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Graphomotor Fluency in Child and Adolescent ADHD: Neuropsychological Factors and Implications for Assessment

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GRAPHOMOTOR FLUENCY IN CHILD AND ADOLESCENT ADHD: NEUROPSYCHOLOGICAL FACTORS AND IMPLICATIONS FOR ASSESSMENT

by

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Declaration of Originality

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Abstract

Attention-Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder of complex etiology that typically presents behaviourally with symptoms of inattention, hyperactivity, and impulsivity. Among associated features, executive dysfunction, learning difficulties, and motor problems are common of the disorder. The present study involved two parts, where Part I sought to determine the optimal methodology to be used for Part II. Within the context of childhood ADHD, Part II of the study investigated 1) the effects of cognitive control on kinematic graphomotor fluency, 2) whether graphomotor fluency development is attenuated in children with ADHD, 3) which neuropsychological factors would best predict improvement in graphomotor fluency, and 4) the predictive ability of graphomotor improvement in identifying ADHD. Results indicated the following: 1) participants with and without ADHD demonstrated similar graphomotor fluency as cognitive control demands and figural complexity increased, 2) participants with ADHD evidenced attenuated procedural learning relative to controls when learning a novel grapheme, 3) the neuropsychological factors of verbal skills, processing speed, and fine motor skills were not predictive of improvement in graphomotor fluency, and 4) change in graphomotor fluency improvement did not demonstrate adequate ability to differentiate between those with and without ADHD. Implications, limitations, and additional considerations are discussed.

Dedication

To my wife, Laura, my family, and my friends. Thank you for your patience and support. I could not have completed this without each of you.

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List of Abbreviations

ADHD – Attention Deficit/Hyperactivity Disorder

ADHD-C – ADHD with a Predominantly Combined Presentation

ADHD-HI – ADHD with a Predominantly Hyperactive-Impulsive Presentation

ADHD-PI – ADHD with a Predominantly Inattentive Presentation

ANOVA – Analysis of Variance

AUC – Area Under the Curve

BAARS-IV – Barkley Adult ADHD Rating Scale-IV

BASC-2 – Behavioral Assessment System for Children-2nd Edition

BD – Block Design

BOLD – Blood-Oxygen-Level Dependent

CD – Conduct disorder

DA – Dopamine

DAT1 – Dopamine Transporter Gene 1

DBH – Dopamine-β-hydroxylaxe

DCD – Developmental Coordination Disorder

DCDQ'07 – Developmental Coordination Disorder Questionnaire 2007

df – Degrees of Freedom

DMN – Default Mode Network

DRD2, 4, & 5 – Dopamine D2, 4, & 5 Receptor Genes, respectively

DS – Digit Span

DSM – Diagnostic and Statistics Manual (-IV for 4th edition and -5 for 5th edition)

DV – Dependent Variable

EEG – Electroencephalogram

EF – Executive Functioning

fALFF – Fractional Amplitude of Low-Frequency Fluctuation

fMRI – Functional Magnetic Resonance Imaging

FSIQ – Full Scale Intelligence Quotient

HKD – Hyperkinetic Disorder

ICD – International Classification of Disease

ID – Identification Number

IQ – Intelligence Quotient

LD – Learning Disability

M – Mean

MANOVA – Multivariate Analysis of Variance

MHPG – 3-Methoxy-4-Hydroxyphenylethylene Glycol

MRA – Multiple Regression Analysis

MRI – Magnetic Resonance Imaging

MS – Mean Squared

n – Number of Subjects in Group

NA – Noradrenaline

NE – Norepinephrine

NIMH – National Institute of Mental Health

NJ – Normalized Jerk

ODD – Oppositional Defiant Disorder

OV – Outcome Variable

PET – Positron Emission Tomography

PV – Predictor Variable

RDoC – Research Domain Criteria

ReHo – Regional Homogeneity

ROC – Receiver Operating Characteristic

SD – Standard Deviation

SES – Socioeconomic Status

SS – Symbol Search

SS – Sum of Squares

SV – State Variable

TV – Test Variable

VC – Vocabulary

WRAT-4 – Wide Range Achievement Test-4th Edition

Zn – Zinc

Chapter 1: Introduction

Graphomotor Fluency in Child and Adolescent ADHD: Neuropsychological Factors and Implications for Assessment

Attention-deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder commonly characterized diagnostically by symptoms of inattention, hyperactivity, and impulsivity. Prevalence estimates of childhood ADHD have increased in some cultures. Prevalence and gender ratio discrepancies appear to decrease with age, and the economic impact of treatment and non-treatment is substantial in both childhood and adulthood. The etiology of ADHD is complex and likely multifactorial in nature, although over the past 20 years genetic and neurological causes within the context of environmental factors have been extensively researched as sources of pathogenesis. Neuropsychological studies have identified several risk factors and neurocognitive deficits that are highly associated with ADHD, including deficits in executive functioning (e.g., cognitive control), learning, and motor functioning. Graphomotor research utilizing digitizing technology and kinematic analysis has been a burgeoning area of ADHD research over the past decade and provides the opportunity to study neuropsychological aspects of ADHD in an integrated fashion, noting the highly complex nature of handwriting as it involves a combination of executive, motor, language, and various sensory functions.

Assessment and diagnosis currently rely on psychological interview, behavioural observations, and ADHD-focused rating scales completed by multiple informants. However, neuropsychological assessment gathers information relevant to understanding the child holistically and uses objective measurements of cognitive functioning and

unique data that are not provided by rating scales. Further, the National Institute of Mental Health (NIMH) has developed the Research Domain Criteria (RDoC), which is an initiative to create a framework of objective neurobiological measures (i.e., biomarkers) for the identification and classification of psychopathology. The field of clinical neuropsychology is thus well positioned to respond to this strategy, as understanding brain-behaviour associations is the purview of neuropsychology.

The present study included two parts and five studies. Part I involved recruiting undergraduate student participants in order to determine which kinematic research paradigm would likely elicit the greatest effects of cognitive control on graphomotor fluency (representing Part I, Study 1). Part II of this study required the recruitment of children and adolescents ages 9 to 15 with and without ADHD, with ADHD participants discontinuing stimulant medication 24 to 48 hours prior to taking part in the study. The methodologies in Part II of this study were designed to determine the following: Study 2 – the effects of cognitive control on kinematic graphomotor fluency; Study 3 – if learning a new graphomotor program is attenuated in children with ADHD; Study 4 – the neuropsychological abilities that best predict improvement in graphomotor fluency; and Study 5 – the predictive ability that relative change in graphomotor fluency has in identifying children with and without ADHD.

ADHD: Epidemiology, Course, and Outcome

Attention-deficit Hyperactivity Disorder (ADHD) is a heterogeneous neurodevelopmental disorder characterized diagnostically by symptoms of inattention, hyperactivity, and impulsivity (American Psychiatric Association, 2013; Wasserman & Wasserman, 2012). The earliest reference in the medical literature to such a condition

dates back at least to the late 1700s (Barkley & Peters, 2012). ADHD affects approximately 3.4% to 5% of children across a variety of cultures worldwide (G. Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007; G. V. Polanczyk, Salum, Sugaya, Caye, & Rohde, 2015) and 9% of children between the ages of 3 and 17 in the United States (Bloom, Cohen, & Freeman, 2012). Also within the United States, data indicate relative increases in diagnosis of ADHD between 2001 and 2010 among children of diverse ethnic and socioeconomic backgrounds (particularly among Caucasian children), as well as a 3:1 male-to-female ratio (Getahun et al., 2013; Morgan, Staff, Hillemeier, Farkas, & Maczuga, 2013).

Data demonstrating the persistence of ADHD into adulthood have been mixed over the years, with estimates ranging between 4% (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998) and 85% (Barkley, Fischer, Smallish, & Fletcher, 2002). However, persistence estimates are noted to vary significantly based on a variety of methodological and participant factors (Barkley, 2006) and many estimates are likely conservative given the strict use of diagnostic criteria from the fourth edition of the Diagnostic and Statistics Manual of Mental Disorders (i.e., DSM-IV) (American Psychiatric Association, 2000; Asherson et al., 2012; Root & Resnick, 2003; Sibley et al., 2012). Despite variable estimates of persistence, there is consensus that most childhood ADHD persists into adulthood (Kooij et al., 2010). A relatively recent meta-analysis of epidemiological data estimated the prevalence of ADHD in adults at approximately 2.5% (Simon, Czobor, Balint, Meszaros, & Bitter, 2009). Unfortunately, methodological and participant variability of the studies involved in this meta-analysis precluded the ability to draw firm

conclusions regarding persistence of ADHD into adulthood. Further, this estimate was noted as being conservative given strict diagnostic guidelines.

Collectively, children and adults with ADHD tend to experience greater academic, psychiatric, social, occupational, and medical problems beyond diagnostic symptomatology when compared with the general population (Barkley, 2006). A prospective, 33-year follow-up study found that adults who were diagnosed with ADHD at approximately age 8 with no history of oppositional or conduct problems evidenced lower academic achievement, lower occupational level, lower median annual salary, lower self-ratings of social functioning, and a higher divorce rate as compared to matched controls also followed into adulthood (Klein et al., 2012). Other studies have identified similar occupational, academic, and functional difficulties in children, adolescents, and adults with ADHD, including greater unemployment, lower productivity (e.g., poor performance and absenteeism), poor workplace behaviour, greater risk of driving faults, lower subjective ratings of quality of life, and poor social functioning (Classen & Monahan, 2013; Kupper et al., 2012; Skirrow & Asherson, 2013; Staikova, Gomes, Tartter, McCabe, & Halperin, 2013; Yang, Tai, Yang, & Gau, 2013). Research demonstrating adverse health outcomes for children and adults with ADHD has also mounted over the years, indicating relatively greater risk of substance use (particularly cigarettes), more frequent injuries requiring medical attention (e.g., body cavity insertions in very young children, burns in older children, and injuries sustained from vehicular motor accidents in adolescents and adults), more sleep problems (e.g., increased sleep time to fall asleep, more frequent waking after sleep, and increased motor activity during sleep) due to behavioural hygiene and/or central nervous system factors, and obesity

(Cortese, Ramos Olazagasti, et al., 2013; H. K. Lee et al., 2014; Nigg, 2013; Owens et al., 2013; Pingault et al., 2013). In the United States, the annual economic impact of childhood ADHD has been estimated at between $21 and $44 billion due to related health care costs and between $15 and $25 billion with regard to educational spending (Doshi et al., 2012). The annual incremental costs of adult ADHD in the United States was greater than that of children between 1990 and 2011, with lost productivity and income estimated between $87 and $138 billion (Doshi et al., 2012). In addition, between 1998 and 2010, resource utilization due to clinical diagnostic, administrative, and medical treatment costs was four times greater in the United Kingdom for those affected by ADHD versus unaffected individuals (Holden et al., 2013). Taken together and given the significant social, economic, and individual impact of ADHD, early identification and treatment of ADHD appear crucial (Barkley, 2006; Doshi et al., 2012).

Several risk factors for the development, severity, and outcome of those with ADHD have been identified throughout the years, although the degree of risk or effect conferred by specific factors may be developmental in nature (Cherkasova, Sulla, Dalena, Ponde, & Hechtman, 2013). Early risk factors predicting the development and outcome of those with ADHD include genetic factors, low birth weight, language and motor delay, maternal factors (e.g., education, stress, age, and substance use during pregnancy), and socioeconomic factors. Concerning the severity of ADHD symptomatology, cognitive functioning (e.g., executive functioning) and psychiatric comorbidity (e.g., Oppositional Defiant Disorder and Conduct Disorder) become significant risk factors affecting the outcome of those with ADHD in the pre-school, school-aged, and adolescent years (Cherkasova et al., 2013; Gurevitz, Geva, Varon, & Leitner, 2014; Willoughby, Pek,

Greenberg, & Family Life Project, 2012). Current evidence indicates that although treated individuals with ADHD do not improve to levels of functioning identical to unaffected persons, compared with untreated individuals with ADHD, they experience benefits in functional areas beyond symptom relief, including reduced drug abuse, improved academic functioning, improved social functioning, reduced rates of obesity, and better occupational outcomes (M. Shaw et al., 2012). Varying degrees of effectiveness in treating the symptoms diagnostic of ADHD have been found for non-pharmacological (e.g., vitamin supplementation, diet, and biofeedback techniques) and psychosocial interventions (e.g., executive functioning and parent training), but stimulant medications continue to be the most frequently used pharmacological treatment for those with ADHD (Barkley, 2006; Halperin et al., 2013; Sonuga-Barke et al., 2013).

Etiology. The etiology of ADHD is complex and multifactorial in nature. However, extensive research over the past 20 years has implicated interactions between genetic and environmental factors and their resulting neurological corollaries as primary agents of pathogenesis and symptom expression in ADHD (Barkley, 2006; Cortese, 2012; Koziol, Budding, & Chidekel, 2013; McLoughlin, Palmer, Rijsdijk, & Makeig, 2014; Merwood et al., 2014).

Genetics and neurotransmitter systems. The extant research has not implicated chromosomal abnormalities as causing ADHD, but several lines of research (i.e., family, adoption, twin, and genetic studies) provide evidence that ADHD has a high degree of heritability and in turn a significant genetic component to the development and phenotypic expression of the disorder (Barkley, 2006). Heritability estimates have been reported as high as 0.76 (Faraone et al., 2005). Further highlighting the heritable nature of

the disorder are findings that asymptomatic siblings present with neuroanatomical findings that trend toward the same cortical volumetric reductions (see below) that are seen in their affected siblings (Durston et al., 2004; von Rhein et al., 2015). Many genes involved with the dopaminergic, noradrenergic, serotonergic, cholinergic, and other systems have been investigated for their potential involvement in ADHD, and although no single neurotransmitter system is likely to account for the complex phenotypic expressions and heterogeneity associated with ADHD (Cortese, 2012; L. Yang et al., 2013; Zayats et al., 2015), the greatest focus has been on genes affecting the dopaminergic system and its functioning (Arnsten, Berridge, & McCracken, 2009; Banaschewski, Becker, Scherag, Franke, & Coghill, 2010; Barkley, 2006; Biederman, 2005; Faraone et al., 2005; Spellicy et al., 2012; Spencer et al., 2013). The dopamine transporter (DAT1), dopamine-β-hydroxylase (DBH), and the dopamine D5 receptor (DRD5) genes, for example, have been found to be associated with ADHD, with DAT1 and DBH demonstrating strong familial transmission (Daly, Hawi, Fitzgerald, & Gill, 1999). A recent meta-analysis also found DRD5, DRD2, and DRD4 polymorphisms as conferring a high risk for the development of ADHD (J. Wu, Xiao, Sun, Zou, & Zhu, 2012). Although genetic factors are clearly implicated, the current state of "findings from genetic studies of ADHD are still inconsistent and inconclusive," thus preventing firm conclusions to be drawn regarding which genetic system(s) are involved in its pathogenesis (Li, Chang, Zhang, Gao, & Wang, 2014).

Despite inconsistency in studies aimed at identifying genes associated with the development of ADHD, genetic studies strongly support dysregulation and/or availability of noradrenaline and dopamine neurotransmitters as mechanisms associated with the core

neuropsychological deficits of ADHD (del Campo et al., 2013; Konrad & Eickhoff, 2010). For example, the combination of dopamine (DA) and noradrenaline (NA) appear to affect abilities related to inhibition via prefrontal cortex involvement, whereas DA alone and its effect on the subcortical circuitry of the basal ganglia is suspected to influence attentional abilities (del Campo, Chamberlain, Sahakian, & Robbins, 2011). In addition, children and adolescents with ADHD who possessed two copies of the 10-repeat DAT1 allele were shown to commit more errors and demonstrated greater response variability on a task of sustained attention than either participants with only one copy of the 10-repeat DAT1 allele or unaffected controls (Bellgrove, Hawi, Kirley, Gill, & Robertson, 2005). Mutations of the DAT1 gene have also been found to be related to poor working memory performance in adult ADHD (Brown et al., 2011) and performance on tasks of executive functioning in adults without ADHD (Gordon, Devaney, Bean, & Vaidya, 2015).

Structural neuroimaging. The advent and subsequent popularity of modern neuroimaging techniques such as magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI) have provided researchers with the ability to study the neuroanatomical structures of living individuals in a non-invasive manner. In the case of ADHD, early studies focused on the volumetric differences within specific cortical regions, subcortical nuclei, and major white matter bundles, which were thought to be implicated in the symptomatic expression of ADHD (Barkley, 2006). Across both children and adults, the most replicated abnormalities – the extent of which suggested widespread neurological differences in those with ADHD – include volumetric reduction of the dorsolateral prefrontal cortex, anterior cingulate, basal ganglia, corpus callosum, and, particularly, the

cerebellum (Bledsoe, Semrud-Clikeman, & Pliszka, 2011; McAlonan et al., 2007; Seidman et al., 2006; Stoodley, 2014; Valera, Faraone, Murray, & Seidman, 2007).

Global white matter differences have not been consistently demonstrated in those with ADHD compared with controls (Amico, Stauber, Koutsouleris, & Frodl, 2011; Batty et al., 2010; Castellanos et al., 2002; Durston et al., 2004; McAlonan et al., 2007; Narr et al., 2009), although volumetric and white matter microstructural integrity differences have been more consistently demonstrated in the corpus callosum and more specific pathways such as the frontal-striatal system, respectively (de Zeeuw, Mandl, Hulshoff Pol, van Engeland, & Durston, 2012; Hynd et al., 1991; McAlonan et al., 2007; Seidman, Valera, & Makris, 2005; Tamm, Barnea-Goraly, & Reiss, 2012). A recent meta-analysis identified reduced white matter structural integrity in all age groups of individuals with ADHD, including the white matter tracts of the right anterior corona radiata, areas of the corpus callosum, left- and right-hemispheric internal capsule, and the left cerebellum (van Ewijk, Heslenfeld, Zwiers, Buitelaar, & Oosterlaan, 2012). Research has also provided evidence that white matter differences are related to at least some of the neurocognitive deficits associated with ADHD (Hong et al., 2014; Onnink et al., 2015; Shang, Wu, Gau, & Tseng, 2013; Treit, Chen, Rasmussen, & Beaulieu, 2013; van Schouwenburg et al., 2014; Y. H. Wu, Gau, Lo, & Tseng, 2014).

There is also accumulating evidence indicating that the neuroanatomical differences between those with ADHD and unaffected individuals are best viewed from a developmental standpoint. For example, widespread cortical and laminar thinning involving frontal, parietal, temporal, limbic, and occipital lobes in those with ADHD appear to be consistent anatomical markers for the disorder (Almeida Montes et al., 2013;

Batty et al., 2010; Hoekzema et al., 2012; Makris et al., 2007; Narr et al., 2009; Schweren et al., 2015; Shaw, 2015). When viewed longitudinally and within neurodevelopmental contexts, those with ADHD have demonstrated regional specific maturational order that is similar to unaffected individuals, but with an overall delay in the developmental trajectory of cortical thickness and surface area as a whole, as well as in developmental differences in prefrontal and subcortical regions (e.g., basal ganglia) and functionally connected neural networks (see below) (Sato, Hoexter, Castellanos, & Rohde, 2012; Shaw et al., 2014; Shaw et al., 2007; Shaw et al., 2006; P. Shaw et al., 2012; Tomasi & Volkow, 2014). Interestingly, similar global and prefrontal cortex developmental trajectories have been found in children without ADHD who demonstrate symptoms of hyperactivity and impulsivity (Shaw et al., 2011), and altered cortical, cerebellar, and white matter maturation has also been linked to increased ADHD symptomatology (Cortese, Imperati, et al., 2013; Ghassabian et al., 2013; Mackie et al., 2007; Shaw et al., 2013).

Functional neuroimaging. One of the major limitations of structural neuroimaging studies is that associations between structure (e.g., volumetric differences and microstructural integrity) and function (e.g., ADHD symptomatology) can only be inferred due to the correlational nature of these techniques. Functional neuroimaging techniques such as electrophysiological recording (e.g., EEG), functional magnetic resonance imaging (fMRI), and positron emission tomography (PET) are also correlational, but these methods provide additional evidence for associations between underlying structures and function because electrical and metabolic activity within cerebral structures can be measured while individuals engage in specified activities.

Overall, functional neuroimaging findings have been largely concordant with structural findings associated with ADHD, implicating areas believed to be involved in attention, inhibition, and motor control (Barkley, 2006; Brossard-Racine, Majnemer, & Shevell, 2011; Seidman et al., 2006; Shaw et al., 2006; Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998). For example, compared with healthy children and adolescents, children and adolescents with ADHD tend to show patterns of reduced activation in the prefrontal cortex, basal ganglia, and cerebellum when performing tasks related to attention, inhibition, motor control, and executive function (Bush et al., 1999; Durston et al., 2003; Geburek, Rist, Gediga, Stroux, & Pedersen, 2013; Mostofsky et al., 2006; Posner et al., 2011; Rubia et al., 1999; Shi et al., 2012; Smith, Taylor, Brammer, Toone, & Rubia, 2006; Teicher et al., 2000; Tsujimoto et al., 2013; Vaidya et al., 1998; R. A. Yeo et al., 2003). Similar to structural findings, increasingly worse performance on tasks tapping cognitive functions that have been noted to be weaker in children and adults with ADHD have been associated with increasingly greater reductions in metabolic activity in homologous cortical regions (Ko et al., 2013; Woltering, Liu, Rokeach, & Tannock, 2013; Yasumura et al., 2014). A pair of recent meta-analyses of fMRI studies investigating functional abnormalities in those with ADHD identified groups of cortical and subcortical regions that were associated with more circumscribed deficits, including inhibition (inferior frontal cortex, supplementary motor area, and anterior cingulate), attention (dorsolateral prefrontal cortex, parietal lobe, and cerebellum), and timing (left inferior prefrontal cortex, insula, cerebellum, and left inferior parietal lobe) (Hart, Radua, Mataix-Cols, & Rubia, 2012; Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013).

Together, structural and functional imaging findings indicate widespread neuroanatomical and neurophysiological differences in those with ADHD.

Neural networks underlying ADHD. As described above, earlier investigations into the structural and functional neuroanatomical correlates of ADHD were largely cortico-centric in nature and/or focused on circumscribed regions or subcortical nuclei (Koziol, Budding, & Chidekel, 2013). However, research investigating normal and atypical neurocognitive functioning has shifted from a focus on regional differences in structure and function towards considering interconnected brain networks, with considerable evidence accumulating through functional and resting state neuroimaging methodologies to suggest that behaviour, neurocognitive functioning, and "thinking" require large-scale, whole brain, reciprocal interactions involving the cortex, subcortical nuclei (i.e., the basal ganglia), and cerebellum (Arsalidou, Duerden, & Taylor, 2013; Konrad & Eickhoff, 2010; Koziol, Budding, & Chidekel, 2013; Sepulcre, Sabuncu, Yeo, Liu, & Johnson, 2012). Understanding the topological and functional organization of the brain's networks within a developmental framework is in turn critical to understanding both normal and abnormal neurocognitive functioning (Grayson et al., 2014). Indeed, some researchers have gone so far as to say that "brain network associated dysfunctions have been found to be central in ADHD pathophysiology" (De La Fuente, Xia, Branch, & Li, 2013, p.5).

Early investigations of large-scale brain networks identified what has been termed the default mode network (DMN), which can be described as a resting state, baseline level of neurophysiological activity within a consistently defined neural network involving several interacting subnetworks (Buckner, Andrews-Hanna, & Schacter, 2008;

Raichle et al., 2001). Research has demonstrated that when performing a specific task, the DMN becomes suppressed whereas other networks become more active. One example is the cognitive control network. The cognitive control network consists of the dorsal anterior cingulate cortex, supplemental motor area, dorsolateral prefrontal cortex, anterior insular cortex, and posterior parietal cortex. While the DMN demonstrates reduced activity when performing tasks, the cortical and subcortical regions of the cognitive control network and its related functions (e.g., working memory, response inhibition, and cognitive set shifting) become increasingly active (Buckner et al., 2008; Posner, Park, & Wang, 2014; Raichle et al., 2001). Interestingly, a failure to suppress the DMN has also been associated with attentional lapses in healthy individuals (Weissman, Roberts, Visscher, & Woldorff, 2006). Taken together, these observations led to the speculation that differences within or between the DMN and other neural networks may explain the attentional and variable performance profile demonstrated by those with ADHD (Sonuga-Barke & Castellanos, 2007). Indeed, there is evidence indicating decreased homogeneity and altered connectivity patterns of the DMN in both adults and children with ADHD, most notably of which relate to reduced magnitude of connectively patterns between the cognitive control network and the DMN (X. Cao et al., 2009; Castellanos et al., 2008; Choi, Jeong, Lee, & Go, 2013; Fair et al., 2010; Franzen et al., 2013; Sun et al., 2012). Associations between reduced suppression of the DMN and distractibility (Fassbender et al., 2009) as well as altered DMN connectivity and problems with inhibition, reaction time variability, and impulsivity in children with ADHD have also been identified (Costa Dias et al., 2013; Feige et al., 2013; Mennes et al., 2011).

Brain networks other than the DMN have more recently been investigated as sources of pathology in the case of ADHD and other psychological disorders. A large-scale study involving 1,000 healthy adult participants defined seven consistently identifiable large-scale brain networks using measures of functional connectivity. These include the frontal-parietal network, dorsal attentional network, ventral attentional network, visual network, limbic network, sensorimotor network, and the default mode network (B. T. Yeo et al., 2011). Most of these networks also demonstrated connections with the cerebellum that were proportionally represented in the cerebellum relative to the extent in which each network was represented within the cerebrum (Buckner, Krienen, Castellanos, Diaz, & Yeo, 2011). Similar global networks have also been identified in children and appear to develop in a predictable way, although network characteristics change from strong, short-distance modular connectivity in childhood to greater long-range interconnectivity with maturity (Supekar, Musen, & Menon, 2009; Uddin, Supekar, & Menon, 2010; Uddin, Supekar, Ryali, & Menon, 2011). Despite this latter difference, neural networks in both children and adults have been found to be structurally and functionally organized in a "rich club" fashion, such that areas of the brain with a high degree of interconnectivity are also highly interconnected with other areas with a high degree of interconnectivity (Grayson et al., 2014). Understanding the underpinnings of how the brain's neural networks develop thus has implications for understanding neurodevelopmental disorders, including ADHD (Chu-Shore, Kramer, Bianchi, Caviness, & Cash, 2011; Supekar et al., 2009). In fact, there is speculation that neurodevelopmental disorders such as ADHD "may be manifestations of delay or disruption in the development of these short- and long-range connectivity patterns" (Koziol, Budding, &

Chidekel, 2013, p. 29). There is some evidence to support this contention. For example, one recent study found that children with ADHD had small-world neural network properties that evidenced stronger local connectivity and attenuated global interconnectivity as compared with matched controls, suggesting a maturational delay in long-range interconnectivity in children with ADHD (M. Cao, Shu, Cao, Wang, & He, 2014). Lastly, although all seven previously identified networks may have involvement to varying degrees in the cognitive and behavioural phenotypic expression of ADHD, recent studies provide support for the roles of the default mode, reward sensitivity, cognitive control, frontal-parietal, ventral and dorsal attentional, and sensorimotor networks in both the dysexecutive (e.g., attention, working memory, and cognitive control) and motor control aspects of the disorder (Castellanos & Proal, 2012; Cortese et al., 2012; Costa Dias et al., 2015; Koziol, Budding, & Chidekel, 2013; Mills et al., 2012; van Rooij et al., 2015; von Rhein et al., 2015; Wang et al., 2013).

