KELLEHER, RACHAEL TERESA, Ph.D. The Developmental Roles of Inhibition and Working Memory Across Childhood on Preadolescent ADHD Behaviors (2018). Directed by Dr. Susan P. Keane, 117 pp.

The ADHD literature suggests that impaired executive functions (EF) of response inhibition and working memory (WM), support behaviors of impulsivity, distractibility, and the inability to suppress behavioral hyperactivity. However, methodological approaches commonly used in ADHD research do not examine causal effects of impaired EFs on ADHD behaviors. Moreover, most studies fail to use a developmental approach in attempting to understand how EFs account for ADHD behaviors. To knowledge, no studies have conducted longitudinal, mediational, and moderational tests on key EF's of inhibition and WM to outcomes of ADHD. The current study examined two longitudinal path analysis models assessing whether 5-year inhibition and 10-year WM predict to symptom expressions of ADHD. Specifically, one model tested if ADHD behavioral expressions were moderated by the interaction term of inhibition and WM. The other model assessed if WM mediated the relation between 5-year inhibition to 10-year ADHD behaviors. The model examining the mediational role of WM best fit the data whereas the moderation model did not. Support was found for the mediational role of WM but only for behaviors of inattention. Further, lower 5-year inhibition did not directly predict to greater 10-year ADHD behaviors. Although results of the study supported the hypotheses of the longitudinal contributory effects of earlier EFs on ADHD behaviors, future studies should focus on cross-lagged longitudinal designs to more precisely understand the complex effects of developing EFs on ADHD behavioral expressions.

THE DEVELOPMENTAL ROLES OF INHIBITION AND WORKING MEMORY

ACROSS CHILDHOOD ON PREADOLESCENT ADHD BEHAVIORS

by

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A Dissertation Submitted to
the Faculty of The Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro
2018

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CHAPTER I

INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common childhood disorders that often persists through adolescence and into adulthood (Wilens & Spencer, 2010). Children with ADHD frequently have difficulty concentrating, paying attention, and controlling hyperactive behavior. Such children often experience problems in school, home, and peer relationships due to difficulties with sitting still, listening quietly, focusing, and retaining information. These behavioral issues were once attributed to behavioral unwillingness. However, neurological findings suggest that children with ADHD may experience delayed brain maturation in brain regions associated with attention, thinking, and executive functioning (EF) (Shaw et al., 2007). With this greater understanding, the DSM-5 now lists ADHD as a neurodevelopmental disorder and not a disruptive behavior disorder.

As such, the disorder of ADHD is characterized by impaired EF in children that exhibit greater behavioral symptoms of inattention and/or hyperactivity/impulsivity compared to typically developing peers. In general, EFs are neurocognitive processes that support goal-directed behavior (Willcutt, Doyle, Nigg, Faraone & Pennington, 2005). Abnormal neurodevelopment in brain networks related to EFs, such as inhibition and working memory (WM), have been associated with ADHD (Barkley, 1997) where EF impairments support behaviors of impulsivity, distractibility, and the inability to suppress

behavioral hyperactivity. Although many researchers acknowledge the role of EF in ADHD, methodological approaches commonly used in ADHD and EF research do not address theorized causal effects of EF on ADHD development. For example, many empirical studies that examine EF and ADHD do not use a theoretical framework and usually report group differences in EF performance as a function of group status (clinical vs. controls) (Berlin, Bohlin, Nyberg, & Janols, 2004; Johnstone, Roodenrys, Phillips, Watt & Mantz, 2010; Mullins, Bellgrove, Gill & Robertson, 2005; Overtoom et al., 2002; Willcutt, Pennington, Olson, Chhabildas & Hulslander, 2005). In addition, ADHD studies are often cross-sectional, use small samples, examine only male children, or fail to control for comorbidities (Kerns, McInerney, & Wilde, 2001; Klingberg et al., 2005; Mullins et al., 2005; Pliszka, Liotti, & Woldorff, 2000; Rapport, Alderson, Kofler, Sarver, Bolden, & Sims, 2008). In general, EF studies also have similar limitations.

Another interesting limitation is that most studies fail to use a developmental approach in attempting to understand how EFs account for ADHD behaviors, even though many EFs are theorized to rapidly develop during childhood and ADHD is a neurodevelopmental disorder that affects the developing child. This highlights a major need by the developmental and clinical fields to examine across time how EFs contribute to behavioral expressions of ADHD as outlined in the DSM. To my knowledge, no studies have conducted longitudinal, mediational, and moderational tests on key EF's implicated in pathways of ADHD. This is surprising given that models of EF and ADHD suggest that delays and/or impairments in developing EFs account for observed behaviors in ADHD. Therefore, the purpose of this study was to contribute to the literature by

testing major EFs implicated in pathways of ADHD behaviors on a continuum, using a

longitudinal approach. The current study proposes to test two hybrid developmental

models integrating theories of EF and ADHD.

CHAPTER II

LITERATURE REVIEW

Attention Deficit Hyperactivity Disorder (ADHD)

ADHD is a neurodevelopmental disorder that emerges during early childhood and often persists into adulthood. It is distinguished by behaviors of impulsivity, hyperactivity, and inattention. Symptoms of ADHD fall into two general categories: inattention and hyperactivity/impulsivity. Diagnostic criteria specify that symptoms must be exhibited frequently and for at least 6 months in either or both categories. The pattern of behaviors must be present across two or more settings. In addition, these behaviors must be the primary reason for experienced difficulties across settings such as school, work, and home (American Psychiatric Association, 2013). These behavioral symptoms are also defined as developmentally inappropriate levels of inattention, hyperactivity, and impulsivity that are chronic and pervasive across settings (Frazier, Youngstrom, Glutting, & Watkins, 2007; Marton, Wiener, Rogers, & Moore, 2012).

Although subtypes of inattention, hyperactive/impulsive, or combined types are no longer specified in the current DSM-5, diagnosis requires identification of specific behavioral presentations. For the inattention presentation, behavioral symptoms include: (1) failure to give close attention to details and making careless mistakes; (2) difficulty sustaining attention or remaining focused during long activities or tasks; (3) difficulty listening when spoken to; (4) difficulty following through with instructions and

completing work; (5) difficulty organizing and managing tasks/activities; (6) avoidance or disliking of tasks that require sustained mental effort; (7) frequent loss of important things needed for tasks/activities; (8) susceptibility to distraction by stimuli; and (9) forgetfulness in daily activities (American Psychiatric Association, 2013). Behavioral symptoms for hyperactivity/impulsivity include: (1) fidgeting with hands or feet, restlessness; (2) difficulty staying seated in situations where it is expected; (3) running or climbing on things inappropriately; (4) difficulty playing or engaging in leisure activities quietly; (5) acting "on the go" or "driven by a motor"; (6) excessive talking; (7) blurting answers before questions are finished or difficulty waiting turn in conversations; and (8) interrupting others (American Psychiatric Association, 2013). In children, at least six symptoms for either or both behavioral presentations must be met as reported by teachers, parents, and/or children to meet diagnostic criteria, whereas for adults, only five symptoms need to be present (American Psychological Association, 2013).

As mentioned before, behavior problems that stem from ADHD lead to significant difficulties associated with academic achievement, social and family relationships, and self-regulation of emotions (Barkley, 2013). Increasing prevalence rates based on recent estimates in the United States highlight that ADHD is a significant public health concern (Pelham, Foster, & Robb, 2007). Using data from the National Survey of Children's Health from 2003 to 2011, the Centers for Disease Control and Prevention (CDC) report that 8.8% of children ages 4-17 have a current diagnosis of ADHD based on parent-report. The average current age for diagnosis was 6.2 years of age and children with severe, moderate, and mild presentation of symptoms were diagnosed at the average ages

of 4.4, 6.1, and 7.0 years old, respectively (CDC, 2014). United States rates of child

ADHD have increased 3 to 6% each year (CDC, 2014), indicating ADHD as one of the

most commonly diagnosed psychiatric disorders in children and adolescents. Of those

diagnosed in childhood, it is expected that between 15-65% will continue with the

disorder into adulthood (Faraone, Biederman, & Mick, 2006). Prevalence rates using

DSM-IV criteria suggest that 6 to 7% of children are affected (Willcutt et al., 2012) and a

pooled worldwide prevalence rate is estimated at 5.29% (Polanczyk, de Lima, Horta,

Biederman, & Rohde, 2007; Rucklidge, 2008). Rates are similar across countries

(Faraone, Sergeant, Gillberg, & Biederman, 2003), and boys are three times more likely

to be diagnosed than girls (Sarkari, 2004). However, gender differences in rates of

ADHD disappear by adulthood (Barkley, 2013; Simon, Czobor, Bálint, Mészáros, &

Bitter, 2009).

A more recent review of the literature on sex differences in ADHD reveal no

significant differences in terms of symptom presentation and psychosocial functioning

(Rucklidge, 2008). Observations of the increased prevalence of males with ADHD in

childhood may be an artifact in that females are less likely to manifest symptoms of

hyperactivity but are more likely to show symptoms of inattention that may be more

difficult and take longer to detect (Barkley, 2013). In a meta-analysis conducted by

Gershon and Gershon (2002), boys were reported to exhibit higher levels of

hyperactivity, impulsivity, and inattention problems compared to girls. Females who

received a diagnosis were more likely to present as inattentive. These finding suggest that

symptoms of hyperactivity and impulsivity are more likely to be detected sooner and in

males in contrast to symptoms of inattention which may not be as behaviorally evident, and which is more likely to be ascribed to females (Quinn, 2008).

Comorbidities with ADHD are common (Sarkari, 2004). The three most prevalent co-occurring disorders are Oppositional Defiant Disorder (ODD) at 41%, Depression/Dysthymia at 22%, and Generalized Anxiety Disorder at 15% (CDC, 2014; Elia, Ambrosini, & Berrettini, 2008). As such, symptoms of mood or disruptive behavioral disorders decrease diagnostic clarity because mood disorder symptoms can mimic symptoms of inattention (e.g., difficulties with attention, concentration, and WM) and disruptive behavior problems can also be characteristic of hyperactive and/or impulsive behaviors. Such high levels of comorbidity raise the issue of whether ADHD is an independent disorder given the possibility of shared etiological deficits. However, research findings suggest that comorbid disorders of ODD, anxiety, and depression are only associated with ADHD at the observable trait level—in contrast to shared genetic or biological mechanisms (Rommelse et al., 2008). Moreover, ODD outcomes are more strongly associated with poor social advantage, familial adversity, and parental psychopathology compared to individuals with ADHD (Mash & Wolfe, 2003).

Despite increasing prevalence rates of ADHD in childhood, the stability of ADHD into adulthood, and the high co-occurrence rate with other disorders, there has been no systematic attempt to estimate societal costs due to ADHD (CDC, 2014). ADHD is one of the most common referrals for primary care doctors and special educators. It is considered a prominent problem in education that is associated with poor academic achievement, and it is associated with negative social outcomes such as poor peer and

family relationships, limited vocational achievement, and increased substance use, marital problems, and criminal behavior (Harpin, 2005; Pelham et al., 2007). In a limited review of published studies on ADHD, Pelham et al. (2007) examined the economic impact of the disorder in the United States by assessing costs associated with mental and medical health treatment, education, delinquency, crime and substance use. Using a prevalence rate of 5%, a conservative estimate was derived in which the annual cost due to the disorder ranged from $36 to $52.4 billion. Taken together, ADHD is a serious disorder that causes significant impairment in children's quality of life and it imposes a significant cost to society and the individual. Given this, research continues to focus on identifying the unknown causes of ADHD, in efforts to improve treatments.

With the DSM-5, ADHD has been reclassified as a neurodevelopmental disorder based on neurobiological research implicating genetic and neurobiological components in the etiology of the disorder relative to brain areas that are responsible for emotional control and learning- (Tannock, 2013, Willcutt et al., 2012). Current models of ADHD emphasize the notion of neurologically-based regulatory deficits that may account for observed symptoms. Specifically, it is thought that impairments in EF underlie problems of concentration, time management, organization, and general self-management of behaviors, which can present as inattention, hyperactivity, and/or impulsivity (Hosenbocus & Chahal, 2012; Kerns et al., 2001; Klingberg et al., 2005). As such, the ADHD literature points to deficits of two key EF's implicated in the etiology of ADHD: inhibition and WM.

ADHD and EFs of Inhibition and WM

Although the understanding of ADHD has improved significantly over the last half century, the field has not reached consensus on central deficits that contribute to the disorder. The literature on ADHD has focused on deficits related to processes of arousal, motivation, cognition, emotion, and/or neurological abnormalities in brain network structure and function, as well as gene expression. In general, most research has focused on etiological theories of arousal and cognitive functioning. There is much evidence supporting the observation of EF deficiencies in ADHD (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Walshaw, Alloy, & Sabb, 2010; Willcutt, Doyle, et al. 2005) especially in the domains of WM and inhibition (Barkley, 1997; Hosenbocus & Chahal, 2012; Rapport, Chung, Shore & Isaacs, 2001).

