, 193*(3), 151-160.

Posner, M. I. (1990). Hierarchical distributed networks in the neuropsychology of selective attention.

Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. *Attention and performance X: Control of language processes,* 32, 531-556.

Posner, M. I., & DiGirolamo, G. J. (1998). 18 Executive Attention: Conflict, Target Detection, and Cognitive Control.

Posner, M. I., Rafal, R. D., Choate, L. S., & Vaughan, J. (1985). Inhibition of return: Neural basis and function. *Cognitive neuropsychology*, 2(3), 211-228.

Postle, B. R., Berger, J. S., Goldstein, J. H., Curtis, C. E., & D'Esposito, M. (2001). Behavioral and neurophysiological correlates of episodic coding, proactive interference, and list length effects in a running span verbal working memory task. *Cognitive, Affective, & Behavioral Neuroscience, 1*(1), 10-21.

Purves, D., Augustine, G. J., Fitzpatrick, D., Katz, L. C., LaMantia, A. S., McNamara, J. O., & Williams, S. M. (2001). Circuits within the basal ganglia system. In *Neuroscience. 2nd edition*. Sinauer Associates.

Purves, D., Beau Lotto, R., Mark Williams, S., Nundy, S., & Yang, Z. (2001). Why we see things the way we do: evidence for a wholly empirical strategy of vision. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 356*(1407), 285-297.

Pyrgaki, C., Trainor, P., Hadjantonakis, A. K., & Niswander, L. (2010). Dynamic imaging of mammalian neural tube closure. *Developmental biology*, 344(2), 941-947.

Qiu, A., Crocetti, D., Adler, M., Mahone, E. M., Denckla, M. B., Miller, M. I., & Mostofsky, S. H. (2009). Basal ganglia volume and shape in children with attention deficit hyperactivity disorder.

American Journal of Psychiatry, 166(1), 74-82.

Rachlin, H. (2000). *The science of self-control.* Harvard University Press.

Rapin, I. (1964). Evoked responses to clicks in a group of children with communication disorders. *Annals of the New York Academy of Sciences*.

Rapport, M. D., Orban, S. A., Kofler, M. J., & Friedman, L. M. (2013). Do programs designed to train working memory, other executive functions, and attention benefit children with ADHD? A meta-analytic review of cognitive, academic, and behavioral outcomes. *Clinical psychology review, 33*(8), 1237-1252.

Redgrave, P., Prescott, T. J., & Gurney, K. (1999). The basal ganglia: a vertebrate solution to the selection problem?. *Neuroscience, 89*(4), 1009-1023.

Reisman, J. E. (1993). Development and reliability of the research version of the Minnesota Handwriting Test. *Physical & Occupational Therapy in Pediatrics, 13*(2), 41-55.

Rémi, C., Amara, M., Courtellement, P., & de Brucq, D. (1997). An experimental program for the automatic study of handwriting learning at elementary school. In *Proc. Eighth Biennial Conference of the International Graphonomics Society*.

Richards, J. E., & Cronise, K. (2000). Extended visual fixation in the early preschool years: Look duration, heart rate changes, and attentional inertia. *Child Development, 71*(3), 602-620.

Richards, J. E., & Gibson, T. L. (1997). Extended visual fixation in young infants: Look distributions, heart rate changes, and attention. *Child Development*, 1041-1056.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial

frontal cortex in cognitive control. *science*, 306(5695), 443-447.

Rie, H. E., & Rie, E. D. (Eds.). (1980). *Handbook of minimal brain dysfunctions: A critical view*. John Wiley & Sons.

Robison, L. S., Ananth, M., Hadjiargyrou, M., Komatsu, D. E., & Thanos, P. K. (2017). Chronic oral methylphenidate treatment reversibly increases striatal dopamine transporter and dopamine type 1 receptor binding in rats. *Journal of neural transmission*, 124(5), 655-667.

Rodriguez-Jimenez, R., Avila, C., Jimenez-Arriero, M. A., Ponce, G., Monasor, R., Jimenez, M. E. E. A., ... & Palomo, T. (2006). Impulsivity and sustained attention in pathological gamblers: influence of childhood ADHD history. *Journal of Gambling Studies*, 22(4), 451-461.

Roeltgen, D. P. (2003). Agraphia. In K. M. Heilman and E. Valenstein (eds.), *Clinical Neuropsychology, 4th edn*. (pp. 126–145). New York: Oxford University Press

Rommelse, N. N., Altink, M. E., De Sonneville, L. M., Buschgens, C. J., Buitelaar, J., Oosterlaan, J., & Sergeant, J. A. (2007). Are motor inhibition and cognitive flexibility dead ends in ADHD?. *Journal of abnormal child psychology, 35*(6), 957-967.