Neuropsychological Functioning

Cognitive disturbances as demonstrated by those with ADHD have not been shown to be unitary in nature and likely involve interacting components that give the impression of specific cognitive deficits (Koziol, Budding, & Chidekel, 2013). However, neuropsychological research has been able to identify several cognitive features of ADHD that do not appear to be accounted for by psychiatric comorbidity (Seidman et al., 1995). Widely studied examples of cognitive disturbances found in those with ADHD include executive dysfunction (e.g., relatively weaker abilities in inhibition, organization, and planning), learning problems, motor skill deficits, variability of performance (e.g., reaction time and task-specific intraindividual variability), weaker working memory

performance (e.g., auditory and visual working memory), and timing deficits (Alderson, Hudec, Patros, & Kasper, 2013; Alderson, Kasper, Hudec, & Patros, 2013; Antonini, Narad, Langberg, & Epstein, 2013; Borella, Chicherio, Re, Sensini, & Cornoldi, 2011; Chiang, Huang, Gau, & Shang, 2013; Dovis, Van der Oord, Wiers, & Prins, 2013; Goth-Owens, Martinez-Torteya, Martel, & Nigg, 2010; Jacobson, Ryan, Denckla, Mostofsky, & Mahone, 2013; Karalunas, Huang-Pollock, & Nigg, 2012; Kasper, Alderson, & Hudec, 2012; Noreika, Falter, & Rubia, 2013; Pazvantoglu et al., 2012; Roberts, Milich, & Fillmore, 2012; Salum et al., 2014; Schreiber, Possin, Girard, & Rey-Casserly, 2014; Thaler, Bello, & Etcoff, 2013). It is also well documented that children with ADHD demonstrate difficulties with what some term lower-level executive functions, particularly processing speed as measured by tasks of organized visual search and graphomotor (i.e., handwriting) speed (Calhoun & Mayes, 2005; Mayes, 2006; Mayes & Calhoun, 2004; Mayes, Calhoun, Chase, Mink, & Stagg, 2009; Mayes, Calhoun, & Crowell, 1998; Salum et al., 2014). Germane to the present study are neurocognitive differences related to executive functioning (EF), learning, and motor control.

Executive functioning and cognitive control. In its current form, the concept of executive functioning is rather nebulous, with at least 18 different definitions found within the literature whose subcomponents can further be divided into even more basal and interrelated processes (Koziol, Budding, & Chidekel, 2013; Wasserman & Wasserman, 2012, 2013). There is also little evidence neuroanatomically that even subcomponents of EF represent unitary constructs, noting that different brain networks are recruited during tasks that purport to measure the same construct, such as is seen in tasks of response inhibition (Criaud & Boulinguez, 2013; Koziol, Budding, & Chidekel,

2013; Simmonds, Pekar, & Mostofsky, 2008; Stuss, 2011). Nevertheless, understanding the executive dysfunction within the ADHD population is important as research suggests that adaptive limitations in occupational functioning may be mediated by executive dysfunction (Langberg, Dvorsky, & Evans, 2013; Stavro, Ettenhofer, & Nigg, 2007). Further, aspects of executive functioning have been shown to represent endophenotypes and risk factors for the development, persistence, and severity of ADHD-related symptomatology (Arnett, Macdonald, & Pennington, 2013; McAuley, Crosbie, Charach, & Schachar, 2014; C. J. Miller, Miller, Healey, Marshall, & Halperin, 2013; M. Miller, Ho, & Hinshaw, 2012; M. Miller, Loya, & Hinshaw, 2013; Pauli-Pott, Dalir, Mingebach, Roller, & Becker, 2014; Petersen et al., 2013; Rajendran, Rindskopf, et al., 2013; Rajendran, Trampush, et al., 2013; Robinson & Tripp, 2013; van Lieshout, Luman, Buitelaar, Rommelse, & Oosterlaan, 2013).

Given the difficulty that children with ADHD have in inhibiting prepotent responses and ignoring competing environmental influences, behaviourally defined attentional deficits may be better characterized as deficits in response inhibition (i.e., cognitive control) that negatively affect executive function and behavioural output (Barkley, 2006; Koziol & Budding, 2009). Consistent with this view is a hybrid model of ADHD that integrates aspects of cognitive control, executive functioning, and motor/behavioural control in which the attentional and other core cognitive deficits demonstrated by those with ADHD may be better characterized as disturbed executive functioning (Barkley, 2006). Foremost in this model is the construct of cognitive control, which is posited to have a direct effect on behavioural output (i.e., motor control) and also modify the direct relation between executive functions and motor and behavioural

output. Cognitive control as defined here consists of three abilities: the ability to inhibit responses in which some form of reinforcement is immediately available, the ability to interrupt a reinforcing response pattern, and the ability to persist in a response pattern despite competing stimuli (i.e., interference control) (Barkley, 2006). Proposed executive functions that are negatively affected by deficits in cognitive control are described as sensory-motor-based working memory; verbal working memory representing internalized speech; regulation of affect, motivation, and arousal; and the ability to deconstruct and reconstruct behaviours. In sum, this model posits that faulty cognitive control, over time and with maturation, disturbs the underlying cognitive constructs that set the stage for the subsequent development of executive abilities. This disruption of executive abilities in turn manifests as the deficits in self-governed behaviour as observed in those with ADHD (Barkley, 2006). Recent research appears to support this conceptualization of cognitive control and its relation with executive functioning, its development, and associated deficits in ADHD (de Zeeuw, Weusten, van Dijk, van Belle, & Durston, 2012; Forster, Robertson, Jennings, Asherson, & Lavie, 2014; McAuley et al., 2014; Nigg & Casey, 2005; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Molly A. Nikolas & Nigg, 2013; Oie, Skogli, Andersen, Hovik, & Hugdahl, 2014; Palladino & Ferrari, 2013; Pani et al., 2013; Qian, Shuai, Chan, Qian, & Wang, 2013).

Learning and ADHD. Individuals with ADHD present within the full range of intellectual functioning, with participants from studies suggesting disproportionately lower intellectual functioning likely representing a specific subset of the ADHD population (Barkley, 2006; Biederman, Fried, Petty, Mahoney, & Faraone, 2012). Although much debate exists regarding their assessment and determination, learning

disabilities, which represent unexpected academic underachievement, are a frequent comorbidity in children with psychiatric and behavioural disturbances, and they are particularly common in ADHD (Barkley, 2006; Fletcher, Lyon, Fuchs, & Barnes, 2007; Ponde, Cruz-Freire, & Silveira, 2012). Some estimates place comorbid learning disabilities as occurring in over 70% of children with ADHD, with Disorders of Written Expression being the most commonly identified (Mayes & Calhoun, 2006). Further, when ADHD is comorbid with other psychiatric or behaviour disorders (e.g., in the presence of clinical anxiety, depression, Oppositional Defiant Disorder, or adjustment disorder), there is evidence that the probability of a learning disability increases substantially (Mayes & Calhoun, 2006). Even in the absence of a traditional specific learning disability, ADHD has been characterized by some as a disability of learning (Cutting, Koth, Mahone, & Denckla, 2003). That is, instead of viewing learning difficulties in ADHD solely as an aspect of academic underachievement, children with ADHD have demonstrated difficulties learning and automatizing cognitive and motor skills (Koziol, Budding, & Chidekel, 2013). Early research identified delays in the automatization of simple arithmetic in which children with ADHD most often relied on less automatized calculation strategies (Ackerman, Anhalt, Holcomb, & Dykman, 1986). In this case, whereas typically developing children mostly employed an automatized memory retrieval strategy for arithmetic, children with ADHD most often relied on counting, which represented a less mature, less automatized, and more laborious calculation strategy. In addition, process analyses of word list learning tasks have indicated that susceptibility to retroactive interference negatively affects recall of

previously learned verbal information more in ADHD than in controls (Cutting et al., 2003).

More recently, deficits in implicit, procedural learning (i.e., "the learning of procedures, rules, or skills manifested through performance rather than verbalization" [Zillmer, Spiers, & Culbertson, 2008, p. 234]) have been identified in children with ADHD. In one study, researchers used a serial motor sequence learning task to study implicit learning in children with ADHD (Barnes, Howard, Howard, Kenealy, & Vaidya, 2010). Analyses indicated that children with ADHD demonstrated a variable rate of learning relative to controls as indicated by longer reaction times when completing consistent sequences (i.e., a reduced priming effect) that could not be attributed to poor perceptual-motor abilities. Similar procedural learning differences have been identified in young adults with ADHD. In a study designed to investigate procedural learning over time, young women diagnosed with ADHD were hypothesized to demonstrate reduced learning curves and poorer accuracy within a motor sequence learning paradigm relative to controls (Adi-Japha, Fox, & Karni, 2011). The data yielded several interesting findings, including that 1) control participants demonstrated a significant improvement in speed and accuracy whereas ADHD only demonstrated significant improvement in speed and 2) ADHD participants were significantly less accurate at 24 hour and 2-week post-training follow-ups, even after controlling for comorbid learning disability. These researchers concluded that data supported "the notion of a latent memory consolidation phase in motor sequence learning in individuals with ADHD" (Adi-Japha et al., 2011, p. 1017). Researchers have proposed that delayed skill acquisition as demonstrated by these and other studies (e.g., see Aman, Roberts, & Pennington, 1998; Karatekin, White, &

Bingham, 2009) may occur due to deficits in sustained attention, executive functions, or generally delayed skill acquisition associated with protracted development in those with ADHD (Adi-Japha et al., 2011; Burden & Mitchell, 2005; Lange et al., 2007). Lange and colleagues (2007), for example, suggested that children and adults with ADHD may "have difficulties in skills whose acquisition starts as a [labored] and conscious learning process that becomes automatic following consistent and frequent practice" (p. 256). The complex networks underlying procedural learning and executive processes may provide a neuroanatomical explanation for the learning and automatization deficits seen in children with ADHD (Barnes et al., 2010; Koziol, Budding, & Chidekel, 2013). The cerebellar and frontal-striatal systems, which are affected in those with ADHD, are highly involved in the acquisition of motor skills and thus may explicate why those with ADHD demonstrate relatively greater difficulties in automating motor and cognitive skills (Koziol, Budding, & Chidekel, 2013).

Motor functioning. It is still unclear whether developmental motor milestones are generally delayed in those with ADHD (Barkley, 2006). However, motor problems are often found in the disorder and the pervasive nature of motor problems in ADHD is emphasized by the high comorbidity with Developmental Coordination Disorder (DCD) (between 30% and 50%) and evidence of shared genetic and neurophysiological components (Fliers, Vermeulen, et al., 2009; Kadesjo & Gillberg, 2001; Martin, Piek, & Hay, 2006; Piek, Pitcher, & Hay, 1999). A recent neuroimaging study found similar patterns of reduced functional connectivity within neural circuitry underlying both motor and attentional abilities (e.g., "bilateral inferior frontal gyri, right suparamarginal gyrus, angular gyri, insular cortices, anygdala, putamen, and pallidum") in children with ADHD

and/or DCD (McLeod, Langevin, Goodyear, & Dewey, 2014, p. 571). Other researchers, however, have shown that despite these similarities, motor performance is worse in those with comorbid ADHD and DCD than those with ADHD alone (I. C. Lee, Chen, & Tsai, 2013; Pitcher, Piek, & Barrett, 2002). According to the fifth edition of the Diagnostic and Statistics Manual (DSM-5), DCD is characterized by a delay in the acquisition of developmentally appropriate motor skills that significantly interferes with daily functioning, and whose onset of symptoms begins during early development and cannot better be explained by other disorders (American Psychiatric Association, 2013). Even when ADHD is not in the presence of comorbid DCD, it is clear that children with ADHD demonstrate motor impairments, neurological soft signs, and developmental delays more frequently than the general population (Brossard-Racine, Majnemer, & Shevell, 2011; Chan et al., 2010; Cole, Mostofsky, Larson, Denckla, & Mahone, 2008; Dyck & Piek, 2014; Iwanaga, Ozawa, Kawasaki, & Tsuchida, 2006). Motor coordination problems have been documented as occurring in an estimated 30% to 50% of children with ADHD (Fliers et al., 2008) and appear to persist into adulthood (Lis et al., 2010; Stray et al., 2013). Motor deficits that have been repeatedly identified include poor handwriting; decreased speed and accuracy of complex (but not simple) fine and tactual motor performance; deficits in balance, dexterity, coordination, and gross motor skills; and general inefficiencies in motor control and timing (Chen et al., 2013; Fliers et al., 2008; Harvey et al., 2007; Meyer & Sagvolden, 2006; Piek et al., 1999; Rosch, Dirlikov, & Mostofsky, 2013; Zelaznik et al., 2012). In addition, differences between the motor performance of children with ADHD relative to unaffected children has been shown to worsen as motor task complexity increases (Scharoun, Bryden, Otipkova, Musalek, &

Lejcarova, 2013). Although motor deficits are not specific to ADHD, qualities of specific motor abilities have shown some ability to differentiate the presence of ADHD versus other psychiatric (e.g., pediatric Bipolar Disorder) and neurodevelopmental disorders (e.g., Autism Spectrum Disorder) (Johnson et al., 2013; Mahone et al., 2006; Udal et al., 2009). Despite these significant and replicable findings in the literature, motor problems have traditionally gone under-treated and under-recognized clinically in children with ADHD (Fliers, Franke, et al., 2009).

Handwriting in ADHD and kinematic analysis. Handwriting problems are commonly found in those with ADHD (Barkley, 2006). The volitional control of handwriting is a complex, integrative process involving cognitive, motor, and biophysical aspects of functioning that are organized hierarchically and in parallel in order to produce meaningful visual-spatial output (Plamondon, 1995; Van Galen, 1991). Using a motor program metaphor, high level representations of graphomotor output are retrieved and converted into motor control commands sent to the neuromuscular system that are modified in real-time based on multimodal sensory feedback (Dooijes, 1983; Hepp-Reymond, Chakarov, Schulte-Monting, Huethe, & Kristeva, 2009; Lacquaniti, 1989; R. G. J. Meulenbroek & Van Galen, 1988; Portier, Van Galen, & Thomassen, 1993). The integrative nature of handwriting thus involves an extensive network of central nervous system components, including the primary motor cortex, premotor cortex, supplemental motor area, basal ganglia, cerebellum, language areas, and spinal cord (Plamondon, 1995). As indicated previously, many of these same neural systems associated with handwriting have been implicated in the pathophysiology of ADHD.

Research has consistently shown that the handwriting of children with ADHD can be characterized as impaired, often illegible, and less organized than the handwriting of unaffected children, which can in turn result in low academic achievement (Brossard-Racine, Majnemer, Shevell, & Snider, 2008; Brossard-Racine, Majnemer, Shevell, Snider, & Belanger, 2011). In addition, those with ADHD tend to demonstrate more handwriting errors (e.g., spelling corrections and letter transpositions) compared to those with other neurodevelopmental disorders (Johnson et al., 2013). Interestingly, the poor qualitative writing observed in this population does not appear to be related to purely visual-perceptual or linguistic difficulties, but instead likely involve processes related to the basic parameter setting (e.g., regulation of force, speed, and size of graphomotor movements); motor control; and timing aspects of handwriting (Adi-Japha et al., 2007; Brossard-Racine et al., 2008; Marcotte & Stern, 1997; Rommelse, Altink, Oosterlaan, Beem, et al., 2008; Schoemaker, Ketelaars, van Zonneveld, Minderaa, & Mulder, 2005).

In addition to the qualitative difficulties evidenced in the handwriting of children with ADHD, the use of objective methods to study handwriting movements, such as kinematic analysis, has allowed additional inferences regarding the cognitive, psychomotor, and biophysical processes underlying the graphomotor function in children with ADHD. Kinematic analysis involves the objective quantification of "time changes of position, velocity, and acceleration" (Viviani & Terzuolo, 1982, p. 431). Many technological options are available to perform kinematic analysis, but the use of digitizing tablets to capture handwriting signals has predominated in the field of graphonomic research over the past 30 years (for a review of early graphomotor research, including the use of digitizing tablets, see Graham & Weintraub, 1996). Various

kinematic measures can be derived from parameters of time, acceleration, velocity, pen pressure, and others, to (a) describe abilities related to degree of movement automatization and fluency (e.g., number of changes in acceleration or velocity); (b) quantify the relative decelerations and accelerations of handwriting movements (e.g., simple velocity and acceleration profiles); (c) indicate stability, coordination, and consistency of an individual's handwriting (e.g., jerk); (d) indicate the sharing of processing resources, difficulty of writing trajectories, and presence of dysmetria (e.g., movement time, speed, and velocity profiles); (e) quantify fine motor hypotonia and general proficiency; and (f) indicate the smoothness and efficiency of movements (Mergl, Tigges, Schroter, Moller, & Hegerl, 1999; Phillips, Ogeil, & Best, 2009; Portier & Van Galen, 1992; Schroter et al., 2003; Van Galen, 1991). Procedural aspects of motor functioning in those with ADHD have also been studied using kinematic analyses of graphomotor functioning. Kinematic graphomotor writing fluency (i.e., the degree of graphomotor program automatization) in these instances is often operationalized as the number of changes in direction of velocity or acceleration as recorded by digitizing technology and analyzed by appropriate software. Velocity profiles of fluent, automatized handwriting appear as smooth asymmetrical bell-shaped curves with few changes in velocity/acceleration direction, whereas dysfluent, unautomatized handwriting evinces velocity profiles with multiple "jagged peaks" and many changes in the direction of velocity/acceleration. An analogous measure is that of normalized jerk. See Figures 1 and 2 for examples of fluent versus dysfluent handwriting of the word "hello" written in cursive. In figure 2, however, the word was written with simulated hand tremor.

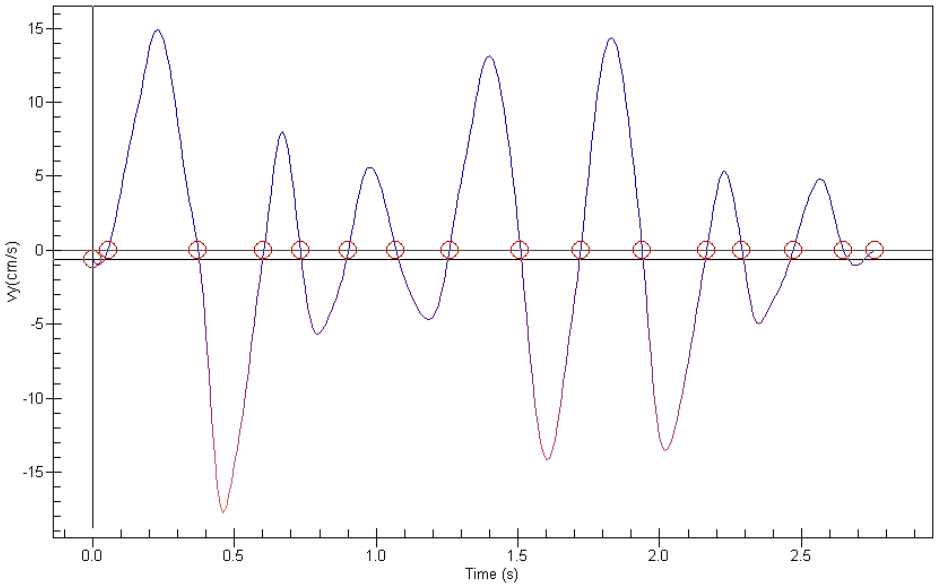

Figure 1. Velocity profile of the word "hello" written fluently.

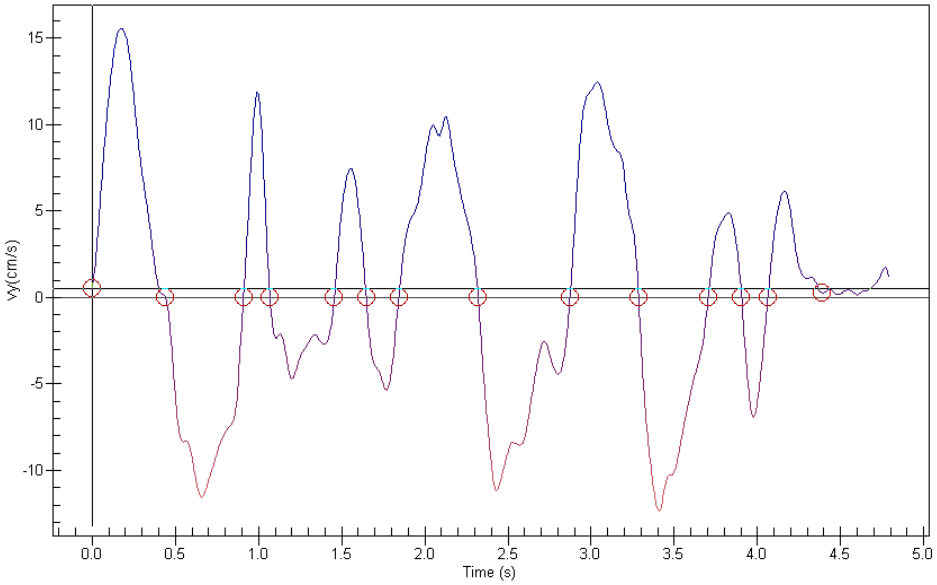

Figure 2. Velocity profile of the word "hello" written with simulated dysfluency.

Kinematic analyses of handwriting performance in the ADHD population have generated additional research questions and demonstrated previously unknown differences in the graphomotor output of this population. For example, children with ADHD who do not have comorbid DCD have demonstrated significantly greater variability in stroke size compared to typically developing children, and other kinematic

aspects such as ballisticity (i.e., degree to which movements can be characterized as sudden and/or bouncing) correlate with ADHD diagnostic symptomatology (Frings et al., 2010; Langmaid, Papadopoulos, Johnson, Phillips, & Rinehart, 2012). A recent kinematic study of adults with ADHD who were counterbalanced on and off stimulant medication identified significantly greater between-trial variability in graphomotor program execution regardless of medication status (Duda, Casey, & McNevin, 2014). Interestingly, this increased variability was only found while learning a novel grapheme and not when executing a putatively automatized grapheme (i.e., writing the word "hello"). Consistent with findings mentioned above, this significantly greater graphomotor fluency variability observed only during a novel graphomotor learning task may indicate differences in automatizing a graphomotor program. Although medication alone does not appear sufficient to remediate all handwriting problems (Brossard-Racine et al., 2015), qualitative handwriting performance tends to improve in children with ADHD after taking stimulant medication (Brossard-Racine et al., 2015; Lerer, Artner, & Lerer, 1979; Tucha & Lange, 2001; Whalen, Henker, & Finck, 1981). Notwithstanding, kinematic analyses assessing the objective, process related aspects of graphomotor functioning have found that the handwriting in these children appears more dysfluent and less automatized when taking stimulant medication versus when they are off medication (Flapper, Houwen, & Schoemaker, 2006; Tucha & Lange, 2001, 2004, 2005). This pattern of fluency and dysfluency related to medication status, however, has not been demonstrated in adults with ADHD (Duda, 2012; Tucha & Lange, 2004). In addition, the kinematic fluency of children with ADHD in these studies was no different from that of unaffected control children when off stimulant medication, and it does not appear that

these findings were due to direct medication effects (Tucha & Lange, 2004). Rather, this decreased fluency and automaticity may be the result of a secondary effect resulting from enhanced attention, from greater cognitive control (Lange et al., 2006; Tucha & Lange, 2001, 2004; Tucha, Paul, & Lange, 2003), or from possibly other cognitive, motor, or psychomotor processes influenced by stimulant medication. For example, other researchers have noted that accuracy is achieved before speed and fluency when learning a complex task (Flapper et al., 2006). In turn, children with ADHD would first need to engage sufficient attentional resources and motor skills for an extended period of time before generating handwriting that is both fluent and accurate. Noting that attentional, learning, and motor skills are often improved in children, adolescents, and adults with methylphenidate treatment (Bart, Daniel, Dan, & Bar-Haim, 2013; Brossard-Racine, Shevell, Snider, Belanger, & Majnemer, 2012; Fox, Adi-Japha, & Karni, 2014; Tucha, Mecklinger, Laufkotter, et al., 2006; Tucha, Prell, et al., 2006), findings of kinematic graphomotor dysfluency during stimulant treatment may indicate a re-calibration of graphomotor programs within the context of improved neurocognitive functioning. Lastly, it is also of interest that research has demonstrated a higher probability of positive stimulant medication response with respect to the diagnostic features of ADHD when problems with motor control are present (Stray, Ellertsen, & Stray, 2010).

Interrelationships between EF, Learning, and Motor Functioning

During the initial development and learning of motor and cognitive skills, the executive-based frontal-striatal system is highly involved in guidance (Leisman, Braun-Benjamin, & Melillo, 2014; Stuss, 2011). Over time, there is a transfer from effortful to automatic processing in which the cerebellum plays a greater role; whether that role

involves motor, executive, learning, affective, or motivational aspects of functioning, as nearly every cortical region possesses reciprocal cerebellar connections (Koziol, Budding, Andreasen, et al., 2013; Koziol, Budding, & Chidekel, 2013; Schmahmann, 2010). Although unanswered questions remain, there appears to be a strong association between the development of both motor and cognitive functioning as indicated by the neuroanatomical associations highlighted above and studies indicating associations between cognitive control and motor skills (Koziol, Budding, & Chidekel, 2013; Leisman et al., 2014; Livesey, Keen, Rouse, & White, 2006). These interrelationships are further indicated by findings demonstrating significant predictive associations between motor skills and later cognitive, social, adaptive, and executive functioning (Piek, Dawson, Smith, & Gasson, 2008; Rigoli, Piek, Kane, & Oosterlaan, 2012; Schoemaker, Lingam, Jongmans, van Heuvelen, & Emond, 2013). For example, a large study involving nearly 7,000 children from the Netherlands between the ages of 7 and 9 years demonstrated that those with more severe motor difficulties in manual dexterity, ball skills, and balance demonstrated greater deficits in social, academic, daily, and cognitive functioning than those defined as having moderate motor difficulties, and both groups performed worse on outcome measures than control children in nearly all domains (Schoemaker et al., 2013).