There is ample research to show that impaired inhibitory responding is associated with ADHD. Meta-analytic reviews have found that groups with ADHD exhibit significant impairment on EF tasks of response inhibition (Nikolas & Nigg, 2013; Pauli-Pott & Becker, 2011; Sergeant, Geurts, & Oosterlaan, 2002; Willcutt, Doyle, et al., 2005). Single studies also lend support to this observation where children with ADHD exhibit impaired performance on inhibitory tasks that require the suppression of a behavioral response, such as Stop-Signal, Go-NoGo, and Continuous Performance tasks (Kerns et al., 2001; (Oosterlaan, Logan, & Sergeant, 1998; Pauli-Pott & Becker, 2011; Schachar, Mota, Logan, Tannock, & Klim, 2000; Sergeant et al., 2002; Sonuga-Barke, Dalen, Daley, & Remington, 2002). Similarly, there are several studies that show an association between ADHD and low inhibitory performance on tasks requiring

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interference control (or inhibitory control), such as Stroop task, Puppet-Says, and Luria's Hand Game (Johnstone et al., 2010; Lansbergen, Kenemans, & van Engeland, 2007; Mullane, Corkum, Klein, & McLaughlin, 2009; Oosterlaan et al., 1998; Pliszka et al., 2000; Schachar et al., 2000; Sergeant et al., 2002; Willcutt, Doyle, et al., 2005). Delay aversion studies also lend support to impaired inhibitory responding where children with ADHD have difficulty delaying gratification when reward contingencies are delayed (Forbes et al., 2009; Marco et al., 2009; Sonuga-Barke, 2003; Sonuga-Barke, Wiersema, van der Meere & Roeyers, 2010). As a result, expression of impulsive behaviors is viewed as being driven by other immediate rewards in the environment (many times the escape from delay).

The EF deficit model has not only focused on inhibition as a key deficit in ADHD, but it also highlights the critical role of WM which supports EF behaviors, (such as successfully planning, appropriately initiating and discontinuing behaviors, and attending to relevant information) that allow for the successful execution of goal-directed behavior, and behavioral /emotional self-regulation (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006). WM is the EF responsible for the temporary storage, maintenance, and manipulation of information in mind that guides behaviors such as learning, listening, and following directions (Alderson et al., 2017; Kofler et al 2018). Some researchers conceptualize WM as an attentional controller or Central Executive (CE) directing attentional focus via the storage and rehearsal components of WM and protecting relevant information from interfering irrelevant information through interference control mechanisms (Alderson et al., 2017; Baddeley, 2010; Engle & Kane, 2004).

Much research points to WM capacity as supporting ADHD behaviors such as impulse control (Raiker, Rapport, Kofler, & Sarver, 2012), hyperactivity (Rapport, Bolden, Kofler, Sarver, Raiker, & Alderson, 2009), and organizational problems (Kofler et al., 2018). Meta-analysis of underlying EF deficits and ADHD reveal WM difficulties in children with ADHD (Martinussen et al. 2005; Walshaw et al. 2010; Willcutt, Doyle, et al. 2005). In addition, Kofler et al., (2018), found a large effect size (d=1.24) for WM deficits in school children aged 8-13 with ADHD. Impaired WM corresponded with greater parent- and teacher-reported symptoms of ADHD, as well as organizational problems across settings (school and home).

Research suggests that WM and ADHD deficits may bear some neurobiological and genetic underpinnings. Structural brain abnormalities in the fronto-striatal and limbic pathways, as well as volume deficits in the caudate grey matter are associated with impaired WM functioning in adults diagnosed with ADHD in childhood and/or adolescence (Roman-Urrestarazu et al 2016). Genetic variations linked to WM have also been observed in children with ADHD. In a study conducted by Trampush, Jacobs, Hurd, Newcorn, and Halperin (2014), the investigators found evidence that the interactions of Dopamine D1 and D2 receptor gene polymorphisms with WM functioning in childhood moderated ADHD symptom expression later in adulthood.

The literature suggests that children and adolescents with ADHD have difficulties with specific functions of WM. There is evidence showing an association between children with ADHD and difficulty with WM functions involving storage, maintenance, and manipulation indexed by simple and complex span tasks (Alderson et al., 2017;

Trampush et al., 2014). Deficits have been observed in ADHD children's performance on simple span tasks such as 1-back (Alderson et al., 2017), Block Span (Skogan et al., 2014) and digit span forward (Fair, Bathula, Nikolas, & Nigg, 2012; Kasper, Alderson, & Hudec, 2012), which measure basic recall, storage, maintenance, and retention. Deficits have also been observed with complex span tasks such as 2-back (Elosúa, Del Olmo, & Contreras, 2017), Digit Span Backwards (Gau & Shang, 2010; Kasper et al., 2012; Rosenthal, Riccio, Gsanger, & Jarratt, 2006), and Coding and Arithmetic tasks from the WISC-R (Fried et al., 2016) which measure manipulation of information, short term memory, and information processing capacity (Whitaker, 2013) or executive attention (Kofler et al., 2014).

Although WM deficits have been found for maintenance and storage functions, there is accumulating evidence to suggest that children with ADHD (and even their unaffected siblings) have differentially weaker WM functioning regarding manipulating information compared to their normally developing peers (Rommelse et al., 2008; Trampush et al., 2014). Moreover, a meta-analysis by Martinusen et al. (2005) on WM and ADHD showed strong effects for WM manipulation difficulties compared to storage and maintenance. As such, deficits in the CE component of WM (responsible for coordinating maintenance and storage of information for purposes of later processing with assistance by interference control mechanisms) are thought to account for ADHD symptoms of inattention and hyperactivity/impulsivity (Kofler et al., 2014; Kofler et al., 2018; Rapport et al., 2009). Taken together, WM functional components have been implicated in ADHD behaviors as well as related to processes of inhibition (via the CE).

Thus, WM presents, especially with respect to functions of manipulating information, as a possible mediating variable to ADHD behaviors (Barkley, 1997; Kofler et al., 2014; Rapport et al., 2008).

In sum, there is much research indicating that EF deficits of inhibition and WM are linked to difficulties in behavioral self-management resulting in behavioral symptoms of ADHD. A limitation of the previous studies is they are based on small sample sizes and cross-sectional designs. Nonetheless, consistent replication of findings among several studies highlights the role of inhibition and WM in ADHD. Further, there is research suggesting biological and neural correlates to these EF deficits of inhibition and WM in children with ADHD and ADHD probands (Gau & Shang, 2010; Nikolas & Nigg, 2015; Sonuga-Barke, Bitsakou, & Thompson, 2010). Considering findings implicating inhibition and WM as underlying behaviors of ADHD, for purposes of this study, I will be examining the role of these two factors on behavioral expressions or symptoms of ADHD.

Barkley and Rapport Models of ADHD

Barkley (1997) and Rapport (2001) propose two seminal EF models of ADHD that are commonly accepted. Barkley argues that inhibition (also termed behavioral or response inhibition) is the core deficit in ADHD, whereas Rapport argues that WM is the core deficit. Detailed overviews of these models are found in the sections below.

Barkley's Model of Inhibition and ADHD

One of the more widely accepted models of ADHD in research and clinical settings is Barkley's ADHD model of behavioral inhibition (Barkley, 1997). In his

model, Barkley focuses on inhibition as the key deficit that accounts for symptoms of the disorder. Barkley takes a neuropsychological perspective and posits that ADHD is inherently a disorder of self-regulation or EF dysfunction in that an individual is not able to activate a self-directed action without suppressing an ongoing response (Barkley, 1997). Barkley defines inhibition as the stopping of prepotent response or a response tied to an immediate positive reinforcement. The stopping of a prepotent response allows for the initiation of another response that does not have an immediate reinforcement associated with it (sub-dominant response). Inhibition can be broken down into two main functions. Inhibition serves to (1) suppress or interrupt an initial or ongoing prepotent response that creates a delay in the response to an event (response inhibition) allowing for a subdominant response to be activated, and (2) it protects this period of delay from distraction by interfering information, thereby allowing time for other EFs to formulate an appropriate response (interference control) (Barkley, 1999; 2013).

Barkley's ADHD model of inhibition specifies a developmental progression of EF emergence in childhood where the development of inhibitory capacities precedes WM. As such, disruptions in inhibition cause disruptions in later developing WM (Barkley, 2013). In particular, Barkley points to the role of low interference control as failing to protect WM from informational disruptions thereby undermining behavioral control (Barkley, 1997). Impairment of behavioral inhibition in ADHD is a primary deficit that leads to the failure to inhibit prepotent responses when appropriate, perseveration of prepotent responses, and poor interference control. This in turn, affects children with ADHD's abilities to utilize the EF of WM.

Barkley defines WM as the ability to hold information in mind over a period of time to guide a subsequent response (Barkley, 2006) and is divided into non-verbal and verbal categories. Non-verbal WM include functions of storage, rehearsal, and manipulation, and it is theorized to emerge before verbal WM. The main role of non-verbal WM is the holding of all sensory information, visual, auditory, and tactile, pertaining to self-awareness (Barkley, 1997, 2009). From non-verbal WM arises verbal WM or the EF of 'internalization of speech', representing a form of meta-cognition (Barkley, 1997). Internalization of speech begins with the advent of language and involves descriptions of the self, self-instructions, comprehension, rules, and moral guides to behavior (Barkley, 1997; 2009).

Barkley notes that internalization of speech or self-speech starts out as overt during the early preschool years (3 to 5 years of age), but then progresses to be private by the school years (6 to 10 years of age) (Barkley, 2013). Self-speech is tied to motor control, where children's verbal thoughts gain increasing control over motor behavior, leading to rule-governed behavior. In many ways, private speech plays a central role in self-control because it facilitates the planning and sustaining of goal-directed behaviors.

It may be the case that impaired WM undermines the development of internalized speech (Barkley, 1997) and there is evidence to show that children with ADHD struggle with both poor internalized speech and WM. Studies reveal that children with ADHD are more likely to use overt and irrelevant speech during problem solving tasks compared to typically developing peers (Berk, 2001; Winsler, 1998). Difficulties with WM may also impede children's abilities to abide by rule-governed behavior in that they cannot follow

through with instructions that they have difficulty keeping in mind (Barkley, 1997; 2013). The literature in education points out that children with ADHD are unable to follow rule-governed behavior unless there is an immediate consequence (Barry & Kelly, 2006; Brown & La Rosa, 2002). Hence, children may have difficulties holding rules in mind in the absence of an external reminder.

Barkley notes that another aspect of WM that may be deficient in ADHD is temporal ordering. Children with ADHD have difficulty with accurately sensing time and controlling behavior as a function of time. Temporal disorganization in WM results from the compromised ability to represent and sequence events or information in correct temporal order—partly due to inhibitory control failures. WM processes are involved in the binding of retrospective and prospective information that help to generate appropriate responding for long-term goal attainment (Barkley, 1997;2013; McCabe, Roediger, McDaniel, Balota, & Hambrick, 2010). Difficulties inhibiting prepotent (irrelevant) responses, which are often driven by immediate positive or negative reinforcement environmental influences, prevent the delay needed to activate subdominant goal-oriented actions in the time efficient manner needed to achieve positive long-term future consequences (Barkley, 1997).

There is growing empirical evidence to suggest that deficits in timing are found in children with ADHD. Variable response speed and performance are also common features found in ADHD (Castellanos & Tannock, 2002; Leth-Steensen, Elbaz, & Douglas, 2000; Mullins et al., 2005). Children tend to underestimate long periods and overestimate short periods of time impairing motor timing production (Barkley, 1997;

Kerns et al., 2001; Kuntsi, Oosterlaan, & Stevenson, 2001; Mullins et al., 2005; Rubia, Noorloos, Smith, Gunning, & Sergeant, 2003). These deficits have been further associated with low inhibition (Kerns et al., 2001; Sonuga-Barke, Saxton, & Hall, 1998), WM (Rubia et al., 2003; Rubia, Taylor, Taylor, & Sergeant., 1999), and abnormal functioning in the frontal and parietal lobes; areas associated with time perception due to attention and WM (Mullins et al., 2005).

In general, there is substantial cognitive and neurological research suggesting that inhibitory and WM processes are related (Brocki & Bohlin, 2004; Clark et al., 2007; Cornoldi et al., 2001; Engle, 2002; Kane & Engle, 2000; Mecklinger, Weber, Gunter & Engle, 2003; Palladino & Ferrari, 2013; Vaidya et al., 2005). Notably, the WM literature conceptualizes inhibitory functioning as part of WM (Brocki & Bohlin, 2004; Engle, 2002; Kane & Engle, 2000), where attentional and inhibitory processing is involved in the encoding and retrieval processes of WM. In keeping with Barkley's theory, Palladino and Ferrari (2013) suggest that a key inhibitory mechanism in WM is interference control that protects WM from intrusions of irrelevant information that disrupt other WM processes such as retrieval, maintenance, and encoding.