Roodenrys, S., Koloski, N., & Grainger, J. (2001). Working memory function in attention deficit hyperactivity disordered and reading disabled children. *British Journal of Developmental Psychology, 19*(3), 325-337.

Ross DM, Ross SA. *Hyperactivity: research, theory and action*. New York: Wiley; 1976

Roux, F. E., Minkin, K., Durand, J. B., Sacko, O., Réhault, E., Tanova, R., & Démonet, J. F. (2015). Electrostimulation mapping of comprehension of auditory and visual words. *Cortex*, 71, 398408.

Rubia, K., Halari, R., Cubillo, A., Smith, A. B., Mohammad, A. M., Brammer, M., & Taylor, E. (2011). Methylphenidate normalizes fronto-striatal underactivation during interference inhibition in medication-naive boys with attention-deficit hyperactivity disorder. *Neuropsychopharmacology, 36*(8), 1575-1586.

Rubia, K., Smith, A. B., Brammer, M. J., & Taylor, E. (2003). Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *Neuroimage, 20*(1), 351-358.

Rubia, K., Smith, A. B., Taylor, E., & Brammer, M. (2007). Linear age-correlated functional development of right inferior fronto-striato-cerebellar networks during response inhibition and anterior cingulate during error-related processes. *Human brain mapping, 28*(11), 11631177.

Rubia, K., Smith, A., & Taylor, E. (2007). Performance of children with attention deficit hyperactivity disorder (ADHD) on a test battery of impulsiveness. *Child Neuropsychology, 13*(3), 276-304.

Rubin, N., & Henderson, S. E. (1982). Two sides of the same coin: variations in teaching methods and failure to learn to write. *Special Education: Forward Trends, 9*(4), 17-24.

Ruiz-Goikoetxea, M., Cortese, S., Aznarez-Sanado, M., Magallón, S., Zallo, N. A., Luis, E. O., ... & Arrondo, G. (2018). Risk of unintentional injuries in children and adolescents with ADHD and the impact of ADHD medications: a systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 84, 63-71.

Sagvolden, T., Aase, H., Zeiner, P., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. *Behavioural brain research, 94*(1), 61-71.

Sagvolden, T., Johansen, E. B., Aase, H., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences, 28*(3), 397-418.

Sagvolden, T., Johansen, E. B., Wøien, G., Walaas, S. I., Storm-Mathisen, J., Bergersen, L. H., ... & Faraone, S. V. (2009). The spontaneously hypertensive rat model of ADHD–the importance of selecting the appropriate reference strain. *Neuropharmacology, 57*(7-8), 619-626.

Sanders, A. F. (1983). Towards a model of stress and human performance. *Acta psychologica*, 53(1), 61-97.

Santangelo, T., & Graham, S. (2016). A comprehensive meta-analysis of handwriting instruction. *Educational Psychology Review, 28*(2), 225-265.

Sassoon, R. (1990). *Handwriting: A new perspective.* Stanley Thornes.

Schachar, R., Logan, G. D., Robaey, P., Chen, S., Ickowicz, A., & Barr, C. (2007). Restraint and cancellation: multiple inhibition deficits in attention deficit hyperactivity disorder. *Journal of abnormal child psychology, 35*(2), 229-238.

Schaltenbrand, G. (1928). The development of human motility and motor disturbances. *Archives of Neurology & Psychiatry, 20*(4), 720-730.

Scheiffele, P., Fan, J., Choih, J., Fetter, R., & Serafini, T. (2000). Neuroligin expressed in nonneuronal cells triggers presynaptic development in contacting axons. *Cell, 101*(6), 657-669.

Schmidt, R. A. (1976). The schema as a solution to some persistent problems in motor learning theory. In *Motor control* (pp. 41-65). Academic Press.

Schnoes, C., Reid, R., Wagner, M., & Marder, C. (2006). ADHD among students receiving special education services: A national survey. *Exceptional Children, 72*(4), 483-496.

Schoemaker, M. M., Ketelaars, C. E., Van Zonneveld, M., Minderaa, R. B., & PhD, T. M. (2005). Deficits in motor control processes involved in production of graphic movements of children with attention-deficit-hyperactivity disorder. *Developmental Medicine & Child Neurology, 47*(6), 390-395.

Schultz, W., Apicella, P., & Ljungberg, T. (1993). Responses of monkey dopamine neurons to reward and conditioned stimuli during successive steps of learning a delayed response task. *Journal of neuroscience, 13*(3), 900-913.

