Possible Underlying Mechanisms of Hyperactivity in Children with ADHD

Nicole Feirsen
The Graduate Center, City University of New York

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Nicole Feirsen, M.A.
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Date

Anil Chacko, Ph.D.
Chair of Examining Committee

Date

Maureen O’Connor, Ph.D.
Executive Officer

Supervisory Committee:

Anil Chacko, Ph.D.

Nancy Foldi, Ph.D.

Sarah O’Neill, Ph.D.

THE CITY UNIVERSITY OF NEW YORK
ABSTRACT

Possible Underlying Mechanisms of Hyperactivity in Children with ADHD

by

Nicole Feirsen

Advisor: Anil Chacko, Ph.D.

Conceptualizations of Attention-Deficit/Hyperactivity Disorder (ADHD) have evolved significantly over the years. Historically, early conceptualizations of ADHD described hyperactivity as the core symptom of the disorder. However, when the third version of the Diagnostic and Statistical Manual of Mental Disorders (DSM) was published (1980), hyperactivity became a specific qualifier of the disorder and was no longer necessary for a diagnosis. Despite this shift in conceptualization of the disorder, there is an abundance of empirical evidence, both recent and historical, supporting the fact that hyperactivity is an enduring and clinically impairing symptom domain in ADHD. However, much of the extant literature has focused on only the combined and inattentive subtypes and does not account for the purely hyperactive presentation. Despite having numerous validated instruments available to measure hyperactive behavior, most current models of ADHD are purely neurocognitive in nature and fail to sufficiently account for hyperactive symptoms. The aim of this study was to gain a deeper, more nuanced understanding of hyperactivity in children with ADHD, as many questions remain about the relationships between neuropsychological constructs and hyperactivity. A total of 130 participants with ADHD were enrolled and completed working memory and behavioral inhibition tasks while wearing actigraphs. Results showed that unmedicated children with working memory deficits display significantly higher levels of
activity than children without a deficit in this area; however, this relationship was no longer significant after controlling for basic attentional processes. There were no significant relationships between level of hyperactivity and behavioral inhibition. Implications of these findings will be discussed in detail.
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Part I: Introduction to ADHD

Attention Deficit/Hyperactivity Disorder (ADHD) is one of the most prevalent childhood psychiatric disorders and is characterized by the core symptom domains of persistent and developmentally inappropriate levels of inattention and/or hyperactivity/impulsivity, which cause clinically significant levels of impairment across multiple settings (APA, 2013). Specifically, to qualify for a diagnosis, individuals must exhibit at least six of the nine symptoms of inattention (e.g., difficulty sustaining attention, easily distracted, difficulty with organization) and/or six of the nine symptoms of hyperactivity/impulsivity (e.g., often fidgets, talks excessively, has difficulty awaiting turn, always “on the go”) by the age of twelve. According to the DSM-V, there are three distinct clinical presentations of ADHD: combined presentation (ADHD-C), predominately inattentive presentation (ADHD-I), and predominantly hyperactive/impulsive (ADHD-HI) presentation (APA, 2013). As each name suggests, specific patterns of symptoms are unique to each subtype; those with diagnoses of either the combined presentation or the primarily hyperactive/impulsive presentations display excessive levels of motor activity (e.g., tapping, running, talking), whereas those with the predominantly inattentive presentation do not. Symptoms must be present in multiple contexts (e.g., home and school) and should be observed by multiple collaterals (e.g., parents and teachers). Moreover, symptoms must cause impairment in social, academic, or occupational functioning (APA, 2013). It should be noted that the diagnostic information cited above is based on DSM-V criteria, but because the latest edition of the DSM was only published very recently, the data cited throughout the remainder of this paper was based on DSM-IV criteria.

The estimated global prevalence of ADHD in school aged children is approximately 5% (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). In the United States specifically, ADHD is the most commonly diagnosed psychiatric childhood disorder, and prevalence rates
have been estimated as approximately 8-9% (Merikangas et al., 2010). ADHD is more
commonly diagnosed in males than females, with an approximate 2:1 ratio (Rucklidge, 2010),
and females are more likely to present with inattentive rather than hyperactive symptoms
(Polanczyk, et al., 2007). The predominantly inattentive subtype is most frequently diagnosed
(approximately 38-57% of all cases), followed by the combined subtype and
hyperactive/impulsive subtype. Despite the inattentive presentation’s high incidence, children
diagnosed with the combined-type presentation are most likely to be referred for clinical services
(Willcutt, 2012).

ADHD is highly comorbid with other psychiatric disorders. Indeed, ADHD is frequently
associated with comorbid oppositional defiant disorder (ODD), conduct disorder (CD), tic
disorders, and mood disorders (e.g., major depressive disorder [MDD] and anxiety disorders)
(Agosti, Chen, & Levin, 2011). According to the DSM-V, ODD occurs in approximately half of
the population diagnosed with ADHD, and CD occurs in approximately one quarter of
individuals with ADHD (APA, 2013). Moreover, approximately 20-60% of children with
ADHD are also diagnosed with a learning disorder; indeed, they are at a higher risk for reading,
spelling, and math difficulties (Czamara et al., 2013). The presence of a comorbid psychiatric
disorder is predictive of significantly worse long-term outcomes (Agosti, et al., 2011).

Youth with ADHD display overall lower levels of academic achievement when compared to
typically developing controls (Frazier, Youngstrom, Glutting, & Watkins, 2007). They are also
more likely to have to repeat a grade in school and/or prematurely drop out of school (Fried et
al., 2013). Additionally, there is a substantial amount of empirical data that illustrates profound
social deficits associated with ADHD (Hoza, 2007; Landau, Milich, & Deiner, 1998; Nixon,
2001; Wheeler & Carlson, 1994). Evidence also suggests that parent-child interactions and
family relationships are strained in children with ADHD (Lifford, Harold, & Thapar, 2008). Children with ADHD are also more likely to develop substance abuse disorders and are more likely to develop nicotine dependence than their typically developing peers (Lee, Humphreys, Flory, Liu, & Glass, 2011). Moreover, youth with ADHD are more likely to sustain significant injuries (e.g., bone fractures, head injury) than their peers (Merrill, Lyon, Baker, & Gren, 2009), and they are also at a higher risk for being involved in traffic accidents (Pastor & Reuben, 2006). These various impairments are all predictive of negative long-term outcomes (Ollendick, Weist, Borden, & Greene, 1992; Woodward & Fergusson, 2000).

Although ADHD is most often diagnosed in childhood and is categorized as a childhood disorder, it persists into adulthood in approximately two thirds of cases (Turgay et al., 2012). Indeed, the core symptoms associated with ADHD (e.g., inattention, impulsivity, and hyperactivity), as well as mood dysregulation, are frequently seen in adults (Biederman, Mick, & Faraone, 2000). However, the presentation of these symptoms changes throughout the lifespan; while inattentiveness may remain relatively unchanged, hyperactivity varies across developmental stages (Biederman, et al., 2000). Whereas childhood hyperactivity frequently manifests as fidgeting, acting as if one was driven by a motor, and the inability to remain seated, in adulthood, hyperactivity is often experienced as inner restlessness or the inability to relax (Wolraich et al., 2005). Moreover, adult impulsivity is frequently demonstrated in the form of temper outbursts, sexual promiscuity, and impatience (Turgay, et al., 2012). Additionally, in children diagnosed with ADHD, hyperactivity is associated with poorer outcomes in education and occupation in adulthood (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993), indicating that symptoms continue to be impairing as children age. As such, it is likely that if the hyperactivity symptoms were to be reconceptualized and redefined across the lifespan as being
consistent with more developmentally appropriate criteria for adult behaviors, the rates of persistence into adulthood would be even higher (McGough & Barkley, 2004).

Although no precise etiology of ADHD has been identified to date, there are multiple factors that likely contribute to its development. Given that ADHD presentations are so variable, it is probable that several endophenotypes exist. Indeed, there is evidence that genetic, neurochemical, neuroanatomical, and neurocognitive factors are all related to the symptomatology of this disorder.

Strong empirical evidence exists supporting the fact that ADHD is heritable and has a genetic component. Family studies have documented that there is an increased frequency in which ADHD occurs in the biological relatives of a proband with ADHD when compared to the general population (Biederman et al., 1992; Faraone, Biederman, Keenan, & Tsuang, 1991). Twin studies have also been conducted to evaluate the genetic role in ADHD and have demonstrated that genetic factors contribute approximately 70-80% to the presentation of the disorder in international samples (Biederman & Faraone, 2005). Similar findings have also been documented in adoption studies (Sprich, Biederman, Crawford, Mundy, & Faraone, 2000). Although this field is still developing, several genetic markers including polymorphisms within dopamine transporter genes (e.g., DAT1), dopamine receptor genes (e.g., DRD4), serotonin transporter genes (e.g., 5-HTT), serotonin receptor genes (e.g., 5-HT2A), norepinephrine transporter genes (e.g., NET1), synaptosomal-associated proteins (e.g., SNAP25), and catalyzing enzymes (e.g., COMT) have currently been identified as potential etiological candidates for ADHD (Wallis, Russell, & Muenke, 2008). More recently, linkage studies have been conducted to identify more general locations on chromosomes that may contribute to ADHD rather than candidate genes in isolation. Several loci have been identified in these studies, suggesting that
certain chromosomes are likely to contain susceptibility genes for ADHD (Arcos-Burgos et al., 2004; Bakker et al., 2003; Hebebrand et al., 2006; Ogdie et al., 2004). However, it is important to recognize that these genetic factors do not occur in isolation, and the environment (e.g., childhood adversity, maternal prenatal smoking, maternal use of alcohol prenatally, etc.) likely interacts with genetic factors to produce the ultimate outcome (Brookes et al., 2006; Kahn, Khoury, Nichols, & Lanphear, 2003; Retz et al., 2008).

Neurochemical factors have also been implicated in the etiology of ADHD. Specifically, dopaminergic dysregulation has been proposed as one model of ADHD pathogenesis. In general, dopamine circuits are involved in the production of movement, behavioral modulation in response to rewards and reinforcements, and information processing; these domains are consistent with the domains that are often impaired in ADHD (Genro, Kieling, Rohde, & Hutz, 2010). In particular, nigrostriatal pathway dysfunction has been linked to hyperactive symptoms (Krause, Dresel, Krause, Kung, & Tatsch, 2000). Additionally, animal models using various methodologies (e.g., neurotoxins, knockout models, selective breeding) have demonstrated that modification of the DA systems can result in either hyperactivity or hypoactivity (van der Kooij & Glennon, 2007). Brain imaging studies have also shown that areas that are highly innervated by dopaminergic neurons (e.g., caudate and globus pallidus) are significantly smaller in patients with ADHD as compared to controls (Valera, Faraone, Murray, & Seidman, 2007). These structural differences are observed in childhood and continue into adolescence (Castellanos et al., 2002). Moreover, functional imaging studies have demonstrated hypoactivation of the ventral prefrontal and inferior parietal dopaminergic pathways during behavioral and cognitive tasks (Durston, 2003; Durston, Mulder, Casey, Ziermans, & van Engeland, 2006). As stated earlier, genetic studies also lend support to the dopamine dysregulation model. Additionally, data from
pharmacology research suggests that dopamine is involved in symptom presentation. Stimulant medication is the first line drug treatment for ADHD, and stimulants (e.g., methylphenidate) are believed to enhance the neurotransmission of DA by inhibiting the DA transporter, which allows for increased levels of DA to remain in the synapse (Engert & Pruessner, 2008; Spencer, 2004; Volkow et al., 2002).