Supportive research findings by Cornoldi et al. (2001) and Palladino and Ferrari (2013) have found that impaired WM performance in children with ADHD has been explained by low interference control (i.e., intrusion errors of irrelevant information) as opposed to limited recall. Similarly, other WM studies have found a direct relation between interference control and WM storage (Engle, 2002; Mecklinger et al., 2003). Lastly, in a unique cross-sectional study conducted on school age males with ADHD,

Alderson, Rapport, Hudec, Sarver, and Kofler (2010) assessed the mediational effects of both inhibition and WM on group ADHD status, as well as EF performance. The researchers found significant mediational effects for both inhibition and WM, yet WM was the stronger mediator that predicted to ADHD group status (Alderson et al., 2010).

Alderson's findings suggest that WM more strongly mediates the relation between inhibition and ADHD outcomes than was originally thought. These findings may complement a pattern of findings within the literature showing that deficits in inhibition, and not WM, are often associated with ADHD primarily during the preschool and early school-age periods (Brocki & Bohlin, 2004; Brocki, Nyberg, Thorell, & Bohlin, 2007; Kerns et al., 2001; Sonuga-Barke et al., 2002). In contrast, WM deficits are commonly associated with ADHD in older school age children (Brocki et al., 2007; Willcutt, Doyle et al., 2005). Hence, consistent with Barkley's theory, impairments in inhibition are likely to emerge before impairments in WM. Therefore, deficits in inhibition may underlie or mediate ADHD behaviors in early childhood while also adversely affecting WM development later in childhood (Barkley, 1997; Brocki et al., 2007; Gathercole & Alloway, 2007; Gathercole, Pickering, Ambridge, & Wearing, 2004).

Limitations of Barkley's ADHD Model. Although there is ample evidence from the literature to show that weaknesses in behavioral inhibition often distinguish children with ADHD, tests of longitudinal mediation (of behavioral inhibition and WM) would provide greater support for Barkley's model. He hypothesizes that deficits in early inhibition (observed in early childhood) negatively affect the development of later developing EFs. Specifically, deficits in behavioral inhibition, which is considered the

core feature of ADHD, sets off a cascade of EF impairments (e.g., impaired WM) that support behavioral expressions of ADHD. Although Barkley does not specify the term 'mediation' when describing the role of EFs in the manifestation of ADHD behaviors, Barkley does outline how deficits in inhibition and WM account for behavioral expressions of ADHD. As such, it may be argued that the role of EFs may partially cause or have a mediational role on outcomes of ADHD. Notably, most studies examining ADHD do not examine the mediational role of EFs on outcomes of ADHD even though such evidence would further bolster Barkley's theory of ADHD.

Studies examining Barkley's model as well as the role of inhibition and WM in ADHD are mostly cross-sectional in design. Findings provide mixed support regarding the nature of EF deficits associated with ADHD outcomes. For example, there is evidence indicating deficits for both EFs (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Berlin et al., 2004; Mullins et al., 2005), inhibition only (Berlin et al., 2004; Brocki et al., 2007; Ikeda, Okuzumi, & Kokubun, 2013; Kerns et al., 2001; Overtoom et al., 2002;), and WM only (Kuntsi et al., 2001; Martinussen et al., 2005). Therefore, the lack of consistent findings on inhibitory deficits and WM in these types of cross-sectional studies present an unclear picture as to whether inhibition, WM, or both account for ADHD as proposed by Barkley's model.

Rapport's Model of WM and ADHD

In contrast to Barkley's inhibition model of ADHD, Rapport asserts that WM is the central deficit in ADHD where observed inhibitory behaviors occur as a function of WM deficits. Activation of inhibitory responding relies on the identification and

detection of incoming information as irrelevant. This interpretation takes place in WM. Thus, inhibitory processes cannot be activated until WM evaluates whether information is relevant or irrelevant (Alderson, Rapport, Sarver & Kofler, 2008; Rapport et al., 2001). Rapport reasons that the activation of WM occurs before inhibition and therefore WM deficits are central to the etiology of ADHD.

Rapport et al.'s (2008) definition of WM is derived from Baddeley's (2001) WM model. WM is the cognitive ability to temporarily store, rehearse, and manipulate information in mind. Both storage and rehearsal functions have a finite capacity affecting the rate and amount of informational processing in WM. Children with ADHD are hypothesized to show deficits in all three components of WM which are: the phonological loop (verbal rehearsal/storage), visual-spatial sketchpad (non-verbal rehearsal/storage), and Central Executive (CE - the manipulating of information via management of the phonological loop and visual-spatial sketchpad through attentional control) (Baddeley, 2001; Rapport et al., 2001). Both the phonological loop and visual-spatial sketchpad receive and encode incoming information, temporarily store information, and rehearse /maintain information. The CE is the attentional controller and coordinator of these two subsystems. Additionally, the CE manages WM functions in response to changing environmental information and task demands, as well as facilitates the integration of information from WM to long-term memory (Rapport et al., 2008). As such, greater impairment is hypothesized to be found in the CE component of WM, where stored and maintained information is manipulated in mind, in those with ADHD (Kofler, Rapport, Bolden, Sarver & Raiker, 2010; Rapport et al., 2008).

Like Barkley's notion of WM functions of 'sensing of the self' and 'internalization of speech', Rapport's model emphasizes that WM plays a central role in the organization of behavior (Rapport et al., 2001). As described above, WM components facilitate the representation, maintenance, and manipulation of information in mind. WM processes operate as search, access, and recall procedures for a broad network of memory traces linked to specific information about appropriate behavioral actions for current circumstances. Matching of memory traces (recalled and maintained in WM) to incoming information facilitates recognition and activation of situationally-based adaptive behavior. Hence, disruptions occurring at any level involving access, retrieval, and updating of memory traces will lead to disorganized behavioral responding (Rapport et al., 2001).

Although compromised WM in ADHD can be found in both the structural components (phonological loop, visual-spatial sketchpad, CE) and functions (storage, rehearsal, and manipulating information), emphasis is made on two key mechanisms that undermine overall WM functioning. First, Rapport asserts that WM capacity associated with both storage and rehearsal functions in children with ADHD is significantly reduced compared to typically developing children (Rapport et al., 2001; 2008). There is some evidence to support this notion in that meta-analytic studies examining children with ADHD have found small-to-moderate effect sizes on low WM performance on tests assessing storage capacity and rehearsal (Pauli-Pott & Becker, 2011; Willcutt, Doyle, et al., 2005).

Second, Rapport asserts that those with ADHD are susceptible to disruptions affecting WM functions due to low attentional control (via the CE) of informational intrusions thereby affecting WM processes that manipulate information. As such, WM functioning may be further exacerbated when task demands exceed already reduced WM storage and rehearsal capacity. Rapport et al.'s (2008) study offers empirical support for this postulate. The researchers assessed behaviors of attention (laboratory observed visual orienting/distractibility) in male school age children with ADHD and typically developing controls. For ADHD children only, WM demands more easily exceeded their WM capacity for both storage/rehearsal and manipulation of information (on verbal and non-verbal sequencing tasks of varying set sizes). Moreover, while all children demonstrated inattentive behaviors of distractibility as a function of increasing WM demands, children with ADHD demonstrated a significantly lower threshold compared to their typically developing peers. Using latent variable analyses, the researchers parsed out the shared variance between WM components of the phonological loop and visual-spatial sketchpad to index CE functioning. Findings revealed that for children in the ADHD group, lowered CE functioning significantly predicted to lower WM performance and behaviors of distractibility. This finding implicates the role of interference effects on WM performance. In addition, findings from this study suggest that WM deficits account for inattentive behaviors in ADHD. This observation is further supported by neuropsychological profiles found for children with ADHD characterized by reduced WM spans and parent/teacher behavioral ratings of child inattention (Fair et al., 2012).

Rapport asserts that WM impairments can also account for behaviors of hyperactivity/impulsivity in children with ADHD via another mechanism related to dysregulated arousal as opposed to EF. The interaction between low cortical arousal and limited WM storage/rehearsal capacity facilitates rapid rates of fading mental representations. Children's inability to maintain mental representations in mind leads to increased behavioral activity to increase alertness. By updating WM with continuous incoming information that matches the rate of fading mental representations, children may engage in frequent shifting of behavioral activity especially in situations that are non-stimulating. In addition, children involved in challenging tasks that exceed their WM capacities, may lead to the redirection of attention evidenced by escape behavior. This is achieved via hyperactive/ impulsive behaviors (Rapport et al., 2001). These empirical findings support the notion that WM predicts to behaviors of hyperactivity and impulsivity (Kofler, Rapport, Bolden, & Altro, 2008; Rapport et al., 2001; 2008).

Limitations of Rapport's ADHD Model. Like Barkley, Rapport's model of ADHD posits that WM deficits are central to the etiology of ADHD. Despite a few supportive studies conducted by Rapport and his colleagues that show WM impairments as undermining behavioral inhibition (Alderson et al., 2010; Kofler et al., 2010; Rapport et al. 2008), other studies examining inhibition and WM show that these EFs are closely linked or interdependent (Brocki & Bohlin, 2004; Kane & Engle, 2002; Palladino & Ferrari, 2013). In taking a closer look at the ADHD studies by Rapport and his colleagues that show WM deficits to be primary and inhibition deficits to be secondary (Alderson et al., 2010; Kofler et al., 2010; Rapport et al., 2008), caution should be made when

interpreting findings. Generalizability is questionable in that the studies were conducted on small sample sizes (~15) of all male children varying in age (8 to 12), some of whom were comorbid with ODD. Given that these factors increase the risk of type II errors, replication of findings by independent studies on larger samples is needed.

Another major limitation to Rapport's model is that it is does not use a developmental approach to understand the etiology of ADHD. Given that EFs emerge and experience significant changes across childhood, a developmental approach to examining how EFs play a role in childhood disorders such as ADHD is essential. This may be of interest regarding WM in that the development of this EF matures at a slower rate than inhibition (Best & Miller, 2010; Garon, Bryson, & Smith, 2008). As such, it may be the case that WM deficits during early childhood may not come online given that the CE component has yet to be fully developed.

General Limitation of ADHD and EF Research: Lack of Developmental Specificity

Notably, many models of ADHD and EF fail to take an informed developmental approach to identify different pathways to the disorders. Although Barkley's model offers descriptions of developmental presentations of EF and ADHD during childhood, his descriptions are general and not empirically validated. He also fails to describe how ADHD symptoms in infancy, toddlerhood, and even preschool are different, whether in frequency or intensity, from commonly occurring behaviors of distractibility and motor activity more typical in early childhood. Other cognitive models have minimally addressed developmental considerations of how developing EFs in childhood may contribute to ADHD behaviors. Several studies group children into age categories to

index age differences. Yet this methodology does not allow for developmentally sensitive examination of cognitive development during a period where cognitive growth is continually occurring (Casey, Tottenham, Liston, & Durston, 2005). In addition, the few longitudinal studies that have examined ADHD have a clinically based focus where behavioral interventions or medication effects are assessed over time as opposed to the disorder (Jensen et al., 2001; Molina et al., 2009; Swanson et al., 2001). Although theory and research conceptualize ADHD as a neurodevelopmental disorder, employment of these limited methodologies in current research is still commonly used. As such, little progress can be made in understanding developmental pathways that support the manifestation of ADHD behavioral symptoms on a continuum.

Despite little knowledge on the development of ADHD, observed developmental patterns of the disorder indicate that symptoms of hyperactivity appear first in preschool (Barkley, 2013). Hyperactivity and impulsivity symptoms have been observed to remain stable until adolescence where symptoms of impulsivity remain and symptoms of hyperactivity decline (Barkley, 1997; 2013; Biederman, Mick, & Faraone, 2000). Symptoms of inattention are often detected later, during the early school age years when increased demands on attention and concentration take place in school settings. Symptom stability has also been observed for inattention from childhood into adulthood (Barkley, 1997; 2013). Despite much focus on the role of EF in ADHD, research on clinical populations has yet to adopt a developmental approach to expand our knowledge in this area. The following sections review EF development in childhood, briefly summarize

ADHD and EF models discussed, and conclude with two proposed hybrid models that address a few of the major limitations observed in the ADHD and EF child research.

<center>**EF Development in Childhood**</center>

Developmental Integrative Framework of EF

The study of EF has gained popular interest in the fields of developmental and clinical psychology given its role in adaptive functioning and psychopathology. EFs are neurocognitive-based skills that manage emotional and cognitive resources in supporting effortful behaviors in service of self-regulation, and achievement of daily tasks, and long-term goals (Blair, Zelazo & Greenberg, 2005). EF is broadly defined as incorporating mental operations or cognitive processes such as inhibition, WM, planning, self-monitoring, temporal processing, mental flexibility, etc., that support goal-directed behavior (Best & Miller, 2010; Luu, Flaisch, & Tucker, 2000). These broad ranging processes present major challenges to examining the construct of EF, especially regarding how EF, as a whole, develops. Nonetheless, using a neurodevelopmental model of higher cortical maturation, the work of Luria has provided a foundation for current research on EF development. He identified neuropsychological functions of EF associated with language, attention, memory, intelligence along with key brain-behavior relations as a function of childhood development (Chan, Shum, Toulopoulou, & Chen, 2008; Goldstein, Naglieri, Princiotta, & Otero, 2014).