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, 275(5306), 1593-1599.

Seger, C. A. (2008). How do the basal ganglia contribute to categorization? Their roles in generalization, response selection, and learning via feedback. Neuroscience & Biobehavioral *Reviews, 32*(2), 265-278.

Seger, C. A., & Spiering, B. J. (2011). A critical review of habit learning and the basal ganglia. *Frontiers in systems neuroscience, 5*, 66.

Sergeant, J. A. (2005). Modeling attention-deficit/hyperactivity disorder: a critical appraisal of the cognitive-energetic model. *Biological psychiatry, 57*(11), 1248-1255.

Sergeant, J. A., Geurts, H., Huijbregts, S., Scheres, A., & Oosterlaan, J. (2003). The top and the bottom of ADHD: a neuropsychological perspective. *Neuroscience & Biobehavioral Reviews, 27*(7), 583-592.

Sergeant, J. A., Oosterlaan, J., & van der Meere, J. (1999). Information processing and energetic factors in attention-deficit/hyperactivity disorder. In *Handbook of disruptive behavior disorders* (pp. 75-104). Springer, Boston, MA.

Sesack, S. R., & Grace, A. A. (2010). Cortico-basal ganglia reward network: microcircuitry. *Neuropsychopharmacology,* 35(1), 27-47.

Shannon, C. E. (1948). A mathematical theory of communication. *The Bell system technical journal*, 27(3), 379-423.

Shaw, P., De Rossi, P., Watson, B., Wharton, A., Greenstein, D., Raznahan, A., ... & Chakravarty, M. M. (2014). Mapping the development of the basal ganglia in children with attentiondeficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*(7), 780-789.

Shaw, P., Lerch, J., Greenstein, D., Sharp, W., Clasen, L., Evans, A., ... & Rapoport, J. (2006). Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with attention-deficit/hyperactivity disorder. *Archives of general psychiatry, 63*(5), 540-549.

Shaw, P., Stringaris, A., Nigg, J., & Leibenluft, E. (2014). Emotion dysregulation in attention deficit hyperactivity disorder. *American Journal of Psychiatry, 171*(3), 276-293.

Shen, I. H., Lee, T. Y., & Chen, C. L. (2012). Handwriting performance and underlying factors in children with Attention Deficit Hyperactivity Disorder. *Research in developmental disabilities, 33*(4), 1301-1309.

Sherrington, C. S. (1906). Observations on the scratch-reflex in the spinal dog. *The Journal of physiology, 34*(1-2), 1.

Shumway-Cook, A., & Woollacott, M. H. (2007). *Motor control: translating research into clinical practice*. Lippincott Williams & Wilkins.

Simner, M. L. (1981). The grammar of action and children's printing. Developmental Psychology, 17(6), 866.

Simonoff, E., Taylor, E., Baird, G., Bernard, S., Chadwick, O., Liang, H., ... & Jichi, F. (2013). Randomized controlled double-blind trial of optimal dose methylphenidate in children and adolescents with severe attention deficit hyperactivity disorder and intellectual disability. *Journal of Child Psychology and Psychiatry, 54*(5), 527-535.

Sinzig, J., Döpfner, M., Lehmkuhl, G., & German Methylphenidate Study Group. (2007). Long-acting methylphenidate has an effect on aggressive behavior in children with attentiondeficit/hyperactivity disorder. *Journal of child and adolescent psychopharmacology, 17*(4), 421-432.

Slaats-Willemse, D., de Sonneville, L., Swaab-Barneveld, H., & Buitelaar, J. (2005). Motor flexibility problems as a marker for genetic susceptibility to attention-deficit/hyperactivity disorder. *Biological Psychiatry, 58*(3), 233-238.

Smits-Engelsman, B., Galen, G. P. V., & Schoemaker, M. M. (1998). Theory-based diagnosis and subclassification in the developmental coordination disorder. In *Perspectives on the classification of specific developmental disorders* (pp. 245-264). Springer, Dordrecht.

Sobel, L. J., Bansal, R., Maia, T. V., Sanchez, J., Mazzone, L., Durkin, K., ... & Peterson, B. S. (2010). Basal ganglia surface morphology and the effects of stimulant medications in youth with attention deficit hyperactivity disorder. *American Journal of Psychiatry, 167*(8), 977-986.

Sonuga-Barke, E. J. (2002). Psychological heterogeneity in AD/HD—a dual pathway model of behaviour and cognition. *Behavioural brain research, 130*(1-2), 29-36.