In the case of ADHD, motor development has also been found to be associated with variability of performance on executive tasks, which is considered by some as a hallmark of ADHD (Barkley, 2006; Klotz, Johnson, Wu, Isaacs, & Gilbert, 2012; Kofler et al., 2013). In addition, significant positive correlations have been found between ADHD diagnostic symptom severity and motor sequelae (Rommelse et al., 2009). Regarding learning and cognitive skills, children with ADHD tend to perform within

normal limits during list learning tasks in the absence of comorbid LD (Vakil, Blachstein, Wertman-Elad, & Greenstein, 2012), but process analyses have demonstrated deficits in delayed recall with intervening trials that implicate executive dysfunction as negatively affecting prior learning (Cutting et al., 2003). Beyond these associations are more specific findings related to cognitive control, learning, and motor skills in ADHD, which typically involve worse performance in these domains relative to peers as cognitive demands increase (Alderson, Hudec, et al., 2013; Alderson, Kasper, et al., 2013; Egeland, Ueland, & Johansen, 2012; Huizenga, van Bers, Plat, van den Wildenberg, & van der Molen, 2009). Qualitative handwriting performance and performance on tests of motor ability, for example, appear to deteriorate as visual and motor integration demands increase (Egeland et al., 2012; Shen, Lee, & Chen, 2012).

Diagnostic Criteria and Assessment of ADHD

The DSM system of understanding psychopathology represents a categorical approach, which is distinct from a dimensional approach that assumes that all aspects of human behaviour lie on a continuum and that pathology represents extremes along the continuum (American Psychiatric Association, 2000). The International Classification of Diseases (ICD) also represents a categorical diagnostic system and uses the term Hyperkinetic Disorder (HKD) as an analogue to ADHD. Despite similarities in symptom profiles and descriptions, HKD is not synonymous with ADHD, noting greatly different prevalence estimates the two sets of criteria yield (S. I. Lee et al., 2008). In the case of ADHD as defined by the DSM, classification and criteria have largely been based on (a) multi-determined clinical observations and informant reports that possess no consistent reference points to quantify abnormality or impairment, (b) groupings of symptoms with

no theoretical underpinnings of neuropathology resulting in widely heterogeneous presentations, and (c) criteria or subtypes with questionable validity (e.g., few children can be described as globally or pervasively inattentive, hyperactive, or impulsive as required by criteria) (Koziol, Budding, & Chidekel, 2013; Koziol & Stevens, 2012; Licht & Tryon, 2009; Wasserman & Wasserman, 2012; Willcutt et al., 2012). Nevertheless, based on DSM-IV criteria, a diagnosis of ADHD is indicated when an individual demonstrates at least six symptoms of inattention and/or at least six symptoms related to hyperactivity and impulsivity that persist for at least 6 months, symptoms cause functional impairment that is inconsistent with normal development, symptoms occur before the age of seven, and symptoms occur in two or more settings (e.g., school, home, and/or workplace) (American Psychiatric Association, 2000). The newest version of the DSM (i.e., the DSM-5) was published in 2013 and retained most of the diagnostic characteristics of ADHD that were included in the DSM-IV. However, differences include explicit reclassification of ADHD as a neurodevelopmental disorder, relaxed diagnostic criteria pertaining to adults that will likely increase identification of adult ADHD (e.g., fewer needed criteria, presence of symptoms in childhood versus impairment), the removal of Autism as an exclusionary criteria, and the downgrade of subtypes of ADHD to presentations, noting that symptom presentation often changes over time (American Psychiatric Association, 2013; Molly A. Nikolas & Nigg, 2013; Sibley et al., 2012; Taylor, 2013).

Assessment and subsequent diagnosis of ADHD currently rely on psychological interview, behavioural observations, and rating scales that are completed by multiple informants (Barkley, 2006). Interviews with the client and appropriate informants are

important to gain additional information pertaining to social, medical, and occupational functioning to place the individual's current situation in context and to understand if there are additional factors that may be contributing to the individual's presentation. Rating scales used in the assessment of ADHD typically include a combination of broadband assessments of psychopathology (e.g., internalizing versus externalizing symptoms) and those geared toward identifying behaviours specific to the diagnosis of ADHD (Barkley, 2006). Broadband measures can be especially important as they can (a) identify problems that are not necessarily diagnostic of ADHD but still significantly affect the individual's daily functioning and (b) help discriminate those with and without ADHD (Harrison, Vannest, & Reynolds, 2011; Shimoni, Engel-Yeger, & Tirosh, 2012). Using information gathered from a combination of broadband and diagnostic rating scales based on multiple informants has demonstrated impressive sensitivity and specificity in diagnosing and ruling-out ADHD, which is intuitive given that ADHD is a behaviourally defined disorder and these rating scales pertain specifically to those behaviours in question (Vaughn & Hoza, 2012).

Research investigating the neuropsychological functioning of those with ADHD has yielded some patterns that are often observed in the disorder, particularly within the large umbrella domain of executive functioning (Barkley, 2006). However, the overall neuropsychological profile of those with ADHD appears to be one of variability (Doyle, Biederman, Seidman, Weber, & Faraone, 2000; Wasserman & Wasserman, 2012). For example, some have estimated that deficits in cognitive control may be present in 35% to 50% of those with the combined subtype of ADHD, but this may represent one of various dysexecutive patterns that corresponds to only a subset of the disorder, which in turn

makes differentiation between those with and without ADHD difficult (Huang-Pollock, Karalunas, Tam, & Moore, 2012; Nigg et al., 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Further, executive dysfunction, including disinhibition and attention deficits, is not specific to ADHD (Goldstein & Reynolds, 2011; Mahone & Schneider, 2012; Wasserman & Wasserman, 2012).

Poorer performances on combinations of neuropsychological tests or tests with multiple components have yielded improved ability to differentiate those with ADHD from typically developing individuals, but these findings mostly demonstrated reduced performance rather than truly deficient performance and less than ideal sensitivity and specificity (Cassuto, Ben-Simon, & Berger, 2013; Englund, Decker, Allen, & Roberts, 2013; Hinshaw, Carte, Sami, Treuting, & Zupan, 2002; Mayes & Calhoun, 2007; Rubia, Smith, & Taylor, 2007). Taken together, neuropsychological test performance has yielded limited ability to identify ADHD as defined behaviourally, and studies have demonstrated mixed results regarding acceptable levels of either sensitivity or specificity, regardless of DSM defined ADHD subtype (Abreu et al., 2013; Doyle et al., 2000; Hinshaw et al., 2002; Munkvold, Manger, & Lundervold, 2014; Nigg et al., 2005; Molly A. Nikolas & Nigg, 2013; Wasserman & Wasserman, 2012; Willcutt et al., 2005). Part of this difficulty may be due to the fact that performances on neuropsychological tests are largely multi-determined with dynamic recruitment of various brain regions despite the appearance of measuring a unitary cognitive construct (Koziol & Budding, 2009; Koziol, Budding, & Chidekel, 2013), but poor predictive ability is also likely a result of ADHD being a behaviourally defined disorder rather than a cognitively defined disorder (Koziol & Stevens, 2012; Wasserman & Wasserman, 2012). This latter point is further emphasized

when noting that although ADHD is currently defined behaviourally and identified via rating scales, behaviour rating scales alone do not provide sufficient information regarding whether or not a child has deficits in subcortical circuit functioning (which are implicated in the disorder) or if these particular individuals will respond to pharmacological treatment (Carmichael et al., 2015). Neuropsychological tests have also traditionally demonstrated limited ecological validity, which further reduces their diagnostic utility (Koziol, Budding, & Chidekel, 2013; Torralva, Gleichgerrcht, Lischinsky, Roca, & Manes, 2013). As such, the field of neuropsychology is currently in need of test batteries that will tap into the foundational components of ADHD within a neuroanatomically informed framework to include tests of reward sensitivity, tests quantifying procedural learning, developmentally-oriented motor functioning batteries that include aspects of neurological soft signs, and tests more specifically associated with aspects of executive functioning (e.g., self-regulation, emotional response inhibition, and cognitive control) that are tied to current knowledge of neuroscience (Carmichael et al., 2015; Fosco, Hawk, Rosch, & Bubnik, 2015; Koziol, Budding, & Chidekel, 2013; Wasserman & Wasserman, 2012). Even if not diagnostic of the disorder, however, neuropsychological measures provide valuable information regarding the individual's functioning that are not provided by rating scales or interview that help facilitate treatment implementation (DeBono et al., 2011; Pritchard, Koriakin, Jacobson, & Mahone, 2014; Toplak, West, & Stanovich, 2013).

Noting the longstanding limitations of categorical diagnosis of psychopathology in general and the weaknesses of the DSM in particular, the National Institute of Mental Health (NIMH) developed the Research Domain Criteria (RDoC), which are part of a

strategy to create objective, dimensionally-based neurobiological measures (i.e., biomarkers) for the description and subsequent identification of psychopathology (Insel et al., 2010). Further, the RDoC espouses a neurodevelopmental framework in which understanding typical developmental trajectories, sensitive periods or limited windows of development, and the continuous interaction between the environment and neurobiological systems (e.g., timing of injury or trauma relative to brain development) become pivotal to understanding the development, maintenance, and treatment of psychopathology and neurodevelopmental disorders such as ADHD (Casey, Oliveri, & Insel, 2014). The field of neuropsychology is thus well positioned to respond to the RDoC strategy, as understanding brain-behaviour associations is the purview of neuropsychology (Koziol, Budding, & Chidekel, 2013) and neuropsychological assessment has already been demonstrated to facilitate endophenotypic research in ADHD, particularly when considering measures of response inhibition, reaction time variability, and temporal processing (Henriquez-Henriquez et al., 2014; M. A. Nikolas & Nigg, 2015; Rommelse, Altink, Martin, Buschgens, Buitelaar, et al., 2008; Rommelse, Altink, Martin, Buschgens, Faraone, et al., 2008; Rommelse, Altink, Oosterlaan, Beem, et al., 2008; Rommelse, Altink, Oosterlaan, Buschgens, et al., 2008).

Neuroimaging techniques such as fMRI, PET, and DTI have traditionally been unable to demonstrate adequate sensitivity or specificity in identifying/ruling-out ADHD or other psychiatric disorders, and are thus unsuitable for clinical, diagnostic decisions (Weyandt, Swentosky, & Gudmundsdottir, 2013). However, recent advances in neuroimaging and statistical techniques have allowed the analysis of large-scale brain networks as a means to identify potential biomarkers for psychopathology and correctly

classify those with and without a particular disorder, including ADHD (Deco & Kringelbach, 2014). One study using combined measures of functional connectivity, fractional amplitude of low-frequency fluctuation (fALFF, which "reflects the 'energy' of the BOLD signal at each voxel' [p. 2]), and regional homogeneity (ReHo, which characterizes the degree of synchronization between local neuronal units) achieved over 76% diagnostic accuracy and sensitivity and specificity of 63.27% and 85.11%, respectively, in a database sample of 101 children with ADHD and 144 healthy controls (Cheng, Ji, Zhang, & Feng, 2012). Consistent with earlier imaging research, the underlying networks that differentiated the groups primarily involved frontal and cerebellar networks. Other research using similar methodologies but controlling for artifacts such as movement during scan have achieved even greater predictive accuracy that varied based on ADHD subtype; the best procedure demonstrated 82.7% classification accuracy, 78.9% sensitivity, and 86.5% specificity for the primarily inattentive type of ADHD (ADHD-PI) (Fair et al., 2013). Other imaging studies have been able to correctly classify those with ADHD versus unaffected peers with varying, yet lower levels of accuracy, sensitivity, and specificity (Colby et al., 2012; Dai, Wang, Hua, & He, 2012; Dey, Rao, & Shah, 2012). Other recently investigated biomarkers that have been found to be associated with, but not yet diagnostic of ADHD, include parasympathetic and sympathetic activity (Musser, Galloway-Long, Frick, & Nigg, 2013), oculomotor measures during attention tasks (Fried et al., 2014), EEG activity (Kim et al., 2015; Mazaheri et al., 2014), and peripheral levels of MAO (monoamine oxidase), NE (norepinephrine), MHPG (3-Methoxy-4-hydroxyphenylethylene glycol), Zn (zinc), ferrintin, and cortisol (Scassellati, Bonvicini, Faraone, & Gennarelli, 2012).

The Present Study and Hypotheses

Still lacking in the ADHD literature are studies that examine the effects of varying the level of cognitive control demands on graphomotor fluency using animated stimuli on a digitizing tablet. Although studies have investigated the development of graphomotor fluency in typically developing individuals, children with DCD, and adults with ADHD (Chang & Yu, 2010; Duda, Casey, & McNevin, 2015; Portier & Van Galen, 1992; Rueckriegel et al., 2008; Zesiger, Mounoud, & Hauert, 1993), no study has investigated this development in children with ADHD, and none has investigated possible neuropsychological factors that best predict this development. Neuropsychological testing has lacked sufficient sensitivity and, particularly, specificity in identifying ADHD, which may be due at least in part to a lack of motor skill and procedural learning tests that tap into the motor and executive neuropsychological sequelae associated with ADHD. In turn, there is a need for research investigating alternative methods of assessing motor problems in ADHD, which include automatization deficits seen in the disorder.

To address these issues, the present study included two parts and five studies. Part I, Study 1 was designed to determine which kinematic research paradigms would elicit the greatest effects of cognitive control demands on graphomotor fluency such that only two of the tested eight task types would be selected for Part II. Increased cursor tracing/following time and path complexity combined with task performance demands were expected to require increased cognitive control requirements and thus elicit reduced graphomotor fluency. The methodologies in Part II were designed to determine the following: Study 2 – the effects of increased cognitive control on kinematic graphomotor fluency in children and adolescents with ADHD relative to controls; Study 3 – whether

learning a novel grapheme (i.e., a new graphomotor program) is attenuated in children and adolescents with ADHD who have discontinued stimulant medication; Study 4 – which neuropsychological abilities are related to, and best predict improvement in graphomotor fluency; and Study 5 – the predictive ability that relative change in graphomotor fluency has in identifying children with and without ADHD. The following were specific hypotheses for each study in Part II:

Hypotheses **Part II, Study 2.** Two findings were expected. First, increased requirements of cognitive control were predicted to negatively affect graphomotor fluency in all participants (e.g., see Tucha et al., 2003). Second, given the nature of ADHD as a disorder of executive functioning and as could be predicted by Barkley's (2006) hybrid model of ADHD, the graphomotor fluency of participants with ADHD was expected to be significantly different from that of control participants as cognitive control demands increased (i.e., an interaction effect between group membership and cognitive control requirements).

Hypothesis **Part II, Study 3.** Motor and learning problems have been demonstrated in children and adults with ADHD. More specifically regarding graphomotor performance, recently published research indicates that graphomotor fluency development within the context of learning a new grapheme appears to be attenuated in adults with ADHD (Duda et al., 2015). As such, a significant group by practice interaction effect was predicted in which children and adolescents with ADHD would demonstrate a reduced ability to automatize a newly learned grapheme relative to their unaffected peers, and despite being given the same number of practice trials.

Hypothesis **Part II, Study 4.** Research with typically developing individuals has demonstrated that verbal abilities strongly correlate with kinematic aspects of handwriting, in which stronger verbal abilities relate to better performance (Mergl et al., 1999). In addition, measures of executive functioning and fine motor skills have been found to predict graphomotor fluency performance in children (Noda et al., 2013). As such, all three of these neuropsychological constructs were expected to be associated with graphomotor fluency while learning a new grapheme, and each was expected to have predictive value related to relative improvement in graphomotor fluency with practice. Given the current lack of research in this particular area, no a priori hypothesis was salient regarding which constructs would most strongly predict graphomotor fluency improvement.

Hypothesis **Part II, Study 5.** Historically, neuropsychological tests have not demonstrated adequate predictive ability, sensitivity, or specificity with regard to identifying ADHD. However, most neuropsychological tests attempt to tap more unitary cognitive constructs that by themselves may not be sensitive to the integrated nature of neuropsychological sequelae associated with ADHD. Noting this and given the diverse and integrative nature of handwriting – which involves verbal, executive, motor, and procedural learning abilities – relative change in graphomotor fluency was expected to demonstrate adequate ability to classify participants as ADHD or non-ADHD as demonstrated by moderate sensitivity and specificity (i.e., area under the curve [AUC], sensitivity, and specificity $\geq .70$). Additional support for this contention is provided by research indicating some ability for relative change in graphomotor fluency to identify

adults with ADHD (Duda, Casey, & Millis, 2014). See Table 1 for an outline describing each study.

Table 1

Description of each Study and Corresponding Hypothesis

	Study Description and Aim	**Hypothesis**
Part I		
Study 1	Determine which 2 of 8 graphomotor paradigms conducted on a digitizing tablet would elicit the greatest and least graphomotor dysfluency. These two tasks would in turn be used in Part II, Study 2. The task eliciting the greatest dysfluency would represent the "high" cognitive control task whereas the task eliciting the least dysfluency would represent the "low" cognitive control task.	Increased tracing time and figure complexity combined with task performance requirements would elicit the greatest cognitive control demands and result in the most dysfluency.
Part II		
Study 2	Using the two tasks determined in Part I, Study 1, examine the effects of increased cognitive control demands on the kinematic graphomotor fluency of child and adolescent participants with and without ADHD (within the context of discontinued use of stimulant medication).	Both groups were expected to produce greater dysfluency during the high cognitive control tasks, although an interaction was predicted in which participants with ADHD were expected to be significantly more affected and demonstrate even greater dysfluency than controls.
Study 3	By writing a novel grapheme on a digitizing tablet 30 times, examine the development of a novel graphomotor program in child and adolescent participants with and without ADHD.	Control participants would demonstrate significantly greater improvements in fluency with practice.
Study 4	Determine which neuropsychological constructs (i.e., verbal ability, processing speed, or fine motor skills) would best predict the improvement in graphomotor fluency observed in Study 3.	As this is exploratory in nature, no a priori hypothesis was proposed, although all variables were expected to be associated with graphomotor fluency improvement.
Study 5	Examine the potential predictive ability that relative change in graphomotor fluency may have in identifying children with and without ADHD.	Proportion of change between the beginning and end of practice was predicted to have moderate predictive ability, sensitivity, and specificity with regard to the classification of ADHD (i.e., AUC, sensitivity, and specificity ≥ 0.70)

Chapter 2: Method – Part I, Study 1

Participants

Power analysis ([1 – β] = .80) using G*Power software (Buchner, Erdfelder, Faul, & Lang, 2009) indicated that using the proposed methodological design and statistical analyses, 81 participants were needed to detect a statistically significant difference (α = .05) of small effect size ($\omega^2_{partial}$ = 0.02). Although child and adolescent controls would have been ideal for Part I pilot study data collection, the large number of participants required for adequate statistical power did not make this viable. As such, a convenience sample of university students was recruited for Part I, Study 1.

Following ethics clearance, undergraduate student participants were recruited through the University of Windsor's Psychology Participant Pool. In order to minimize confounds related to extraneous visual and/or motor disturbances, participants included only those with normal (or corrected-to-normal) vision, and no existing neurological condition affecting graphomotor performance (e.g., cerebral palsy affecting the upper extremities, tendinitis, or carpal tunnel syndrome). Results were monitored throughout data collection and due to graphically obvious task-based graphomotor fluency differences, data collection was discontinued after data were collected from 76 participants. As compensation for completing the study, participants received 1.5 bonus points towards their final grade of a qualifying course based on participation time of approximately 90 minutes.

Materials and Apparatus

A WACOM Cintiq 21UX digitizing tablet was used to record the handwriting movements of participants. The digitizing tablet has an active display area of 17" by

12.75" and spatial resolution of 5080 lines per inch. The tablet provided real-time on-screen visual feedback using a special non-inking pen. MovAlyzeR software (NeuroScript, LLC; Tempe, AZ, USA) was utilized to quantify handwriting movements with a maximum sampling rate of 200 Hz, and x-y coordinates were low-pass filtered at 12 Hz. Handwriting movements were broken down by MovAlyzeR software into strokes using interpolated vertical velocity zero crossings. In this sense, a stroke, representing a "unit" of handwriting, can be defined as "a segment bounded by time moments at which the vertical component of the velocity changes sign" (Teuber, Thomassen, & Van Galen, 1983, p. 168). The digitizing tablet was calibrated and accuracy maximized according to MovAlyzeR software protocol (NeuroScript, LLC; Tempe, AZ, USA).

Normalized Jerk (NJ) was the kinematic dependent variable (DV) of interest and was derived using MovAlyzeR software. NJ is a measure of writing smoothness and fluency and represented the operational definition of degree of graphomotor procedural learning and automatization. In addition, automatization was conceptualized as a continuous variable occurring on a continuum of automaticity and not a dichotomous construct in which performance was either "fluent/automatized" or "dysfluent/non-automatized." NJ values can theoretically range between 0 and infinity. The NJ variable is similar to the dysfluency measure of "number of inversions of acceleration/velocity" used in much of the research utilizing kinematics to investigate graphomotor problems in those diagnosed with ADHD (Flapper et al., 2006; Schoemaker et al., 2005; Tucha & Lange, 2001, 2004, 2005; Tucha, Mecklinger, Laufkotter, et al., 2006; Tucha et al., 2003; Tucha, Prell, et al., 2006) in that NJ "is the change of acceleration per time" (Teulings et al., 1997, p. 160). NJ, however, has the advantage of allowing comparisons of words or

symbols of varying size and movement durations because it is normalized (Teulings, Contreras-Vidal, Stelmach, & Adler, 1997). High NJ scores indicate more dysfluent movement and low NJ scores indicate smoother, fluent, and more automatized movement (Teulings et al., 1997; Yan, Rountree, Massman, Doody, & Li, 2008). As one practices a grapheme, graphomotor fluency and automatization increase as indexed by *lower* values of NJ (Portier & Van Galen, 1992; Hanneke van Mier, Hulstijn, & Petersen, 1993) because these lower values suggest fewer "stops and starts" during movement production and better sensorimotor coherence.

Due to the experimental nature of this measure and variability resulting from different data-capturing tools (e.g., monitor-style digitizing tablets versus hand-held digitizing tablets, visual feedback with trace using non-inking pens versus use of inking pens or pens with no visual feedback) and individual computer processing differences, there currently exists no systematically determined reliability or validity data for this specific tool. However, dozens of studies with various clinical populations (e.g., ADHD, Parkinson's Disease, DCD, and other disorders) have yielded replicable results using kinematic analysis (for examples, see Chang & Yu, 2010; Gangadhar et al., 2009; Schoemaker et al., 2005; Smits-Engelsman, Wilson, Westenberg, & Duysens, 2003; Tucha & Lange, 2001, 2004; Tucha, Mecklinger, Thome, et al., 2006).

Current and childhood ADHD symptomatology was acquired via participant self-report using the Barkley Adult ADHD Rating Scale-IV (BAARS-IV; Barkley, 2011). Based on DSM-IV diagnostic criteria, the BAARS-IV is a self-report questionnaire designed to evaluate current and/or childhood ADHD symptoms in adults between the ages of 18 and 81 years. The normative sample of the BAARS-IV consists of 1,249

adults between the ages of 18 and 96 and closely approximates the U.S. adult population from the 2000 U.S. Census regarding "regional distribution, sex, race/ethnic group, marital status, employment status, total household income, and education" (Barkley, 2011, p. 14). According to the manual, ratings are considered clinically significant if scores are at or above the 93rd percentile in the domains of inattention, hyperactivity, or impulsivity. The BAARS-IV has satisfactory reliability as indicated by high internal consistency (Cronbach's α = .92 and .95 for current and childhood ADHD symptom scores, respectively) and two- to three-week test-retest reliability (.75 and .79 for current and childhood ADHD symptom scores, respectively). According to the technical manual, the BAARS-IV also has good validity as evident from factor analyses, correlations with current ADHD measures that are psychometrically robust, and group comparisons demonstrating concurrent validity with various other assessment instruments.

All demographic and research data were kept confidential and in secure locations. Participant data were recorded in separate encrypted files, one of which contained specific identifying information (i.e., participant name, participant ID, contact information, appointment date, appointment time, and the date in which data were to be anonymized) and the other of which contained research data (i.e., participant ID, demographic information, and research data). However, the participant's name and participant ID were only held within the first spreadsheet for one week, after which time these data were deleted. Keeping these separate spreadsheets with identifying information attached to research data afforded participants the opportunity to remove their data from the study during this time if they chose to do so. After one week, the link connecting identifying personal information with demographic and research data was removed,

which effectively anonymized the data such that only arbitrary participant identification numbers were associated with research data. All paper forms were de-identified (i.e., coded with a randomly assigned participant identification) and stored within a locked room and filing cabinet.

Procedure

All data for each participant were collected in one session. Participants took part in an interview with the researcher to acquire appropriate demographic information (i.e., age, handedness, sex, and first language), medical information (e.g., information to screen for vision-related and/or neurological problems affecting the ability to perform the tasks), and complete the BAARS-IV self-report measure.

To become familiarized with using the digitizing tablet and pen, all participants were allowed to write their name on the digitizing tablet three times. This also allowed the researcher the ability to emphasize using and holding the pen "naturally" and in this same manner throughout the experiment. All participants were given the opportunity to manipulate the tablet and chair to a comfortable writing position. All instructions were given orally and short written instructions appeared on the digitizing tablet throughout the duration of the experiment.

Prior to engaging in practice trials of the cognitive control tasks, the appearance and action of the stimuli on the tablet, as well as task demands, were described to each participant. Participants were then given the opportunity to practice one trial each of the seven of eight possible tasks in order to become familiar with those tasks. Order of practice administration for each of these seven tasks was as follows: Free Hand, Slow Linear Pattern, Moderate Linear Pattern, Fast Linear Pattern, Slow Wave Pattern,

Moderate Wave Pattern, and Fast Wave Pattern (described in further detail below). See Appendix A for verbiage used to explain the study to all participants.

All cognitive control task stimuli were comprised of a green box on the right hand of the screen labeled "Start" and a red box on the left hand of the screen labeled "End." In all eight possible tasks, participants moved from their right to their left. Seven cognitive control tasks also included a stimulus element consisting of a thick black bar (termed the "Cursor") that moved across the screen at three different speeds (slow, moderate, and fast) and in two possible patterns: a straight line (i.e., the Linear Pattern task) or a wavy line (i.e., the Wave Pattern task). Participants were instructed to place the pen at the center of the green box (Start) as soon as the Cursor appeared on the screen. After placing the pen at the center of the green Start box, the participants followed the Cursor as closely as possible at its midpoint - without touching it or going past it - until they reached the center of the red End box. As soon as they reached the center of the red box, they were instructed to lift their pen and move to the starting position - without touching the pen to the tablet - to wait for the next task or trial.