More recently, Garon et al. (2008) has proposed a developmental framework of EF based on age-related changes of core EFs that correspond with rapid development of attention networks and skills during the first five years of life. Garon and colleagues base

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their framework using Posner and Rothbart's (2000) Attention Model describing the Executive Attention Network. Posner's model and other neurocognitive models, like the Supervisory Attentional System (Shallice, Burgess, & Robertson, 1996), suggest that not only is attention the basis of EF development, but that it underlies EF. Hence, processes of executive attention may be the source of common variance underlying EF dissociable components, which then integrate over time (i.e., general EF functioning), supporting the notion of a unitary EF mechanism (Garon et al., 2008; Miyake et al., 2000).

Currently, a prevailing and widely accepted theory in the EF field is the unity/diversity theory of EF. This theory asserts that EF is hierarchically organized as a functional unit composed of separable components (Miyake et al., 2000). There are three core EF components identified in the adult and child literatures: inhibition, WM, and set-shifting (Lehto, Kooistra, & Pulkkinen, 2003; Miyake et al., 2000). These EF components have different developmental trajectories evidenced by different developmental timing of EF abilities in early childhood (Garon et al., 2008). Partial integration of these components across childhood and into adulthood serves as a unitary coordinating EF mechanism of the non-integrated EF components (Garon et al., 2008; Miyake et al., 2000).

As mentioned above, attentional abilities are the first to emerge and develop in infancy and are theorized as foundational for emerging EFs. The first core EF to emerge is WM, which appears within the first six months of infancy. WM has a protracted rate of development across childhood and begins to reach adult-like levels of functioning starting in adolescence (Best & Miller, 2010; Garon et al., 2008). Inhibition, the second

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core EF, also emerges in early infancy, with rapid development taking place during the second half of infancy, from 6 months to 2 years, and from 3 to 5 years. Lastly, set-shifting, the third core EF, emerges in children as young as 8 years of age (Garon et al., 2008; Lehto et al., 2003) and may have a later developing trajectory than inhibition and WM. Because research on set-shifting is lacking and it is thought to develop later in childhood, coupled by the established focus on inhibition and WM by the ADHD literature, examining set-shifting is beyond the scope of this study. Following Garon et al.'s (2008) integrated developmental EF framework, the next two sections will review the literature on childhood development of the two core EFs of inhibition and WM. Although WM emerges before inhibition in infancy, inhibition exhibits greater developmental gains (during the preschool period) before WM and will therefore be reviewed first.

Inhibition. There are different definitions of inhibition that describe several sub-processes. For example, response inhibition involves the deliberate suppression of a motor response and it is considered a primary mechanism of behavioral control in preschool (Garon et al., 2008). However, inhibition may also involve the suppression of cognitive and emotional information (Barkley et al., 2001; Miyake et al., 2000; Sonuga-Barke, Wiersema, et al., 2010). In general, inhibition is defined as the suppression of responses that are automatic, prepotent, ongoing, or dominant followed by the activation of a sub-dominant response in service of an immediately less rewarding goal (Barkley et al., 2001; Diamond, 1990).

Early inhibition skills begin with the ability to suppress a motor response seen in infancy by caregivers when infants respond to a caregiver's "stop" or "don't" request. The ability to stop automatic or reflexive responses in infancy, however, is also evidenced by antisaccade tasks where the infant stops his/her visual orientation toward a visual target. Mastery of visual shifting (or producing full antisaccade eye movements) is achieved by late toddlerhood (Garon et al., 2008; Scerif, Cornish, Wilding, Driver, & Karmiloff-Smith, 2004). By toddlerhood, children can also delay a desired response for a reward and by preschool choose to delay in service of a greater reward (Carlson, 2005; Garon et al., 2008; Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996). By the third year of life, Carlson (2005) suggests that the ability to coordinate the suppression of a dominant response and the activation of a sub-dominant response rapidly emerges.

Inhibition not only consists of behavioral suppression, but also interference control that requires the suppression of distracting irrelevant information so that an organized appropriate response can be formulated (Barkley, 1997; Vaidya et al., 2005). As a result, disruptions of interfering information usually translate to slower or incorrect task responses (Vaidya et al., 2005). Successful inhibition performance involves the ability to detect conflicting information and resolve informational conflicts, signaling a critical milestone for EF functioning which begins to emerge during the early preschool period (Garon et al., 2008; Posner & Rothbart, 2000). Thus, inhibition requires selective attention that allows for increased focus of relevant information necessary to address task demands (Garon et al., 2008).

A common laboratory task used to assess interference control is the Stroop task. One version of this task involves showing words of color names (e.g., blue, green, red, yellow) printed in different colored ink. In the conflict condition, participants state the color of the ink and refrain from stating the color name they read. In general, Stroop tasks require the inhibition of an automatic response in service of activating a conflict resolving (subdominant) response. Stroop tasks are therefore considered a good measure of interference control wherein successful performance is based on the cognitive ability to protect against the activation of irrelevant information that competes with the processing of relevant information (Barkley, 1997; Nigg, 2000).

Inhibitory control tests have been developed for a variety of ages in childhood and research shows that inhibitory ability increases from ages 3 to 7 years old (Carlson, 2005; Cohen et al., 2004; Garon et al., 2008). Younger preschool children can successfully perform basic Stroop tasks (e.g., Shape Task) involving the selection of smaller shapes versus larger ones (Kochanska et al., 1996). Other Stroop tasks such as Day/Night or Snow/Grass are more difficult because inhibition of the semantic tendency to state a word that goes with an image is required. Children are told to quickly state the opposite image when presented with a central image (i.e., picture of sun, child says night). Findings show that Stroop tasks are more challenging for 3-year-olds than for 5-year-olds with increasing interference control abilities observed through the early school age years (Carlson, 2005; Garon et al., 2008).

As previously mentioned, children experience significant gains in inhibition during the mid- to late- preschool years as evidenced by their increased control of motor

and verbal behavior, as well as their ability to hold mental representations in mind, such as rules, to guide behavior (Carlson, 2005; Garon et al., 2008; Kochanska et al., 1996). Evidence of this can be seen from Murray and Kochanska's (2002) tower task study that showed over half a sample of 4-year-olds were able to demonstrate appropriate levels of turn-taking behavior. The tower task has been used to gage inhibitory responding to social demands and expectations in preschoolers in which children must apply social rules of turn-taking with the experimenter while doing a fun activity of building a tall tower with blocks.

Of note, the ability of applying an internalized rule to regulate behavior is an advanced inhibitory skill that relies on skills of attentional and inhibitory control, as well as WM. The child must have the storage and rehearsal capacity to remember the rule during the task and execute the appropriate response by attending to changing task demands. Consistent with a dynamic view of development, early attention, inhibition, and WM are theorized to build upon and mutually influence each other. As such, inhibitory tasks during the preschool period may be considered to reflect "complex response inhibition" abilities (Garon et al., 2008) where attentional, inhibitory, and WM skills are used. Mastery of these tasks reflects improved coordination and integration of these cognitive processes.

Due to the integrated development of EF, inherent challenges of task impurity problems commonly emerge in the assessment of EF. For example, the Day/Night Stroop task measures child inhibition by requiring children to suppress the dominant response of naming the larger picture they see and instead enlist the subdominant response of naming

the smaller picture they see as specified by the task instructions. The task also has WM demands in which children must learn and remember the task rules. The WM processing requirements of the task may be challenging for young preschool children, compromising their inhibitory abilities and task performance (Ikeda et al., 2013). Nevertheless, there is some research to suggest that increases in inhibitory abilities have a stronger influence on complex inhibitory task performance during the preschool period than concurrent increases in WM skills associated with storage capacity and load (Diamond, Kirkham, Anso, 2002; Garon et al., 2008; Simpson & Riggs, 2005). Thus, after accounting for preschoolers' WM skills, the extent to which inhibitory abilities have developed determines complex inhibitory performance.

Despite research examining the developmental sensitivity of EF inhibitory tasks among preschool children, there is less research, especially longitudinal research, examining inhibition beyond the preschool years. Findings from a few studies support the notion that inhibition stabilizes by the early school age period but that steady improvements continue into adolescence (Best & Miller, 2010; Lehto et al., 2003). Thus, inhibitory skills likely remain stable across childhood (Barkley, 1997; Best & Miller, 2010). Single studies and meta-analytic findings reveal increased inhibitory abilities from ages 5 to 8, which is associated with suppressing a prepotent response (Best & Miller, 2010; Romine & Reynolds, 2005), along with steady improvements into adulthood on impulsivity measures indexed by commission errors, accuracy, and speed (Best & Miller, 2010; Steinberg et al., 2008). In general, research indicates that more mature inhibitory abilities are closely associated with WM functioning, suggesting EF integration.

Therefore, the next section will describe the development of WM and its integrated functioning beginning in early childhood.

Working Memory. WM is commonly defined as the ability to recall, update, and manipulate information over a short time period in the absence of external cues (Alloway, Gathercole, & Pickering, 2006; Baddeley, 2001; Best & Miller, 2010). Baddeley's (2001) model of WM defines three parts to WM: the phonological loop stores auditory information, the visual-spatial sketchpad stores visual-spatial information and the CE manages attentional processes and, by some researchers, is analogous to the executive attention network (Blair & Ursache, 2011; Reuda et al., 2011). The CE deals with the coordination of attention involving the focusing, division and shifting of attention. However, Baddeley along with other researchers further point to inhibitory control (or interference control) mechanisms as also being involved in CE functioning (Baddeley, 2001; Blair & Ursache, 2011; Rueda, Posner, & Rothbart, 2011). As such, the CE manages the processing and/or manipulation of information within WM (Baddeley, 2001; Garon et al., 2008).

Development of storage and rehearsal functions emerge before the more advanced function of manipulating information in mind. WM research on infants suggests that infants can retain information in mind based on findings from toy-hiding tasks (Garon et al., 2008). Span tasks are commonly used to assess WM development in toddlers and preschoolers where increases in digit and word span performance have been observed from ages 3 to 5 years (Espy & Bull, 2005, Garon et al., 2008). In general, increases in WM capacity continues throughout childhood (Garon et al., 2008).

WM functions of updating and manipulating information in mind have been observed as early as toddlerhood via box-cup scrambler and self-ordered pointing tasks (Diamond, Prevor, Callender, & Druin, 1997; Garon et al., 2008). Gains in this ability have been observed (e.g., backward span tasks) in children from ages 3.5 to 7 years of age (Carlson, 2005, Garon et al., 2008). These findings indicate the establishment of all WM functions by early childhood and that continued development pertains to increasing coordination of these functions. Moreover, WM gains associated with the function of manipulating information has been observed during the preschool years. It is theorized that this cognitive ability results from increased coordination of attentional and inhibitory processes that are also taking place. As increased coordination and integration takes place during the preschool and early school age years, attentional control becomes more efficient in the selection of relevant information into WM. This improved efficiency may then support more advanced WM functions pertaining to the management of information in mind. Given that complex WM tasks appear to require functions of attention and inhibition that assist with the maintenance and manipulation of information (Best & Miller, 2010; D'Esposito & Postle, 1999; Garon et al., 2008), it is likely that advanced WM enlists more executive processes that are supported by increased prefrontal cortex functioning. Hence, development of WM likely relies on the development of the CE reflecting the coordination of different WM functions.

Gathercole et al. (2004) lend evidence to the developing role of the CE in young children. The researchers conducted a confirmatory factor analyses of Baddeley's WM model on preschool and school-age children and found support that all three WM

components (phonological buffer, visual-spatial sketchpad, CE) and functions (storage, rehearsal, manipulating information) were established by early school age. Moreover, WM improvements have been observed as linearly increasing in children's abilities to manipulate increasing information loads from ages 4 to 14, reaching adult-like levels by age 15 (Best & Miller, 2010; Gathercole et al., 2004). Thus, the development of later WM is characterized by stable gradual increases in the capacity to store, maintain, and process information coinciding with stable improvements in inhibitory and attentional abilities across childhood (Best & Miller, 2010; Garon et al., 2008).

In sum, EF research findings indicate a general pattern of development characterized by rapid gains in inhibition during early childhood followed by gradual increases into late childhood. This pattern is consistent with the rate of increased development of the prefrontal cortex functioning associated with attentional and inhibitory behaviors across childhood. In contrast, WM reflects a more protracted linear growth from early childhood into adolescence. This steady trajectory may be contingent upon the degree of development by other foundational cognitive processes. Research indicates that the advanced development of attention and inhibition across middle and late childhood support WM's steadily increasing capacity to processes greater amounts of information. Although Garon's integrated framework of EF development provides observed evidence for the developmental EF sequence of inhibition and WM in childhood, more research is needed to better explain how these and other EFs emerge, develop, and integrate to support the development of more advanced EF. The following

section will address other developmental considerations that may inform theoretically-based cognitive models and future research.