Sonuga-Barke, E. J. S., Houlberg, K., & Hall, M. (1994). When is "impulsiveness" not impulsive? The case of hyperactive children's cognitive style. *Journal of Child Psychology and Psychiatry*, 35(7), 1247-1253.

Sonuga-Barke, E. J., Becker, S. P., Bölte, S., Castellanos, F. X., Franke, B., Newcorn, J. H., ... & Simonoff, E. (2022). Annual Research Review: Perspectives on progress in ADHD science–from characterization to cause. *Journal of Child Psychology and Psychiatry*.

Sonuga-Barke, E. J., Sergeant, J. A., Nigg, J., & Willcutt, E. (2008). Executive dysfunction and delay aversion in attention deficit hyperactivity disorder: nosologic and diagnostic implications. *Child and adolescent psychiatric clinics of North America, 17*(2), 367-384.

Sonuga-Barke, E. J., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion—I. The effect of delay on choice. *Journal of Child Psychology and Psychiatry, 33*(2), 387-398.

Sowell, E. R., Thompson, P. M., Tessner, K. D., & Toga, A. W. (2001). Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex: Inverse relationships during postadolescent brain maturation. *Journal of Neuroscience, 21*(22), 8819-8829.

Stanford, M. S., Mathias, C. W., Dougherty, D. M., Lake, S. L., Anderson, N. E., & Patton, J. H. (2009). Fifty years of the Barratt Impulsiveness Scale: An update and review. *Personality and individual differences, 47*(5), 385-395.

Stanton, K., Forbes, M. K., & Zimmerman, M. (2018). Distinct dimensions defining the Adult ADHD

Self-Report Scale: Implications for assessing inattentive and hyperactive/impulsive symptoms. *Psychological assessment, 30*(12), 1549.

Steel, R. M., Bastin, M. E., McConnell, S., Marshall, I., Cunningham-Owens, D. G., Lawrie, S. M., ... & Best, J. J. (2001). Diffusion tensor imaging (DTI) and proton magnetic resonance spectroscopy (1H MRS) in schizophrenic subjects and normal controls. *Psychiatry Research: Neuroimaging, 106*(3), 161-170.

Stephenson-Jones, M., Samuelsson, E., Ericsson, J., Robertson, B., & Grillner, S. (2011). Evolutionary conservation of the basal ganglia as a common vertebrate mechanism for action selection. *Current biology, 21*(13), 1081-1091.

Sternberg, S. (1969). Memory-scanning: Mental processes revealed by reaction-time experiments. *American scientist, 57*(4), 421-457.

Steuer, I., & Guertin, P. A. (2019). Central pattern generators in the brainstem and spinal cord: an overview of basic principles, similarities and differences. *Reviews in the Neurosciences, 30*(2), 107-164.

Stevenson, J. C., Everson, P. M., Williams, D. C., Hipskind, G., Grimes, M., & Mahoney, E. R. (2007). Attention deficit/hyperactivity disorder (ADHD) symptoms and digit ratios in a college sample. *American Journal of Human Biology, 19*(1), 41-50.

Stewart, T., Choo, X., & Eliasmith, C. (2010). Symbolic reasoning in spiking neurons: A model of the cortex/basal ganglia/thalamus loop. In *Proceedings of the Annual Meeting of the Cognitive Science Society* (Vol. 32, No. 32).

Still, G. F. (1902). The Goulstonian Lectures. *Some abnormal psychical conditions in children*, 1008-

1012.

Stocco, A., & Lebiere, C. (2014). Inhibitory synapses between striatal projection neurons support efficient enhancement of cortical signals: A computational model. *Journal of computational neuroscience, 37(*1), 65-80.

Stocco, A., Lebiere, C., & Anderson, J. R. (2010). Conditional routing of information to the cortex: A model of the basal ganglia's role in cognitive coordination. *Psychological review, 117*(2), 541.

Stoet, G. (2010). PsyToolkit - A software package for programming psychological experiments using Linux. *Behavior Research Methods, 42*(4), 1096-1104.

Stoet, G. (2017). PsyToolkit: A novel web-based method for running online questionnaires and reaction-time experiments. *Teaching of Psychology, 44*(1), 24-31.

Stott, D. H., Moyes, F. A., & Henderson, S. E. (1985). *Diagnosis and remediation of handwriting problems.* Brook Educational Pub..

Sugihara, T., Diltz, M. D., Averbeck, B. B., & Romanski, L. M. (2006). Integration of auditory and visual communication information in the primate ventrolateral prefrontal cortex. *Journal of Neuroscience, 26*(43), 11138-11147.