The linear distance traced for each cognitive control task was the same. However, the time to complete each task varied by task type to include Free Hand and 2-second ("fast" task speed), 4-second ("moderate" task speed), and 6-second ("slow" task speed) interval tasks. Combining features of speed and pattern resulted in the creation of eight specific tasks and all participants completed six trials of each task. The eight tasks conducted by all participants included (1) a Free Hand task in which participants connected the center of the green box to the center of the red box while moving as quickly as possible and while comfortably maintaining accuracy; (2) a Slow Linear

Pattern task in which participants followed the Cursor moving in a straight line at a 6-second interval; (3) a Moderate Linear Pattern task in which participants followed the Cursor moving in a straight line at a 4-second interval; (4) a Fast Linear Pattern task in which participants followed the Cursor in a straight line at a 2-second interval; (5) a Slow Wave Pattern task in which participants followed the Cursor in a Wave pattern at a 6-second interval; (6) a Moderate Wave Pattern task in which participants followed the Cursor in a Wave pattern at a 4-second interval; (7) a Fast Wave Pattern task in which participants followed the Cursor moving in a Wave pattern at a 2-second interval; and (8) a Random task in which participants performed Slow, Moderate, and Fast Linear and Wave Pattern tasks one time each in a randomly presented sequence. See Figures 3, 4, and 5 for examples of Free Hand, Linear, and Wave pattern task appearance, respectively.

Figure 3. Configuration of starting and ending points of the Free Hand Task. Note that there was no cursor to follow for this task and participants connected the boxes in a straight line at a self-determined pace.

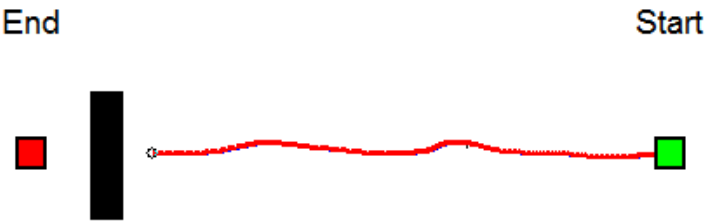

Figure 4. Sample trace of Linear Pattern task, showing the start point, end point, and linear path of the cursor that was followed by participants.

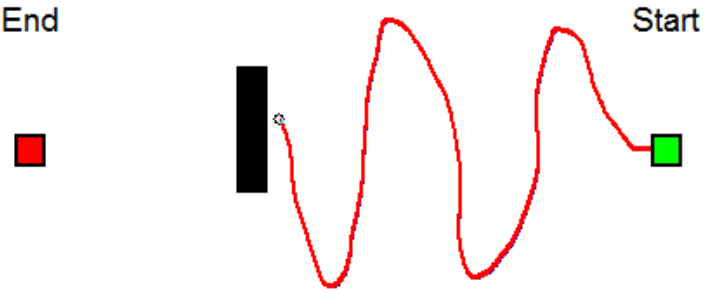

Figure 5. Sample trace of Wave Pattern task, showing the start point, end point, and Wave pattern of the cursor that was followed by participants.

Due to the extremely large number of possible permutations for the eight different cognitive control tasks (i.e., 40,320 possible order permutations), order of administration could not be completely counterbalanced. To control for order effects, a form of the Latin squares method was used to create 24 different Order Set combinations of the cognitive control tasks. See Tables 2 and 3 for details regarding Item and Order Set creation, their association with the eight different task types described above, and descriptive statistics for each Order Set combination used within the study.

Table 2

Task Set and Sequence Combination Descriptions

Task Set	Description	Sequence	Sequence Description
I	Free Hand	a	Slow → Moderate → Fast
II	Linear	b	Slow → Fast → Moderate
III	Wave	c	Moderate → Fast → Slow
IV	Random	d	Moderate → Slow → Fast
		e	Fast → Slow → Moderate
		f	Fast → Moderate → Slow

Note. Task Sets II and III combine with sequence to represent order and task type performed and the resulting Order Set Number (See Table 3 below). For example, combining Task Set II with Sequence "a" (i.e., IIa) indicates performance of the Slow Linear (Task 2), Moderate Linear (Task 3), and Fast Linear (Task 4) tasks, in that order. Combining Task Set III with Sequence "a" (i.e., IIIa) indicates performance of the Slow Wave (Task 5), Moderate Wave (Task 6), and Fast Wave (Task 7) tasks, in that order.

Table 3

Order Sets and Descriptive Statistics

Order Set #	Sequence	Frequency Used	Order Set #	Sequence	Frequency Used
1	I, IIa, IIIa, IV	4	13	I, IIb, IV, IIIb	3
2	IIa, I, IIIc, IV	4	14	IId, I, IV, IIId	3
3	IIIa, I, IIa, IV	4	15	IIId, I, IV, IId	3
4	IV, I, IIe, IIIe	4	16	IV, I, IIIf, IIf	3
5	I, IIIa, IIb, IV	3	17	I, IIIb, IV, IIa	3
6	IIb, IIIc, IV, I	3	18	IIe, IIId, I, IV	3
7	IIIb, IIc, IV, I	3	19	IIIe, IId, I, IV	3
8	IV, IIe, IIIe, I	3	20	IV, IIf, I, IIIf	3
9	I, IV, IIc, IIIa	3	21	I, I, IIIb, IIb	3
10	IIc, IV, I, IIIc	3	22	IIf, IV, IIId, I	3
11	IIIc, IV, I, IIc	3	23	IIIf, IV, IId, I	3
12	IV, IIIe, I, IIe	3	24	IV, IIIf, IIf, I	3

Chapter 3: Results – Part I, Study 1

All statistical analyses were performed using IBM SPSS Statistics, Version 21. A Repeated Measures ANOVA was conducted to identify statistically significant differences within the mean normalized jerk (NJ) of the eight cognitive control tasks. Linear contrasts were used as follow-up analyses to identify and quantify the tasks that

elicited the greatest and least amount of dysfluency. Higher values of mean NJ represented greater dysfluency (i.e., less automaticity) and lower values represented less dysfluency (i.e., greater automaticity). Due to multiple comparisons (see below), a Bonferonni correction was used, resulting in an adjusted alpha level of .02 to indicate statistical significance. Interpretations of effect sizes using ω^2 and $\omega^2_{partial}$ were based on Kirk's (2003) suggestions due to a lack of effect sizes reported in the literature. As such, effect sizes of 0.010 to 0.058, 0.059 to 0.137, and 0.138 or greater were interpreted to indicate small, medium, and large associations, respectively.

Analysis of Assumptions and Data Cleaning

Individual trials within each task and for all participants were examined for potentially invalid or extremely influential data points. These data points were interpreted as not representative of the typical intraindividual graphomotor fluency performance for a particular participant and were removed based on the following procedure: Trials with NJ values of 0 or >10,000 (if only one such value were present and did not represent the typical performance of the participant) and trials that were deemed invalid by observation. See Table 4 for descriptive statistics of valid number and proportion of valid trials retained for each task type.

Table 4

Descriptive Statistics of Valid Trials by Task Type

Task	% Valid Trials	Mean # Valid Trials	SD # Valid Trials	Range of Valid Trials
Free Hand	93.64%	5.62	0.52	4 to 6
Linear Slow	91.23%	5.47	0.58	4 to 6
Linear Moderate	93.42%	5.61	0.57	4 to 6
Linear Fast	92.32%	5.54	0.55	4 to 6
Wave Slow	93.20%	5.59	0.55	4 to 6
Wave Moderate	95.61%	5.74	0.47	4 to 6
Wave Fast	97.81%	5.87	0.38	4 to 6
Random	98.90%	5.93	0.25	5 to 6
Grand Total	94.50%	45.37	1.73	40 to 48

Note. Six trials conducted per task.

Independence of observations was assumed given the consistent and individual administration of all experimental tasks, the novel nature of the experiment, and the lack of known systematic communication between participants. Homogeneity of variance was not tested noting no between-subject analyses. Regarding the normality of the data, only the mean NJ of the Slow Wave Pattern task was normally distributed. Data distributions and their corresponding dependent variables were non-normal for the remaining seven tasks as indicated by statistically significant Shapiro-Wilk statistics and significant skewness (i.e., skewness values $\geq |2|$) and kurtosis (i.e., kurtosis values $\geq |3|$). In particular, the Free Hand, Linear Slow Pattern, and Linear Fast Pattern tasks were significantly Leptokurtic and positively skewed. Taken together, these analyses of assumptions indicated a violation of the assumption of normality. The assumption of sphericity was also violated as shown by a significant Mauchley's test, $\chi^2(27) = 484.40$, $p < .05$. Mean NJ scores were standardized and 13 data points were identified as outliers (i.e., $\geq |2.5|$ SD). After removal of the 13 outlier data points, normality was improved but the assumptions of normality and sphericity were still violated, $\chi^2(27) = 375.74$, $p < .05$.

The original data set (i.e., prior to the removal of outlier data points) was transformed using the square root of each observation. This transformation improved normality in all but three variables, but significant skewness and kurtosis remained and the assumption of sphericity continued to be violated, $\chi^2(27) = 190.23$, $p < .05$. Transformed mean NJ scores were standardized and eight data points were identified as outliers and subsequently removed. The removal of these outliers resulted in the elimination of any significant skewness or kurtosis on all of the dependent variables although Sharpiro-Wilk statistics were still significant for the Free Hand and Linear Fast Pattern tasks and the assumption of sphericity remained violated, $\chi^2(27) = 125.56$, $p < .05$. Noting the improvements in normality, skewness, and kurtosis associated with data transformation and the removal of outliers, as well as the relatively large sample size, primary data analyses were conducted using transformed variables and after the removal of outliers. In addition, as recommended by Field (2009), degrees of freedom for the following repeated measures ANOVA were corrected using Greenhouse-Geisser estimates noting (a) the violation of the assumption of sphericity and (b) a Greenhouse-Geisser correction ε (epsilon) of 0.620, which is closer to 1 than the lower limit of ε (lower-bound = 0.143). See Tables 5 and 6 below for analyses of assumptions before and after data transformations, respectively.

Table 5

Analyses of Assumptions Prior to Data Transformation

	With Outliers (*N* = 76)				Without Outliers (*N* = 67)			
Task (DV = Mean NJ)	**Skewness**	**Kurtosis**	**Shapiro-Wilk**	**Mauchley's Sphericity (*p*)**	**Skewness**	**Kurtosis**	**Shapiro-Wilk**	**Mauchley's Sphericity (*p*)**
Free Hand	***7.12***	***56.76***	***<.001***	***<.001***	***2.03***	***4.13***	***<.001***	***<.001***
Linear Slow	1.74	***3.73***	***<.001***		1.16	1.28	***<.001***	
Linear Moderate	1.00	0.80	***<.001***		1.04	0.91	***<.001***	
Linear Fast	***5.24***	***33.53***	***<.001***		***2.68***	***8.66***	***<.001***	
Wave Slow	0.48	0.34	.14		0.11	-0.66	.27	
Wave Moderate	0.84	0.31	***<.001***		0.74	0.17	***<.001***	
Wave Fast	0.51	-0.48	***.02***		0.62	-0.23	***.02***	
Random	0.96	1.21	***<.001***		0.49	-0.33	.11	
Grand Mean	1.03	2.36	***<.001***		0.22	-0.77	.18	

Note. Bold and italicized values within the table represent violations of assumptions (skewness ≥ |2|, kurtosis ≥ |3|, Shapiro-Wilk $p < .05$, and Mauchley's test of sphericity $p < .05$).

Table 6

Analyses of Assumptions Subsequent to Data Transformation

	Data Transformed (N = 76)				Data Transformed & Without Outliers (N = 69)			
Task (DV = Mean NJ)	**Skewness**	**Kurtosis**	**Shapiro-Wilk**	**Mauchley's Sphericity (p)**	**Skewness**	**Kurtosis**	**Shapiro-Wilk**	**Mauchley's Sphericity (p)**
Free Hand	***3.48***	***18.64***	***<.001***	***<.001***	1.12	0.68	***<.001***	***<.001***
Linear Slow	0.66	0.59	0.06		0.35	-0.02	0.47	
Linear Moderate	0.25	-0.32	0.56		0.29	-0.09	0.68	
Linear Fast	***2.78***	***11.64***	***<.001***		1.20	2.11	***<.001***	
Wave Slow	-0.57	0.46	***0.05***		-0.34	0.59	0.29	
Wave Moderate	0.18	0.02	0.09		0.24	0.27	0.09	
Wave Fast	-0.04	-0.58	0.62		-0.06	-0.57	0.63	
Random	0.27	-0.08	0.86		0.05	-0.44	0.96	
Grand Mean	0.37	0.41	0.37		0.10	-0.72	0.38	

Note. Bold and italicized values within the table represent violations of assumptions (skewness ≥ |2|, kurtosis ≥ |3|, Shapiro-Wilk $p < .05$, and Mauchley's test of sphericity $p < .05$).

Participant descriptive information. Participants were primarily right handed by self-report (95%), a majority self-identified as women (86%), and most participants self-identified as Caucasian (70%). Only one participant reported any current chronic or acute medication condition (sleep apnea). Nine participants (12%) reported at least one current psychiatric diagnosis, 24 participants (32%) reported significant ADHD symptomatology on at least one scale of the BAARS-IV (which is a considerably higher proportion of college students reporting significant ADHD symptomatology than has been observed in large studies; see Garnier-Dykstra, Pinchevsky, Caldeira, Vincent, & Arria, 2010), and five participants reported having taken psychoactive medication prior to taking part in the study. Despite this, all participants who reported psychiatric diagnoses, significant ADHD symptomatology, and/or medication use were retained for purposes of the overall analyses noting that diagnoses or medication usage could not be confirmed, there is a lack of research indicating differences related to graphomotor fluency (specifically) in those with the kinds of psychiatric illnesses reported (e.g., see Mergl, Juckel, et al., 2004; Mergl, Mavrogiorgou, Juckel, Zaudig, & Hegerl, 2004; Morrens, Hulstijn, Van Hecke, Peuskens, & Sabbe, 2006; Sabbe, Hulstijn, Van Hoof, & Zitman, 1996), and the BAARS-IV is a screening tool designed to maximize sensitivity in identifying those who might have ADHD, and is thus not diagnostic of the disorder. In addition, while noting that no study has utilized the current methodologies to study graphomotor fluency and direct comparisons cannot be made, medication use (particularly stimulant medication) has not consistently been found to affect graphomotor fluency in adults with ADHD (see Duda et al., 2015 and Tucha & Lange, 2004). See

Table 7 for complete descriptive statistics of participant demographic, psychiatric, and medication information.

Table 7

Participant Descriptive Statistics

		n	*Mean*	*SD*
Handedness	Right	72	-	-
	Left	4	-	-
Sex	Women	66	-	-
	Men	10	-	-
Race/Ethnicity				
	Asian	8	-	-
	Black/African/Caribbean	2	-	-
	Caucasian/European/White	53	-	-
	Hispanic/Latina/Latino	1	-	-
	Middle Eastern	9	-	-
	Multiracial	1	-	-
	Native/Aboriginal	2	-	-
Psychiatric Diagnosis(es)				
	ADHD	2	-	-
	Bipolar Disorder	1	-	-
	Unipolar Depression	1	-	-
	Generalized Anxiety Disorder	2	-	-
	Personality Disorder	1	-	-
	Multiple	2	-	-
Psychoactive Medications				
	SNRI	2	-	-
	SSRI	2	-	-
	Stimulant	1	-	-
Age (Years)		-	23.24	5.34
	Range	18.00 to 48.42	-	-
BAARS-IV				
	Current Total ADHD Score	-	28.76	7.70
	Current Inattention Score	-	14.84	4.29
	Current Hyperactivity Score	-	8.29	2.48
	Current Impulsivity Score	-	6.66	2.39
	Clinically Significant	24	-	-

Note. SNRI = Selective Norepinephrine Reuptake Inhibitor; SSRI = Selective Serotonin Reuptake Inhibitor

Analysis of Order Set Effects

A grand mean for combined transformed NJ performances across all eight tasks was calculated to assess potential order set effects. No statistically significant order effect was found using omnibus One-Way ANOVA, $F(23, 75) = 1.24$, $p = .260$, $\omega^2 = 0.067$. However, this may be due to low power (observed $\beta = .774$) given the number of levels of the independent variable (i.e., 24 order sets) and low frequency (i.e., a small n) with which each order set was utilized. See Table 8 for descriptive statistics regarding grand mean NJ performance between order sets.

Table 8

Descriptive Statistics for Grand Mean NJ Performance across Order Sets

Order Set #	*M*	*SD*	*n*
1	7.26	1.23	4
2	8.12	1.23	4
3	7.84	1.93	4
4	8.11	1.14	4
5	7.45	0.95	3
6	10.06	1.42	3
7	7.01	0.83	3
8	7.85	1.27	3
9	6.46	0.39	3
10	8.24	1.67	3
11	10.05	0.50	3
12	8.70	1.59	3
13	7.93	2.11	3
14	7.28	2.08	3
15	9.05	0.92	3
16	7.51	1.83	3
17	7.98	1.03	3
18	9.16	0.19	3
19	7.77	2.99	3
20	8.75	0.44	3
21	7.16	0.69	3
22	7.41	0.70	3
23	8.38	1.80	3
24	8.83	0.52	3
Total	8.07	1.46	76

Primary Data Analyses

Omnibus Repeated Measures ANOVA indicated that graphomotor fluency was significantly affected by task type, $F(4.34, 295.09) = 139.11$, $p < .001$, $\omega^2_{partial} = 0.897$. To limit the number of comparisons made within task types and minimize experiment-wise error, a comparison of descriptive statistics combined with a visual analysis of results using box plots was conducted. See Table 9 for descriptive statistics of graphomotor fluency performance across all tasks and Figure 6 for box plots used for graphical analysis.

Table 9

Graphomotor Fluency Descriptive Statistics for All Task Types – Transformed Mean NJ

Task	*N*	*M*	*SD*
Free Hand	69	5.38	2.85
Slow Linear	69	6.94	2.51
Moderate Linear	69	6.21	2.02
Fast Linear	69	3.67	1.33
Slow Wave	69	14.44	4.00
Moderate Wave	69	6.91	1.66
Fast Wave	69	6.13	1.75
Random	69	8.10	2.01

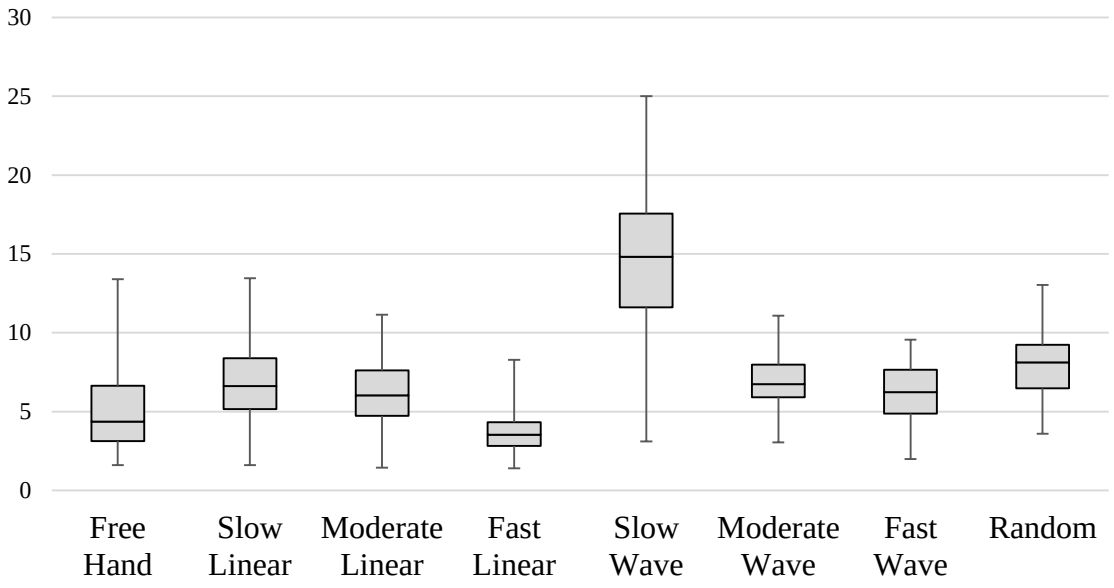

Figure 6. Box-plots of transformed mean Normalized Jerk for each task type.

Descriptive statistics and box plots were interpreted to suggest that the greatest difference in transformed mean NJ due to task type existed between the Fast Linear Pattern task and the Slow Wave Pattern task. A repeated measures contrast was performed, indicating a statistically significant difference in graphomotor fluency between these two tasks with a large effect size, $F(1, 68) = 494.91$, $p < .001$, $\omega^2_{partial} = 0.879$. Results in turn demonstrated that participants evidenced greater graphomotor dysfluency during the Slow Wave Pattern task versus the Fast Linear Pattern task. Additional custom repeated measures linear contrasts were conducted for exploratory and interpretive purposes. Contrasts of interest included a comparison of the Fast Linear Pattern task with the Random task (as this represented the next largest mean and graphically disparate group comparison), all Linear Pattern tasks with all Wave Pattern tasks, as well as all patterned tasks versus the random task. Although statistically significant differences were found in each comparison, the greatest effect size was observed in the original comparison of the Fast Linear Pattern task with the Slow Wave

Pattern task. See Table 10 for a summary of these additional custom repeated measures linear contrasts.

Table 10

Additional Custom Linear Contrasts

	SS	*df*	*MS*	*F*	*p*	$\omega^2_{partial}$
Fast Linear vs. Random	1380.85	1	1380.85	261.12	<.001	.783
Error term	380.75	72	5.29			
Linear Pattern vs. Wave Pattern	7885.14	1	7885.14	139.42	<.001	.665
Error term	3944.21	70	56.35			
All Pattern vs. Random	1264.08	1	1264.08	9.08	.004	.105
Error term	9608.30	69	139.251			

Note. "All Pattern" represents the Slow, Moderate, and Fast Linear and Slow, Moderate, and Fast Wave Pattern tasks, combined.

Chapter 4: Discussion – Part I, Study 1

The purpose of Part I, Study 1 was to determine which two of eight designed graphomotor tasks would elicit the greatest and least graphomotor dysfluency for subsequent use in Part II, Study 2, in turn representing "High" and "Low" cognitive control tasks, respectively. Results demonstrated that the greatest graphomotor fluency differences existed between the Slow Wave Pattern task and the Fast Linear Pattern task. Based on these findings, the Slow Wave Pattern task was interpreted as best representing the "High" cognitive control task, whereas the Fast Linear Pattern task was interpreted as best representing the "Low" cognitive control task. These conclusions are consistent with past research indicating reduced graphomotor fluency and generally reduced performance on kinematic variables in the presence of greater figure complexity (Hollerbach, 1981; Ruud G. J. Meulenbroek & Thomassen, 1993; R. G. J. Meulenbroek & Van Galen, 1988;

Morasso, Ivaldi, & Ruggiero, 1983; Van Galen, 1991) and greater cognitive control demands (Tucha & Lange, 2005; Tucha, Tucha, & Lange, 2008). In addition, relative to the Fast Linear Pattern task, participants (observationally) appeared to demonstrate greater focus in an effort to perform the Slow Wave Pattern without making errors (i.e., trying to stay behind the cursor without touching it), which would be consistent with at least two aspects of Barkley's (2006) description of cognitive control, which includes maintaining task persistence and inhibiting prepotent responses. However, these observations would need to be confirmed through systematic data collection and analysis, which was not done as part of this study. Lastly, these tasks may also be viewed as representing high and low neuromotor complexity with additional cognitive components. Examples of additional cognitive components likely involved in the performance of this task include focused/sustained attention, self-monitoring, adjusting responses based on sensory feedback, visual-motor integration, and visual/spatial perception. Nevertheless, although results for the purposes of Part I, Study 1 appear clear and the two identified graphomotor tasks were adopted for use in Part II, Study 2, several observations related to participant demographic variables and potential limitations warrant further discussion.

As detailed above, participants were predominantly right-handed, women, and Caucasian. Although the preponderance of these participant characteristics could limit the generalizability of findings to the larger population, differences specifically related to sex and handedness have not been found to significantly relate to overall performance on kinematic variables (Mergl et al., 1999; H. van Mier, 2006). Unfortunately, there currently exists no research to clarify if race or ethnicity potentially plays a role in graphomotor fluency performance as measured by NJ. It could be reasonably concluded

that race or ethnicity per se has no direct effect on kinematic graphomotor fluency measures, but further research is needed to clarify if performance on these variables is in fact sensitive to this particular demographic variable.

In addition, a larger-than-expected proportion of participants reported significant ADHD symptomatology on at least one scale of the BAARS-IV. Although the BAARS-IV is by no means diagnostic of ADHD when used in isolation, this again raises concerns regarding the generalizability of these findings to the larger population. Subsequent exploratory analyses using One-Way ANOVA were conducted comparing graphomotor fluency performance on each task between those who screened positive (n = 24) and those who screened negative for ADHD (n = 52) on the BAARS-IV. These analyses revealed no statistically significant findings or findings that neared significance. This was the case even while not making corrections to the alpha level in order to reduce experiment-wise error due to multiple comparisons. These subsequent analyses could be reasonably interpreted to suggest that despite the higher proportion of participants with significant ADHD symptomatology than what would be expected in a university student sample, generalizability does not appear to be significantly called into question noting no notable group differences on graphomotor fluency measures in any of the experimental tasks.

Lastly, although attempts were made to remove trials deemed invalid based on observations and extreme or unrealistic values (i.e., 0 or ≥10,000), this was not conducted in a pre-defined, systematic manner. As such, predefined, standardized, and operationalized definitions of error types, invalid trials based on observations, and extreme values would likely improve replication and standardization in future work.

Noting this, these data were collected and analyses conducted during Part II, Study 2 (see below).

Chapter 5: Method – Part II, Studies 2, 3, 4, and 5

Participants

All power analyses were conducted using G*Power software (Buchner et al., 2009). For Studies 2 and 3, power analysis ($1 - \beta = .80$) indicated 34 total participants were needed in each study to detect a statistically significant difference ($\alpha = .05$) of medium effect size ($\omega^2 = 0.059$) using the proposed statistical analyses (i.e., 2 x 2 Factorial Mixed Design Repeated Measures ANOVA). In Study 4, 43 total participants were identified as necessary to have sufficient power (H_1 $p^2 = .27$; H_0 $p^2 = 0$; $1 - \beta = .80$; $\alpha = .05$) to conduct multiple regression analysis with three predictor variables. However, per recommendations by Stevens (2009) that at least 15 participants should be recruited per predictor variable, 45 participants were targeted for recruitment. Lastly, based on convention, 100 participants (50 with ADHD and 50 without ADHD) were targeted for recruitment in order to perform a receiver operating characteristic (ROC) curve analysis for Study 5.