Childhood EF: Methodological and Developmental Considerations. Despite notions that more advanced EFs involve integrated use of basic EF components, there is very little longitudinal research examining the effects of earlier EFs on later ones. Given that EF development is considered dynamic with mutual influence among components, assessing the order and manner by which earlier EFs become integrated is challenging. A major barrier in developmental research examining EF is that most studies of EF in childhood share the same methodological problems found in studies of the ADHD literature. Issues such as small sample sizes and cross-sectional designs, along with heavier a focus on early childhood and adolescence as opposed to middle childhood, highlights the need to examine EF development more continuously after the preschool period into adolescence. Doing so would help researchers establish a better developmental account of EFs across all of childhood.

Another limitation is the issue of task impurity where the same EF tasks have been used to measure different EFs. For example, the Stroop and tower task have both been used to assess behavioral inhibition (Ikeda et al., 2013; Kerns et al., 2001), interference control (Bugg, Jacoby & Toth, 2008; Lansbergen et al., 2007; Long & Prat, 2002; Vaidya et al., 2005), and WM (Dehn, 2011; Wolf & Bell, 2004). Thus, careful interpretation of EF findings is essential. In addition, future studies examining EF development will need to provide clearer definitions of EF components that may help behaviorally operationalize EF constructs, as well as alleviate task impurity issues. Thus,

addressing these theoretical and methodological issues via improved developmental models and research design would improve understanding of EF development.

Despite these challenges, findings from EF childhood studies are consistent with the unity/diversity theory of EF. Findings that show different rates of development for different EF components lend support to the notion that EF has separable components (Best & Miller, 2010; Garon et al., 2008). There is also evidence to support a unitary characteristic of EF reflective of integrated functioning, where partial integration among EF components, observed via increased coordination and consolidation, underlies more advanced cognitive functioning. For example, complex inhibitory tasks involve coordinated EF skills of attention and WM (Garon et al., 2008); or, advanced WM functioning is thought to involve a CE component that manages information through interrelated processes of attentional and inhibitory control (Baddeley, 2001; Best & Miller, 2010; Garon et al., 2008).

Lastly, as mentioned before, Garon's integrated framework draws emphasis to the idea that earlier development of EFs facilitate or affect the further development of other EFs. Although far more research is needed to assess how initial EFs affect the development of other EFs, there are some findings to support this notion. For example, early attentional control may facilitate children's later inhibition skills. In a study conducted by Eigsti et al (2006), preschool children who exhibited greater attentional control, via the redirection of attention from rewarding stimuli, demonstrated more efficient and accurate performance on inhibition tasks in adolescence (Eigsti et al., 2006). There is also some evidence to suggest that early inhibition skills may affect the continual

development of WM (Engle, 1996; Rapport et al., 2008). For example, young children's varying interference control abilities have been associated with both lower and higher WM spans for children ranging from preschool to early school age (Espy & Bull, 2005). Moreover, findings from ADHD and WM studies demonstrate that low interference control undermines WM processes of retrieval, maintenance, and encoding (Cornoldi et al., 2001; Engle, 2002; Mecklinger et al., 2003; Palladino & Ferrari, 2013).

More research is needed to examine the longitudinal and concurrent interrelations among EFs such as inhibition and WM. Longitudinal research is needed to examine whether early inhibitory abilities influence later and more advanced WM abilities. Thus, a new model of EF would not only specify the developmental, and perhaps hierarchical, sequence of different core EFs, but it would also identify important sub-processes within core EFs, such as response suppression and interference resolution, that may play a mechanistic role in the development of more advanced EFs.

Summary of ADHD Models and EF Development

Both Barkley and Rapport present a somewhat fixed deficit view of ADHD. Barkley asserts that inhibition must precede and underlie more-advanced EFs, such as WM. Inhibition is theorized to provide the necessary delay needed by WM to process relevant information in service of an appropriate goal directed response. In contrast, Rapport asserts that WM precedes and underlies inhibitory processes because WM is responsible for the interpretation of behavioral information that determines if an inhibitory response is necessary. Their models specify central deficits of a neurodevelopmental disorder (ADHD) that has significant variability in symptom

presentation. Although both researchers acknowledge multiple pathways to ADHD symptom expression, the centrality of a primary deficit is not entirely consistent with a dynamic view of EF development where EF processes may mutually affect each other and become integrated during development (Garon et al., 2008; Miyake et al., 2000). It may be the case that there are a host of central EF deficits that may not only predict to different behavioral presentations of ADHD, but there also may be a sequence of EF deficits, mirroring the order of specific emerging and developing EFs across childhood, that ultimately characterizes pathways to ADHD.

Barkley's and Rapport's models of ADHD may stand to gain from studies examining the development of EFs. As such, Garon et al., (2008) have proposed a comprehensive integrative framework of EF development which highlights how EFs develop across childhood based on the child EF literature. In general, Garon and colleagues (2008) assert that there are three core EF components present in early childhood: inhibition, WM and set-shifting. Regarding one of the first developing EFs, inhibitory responding, Garon and colleagues' developmental framework of EF conceptualize the development of inhibition as emerging and experiencing rapid gains during the preschool period with slower and steady improvements occurring after the early school age years. They also posit that the development of WM begins in infancy but develops in a more linear protracted fashion across childhood and into early adulthood. Moreover, integration of inhibitory processes with WM in later childhood facilitate more advanced and improved WM functioning, where children are able to manipulate information in service of achieving effortful goal directed behavior. As such, this study

will focus on the two core EFs, inhibition and WM, identified by the ADHD literature as central deficits to the disorder as well as identified by the EF literature as emerging and developing first in childhood.

Both Barkley's and Rapport's deficit models suggest inhibition and WM, respectively, as mediating mechanisms in the expression of ADHD behaviors. However, contrary to their perspectives that one EF is more central than the other in the etiology of ADHD, I propose a developmental approach, using Garon's framework, suggesting these EFs may both have influential roles in the manifestation of ADHD symptom expression. Using the research examining these three theories, the current study will present and test two new hybrid models to examine whether early preschool inhibition predicts to later WM performance in preadolescence and whether later WM then then predicts to behavioral or symptom expressions of ADHD.

New Models of ADHD

For the dissertation, I presented two hybrid models of ADHD that incorporated the major EFs highlighted by Barkley, Rapport, and Garon. These two models assessed the extent to which early childhood inhibitory responding positively and directly affects preadolescent WM performance. The models also assessed whether later WM abilities account for concurrent ADHD symptom expression in preadolescence. One model assessed for partial indirect effects by WM on later ADHD symptoms. The other model assessed the moderating role of early inhibition and WM on ADHD symptom expression. In general, these models adopted a developmental approach to help address whether EF deficits underlie symptom expression of ADHD while incorporating a flexible view of

more than one key EF as playing a role in the development of ADHD behaviors. The sections below outline and provide a rationale for the aims of the study, as well as identify the specific hypotheses for each aim.

<center>**Research Aims and Hypotheses**</center>

Model 1

The first model (See Figure 2.1) proposed that preadolescent WM mediates the relation between early school age inhibition and preadolescent behaviors of ADHD. I proposed that the development of inhibition skills during the early school age period may affect the protracted development of WM and that specifically, lower inhibitory skills may undermine WM development over time. Accordingly, observed lower WM in preadolescence is expected to support concurrent behaviors of ADHD inattention and impulsivity/hyperactivity. Further, WM is also expected to partially mediate the relation between early inhibition and preadolescent behaviors of ADHD inattention and impulsivity. Confirmation of the hypotheses below would lend evidence toward the central EF roles of inhibition and WM in the expression of ADHD behaviors.

Aim 1: Prediction of 5yr Inhibition to 10yr WM. Significant development of inhibition has been observed to precede the more protracted development of WM (Best & Miller, 2010; Garon et al., 2008). Specifically, inhibitory functions such as behavioral suppression and interference control are theorized to play central roles in WM. Respectively, they allow for necessary delays and efficient processing of information in WM needed for appropriate behavioral responding or goal directed-activity (Alderson et al., 2010; Barkley, 1997). Given that the development of inhibition and WM are mostly

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stable across childhood (Garon et al., 2008), it may be the case that lower inhibitory skills of suppression and interference control in childhood constrain the development of more advanced WM that takes place in preadolescence. *Hypothesis 1.* The current study hypothesized that children's inhibition skills at age 5 will show a positive direct effect to their later WM performance at 10 years of age.

Aim 2: Prediction of 5yr Inhibition to 10yr Hyperactivity/Impulsivity. Low behavioral inhibition is characterized by difficulty suppressing a dominant response in lieu of a more appropriate subdominant response (Barkley, 1997). As such, low behavioral inhibition is thought to be stimulus driven by positive reinforcements. Uninhibited behaviors often manifest as hyperactive/impulsive often observed as increased activity level, talkativeness, out of seat behavior, and difficulty delaying gratification (Barkley, 1997; Kochanska et al., 1996). During the preschool period, gains in inhibitory control are demonstrated by children's decreased impulsivity and increased verbal and motor control. These gains are supported by greater attention to relevant information (as opposed to less relevant but more positively rewarding information) in the environment that guides appropriate behavioral responding (Best & Miller, 2010; Carlson, 2005; Garon et al., 2008; Kochanska et al., 1996; Steinberg et al., 2008). Thus, difficulties in suppressing dominant behavioral responses in late preschool and early school age may set the stage for continued difficulty in suppressing dominant responses driven by environmental reinforcements of reward and punishment across childhood. This is consistent with developmental evidence showing the stability of inhibitory abilities across childhood and adolescence (Garon et al., 2008; Rueda et al., 2011). As

such, lower levels of inhibition in late preschool or during the early school years is thought to predict to preadolescent observations of hyperactivity and impulsivity. *Hypothesis 2.* The current study hypothesized that children's inhibition skills at age 5 will show a negative direct effect to later behaviors of hyperactivity and impulsivity at age 10.

Aim 3: Prediction of 5yr Inhibition to 10yr Inattention. Lower levels of inhibition during the early school age years may not only result from low behavioral suppression, but also low interference control. Interference control protects appropriate behavioral responding from disruptions of competing irrelevant information or stimuli in the environment (Barkley, 1997; Marchetta et al., 2008). Thus, increased disruptions of competing stimuli or events may result in inattentive-like behaviors such as distractibility, day dreaming, missing important details, forgetfulness, or not appearing to listen. Notably, some cross-sectional studies have shown a link between interference control and behaviors of distraction (Kofler et al., 2010; Palladino & Ferrari, 2013). Given that ADHD inattentive behaviors have been observed to remain stable across the school-age years into adulthood (Barkley, 1997; 2013), lower inhibition skills in the early childhood may support ADHD inattentive behaviors over time. *Hypothesis 3.* The current study hypothesized that children's inhibition skills at age 5 will show a negative direct effect to later ADHD behaviors of inattention at 10 years of age.

Aim 4: Prediction of 10yr WM to 10yr Inattention. The Central Executive (CE) is a theoretical structure in WM that controls attentional and inhibitory processes and manages the flow of information to and from specialized storage/rehearsal systems

(McLeod, 2008). Difficulties in WM may reflect disrupted informational processing of the CE associated with recall, rehearsal, planning, sense of timing, and problem solving. Accordingly, such disruptions may support inattentive behaviors of ADHD such as distraction, forgetfulness, and trouble focusing or concentrating (Barkley, 1997; Rapport et al., 2008). In addition, demands that exceed WM limits of storage, rehearsal, and manipulation of information capacities may also result in behaviors of distraction, difficulties with concentration, trouble focusing, and low sustained effort. There is some evidence to suggest that impairments in the CE (reflective of the coordination and manipulation of information) and storage/rehearsal functions of WM are associated with inattentive symptoms of ADHD in children (Kofler et al., 2010; Rapport et al., 2008). Other ADHD studies have also shown an association between low WM performance and ADHD outcomes in general (Pauli-Pott & Becker, 2011; Willcutt, Doyle, et al., 2005) to include symptoms of behavioral inattention (Fair et al., 2012; Kofler et al., 2010; Nikolas & Nigg, 2013; Rapport et al., 2008). Given this, difficulties with WM in preadolescence may become more evident because of increased academic and social demands.

Hypothesis 4. The current study hypothesized that preadolescent WM performance at age 10 will show a negative direct effect to concurrent ADHD behaviors of inattention.

Aim 5: Prediction of 10yr WM to 10yr Hyperactivity/Impulsivity. Disruptions in WM may also contribute to behaviors that are impulsive or hyperactive. Low interference control in WM may limit informational processing needed to organize and generate an appropriate and effortful behavioral response. Instead, disrupted WM may lead to behaviors that are determined by immediate positive or negative reinforcements in

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the environment as opposed to rule-guided behavior. Therefore, stimulus driven and/or escape-like behaviors may result which may be viewed as impulsive (reactive) or hyperactive (increases in motor/verbal activity). Moreover, there is some evidence to support the association between low WM and behaviors of hyperactivity and impulsivity (Kofler et al., 2008; Rapport et al., 2001; Rapport et al., 2008). Given this, low WM performance in preadolescence may also support behaviors of hyperactivity/impulsivity when social and emotional demands increase during this period. ***Hypothesis 5.*** The current study hypothesized that preadolescent WM performance at age 10 will show a negative direct effect to concurrent ADHD behaviors of impulsivity and hyperactivity.