Surmeier, D. J., Mercer, J. N., & Chan, C. S. (2005). Autonomous pacemakers in the basal ganglia: who needs excitatory synapses anyway?. *Current opinion in neurobiology, 15*(3), 312-318.

Sutcubasi, B., Metin, B., Kurban, M. K., Metin, Z. E., Beser, B., & Sonuga-Barke, E. (2020). Resting-

state network dysconnectivity in ADHD: A system-neuroscience-based meta-analysis. *The World Journal of Biological Psychiatry, 21*(9), 662-672.

Swanson, H. L., & Beebe-Frankenberger, M. (2004). The relationship between working memory and mathematical problem solving in children at risk and not at risk for serious math difficulties. *Journal of educational psychology, 96*(3), 471.

Swedlow, J. R., Goldberg, I. G., Eliceiri, K. W., & OME consortium. (2009). Bioimage informatics for experimental biology. *Annual review of biophysics*, 38, 327.

Szatmari, P. (1992). The epidemiology of attention-deficit hyperactivity disorders. *Child and Adolescent Psychiatric Clinics,* 1(2), 361-371.

Thapar, A., Cooper, M., Eyre, O., & Langley, K. (2013). Practitioner review: what have we learnt about the causes of ADHD?. *Journal of Child Psychology and Psychiatry, 54*(1), 3-16.

Thelen, E., Kelso, J. S., & Fogel, A. (1987). Self-organizing systems and infant motor development. *Developmental review, 7*(1), 39-65.

Thorndike, E. L. (1910). The contribution of psychology to education. *Journal of Educational Psychology, 1*(1), 5.

Tipper, S. P. (1985). The negative priming effect: Inhibitory priming by ignored objects. *The quarterly journal of experimental psychology, 37*(4), 571-590.

Towse, J. N., Redbond, J., Houston-Price, C. M., & Cook, S. (2000). Understanding the dimensional change card sort: Perspectives from task success and failure. *Cognitive Development, 15*(3), 347-365.

Tredgold, A. F. (1908). *A textbook of mental deficiency*. Baltimore: Williams and Wilkins.

Tseng, M. H., & Murray, E. A. (1994). Differences in perceptual-motor measures in children with good and poor handwriting. *The Occupational Therapy Journal of Research, 14*(1), 19-36.

Tucha, O., & Lange, K. W. (2001). Effects of methylphenidate on kinematic aspects of handwriting in hyperactive boys. *Journal of Abnormal Child Psychology, 29*(4), 351-356.

Turvey, M. T. (1977). Preliminaries to a Theory. *Perceiving, acting, and knowing: Toward an ecological psychology*, 211.

Uddin, L. Q., Kelly, A. C., Biswal, B. B., Margulies, D. S., Shehzad, Z., Shaw, D., ... & Milham, M. P. (2008). Network homogeneity reveals decreased integrity of default-mode network in ADHD. *Journal of neuroscience methods, 169*(1), 249-254.

Uexküll, J.V., 1926. *Theoretical Biology.* Harcourt, Brace & Co., New York

Ugawa, Y., Shimo, Y., & Terao, Y. (2020). Future of tanscranial magnetic stimulation in movement disorders: introduction of novel methods. *Journal of Movement Disorders, 13*(2), 115.

Uleman, J. S., & Bargh, J. A. (Eds.). (1989). *Unintended thought*. Guilford Press.

Utter, A. A., & Basso, M. A. (2008). The basal ganglia: an overview of circuits and function. *Neuroscience & Biobehavioral Reviews, 32*(3), 333-342.

Valera, E. M., Faraone, S. V., Murray, K. E., & Seidman, L. J. (2007). Meta-analysis of structural imaging findings in attention-deficit/hyperactivity disorder. *Biological psychiatry, 61*(12), 1361-1369.

Van De Voorde, S., Roeyers, H., Verté, S., & Wiersema, J. R. (2011). The influence of working memory load on response inhibition in children with attention-deficit/hyperactivity disorder or

reading disorder. *Journal of Clinical and Experimental Neuropsychology, 33*(7), 753-764.

Van Galen, G. P. (1991). Handwriting: Issues for a psychomotor theory. *Human movement science*, 10(2-3), 165-191.

Vanden, B., & Gary, R. (2007). *APA dictionary of psychology*. Washington, DC.

VanSant, A. F. (1990). Life-span development in functional tasks. *Physical Therapy, 70*(12), 788-798.