The noradrenergic system has also been implicated in the pathophysiology of ADHD. Noradrenaline is projected throughout the prefrontal cortices and is believed to play an important role in higher-order cognitive functioning (Del Campo, Chamberlain, Sahakian, & Robbins, 2011; Robbins & Arnsten, 2009). These higher-order cognitive operations (e.g., working memory, inhibitory control, executive planning, etc.) are often impaired in children with ADHD (Chamberlain et al., 2011). Additionally, most of the first-line stimulant treatments (e.g., MPH, amphetamine) for ADHD increase the amount of NE available in the prefrontal cortex; moreover, atomoxetine, a selective norepinephrine reuptake inhibitor, has been empirically demonstrated to reduce ADHD symptoms while increasing NE levels in the prefrontal cortex (Del Campo, et al., 2011). In a similar vein, guanfacine, an α2-adrenergic receptor agonist, has also been shown as effective in treating ADHD symptomatology (Scahill et al., 2001).

Unfortunately, due to a lack of suitable PET radioligands, no functional imaging of this nature has been completed to date (Zimmer, 2009).

Developmental structural and functional neuroanatomical factors have also been documented in ADHD. Children with ADHD have smaller brain volumes than typically developing controls with prominent significant differences seen in the cerebellum (Valera, et al., 2007). Longitudinal data has shown that children with ADHD have reduced cortical thickness, particularly within the medial and superior prefrontal and precentral regions (which are associated with attentional
control), with normalization only in children with the greatest clinical improvement (Shaw et al., 2006). However, more recent data has indicated that gray matter reductions, specifically in the dorsal attentional network, limbic areas, right caudate, right thalamus, and bilateral cerebellar hemispheres, endure into adulthood regardless of symptom persistence (Proal et al., 2011). The fronto-striatal network has been linked to executive functioning and attentional control, and the parietal and temporal regions also appear to contribute to these functions (Rubia, Smith, Brammer, & Taylor, 2007). Functional neuroimaging studies have illustrated an association between the right inferior prefrontal cortex and caudate and performance on tasks of cognitive control in ADHD (Silk et al., 2005; Smith, Taylor, Brammer, Toone, & Rubia, 2006).

Additionally, significant patterns of generalized frontal hypoactivity (particularly within the anterior cingulate, dorsolateral prefrontal, and inferior prefrontal cortices) have been consistently identified in patients with ADHD during various executive and non-executive tasks (Dickstein, Bannon, Castellanos, & Milham, 2006; El-Sayed, Larsson, Persson, & Rydelius, 2002). Neuroimaging research has also garnered support for the role of the fronto-cerebellar circuit in ADHD. The cerebellum projects to the prefrontal cortex and the basal ganglia, both of which are implicated in ADHD. Functional MRI studies have shown cerebellar hypoactivity to be associated with deficits in cognitive control, timing, and working memory (Durston, van Belle, & de Zeeuw, 2011). Alterations in the cingulate cortex have also repeatedly been correlated with impairment in the domains of response selection, cognitive processing, response inhibition, error detection, and motivation in ADHD (Rubia, 2011; Smith, Taylor, Brammer, Halari, & Rubia, 2008). Additionally, diffusion tensor imaging (DTI) studies have consistently found disturbances in white matter integrity in children with ADHD. Indeed, when compared to healthy controls, significant white matter differences have been found in the right anterior corona radiata,
forceps minor close to the genu of the corpus callosum, right and left internal capsule, and left cerebellum (Cortese & Castellanos, 2012; van Ewijk, Heslenfeld, Zwiers, Buitelaar, & Oosterlaan, 2012).

Therefore, there is a plethora of evidence supporting the biological influences, including genetic, neurochemical, and neuroanatomical factors, in the etiology of ADHD. Despite the abundance of empirical research, the underlying etiology/etiologies remain unclear. It is likely that these factors interact to create multifaceted, complex presentations of ADHD. At this stage, there is a dearth of evidence directly linked to the pathophysiology of the hyperactive component of the disorder. Indeed, much of the extant imaging data has investigated the attentional networks associated with ADHD, and very few studies have focused specifically on the fundamental mechanisms that contribute to the production of clinically elevated motor hyperactivity. This paper will selectively focus on the hyperactive symptoms of ADHD and their role in the manifestation of the disorder.

Part II: Hyperactivity and ADHD
Conceptualizations of ADHD have evolved significantly over the years. Historically, early iterations of ADHD described hyperactivity as the core symptom of the disorder. Indeed, early clinical accounts utilized titles emphasizing the motor component of the disorder when describing the syndrome. In 1932, Franz Kramer and Hans Pollnow published about a disorder called hyperkinetic disease of infancy. They described it as a condition in which children experienced motor restlessness, perpetual movement without any obvious goal, inability to remain still, an urgent need to run or climb, impulsivity, and difficulty concentrating (as cited in Lange, Reichl, Lange, Tucha, & Tucha, 2010). Then, in 1968 in the second edition of the DSM, hyperkinetic reaction of childhood was introduced to the literature. This disorder was characterized by clinically elevated levels of physical activity, restlessness, distractibility, and
short attention span, especially in young children (as cited in Lange, et al., 2010). When the third version of the DSM was published (1980), there was a noted shift in the conceptualization of this type of disorder. Here, the attentional component became more prominent and the hyperactive component became more secondary; hyperactivity became a specific qualifier of the disorder and was no longer necessary for a diagnosis (as cited in Lange, et al., 2010). The disorder continued to evolve over time, and the current diagnostic criteria according to the DSM-V states that children must present with either 6 symptoms of inattention (inattentive subtype), 6 symptoms of hyperactivity (hyperactive/impulsive subtype), or both (combined type) (APA, 2013). Thus, clinically impairing hyperactivity has been empirically described for a long period of time. However, although hyperactivity remains an important component of ADHD, symptoms of hyperactivity are not required for a DSM diagnosis, indicating that there are likely distinct endophenotypes for those who experience significant hyperactivity versus those who do not.

Measurement of hyperactivity

Hyperactivity is measured using a broad range of instruments. Rating scales have been empirically validated and used to measure hyperactivity for several decades; typically, parents, teachers, and/or patients rate items on likert-like scales that assess domains such as restlessness, impulsivity, excitability, fidgeting, tempo of actions, and overall activity level (Faries, Yalcin, Harder, & Heiligenstein, 2001; Poggio & Salkind, 1979; Sprafkin, Gadow, & Nolan, 2001; Spring, Blunden, Greenberg, & Yellin, 1977; Trites, Blouin, & Laprade, 1982; Zukow, Zukow, & Bentler, 1978). Analogue measures have also been used to measure hyperactivity in children as an attempt to increase ecological validity and objectivity (Barkley, 1991). These paradigms attempt to create real-world-like settings within a laboratory (e.g., playroom, classroom) to
measure hyperactivity in naturalistic environments to increase external validity. They typically assess motor movement, activity shifts, vocalizations, off-task behavior, number of times a child gets out of his/her seat, and fidgeting (Barkley, DuPaul, & McMurray, 1990; Milich, Loney, & Roberts, 1986; Roberts, 1990). Another methodology employed for measuring hyperactivity is through structured direct observation protocols in actual naturalistic environments (e.g., school). These protocols traditionally utilize behavioral coding systems via reliable raters and focus on similar aspects of behavior as other techniques such as the number of verbalizations, motor movement, translocation, fidgeting, and physical contact made by children (Luk, Leung, & Yuen, 1991; Whalen et al., 1978). Pedometers and actigraphs are also commonly used to precisely measure the amount of truncal and extremity movement (Konrad, Günther, Heinzel-Gutenbrunner, & Herpertz-Dahlmann, 2005; Plomin & Foch, 1981; Rapport et al., 2008b; Tsujii et al., 2009).

**Trajectory of Hyperactivity over the Lifespan**

As previously discussed, ADHD is now recognized as a disorder that frequently persists throughout the lifespan. However, it is also noted that symptoms likely change in quality as individuals age. Therefore, the criteria used to diagnose a preschooler may not accurately reflect the diagnostic criteria appropriate for school-age children, adolescents, as well as adults. This may be especially true within the domain of hyperactivity, as developmental norms change drastically. Therefore, some normative developmental changes in hyperactive symptom presentation are expected given the age-dependent decline in normative levels of hyperactivity within the typically developing population (Martel, von Eye, & Nigg, 2012). It is especially problematic that some symptoms are developmentally unfitting when applied to adolescents and adults (e.g., “runs about or climbs excessively,” or “plays quietly”) and that the criteria required
for diagnosis remains inflexible (McGough & Barkley, 2004). Indeed, when measured using the criteria developed for childhood diagnosis in adolescents and adults, the rate of hyperactive-related remission is higher than the rate of inattention-related remission (Biederman, et al., 2000; Kessler et al., 2010) despite continued impairment. As such, although it may initially appear that hyperactive symptoms associated with ADHD remit at a higher rate than the inattentive symptoms, this account is likely misleading due to use of inappropriate developmental standards. Chacko and colleagues (2009) have recently proposed the idea of using a diagnostic system that identifies symptoms based upon the developmental quality of the behavior rather than solely relying on symptom frequency. It is not surprising that when using this type of developmental lens, it has been shown that hyperactive symptoms do, in fact, often persist when the diagnostic criteria are adjusted to reflect more appropriate norms and standards (e.g., higher focus on impulsive behaviors [i.e., interrupting others] rather than motoric activity [i.e., driven by a motor]; Martel, et al., 2012; Solanto, Wasserstein, Marks, & Mitchell, 2011). Indeed, adults with ADHD endorse a subjective “inner” hyperactivity characterized by feelings of internal restlessness, difficulty sitting for extended periods, inability to relax, and impatience (Turgay, et al., 2012).

Hyperactivity and Functional Impairment

In general, children across all subtypes of ADHD perform more poorly on academic and behavioral measures than their typically developing peers. Despite the fact that very little research has been conducted on the direct impact of hyperactivity on functional impairment in children with ADHD, differences in profiles have been observed between subtypes of the disorder. Notably, children whose symptoms are best characterized by hyperactivity/impulsivity tend to experience significantly more peer rejection, relational aggression, and frequent
accidental injuries than their primarily inattentive peers (Willcutt et al., 2012). Additionally, oppositional behaviors and maladaptive behavioral conduct are typically more closely associated with the hyperactive or combined-type presentations (Graetz, Sawyer, Hazell, Arney, & Baghurst, 2001; Newcorn et al., 2001; Wood, Rijsdijk, Asherson, & Kuntsi, 2009). Moreover, hyperactive/impulsive symptoms are significantly correlated with classroom disruption, even after controlling for the presence of oppositional symptoms (Garner et al., 2013). Generally, the literature has consistently demonstrated that children with ADHD-HI type experience greater behavioral and social impairment, while children with ADHD-PI type experience greater academic impairment (Chhabildas, Pennington, & Willcutt, 2001; Crystal, Ostrander, Chen, & August, 2001; Gadow et al., 2004; Mitsis, McKay, Schulz, Newcorn, & Halperin, 2000). Because the combined type includes a significant number of symptoms from both domains, children with this profile tend to display the highest levels of overall impairment (academic and behavioral) (Gadow, et al., 2004).