Aim 6: Mediation of 5yr Inhibition to 10yr Inattention by 10yr WM. EF development across childhood indicates that inhibition undergoes rapid development during the preschool period, whereas WM development is protracted with adult levels of functioning appearing by age 15 (Best & Miller, 2010; Garon et al., 2008; Gathercole et al., 2004). This developmental sequence is supported by findings that show advanced WM processes as relying on established attentional and inhibitory mechanisms within the CE (Baddeley, 2001; Blair & Ursache, 2011; Gathercole, 1998; Rueda et al., 2011). As such, lower CE functioning may be attributed to less developed inhibitory skills early in childhood. Low interference control in the CE may result in frequent disruptions of irrelevant information in WM. These disruptions in turn impede WM functions of manipulating information associated with temporal ordering, planning, reasoning, problem solving, and using internalized speech to guide behavior (Barkley, 1997; 2013; Rapport et al. 2001; 2008). Impaired behavioral suppression may also undermine WM

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functioning. Inhibitory difficulties with behavioral suppression may preclude the delays needed for appropriate information processing and goal-directed responding (Barkley 1997). Hence, low WM functioning through low interference control and/or behavioral inhibition may result in ADHD behavioral expressions of inattention such as low concentration, low persistence, difficulty in resisting distractions, difficulty in organizing complex tasks, and forgetfulness.

Although there is no evidence to my knowledge supporting the mediational role of WM on the effect of early inhibition and later ADHD behaviors of inattention, there is some cross-sectional evidence to support the mediational role of WM on the relation between inhibition and ADHD behaviors of inattention in children (Kofler et al., 2010; Rapport et al., 2008). Given the developmental sequencing of inhibition and WM, it may be the case that early inhibition underlies the development of more advanced WM. Later WM capacity may then support more complex EF behaviors seen in later childhood. As such, lower WM functioning may enable lapses in behavioral and cognitive regulation which manifest as behavioral expressions of ADHD inattention. This may be especially seen during the preadolescent period when greater academic, social, and emotional demands are being made on WM. *Hypotheses 6.* The current study hypothesized that preadolescent WM performance at age 10 will partially mediate the relation between inhibition skills at age 5 and 10 year-ADHD behaviors of inattention.

Aim 7: Mediation of 5yr Inhibition to 10yr Hyperactivity/Impulsivity by 10yr WM. As explained above, earlier inhibition contributes and becomes somewhat integrated with developing WM in childhood (Baddeley, 2001; Gathercole, 1998).

Advanced WM processes rely on inhibitory mechanisms within the CE, such as interference control (Baddeley, 2001; Blair & Ursache, 2011; Gathercole, 1998; Rueda et al., 2011) which in turn may facilitate disruptions of irrelevant information as well as impaired behavioral suppression in WM (Barkley, 1997; 2013; Rapport et al., 2001; 2008). This may lead to failures of attending to relevant information in mind to inform appropriate behavioral responding as well as suppressing a dominant motor or verbal response driven by immediate environmental contingencies of reward and punishment resulting in impulsive and/or hyperactive behaviors (Barkley, 1997). Hence, low WM functioning thru low interference control and/or behavioral suppression may result in ADHD behavioral expressions of impulsivity or hyperactivity such as excessive talkativeness, restlessness, a need to stay busy, blurting out inappropriate comments, or interrupting others. Once again, there is no evidence to my knowledge supporting the mediational role of WM on the effect of early inhibition and later ADHD behaviors of impulsivity and hyperactivity. Rapport and colleagues (2009) offer cross-sectional evidence to support the mediational role of WM on the relation between inhibition and ADHD behaviors of hyperactivity/impulsivity. As such, lower WM functioning in preadolescence may help support concurrent ADHD behaviors of impulsivity and hyperactivity. *Hypotheses 7.* The current study hypothesized that preadolescent WM performance at age 10 will partially mediate the relation between inhibition skills at age 5 and 10 year-ADHD behaviors of impulsivity/hyperactivity.

Model 2

The second model (See Figure 2.2) proposed that inhibition during the early school age years would moderate the effect of preadolescent WM on ADHD behaviors. Lower inhibition skills in early childhood may limit the development of WM gains experienced in preadolescence (Garon et al., 2008; Jones, Rothbart, & Posner, 2003). Hence, lower levels of WM in preadolescence is expected to exacerbate, in non-linear increases, the presence of concurrent ADHD behaviors of inattention and hyperactivity/impulsivity. The first five aims for the second model were the same as the first model described above.

Aim 8: Moderation of 10yr Inattention by 5yr Inhibition and 10yr WM. Given the developmental and integrative sequence of EF development across childhood, early inhibitory skills are theorized to underlie the protracted development of WM (Garon et al., 2008). Low inhibitory skills in the early school age years and its continued stability over childhood may provide less than optimal conditions for the development of WM which relies on inhibitory mechanisms of behavioral suppression and interference control (Barkley, 1997; Garon et al., 2008). Early and sustained difficulties with interference control may facilitate the processing of irrelevant information in WM undermining its more advanced development. Hence, lower inhibitory skills in childhood may negatively interact with more complex WM functioning observed in preadolescence. Poor WM performance during this period may not only contribute to inattentive behaviors of ADHD such as distraction, difficulty concentrating, and forgetfulness, but the presence of these behaviors may become exacerbated as WM demands increase during

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preadolescence. ***Hypothesis 8.*** The current study hypothesized that the interaction term involving 5-year inhibition and 10-year WM will show a non-linear effect on 10-year ADHD behaviors of inattention. It was expected that lower inhibition skills with lower WM skills will enhance the presence of ADHD behaviors of inattention.

Aim 9: Moderation of 10yr Hyperactivity/Impulsivity by 5yr Inhibition and 10yr WM. As mentioned before, low inhibitory skills during the early school age years and its continued stability over childhood may provide less than optimal conditions for the development of WM which relies on inhibitory mechanisms of behavioral suppression and interference control (Barkley, 1997; Garon et al., 2008). Difficulty with the inhibitory mechanism of behavioral suppression in WM may prevent the necessary delay needed for WM to organize an appropriate behavioral response. As such, resulting behaviors may then be determined by immediate environmental contingencies of reward and punishment, characterized as impulsive and/or hyperactive. Hence, lower inhibitory skills early in childhood may interact with later WM skills that are not as developed, negatively affecting behavioral control. Preadolescents may find it more challenging to hold rule guided and goal directed behavior in mind (e.g., WM) resulting in elevated behaviors of hyperactivity/impulsivity. ***Hypothesis 9.*** The current study hypothesized that the interaction term involving 5-year inhibition and 10-year WM will show a non-linear effect on 10-year ADHD behaviors of hyperactivity/impulsivity. Specifically, it was expected that lower inhibition skills with lower WM skills will enhance the presence of ADHD behaviors of hyperactivity/impulsivity.

Figure 2.1 Conceptual Parent Mediation Model

The following models (Figures 2.1 and 2.2) control for 2-year externalizing behaviors, sex, and prior levels of ADHD inattention and hyperactivity/impulsivity at 4 years of age. Statistical comparison of the models cannot be done; however, both models can be compared to their respective baseline models. Statistically significant differences between the baseline and proposed models indicate that the proposed models fit the data better than the baseline model. Fit statistics such as RMSEA, CFI, and SRMR were used to show how well the proposed models fit the data.
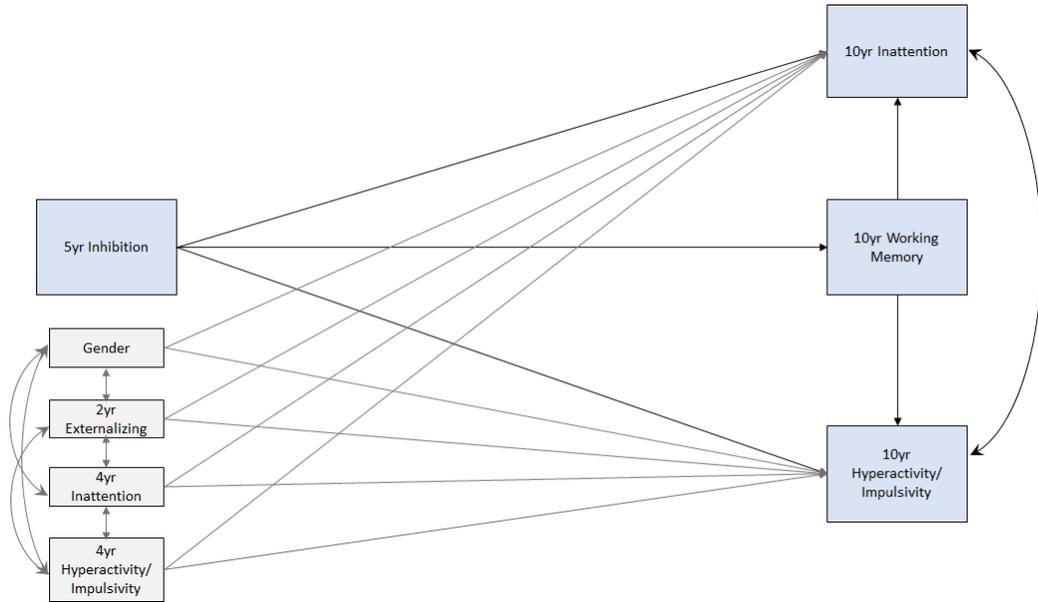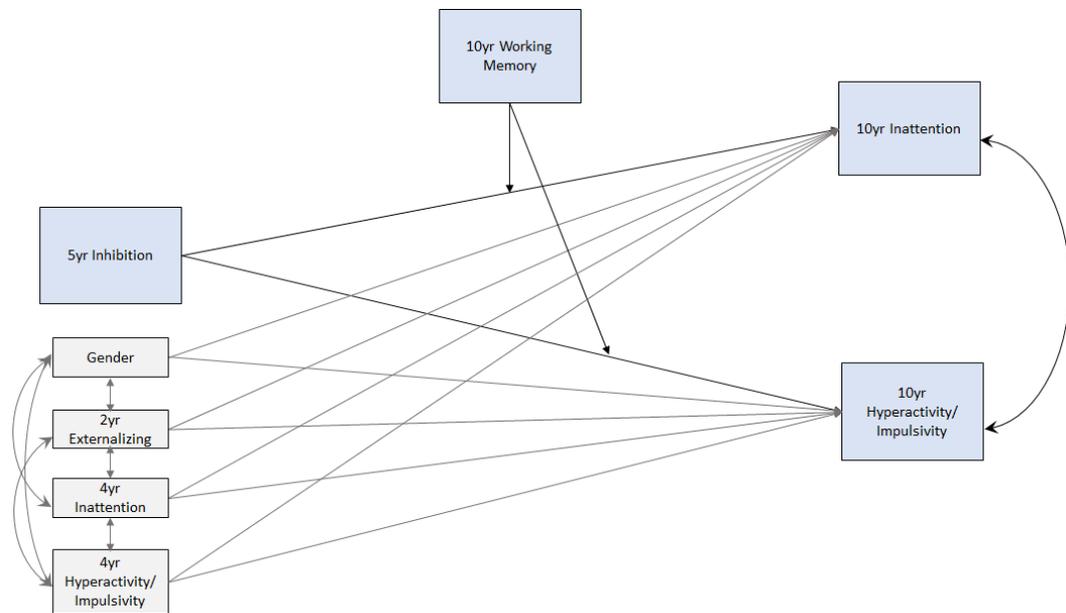
Figure 2.2. Conceptual Parent Moderation Model

CHAPTER III

RESEARCH METHOD

Participants

The current study used data, already collected, from three cohorts of children who were part of an ongoing longitudinal study of social and emotional development. The goal for recruitment of the larger study was to obtain a sample of children who were at risk for developing future externalizing behavior problems, and who were representative of the surrounding community in terms of race and socioeconomic status (SES). All cohorts were recruited through child day care centers, the County Health Department, and the local Women, Infants, and Children (WIC) program in North Carolina. Potential participants for cohorts 1 and 2 were recruited at two-years of age (cohort 1: 1994-1996 and cohort 2: 2000-2001) and screened using the Child Behavior Checklist (CBCL 2-3; Achenbach, 1992), completed by the mother, to over-sample for externalizing behavior problems. Children were identified as being at-risk for future externalizing behaviors if they received an externalizing T-score of 60 or above. Efforts were made to obtain approximately equal numbers of males and females. This recruitment effort resulted in a total of 307 selected children. Cohort 3 was initially recruited when infants were six-months of age (in 1998) for their level of frustration, based on laboratory observation and parent report, and were followed through the toddler period (Calkins, Dedmon, Gill, Lomax, & Johnson, 2002 for more information). Children whose mothers completed the CBCL at two-years of age were joined with participant families from cohorts 1 and 2 (*N*

= 140). The entire sample total was $N = 447$, of which 37% of the children were identified as being at risk for future externalizing problems and 15% ($N = 447$) were identified as being at risk for future internalizing problems. There were no significant demographic differences between cohorts regarding gender, χ^2 (2, $N = 447$) = .63, p = .73, race, χ^2 (2, $N = 447$) = 1.13, p = .57, or two-year SES, F (2, 444) = .53, p = .59.