Vasconcelos, M., Hollis, K., Nowbahari, E., & Kacelnik, A. (2012). Pro-sociality without empathy. *Biology letters, 8*(6), 910-912.

Vaurio, R. G., Simmonds, D. J., & Mostofsky, S. H. (2009). Increased intra-individual reaction time variability in attention-deficit/hyperactivity disorder across response inhibition tasks with different cognitive demands. *Neuropsychologia, 47*(12), 2389-2396.

Velanova, K., Wheeler, M. E., & Luna, B. (2008). Maturational changes in anterior cingulate and frontoparietal recruitment support the development of error processing and inhibitory control. *Cerebral cortex, 18*(11), 2505-2522.

Verbruggen, F., & Logan, G. D. (2008). Response inhibition in the stop-signal paradigm. *Trends in cognitive sciences, 12*(11), 418-424.

Viholainen, H., Ahonen, T., Lyytinen, P., Cantell, M., Tolvanen, A., & Lyytinen, H. (2006). Early motor development and later language and reading skills in children at risk of familial dyslexia. *Developmental Medicine and Child Neurology, 48*(5), 367-373.

Volkow, N. D., & Swanson, J. M. (2003). Variables that affect the clinical use and abuse of methylphenidate in the treatment of ADHD. *American Journal of Psychiatry, 160*(11), 19091918.

Volkow, N. D., Fowler, J. S., Wang, G. J., Swanson, J. M., & Telang, F. (2007). Dopamine in drug abuse and addiction: results of imaging studies and treatment implications. *Archives of neurology*, 64(11), 1575-1579.

Volkow, N., & Li, T. K. (2005). The neuroscience of addiction. *Nature neuroscience, 8*(11), 1429-1430.

Wade, M. G. (1976). Effects of methylphenidate on motor skill acquisition of hyperactive children. *Journal of learning disabilities, 9*(7), 443-447.

Watling, R., Deitz, J., Kanny, E. M., & McLaughlin, J. F. (1999). Current practice of occupational therapy for children with autism. *The American Journal of Occupational Therapy, 53*(5), 498505.

Weisz, S. (1938). Studies in equilibrium reaction. *The Journal of Nervous and Mental Disease, 88*(2), 150-162.

Welsh, T. N., Elliott, D., Anson, J. G., Dhillon, V., Weeks, D. J., Lyons, J. L., & Chua, R. (2005). Does Joe influence Fred's action?: Inhibition of return across different nervous systems. *Neuroscience Letters, 385*(2), 99-104.

Welsh, T. N., Lyons, J., Weeks, D. J., Anson, J. G., Chua, R., Mendoza, J., & Elliott, D. (2007). Within- and between-nervous-system inhibition of return: Observation is as good as performance. *Psychonomic Bulletin & Review, 14*(5), 950-956.

Wiecki, T. V., & Frank, M. J. (2013). A computational model of inhibitory control in frontal cortex and basal ganglia. *Psychological review, 120*(2), 329.

Wierenga, L. M., Langen, M., Oranje, B., & Durston, S. (2014). Unique developmental trajectories of cortical thickness and surface area. *Neuroimage*, 87, 120-126.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review. *Biological psychiatry, 57*(11), 1336-1346.

Wilson, D. M. (1961). The central nervous control of flight in a locust. *Journal of Experimental Biology, 38*(2), 471-490.

Wodka, E. L., Mark Mahone, E., Blankner, J. G., Gidley Larson, J. C., Fotedar, S., Denckla, M. B., & Mostofsky, S. H. (2007). Evidence that response inhibition is a primary deficit in ADHD. *Journal of clinical and experimental neuropsychology, 29*(4), 345-356.

Wojcik, J., Schwabedal, J., Clewley, R., & Shilnikov, A. L. (2014). Key bifurcations of bursting polyrhythms in 3-cell central pattern generators. *PloS one, 9(*4), e92918.

Wolpert, D. (2011). Daniel Wolpert: The Real Reason for Brains. TED.

Wolpert, D. M., & Flanagan, J. R. (2001). Motor prediction. *Current biology, 11*(18), R729-R732.

Wood, A. C., & Neale, M. C. (2010). Twin studies and their implications for molecular genetic studies: endophenotypes integrate quantitative and molecular genetics in ADHD research. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(9), 874-883.

Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice, 17*(4), 281.

Wright, A., & Diamond, A. (2014). An effect of inhibitory load in children while keeping working memory load constant. *Frontiers in psychology*, 5, 213.

Wundt, W. M., & Judd, C. H. (1902). *Outlines of psychology*. W. Engelmann.