Following clearance through the University of Windsor's Research Ethics Board, child and adolescent participants - referred to going forward as "participants" when referenced collectively as child and adolescent participants - were recruited through community media advertising, psychological and medical clinics, community organizations, and the University of Windsor's Psychology Participant Pool. Participant eligibility requirements included only those who were between the ages of 9 and 15 years, had normal or corrected-to-normal vision, were able to perform intellectual and

motor tasks without significant difficulty (e.g., did not have an intellectual disability or neurological condition that would impair their ability to write), and were able to participate in handwriting tasks that lasted approximately 30 minutes. As compensation for participation, parents of participants received a written report of their child's performance on standardized measures (see Materials and Apparatus section below for information regarding measures used) and $10 if recruited through the community. If recruited through the University of Windsor's Psychology Participant pool, parents of participants received a written report and 2 bonus points towards their final grade of a qualifying university course. Participants received $10 for their participation in the study regardless of recruitment source. If concerns were raised regarding participants' current psychosocial functioning based in interview data or parent responses on rating scales, a recommendation for formal psychological evaluation was made. Altogether, 46 participants took part in the study. Data, however, were analyzed from 40 participants after removal of six participants due to invalid performance or for statistical reasons (described further below and in the Procedures and Results sections).

Based on group assignment criteria described in the Procedures section below, 16 participants met inclusion criteria for the ADHD group (seven of whom had a preexisting diagnosis of ADHD) and 30 were assigned to the control group. Of note is that only three participants in the ADHD group did not meet strict DSM-5 diagnostic criteria for ADHD. This was due to one participant having impairments reported in only one setting, and two participants demonstrating five instead of six symptoms of inattention or hyperactivity-impulsivity as required by DSM-5 criteria. Otherwise, all other DSM-5 diagnostic criteria were met by these three participants placed in the ADHD group based on their Conners 3

ADHD Index score. As described further below, the psychosocial functioning of these three participants was also very similar to ADHD participants in that each had at-risk or clinically significant difficulties in multiple areas of functioning, which further supported their placement within the ADHD group.

Materials and Apparatus

Demographic, sleep, and neuropsychological assessment. For purposes of sample description and group matching, demographic information (e.g., age, sex, handedness, socioeconomic status [SES], and medical and psychiatric histories) and sleep history were collected via an interview with researchers. See Appendix B for complete information collected and the interview form used. SES was estimated using the four factor index of social status (Hollingshead, 1975), with higher calculated scores based on education, occupation, and marital status indicating higher socioeconomic status. Neuropsychological assessment consisted of measures in the following domains: broad psychosocial functioning, ADHD and Developmental Coordination Disorder (DCD) symptomatology, general intellectual functioning, processing speed, working memory, performance validity, academic screening, and fine motor skills.

Overall psychosocial functioning was assessed by parent-report using the Behavioral Assessment System for Children – 2nd Edition (BASC-2) (Reynolds & Kamphaus, 2004). The BASC-2 provides information regarding an individual's psychosocial functioning in the broadband domains of internalizing behaviours, externalizing behaviours, behavioural symptoms, and adaptive functioning. These composite scores are also associated with individual scales with related content, which include symptoms of hyperactivity, aggression, conduct problems, anxiety, depression,

somatization, withdrawal, and attention problems, as well as abilities related to adaptability, social skills, leadership, functional communication, and performing activities of daily living. According to the manual, the BASC-2 has excellent psychometric properties, including good reliability (internal consistency values in the .80 range for individual scales and in the .90 range for composite scales; six-week test-retest reliabilities in the .80 range for composite scores and between .70 and .80 for individual scales; median interrater reliabilities in the .70s for both individual and composite scales) and well-established validity (e.g., individual scale and composite score correlations with other psychometrically sound measures of psychosocial functioning ranging between .70 and .90). The BASC-2 yields T scores (M = 50, SD = 10), which were derived based on age and general population normative data. According to the manual, higher T scores for Internalizing Problems, Externalizing Problems, and Behavioural Symptoms Index composites and their corresponding individual scales indicate more problems. In turn, scores ≥ 60 were interpreted as indicating at-risk symptoms and scores ≥ 70 were interpreted as indicating clinically significant problems. For the Adaptive Skills Composite and its corresponding individual scales, however, Lower T scores indicate worse functioning in those areas, with T scores ≤ 39 interpreted as at-risk and T scores ≤ 29 interpreted as clinically significant.

ADHD symptomatology was assessed using the long-form version of the Conners 3 (Conners, 2008) parent rating form with DSM-5 update. The Conners 3 possesses good psychometric properties, with internal consistency coefficients ranging between .77 and .97, two- to four-week test-retest reliabilities ranging between .71 to .98, and interrater reliability coefficients from .52 to .94. The manual also reports evidence of acceptable

discriminative and construct validity. The Conners 3 also yields T scores (M = 50, SD = 10), with higher scores indicating greater problems or a higher level of symptomatology in the measured area. Based on standardized interpretation described in the manual, clinical and symptom T scores ≥ 65 were interpreted as indicating an elevated score with more concerns than are typically reported, and T scores ≥ 70 were interpreted as indicating very elevated symptoms with many more concerns reported than is typical for an individual's age and sex. All T scores were derived using age- and sex-based normative data.

Symptomatology associated with DCD was assessed using the Developmental Coordination Disorder Questionnaire 2007 (DCDQ'07) (Wilson & Crawford, 2012). The DCDQ'07 is a parent-report measure of their child's motor functioning that yields raw scores in three domains: control during movement, fine motor/handwriting, and general coordination. Raw scores are added to derive a total raw score, with scores below the cutoff (thus indicating more problems in that domain) indicating the presence of DCD and scores above the cutoff (thus indicating fewer or no problems in that domain) suggesting that DCD is not present. A cutoff score maximizing sensitivity and specificity is provided for various age ranges of children and adolescents between 5 and 15 years of age. According to the manual, the DCDQ'07 possesses good overall psychometric properties. This includes good internal consistency reliability (Cronbach's α = .89), strong evidence of construct and concurrent validity, and an overall sensitivity and specificity of 84.6% and 70.8%, respectively.

An estimate of general intellectual functioning (i.e., IQ) was derived using the Block Design (BD) and Vocabulary (VC) subtests of the Wechsler Intelligence Scale for

Children-Fourth Edition (WISC-IV; Wechsler, 2003). This short form estimate of IQ yields reliability and validity coefficients with the WISC-IV Full Scale IQ (FSIQ) of .916 and .874, respectively (Sattler, 2008). To calculate estimated IQ, scaled scores (M = 10, SD = 3) derived from age-based normative data of the BD and VC subtests were summated. A standard score (M = 100, SD = 15) was then determined based on this sum of scaled scores using resources provided by Sattler (2008). Per construct validity data provided within the WISC-IV Technical and Interpretive Manual, the BD subtest can be conceptualized as a measure of nonverbal, visual-perceptual reasoning, and the VC subtest represents a general measure of verbal ability. The VC subtest was selected as a measure of general verbal ability to be used in Study 4 noting its strong validity and similarity to tests of verbal ability used in a previous kinematic study demonstrating an association between verbal skills and graphomotor performance (Mergl et al., 1999). Additional subtests utilized from the WISC-IV included the Symbol Search (SS) subtest as a measure of processing speed and the Digit Span (DS) subtest as a measure of working memory. The SS subtest was selected as a measure of processing speed to be used in Study 4 noting its theoretical association with learning and executive functioning (Koziol, Budding, & Chidekel, 2013; Noda et al., 2013). The WISC-IV Technical and Interpretive Manual (Wechsler, 2003) provides extensive data supporting the acceptable to excellent reliability and validity of these measures and their associated constructs. Reliable digit span was calculated based on DS performance as a measure of performance validity, with scores ≤6 interpreted as suggestive of invalid performance in non-clinical populations (Kirkwood, Hargrave, & Kirk, 2011). No such cutoff was used for participants in the ADHD group, noting that cross-validation studies are necessary to

substantiate the use of reliable digit span in children and adolescents with a history of ADHD (e.g., see Welsh, Bender, Whitman, Vasserman, and MacAllister, 2012).

Academic screening was conducted using the Wide Range Achievement Test-Fourth Edition (WRAT-4) (Wilkinson & Robertson, 2006). Selected subtests and academic domains tested included Word Reading, Spelling, and Math Computation. The WRAT-4 has excellent psychometric properties, including good to excellent internal consistency reliability (median α between .87 and .96 for subtests), good test-retest reliability within one month (α = .86, .89, and .88 for Word Reading, Spelling, and Math Computation, respectively), and good internal and external evidence of validity when examining item content and as compared to other psychometrically validated measures of academic achievement (Wilkinson & Robertson, 2006).

Fine motor skills were quantified using the Grooved Pegboard Test (Klove, 1963; Reitan, 1969). The Grooved Pegboard Test is a test of fine motor speed, hand-eye coordination, and dexterity that is widely used in neuropsychological assessment. The test requires that participants place a small metal peg with a round and flat side into a similarly shaped hole in a pegboard, first with their dominant (e.g., right) and next with their non-dominant (e.g., left) hand. Raw scores are recorded in seconds and T scores (M = 50, SD = 10) were calculated based on normative data provided by Knights and Moule (1968) such that higher scores indicated better fine motor skills. Although no reliability information is available regarding performance in children and adolescents, test-retest reliability has been found to be marginal (.67) to high (.86) over intervals of 4 to 24 months in adults, respectively (Strauss, Sherman, & Spreen, 2006). This test also shows modest to moderate evidence of construct, criterion, and ecological validity with other

measures of motor functioning (Strauss et al., 2006), and was thus chosen as a measure of fine motor skills to be used in Study 4.

Handwriting analysis. A WACOM Cintiq 21UX digitizing tablet and MovAlyzeR software, as detailed in Part I, were again used to record and process handwriting movements (see the Methods – Part I, Materials and Apparatus section above for specific information).

Procedures

Research assistants. In addition to the principal investigator, eight research assistants, including two appointed lead research assistants, conducted scheduling, consent and interview procedures, parent and child assessment, data entry, and report writing. All research assistants were provided extensive training for experimental and assessment tasks. A comprehensive manual detailing all procedures and decisions was also created and provided to research assistants. Each research assistant was required to pass a "check out" procedure demonstrating appropriate administration of standardized tests, operation of experimental tasks, and recording of participant behaviours during experimental tasks. Regular communication was maintained and update meetings were held virtually (e.g., group emails) and in-person to address any issues that arose.

Eligibility, interview, and parent-report measures. Prior to scheduling and participating in the study, all parents of potential participants were contacted to determine eligibility. Verbiage was used that allowed parents to answer "yes" or "no" with regard to their child's overall eligibility while limiting disclosure of their child's personal identifying and sensitive health information (e.g., this would allow the parent to respond without disclosing information regarding their child's name, age, vision status, presence

of motor or intellectual impairment, or ability to participate in handwriting tasks lasting 30 minutes). After eligibility was determined, participants were assigned an identification number (ID) that was listed with their name on an encrypted spreadsheet separate from research data. This connection between participant name and ID was maintained for 48 hours from the time they completed the research study, thus allowing participants the opportunity to withdraw their data from the study during this time. After 48 hours, the link between name and ID was removed, thus completely anonymizing participant data.

Participants who were taking stimulant medication were asked to discontinue medication for 24 to 48 hours prior to testing. The time-frame of medication discontinuation was based on prescription drug information indicating very low drug plasma concentrations between 24 and 48 hours after taking stimulant medication (see U.S. Food & Drug Administration, 2007). As such, all participants in this study were either stimulant medication naïve or had discontinued stimulant medication prior to, and during participation.

All procedures were completed during one session. Informed consent was obtained from parent participants and assent was obtained from participants. The interview was conducted and demographic data collected with both the parent and participant present. Data regarding existing diagnoses of neurodevelopmental or psychiatric disorders that were not screened as part of the study were recorded as indicating the participant was "at risk" for the condition described (e.g., Generalized Anxiety Disorder, Obsessive Compulsive Disorder, or others). After completion of the interview, participants took part in neuropsychological and handwriting assessment with one research assistant while parent participants completed rating scales and follow-up

questions with another research assistant. Parent participants completed rating scales in the following order: BASC-2, Conners 3, and DCDQ'07. Research assistants subsequently ensured all forms were complete and caregivers' questions were answered. After completion of the BASC-2 and Conners 3, critical items were reviewed (e.g., questions related to destructive, violent, or potentially suicidal behaviour) to determine if follow-up questioning was necessary to ensure safety (note: no significant concerns related to critical items were reported by any parents or participants). After completion of the Conners 3 and for diagnostic coding purposes, parents were asked if their child experienced problems with attention and/or hyperactivity before the age 12. See Figure 7 for the decision flow chart for determining questioning and coding for this diagnostic criteria.

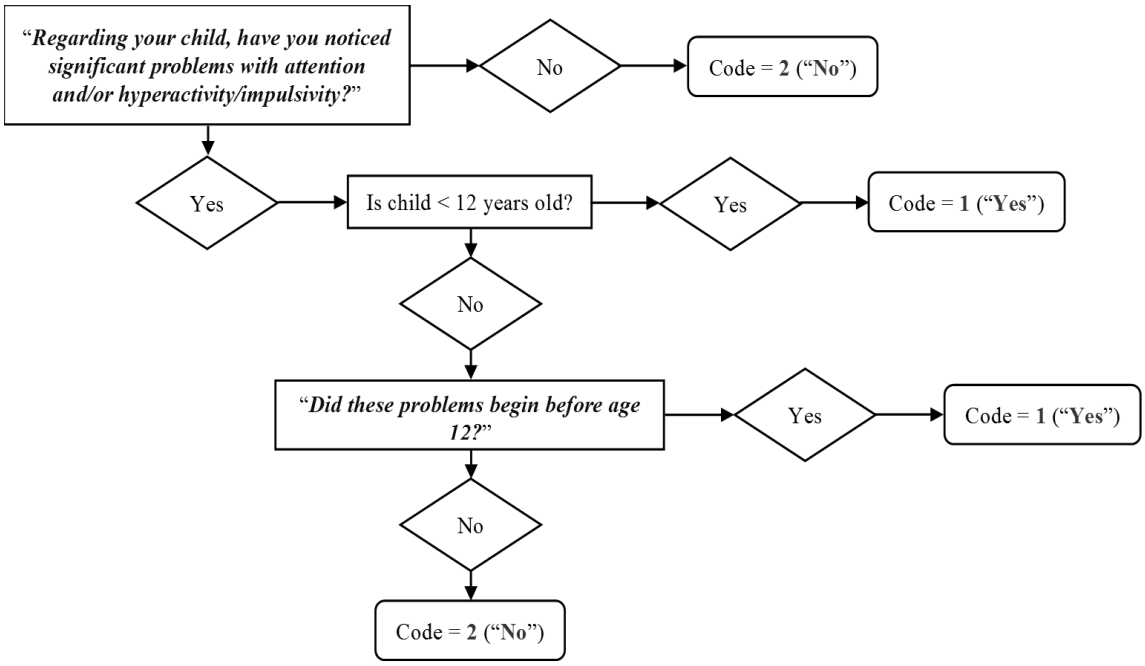

Figure 7. Decision chart for determining if a participant was coded as potentially meeting or not meeting diagnostic criteria for experiencing symptoms before the age of 12 years.

After completing the study, parents and participants were debriefed regarding hypotheses and provided the opportunity to ask questions regarding the study.

Group assignment. Participants assigned to the ADHD group included those who 1) were previously diagnosed with ADHD and continued to meet DSM-5 criteria based on a combination of clinical interview and rating scales, 2) did not have a preexisting diagnosis of ADHD but met DSM-5 criteria based on information gained from clinical interview and rating scales, or 3) had a Conners 3 ADHD Index Probability Score ≥71. The ADHD Index consists of 10 selected items and yields a Probability score indicating the likelihood that responses are consistent with a diagnosis of ADHD. Per guidelines outlined within the Conners 3 manual, Probability scores ≥71 indicate a high probability that a classification of ADHD is warranted, and was thus used as the cutoff score for the current study. Classification of ADHD presentation as primarily inattentive, primarily hyperactive-impulsive, or combined was based on either DSM-5 symptom criteria for those who met full criteria or, for those who did not meet full DSM-5 criteria, Conners 3 Inattention and/or Hyperactivity/Impulsivity domain scale T scores ≥65. That is, those who screened positively for ADHD based on the Conners 3 ADHD Index Probability score with only an Inattention T score ≥65 were coded as having a primarily inattentive presentation, those with only a Hyperactivity/Impulsivity T score ≥65 were coded as having a primarily hyperactive-impulsive presentation, and those who had both Inattention and Hyperactive/Impulsivity T scores ≥65 were coded as having a combined presentation. All participants not meeting any of these criteria were assigned to the control group.

Noting the high comorbidity of Specific Learning Disorder and Disruptive Behaviour Disorders (e.g., Oppositional Defiant Disorder and Conduct Disorder) in those with a history of ADHD, participants were screened for possible Specific Learning Disorder and Disruptive Behaviour Disorders based on results from the Conners 3 Learning Problems scale, symptoms counts for Oppositional Defiant Disorder (ODD), Conduct Disorder (CD) symptom counts, parent interview, and discrepancy analysis between derived standard scores of estimated IQ and WRAT-4 Word Reading, Spelling, and Math Calculations performance (i.e., an estimated IQ that is greater than 29 standard score points higher than performance on academic tests). Participants were coded as "at risk" for specific learning disorder if they had a T Score of ≥65 on the Learning Problems scale or met the discrepancy analysis criterion, and "at risk" for ODD or CD if they met DSM 5 symptom count criteria based on Conners 3 ratings.

Neuropsychological assessment and handwriting tasks. Order of administration considerations were made between neuropsychological assessment and overall experimental handwriting tasks, and among the three experimental handwriting tasks. Participants with an odd ID were assigned neuropsychological assessment first and handwriting tasks second, and even numbered participants were assigned handwriting tasks first and neuropsychological assessment second. Experimental handwriting tasks were ordered such that all participants completed the High or Low cognitive control tasks first, followed by the Learning task. High and Low cognitive control tasks were counterbalanced in an alternating fashion in order to ensure that half of the participants completed the High cognitive control task first and half completed the Low cognitive control task first. Statistical analysis indicated that order of administration of

neuropsychological testing, experimental handwriting tasks, and cognitive control tasks (i.e., High versus Low paradigms) were similar overall. However, a greater proportion of participants with ADHD completed neuropsychological testing first and experimental handwriting tasks second. See Table 11 below for descriptive statistics regarding order of administration.

Table 11.

Orders of Administration

	Control		ADHD	
	n	Proportion	*n*	Proportion
Order of Administration				
NP → Tablet	12	50%	10	62%
Tablet → NP	12	50%	6	38%
Cognitive Control: High → Low	12	48%	10	62%
Cognitive Control: Low → High	12	52%	6	38%

The neuropsychological tests and procedures were conducted in the following order: BD, DS, VC, and SS subtests of the WISC-IV; Word Reading, Spelling, and Math Calculations subtests of the WRAT-4; and the Grooved Pegboard Test. Prior to engaging in experimental handwriting tasks, participants were provided the opportunity to become familiar with the tablet and pen and adjust their chair and the tablet to a comfortable position. During this time, participants were asked if they had already been taught how to write in cursive and this information was recorded. Participants were then instructed how to complete the cognitive control tasks and given an opportunity to practice. Practice consisted of writing their name on the tablet five times, following a cursor in a straight line three times, and following a cursor in a wavy pattern three times. Practice Linear and Wavy Pattern tasks used were the Moderate Linear pattern and Moderate Wave pattern tasks described in Part I, Study 1. See Appendix A for verbiage and procedures used to

orient the participant to the use of the tablet and how to conduct the cognitive control tasks.

The High and Low Cognitive Control tasks were identical to those described in Part I, Study 1, consisting of the Slow Wave and Fast Linear tasks, respectively. The Learning task involved writing a novel grapheme within a box 30 times. A card with the novel grapheme was present throughout the experiment. Participants were allowed to position the card in a location that maximized their ability to look at the grapheme while completing the task. See Figure 8 for an example of the novel symbol written inside the box as captured by the digitizing tablet and MovAlyzeR software. See Appendix C for specific verbiage used to explain the Learning task.

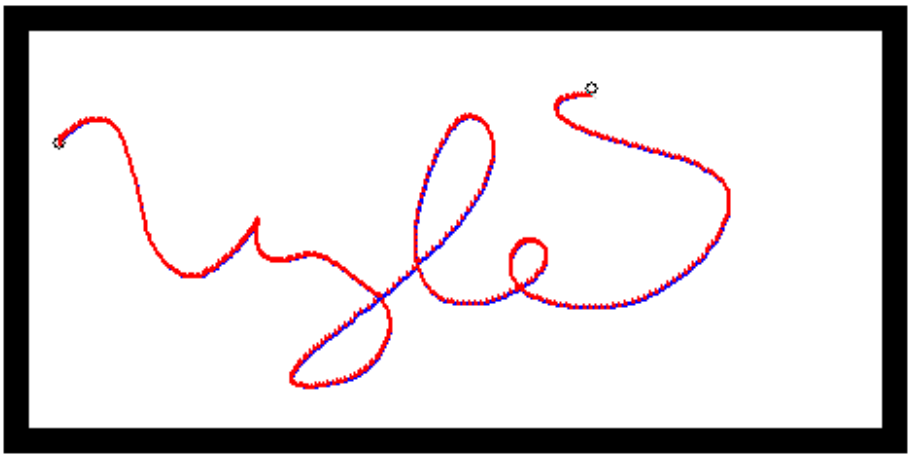

Figure 8. Example image capture of novel grapheme practiced 30 times during the Learning task.

Graphomotor fluency and automaticity was again the construct of interest captured by the digitizing tablet, which was operationalized as Normalized Jerk (NJ). For Study 2, this Dependent Variable (DV) of interest was operationalized as the mean NJ for the 20 trials performed in each of the High Cognitive Control and Low Cognitive Control tasks. In Study 3, beginning learning fluency was defined as the mean NJ of the first 3

valid trials performed within each participant's first 10 trials (i.e., Beginning), and ending learning fluency was defined as the mean NJ of the last 3 valid trials performed within each participant's last 10 trials (i.e., Ending). The DV or outcome variable of interest for Study 4 was improvement in graphomotor fluency with practice (i.e., Change). This DV was operationalized as the proportion of change between Beginning and Ending performance. Note that a proportion of change was utilized as opposed to a raw difference score as individuals show significant variability in the degree to which graphomotor fluency and automatization are demonstrated. As such, what appears to be a significant change for one individual based on a raw difference score may not be the same for an individual based on a proportion of change from Beginning to Ending performance. Predictor variables for Study 4, as detailed above, included verbal ability (Vocabulary scaled score subtest performance on the WISC-IV), processing speed (Symbol Search subtest scaled score performance on the WISC-IV), and fine motor skills (dominant hand fine motor skill T score performance on the Grooved Pegs). Lastly, Change was used as the testing variable for purposes of the ROC Curve analysis conducted as part of Study 5. See Table 12 below for descriptions and determinations of each dependent, outcome, independent, predictor, testing, and state variable used in each study.

Table 12

Descriptions of Variables used in each Study.

Variable Name	Description	Calculation	Variable Type & Associated Study
NJ	Normalized Jerk; represents graphomotor fluency and automaticity	Third time derivative calculated by MoValyzeR Software	Utilized throughout
Low Cognitive Control	Graphomotor fluency and automatization during within the context of low cognitive control demands	Average NJ of 20 trials conducted during the Low Cognitive Control task	DV: Study 2
High Cognitive Control	Graphomotor fluency and automatization during within the context of high cognitive control demands	Average NJ of 20 trials conducted during the High Cognitive Control task	DV: Study 2
Beginning	Graphomotor fluency and automatization at the beginning of practice while learning the novel grapheme	Average NJ of the first 3 valid trials within the first 10 total trials performed	DV: Study 3
Ending	Graphomotor fluency and automatization at the ending of practice while learning the novel grapheme	Average NJ of the last 3 valid trials within the last 10 total trials performed	DV: Study 3
Change	Proportion of change between the beginning and ending of practice of the novel grapheme	Calculated by subtracting Ending NJ from Beginning NJ and dividing by Beginning NJ	OV: Study 4 TV: Study 5
Group	Group membership based on the presence of absence of an ADHD diagnosis	Based on diagnostic criteria according to study protocol	IV: Studies 2 & 3 SV: Study 5
VC	Verbal ability as indicated by performance on the Vocabulary subtest of the WISC-IV	Scaled score derived from age-based normative data	PV: Study 4
SS	Processing speed ability as indicated by performance on the Symbol Search subtest of the WISC-IV	Scaled score derived from age-based normative data	PV: Study 4
Pegs	Fine motor skill ability as indicated by performance on the Grooved Pegboard test	T score derived from age- and sex-based normative data	PV: Study 4

Note. DV = Dependent Variable. OV = Outcome Variable. TV = Test Variable. SV = State Variable. PV = Predictor Variable.

Observations of participant behaviours while performing handwriting tasks were systematically recorded to identify invalid trials for removal, and for potential descriptive and explanatory purposes. This was conducted using a form designed by the principle investigator based on observations made during Part I, Study 1. Specific error behaviours observed were categorized into two separate classes: cognitive control errors and invalid trial errors. Cognitive control errors were only pertinent to, and recorded for, cognitive control tasks, as no such errors were possible during the novel symbol learning task. Per-trial cognitive control errors were counted in separate columns whenever a participant 1) touched the tablet too soon (i.e., "too soon") or 2) touched the cursor (i.e., "# touched"). Per-trial invalid trial errors were recorded and counted in separate columns and included the following error types: start-stop-restart, wrong pattern, timeout (long), time out (short), and other. Corrective feedback was provided to participants each time an error was made. See Table 13 below for descriptions of each invalid trial error type. See Appendix D for the form used to collect participant behaviour data.

Table 13

Descriptions of Invalid Trial Error Types

Start-stop-restart	Participant began the task, stopped and lifted pen, then restarted drawing, but MovAlyzeR, before the trial was completed, reset the task and continued to the next trial.
Wrong pattern	Cognitive control tasks: The participant did not grossly follow or did not attempt to follow the pattern of the cursor. Learning task: The overall image was not grossly identifiable as the novel grapheme and/or did not contain the six elements depicted on the card. This included having too few or too many elements.
Timeout (long)	The participant did not complete the trial within the allotted two-minute time frame and MovAlyzeR software reset to begin the next trial.
Timeout (short)	The participant started the task very briefly but stopped and MovAlyzeR software reset to begin the next trial.
Other	Any errors not captured by the above error types.

Error validation. After completion of all tasks, errors observed and recorded during experimentation were subsequently validated against each individual trial captured with MovAlyzeR software. When a trial was verified as invalid based on observations, its handwriting data for those individual trials were removed from analyses.