Of the 447 originally selected participants, six were dropped because they did not participate in any two-year data collection. An additional 12 families participated at recruitment, did not participate at two-year, but did participate at later years. Data collection for all three cohorts occurred during the following years: cohort 1: 1996-1997, cohort 2: 2000-2001, cohort 3: 1999-2001. At four years of age, 399 families participated (data collection- cohort 1: 1998-1999, cohort 2: 20002-2003, cohort 3: 2001-2003). Families lost to attrition included those who could not be located, moved out of the area, declined participation, or did not respond to phone and letter requests to participate. There were no significant differences between families who did and did not participate at age four in terms of gender, χ^2 (1, $N = 447$) = 3.27, $p = .07$, race, χ^2 (1, $N = 447$) =.65, $p =.42$, two-year SES, t (432) = -.92, $p =.36$, or two-year externalizing T score, t (445) =.45, $p =.65$. At age five, 365 families participated, including four that did not participate in the four-year assessment (cohort 1: 2000-2001, cohort 2: 2003-2004, cohort 3: 2003-2004). Again, there were no significant differences between families who did and did not participate in terms of gender, χ^2 (1, $N = 447$) = .76, $p = .38$, race, χ^2 (1, $N = 447$) = .14, $p = .71$, 2-year SES, t (432) = -1.93, $p = .06$, and 2-year externalizing T score, t (445) = 1.39, $p = .17$. At seven years of age, 350 families participated, including 19 that

did not participate in the five-year assessment (cohort 1: 2001-2002, cohort 2: 2005-2006, cohort 3: 2005-2006). Again, there were no significant differences between families who did and did not participate in terms of gender, χ^2 (1, $N = 447$) = 2.12, $p = .15$, race, χ^2 (3, $N = 447$) = .19, $p = .67$, and two-year externalizing T score, t (445) = 1.30, $p = .19$. Families with lower 2-year SES, t (432) = -2.61, $p < .01$, were less likely to participate in the seven-year assessment. At age ten, 357 families participated, including 31 families that did not participate in the seven-year assessment (cohort 1: 2005-2006, cohort 2: 2008- 2009, cohort 3: 2007-2009). No significant differences were noted between families who did and did not participate in the ten-year assessment in terms of child gender, χ^2 (1, $N = 447$) = 3.31, $p = .07$; race, χ^2 (3, $N = 447$) = 3.12, $p = .08$; 2-year SES, $t(432) = .02$, $p = .98$; or 2-year externalizing T score, t (445) = -.11, $p = .91$.

Procedures and Measures

Children and their mothers came to a university-based laboratory in Greensboro, NC to participate in the study. Consent from mothers and, beginning at age 4, verbal assents from children, were obtained before data collection began. Mothers completed questionnaires in a private setting. Mother-child dyads also participated in several tasks that assessed emotional and physiological regulation not used in the present study. Although laboratory procedures were not identical across time points, the study used analogous age-appropriate tasks for children and their mothers (when children were 4, 5, and 10 years of age) to maintain measurement equivalence. Mothers received an honorarium of $50 for every laboratory visit they participated in across the years of the study, and children received small age-appropriate toys for their participation.

5-Year Laboratory Visit

Children and their mothers returned to the laboratory for a 2-hour visit when children were 5 years old. The current study will use the following measures and laboratory tasks measuring behavioral inhibition (suppression of a behavioral response) and interference control.

Walk the Line. Children participated in a behavioral suppression task that involved walking on a ribbon strip taped to the laboratory floor (the line). Children walked on the ribbon for three trials. The first baseline trial was defined by children walking on the ribbon at their regular pace. For the second and third trials, children were instructed to walk on the ribbon more slowly. The experimenter timed each trial. Behavioral inhibition was indexed by the proportion of the slowdown from duration scores of trial 2 and trial 3 to the total time of trials 2 and 3 using the following formula: T3-T2/T2+T3 (Gandolfi, Viterbori, Traverso, & Usai, 2014). Using the proportion of the slowdown normalizes the varying completion times among participants. Greater scores indexed greater behavioral inhibition.

Star Tracing Task. Children participated in another behavioral suppression task involving drawing. Children drew a star within two lines outlining the shape of a star on a worksheet. The baseline condition consisted of children drawing between the lines of the star at their own pace. The second trial consisted of children drawing between the lines of the same star shape, but more slowly. The experimenter timed how long children drew their star figures. The proportion of the slowdown from duration scores from baseline and the first trial was calculated by the following formula: T2-T1/T1+T2 (Bachorowski &

Newman, 1985; Gandolfi et al., 2014). Using the proportion of the slowdown normalizes the varying completion times among participants. Greater scores indexed greater behavioral inhibition.

Shapes (Stroop-Task). Children participated in an interference control task involving pictures of animals, shapes, and letters. The first picture drawing was of a large chicken with little triangle shapes inside the chicken figure. The experimenter asked the children whether there was "something funny about the picture?" If children were unable to identify the triangle shape as incongruous, the experimenter assisted children in correctly identifying the triangles. Children did not proceed to the first trial until they correctly identified the triangles. Children were then instructed to name the figure inside the larger picture as fast as they could. The experimenter recorded accuracy scores from children's responses for 20 pictures. Higher accuracy scores were used to index interference control.

Inhibition Composite. A composite variable for inhibition was created using performance ratings from the Walk the Line, Star Tracing, and Stroop Shape tasks. Full z-score standardization of the variables was not used because z-score transformations do not preserve covariance information needed for SEM estimation. Instead, the Proportion (or Percentage) of Maximum Scaling (POMS) transformation was used to scale each differently measured variable indexing inhibition on the same metric (Little, 2013; Moeller, 2015). This scale transformation is a monotonic transformation, meaning the transformation does not affect rank order of individual data or the associations among variables; it also preserves mean-level changes over time. The POMS transformation is

commonly used for rescaling questionnaire items on different Likert scales such as converting a narrow scale to a wider scale by converting a variable's scale to range from 0 to 1 if a proportion score, or 0 to 100 if a percentile score (Little, 2013). Thus, the POMS transformation was similarly used to convert the narrow percentile scale of the Stroop Shape task with percentile values ranging from 0 to 100, to the wider scales of both the Walk the Line and Star Tracing tasks which yield percentile values from -100 to +100. The POMS transformation formula used was as follows:

$$POMS = [(observed- minimum)/(maximum-minimum)] \times 100$$

Resulting scores were interpreted as the percentage of the possible maximum score. After POMS scores were derived for the Stroop Shape, Walk the Line, and Star Tracing tasks, the scores were averaged to comprise an overall inhibition composite score. Higher percentile scores indicated greater behavioral inhibition.

10-Year Laboratory Visit

Preadolescents and their mothers returned to the laboratory for two 3-hour laboratory visits: Temperament and IQ-Achievement visit when children were 10 years old. For the IQ assessment, clinically trained experimenters administered the Wechsler Intelligence Scale for children--Fourth Edition (WISC-IV). The current study will use the following measures and laboratory tasks outlined below indexing WM and ADHD symptom expression.

WISC-IV. Children completed the Digit Span and Letter Number Sequencing subtests that make up the WM Index. For the Digit Span task, the experimenter read

aloud a series of numbers and asked preadolescents to recall the sequence of numbers they heard in order and reverse order. Of note, Digit Span Backwards is known to better index WM skills compared to Digit Span Forward which is better known to index attention and auditory recall (Rosenthal et al., 2006). For the Letter-Number Sequencing subtest, preadolescents heard a sequence of numbers and letters provided by the experimenter. Preadolescents recalled the letter-number information in both numerical and alphabetical order. Scaled scores (*M*=10; *SD*=3; range: 1-19) were calculated for each subtest.

D-KEFS Tower Task. The Tower Task required preadolescent participants to move five disks of different sizes arranged on three pegs from a starting position to an end position. Participants were instructed to move the pegs to achieve an end position visually represented to them via a picture. Participants could move the disks adhering to two rules: (1) they were only allowed to move one disk at a time; and (2) they were not allowed to place a larger disk on a smaller disk. The Tower Task involves WM wherein one must use his/her recall and planning skills. As such, the D-KEFS total achievement scaled score was used (*M*=10; *SD*=3; range: 1-19).

WM Composite. To best index WM, scaled scores for Digit Span Backwards, Letter Number Sequencing, and Tower Task, which are on the same scale, were averaged to form a WM composite. Moreover, a constant of 4 was multiplied to the composite score to make this variable's variance more homogenous with the other variable variances (composite score range: 4-76). This was done to prevent failed estimation of

the structural equation model (Kline, 2005). Higher mean scores reflected greater WM skills.

ADHD Rating Scale-IV (ADHD-RS). Mothers completed the ADHD-RS (DuPaul, Anastopoulos, Power, Murphy, & Barkley, 1994), an 18-item questionnaire consisting of 9 inattention- and 9 hyperactive/impulsive- symptoms listed in the DSM-IV criteria for ADHD. Mothers rated each item about their preadolescent over the past 6 months on a 0 (not at all) to 3 (very often) frequency scale. Items were summed to yield a total Inattention score and a Hyperactivity/Impulsivity score (range: 0-27). Good internal consistency scores have been found for this measure on both the school and parent versions. Coefficient alphas for inattention were .86 and .96, respectively; and .88 for hyperactivity/impulsivity (DuPaul, Power, Anastopoulos, & Reid, 1998). Consistent with previously reported reliability scores, the current sample's coefficient alphas were as follows: Inattention subscale, consisting of 9 items, (α =.93) and Hyperactivity/ Impulsivity subscale, consisting of 9 items, (α =.91). Higher summation scores for each presentation type indicated greater ADHD symptom expression.

Covariates

To account for third variable influences that may also affect the study's outcome variables (expressions of ADHD in preadolescence), the current study controlled for the following variables: sex, initial levels of externalizing behavior at age 2, and 4-year ADHD symptom scores of Inattention and Hyperactivity/Impulsivity from the ADHD-RS (described above). Parental observations of ADHD behaviors and high levels of externalizing behaviors in early childhood frequently co-occur with ADHD behaviors

later in childhood. As such, these factors are likely to have a biasing effect on outcome variables of ADHD inattention and hyperactivity/impulsivity in preadolescence. Similarly, child sex is also known to be associated with ADHD presentation type. Hence, controlling for early ADHD symptoms and externalizing behaviors as well as participant sex will better isolate any significant effects resulting from other study variables (inhibition and WM).

Sex. Child sex was coded as follows: male (1) and female (2) and was initially included in models given that the literature indicates that males typically exhibit higher levels of hyperactivity and impulsivity than females and conversely, females are more likely to show ADHD symptoms of inattention (Barkley, 2013; Gershon & Gershon, 2002). No associations were found between child sex and later ADHD symptom behaviors and therefore child sex was removed from the models. This finding was surprising given that sex differences are well documented in the literature (Arnett et al., 2015). However, it may be the case that sex differences are likely to disappear as children get older (Barkley, 2013; Simon, Czobor, Bálint, Mészáros, & Bitter, 2009) and/or when children exhibit greater ADHD symptom severity which may occur less within a community sample.

Externalizing. To control for initial levels of externalizing behavior in toddlerhood, the current study used data from the Child Behavior Checklist -2/3 (CBCL; Achenbach, Edelbrock & Howell, 1987). Mothers completed the CBCL, a 99-item questionnaire, at the first laboratory visit when children were 2 years of age. This questionnaire assessed child behavior problems and social competencies observed during

the two previous months. Items were on a 0 to 2 scale (i.e., 0 = not true; 1 = somewhat or sometimes true; 2 = very true). The externalizing non-gendered norm raw scale scores were used based on Achenbach (1991) recommendation that raw scores on the CBCL behavior syndromes and problems scales be used in research.

ADHD-RS. To control for prior ADHD behaviors earlier in childhood, the study used rating scores from the ADHD-RS 18-item questionnaire completed by participant mothers when child participants were 4 years of age. See above for more information on this measure. Sum scores from the Inattention and Hyperactivity/Impulsivity subscales were used.

CHAPTER IV

RESULTS

Analyses of the data are organized into two sections. First, preliminary analyses are reported for all indicator variables; and second, primary analyses of the current study identify the best fitting models and describe longitudinal predictions and mediations/moderations observed.

Preliminary Analyses

Analytic Approach

Means, standard deviations, ranges, and correlations were analyzed for all variables as presented in Table 4.1. Skewness and kurtosis tests for normality were conducted and slight- to moderate- non-normality were found for some variables with skewness between 1 and 2 and kurtosis between 1 and 2.2. Slight to moderate skew and kurtosis are defined by values between 1 and 2 and 1 and 7, respectively (Curran, West, & Finch, 1996; Gao, Mokhtarian, & Johnston, 2008). Because departure from normality was not severe and full information maximum likelihood estimation (FIML), used in the analyses, is robust to violations of normality assumptions and incomplete data (Enders, 2001); no variable transformations were conducted because doing so may distort linear relationships among the variables or may render the models empirically unidentifiable (Gao et al 2008).