Yager, L. M., Garcia, A. F., Wunsch, A. M., & Ferguson, S. M. (2015). The ins and outs of the striatum: role in drug addiction. *Neuroscience*, 301, 529-541.

Yan, J. H., & Thomas, J. R. (2002). Arm movement control: differences between children with and without attention deficit hyperactivity disorder. *Research quarterly for exercise and sport, 73*(1), 10-18.

Zelazo, P. D., Carter, A., Reznick, J. S., & Frye, D. (1997). Early development of executive function: A problem-solving framework. *Review of general psychology, 1*(2), 198-226.

Zesiger, P., Orliaguet, J. P., Boë, L. J., & Mounoud, P. (1995). The influence of syllabic structure in handwriting and typing production. *Les Cahiers de l'ICP. Rapport de recherche, (4),* 77-87.

Zhang, Y., Bertolino, A., Fazio, L., Blasi, G., Rampino, A., Romano, R., ... & Sadée, W. (2007). Polymorphisms in human dopamine D2 receptor gene affect gene expression, splicing, and neuronal activity during working memory. *Proceedings of the National Academy of Sciences, 104*(51), 20552-20557.

Ziviani, J., & Elkins, J. (1984). An evaluation of handwriting performance. *Educational review, 36*(3), 249-261.

Appendix

Effect Size Motor Inhibition Task

Exp 1 **Significant** Difference that participants switched button presses on significant trial

motor inhibition effect present

t(17) = 4.3, p = 0.001, d = 1.0

Effect Size d_{Cohen} = 1.2 Large

Exp 2 **Control**, **Significant** Difference participants switched button presses on significant trial

motor inhibition effect present

t(24) = 5.574, p = 0.00001, d = 1.1

Effect Size d_{Cohen} = 2.3 Large

ADHD, **Significant** difference to repeat button presses on significant trials

Motor inhibition effect not present

t(15) = 3.4, p = 0.004, d = 0.85.

Effect Size d_{Cohen} = 3.112 Large

Exp 3 Not **Significant** Difference that participants switched button presses on significant trial

motor inhibition effect not-present

t(34) = 1.1, p = 0.3, d = 0.18.

Effect Size d_{Cohen} = 0.46 Small

Experiment 1. Motor Induce Motor Inhibition Task checking for bias on key presses

Experiment 1 N=18	Test Used	H_o	Effect Size
All A button (left) and B Button (right) presses Response times	Paired Sample T-Test	Results of the paired-t test indicated that there is a non-significant difference between A-Button presses (M = 532.3 ,SD = 155.9) and B-Button Presses (M = 511.1 ,SD = 113.9), t(23) = 1.2, p = .245.	The observed effect size d is small, 0.24

Count of Left Button presses and Right button presses	Paired Sample T-Test	Results of the paired-t test indicated that there is a non significant small difference between Before (M = 39.1 ,SD = 8) and After (M = 43.8 ,SD = 10.6), t(23) = 1.2, p = .228.	The observed effect size d is small, 0.25.
Ambiguous Lines Small, Medium, and Large Response Times	One-Way ANOVA and Tukey HSD	Since p-value 0.659296 > α, H0 is accepted. The averages of all groups are assumed to be equal.	The observed effect size f is small (0.11).

Experiment 2 N=41 25 Control, 16 ADHD	Test Used	H_o	Effect Size
All A button (left) and B Button (Right) ADHD and Control	One-Way ANOVA and Tukey HSD	Since p-value 0.333481 > α, H0 is accepted. The averages of all groups are assumed to be equal.	The observed effect size f is medium (0.21).

Group	All Key Presses Right Side ADHD	All Key Presses Left Side Control Group	All Key Presses Right Side Control
All Key Presses Left Side ADHD	34.01	41.59	61.68
All Key Presses Right Side ADHD	0	75.6	95.69
All Key Presses Left Side Control Group	75.6	0	20.09

Experiment 2 N=41 25 Control, 16 ADHD	Test Used	H_o	Effect Size
Ambiguous Lines Small, Medium, and Large (ADHD) Ambiguous Lines Small, Medium, and Large (Control)	One-Way ANOVA and Tukey HSD	Since p-value 0.624162 > α, H0 is accepted. The averages of all groups are assumed to be equal.	The observed effect size f is small (0.17).