Part III: Underlying Mechanisms of Hyperactivity in ADHD
Clearly there is an abundance of empirical evidence, both recent and historical, supporting the fact that hyperactivity is an enduring and clinically impairing symptom domain in ADHD. Despite having numerous validated instruments available to measure hyperactive behavior, most current models of ADHD are purely neurocognitive in nature and fail to sufficiently account for hyperactive symptoms. Indeed, many questions remain regarding the underlying factors that contribute to the manifestation of hyperactivity. Are there subtypes of hyperactivity? Might hyperactivity be moderated by neuropsychological constructs (e.g., response inhibition, executive functioning)? Although they are sparse in number, several models have attempted to answer these questions.
Dynamic Developmental Theory of ADHD (Sagvolden, Johansen, Aase, & Russell, 2005): This model describes hyperactivity as incidental movement in the context of altered reinforcement and extinction processes. It suggests that ADHD results from a combination of a hypoactive dopaminergic system and impaired learning processes (namely reinforcement and extinction). Specifically, the authors hypothesize that an overall reduction of dopamine levels leads to deficient behavioral reinforcement and extinction processes, which in turn, produces the excessive behavior associated with hyperactivity and disinhibition. In this view, individuals with ADHD have shorter and steeper delay-of-reinforcement gradients than their typically developing peers as a result of an underactive mesolimbic dopaminergic reward system; therefore, rewards will only be effective if they are administered in extremely close temporal proximity to the behavior (there will be no phasic burst of dopamine for rewards that are provided after this significantly reduced period). This in itself will not produce hyperactivity. Instead, hyperactivity occurs because these individuals experience less efficient extinction than their peers because they do not undergo an adaptive, coordinated phasic decrease in dopamine after the reinforcement ceases; thus, they continue to display the originally reinforced behavior for a longer period after the reinforcement is eliminated. Thus, acquired responses do not undergo extinction, but rather accrue as a function of contact with different reinforcement contingencies (Johansen, Aase, Meyer, & Sagvolden, 2002). Accordingly, this theory predicts that inefficient extinction processes promotes an increased number of responses and increased behavioral variability, even in the context of a dysfunctional reinforcement response. As such, it is believed that hyperactivity is developed and supported by a combination of successful, albeit aberrant, reinforcement processes and inadequate extinction, thereby increasing the frequency of acquired responses without eliminating the older, irrelevant responses. This pattern will ultimately lead to
an accumulation of responses that appear to manifest as excessive, non-goal directed motor
activity (hyperactivity) in the absence of any obvious reinforcer.

Although this model appears promising, it has not yet been supported by an abundance of
empirical evidence. The same group of researchers who created the model completed a small
study in which they demonstrated that when compared to typically developing children, children
with ADHD displayed response bursts of activity after an initial learning period, but unlike their
counterparts, they continued to emit this activity even after an extinction procedure (Sagvolden,
Aase, Zeiner, & Berger, 1998). To this author’s knowledge, the only other empirical support has
come from animal models of ADHD. Similar patterns of increased rates of responding and motor
activity in the context of decreased rates of reinforcement and slower extinction rates have been
shown in the Spontaneously Hyperactive Rat (SHR), a commonly utilized animal model for
ADHD (Brackney, Cheung, Herbst, Hill, & Sanabria, 2012; Hill, Herbst, & Sanabria, 2012;
Johansen & Sagvolden, 2004). However, despite the dearth in empirical evidence, this model is
theoretically consistent with the neurochemical research pertaining to dopamine dysregulation
described above; pathologically low tonic baseline dopaminergic activity may produce a “floor
effect” that damages the extinction process (i.e., extinction is associated with a phasic decrease
in dopaminergic activity, and the neuronal firing rate cannot be further reduced).

In addition to lacking significant empirical support, this model may also lack external
validity. The only studies that have been conducted thus far have been performed in highly
controlled laboratory settings that do not necessarily equate to real-life situations. Clinically, it is
difficult to explain all hyperactive behavior as the result of inadequate extinction, and it would be
difficult and quite challenging, if not impossible, to demonstrate causality in a more naturalistic
setting.
Hyperactivity as a Minimization of Delay Aversion: Similar to the previously described model, this model is predicated on the idea that children with ADHD have significantly different responses to the effects of reinforcement than their typically developing peers (Sonuga-Barke, Taylor, Sembi, & Smith, 1992). However, unlike the previous model, it focuses more on timing of reward provision rather than the extinction procedure. It is based on the theory that there is a pronounced sensitivity to pre-reward delay in children with ADHD. In one of the seminal studies on this model, Sonuga-Barke and colleagues (1992) aimed to elucidate the etiology of this delay of reward sensitivity; their results suggested that children with ADHD typically choose smaller, more immediate rewards over larger delayed rewards not because of impulsivity or reward maximization, but rather due to an aversion to delays. Therefore, they suggest that hyperactive behavior serves the purpose of minimizing the aversive quality of delays. Indeed, the model considers hyperactivity to be a “motivational style” in which children are driven to avoid or escape delay due to its inherently unpleasant nature. Excessive activity embodies a functional expression of their aversion to delay; the model predicts that children with ADHD will consistently choose immediate rewards over delayed rewards, and when there is no immediate choice available, they will express physical activity to reduce their perception of time during delay (Sonuga-Barke, 2002). In doing so, they create alternative sources of stimulation and/or reinforcement for themselves.

There is a substantial amount of empirical evidence supporting the theory that children with ADHD are delay-averse. Kuntsi, Oosterlaan, and Stevenson (2001) found that participants with ADHD consistently chose smaller, more immediate rewards over larger delayed rewards in an effort to minimize the length of the delay period, understanding that they were sacrificing a more significant reward. Moreover, when these children experienced delay periods, they
engaged in other behaviors (e.g., talking, moving) during the wait. Although these results may be interpreted as a deficit in inhibition, several other studies have shown a double dissociation between performance on tasks of inhibitory control and delay aversion (Sonuga-Barke, Houlberg, & Hall, 1994; Sonuga-Barke, Williams, Hall, & Saxton, 1996). Indeed, these studies suggest that children with ADHD are able to wait for rewards under certain task demands as much as controls even under circumstances in which inhibition is required, and in situations with different task demands in which no inhibition is necessary, they fail to wait for rewards. More generally, these studies have also demonstrated that children with ADHD typically choose to forgo rewards to decrease the amount of delay. Solanto and colleagues (2001) directly compared performance on tasks of behavioral impulsivity and delay aversion and found that delay aversion is associated with a broad range of ADHD characteristics, including hyperactivity, whereas inhibitory failure is associated with the distinct domain of executive functioning. Others (Sonuga-Barke, Dalen, & Remington, 2003; Thorell, 2007) have replicated similar results and found that executive dysfunction and delay aversion are independent constructs and that executive impairment was independently related to symptoms of inattention, whereas delay aversion was independently associated with symptoms of hyperactivity.

A major limitation to this theory is that it fails to account for hyperactivity in the absence of delays. Although the theory intuitively makes sense for instances in which children with ADHD are forced to wait for a reinforcer, it does not explain why hyperactivity occurs in settings that are reinforcing to a child. Indeed, this model contradicts anecdotal evidence that children with ADHD appear less hyperactive in novel, unknown settings that are not inherently reinforcing (e.g., doctor’s office) than in settings in which they are comfortable and more likely to be reinforced (e.g., at home, with friends, etc.). Therefore, it is possible that this theory
accounts for some hyperactive behaviors in children with ADHD, but it may not be broad enough to explain all instances of hyperactivity across settings and conditions in this population.

Hyperactivity as a Manifestation of Subcortical Impairment: This model purports that the core symptoms of ADHD, including hyperactivity, are caused by subcortical neural dysfunction (Halperin & Schulz, 2006). The authors hypothesize that this noncortical dysfunction begins early in development and remains relatively stable throughout the lifespan. Accordingly, it is not believed to be associated with the reduction in ADHD symptomatology that frequently occurs as children age. Instead, it is believed that the development of the prefrontal cortex provides compensation for some of the deficits secondary to subcortical dysfunction via top-down executive regulation. Originally, the model suggested that typical reductions in overt hyperactivity in children with ADHD over time are the result of a normalization of caudate volume within the basal ganglia, which has reciprocal projections to the prefrontal cortex. However, data from a longitudinal study indicated otherwise. Halperin and colleagues (2008) found that children with ADHD across age groups fidgeted and expressed hyperactivity significantly more than controls even in light of the generally accepted belief that activity level declines during adolescence in individuals with ADHD. Additionally, they found that activity levels were elevated for participants who continued to meet criteria for ADHD in adolescence as well as those who did not. As such, the authors revised their theory to reflect this new data. They now suggest that the underlying mechanism for hyperactivity in ADHD is more central to the core of the disorder (i.e., subcortical dysfunction) and is not mediated by compensatory mechanisms produced by the prefrontal cortex (i.e., executive functions).

Although there is not a tremendous amount of empirical support directly backing this theory, another longitudinal study found that hyperactivity in middle childhood significantly
contributes to the presence of inattentiveness in early adolescence, but inattentiveness in childhood is not predictive of hyperactivity in adolescence; this suggests that hyperactivity is a basic, early core symptom of ADHD (Greven, Asherson, Rijsdijk, & Plomin, 2011).

Additionally, a recent longitudinal study examined the temporal relationship between ADHD symptom severity and neuropsychological functioning in preschoolers with significantly elevated levels of hyperactivity (at risk for developing ADHD) and a typically developing group (Rajendran et al., 2013). The results of this study indicated that improvement in neuropsychological functioning in the hyperactive group preceded significant symptom reduction, which insinuates that more advanced neurodevelopment is critical for the diminution of symptoms and is consistent with Halperin and Schulz’s (2006) neural dysfunction theory. This theory is currently promising but requires further empirical study before its merits can be objectively assessed.

