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Development of graphomotor fluency in adults with ADHD: Evidence of attenuated procedural learning

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ABSTRACT

Purpose: The present study sought to determine if adults with ADHD demonstrate reduced graphomotor learning relative to controls.
Method: Twenty-eight control adults ($n = 14$) and adults with ADHD ($n = 14$) were recruited and wrote a novel grapheme on a digitizing tablet 30 times. Participants with ADHD were counterbalanced on and off stimulant medication.
Results: Control participants, $F(1,13) = 13.786$, $p = .003$, $\omega^2_{partial} = .460$, and participants with ADHD on medication, $F(1,13) = 10.462$, $p = .007$, $\omega^2_{partial} = .387$, demonstrated significant improvement in graphomotor fluency with equivalent practice whereas participants with ADHD off medication did not, $F(1,12) = 0.166$, *NS*.
Conclusions: Results indicate that graphomotor program learning in adults with ADHD may occur more slowly than typically developing peers. Findings have implications for providing accommodations to adults with ADHD, potential benefits of stimulant medication, and using digitizing technology as a neuropsychological assessment instrument.

1. Introduction

Attention-Deficit Hyperactivity Disorder (ADHD) is a heterogeneous neurodevelopmental disorder characterized diagnostically by symptoms of inattention, hyperactivity, and impulsivity (American Psychiatric Association, 2013). Current conceptualizations assert that most childhood ADHD persists into adulthood (Kooij et al., 2010) and a relatively recent meta-analysis estimated the prevalence of adult ADHD at approximately 2.5% (Simon, Czobor, Balint, Meszaros, & Bitter, 2009). Compared with unaffected peers, adults with ADHD tend to experience greater academic, psychiatric, social, occupational, and medical problems above and beyond diagnostic symptomatology (Barkley, 2006), including lower occupational level, greater unemployment, lower productivity, lower median annual salary, more frequent driving accidents, lower self-ratings of social functioning, higher psychiatric comorbidity, and a higher divorce rate (Klein et al., 2012; Kupper et al., 2012; Skirrow & Asherson, 2013; Yang, Tai, Yang, & Gau, 2013).

The etiology of ADHD is complex and multifactorial in nature, with research implicating interactions between genetic and environmental factors and their neurological corollaries as primary causal agents (Cortese, 2012; Koziol, Budding, & Chidekel, 2013; Kuntsi et al., 2014; McLoughlin, Palmer, Rijsdijk, & Makeig, 2014; Merwood et al., 2014). The complex phenotypic expression of ADHD is likely polygenic and dopaminergic, noradrenergic, serotonergic, cholinergic, and other neurotransmitter systems have been investigated (Cortese, 2012; Yang, Neale, et al., 2013). Significant attention, however,

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has been paid to the dopaminergic system and its functioning (Arnsten, Berridge, & McCracken, 2009; Banaschewski, Becker, Scherag, Franke, & Coghill, 2010; Biederman, 2005; Spencer et al., 2013).

Neuroimaging research using structural, functional, and functional-connectivity paradigms have converged to reveal widespread neuroanatomical differences in both children and adults with ADHD. The most replicated structural abnormalities in ADHD include volumetric reductions of the dorsolateral prefrontal cortex, anterior cingulate, basal ganglia, corpus callosum, and the cerebellum (Bledsoe, Semrud-Clikeman, & Pliszka, 2011; McAlonan et al., 2007; Seidman et al., 2006; Valera, Faraone, Murray, & Seidman, 2007). In addition, a recent meta-analysis identified reduced white matter structural integrity in all age groups of individuals with ADHD in more specific tracts, including the corona radiata, corpus callosum, left- and right-hemispheric internal capsule, and the left cerebellum (van Ewijk, Heslenfeld, Zwiers, Buitelaar, & Oosterlaan, 2012). When viewing these structural differences longitudinally, a developmental pattern emerges in which individuals with ADHD demonstrate regional specific maturational order that is similar to that of unaffected individuals but with an overall protracted developmental trajectory of cortical thickness that is particularly delayed in prefrontal regions (Sato, Hoexter, Castellanos, & Rohde, 2012; Shaw et al., 2007, 2013). Altered cortical, cerebellar, and white matter maturation has in turn been linked to increased ADHD symptomatology (Cortese et al., 2013; Ghassabian et al., 2013; Mackie et al., 2007; Shaw et al., 2013).