Group	x2	x3	x4	x5	x6
x1	68.94	76.78	20.75	56.26	75.33
x2	0	7.84	89.69	12.68	6.38
x3	7.84	0	97.53	20.53	1.46
x4	89.69	97.53	0	77	96.07
x5	12.68	20.53	77	0	19.07

Experiment 3 N=35	Test Used	H_o	Effect Size
All A button (left) and B Button (right) presses Response times	Paired Sample T-Test	Results of the paired-t test indicated that there is a non-significant difference between Before (M = 684.9 ,SD = 173.7) and After (M = 675.6 ,SD = 200.9), $t(34)$ = 0.4, p = .673.	The observed effect size d is very small, 0.072. This indicates that the magnitude of the difference between the average of the differences and the expected average of the differences is very small.
Ambiguous Lines Small, Medium, and Large	One-Way ANOVA and Tukey HSD	Since p-value 0.781809> α, H0 is accepted. The averages of all groups are assumed to be equal.	The observed effect size f is small (0.13).

Experiment 4.

The mean number of times that the ADHD group rolled the dice off the table was 4.7 (SD = 3.5) times. For the control group, this figure was 0.69 (SD = 1.21). These values were significantly different, $t(176) = 9.48$, $p < 0.001$.

Effect Size d_{Cohen} = 2.53 Large

Experiment 5.

Fractal Method T-Test the 16-member ADHD group (M = 1.42, SD = 0.08) when compared with 162 member control group (M = 1.32, SD = 0.1) demonstrated more deviation in their handwriting $t(176)=4.186$, $p = 0.001$

Effect Size d_{Cohen} = 0.11 below small

Quadratic Method the ADHD group (M = 0.08, SD = 0.05), when compared with the control group (M = 0.04, SD = 0.02), demonstrated significantly less motor inhibition resulting in more deviation in their handwriting $t(176)=7.491$, $p = 0.001$

Effect Size d_{Cohen} = 1.62 Large

There was also a strong correlation between the two measures ($r(176) = 0.71$, $p = 0.001$), suggesting that the two measures index a related phenomenon.

Experiment 6.

There was a significant difference between the ADHD group (M = 76.9%, SD = 18.9%) and the control group (M = 98.0%, SD = 4.68%), $t(176) = 11.2$, $p < 0 .001$].

Effect Size d_{Cohen} = 2.864Large

The one-way ANOVA was conducted to see if the ADHD group could not complete the multiplication of addition questions in this experiment. The groups consisted of 178 total participants: 168 in the control group and 16 in the ADHD group.

The means for the

X_1 control group, non-critical trials mean = 99.4% success rate,

X_2 control group, critical trials mean = 98.3% success rate.

X_3 ADHD group for non-critical trials mean = 99.4% success rate

X_4 ADHD group for critical trials mean = 76.9% success rate.

The test statistic F equals 105.280555, which is not in the 95% region of acceptance: [$-\infty$: 2.6301] The DF (between groups= 3) and (within groups was 354).

p-value equals -4.44089e-16, [$p(x \leq F) = 1$]. This means that the chance of type1 error (rejecting a correct H0) is small: -4.441e-16 (-4.4e-14%)

The observed effect size f is large (0.94). That indicates that the magnitude of the difference between the averages is large. The $\eta 2$ equals 0.47. This means that the group explains 47.2% of the variance from the average (similar to R2 in the linear regression)

Tukey HSD / Tukey Kramer

The means of the following pairs are significantly different: x1-x4, x2-x4, x3-x4.

Pair	Difference	SE	Q	Lower CI	Upper CI	Critical Mean	p-value
x1-x2	0.01078	0.003816	2.8263	-0.0031	0.02471	0.01393	0.1905
x1-x3	0.000539	0.009024	0.05973	-0.0324	0.03348	0.03294	1
x1-x4	0.2251	0.009024	24.9491	0.1922	0.2581	0.03294	1.323e-10
x2-x3	0.01025	0.009024	1.1353	-0.0227	0.04319	0.03294	0.8531
x2-x4	0.2144	0.009024	23.7541	0.1814	0.2473	0.03294	1.323e-10
x3-x4	0.2246	0.01218	18.443	0.1802	0.2691	0.04446	1.323e-10

The test priori power is strong: 0.9856.

There was no significant difference between ADHD and control groups for Multiplication, meaning that children with ADHD can complete their math tasks at a comparable success rate as the control group.

Students were tested in prior lessons and ADHD students could add at a prior rate of 99.4% and Non ADHD students had a success rate on addition of 100%.

Experiment 7.

Significant Difference between response rate was 48.2% (SD = 7.0) and chance value of 50%, t(65) = 2.2 p = 0.042.

Experiment 8.

The mean Repeat response rate was 47.2% (SD = 7.2). This value was significantly different to the chance value of 50%, t(63) = 2.3 p = 0.042.