Additionally, given that the literature has also demonstrated significant neural differences in cortical and cerebellar regions between children with ADHD and typically developing children, this model of a subcortical etiology of hyperactivity remains debatable. It is possible that the interaction of growth and development of multiple brain regions (or lack thereof) contributes to ADHD symptom presentation and that hyperactivity cannot solely be attributed to subcortical neural dysfunction. Moreover, given the extreme variability in both symptom presentation and brain development, it is possible that several neurodevelopmental endophenotypes exist regarding ADHD-related pathology. In addition, the model does not account for environmental factors that may influence neural, behavioral, and cognitive development.
Developmental Delay in Response Inhibition: This theory suggests that the core symptom of ADHD is a deficit in response inhibition, which leads to subsidiary impairment in other neuropsychological domains; these secondary impairments, in turn, lead to decreased control of motor behavior (Barkley, 1997). This developmental delay in inhibitory processes produces excessive motor activity when children with ADHD cannot inhibit task-irrelevant behavior and fail to regulate the appropriate goal directed behavior. Specifically, executive functions (working memory, internalization of speech, self-regulation of affect-motivation-arousal, and reconstitution) guide the motor system in the execution of goal-directed behavior. Accordingly, because these executive functions are impaired in individuals with ADHD due to an overarching deficit in inhibition, the motor system lacks the refined control and management displayed by typically developing individuals and instead produces excessive, aimless gross motor activity. The motor system also consequently lacks the capacity to generate novel, appropriate, and complex responses in challenging situations. As such, the model suggests that the deficient behavioral inhibition system reduces the effective usage of executive abilities, and therefore, the reduced capacity for inhibition secondarily disturbs the regulation of motor activity. Indeed, it is hypothesized that children’s hyperactive behaviors are associated with the inability to filter nonessential information; once the nonessential information enters the limited-capacity working memory system, it essentially encourages non-goal directed behavior that is inconsistent with the situational demands. Furthermore, as the disorder is characterized by inhibition and executive deficits in this model, it makes sense that it describes the behavior of individuals with ADHD as being predominantly controlled by the immediate context rather than by a systematic evaluation of possible behavioral options.
The empirical evidence supporting Barkley’s model is robust. Various meta-analyses and well-designed studies have consistently identified deficits in executive functioning, particularly in response inhibition, in children with ADHD (Avila, Cuenca, Felix, Parcet, & Miranda, 2004; Berlin, Bohlin, Nyberg, & Janols, 2004; Scheres et al., 2004; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Additionally, neuroimaging and EEG studies have confirmed functional differences in children with ADHD versus controls on tasks of inhibition (Berger, Alyagon, Hadaya, Atzaba-Poria, & Auerbach, 2013; Schulz et al., 2004; Suskauer et al., 2008). Moreover, Konrad and colleagues (2000) compared the neuropsychological and behavioral performances of children with ADHD and traumatic brain injuries (TBI); they found that children with TBI who exhibit significant hyperactivity post-injury displayed similar patterns of impairment on tasks of inhibition as compared to healthy controls and children with TBI who have developmentally normal levels of motor activity. Although these results are only correlational, they are suggestive of a shared underlying etiology that impacts both motor activity and inhibition. However, other results have been equivocal and have reported nonsignificant relationships between behavioral inhibition measures and parent and teacher ratings of hyperactivity (Kuntsi, et al., 2001; Nigg, 1999).

Recently, Alderson and colleagues (2012) directly investigated the relationship between motor activity and behavioral inhibition in children with ADHD. They experimentally manipulated behavioral inhibition by administering a stop-signal task (common experimental measure of inhibition) and two additional tasks that were created to be identical to the stop-signal except for the demands placed on behavioral inhibition specifically (purely choice reaction-time tasks with no inhibition-related properties). They administered these tasks to children with and without ADHD and observed their effects on the objectively measured activity level (via
Actigraph). They predicted that children with ADHD would have fewer available inhibitory resources to moderate excessive motor behavior (e.g., limb movements, restlessness) when these resources were occupied by stop-signal task demands. Their results indicated that in general, children with ADHD were more active across experimental conditions than the healthy controls. They also found that both groups of participants displayed significantly more activity during experimental conditions (stop-signal task and the two choice reaction time conditions) relative to control conditions (paint program), and activity level remained stable across experimental conditions. However, contrary to their original hypothesis, they found that increasing inhibitory demands (performance on the stop-signal task versus the two active control conditions) did not produce increased amounts of motor activity and did not differentially affect children with ADHD and typically developing children. They interpreted these results as being more suggestive of an underlying deficit in the controlled attentional processes associated with the central executive component of working memory. Indeed, they proposed that increased hyperactivity observed during tasks of behavioral inhibition is primarily due to increased attentional demands rather than a higher-level inhibitory deficit. This theory has been supported by other empirical studies, which have suggested that poor performance on behavioral inhibition tasks is secondary to basic cognitive processing deficits (Alderson, Rapport, Sarver, & Kofler, 2008; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005).

One limitation of this model is that it fails to causally explain why children with ADHD have higher baseline levels of motor activity than typically developing children in the absence of high attentional demands if the central executive is indeed responsible for increased rates of activity. If the central executive is truly regulated by inhibitory processes as Barkley proposed, the data from the studies above are consistent with his model. However, the relationship between
inhibitory and working memory systems is still theoretical and debatable at this time, and it is possible that the relationship between these functions is not as linear, unidirectional, or simple as Barkley posited. Accordingly, more data is needed regarding the causal relationship between inhibition and working memory to address potential gaps in this theory. Moreover, many of the tasks utilized to measure both behavioral inhibition and working memory are highly contrived and may not have great external validity, and this may account for equivocal data regarding the relationship between hyperactivity and inhibition. Therefore, it is difficult to ascertain how this data is translated in an ecologically valid manner.

**Working Memory Model of Hyperactivity:** In accordance with Alderson et al.’s theory (2012), this model hypothesizes that deficits in underlying working memory mechanisms generate increased movement in children with ADHD to augment overall arousal required for task performance (Rapport, et al., 2008b). Specifically, it postulates that increased rates of motor activity help compensate for the chronic cortical hypoarousal (discussed earlier) associated with ADHD. Accordingly, as task demands increase, so does the amount of cortical activation required for successful completion. The model predicts that increasing the working memory demands of particular tasks will increase the amount of excessive motor activity as a compensatory means to stimulate reduced baseline cortical activity in children with ADHD. Working memory, in this model, is described in terms of Baddeley’s model; namely, working memory is comprised of two slave systems (phonological loop and visuospatial sketchpad) and a central executive that governs and coordinates the two subsystems and reacts to the changing attentional demands of any given situation (Baddeley, 1992). The investigators evaluated the level of motor activity using Actigraphs during working memory and control tasks. They found that children with ADHD and healthy controls both demonstrated significantly higher rates of
motor activity while performing phonological and visuospatial working memory tasks as compared to control conditions. They also found that the participants with ADHD moved more than typically developing controls children across both visuospatial and phonological modalities, and their activity level remained stable even in the context of increases in cognitive demand. This was interpreted as reflecting a general trend that increases in activity level between control and working memory tasks represent general task demands associated with central executive processing rather than increases in cognitive load of the subsystems. Indeed, subsequent latent variable analyses revealed that performance on working memory tasks reliant only on central executive functioning (but not phonological or visuospatial storage/rehearsal) was significantly functionally related to increased levels of hyperactivity. Additionally, although the children with ADHD demonstrated higher levels of activity than their peers on control conditions with minimal working memory components, this group discrepancy was fully resolved by removing the influence of working memory demands of those tasks. As such, the authors surmise that motor activity may function as an adaptive mechanism in all children to the extent that arousal is required for central executive processing but becomes excessive in children with ADHD to compensate for persistent cortical hypoarousal.

Although this model was created relatively recently, it has received some empirical support. Indeed, deficiencies in working memory, and more specifically in the central executive system, have been previously documented in children with ADHD (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Rapport et al., 2008a; Willcutt, et al., 2005). Moreover, a recent study examined the relationship between working memory and motor activity in boys with ADHD using several experimental conditions with varying levels of WM demand (n-back task, choice reaction task, and simple reaction time task) and a simple control condition (Hudec et al.,
Results showed that participants with ADHD exhibited higher activity levels compared to healthy controls and that both groups exhibited the greatest activity during conditions with high working memory demands, followed by the reaction time and control task conditions, respectively. These findings were interpreted as being indicative of the fact that increases in motor activity are largely associated with increased demands on working memory systems but that demands on non-executive processes (e.g., basic attention) also elicit increased (of a smaller magnitude) activity. A similar study was conducted with adults with ADHD (Hudec, Alderson, Kasper, & Patros, 2013), and the findings also suggested that increased working memory demands were related to demonstrations of hyperactivity. However, few other studies have directly assessed the relationship between working memory/central executive deficits and hyperactivity, and it will be important to investigate this association in future research. Additionally, this model postulates that hyperactivity increases cortical activity to allow children with ADHD to perform at a higher level, which has been substantiated in one study examining working memory task performance and hyperactivity (Sarver, Rapport, Kofler, Raiker, & Friedman, 2015). However, these results have yet to be replicated. Additionally, no studies have directly used electrophysiological measures during the experiment to the author’s knowledge. Future research will need to measure cortical activity during tasks that systematically tax the central executive while concurrently measuring physical activity. Moreover, the model presently fails to address the possibility of mediating or moderating factors/systems that may impact both the motor activity and working memory systems.

Part IV: Future Directions

Clearly, many questions regarding the nature and etiology of hyperactivity in ADHD remain unanswered. The extant models described above have not yet received enough empirical evidence nor have they filled in all the gaps related to the complexity of the diverse presentation.
of hyperactivity symptomatology to be considered parsimonious, evidence-based models. Additionally, they all provide unique, and sometimes conflicting, perspectives on this symptom domain. Ideally, new, comprehensive models will address the theoretical inconsistencies that currently exist between the models and will also add knowledge to the literature specifically with regards to how neuroanatomy, functional neural networks, neurochemistry, environment, and neuropsychological factors contribute to the manifestation of hyperactivity. It is likely that several novel, distinct, and inclusive models will be required to explain the variability of presentations of children with ADHD. This knowledge, in turn, will have tremendous treatment implications and may produce new and improved interventions for children with ADHD.

As discussed earlier, there is a plethora of research documenting neuroanatomical (both functional and structural) and neurochemical differences between children with ADHD and typically developing controls. However, much of this data has focused specifically on previously established attentional networks rather than possible mechanisms underlying hyperactive symptoms. Going forward, incorporating data from neuroimaging and neurochemical modalities into the extant models is critical in order to gain a fuller picture of the pathophysiology of ADHD-related hyperactivity. Specifically, it would be particularly useful for researchers to compare the profiles of children with ADHD-HI and ADHD-I to isolate differences between the subtypes. This type of study may be particularly challenging to implement given that it would likely be difficult to find children who meet criteria for one subtype but do not display any symptoms of the other symptoms domain (i.e., a child who presents with at least 6 hyperactive symptoms but no inattentive symptoms or vice versa), but if successful, it may ultimately lead to the identification of possible biological bases for hyperactivity. Alternatively, it may be more realistic to divide a sample of children with ADHD
into those who present with high levels of hyperactivity and low levels of inattention and those who present with low levels of hyperactivity and high levels of inattention (e.g., those who do not present with the combined-subtype or those who are not near the cut-points for the combined subtype).

It is also likely that in additional biological factors, environmental factors contribute to the presentation of hyperactivity. To this author’s knowledge, very little (if any), well-designed research studies have investigated environmental factors that are directly related to hyperactivity in children diagnosed with ADHD (rather than ADHD in general). For example, what is the role of parenting in the manifestation of hyperactive symptoms? Research supports correlations between environmental factors such as prenatal exposure to lead (Kim et al., 2013), smoking (Nomura, Marks, & Halperin, 2010; Thakur et al., 2012), maternal stress (Grizenko et al., 2012), and gestational diabetes (Nomura et al., 2012) and the development of ADHD; however, none of the researchers who specifically focus on the etiology of ADHD have incorporated these environmental components into the extant theories. Therefore, future research should aim to fill this gap.