Chapter 6: Results and Discussion – Part II, Studies 2, 3, 4, and 5

All statistical analyses were performed using IBM SPSS Statistics, Version 22. Higher values of mean NJ represented greater dysfluency (i.e., less automaticity) and lower values represented less dysfluency (i.e., greater automaticity). Interpretations of effect sizes using ω^2 (for analyses including between group variance) and $\omega^2_{\text{partial}}$ (for analyses with only within group variance) were based on Kirk's (2003) suggestions due to a lack of effect sizes reported in the literature. As such, effect sizes of 0.010 to 0.058,

0.059 to 0.137, and 0.138 or greater were interpreted to indicate small, medium, and large associations, respectively. Unless otherwise indicated, an alpha level of .05 was used to indicate statistical significance for findings in each study individually.

Data Cleaning Prior to Statistical Analyses

Consistent with the above protocol used in the pilot study, all NJ values that were 0 or ≥10,000 were removed from analyses. Combined with trials deemed invalid by observations, this resulted in removing 5.38%, 10.25%, and 20.17% of the original data points from the Low Cognitive Control, High Cognitive Control, and Learning tasks, respectively. As described further below, data appeared to be missing not at random as more invalid trials were produced by younger participants. As such, imputation techniques were not utilized noting that such techniques assume data are missing at random or missing completely at random.

As highlighted in the discussion section of Part I, Study 1, individual trials in each of the three task types were systematically analyzed to determine the most appropriate cutoff value for removing NJ values characterized as extreme and unduly influential in their overall NJ for a given individual. The aim of this analysis was to determine which cutoff criteria would maximize the removal of extreme values in each task while allowing for an appropriate and defensible degree of intraindividual variability. Allowing for an appropriate degree of variability was important from a developmental neuropsychological perspective because participants may naturally demonstrate more trial by trial variability relative to adults due to less well-developed cognitive and motor abilities. As such, care was taken to avoid removing potentially valid trials and/or important explanatory

variance assuming greater variability and more numerous extreme scores may be developmentally appropriate.

For each participant, per trial Z scores were calculated with removal for each handwriting task. Cutoff values investigated included Z scores with absolute values ≥ |9|, |8|, |7|, |6|, |5|, |4|, and |3|. Analyses indicated that a single cutoff value was not appropriate for all tasks, as each task yielded a different distribution and frequency of putatively extreme scores. As such, cutoff values were selected based on retaining at least 97% of all trials (rounded to the nearest whole number) after removing trials determined to be invalid by observations. This resulted in the proposed removal of all trials in all tasks with NJ values ≥10,000, and the removal of additional trials based on the following Z score cutoffs: Low Cognitive Control Z score cutoff of ≥9, High Cognitive Control Z score cutoff of ≥8, and Learning Z score cutoff of ≥5. See Table 14 below for descriptive statistics pertaining to proportion of remaining trials after removal of extreme values at various Z score cutoffs, and Figure 9 for a graphical analysis of distributions by task and proportion of trials retained and removed by cutoff score.

Table 14

Descriptive Statistics for Trial Removal Analysis

	Low	Cum. Rem.	% Remain.	High	Cum. Rem.	% Remain.	Learning	Cum. Rem.	% Remain.
Possible Trials	800	0	100%	800	0	100%	1200	0	100%
Obs Removed	36	36	96%	74	74	91%	240	240	80%
Total Valid	764	0	100%	726	0	100%	960	0	100%
Removed by ≥10k	8	8	99%	8	8	99%	2	2	100%
Removed by ≥9 Z	18	***26***	***97%***	12	20	97%	5	7	99%
Removed by ≥8 Z	5	31	96%	2	***22***	***97%***	1	8	99%
Removed by ≥7 Z	4	35	96%	4	26	96%	2	10	99%
Removed by ≥6 Z	7	42	95%	2	28	96%	6	16	98%
Removed by ≥5 Z	13	55	93%	3	31	96%	5	***21***	***98%***
Removed by ≥4 Z	28	83	89%	13	44	94%	13	34	96%
Removed by ≥3 Z	26	109	86%	25	69	90%	39	73	92%

Note. Obs = Observations. Cum. Rem. = cumulative number of trials removed. Remain. = Remaining proportion of trials (rounded up). Text in bold and italics represent values at chosen cutoffs.

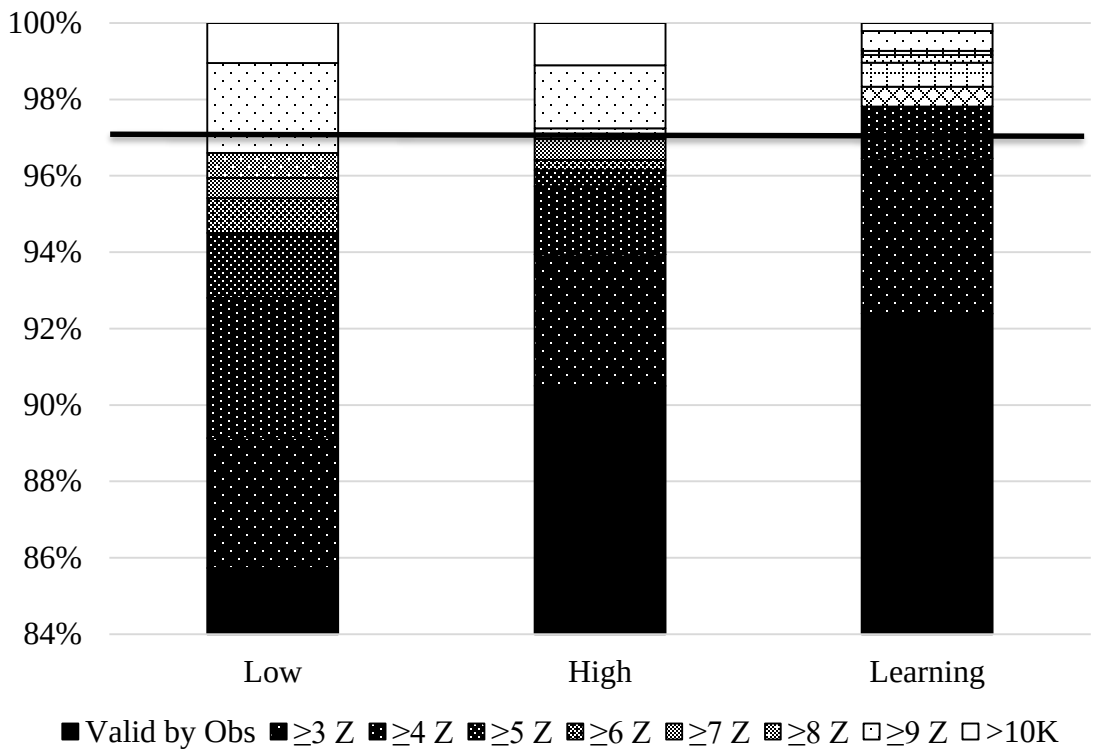

Figure 9. Proportion of trials retained across conditions by Z score cutoff. Areas with darkened backgrounds and white points represent trials retained, whereas areas with a white background and black lines or dots (i.e., higher on the stacked bar chart) represent the proportion of trials removed. The dark horizontal line represents the 97% benchmark. 10K = a normalized jerk of >10,000.

Finally, an omnibus Multivariate Analysis of Variance (MANOVA) was conducted to ensure that there was no significant between-group difference (i.e., control versus ADHD participants) or within-task differences (i.e., Low Cognitive Control, High Cognitive Control, and Learning tasks) in the number of trials removed by proposed Z score cutoffs. Box's M test was not significant (p = .900), indicating that multivariate homogeneity of variance was not violated. The data were non-normal, but interpretation was not thought to be significantly compromised noting that multivariate homogeneity of variance was not violated, sample size was adequate, and all skewness, kurtosis, and variance statistics were within acceptable parameters. There was no statistically

significant task main effect for the number of trials removed, *Wilk's* λ = .985, $F(2, 37) < 1.00$, p = .754, $\omega^2_{multivariate}$ = -.037, and no statistically significant group by task interaction was present, *Wilk's* λ = .983, $F(2, 37) < 1.00$, p = .754, $\omega^2_{multivariate}$ = -.037. As such, prior to statistical analyses to test research hypotheses, all trials identified as "extreme" were removed from each task based on the Z score cutoffs described above.

Initial participant data removal. Data from one control participant were removed due to observably insufficient cooperation and engagement during handwriting tasks. In addition, data from three control participants were removed due to suboptimal performance based on Reliable Digit Span. Two additional control participants were removed in order to maintain relatively equal group sizes to maximize the robustness of statistical analyses used below (see Field, 2009), resulting in a sample of 16 participants with ADHD and 24 control participants (N = 40). Rather than randomly removing these two additional control participants, removal was conducted in order to maximize group equivalency based on demographic factors (e.g., age, SES, and sex) and general intellectual functioning (i.e., Estimated FSIQ based on WISC-IV performance).

Participant descriptive information. Participants were right-hand dominant by self-report (83%), a majority identified as male (63%), and most identified as being of Caucasian/White/European descent (65%). Five participants reported learning English as a second language, and all but one participant reported English as being the language in which they were most fluent. Participants with a diagnosis of ADHD came from lower SES households on average, but this difference was not statistically significant from that of control participant SES, $F(1, 38) = 1.63$, p = .209, ω^2 = 0.016. Control participants were slightly older than participants with ADHD on average, although this difference was

also not statistically significant, $F(1, 38) = 2.14$, $p = .152$, $\omega^2 = 0.028$. See Table 15 for complete descriptive statistics detailing participant demographic information.

Table 15.

Participant Descriptive Statistics: Demographic Information

	Control			ADHD		
	n	*M*	*SD*	*n*	*M*	*SD*
Total	24			16		
Handedness						
Right	19			14		
Left	5			2		
Learned Cursive (Yes/No)	19/5			11/5		
Sex						
Female	10			5		
Male	14			11		
Race/Ethnicity						
Asian	1			0		
Black/African/Caribbean	1			2		
Caucasian/European/White	14			12		
Hispanic/Latina/Latino	0			0		
Middle Eastern	2			0		
Multiracial	1			1		
Native/Aboriginal	4			0		
Other	1			1		
English as a Second Language[†]	3			2		
Socioeconomic Status		45.85	8.59		40.31	8.66
Age		12.18	2.04		11.24	1.86

Note. [†]All who reported English as a Second Language also reported being most fluent in English.

Relative to control participants and consistent with the literature, participants diagnosed with ADHD reported, or were screened, as being "at risk" for a greater variety, number, and proportion of neurodevelopmental and/or psychiatric disorders. Indeed, all

but one participant with ADHD screened positive as being "at risk" for an additional comorbid diagnosis. Seven total participants with ADHD screened positive for two additional potential comorbidities, and three participants with ADHD screened positive for three additional potential comorbidities. Most participants with ADHD were medication naïve (i.e., never having taken medication for the treatment of ADHD) (n = 14), and no participants in the control group reported use of stimulant medication. The two participants with ADHD treated with stimulant medication had a preexisting diagnosis of ADHD. See Table 16 below for complete descriptive statistics for all participant diagnostic information and medication use.

Table 16.

Participant Descriptive Statistics: Diagnostic Information and Medication Use

	Control	ADHD
	n	*n*
Psychiatric Diagnosis/Comorbidities[†]:		
ADHD Combined	0	7
ADHD Inattentive	0	3
ADHD Hyperactive-Impulsive	0	6
At-Risk Additional Diagnosis/Comorbidities:		
Conduct Disorder	0	2
Developmental Coordination Disorder	3	9
Oppositional Defiant Disorder	0	6
Specific Learning Disability	3	12
Obsessive Compulsive Disorder	1	1
Generalized Anxiety Disorder	0	2
Speech/Language Disorder	1	1
Tic Disorder	0	1
Prescribed Stimulant medication (Total):	0	2
Vyvanse	0	1
Adderall	0	1
Stimulant Medication Naïve	24	14
Other Medications and Supplements:		
Epival	1	0
Melatonin	0	3

Note. [†]Psychiatric Diagnosis/Comorbidities based on positive findings on screens used as part of this assessment or parent-report of an existing diagnosis

Quality and quantity of sleep reported between participant groups was similar. However, participants with ADHD were reported to have a significantly greater number of sleep problems relative to control participants, $F(1, 38) = 7.05$, $p = .012$, $\omega^2 = 0.131$, and the effect size was large. This may correspond to data described above indicating only participants with ADHD reported taking melatonin to aid with sleep. See Table 17 below for sleep information.

Table 17.

Participant Sleep Statistics

	Control			ADHD		
	n	*M*	*SD*	*n*	*M*	*SD*
Reported Hours of Sleep		8.59	0.89		8.66	1.56
Quality of Sleep						
Poor	2			1		
Fair	2			5		
Good	20			10		
Typicality of Sleep						
Less than Normal	8			5		
Normal	12			7		
More than Normal	4			4		
Number of Sleep Problems		0.58*	0.88		1.44	1.15
Problems falling asleep	6			7		
Excessive daytime sleepiness	2			3		
Frequent awakening	1			8		
Irregular sleep schedule	1			1		
Snoring or breathing problems	4			4		
Reported Diagnosis of Sleep Apnea	0			1		

Note. * = statistically significant difference between controls and participants with ADHD, $p < .05$

With the exception of the Social Skills subscale of the BASC-2, participants with ADHD demonstrated significantly poorer psychosocial functioning and diagnostic concern relative to control participants. Further, control participants were rated within

normal limits in all areas of psychosocial functioning and diagnostic concern on the BASC-2 and Conners 3, whereas participants with ADHD were, on average, rated within the "at risk" or "clinically significant" range in 17 of these 28 areas. Participants with ADHD were also rated by their caregivers as having significantly lower abilities in all areas of motor functioning compared with control participants as indicated by the DCDQ'07. See Table 18 below for detailed results pertaining to caregiver ratings of participant psychosocial and motor functioning.

Table 18

Participant Psychosocial and Motor Functioning by Caregiver Report

		Controls		ADHD	
		M	*SD*	*M*	*SD*
BASC-2	Externalizing	43.96*	6.13	59.38	8.55
	Internalizing	43.50*	7.33	60.38†	13.55
	Behavioural Symptom Index	43.29*	6.10	64.06†	8.24
	Adaptive Skills	57.75*	6.94	45.06	9.03
	Hyperactivity	43.13*	6.02	64.88†	10.32
	Aggression	44.54*	5.54	55.75	11.29
	Conduct Problems	46.04*	6.83	54.69	8.57
	Anxiety	46.75*	10.55	59.31	14.16
	Depression	45.00*	7.22	62.25†	11.73
	Somatization	42.54*	5.93	56.69	12.32
	Atypicality	43.33*	4.45	60.56†	12.40
	Withdrawal	47.29*	13.11	60.06†	19.70
	Attention Problems	45.50*	7.62	62.00†	6.57
	Adaptability	56.00*	7.70	44.94	8.81
	Social Skills	56.42	7.66	51.56	11.47
	Leadership	58.46*	7.99	48.81	11.85
	Activities of Daily Living	55.13*	7.32	40.94	7.77
	Communication	57.04*	6.28	42.75	11.52
Conners 3	Inattention	48.96*	8.34	73.31‡	10.21
	Hyperactivity-Impulsivity	48.08*	6.49	76.81‡	14.25
	Learning Problems	46.08*	7.98	67.69†	14.40
	Executive Function	49.83*	8.27	66.06†	11.33
	Defiance/Aggression	47.67*	5.79	60.94	16.29
	Peer Relations	52.04*	12.64	66.31†	19.07
	DSM-5 ADHD Inattentive	49.13*	7.60	72.00‡	9.77
	DSM-5 ADHD Hyperactive-Impulsive	48.54*	7.34	75.25‡	15.55
	DSM-5 Conduct Disorder	46.71*	4.25	53.69	11.11
	DSM-5 Oppositional Defiant Disorder	49.08*	6.31	66.19†	15.49
	Conners 3 ADHD Index	20.71*	16.13	85.50‡	13.02
	Conners 3 Global Index	47.42*	15.04	73.19‡	10.51
DCDQ'07	Control During Movement	26.92*	4.43	22.56	6.95
	Fine Motor Skills	17.00*	3.72	12.50	5.09
	General Coordination	21.33*	4.01	16.69	5.28
	Total	65.25*	9.90	51.75	15.24

Note. Statistically significant between-group difference using ANOVA, *$p < .05$. † = At-Risk, Abnormal, or Elevated problems. ‡ = Clinically Significant, Abnormal, or Very Elevated problems.

Control and ADHD participants were nearly equivalent in all areas of cognitive functioning, including visual-perceptual reasoning, verbal ability, working memory, processing speed, and general intellectual functioning. Academic performance was also

largely equivalent between groups, although participants with ADHD performed significantly lower on math computation relative to controls, $F(1, 38) = 7.55$, $p = .009$, $\omega^2 = 0.141$. Fine motor skill performance was similar between groups, although both groups' performance was lower than the normative sample used to derive scores. See Table 19 below for complete details pertaining to participant cognitive, academic, and fine motor skill performance.

Table 19

Participant cognitive, Academic, and Fine Motor Skill Performance

		Controls		ADHD	
		M	*SD*	*M*	*SD*
WISC-IV					
	Block Design	11.42	2.67	10.81	3.73
	Vocabulary	11.33	2.76	10.69	2.47
	Digit Span	11.33	3.90	10.31	2.05
	Digit Span Forward	10.46	3.92	9.19	2.81
	Digit Span Backward	10.50	3.41	9.44	3.20
	Symbol Search	10.46	2.92	9.50	2.48
	Estimated FSIQ	108.00	12.30	104.31	15.78
WRAT 4					
	Word Reading	110.00	16.24	102.69	15.92
	Spelling	110.21	17.14	100.50	17.10
	Math Calculation	103.08*	16.98	89.75	11.42
Grooved Pegs					
	Dominant	45.20	11.60	42.18	8.68
	Non-Dominant	42.91	18.73	41.26	9.81

Note. Statistically significant between-group difference using ANOVA, *$p < .05$.

Identification of Potential Covariates

Bivariate correlational analysis was conducted to identify potential covariates for subsequent data analyses. In order to avoid the identification of potentially spurious correlations, only global variables determined to be reasonably related to dependent and outcome variables (i.e., Low Cognitive Control, High Cognitive Control, Beginning,

Ending, and Change) were selected. Variables investigated as potential covariates included Age, Socioeconomic Status (SES), and WISC-IV Estimated FSIQ. Age was significantly negatively correlated with Beginning fluency performance, r = -.432, p = .005, r^2 = .187, indicating that older participants had better fluency at the beginning of practice. A statistically significant negative correlation was also found between Estimated FSIQ and Beginning NJ performance, r = .373, p = .018, r^2 = .139, in which participants with higher Estimated FSIQs had better fluency at the beginning of practice. There was, however, no association found between Age and Estimated FSIQ, r = -.007, p = .965. Given that no significant group differences were evident on any of these variables, none were entered as covariates for any of the below analyses. See Table 20 below for correlation matrix of variables investigated as potential covariates.

Table 20

Correlation Matrix of Variables Investigated as Potential Covariates (Pearson r)

	Low Cognitive Control	High Cognitive Control	Beginning	Ending	Change
Age (Years)	-.013	-.136	-.432*	-.288	.083
SES	.176	-.007	-.301	-.139	.027
FSIQ	-.020	0.42	-.373*	-.073	-.246

Note. SES = Socioeconomic Status. FSIQ = Estimated Full Scale Intelligence Quotient. *p < .05.

Results – Study 2

Analysis of assumptions. Independence of observations was again assumed given that tasks were administered individually and the novelty of the experiment. Sphericity was not assessed noting the presence of only two repeated measures and thus only one difference score. The assumptions of normality and homogeneity of variance were assessed in four conditions: all data, data without outliers (i.e., Low Cognitive

Control and High Cognitive Control NJ values with standardized residual Z scores of ≥|3|), all transformed data (using square root transformations as detailed above in Part I, Study 1), and transformed data without outliers. Consistent with pilot study analyses, data were non-normally distributed on the Low Cognitive Control and High Cognitive Control DVs as indicated by statistically significant Shapiro-Wilk tests and inspection of histograms. Non-normality persisted even after removing data points determined to be outliers and using data transformation methods. Homogeneity of variance was also violated in all cases except when outliers were removed and data were not transformed, but non-normality and significant problems with kurtosis were evident. Together, it was determined to continue with the main analysis using Mixed Design Repeated Measures ANOVA and transformed data that included outliers. This was done to maximize power by including all participants and promote within-study interpretability of findings relative to those in Part I, Study 1. However, noting potentially significant violations of the assumptions of ANOVA, the original dataset (i.e., non-transformed variables with outlier data present) was subsequently analyzed with nonparametric statistics in order to provide evidence to further support or refute findings based on parametric analyses. See Tables 21 and 22 below for statistics used to assess the assumptions of normality and homogeneity of variance.

Table 21

Analyses of Assumptions for Pre-Transformed Variables – Part II, Study 2

	With Outliers (N = 40) (Control n = 24, ADHD n = 16)				Without Outliers (N = 38) (Control n = 23, ADHD n = 15)			
Task	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test
Low Cognitive Control	***2.66***	***7.18***	***<.001***	.842	***2.52***	***6.20***	.19	.13
High Cognitive Control	1.80	***3.65***	***<.001***	***.005***	1.47	1.88	***.04***	.10

Note. Bold and italicized values within the table represent violations of assumptions (skewness ≥ |2|, kurtosis ≥ |3|, Shapiro-Wilk $p < .05$, and Levene's test of homogeneity of variance $p < .05$).

Table 22

Analyses of Assumptions Post Variable Transformations – Part II, Study 2

	With Outliers (N = 40) (Control n = 24, ADHD n = 16)				Without Outliers (N = 38) (Control n = 23, ADHD n = 15)			
Task	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test
Low Cognitive Control	1.80	2.55	***<.001***	.74	1.76	2.27	***<.001***	.10
High Cognitive Control	1.12	1.15	***.01***	***.01***	0.92	0.49	***.01***	.097

Note. Bold and italicized values within the table represent violations of assumptions (skewness ≥ |2|, kurtosis ≥ |3|, Shapiro-Wilk $p < .05$, and Levene's test of homogeneity of variance $p < .05$).

Primary data analysis. A 2 x 2 Factorial Mixed Design Repeated Measures ANOVA was conducted to compare the graphomotor fluency performance of control participants and participants with ADHD within the context of varying levels of cognitive control demands. Group membership (i.e., controls and ADHD participants) represented the between groups factor whereas level of cognitive control (i.e., Low Cognitive Control versus High Cognitive Control) represented the within groups factor. Consistent with Part I, Study 1 pilot results, a statistically significant main effect was found for level of cognitive control in which participants overall demonstrated greater graphomotor

dysfluency during the High Cognitive Control task (M = 15.29, SD = 4.49) relative to the Low Cognitive Control task (M = 8.23, SD = 6.69), regardless of group membership, $F(1, 38) = 37.00$, $p < .001$. The effect size was large, $\omega^2_{partial} = .474$. However, there was no statistically significant group main effect $F(1, 38) < 1.00$, $p = .559$, $\omega^2 = -.017$, and no statistically significant group by level of cognitive control interaction was present, $F(1, 38) < 1.00$, $p = .893$, $\omega^2 = -.013$, indicating both groups were similarly affected by the level of cognitive control demands present in each task. See Table 23 below for source information pertaining to this analysis and Figure 10 for a graphical depiction of results.

Table 23

Source Table for Part II, Study 2 Analysis using ANOVA

	SS	*df*	*MS*	*F*	*p*
Low vs. High Cognitive Control	949.42	1	949.42	37.00	<.001
Error term	975.00	38	25.66		
Cognitive Control x Group Interaction	0.47	1	0.47	0.02	.893
Error term	975.00	38	25.66		

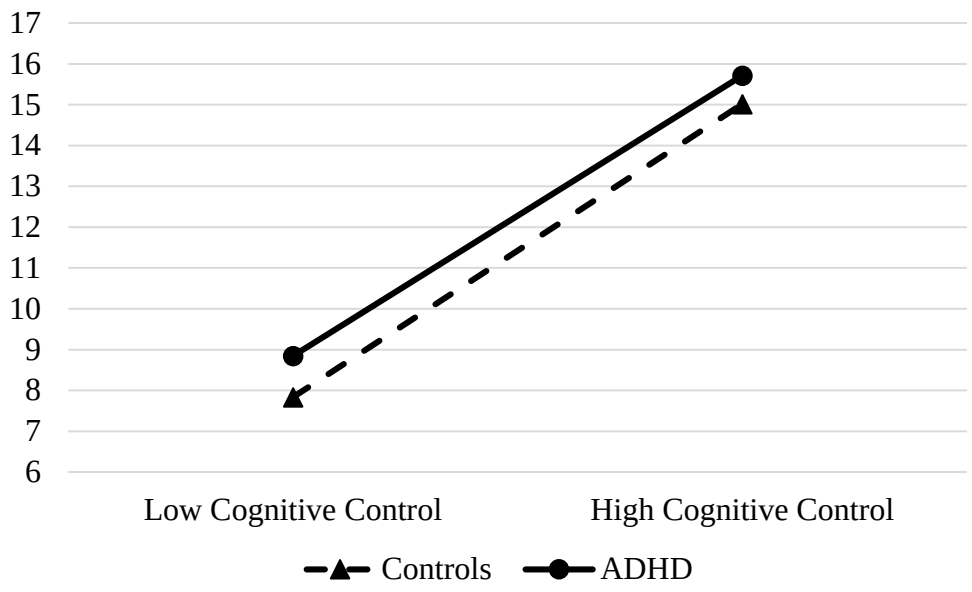

Figure 10. Main effect for level of cognitive control (horizontal axis) for each group (lines) on mean graphomotor fluency (vertical axis).

As previously noted, the assumptions of ANOVA were violated, thus raising concern for the interpretability and validity of parametric analysis. As such, follow-up nonparametric analyses were conducted using the Mann-Whitney U test for between groups comparisons and a Paired-Samples Sign test for within task analysis. Effect size was estimated using *r*, such that effect sizes of 0.1, 0.3, and 0.5 were interpreted as indicating a small, medium, and large effect size, respectively. A Bonferonni correction was applied to the originally proposed alpha level of .05 due to multiple comparisons, resulting in an adjusted alpha of .017. All assumptions of the Mann-Whitney U test and Paired-Samples Sign test were assessed and met (i.e., at least ordinal level DVs, a dichotomous independent variable, independence of observations, similarly shaped distributions, and, as unique to the Paired-Samples Sign test, difference scores were from a continuous distribution). Nonparametric findings were concordant with those of parametric analysis. There was a significant main effect for level of cognitive control, in

which greater graphomotor dysfluency was demonstrated by all participants during the High Cognitive Control task (*Mdn* = 14.93) relative to the Low Cognitive Control task (*Mdn* = 4.99), *T* = 6, *z* = -4.27 *p* < .001, and the effect size was large, *r* = -.67. The graphomotor fluency of participants with ADHD (*Mdn* = 4.88) was not significantly different from that of control participants (*Mdn* = 5.08) in the Low Cognitive Control condition, *U* = 185.00, *z* = -0.193, *p* = .859, *r* = -.03. Graphomotor fluency between ADHD (*Mdn* = 13.26) and control (*Mdn* = 15.24) participants was also not significantly different from one another when performing the High Cognitive Control task, *U* = 180.00, *z* = -0.331, *p* = .754, *r* = -.05. When viewed in light of mean rankings that were largely equivalent between groups and within conditions, findings appeared consistent with parametric analysis indicating no statistically significant group by level of cognitive control interaction. See Table 24 for additional summary information regarding nonparametric analyses.