Functional neuroimaging findings have been largely concordant with structural findings associated with ADHD, implicating areas believed to be involved in executive functioning, attention, inhibition, and motor control (Brossard-Racine, Majnemer, & Shevell, 2011; Seidman et al., 2006; Shaw et al., 2006; Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998). A pair of recent meta-analyses of functional magnetic resonance imaging (fMRI) studies identified cortical and subcortical regions associated with deficits in 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). Lastly, functional connectivity studies investigating large-scale brain networks have provided support for the roles of the default mode, frontal-parietal, ventral and dorsal attentional, and sensorimotor networks in both the dysexecutive and motor control deficits associated with ADHD (Castellanos & Proal, 2012; Cortese et al., 2012; Koziol, Budding, & Chidekel, 2013; Mills et al., 2012; Wang et al., 2013).

Several non-diagnostic features have been identified in the ADHD population, including motor impairments and increased rates of neurological soft signs (Brossard-Racine et al., 2011; Chan et al., 2010; Cole, Mostofsky, Larson, Denckla, & Mahone, 2008; Iwanaga, Ozawa, Kawasaki, & Tsuchida, 2006). Consistently identified motor deficits include poor handwriting; decreased speed and accuracy of complex 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; Garcia Murillo, Cortese, Anderson, Di Martino, & Castellanos, 2015; Harvey et al., 2007; Meyer & Sagvolden, 2006; Piek, Pitcher, & Hay, 1999; Rosch, Dirlikov, & Mostofsky, 2013). Of particular interest are motor skill and procedural learning differences in the ADHD population.

ADHD has been characterized by some as a disability of a learning (Cutting, Koth, Mahone, & Denckla, 2003) even in the absence of a traditional, specific learning disability. 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). 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 those with ADHD. A study involving serial motor sequence learning in children with ADHD identified variable rates of learning and reduced priming effects that could not be attributed to poor perceptual-motor abilities (Barnes, Howard, Howard, Kenealy, & Vaidya, 2010). Similarly, a study of procedural learning in young women diagnosed with ADHD found evidence for "the notion of a latent memory consolidation phase in motor sequence learning in individuals with ADHD" (Adi-Japha, Fox, & Karni, 2011, p. 1017). Others have proposed that delayed skill acquisition in the ADHD population 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 (Adi-Japha et al., 2011; Burden & Mitchell, 2005; Lange et al., 2007). The complex networks underlying procedural learning and executive processes, which include cerebellar and frontal-striatal systems, may provide a neuroanatomical explanation for procedural learning and skill acquisition difficulties found in ADHD when considering that these systems are also implicated in the expression of ADHD symptomatology (Barnes et al., 2010; Koziol, Budding, Andreasen, et al., 2013; Koziol, Budding, & Chidekel, 2013; Lange et al., 2007; Leisman, Braun-Benjamin, & Melillo, 2014).

Procedural aspects of motor functioning in those with ADHD have also been studied using kinematic analyses of graphomotor functioning (i.e., handwriting). Kinematic analysis is used to analyze handwriting by objectively quantifying graphomotor movement changes over time (Viviani & Terzuolo, 1982) that in turn yields information that describes several aspects of handwriting, including degree of movement automatization (i.e., graphomotor program fluency) (Mergl, Tigges, Schroter, Moller, & Hegerl, 1999; Portier & Van Galen, 1992). In the case of ADHD, several studies have indicated that relative to controls, children with ADHD (but not adults) execute graphomotor programs in a more dysfluent, less automatized fashion, but only when taking prescribed dosages of stimulant medication (Flapper, Houwen, & Schoemaker, 2006; Tucha & Lange, 2001, 2004, 2005). In addition, children with ADHD who did not present with comorbid Developmental Coordination Disorder