Although many of the older existing cognitive models of ADHD discount the role of hyperactivity, some of the more recent research models (e.g., Alderson et al., 2012 and Rapport et al., 2008) have begun to systematically investigate the relationship between neuropsychological factors and clinically elevated levels of activity. Interestingly, both the working memory and behavioral inhibition models of ADHD point to an etiological underlying deficiency in basic cognitive processing (e.g., central executive) that is associated with the manifestation of ADHD. The behavioral inhibition model posits that hyperactivity is the functional consequence of an inability to filter extraneous information from entering the working
memory system due to an impaired primary behavioral inhibition mechanism, which leads to an increase in non-goal-directed behavior. The working memory model, on the other hand, describes hyperactivity as the result of adaptive, compensatory working memory processes. Alderson and colleagues (2010) directly compared the functional working memory and behavioral inhibition models of ADHD by investigating the directional relationship between constructs; they statistically isolated the domain-general central executive and subsystem processes and then tested each as potential mediators of behavioral inhibition. They found that deficits in the central executive functioning of children with ADHD persist after controlling for behavioral inhibition deficits, and that impairment in behavioral inhibition may be the result of more complex executive functions such as working memory. Similarly, Raiker and colleagues (2012) also compared the behavioral inhibition and working memory models to determine which model could more accurately predict impulsive behavior in children with ADHD. They found that deficits in the central executive system accounted for impulsivity significantly more than behavioral inhibition deficits, which is consistent with Alderson et al.’s findings.

Several theories explaining the manifestation of hyperactivity in ADHD can be found within the extant literature. Currently, there is a growing body of evidence supporting the theory that deficits in basic cognitive processing may be at the core of ADHD symptoms, including hyperactivity. These recent models suggest that an underlying cognitive deficit may account for the clinically elevated levels of motor activity seen in children with ADHD. At present, it appears that there is a great potential for the central executive, the governing body of the attentional working memory construct, to be contributing to the presence of maladaptive overactivity associated with the disorder. However, this model is relatively new and requires more rigorous research for validation. It will be important to continue to investigate this model,
as there are many significant clinical implications regarding treatment of impairing hyperactivity in children with ADHD. Indeed, if hyperactivity is a result of a central executive-based deficit, theoretically, cognitive remediation or other cognitive-based interventions should be viable treatment options. Therefore, a clear understanding of this relationship continues could change the standard of treatment for children with ADHD.

In summary, the literature suggests that a basic cognitive processing deficit is related to the manifestation of hyperactivity in ADHD, but the exact nature of this relationship remains nebulous at best. In order to have a clearer understanding of potentially shared functional circuitry between basic attentional processes and hyperactivity, it is necessary for future research to elaborate on the relationship between the two systems. As such, the goal of this study was to systematically evaluate patterns of motor activity in school-aged children with ADHD as they completed both behavioral inhibition and working memory tasks; specifically, this study aimed to enhance the current literature with regard to etiology of ADHD symptomology by potentially identifying a shared mechanism between cognitive and motoric impairment and potentially provide empirical support for either the Working Memory Model of Hyperactivity and/or the Developmental Delay in Response Inhibition Model.

Specific Aim 1:
The first aim of this research was to determine whether variability in the central executive (CE) component of WM correlates with variability in activity level in children with ADHD. Specifically, this study aimed to identify possible subgroups of children with ADHD with regard to varying patterns of CE deficits and corresponding activity levels. This was investigated by examining the level of CE impairment (based on performance on WM tasks) along with level of hyperactivity (based on actigraph measurements). It was also examined by determining whether children without CE deficits (defined as being more than 1 standard deviation from the norm, see
(Alloway, Gathercole, Kirkwood, & Elliott, 2009) for rationale) display different patterns of activity than children with CE deficits. Because it has previously been speculated that increased motor activity is required for the successful completion of tasks involving the CE (Rapport, et al., 2008b), it was hypothesized that children with higher levels of baseline CE deficits would display higher levels of motor activity during WM tasks than those with lower levels of baseline deficits. It was also hypothesized that children with and without baseline CE deficits would show significantly different patterns of motor activity during WM tasks.

Specific Aim 2:
The second aim of this research was to determine whether different activity level profiles are associated with different levels of successful completion of WM tasks while controlling for basic attentional capacity differences. It was hypothesized that the children with the lowest levels of motor activity would have significantly different rates of success in completing WM tasks than children with the highest levels of motor activity. Completion of working memory tasks was defined by the maximum number of items that were correctly answered in each WM subtest.

Specific Aim 3:
The third aim of this study was to determine whether variability in behavioral inhibition (BI) correlates with variability in activity level in children with ADHD. Specifically, this study aimed to identify possible subgroups of children with ADHD with regard to varying patterns of BI deficits and corresponding activity levels. This was investigated by examining the level of BI impairment (based on performance on a continuous performance task) along with the level of hyperactivity. It was also examined by determining whether children without BI deficits (defined as being more than 1 standard deviation from the norm) display different patterns of activity than children with BI deficits (see Halperin, Sharma, Greenblatt, & Schwartz, 1991 for norms). It was hypothesized that children with higher levels of baseline BI deficits would
display higher levels of motor activity during BI tasks than those with lower levels of baseline deficits. It was also hypothesized that children with and without baseline BI deficits would show significantly different patterns of motor activity during BI tasks.

**Specific Aim 4:**
The fourth specific aim was to determine whether there is a cumulative effect of having both CE and BI deficits with regard to activity levels. It was hypothesized that children with deficits in both areas would display significantly different patterns of hyperactivity than those with deficits in only one domain and those without either BI or CE deficits.

**Method**

**Participants**

Children and their families were recruited through community advertisements for a larger clinical trial assessing the benefit of combined and sequenced effects of Cogmed WM training and behavioral parent training (BPT) for youth with ADHD (Title: Combined cognitive remediation and behavioral intervention for ADHD; [http://clinicaltrials.gov/ct2/show/NCT01137318](http://clinicaltrials.gov/ct2/show/NCT01137318)). Inclusion criteria included: 1) children between the ages of 7-10 years; 2) a diagnosis of ADHD through consensus diagnosis based on the following: a) parent and teacher ratings on the Disruptive Behavior Disorder Rating Scales (DBD; Pelham, Gnangy, Greenslade, & Milich, 1992), b) impairment using the Impairment Rating Scale ([Fabiano et al., 2006](http://www.ncbi.nlm.nih.gov/pubmed/17196430)), and c) a semi-structured interview with the parent using the Kiddie-SADS (Kaufman, Birmaher, Brent, Rao, & Ryan, 1996); 3) fluency in English (parent and child) and; 4) internet access at home. Exclusion criteria included: 1) mental retardation or severe mental illness (i.e., psychosis, bipolar, major depressive disorder) or autism spectrum disorder; 2) child or parent presented with emergency psychiatric requiring immediate services (e.g., suicidal or homicidal intent), and; 3) the child’s estimated Full Scale IQ was less than 80 on
two subtests of the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999).

A total of 130 participants were included, with a mean age of 8.4 years (standard deviation [SD] = 1.33 years). 75.4% of the sample was male. Average FSIQ was 104 (SD=16). 22% of the sample was taking medication for ADHD. 56.4% of the sample met DSM-IV criteria for ADHD: Combined presentation, 43.6% met criteria for primarily inattentive presentation, and there were no children in this sample with primarily hyperactive/impulsive presentation. 40.2% had comorbid ODD, and 8.5% had comorbid CD. 41.0% identified as Caucasian, 12.8% identified as “multi-cultural,” 21.4% identified as African American/Black, 23.9% identified as Asian, 0.85% identified as American Indian/Alaska Native, and 32.0% identified as Hispanic or Latino. With regard to parental marital status, 66.1% were married, 13.4% were never married/single, 14.1% were divorced, and 6.2% were married but separated. Using the Nakao and Treas Socioeconomic Prestige Index (1994), mean socioeconomic status was 57.16 (SD=17.12). Detailed information about the sample demographics can be found in Table 1.

Measures

Measures used to assess inclusion and exclusion criteria (not used in data analyses)

**Kiddie-SADS PL.** The Kiddie-SADS (Kaufman, Birmaher, Brent, Rao, & Ryan, 1996) is a reliable, commonly used, semi-structured interview that assesses a wide array of pediatric psychiatric conditions according to DSM-IV criteria, including: ADHD, disruptive behavior disorders, mood and anxiety disorders, psychosis, and substance use. The substance use module was not administered, as it was not appropriate for this age group. The Kiddie-SADS were administered by trained student clinicians. The Kiddie-SADS, although administered to the parent, specifically addresses issues related to the child’s behavior in school, in addition to at home. A strength of this measure, as compared to other more structured interviews, is that it
allows for greater latitude on the part of the interviewer to query about various behaviors and settings, and in the end, the presence/absence of symptoms is determined by the clinician, based upon all available information.

**Disruptive Behavior Disorders Rating Scale (Parent and Teacher forms).** Symptoms were measured using the Disruptive Behavior Disorders Rating Scale (DBD-P and DBD-T) Parent and Teacher Forms (Pelham, Gnagy, Greenslade, & Milch, 1992). The DBD is a 45-item measure that asks parents and teachers to rate symptoms of ADHD, ODD, and CD on a four-point scale (i.e., Not at all, Just a little, Pretty Much, or Very Much), with higher scores indicating a greater frequency of problems. The DBD evidenced internal consistency (Cronbach’s alpha=.82-.85 for parent ratings and .91-.96 for teacher ratings). Test-retest reliability from pre/post treatment ranged from .49-.61 for parent ratings. Interrater reliability ranged from .14 to .26 in parent and teacher reports, respectively. Concurrent reliability with the Diagnostic Interview Schedule for Children (DISC) ranged from .38-.62.

**Impairment Rating Scale.** Parent and teacher ratings of symptom severity and need for treatment in important functional domains were measured using the Impairment Rating Scale (IRS; Fabiano et al., 2006). The IRS measures impairment across domains of functioning as well as the overall need for treatment. Parents and teachers place an “x” on a seven-point visual analogue scale to signify each child’s functioning along a continuum of impairment that ranges from zero (Not a problem at all. Definitely does not need treatment or special services.) to six (Extreme problem. Definitely needs treatment and special services). The IRS exhibits acceptable temporal stability (r = .54-.76 for one year and .66-.98 for two months), has adequate internal consistency (Cronbach’s alpha= 0.84) correlates with other measures of impairment (r=.62-.77
for the CGAS and IRS), and correlates moderately with behavioral observations while
demonstrating convergent and discriminant validity (Fabiano et al., 2006).