Table 24

Summary of Nonparametric Test Results for Part II, Study 2

		Mann-Whitney U Test		Paired-Samples Sign Test		
	N	Mean Rank	Sum of Ranks	Negative Differences	Positive Differences	Ties
Low Cognitive Control						
Controls	16	20.21	485			
ADHD	24	20.94	335			
High Cognitive Control						
Controls	16	21.00	504			
ADHD	24	19.75	316			
High Cognitive Control – Low Cognitive Control				6	34	0
Controls				3	21	0
ADHD				3	13	0

Supplementary data analyses. Lastly, results were qualified by analyzing potential differences in the number of cognitive control errors and invalid trials based on observations between control and ADHD groups using a 2 x 2 Factorial Mixed Design Repeated Measures ANOVA. A statistically significant main effect was evident in that more cognitive control errors were committed during the High Cognitive Control task (M = 7.77, SD = 7.46) relative to the Low Cognitive Control Task (M = 3.10, SD = 2.25), $F(1, 38) = 17.96$, $p < .001$, $\omega^2_{partial} = .298$. There was, however, no statistically significant group by task interaction with regard to number of cognitive control errors committed, $F(1, 38) = 1.27$, $p = .267$, $\omega^2 = .005$. Conceptualizing invalid trials based on observations as an additional type of cognitive control error, these were added to previously described cognitive control error types to form a grand cognitive control error variable. A main effect for level of cognitive control was again observed in that significantly more grand cognitive control errors were made during the High Cognitive Control task (M = 9.63, SD = 7.52) versus the Low Cognitive Control task (M = 4.00, SD = 2.69), $F(1, 38) = 25.75$, $p < .001$, $\omega^2_{partial} = .382$, but there was still no significant group by task interaction, $F(1, 38) = 1.56$, $p = .219$, $\omega^2 = .009$.

Discussion – Study 2

Part II, Study 2 sought to examine the effects of systematically varying cognitive control demands on the graphomotor fluency and automaticity of participants with and without ADHD. Based on a review of the literature and Barkley's hybrid model of ADHD, it was proposed that all participants would demonstrate worse graphomotor fluency when engaging in the High relative to the Low Cognitive Control task. However,

participants with ADHD were hypothesized to be more negatively affected by these increased demands as indicated by a significant interaction effect.

Consistent with results from the pilot study in Part I, graphomotor fluency and automaticity as measured by NJ was negatively affected by increased cognitive control demands in that all participants demonstrated significantly greater dysfluency when performing the High Cognitive Control versus the Low Cognitive Control task. Further, a greater number of errors were committed during the High Cognitive Control task relative to the Low Cognitive Control task whether errors were conceptualized as cognitive control errors alone or in combination with trials deemed invalid by observation. These particular findings appear to represent an extension of the existing literature demonstrating greater dysfluency when executing well-learned graphomotor programs under conditions of increasingly greater, participant-driven cognitive control demands, such as writing under visually- or mentally-guided control (Tucha & Lange, 2005; Tucha et al., 2003). The greater number of errors committed by participants during the High Cognitive Control task further supports its identification as a more complex task, and increased frequency of errors could be reasonably predicted with increased task complexity.

Contrary to the main hypothesis of Study 2 and confirmed by both parametric and nonparametric statistical analysis, there was no differential effect of cognitive control demands on the graphomotor fluency of participants with ADHD relative to those without ADHD. Indeed, the graphomotor fluency of both control participants and participants with ADHD were similarly affected in terms of increased graphomotor dysfluency as cognitive control demands increased. Further, although the sample of

participants with ADHD in this study made more errors on average, this difference was not statistically significant and thus cannot be characterized as an actual difference between groups. Given the current data, it cannot be strongly argued that Barkley's hybrid model of ADHD extends to the graphomotor fluency domain as measured by kinematic analysis. However, limitations within the current study prevent firm conclusions from being drawn, but also highlight additional opportunities for future research.

The primary limitation of the current study is the relatively small sample of participants. Not only does this result in problems with sample bias and lack of generalizability (described further below), but small sample size also resulted in inadequate power to detect statistically significant group by task interaction effects for both primary hypotheses related to group membership and level of cognitive control demands (observed power β = .052) and potential interaction effects associated with the number of cognitive control errors committed by participants in each task type (observed power β = .196). Given the current data, it is also quite possible that the tasks used within this study – although adequate in eliciting significant differences in graphomotor dysfluency – were not sufficiently cognitively complex to elicit an interaction effect between ADHD and control participants. Together, future research examining the potential effects of cognitive control demands on graphomotor fluency and associated errors would benefit from samples sufficient in size and more cognitively challenging tasks that may be more sensitive to the neuropsychological sequelae associated with ADHD. Possible tasks that could be considered for future research and may be more challenging include dual activity tasks, tasks that incorporate distractions within the

current activity, or similar tasks with increased duration of tracking time to place greater demands on sustained attention and cognitive control. In addition, results of the current study were within the context of ADHD participants discontinuing stimulant medication. As such, the current methodology could also be used in future research to examine the effects of stimulant medication treatment on graphomotor fluency and error performance. Lastly, previous research has investigated the quality of graphomotor output within the context of medication status, with findings indicating poorer qualitative performance and improved kinematic performance when off medication. Although the number of errors could represent a proxy for quality of performance, these data do not adequately reflect the quality of the design produced by participants. As such, and within the context of medication status, future research would benefit from additional qualitative analysis of graphomotor output when completing similar animated tasks.

Despite predictions based on Barkley's model not being confirmed, results from the current study were consistent with other kinematic research involving children and adults with and without ADHD. For example, as demonstrated by Tucha and Lange (2001), children with ADHD who had discontinued stimulant medication produced similarly fluent graphomotor programs to those of peers without a diagnosis of ADHD. This occurred when participants wrote a common word on a digitizing tablet. As such, given current results, it could be said that whether executing a graphomotor task that is based on previously learned graphemes or animated patterns, the graphomotor fluency of children with ADHD who are not taking stimulant medication is similar to that of children without the disorder. In addition, as indicated by parent-report measures of inattention revealing significantly worse attention demonstrated by participants with

ADHD versus those without ADHD, results of the current study provide further evidence that graphomotor fluency appears independent of attentional functioning (Tucha, Mecklinger, Walitza, & Lange, 2006). Lastly, despite non-significant findings, the current study also has merit in that several possible graphomotor paradigms were systematically investigated and their effects on graphomotor fluency determined. In turn, other research could implement these protocols to investigate additional phenomena in clinical and non-clinical populations.

Although not formally investigated as a part of the current study, it is of note that despite child and adolescent participants (i.e., controls and ADHD combined) not producing a significantly greater proportion of invalid trials relative to pilot study participants when combining performance on both Low and High Cognitive Control tasks, $F(1, 114) < 1.00$, $p = .736$, $\omega^2 = -.008$, a statistically significant interaction was present in that child and adolescent participants produced a significantly lower proportion of valid trials as cognitive control demands increased, $F(1, 114) = 6.06$, $p = .015$, $\omega^2 = 0.042$. Even more, this finding was within the context of no statistically significant main effect for task type, $F(1, 114) = 2.885$, $p = .092$, $\omega^2_{partial} = .017$, and results remained constant even after removing participants with ADHD. Although child and adolescent participants completed more trials and in turn had more opportunity to make errors, results from pilot participants were significantly more affected by even a single error, thus potentially offsetting this viable confound. When viewed from the perspective of developmental neuropsychology, it is not unreasonable to speculate that increased errors would be committed by child and adolescent participants relative to young adults. For example, normative data from many neuropsychological tests indicate that it is typical for

younger patients to produce more errors than older patients. Indeed, this was also the case in the current study, in which age was significantly negatively correlated with total number of errors, r = -.412, p = 007, r^2 = .170, demonstrating this same effect. However, future research with samples sufficient in size to account for variance associated with participant age is necessary to clarify these specific findings. This is especially the case when noting the broad age range of child and adolescent participants used in the current study, which stretched between prepubertal years and adolescence. See Figure 11 below for a graphical comparison of proportion of valid trials between pilot and participants.

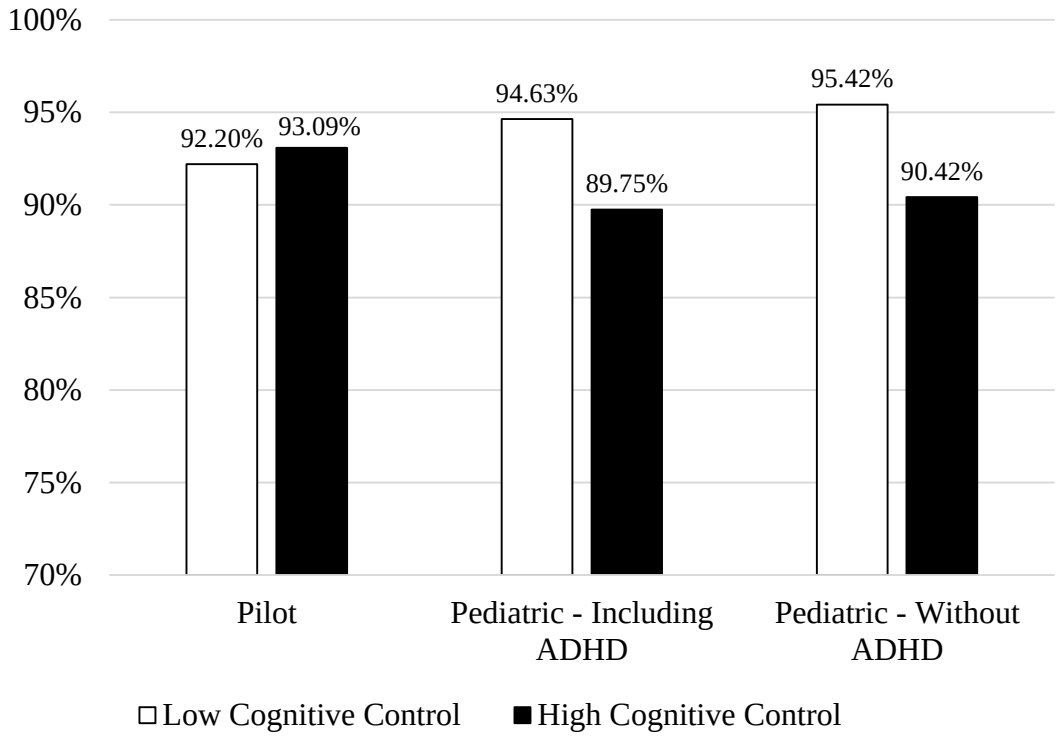

Figure 11. Comparison of Proportion of Valid Trials between Pilot Study Young Adults and Participants. Pilot = young adult participants from the pilot study (Part I). Pediatric = participants from experimental study (Part II).

Results – Study 3

Prior to the analysis of assumptions and primary data analyses, group sizes were equalized and participants were closely matched on age and general intellectual functioning. Matching of participants based on age and general intellectual functioning was conducted due to significant correlations found between the DV Beginning and participant variables of Age and Estimated FSIQ. Equalization of groups was completed to minimize potential concerns related to significant or non-significant findings due to issues associated with statistical power (β) resulting from different group sizes. See Table 25 below for selected descriptive statistics regarding the matched participant sample used for subsequent analysis in Study 3.

Table 25

Descriptive Statistics of Matched Participant Sample – Part II, Study 3

	Control			ADHD		
	n	*M*	*SD*	*n*	*M*	*SD*
Sex						
Female	8			5		
Male	8			11		
Handedness						
Right	13			14		
Left	3			2		
Learned Cursive						
Yes	12			11		
No	3			4		
Age		11.89	2.03		11.24	1.86
Estimated FSIQ		104.69	9.71		104.31	15.78
SES		40.41	14.02		40.31	11.15

Analysis of assumptions. Given the novelty of the experiment and lack of known systematic between-participant communication, independence of observations was assumed. The assumption of sphericity was not assessed noting the presence of only two repeated measures and thus only one difference score. The assumptions of normality and homogeneity of variance were assessed in four conditions: all data, data without outliers (i.e., Beginning and Ending NJ values with standardized residual Z scores of $\geq|3|$), all transformed data (using square root transformations as detailed above in Part I, Study 1), and transformed data without outliers. Beginning and Ending data were non-normally distributed as indicated by statistically significant Shapiro-Wilk tests and inspection of histograms indicating a highly positively skewed distribution. Non-normality persisted for Ending performance after removing outlier data and using data transformation methods, but normality improved for Beginning data under these circumstances. Homogeneity of variance was violated only for the Beginning DV, but was corrected after removing outlier data and/or with transforming data. Although violations of the assumptions of ANOVA were greatly reduced with data transformation and removal of outlier data points, the severity of non-normality combined with the small sample size and different distribution shapes of the data from the Beginning to the Ending of practice caused significant concern regarding the interpretability and reliability of results. As such, nonparametric statistical techniques were chosen for primary analyses.

Between-group comparisons were completed using the Mann-Whitney U test whereas within-task analysis was completed using the Wilcoxon Signed-Rank test. Effect sizes were estimated using *r*, such that effect sizes of 0.1, 0.3, and 0.5 were interpreted as indicating a small, medium, and large effect size, respectively. A Bonferonni correction

was applied to the originally proposed alpha level of .05 due to multiple comparisons, resulting in an adjusted alpha level of .017. All assumptions of the Mann-Whitney U test and Wilcoxon Signed-Rank test were assessed and met (i.e., at least ordinal level DVs, a dichotomous independent variable, independence of observations, similarly shaped distributions between groups, and, as unique to the Wilcoxon Signed-Rank test, difference scores that were symmetrical in distribution in each group). See Tables 26 and 27 below for statistics used to assess the assumptions of normality and homogeneity of variance.

Table 26

Analyses of Assumptions for Pre-Transformed Variables – Part II, Study 3

	With Outliers (N = 32) (Control n = 16, ADHD n = 16)				Without Outliers (N = 31) (Control n = 16, ADHD n = 15)			
Task	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test
Beginning	***2.58***	***8.29***	***<.001***	***.035***	1.60	***3.12***	***.001***	.320
Ending	1.88	***3.14***	***<.001***	.229	2.02	3.67	***<.001***	.229

Note. Bold and italicized values within the table represent violations of assumptions (skewness ≥ |2|, kurtosis ≥ |3|, Shapiro-Wilk $p < .05$, and Levene's test of homogeneity of variance $p < .05$).

Table 27

Analyses of Assumptions for Post Variable Transformations – Part II, Study 3

	With Outliers (N = 32) (Control n = 16, ADHD n = 16)				Without Outliers (N = 31) (Control n = 16, ADHD n = 15)			
Task	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test	Skew-ness	Kurtosis	Shapiro-Wilk	Levene's Test
Beginning	1.26	2.53	***.019***	0.196	0.60	0.58	0.527	0.678
Ending	1.11	0.75	***.007***	0.555	1.23	1.16	***0.003***	0.555

Note. Bold and italicized values within the table represent violations of assumptions (skewness ≥ |2|, kurtosis ≥ |3|, Shapiro-Wilk $p < .05$, and Levene's test of homogeneity of variance $p < .05$).

Primary data analysis. A statistically significant main effect was observed for practice in which graphomotor fluency and automatization improved from the Beginning

(Mdn = 525.66) to the Ending (Mdn = 313.11) of practice, $T = 6$, $z = -3.011$, $p = .002$, and the effect size was large, $r = -0.532$. However, as an individual group, participants with ADHD did not demonstrate a statistically significant improvement in graphomotor fluency from the Beginning (*Mdn* = 565.10) to the Ending (*Mdn* = 362.79) of practice, $T = 4$, $z = -1.810$, $p = .074$, $r = -0.32$. In contrast, control participants did show a statistically significant improvement in graphomotor fluency and automatization from the Beginning (*Mdn* = 428.49) to the Ending (*Mdn* = 248.76) of practice, $T = 2$, $z = -2.534$, $p = .009$, and the effect size was medium $r = -0.448$. The statistically significant main effect for practice combined with data indicating only control participants significantly improving from the Beginning to the Ending of practice suggested an interaction effect, but this interpretation was qualified with further nonparametric analyses. Results from follow-up analyses using the Mann-Whitney U test indicated no statistically significant between-group differences when comparing controls with ADHD participants at the beginning, $U = 104.00$, $z = -0.905$, $p = .381$, $r = -.160$, or the ending of practice, $U = 100.00$, $z = -1.055$, $p = .305$, $r = -.187$. When reviewing mean rankings that were largely equivalent between groups and within conditions, as well as negative and positive ranks with similar directionality, results did not appear to support a significant group by practice interaction effect. See Table 28 for additional summary information regarding nonparametric analyses, and Figure 12 for Box Plots demonstrating change in graphomotor fluency with practice.

Table 28

Summary of Nonparametric Test Results for Part II, Study 3

				Mann-Whitney U Test		Wilcoxon Signed-Rank Test		
	n	*Mean* NJ	*SD* NJ	Mean Rank	Sum of Ranks	Negative Ranks	Positive Ranks	Ties
Beginning								
Controls	16	535.16	322.93	15.00	240.00			
ADHD	16	834.83	840.58	18.00	288.00			
Ending								
Controls	16	406.47	400.20	14.75	236.00			
ADHD	16	590.79	593.84	18.25	292.00			
Beginning – Ending								
Main Effect						26	6	0
Controls						14	2	0
ADHD						12	4	0

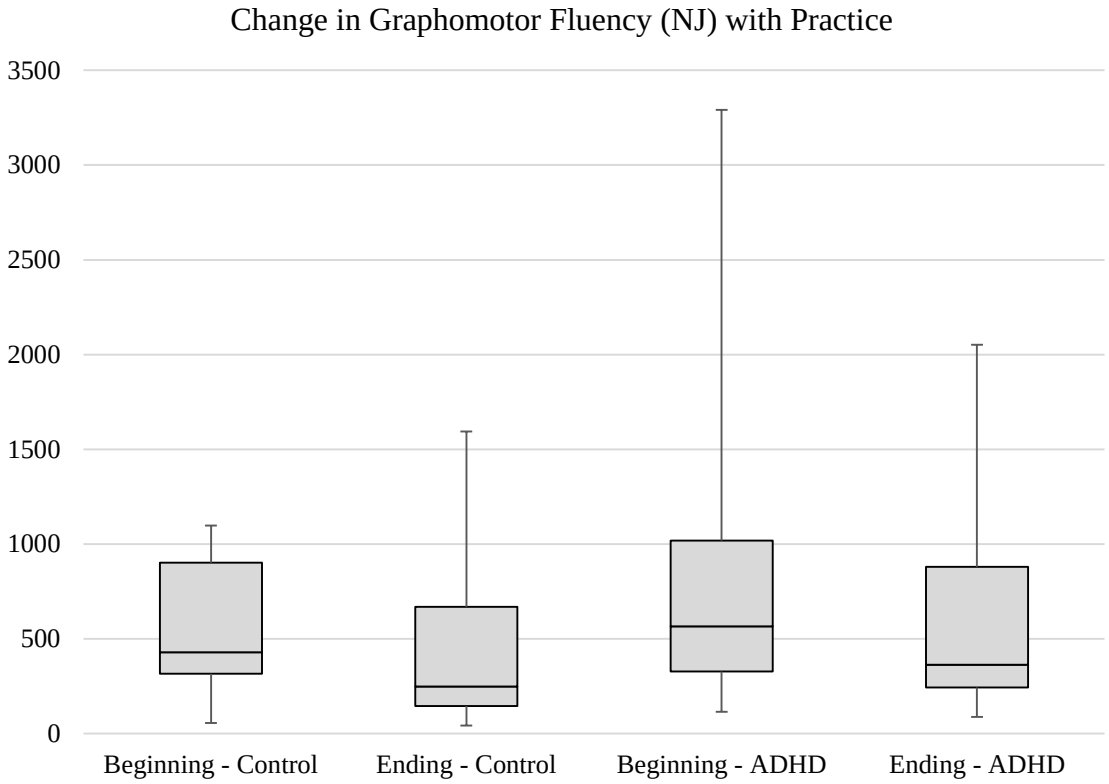

Figure 12. Change in graphomotor fluency performance with practice as measured by the mean Normalized Jerk (NJ) of the first 3 trials (Beginning) and mean NJ of the last 3 trials (Ending)

Discussion – Study 3

Using kinematic analysis of graphomotor fluency and automaticity, the present study sought to determine if participants with ADHD, as compared to children and adolescents without ADHD, would demonstrate similar ability to learn a novel graphomotor program given the same amount of practice. Control participants were expected to demonstrate a statistically significant improvement in graphomotor fluency, whereas ADHD participants – who had discontinued stimulant medication or were medication naïve – were hypothesized to show relatively reduced improvement. Results of the current study were consistent with previous research and hypothesized outcomes: control participants demonstrated a statistically significant improvement in graphomotor fluency and automaticity whereas participants with ADHD did not. These data were interpreted to suggest that children and adolescents with ADHD not taking stimulant medication may exhibit attenuated procedural learning while learning a new grapheme as compared to those without the disorder. The conceptualization of ADHD participant's procedural graphomotor learning as "attenuated" is emphasized: it is not to say no change occurred or that change did not occur in the expected direction. Rather, the procedural learning of participants with ADHD appeared attenuated relative to controls when noting that despite controls showing a statistically significant improvement in graphomotor fluency (i.e., $p = .009$) with a medium effect size (i.e., $r = -0.448$), participants with ADHD demonstrated a nearly statistically significant improvement (i.e., $p = .074$) with a medium effect size (i.e., $r = -0.32$). Combined with information indicating that groups were largely similar on relevant variables including age, SES, Estimated FSIQ, and experience with cursive handwriting, data appear most defensibly interpreted as

reflecting attenuated performance as opposed to no improvement or purely an artifact of low power and small sample size.

Kinematic analysis of handwriting conducted with digitizing technology, as was utilized in the current study, possesses the advantage of directly measuring motor skill acquisition with the use of an objective and quantifiable indicator of procedural learning over time. Methods used in the current study also possessed ecological validity when considering that handwriting is an important aspect of academic performance and children are actively involved in automatizing their handwriting. In addition, automated handwriting is often a required basal skill for future academic endeavors (e.g., being able to take notes while simultaneously paying attention to teacher instructions). However, as a result of several methodological factors, conclusions that children with ADHD exhibit attenuated procedural learning of a novel grapheme based on the current data are held only tentatively at this time pending additional research.

Small sample sizes inherently raise concern for sampling bias. This is indeed a significant consideration within the current study given that only two more control participants showed improvement in graphomotor fluency than did participants with ADHD. Small sample size may also create difficulties with replication and findings of equivalent effect sizes, which have been identified as ongoing concerns within psychological research (Open Science, 2015). In addition, although adequate statistical power and interpretability of data were achieved through nonparametric analysis given the current sample size and data characteristics, a much larger sample may yield more normally distributed data with greater homogeneity of variance that would in turn permit the use of more powerful parametric analyses. The small sample also consisted of a

relatively broad age range of participants, which also clouds interpretation from a developmental perspective. Given neurodevelopmental considerations associated with motor functioning between childhood and puberty, future research would benefit from large samples stratified by narrow age-bands to better control for and understand maturational factors involved in procedural graphomotor learning. Age stratification also becomes an important factor related to participant sex, as male adolescents (but not school-aged children) have been shown to demonstrate an advantage in motor learning compared with female adolescents (Dorfberger, Adi-Japha, & Karni, 2009).

The generalizability of these findings are also limited by the small number of participants with each presentation of ADHD (i.e., primarily inattentive, hyperactive-impulsive, or combined presentation). Effects, therefore, may only pertain to a certain subset of the ADHD population based on primary presentation, and sample sizes including larger numbers of participants with each presentation are needed for clarification. Generalizability is also limited noting that participants with ADHD in the current study demonstrated potentially greater diagnostic comorbidity than would be expected in a typical community sample, including a greater number of reported sleep problems. Most importantly regarding diagnostic comorbidity are limitations associated with the degree to which reduced automatization can be attributed to ADHD itself versus the presence of a comorbid condition. This is especially the case within the context of potentially comorbid DCD, of which 63% of participants screened positive. However, it is also of note that of the four participants with ADHD who did not demonstrate an improvement in graphomotor fluency, two were screened positive for DCD whereas two were not. Noting these observations and combined with other research demonstrating

difficulties with procedural motor learning and increased neuromotor noise in children with a diagnosis of DCD (Huau, Velay, & Jover, 2015), future research must include significantly larger sample sizes in order to determine which effects are related to ADHD versus DCD or other factors (e.g., sleep disturbance).

An additional note regarding generalizability pertains to the observation that participants with ADHD were largely medication naïve (n = 14). This has two primary but interrelated implications. First, at a basic level, findings may not be generalizable to those who have been actively treated with stimulant medication for some time (e.g., years) but discontinue its use for short periods of time. Second, it may follow that because most participants with ADHD were medication naïve, their symptomatology was less severe, thus not requiring intervention with medication. Some support for this assertion exists when contrasting normative cognitive data provided by the WISC-IV Technical and Interpretive Manual (Wechsler, 2003) with performance of ADHD participants in the current study. The WISC-IV Technical and Interpretive manual details the WISC-IV performance of 89 children and adolescents with ADHD between the ages of 8 and 13 years. Compared with these children and adolescents, participants within the current study performed slightly better on WISC-IV measures by an average of approximately 0.6 scaled score points on subscales, and approximately 6.7 standard score points on FSIQ. Performance by ADHD participants described in the manual and within the current study, however, was still in the average range overall (i.e., average scaled scores of 9.4 or better in each measure), and only one participant with ADHD in the current study demonstrated an impaired performance on any individual subtest (i.e., a scaled score of ≤5). In addition, the manual reported statistically significant differences

between ADHD and matched control participants on three of the WISC-IV subtests used within the current study (i.e., VC, DS, and SS) in which matched controls performed better. Although no statistically significant between-group differences were found within the current study, this is due to insufficient power within the current study to detect such a small effect. That being said, the subscale score differences between ADHD and control participants in both studies were similar in magnitude and directionality, and control participants in the current study also performed better than matched controls described in the WISC-IV Technical and Interpretive manual by an average of 0.4 scaled score points on subtests and 2.0 standard score points on FSIQ. Although cognitive data lends some support to the possibility that participants with ADHD in the current study are functioning better than may be expected and thus unmedicated, the caregivers of these same participants reported them as experiencing at-risk or clinically significant levels of ADHD symptomatology, learning problems, and/or psychosocial problems in most areas of functioning measured, thus making this potential confound with generalizability less likely. Nevertheless, this factor should be considered in future research. See Table 29 below for descriptive statistics comparing the average subscale and FSIQ performance of participants described in the WISC-IV Technical and Interpretive Manual with study participants.