**Wechsler Abbreviated Scale of Intelligence** (WASI; Wechsler, 1999). The WASI is a
brief, reliable, screening measure of intellectual abilities for individuals between the ages of 6-89
years. The WASI has excellent psychometric properties, with an average reliability coefficient of
0.98 (for FSIQ) and test-retest reliability of 0.92 (for FSIQ). For participants who had an
intellectual assessment within six months of the screening evaluation, the summary scores of the
IQ test were requested; those who had not completed an IQ test were administered the WASI,
which was administered by student clinicians under the supervision of a licensed clinical
neuropsychologist.

**Outcome Measures**

**Working memory.** The Automated Working Memory Assessment (Alloway, 2007) was
used to objectively measure working memory ability. The AWMA is a computer-based
assessment of WM skills, with a user-friendly interface and with fully automated administration
and scoring. It consists of auditory-verbal and visual-spatial memory tasks which tap the
temporary storage and manipulation of information. The AWMA is suitable for use with
individuals aged between four and 22 years. Test reliability of the AWMA has been measured on
128 individuals (mean = 10.4 years, SD = 5 years) with four weeks separating the two successive
testing administrations. There was a close relationship between the individual’s performance at
the first and the second time of testing, which indicates that there was very little change in the
scores at the two testing points (r’s ranged from .69 to .90); there is also strong internal
consistency (Cronbach’s alpha= 0.81). Validity of the AWMA was established by comparing it
with performance on the WISC-IV Working Memory Index (Alloway, Gathercole, Kirkwood, &
Elliot, 2008). Results indicate that the AWMA has good diagnostic validity, as evidenced by the
high classification accuracy (91%) of the digit span subtest of the WISC-IV. The short form measure of the AWMA (four subtests) was completed to assess nonverbal maintenance (Dot Matrix) and manipulation (Spatial Recall), and verbal maintenance (Digit Recall) and manipulation (Listening Recall) aspects of WM. The Dot Matrix task is a visual span task that utilizes a paradigm in which a red dot appears on a grid for a brief period and then disappears shortly thereafter. Children are instructed to then point to the location in which the dot appeared. The trials get increasingly difficult as the number of dots in a row increases. The Spatial Recall task adds a manipulation component to the task in which children are asked to determine whether two flags point in the same or opposite directions prior to indicating dot locations. The Digit Recall task is a pure digit span task during which children were asked to repeat a string of numbers (of increasing length) verbatim. The Listening Recall task included a paradigm in which children heard at least two sentences (number of sentences increased as children progressed in the task) and then had to determine whether each sentence was true or false. After this, they had to repeat the last word of each sentence in the order in which they heard them. Standard Scores for each of the subtests were generated by the AWMA and used as outcome measures. Additionally, the raw scores were extrapolated from the standard scores to determine the number of items answered correctly.

Behavioral Inhibition. The A-X Continuous Performance Test (AX-CPT; Halperin, Sharma, Greenblatt, & Schwartz, 1991) generates objective measures impulsivity. Letters are presented individually for 200 ms with a 1.5 s interstimulus interval. The child responds when s/he sees an “A” followed by an “X.” A total of 400 letters are presented, and the entire task lasts approximately 12 minutes. The number of commission errors was used as an outcome measure representing behavioral inhibition errors. Reported reliabilities range from .65 to .74 in
previous analyses. Internal consistency has been shown to be sufficient (Cronbach’s alpha = 0.83).

**Motor Activity (Hyperactivity).** Motor activity was recorded throughout the evaluations using two acceleration-sensitive devices with solid-state memory that store movements per minute (Reichenbach, Halperin, Sharma, & Newcorn, 1992). Actigraphs were placed on the non-dominant ankle and waist, and the mean of the median activity counts for the two actigraphs was calculated. In addition, exploratory analyses were run using the ankle actigraph only, as that has been done in several of the cited studies ((Alderson, et al., 2012; Rapport, et al., 2008b). Test-retest reliability was reported as .84. There was a mild positive skew on this measure. Accordingly, scores were square-root transformed to correct for the positive skew. However, because the results did not change significantly after the transformations, only the original analyses are included in this paper. Outliers were not excluded, as those with the highest scores demonstrated the highest level of activity, which was clinically meaningful.

**Procedure**

**Baseline Assessment.** All assessments took place in a university-based research lab, most frequently during after school hours in the early evening or throughout the day on the weekends. Parental informed consent and child assent were received during the first intake session. The university’s Institutional Review Board (IRB) approved the procedures for this study. The data used in this study was gathered during baseline testing only (as this was part of a larger intervention study evaluating the effectiveness of a combined cognitive remediation and behavioral parent training program).

**Parent.** At the study intake, parents and youth were debriefed on the study’s procedures and general aims. Following parental consent and youth assent, semi-structured interviews (K-
were completed by graduate students who received training and supervision on ADHD diagnostic assessments and administration of the measures with the parents to ascertain psychiatric diagnoses, including ADHD. After completion of the interview, parents completed the measures described above, including the DBD and IRS.

**Child.** Children were tested on medication if it was part of their typical treatment regimen. While parents completed the procedures described above, the children worked simultaneously with a second trained and supervised graduate student. The child’s assessment occurred within the university lab setting, in a separate room from his or her parents. At this time, the WASI, AWMA, and the AX-CPT were administered to the child; because this study occurred within the context of a larger study, the child also completed several academic measures (e.g., Wide Range Achievement Test [WRAT]). The children all wore 2 actigraphs throughout the entirety of the evaluation. The order of assessment included: WASI, 2 subtests of the AWMA, WRAT, 2 subtests of the AWMA, and the CPT. The 4 subtests of the AWMA were divided so as to minimize fatigue and potential frustration occurring as a result of the challenging working memory tasks.

**Statistical Analyses**

**Data Analysis**

For Aim 1, Pearson’s product-moment correlation analysis was used to assess the relationship between severity of CE impairment and level of hyperactivity. CE functioning was measured using two AWMA variables: Spatial Recall and Listening Recall. These two subtests have previously been used in the literature to represent the CE construct (Alloway, Gathercole, Kirkwood, & Elliott, 2008; Alloway, et al., 2009). Level of hyperactivity was operationalized as the mean of the median scores for the ankle and waist actigraphs, which has also been used previously in similar protocols (Marks, Himelstein, Newcorn, & Halperin, 1999). As stated
above, analyses were also run using the ankle actigraphs only. However, the results did not change; as such, only the original results using both actigraphs will be reported. A between-subjects ANOVA was used to assess children with ADHD without CE deficits display different patterns of activity than children with CE deficits (as stated earlier, a deficit was defined as being more than 1 standard deviation below the mean). An ANOVA was chosen (as opposed to a t-test) as the analysis of choice because it is a more robust procedure that can tolerate skewed data. Separate analyses were run excluding children who were currently medicated for ADHD.

For Aim 2, Pearson’s product-moment partial correlation analyses were used to assess the relationship between level of hyperactivity and performance on both of the WM subtests (Spatial Recall and Listening Recall). Raw scores were used in order to ascertain the exact number of items answered correctly to reflect the degree of success across trials for each individual participant. To account for basic attentional capacity differences, raw scores on two other AWMA subtests (Dot Matrix and Digit Recall) were used as covariates. These two subtest provide spatial and digit spans, respectively. As in Aim 1, the analyses were also ran excluding the medicated participants.

For Aim 3, Pearson’s product-moment correlation analysis was used to assess the relationship between severity of BI impairment and level of hyperactivity. BI functioning was measured using several AX-CPT variables, as previously documented in the literature (Marks, et al., 1999), that reflect rapid reaction time responses following a distractor sequence and lengthy reaction times following the presentation of one of the two target letters. Another Pearson’s product correlation was also run to examine the relationship between commission errors on the AX-CPT and level of hyperactivity. A between-subjects ANOVA was used to assess children with ADHD without BI deficits display different patterns of activity than children with BI
deficits (as stated earlier, a deficit was defined as being more than 1 standard deviation below the mean). An ANOVA was chosen as the procedure of choice for the same reasons as listed above. Again, separate analyses were performed excluding medicated participants.

For Aim 4, between-groups ANOVAs (with and without the medicated participants) were used to assess whether children with deficits in either and/or both areas (WM and BI) display significantly different patterns of activity than those without CE or BI deficits. The same definitions and variables utilized in Aims 1 and 3 were used in this analysis. Again, an ANOVA was chosen due to its robust properties.

Results

A correlation matrix for all of the included variables is provided in Table 2. Descriptive statistics (e.g., means, ranges, and standard deviations) of all variables for the entire sample are presented in Table 3. Descriptive statistics for the sample excluding the medicated children are presented in Table 4.

Aim 1: Within this sample, 40 children were identified as having a CE deficit, and 89 children were identified as not having a deficit. After excluding the medicated children, 24 children were identified as having a CE deficit, and 64 were identified as not having a deficit. A Pearson product-moment correlation was conducted to determine the relationship between the severity of CE impairment in the visual domain and hyperactivity. No significant relationship was found between level of impairment on the AWMA Spatial Recall subtest and the level of hyperactivity defined by Actigraph measurements ($r = 0.09$, $p = 0.28$). Similarly, a Pearson product-moment correlation was run to determine the relationship between the severity of verbal CE impairment and hyperactivity. Again, there was no significant relationship between level of impairment on the AWMA Listening Recall subtest and level of hyperactivity ($r = 0.09$, $p = 0.29$). The same results were found when medicated children were excluded from the analyses.
for both spatial \((r = -0.03, p = 0.77)\) and verbal \((r = 0.15, p = 0.17)\) CE domains. Furthermore, a
one-way ANOVA did not yield any significant differences between groups (CE deficit, \(n=40\) /no
CE deficit, \(n= 89\)) with regard to level of hyperactivity \((F(1,127) = 0.30, p = 0.58)\) when the
entire sample was included (children with a deficit in either CE domain were combined and
included in the “deficit” group). However, when children who taking psychostimulant
medications were excluded from the analysis, a significant difference emerged between groups,
indicating that those with an identified WM deficit in at least one domain (verbal
and/or visual) displayed a significantly higher level of motor activity \((F(1,86) = 5.59, p = 0.02)\).

**Aim 2:** A Pearson partial product-moment correlation was conducted to determine the
relationship between the performance on the AWMA Spatial Recall task and hyperactivity
defined by Actigraph measurements, using a visual span to control for basic attentional
differences. No significant relationship was found between performance on the AWMA Spatial
Recall subtest and the level of hyperactivity \((r = -0.02, p = 0.87)\), even when medicated children
were excluded from the analyses \((r = -0.08, p = 0.47)\). However, a significant negative
correlation was found between basic visual attention and level of activity \((r = -0.20, p = 0.03)\).
Similarly, a Pearson partial product-moment correlation was run to determine the relationship
between the performance on the AWMA Listening Recall task and hyperactivity, using a digit
span to control for basic attentional differences. Again, there was no significant relationship
between performance on the AWMA Listening Recall tasks and level of hyperactivity \((r = -0.01, 
p = 0.87)\), even when medicated children were excluded from the analyses \((r = -0.20, p = 0.06)\).
That being said, a trending negative correlation was found between basic auditory attention and
level of activity \((r = -0.21, p = 0.05)\).
Aim 3: Within this sample, 19 children were identified as having a BI deficit, and 107 children did not demonstrate a deficit in this area. After the medicated children were excluded, 15 children had BI deficits and 71 did not. A Pearson product-moment correlation was conducted to determine the relationship between the severity of behavioral impulsivity on the AX-CPT and hyperactivity defined by activity measured by Actigraphs. No significant relationship was found between level of BI impairment and the level of hyperactivity ($r = -0.07, p = 0.46$). Another Pearson product moment correlation was run to determine the relationship between commission errors on the AX-CPT and Actigraph-measured hyperactivity. Again, no significant relationship was documented between commission errors and hyperactivity ($r = 0.00, p = 0.99$).