Table 29

Descriptive Statistics Comparing WISC-IV Performance of ADHD Participants in the Current Study with Those Described in the WISC-IV Technical and Interpretive Manual.

	Controls		ADHD	
	Manual	Study	Manual	Study
Block Design	10.4	11.1	9.9	10.8
Vocabulary	10.9	10.6	9.9	10.7
Digit Span	10.5	10.8	9.6	10.3
Symbol Search	10.2	10.9	9.4	9.5
FSIQ	102.7	104.7	97.6	104.3

Note. Average scaled score performances on BD, VC, DS, and SS subtests. Average standard score performance on F. Manual = data reported in the WISC-IV Technical and Interpretive Manual. Study = data obtained within the current study.

Two additional methodological considerations that should be addressed in future research include the complexity of the grapheme used to examine procedural graphomotor learning, and participant experience with cursive handwriting. A significant proportion of trials were not missing at random. As described above in the initial analysis of valid trials, 20% of trials performed during the learning task were deemed invalid by observations (trials invalid by observation per participant: $M = 6.00$, $SD = 5.8$). In the sample of matched participants used for Study 3, approximately 22% of trials were removed prior to analyses due to invalid performance (trials invalid by observation per participant: $M = 6.59$, $SD = 6.12$). This relatively large number of invalid trials may have indicated that the novel grapheme was too complex for many participants. This is especially likely for younger participants given a statistically significant negative association between participant age and the number of trials deemed invalid based on observations, $r = -.433$, $p = .013$, $r^2 = .187$. In turn, results may have differed given a simpler design overall, and future research would benefit from an array of designs with increasing complexity based on neurodevelopmental considerations.

Although not a major concern with the current study given the nearly equal number of participants between groups who reported previously learning cursive handwriting, experience with cursive also has implications for participant's ability to learn the novel grapheme used in the current study given its similarity to letters formed with cursive writing. Given the current matched sample used for Study 3, there was a statistically significant group main effect in which participants who learned cursive (n = 23) demonstrated better overall mean graphomotor fluency across the 30 learning trials than did those who did not previously learn cursive (n = 9), U = 43.00, z = -2.536, p = .010, and the effect size was medium to large, r = -.448. As such, whether or not participants have experience with cursive must be a consideration when conducting future research, which is especially the case in North America noting that many schools no longer teach cursive handwriting as part of the standard curriculum.

In sum, results of the current study appear consistent with the literature indicating differences in procedural motor learning in those diagnosed with ADHD relative to those without the disorder. Given additional supporting research that addresses the limitations described above, findings may have clinical implications for (1) academic accommodations provided to children and adolescents whose performance appraisals depend upon handwriting (e.g., extended time to practice), (2) remedial interventions (e.g., additional time spent learning handwriting in order to improve automatization or how interventions can be tailored to address automatization) (for example, see Tucha & Lange, 2005), and (3) the use of kinematic analysis as a diagnostic tool to identify motor learning problems and/or the presence of ADHD in children and adolescents.

Results – Study 4

As described previously, the outcome variable (OV) of interest for Study 4 was the proportion of change between the Beginning and Ending of practice (i.e., Change), and proposed predictor variables (PVs) included verbal ability (VC), processing speed (SS), and fine motor skills (Pegs). Noting that no statistically significant difference or differences approaching significance were observed between control and ADHD participants on either the PVs (see Table 18 in the Participant Descriptive Statistics subsection of the Results and Discussion – Part II section) or OV, $F(1, 38) < 1.00$, $p = .706$, $\omega^2 = -.022$, subsequent analyses were conducted with the entire sample of 40 participants.

Prior to conducting multiple regression analysis (MRA), bivariate correlational analysis was performed to determine if proposed PVs were related to the OV of interest, Change. Although prior research was interpreted to suggest that each of these variables would yield predictive ability, none of the proposed predictor variables were significantly associated with Change in kinematic graphomotor fluency as measured by NJ. As such, MRA was not performed and results are discussed below. See Table 30 below for correlational data and Table 31 for descriptive statistics for Change in graphomotor fluency and automatization per group.

Table 30

Correlation Matrix of Predictor and Outcome Variables

	Outcome Variable
Predictor Variables	Change: Proportion of Change in Graphomotor Fluency
VC: Verbal Abilities	*Pearson r* = .016 *p* = .922
SS: Processing Speed	*Pearson r* = .170 *p* = .783
Pegs: Fine Motor Skills	*Pearson r* = -.305 *p* = .056

Table 31

Proportion of Change in Graphomotor Fluency and Automatization per Group

	Controls		ADHD	
	M	*SD*	*M*	*SD*
Change	16.43%	43.58%	5.88%	87.4%

Discussion – Study 4

Part II, Study 4 sought to determine which neuropsychological constructs might best predict change in graphomotor fluency in participants who were learning a new grapheme. As informed by previous literature, neuropsychological abilities hypothesized to predict change in graphomotor fluency included verbal skills, processing speed, and fine motor skills. Results of the current study indicate that none of these variables appear significantly associated with relative improvement in graphomotor fluency and automaticity in participants when learning a new grapheme. However, results are currently conceptualized as inconclusive due to limitations associated with study design and participant characteristics.

Regarding study design, participants were asked to practice a new grapheme over 30 trials. During practice, approximately 20% of all trials were deemed invalid by

observation for various reasons, which included incorrectly reproducing the grapheme, attempts to start over when errors were made despite being instructed not to do so, or attempting to erase or redraw various parts of the figure. As such and as mentioned previously, it is likely that the novel grapheme used within the current study was too complex for participants. This was especially the case for younger participants, noting a statistically significant positive correlation observed between participant age and the number of valid trials produced, $r = .450$, $p = .004$, $r^2 = .203$. As such, future research would benefit from a less complex grapheme or graphemes of varying complexity administered based on age.

Participant characteristics most likely confounding results within the current study include age and prior experience with cursive writing. Although age was not significantly associated with the proportion of change demonstrated by participants (both before and after removing one outlier), $r = .082$, $p = .615$, $r^2 = .007$, a statistically significant correlation existed between participant age and Beginning, $r = -.458$, $p = .003$, $r^2 = .210$, and Ending graphomotor fluency performance, $r = -.431$, $p = .006$, $r^2 = .186$. These correlations indicated that older participants had better fluency as indicted by lower NJ at both end points of practice. When all trials were averaged together for an overall measure of automatization across the 30 practice trials, a statistically significant negative correlation existed, again demonstrating better graphomotor fluency with age, $r = -.351$, $p = .026$, $r^2 = .123$. Considering this, raw score changes would thus be lower in older participants and greater in younger ones. These observations and assumptions are also consistent with previous research indicating maturation as being associated with improved automaticity as measured by kinematic variables (Accardo, Genna, & Borean,

2013). There is also evidence that a curvilinear association exists between age and graphomotor fluency, in which fluency and automaticity improve with age into young adulthood and then decline in older adulthood (Adi-Japha & Freeman, 2001; Mergl et al., 1999). Although utilizing proportion of change as the operational definition of Change in graphomotor fluency and automatization provided the advantage of a more equivalent measure of performance fluctuation that attempted to control for participant differences based on age, experience, and intraindividual variability, this method likely masked potential explanatory variance that would more appropriately be captured by analyzing raw score differences of participants stratified by narrow age bands. Indeed, previous research has demonstrated different rates of improvement in motor skill learning based on age due to initially poorer performance in school-aged children relative to adults (Julius & Adi-Japha, 2015). Together, future research should be conducted with narrow-age bands of participants in order to more definitively and accurately interpret data related to change in graphomotor fluency with practice.

In addition, casual observation garnered suspicion that participant experience with cursive writing may have had a significant impact on graphomotor fluency performance and Change with practice. This appears confirmed, at least tentatively, by nonparametric analysis indicating that participants who reported having learned cursive handwriting (n = 30) demonstrated a significant improvement in graphomotor fluency, $T = 8$, $p = .005$, r = -.354, whereas participants who reported having not learned cursive handwriting (n = 10) demonstrated no such significant improvement, $T = 4$, $p = .377$. However, participants who learned cursive (M age = 12.45 years) were significantly older than those who did not (M = 9.86 years), $F(1,38) = 18.10$, $p < .001$, $\omega^2 = 0.299$, thus further complicating the

interpretation of these findings. Lastly, the correlation between Pegs performance and Change achieved statistical significance after including only those who learned cursive, r = -.445, p = .014, r^2 = .198.

In conclusion, it is possible that the failure of any of the proposed neuropsychological abilities to predict improvement in graphomotor fluency is due to Change being related to an aspect of neurocognitive functioning that is completely unassociated with verbal ability, processing speed, and fine motor skills. This may be tenable when considering a construct such as verbal ability (although its close association to the construct of *g* could lead one to challenge this assertion), but it would be expected that a well-validated measure of fine motor skills (i.e., Grooved Pegboard) would reasonably predict performance on a task such as handwriting, which clearly involves fine motor speed and dexterity. In addition, other research has shown that change in quantitative aspects of handwriting may not be consistently related to any single factor, and other neuropsychological abilities, such as visual-motor integration, may be more appropriate for predicting Change in graphomotor functioning (Brossard-Racine et al., 2015). Nevertheless, firm conclusions cannot be drawn at this time regarding the inability of the proposed neuropsychological factors to predict improvement in graphomotor fluency with practice due to the methodological confounds, the broad age range of participants who took part in the current study, and differences in experience associated with cursive handwriting discussed above. See Figure 13 below for a scatter plot diagram highlighting the differences in change in graphomotor fluency based on participant age.

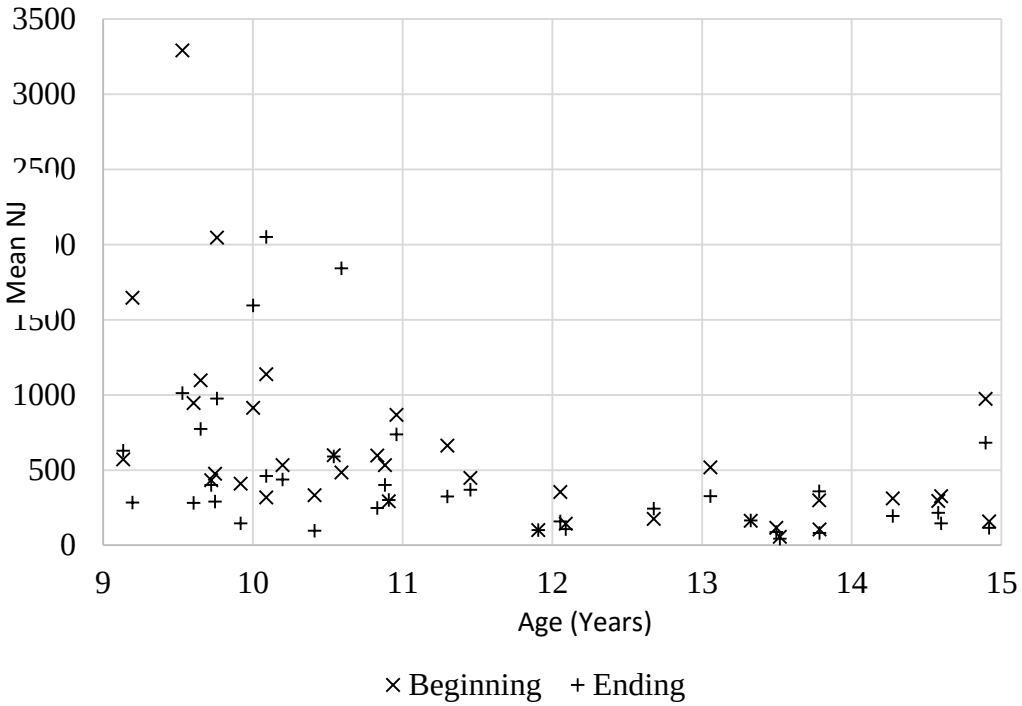

Figure 13. Graphomotor Fluency and Automatization Plotted by Age. Age in years (X axis) and NJ (Y axis).

Results – Study 5

Relative change in graphomotor fluency with practice (i.e., Change) was conceptualized as the test variable (TV) of interest to differentiate those with and without ADHD. No between group difference was present in the proportion of Change from the Beginning to the Ending of practice when comparing participants with and without ADHD, $F(1, 38) < 1.00$, $p = .706$, $\omega^2 = -.022$. As a result, Change could no longer reasonably be assumed to represent a viable TV to differentiate those with and without the disorder. This was confirmed by ROC Curve analysis, which indicated that Change had no predictive ability in differentiating controls and ADHD participants, *AUC* = .510, CI: .318-.703, $p = .912$. See Table 32 for selected sensitivity and specificity data, and Figure 14 for the ROC Curve diagram.

Table 32

Sensitivity and Specificity for Change in Graphomotor Fluency in the Identification of ADHD

Positive if Change ≥:	Sensitivity	Specificity
-379.9%	1.000	0.000
-94.2%	0.938	0.042
-41.6%	0.875	0.125
-6.6%	0.813	0.208
0.5%	0.750	0.292
4.4%	0.688	0.292
17.9%	0.625	0.375
23.3%	0.563	0.417
24.7%	0.500	0.500
26.8%	0.438	0.542
38.2%	0.375	0.750
51.6%	0.313	0.792
57.0%	0.250	0.875
66.7%	0.188	0.917
74.8%	0.125	1.000

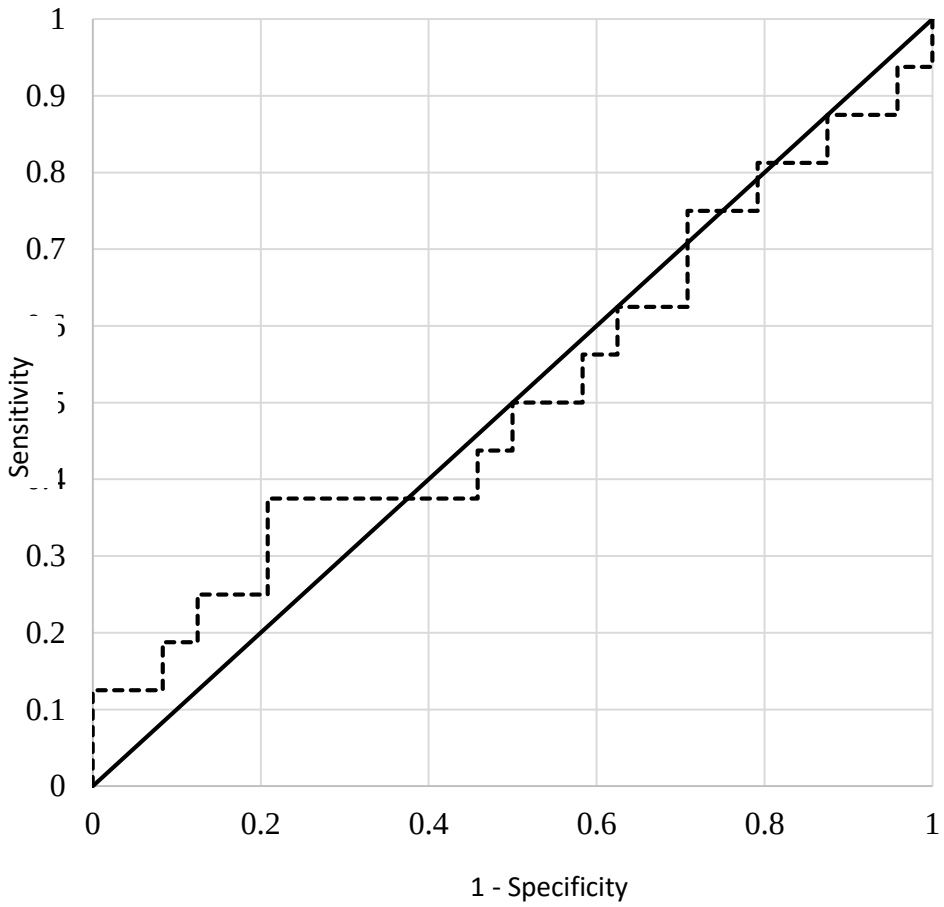

Figure 14. ROC Curve Analysis with Change (i.e., proportion of change from the beginning to the ending of practice) in graphomotor fluency as the test variable to predict the presence of ADHD.

Discussion – Study 5

Study 5 sought to determine the predictive ability that relative change in graphomotor fluency would have in identifying a sample of participants as having or not having ADHD. Results of the current study indicate that Change in graphomotor fluency offers no predictive ability to identify children and adolescents with ADHD. Although results are not encouraging with respect to the use of this measure's ability to identify ADHD in child and adolescent populations, the small sample size and associated participant characteristics preclude the ability to draw definitive conclusions.

Given the available data, it could be reasonably argued that the lack of significant findings is in accordance with the current literature indicating limited ability of direct measures of neuropsychological functioning to identify those with and without ADHD. However, beyond the small sample size used in the current study, perhaps the greatest factor limiting the ability to draw firm conclusions was the large age range of participants. As mentioned previously within the Discussion section of Study 4, younger individuals relative to older ones tended to show significantly greater gains with practice due to initially poorer performance. Although control and ADHD participants were not statistically significantly different in age, combining individuals despite known developmental differences based on age creates significant confounds in interpretation and likely eliminates potential predictive or explanatory variance. Further, children with less well-developed neuromotor systems may in fact show a greater proportion of change from the beginning to the ending of practice due to initially poorer performance. As such, additional research with larger samples of children and adolescents within narrow age bands would be necessary to draw firm conclusions about the ability of relative change in graphomotor fluency to predict ADHD in child and adolescent populations. In addition, developing normative datasets of graphomotor fluency measures with children who are unaffected by neurodevelopmental disorders based on beginning, ending, and overall performance would also be beneficial for identifying procedural learning difficulties.

The hypothesis for Study 5 was based on previous research indicating some ability for relative change in graphomotor fluency to predict the presence of ADHD in adults (Duda, Casey, & McNevin, 2014). Although this study in itself has its own limitations (e.g., small sample size), an additional potential explanation for between study

differences in results is that relative change in graphomotor fluency and the identification of reduced development of automaticity is more sensitive in adult samples due to neurodevelopment that is largely complete. As such, relative change in graphomotor fluency as a predictive measure may simply be less sensitive in children due to normal variability of neurodevelopment of motor systems in children in general, and the protracted development of motor systems in children with ADHD in particular.

Summary and Major Findings

Extensive research has identified ADHD as a neurodevelopmental disorder with a complex etiology and diverse behavioural and neuropsychological manifestations. Although many advances have been made in the conceptualization and diagnosis of ADHD, opportunities remain to better understand the disorder and improve diagnostic clarity and specificity.

The present study sought to provide a greater understanding of the neurodevelopmental aspects of ADHD associated with cognitive control and graphomotor function, as well as inform current diagnostic methodology by examining the ability of a neuropsychological construct (i.e., detected change in graphomotor procedural learning) to identify children and adolescents with ADHD. Pilot study data clearly identified two task paradigms that elicited the greatest and least amount of graphomotor dysfluency based upon dimensions of speed and figure complexity and cognitive components. These were in turn conceptualized as representing tasks of "high" and "low" cognitive control demands, although these tasks may also be viewed as representing tasks of high and low neuromotor complexity with cognitive components. Results were inconsistent with predictions based upon Barkley's hybrid model of ADHD,

but consistent with kinematic research in that participants with ADHD not taking stimulant medication produced graphomotor fluency that was similar to that of controls. This occurred regardless of task complexity and associated cognitive control demands. In addition, findings were consistent with the literature and interpreted to suggest that children and adolescents with ADHD, on average, may automatize graphomotor programs more slowly than those without the disorder. These specific findings, however, were held tentatively noting methodological limitations. No single neuropsychological factor or group of proposed factors was found to predict improvement in graphomotor fluency with practice. These findings were also interpreted as inconclusive noting confounds associated with the wide age range of participants, associated developmental considerations, complexity of the novel grapheme learned, and participants' previous experience (or lack thereof) with cursive handwriting. Lastly, the proposed neuropsychological construct of change in procedural learning, as measured by kinematic analysis, did not demonstrate any diagnostic utility in identifying participants with ADHD. However, given developmental considerations and participant characteristics, these negative findings were conceptualized as inconclusive at this time.

Perhaps of greater importance than the statistically significant and non-significant findings described within the current dissertation was the identification of other important variables that must be considered when conducting graphomotor research within a neurodevelopmental framework. Factors such as age, complexity, and prior experience – as well as their interactions – must be considered in order to make accurate and nuanced interpretations. Future research must consider these variables in order to produce data that are both interpretable and valid.

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Appendices

Appendix A

Experimental Administration Verbiage – Part I, Study 1

1. "Pick up the pen and write on the screen. You can write your name or draw a picture."
    A. If holds in a position natural for handwriting: "For everything you do today with the tablet, hold the pen just like that; just like if you were writing with pencil and paper, and only using your [DOMINANT] hand."
    B. If not holding in a position natural for handwriting: "Is that how you hold a pen to write?" Once adjusted, use instructions above (A).
2. "Just so you know, the buttons on the pen are disabled. So, it doesn't matter if you do or don't push them."
3. "Also, these tablets are used by cartoon artists and a lot of other people. I tell you that because I want you to know that it's pretty sturdy and it's ok to rest your arm on it as you write."
4. "I'm going to first explain to you how these first tasks work, and then we're going to practice. So just put the special pen down and watch me for a moment."
5. "For each of these next tasks, you're going to see two colored boxes on the screen: one green and one red."
6. "You will always move from your RIGHT to your LEFT; green to red."
7. "For all but the freehand task – which I'll explain in a moment – you'll be waiting for a black bar to appear on the screen. That's the cursor." <Hold up a pen to use as a prop for the cursor>

8. “As soon as the cursor appears on the screen, you’ll touch the center of the green square with the tip of the pen and follow behind the cursor.”
9. “You want to follow the cursor as close to it as you possibly can without touching it or going past it. You also want to keep it at about the same distance the whole time.” <Demonstrate with pen and finger>
10. “As you follow along, you want to stay at the middle point of the cursor: not too high or too low.” <Demonstrate with pen and finger>
11. “Then, you’ll stop at the center of the red box.”
12. “For the Free Hand task, you’ll simply draw a straight line connecting the center of the green box to the center of the red box, as quickly as you can while remaining accurate.”
13. “Now, here are a couple of tips and rules and then we’ll start.”
14. “Do NOT touch the screen with the pen until the cursor appears. Have it about this high off the screen.” <Demonstrate about 3cm from tip of pen to the screen> “It’s ok to rest your arm on the tablet while you wait.”
15. “Once the cursor appears and you touch the pen to the screen, do not lift up until you have completed the task. As soon as you get to the center of the red box, lift up the pen and go back to the starting point and wait for the next cursor to appear.” <Demonstrate>
16. “Let’s practice!”

Appendix B

Interview Questionnaire

Participant ID:		What is your current marital status?	
What is your (your child's) birthday?		What are the occupations (or highest previous occupation) of all caregivers (e.g., parents)?	
How would you (your child) describe your (their) sex or gender?		What is the highest level of education for each caregiver?	
What hand do you (does your child) primarily use to write with?		What is the combined annual household income?	
Do you (does your child) currently have a diagnosis of ADHD?		How many hours of sleep did you (your child) get last night?	
If yes, what is your (your child's) specific diagnosis (e.g., ADHD-C, -PI, -HI)?		How would you describe your (your child's) sleep last night qualitatively? Good, fair, or poor?	
Did (you) your child learn English as a second language?		Would you describe last night's sleep as typical or atypical compared to normal? Normal, more sleep than normal, or less sleep than normal?	
What language do you (does your child) speak most fluently?		What term do you (does your child) use to describe your (their) race or ethnicity?	

What medications are you (is your child) currently taking? Please include dosage information and when began.

Medication	Dosage	Purpose	Date began (MM/YYYY)

Do you (does your child) have a current diagnosis or diagnoses affecting the central nervous system or peripheral nervous system that would impair your ability to take part in a writing task?

_____

Do you (does your child) have any chronic medical condition that may affect cognitive functioning (e.g., sickle cell disease, diabetes mellitus) or psychiatric diagnosis (e.g., LD, ODD, DCD, anxiety, Depression)? If so, what are they?

_____

Is there any other information that you (your child) feel may affect your (your child's) participation in this study that you would like me to know?

_____

Screening for Sleep Problems (BEARS)

B: Does your child have any problems going to **B**ed or any problems falling asleep? _____

E: Does your child show symptoms of **E**xcessive daytime sleepiness (seem sleepy during the day and/or have difficulty waking up in the morning)? _____

A: Does your child **A**waken during the night or have any unusual behaviours during the night? _____

R: Does your child have a **R**egular sleep schedule and get enough sleep? _____

S: Does your child **S**nore or have any problems breathing during the night? _____

Appendix C

Experimental Administration Verbiage – Learning task

1. "This next task is going to be a little bit different. You're going to learn how to write the word 'hello' using a language that we made up. <show the participant the symbol on the card and place it on the screen>"
2. "What I want you to do is practice writing this word inside the box that will appear on the screen. You'll write from your left to your right, just like you normally do in English."
3. "It doesn't have to be perfect, but try to make it look as close to this as possible and about the same size."
4. "Also, keep the pen down on the tablet the whole time and don't fix or touch up any errors because you'll have lots of chances to practice. Just keep trying your best each time, but make sure you keep the whole word inside the box."
5. "Now, the computer needs to reset after every time you write the new word, so be sure to let it have time to reset before you start writing again."
6. "Lastly, you can move this little card here with the word to anywhere you want on the screen. Just make sure you can see it well enough. Any questions?"
7. "Just so you know, I'll be standing here over your shoulder watching you write. I don't do that to be weird! I just need to keep an eye on things and make sure the computer doesn't freak out or anything. Ok, let's begin!"

Appendix D

Handwriting Observations Form

Participant ID:

Low Cognitive Control								
		Cognitive Control	Invalid					
Trial #	Valid	# Touched	Too soon	Start-stop-restart	Wrong pattern	Time out (long)	Time out (short)	Other
1								
2								
3								
4								
5								
6								
7								
8								
9								
10								
11								
12								
13								
14								
15								
16								
17								
18								
19								
20								

High Cognitive Control								
		Cognitive Control	Invalid					
Trial #	Valid	# Touched	Too soon	Start-stop-restart	Wrong pattern	Time out (long)	Time out (short)	Other
1								
2								
3								
4								
5								
6								
7								
8								
9								
10								
11								
12								
13								
14								
15								
16								
17								
18								
19								
20								

Novel Symbol Learning						
		Invalid				
Trial #	Valid	Start-stop-restart	Wrong pattern	Timeout (long)	Timeout (short)	Other
1						
2						
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