Furthermore, a one-way ANOVA yielded no significant differences between groups (BI deficit/no BI deficit) with regard to level of hyperactivity ($F(1,125) = 0.95, p = 0.33$). This result did not change after children who were medicated were excluded from the analyses ($F(1,84) = 0.70, p = 0.40$).

Aim 4: Finally, within this sample, nine children were identified as having both WM and BI deficits, as compared to 120 children who were not. After the medicated children were excluded, the number of children who met criteria for having deficits in both areas dropped to 5 (as compared to 84 children who did not meet this criteria). A between-groups ANOVA was run to assess whether children with deficits in both WM and BI displayed different patterns of hyperactivity than those who do not have deficits in either area. Results of this one-way ANOVA did not yield any significant differences between groups with regard to level of hyperactivity ($F(1,127) = 0.30, p = 0.58$), nor did an ANOVA excluding medicated children ($F(1,87) = 0.00, p = 0.97$).
Discussion

**Aim 1**

The first aim of this research was to investigate the specific relationship between the CE component of WM and the manifestation of hyperactivity in children with ADHD. This was done because recent research has suggested that a basic cognitive processing deficit, potentially within the CE, is related to the manifestation of hyperactivity in ADHD (Rapport, et al., 2008b). This question was investigated in two different manners; correlation analyses were employed to examine the overall associations between the two constructs, and a between-groups analysis of variance was utilized to ascertain whether or not differences in activity levels exist between those with identified WM deficits and those without. It was hypothesized that there would be a positive correlation between level of CE impairment and motor activity. It was also hypothesized that there would be significant differences in activity level between subgroups of children with and without identified CE deficits. To the author’s knowledge, this is the first study to compare performances of subgroups of children diagnosed with ADHD (versus only comparing children with ADHD to a healthy control group) in this manner. Contrary to past research, no significant relationships were identified between level of activity and CE functioning, and no group differences were revealed between those with CE deficits and those without. However, when medicated children were excluded from the analysis of variance, a significant difference in activity level emerged; specifically, children with CE deficits exhibited significantly higher levels of motor activity than children without an identified deficit, which is consistent with the literature. This suggests that the medications are likely efficacious in reducing activity levels, and in doing so, they mask an underlying pattern of overlapping symptomatology. Given that the data support the presence of group differences, this may signify the existence of discrete subgroups or endophenotypes within the larger ADHD population.
Aim 2

The second aim of this research was to evaluate the relationships between activity level and performance on WM tasks while controlling for basic attentional capacity. This was done to ensure that differences in underlying basic attention were not contributing to weaknesses in a more complex form of attention, namely working memory, thereby confounding the results. This was accomplished by using tasks that tap purely into the phonological span and visuospatial span (thus eliminating any element of cognitive manipulation) as covariates. It was hypothesized that children with elevated levels of motor activity and comparatively reduced levels of motor activity would have significantly different patterns of task completion after accounting for basic attentional capacity. However, this hypothesis was not supported. No significant relationship was found between activity level and performance on working memory tasks (visuospatial or auditory) after controlling for basic attention. The same was true after the medicated participants were excluded. There was, however, a significant negative correlation between basic visual attention and activity level and a trending negative correlation between basic auditory attention and level of activity. Interestingly, taken together with the significant findings from Specific Aim 1, this suggests that poor performance on WM tasks in children with elevated levels of motor activity may be secondary to impairment in basic attentional processes rather than higher-order CE deficits, per se. This finding directly contradicts the results found in Rapport’s (2008b) study, which posited that the cognitive load (i.e., the amount that each WM subsystem is being taxed) was not functionally related to increased levels of hyperactivity. The current findings are, however, in line with Alderson et al.’s (2012) work, which suggested that increased hyperactivity observed during cognitive tasks is primarily due to increased attentional demands rather than a higher-order executive deficit. Moreover, the current findings are consistent with Halperin and Schulz’s (2006) neural dysfunction theory, which states that as executive functions develop in
children with ADHD, they help compensate for the core deficits associated with frontal-
subcortical functions (e.g., hyperactivity and inattention) that are central to the disorder. The
theory also states that these core symptoms do not ever completely remit, and the current
findings support the idea that deficits in (and the relationship between) basic attention and
hyperactivity are not eliminated by compensatory executive functions.

**Aim 3**

The third aim of this research was to investigate the specific relationships between the
construct of BI and motor activity level, with the goal of identifying possible subgroups of
children with ADHD with unique patterns of BI deficits and corresponding activity levels. This
was investigated via correlation analyses examining the overall associations between the two
constructs as well as with a between-groups analysis of variance to ascertain whether differences
in activity levels exist between those with identified BI deficits and those without. It was
hypothesized that children with higher levels of baseline BI deficits would display higher levels
of motor activity during BI tasks than those with lower baseline deficits and that children with
and without baseline BI deficits would show significantly different patterns of motor activity
during BI tasks. Previous research has produced mixed results, with a select few showing support
for the hypothesis that hyperactivity is related to an underlying BI deficit (e.g., Avila, et al.,
2004; Konrad, et al., 2000) and several others demonstrating equivocal or non-significant results
(e.g., Alderson, et al., 2012; Kuntsi, et al., 2001; Nigg, 1999). The findings from the current
study are in line with the previous studies that did not find any significant relationships between
the two constructs. In the same vein, no significant differences in activity level were identified
between children with and without BI deficits, regardless of medication status.
Aim 4

The final aim of the current study was to determine whether there is a cumulative effect of having both CE and BI deficits with regard to objectively measured activity levels. This was the first study to look at the combined effects of the two constructs, to the author’s knowledge. It was hypothesized that children with deficits in both areas would display significantly different patterns of hyperactivity than those with deficits in only one domain and those without either BI or CE deficits. Again, contrary to the hypothesis, no significant differences across groups were noted. However, the number of participants who exhibited deficits in both areas within the current sample was very small (n = 9). Therefore, it is difficult to draw any meaningful conclusions from this analysis. As such, it will be important to evaluate with a larger sample in the future.

General Discussion

Overall, the aim of this study was to gain a deeper, more nuanced understanding of hyperactivity in children with ADHD given that it has repeatedly been shown throughout the literature to be an enduring and impairing symptom domain. Despite its clinical relevance, most current models of ADHD fail to sufficiently document and account for hyperactive symptoms, and many questions remain about the relationships between neuropsychological constructs and hyperactivity. That being said, a small number of models have been posited to date that directly attempt to better describe and elucidate the nature of hyperactivity in ADHD. These models have tackled this question from several perspectives: neurochemical (e.g., Sagvolden et al., 2005), motivational styles (e.g., Sonuga-Barke et al., 1992), neuroanatomical (e.g., Halperin & Schulz, 2006), and neurocognitive (e.g., Barkley, 1997; Rapport et al., 2008). However, many of these models have not received substantial empirical support, nor have they postulated a parsimonious underlying theory that is able to account for the diverse presentation of hyperactive
symptomatology. With this caveat in mind, there is a growing body of evidence that supporting
the neurocognitive models of ADHD with regard to hyperactivity. Indeed, this literature
suggests that a basic cognitive processing deficit is related to the manifestation of hyperactivity
in ADHD. Therefore, the current study was designed to systematically evaluate patterns of
hyperactivity in school-aged children as they completed cognitive (BI and WM) tasks.

This study documented that unmedicated children with CE deficits display significantly
higher levels of activity than children without a deficit in this area. It also showed negative
correlations between basic attentional processes and level of activity, which is consistent with
Sergeant’s Cognitive-Energetic Model of ADHD (Sergeant, 2004); this model posits that both
bottom-up, non-executive processes (e.g., basic attention) and top-down processes (e.g.,
executive functioning) contribute to the manifestation of symptomatology in ADHD. Taken
together, these results suggest that there may be shared underlying mechanisms between the two
symptom domains (attention/working memory and hyperactivity) or that the two domains are
functionally related at the very least. It also may represent a unique subgroup of children with
ADHD. If Rapport and colleagues’ theory is correct, this elevated level of activity could be
compensatory in nature. This could have clear behavioral treatment implications: these children
may perform better both academically and functionally if allowed to move freely while
completing complex tasks. Moreover, interventions can potentially aim to train children to
channel their motor activity in a manner that encourages cognitive efficiency and productivity
while not contributing to distractibility. Indeed, research from the occupational therapy literature
has supported this notion. Studies have shown that when children with ADHD sit on
therapy/stability balls rather than typical classroom chairs while completing academic tasks, they
exhibit increased in-seat behavior, increased legible word productivity, and decreased disruptive
hyperactivity (Fedewa & Erwin, 2011; Schilling, Washington, Billingsley, & Deitz, 2003). In addition, other studies have suggested that chronic physical activity (e.g., structured exercise) can help to reduce behavioral symptoms and cognitive impairment in children with ADHD (Gapin, Labban, & Etnier, 2011). More recently, a randomized controlled trial comparing a before school physical activity (PA) program to a sedentary classroom-based intervention found that children who were assigned to the PA group demonstrated greater improvement of symptoms of inattention within the home (Hoza et al., 2014). Similarly, several studies (Lidzba et al., 2015; Medina et al., 2010; Pontifex, Saliba, Raine, Picchietti, & Hillman, 2013) have also found completion of acute, intense PA prior to engaging in tasks of sustained attention, reading comprehension, arithmetic, and inhibition was associated with improved performances in children with ADHD. Therefore, this remains an important area for continued systematic investigation in order to facilitate the advent of new behavioral treatments.

With regard to inhibition, this study failed to show any significant relationship between BI and motor activity in school-aged children with ADHD, which is consistent with the results of several other extant studies (Alderson et al., 2012; Kuntsi et al., 2001; Nigg, 1999). While this may reflect the fact that these constructs are truly unrelated, it may also reflect several methodological and theoretical limitations. These limitations will be discussed in detail below, along with future directions.

First, it is important to note that the sample utilized in the current study may be limited in that this was a treatment-seeking sample; this sample tends to have less variability and more severe symptoms, and so this may limit the generalizability of the current results. Therefore, the sample used in the current study may differ from others used in this area of research. Indeed, to the author’s knowledge, no other study on the correlates of executive functioning and motor
activity in youth with ADHD included a treatment-seeking sample (Alderson, et al., 2012; Raiker, et al., 2012; Rapport, et al., 2008b). Additionally, no children included in the current study met DSM criteria for the hyperactive/impulsive subtype of ADHD. Instead, all of the children enrolled in this study were classified as either meeting criteria for the combined subtype or the inattentive subtype. Therefore, it is certainly possible that the relationships between these symptom domains may be significantly correlated in children who display clinically elevated levels of hyperactivity without prominent accompanying attentional deficits. Future research should be focused on the hyperactive subgroup to address this question directly. However, this may be methodologically challenging given that most children who meet criteria for ADHD-HI are young and tend to convert to the combined subtype as they age (Hurtig et al., 2007; Lahey, Pelham, Loney, Lee, & Willcutt, 2005; Millstein, Wilens, Biederman, & Spencer, 1997).

Indeed, the current study’s sample was comprised of a relatively small age range. This is generally consistent with most other studies investigating this area, as they have almost exclusively focused on school-aged children between the ages of 8-12 (Alderson, et al., 2010; Alderson, et al., 2012; Hudec, et al., 2015; Raiker, et al., 2012; Rapport, et al., 2008b). As discussed earlier, ADHD is a lifelong disorder, and the nature of symptoms likely change as children age. Therefore, it is possible that significant relationships will emerge in other age groups. There is robust evidence supporting the fact that the prefrontal cortex continues to develop well after puberty (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; Blakemore & Choudhury, 2006; De Luca et al., 2003). Given that the frontal lobes are strongly involved in the regulation of executive functions (Stuss & Alexander, 2000), it could be inferred that both WM and BI skills mature over time. The literature has indeed substantiated this; aspects of working memory (Conklin, Luciana, Hooper, & Yarger, 2007; Klingberg, 2006; Luciana,
Conklin, Hooper, & Yarger, 2005) and inhibition (Luna, Padmanabhan, & O'Hearn, 2010; Treit, Chen, Rasmussen, & Beaulieu, 2014) have been shown to continue to develop well into late adolescence and early adulthood alongside the maturation of frontal lobe circuitry. As such, it is possible that group differences were not identified in the young sample in this study because executive functions have not reached their full development at this age, and group differences may be come clear over time as executive processes become more sophisticated in a normative population. Indeed, in line with Halperin and Schultz’s theory of subcortical impairment (2006), this differentiation may become more apparent as certain children develop compensatory cognitive and behavioral mechanisms supported by the developing prefrontal cortex while other children continue to display impaired frontal-subcortical networks. This is an area that deserves further study in the future.

It is also important to note that very few participants in this sample met criteria for having a BI deficit (n=19), and even fewer met criteria for having deficits in both WM and BI (n=9). Accordingly, these analyses may not have had enough power to detect group differences. It is therefore currently impossible to state with any certainty that there are no significant group differences in activity level between children with inhibitory deficits and those without. To the author’s knowledge, this is the first study to categorically divide an ADHD sample based on inhibitory deficit status and evaluate group differences regarding level of hyperactivity. Therefore, it will be important to re-evaluate this relationship in a sample with a higher representation of children with inhibitory deficits.

Furthermore, it is important to note that 22% of the current sample was medicated for ADHD, which represents a high proportion of the sample. Other extant studies excluded children who were currently taking medication (Alderson, et al., 2012; Hudec, et al., 2015; Raiker, et al.,
2012; Rapport, et al., 2008b). Although a significant result emerged in the relationship between working memory deficits and level of activity after the medicated children were excluded from analyses, it may be useful to repeat the study with a larger medication-free group going forward.

Other potential confounds that should be considered exist within the measures utilized in the current study. This study only included one WM task and one BI task as outcome measures. Although these measures have each been validated individually, it is possible that they were not sensitive enough to pick up on subtle differences. Many measures of executive functioning, particularly BI and WM, have been psychometrically validated and are available for use. Therefore, it may be the case that other studies (Alderson, et al., 2012; Hudec, et al., 2015; Raiker, et al., 2012; Rapport, et al., 2008b) had significant results due to differences in measures. It may be useful to add several other tasks of both WM and BI in future research to help ensure that the negative results are truly due to a null relationship rather than a methodological flaw. This is also important to consider because, in general, there is a lack of consensus within the literature on how to measure executive functions given the complexity of the constructs and the lack of agreement on definitions (Jurado & Rosselli, 2007). Additionally, many executive functions are highly correlated with each other, which suggests that they may be tapping a common underlying executive ability rather than truly discrete functions (Miyake & Friedman, 2012). Therefore, many questions remain about the purity of extant measures and on how to best capture individual executive functions.

The measures employed in this study had clear limitations in addition to the theoretical problems discussed above. First, both outcome measures were completely computerized, which may limit their generalizability to real world and socially interactive situations. Second, the AWMA includes two verbal subtests in which the computerized program produces spoken
commands; unfortunately, this may have confounded the results because many of the participants
anecdotally reported having difficulty understanding what was being said secondary to
differences in accents. Moreover, the learning/practice trials were relatively short, and many
children initially had trouble understanding the task demands of the nonverbal WM task. As
such, the lack of mastery in the practice trials may have limited performance during test trials.
These factors are important to consider when interpreting the results, as they may represent a
third variable/confound.

Another limitation of this study is that the participants only wore the actigraphs during
their baseline evaluations. Oftentimes, children with ADHD are able to limit their hyperactivity
in novel situations, as discussed earlier. Therefore, it is quite possible that their behaviors during
this initial session signify an underrepresentation of their normal levels activity in their natural
environments. Accordingly, the lack of significant relationships identified in this study may only
be generalizable to similar, new situations. Future studies should be designed to also capture
children’s activity levels in their home and/or school environments. Alternatively, future studies
could potentially repeat the testing over several sessions (possibly using different versions of the
same measures) to eliminate the effect of task novelty on performance.

An important additional methodological limitation exists in the fact that only averages of
activity levels throughout the entire evaluations were used, rather than examining changes in
activity level throughout specific tasks. Unfortunately, the average value may have failed to
capture nuanced group differences and changes; it is conceivable that children with WM and/or
BI deficits exhibited changes in their baseline activity levels during the AWMA and AX-CPT
task administrations, but due to software limitations, this could not be directly investigated. It is
imperative that future studies directly measure changes in activity level specifically as children
are engaging in complex tasks of executive functioning to be able to determine whether or not the higher-order task demands impact level of motor activity.

**Future Directions**

There are many options for future investigations with regard to WM, BI, and motor activity. As stated above, it would be interesting to replicate this study using additional outcome measures to eliminate some of the current methodological cofounds within the current study. Moreover, it will be critical to include a sample of children who meet criteria for the hyperactive/impulsive presentation of ADHD. Furthermore, the actigraph measurements should be implemented in such a way that activity levels can be carefully examined at specific time points throughout the testing. Furthermore, examining the relationships between these constructs longitudinally can help elucidate whether group differences exist as development progresses.

It may also be useful to reconceptualize and examine the data from a different viewpoint; instead of looking for group differences between those with and without executive dysfunction, it may be worthwhile to investigate differences in executive function based on activity level. Specifically, future studies can seek to illustrate whether or not those youths who exhibit significantly elevated levels of motor activity have different patterns of executive functioning than those with low levels of motor activity.

Future research should also assess the relationships between executive functioning and hyperactivity using multi-method, multi-informant methodologies rather than solely relying on performance on computerized tasks in a laboratory setting. Parent- and teacher-ratings of both hyperactivity and executive functioning as well as observational data during tasks involving WM and BI may provide a richer and more complete understanding of the associations between these symptom domains.
Finally, given that many of the extant theories (particularly Rapport et al.’s WM theory) are predicated on the idea that children with ADHD have both functional and structural neurological differences as compared to healthy controls, future research should incorporate functional neuroimaging and neurophysiological methodologies. Understanding differences in patterns of cortical (and/or subcortical) activity during effortful cognitive tasks and how motor activity influences these patterns will be critical to enriching the literature on the etiological bases of symptomatology within ADHD. It may also help identify subgroups within the larger population and consequently influence individualized treatments.
Table 1. *Sample Demographics*

<table>
<thead>
<tr>
<th>Category</th>
<th>Value</th>
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</thead>
<tbody>
<tr>
<td>Age, Mean (SD) in years</td>
<td>8.4 (1.33)</td>
</tr>
<tr>
<td>Sex, % male</td>
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<tr>
<td>Race, %</td>
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<tr>
<td>Caucasian: 41.0</td>
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<tr>
<td>African American/Black: 21.4</td>
<td></td>
</tr>
<tr>
<td>Asian: 23.9</td>
<td></td>
</tr>
<tr>
<td>“Multi-cultural”: 12.8</td>
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</tr>
<tr>
<td>American Indian/Alaska Native: 0.9</td>
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<tr>
<td>Ethnicity, %</td>
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<td>Parental Marital Status, %</td>
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<td>FSIQ, Mean (SD)</td>
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<td>Medicated for ADHD, %</td>
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<tr>
<td>DSM-IV-TR ADHD Subtype, %</td>
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<td></td>
<td>Inattentive: 43.6</td>
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<td>Comorbid ODD, %</td>
<td>40.2</td>
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<td>Comorbid CD, %</td>
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Table 2. Correlation Matrix for all Variables

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<tr>
<th></th>
<th>Actigraph</th>
<th>Listening Recall</th>
<th>Spatial Recall</th>
<th>Listening Recall Raw</th>
<th>Spatial Recall Raw</th>
<th>Dot Matrix Raw</th>
<th>Digit Recall Raw</th>
<th>CPT Impulsivity</th>
<th>CPT Commission Errors</th>
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<td><strong>Actigraph Pearson Correlation</strong></td>
<td>1</td>
<td>-.054</td>
<td>.073</td>
<td>-.212*</td>
<td>-.099</td>
<td>-.102</td>
<td>-.166</td>
<td>-.043</td>
<td>.069</td>
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<tr>
<td>Sig. (2-tailed)</td>
<td></td>
<td>.546</td>
<td>.414</td>
<td>.017</td>
<td>.268</td>
<td>.256</td>
<td>.061</td>
<td>.638</td>
<td>.444</td>
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<tr>
<td>N</td>
<td>128</td>
<td>127</td>
<td>127</td>
<td>127</td>
<td>127</td>
<td>127</td>
<td>128</td>
<td>125</td>
<td>125</td>
</tr>
<tr>
<td><strong>Listening Recall Pearson Correlation</strong></td>
<td>-.054</td>
<td>1</td>
<td>.393**</td>
<td>.877**</td>
<td>.328**</td>
<td>.196</td>
<td>.323**</td>
<td>-.131</td>
<td>-.211**</td>
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<tr>
<td>Sig. (2-tailed)</td>
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<td>.546</td>
<td>.000</td>
<td>.000</td>
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<td>.000</td>
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<td>129</td>
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<td>129</td>
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<td><strong>Spatial Recall Pearson Correlation</strong></td>
<td>.073</td>
<td>.393**</td>
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<td>.348**</td>
<td>.870**</td>
<td>.315**</td>
<td>.354**</td>
<td>.070</td>
<td>.068</td>
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<tr>
<td>Sig. (2-tailed)</td>
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<td>.000</td>
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<td>.000</td>
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<td>.451</td>
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*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).
Table 3. *Descriptive Statistics for Entire Sample*

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Table 4. *Descriptive Statistics Excluding Medicated Children*

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