Model estimations used pairwise deletion to maximize the use of all the data, resulting in a sample of 435 participants. The selected sample was 52% female and 32% non-white (African American, Hispanic, other) participants. Socioeconomic status was indexed using Hollingshead scores (Hollingshead, 1975) yielding a range from 39.61-44.30, suggesting that the sample was predominantly middle class across the 2-, 4-, 5-, and 10-year visits. The average participant age in months at each visit was as follows: 4-year visit (M=53.73; SD=3.73); 5-year visit (M=68.05; SD=3.25); 10-year IQ-Achievement visit (M=127.69; SD=3.03); and, 10-year Temperament visit (M=128.07; SD=3.59). Of note, sample numbers varied by study variable due to missing data explained below (see Table 4.1).

Regarding issues of power, to detect small size effects when testing moderation and/or mediation, a sample size of approximately 315 is needed to have 80 percent power at an alpha of .05 (Aiken & West, 1991; Kenny, 2011). Similarly, a necessary sample size of 200 is necessary to conduct moderate-sized structural equation models of power .80 at an alpha of .05 (Oertzen, Hertzog, Lindenberger, & Ghisletta, 2010).

To summarize scales of the study variables, the scales of covariate variables were as follows: externalizing behaviors at age 2 were measured using raw scale scores and sum scores were used for 4-year ADHD behaviors of inattention and hyperactivity/ impulsivity (see Table 4.1 for score ranges). The same scale used for 4-year ADHD was also used for 10-year ADHD outcome variables. A percentage score was derived for the 5-year inhibition composite predictor variable and scaled scores were used for the 10-year WM composite variable. Of note, a constant of 4 was multiplied to the scaled scores

of WM (see Table 4.1 for range in scores) to ensure homogeneity of variable variance with other variables and prevention of failed model estimation (Kline, 2005).

Correlations between variables were derived (see Table 4.1). All covariates, 2-year externalizing behavior and 4-year ADHD behaviors of inattention and hyperactivity/ impulsivity were significantly and positively associated with each other (r (348) =.37, .44, $p < .001$, respectively) as well as 10-year ADHD inattention and hyperactivity/ impulsivity scores (r (295) = .40, .46, $p < .001$, respectively). Higher inhibition response scores at 5 years of age were positively and significantly related to higher WM scores at 10 years of age, r (271) = .44, $p < .001$ and marginally significant to lower scores of 10-year inattention, r (280) = .10, $p < .10$. Notably, 5-year inhibition scores were not associated with later 10-year hyperactivity/impulsivity scores. Higher WM scores at 10 years of age were significantly and negatively associated with lower inattention and hyperactivity/impulsivity scores at age 10 (r (282) = -.21, $p < .001$; -.17, $p < .01$ respectively).

Missing Data

Data that are missing due to participant attrition as well as study design was addressed in all analyses. Of note, sample numbers for each variable vary due to missing participant data across cohorts and study years (see Table 4.1 for the different N's). Full information maximum likelihood (FIML) was used to address any bias produced by the current study's non-ignorable missing data as well as other types of missing in data. Data were imputed at the item level and using Mplus v. 7.0 (Muthén & Muthén, 1998-2013). FIML was used at the measure and wave levels only. FIML has been shown to produce

unbiased parameter estimates and standard errors when data are missing at random (MAR), missing completely at random (MCAR), when data are nonignorable, and when the amount of missingness is large (e.g., > 25%; Collins, Schafer & Kam, 2001; Graham, 2003). FIML estimates a likelihood function for each participant based on all available variables so that all data are used. Model fit was derived by summing fit functions from every case, allowing the overall model to use fit information from all cases while also using all available variable information.

Primary Analyses

Pathways of 5-year inhibition predicting to 10-year WM predicting 10-year ADHD symptom expression were conducted within a series of nested path analyses conducted in Mplus v.7.0 (Muthen & Muthen, 1998-2013). Nested models allow for competing hypothesized models to be assessed using model comparison. One baseline model of direct effects was compared to a larger, more complex parent model assessing mediation. Similarly, another baseline model of direct effects, containing an interaction variable, was compared to the hypothesized larger, more complex parent model assessing moderation (see Figure 4.1). Of note, the parent mediation and moderation models could not be directly compared because these models are not nested within each other due to being structurally different.

Chi-square difference tests were used to identify whether the parent (larger and more complex) models were better fitting than their respective baseline (smaller and simpler) models. If the difference between the two nested models is significant, then the model with more paths, the parent model, explains the data better (Kline, 2005) (see

Table 4.2). In addition, the following fit statistics were used to help identify the best fitting model regarding how likely a model can reproduce the data well: Confirmatory Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and Standardized Root Mean Square Residual (SRMR). Fit statistic cut offs were based on Hu and Bentler (1999)'s suggestion of using a combination of one of the relative fit indices such as CFI, Tucker Lewis Fit Index (TLI), (where values larger than .90 -.95 are considered good) along with one or both of the following fit indices, SRMR (where good model fit < .08) and/or RMSEA (where good model fit < .06).

Structural Model Comparisons

Model Comparisons. A summary of model fits of the nested models can be found in Table 4.2. Chi^2 difference tests were conducted to determine whether the baseline or parent model was significantly better fitting than the other. Both Chi^2 difference tests between the Baseline Mediation Model and Parent Mediation Model as well as the Baseline Moderation Model and the Parent Moderation Model were significant (for both models, $\Delta \chi^2 (6) = 608$, $p < .001$). Hence the parent models demonstrated significantly better model fit compared to their respective baseline models (See Table 4.2).

Next, a series of fit statistics were used to evaluate how well the parent models fit the data. CFI, RMSEA, and SRMR fit statistics for the Parent Mediation Model were as follows: .99, .04, and .06, respectively. For the Parent Moderation Model, fit statistics of CFI, RMSEA, and SRMR were as follows: .40, .44, and .11, respectively. As such, model fits were very good for the Parent Mediation Model indicating that the model is expected

to reproduce the data consistently. Model fits were within the suggested cutoff fit statistic ranges (Hu & Bentler, 1999), indicating that analyzing significant pathways for that model is appropriate and meaningful. In contrast, fit statistics for the Parent Moderation Model were poor, suggesting that the model is unlikely to reproduce the data consistently and therefore analyzing significant pathways is not meaningful. Hence, only the Parent Mediation Model estimates are interpreted below (see Figure 4.2).

Mediation Model

      Control Variable Effects. Study control variables of 2-year externalizing, and 4-year inattention and hyperactivity/impulsivity positively predicted to outcome variables of 10-year inattention and hyperactivity/impulsivity. 2-year externalizing significantly predicted to 10-year inattention ($\beta = 0.27$, $p < .001$) and 10-year hyperactivity/ impulsivity ($\beta = 0.26$, $p < .001$). In contrast, 4-year inattention only predicted to 10-year inattention ($\beta = 0.35$, $p < .001$) and not 10-year hyperactivity/impulsivity. As a result, the pathway of 4-year inattention predicting 10-year hyperactivity/impulsivity was not included in the Parent Mediation model. Similarly, 4-year hyperactivity/impulsivity positively predicted to 10-year hyperactivity/ impulsivity ($\beta = 0.43$, $p < .001$) but not 10-year inattention, therefore this path was also not estimated in the Parent Mediation model.

      Direct Effects. There was support for the significant direct effect of 5-year inhibition on 10-year WM ($\beta = 0.44$, $p < .001$). In addition, 10-year WM was found to have a significant negative direct effect on 10-year inattention ($\beta = -0.19$, $p < .01$) and only a marginally significant negative effect on hyperactivity/impulsivity ($\beta = -0.10$, $p <$

.10). Contrary to the study's hypotheses, no direct effects were found for early 5-year inhibition on later 10-year inattention and hyperactivity/impulsivity.

Indirect Effects. The current study hypothesized that 10-year WM would partially mediate the relation between 5-year inhibition and 10-year inattention as well as 10-year hyperactivity/impulsivity (See Figure 4.1). Although standard requirements for testing classic mediation with regression analysis indicate the presence of significant direct effects from the predictor to the mediator, a direct effect from the mediator to the outcome, and a direct effect from the predictor to the outcome (Baron and Kenny, 1986); more recent understanding of mediational processes emphasize that the direct path from the predictor to the outcome variable does not have to be significant in the presence of mediation (Hayes, 2009; Shrout & Bolger, 2002). As such, a more effective alternative to test for mediation is to use the bootstrapping approach (MacKinnon, Lockwood, & Williams, 2004).

Because significant pathways were found among the study variables in the expected direction, the indirect effect was therefore tested using a bias-corrected bootstrapping procedure (10,000 draws). MacKinnon and colleagues have shown that this approach generates accurate confidence intervals for indirect effects by reducing Type I error rates and increasing power (MacKinnon, et al., 2004). If a value of zero does not occur within the 95% confidence interval range for the indirect effect, then the indirect effect is considered significant indicating mediation. Hence, the indirect relations involving 5-year inhibition to 10-year WM to 10-year outcomes of inattention and hyperactivity/impulsivity were probed.

As hypothesized, a significant indirect effect was observed for 5-year inhibition to 10-year WM to 10-year inattention. As Figure 4.2 indicates, the standardized regression coefficient between 5-year inhibition and 10-year WM was statistically significant, as was the standardized regression coefficient between 10-year WM and 10-year inattention. The standardized indirect effect was tested using a bootstrap estimation approach of 10,000 samples (MacKinnon et al., 2004), $\beta = -.082$, SE = .027. The 95% confidence interval was computed by determining the indirect effects at the 2.5th and 97.5th percentiles. Given that the range did not include a value of 0, the indirect effect was statistically significant, 95% CI = -.135, -.029. The results indicate that 10-year WM mediated the effect of 5-year inhibition on 10-year inattention. Of note, partial mediation could not be established because the main predictor variable, 5-year inhibition did not significantly predict to the outcome variable of 10-year inattention. Hence, a reduction from the main predictor to the outcome variable when the mediator is accounted for, could not be estimated. Nonetheless, the results indicate that through the indirect effect of 10-year WM, 5-year inhibition has a small significant impact on 10-year inattention whereby an increase of one standard deviation in 5-year inhibition results in an 8.2% standard deviation decrease in 10-year inattention. Contrary to expectations, no indirect effect was observed from 5-year inhibition to 10-year WM to 10-year hyperactivity/ impulsivity (see Table 4.3 for confidence interval estimates).

Table 4.1. Descriptive Information and Correlations

	1	2	3	4	5	6	7
N	400	374	374	335	296	326	326
M	14.24	5.82	7.97	66.83	42.10	6.87	4.72
SD	7.98	4.64	5.23	12.51	7.51	5.74	4.96
Range	46.00	27.00	26.00	61.45	49.33	27.00	27.00
1 Externalizing 2yr							
2 Inattention 4yr	.37***						
3 Hyperactivity/Impulsivity 4yr	.44***	.78***					
4 Inhibition 5yr	-.11†	-.12*	-.10†				
5 WM 10yr	-.15*	-.13*	-.16**	.44***			
6 Inattention 10yr	.40***	.48***	.42**	-.10†	-.21***		
7 Hyperactivity/Impulsivity 10yr	.46***	.48***	.57**	-.08	.17**	.75***	

Note: Descriptive statistics are presented in the upper part of the table. Correlation coefficients are found in the lower triangle of the table.
$^\dagger p < .10$ * $p < .05$ ** $p < .01$ *** $p < .001$

Table 4.2. Fit and Model Comparisons

Models	χ^2	df	p	CFI	RMSEA	SRMR
Baseline Mediation Model	621	14	<.001	0.00	0.32	0.23
Parent Mediation Model	13	8	.224	0.99	0.04	0.06
Baseline Moderation Model	1357	15	<.001	0.00	0.45	0.22
Parent Moderation Model	749	9	<.001	0.40	0.44	0.11

χ^2 *difference tests*

Model comparisons	$\Delta \chi^2$	Δ df	P
Baseline Mediation vs. Parent Mediation	608	6	<.001
Baseline Moderation vs. Parent Moderation	608	6	<.001

Note. Reduced nested models were compared to their parent model.

Table 4.3. Model Estimates and Bootstrap 95% Confidence Intervals

Indirect paths	Estimate	Confidence Interval	
		Lower	Upper
5yr Inhibition → 10yr WM → 10yr Inattention	-.082	-.135	-.029
5yr Inhibition → 10yr WM → 10yr Hyperactivity/Impulsivity	-.045	-.090	.000

Table 4.4 Sex Differences Across Study Variables

	Females			Males			
	N	M	SD	N	M	SD	t-test
Externalizing 2yr	208	13.64	7.47	192	14.89	8.48	ns
Inattention 4yr	201	5.20	4.66	173	6.53	4.52	2.77**
Hyperactivity/Impulsivity 4yr	201	7.51	5.44	173	8.50	4.94	ns
Inhibition 5yr	180	67.50	12.30	155	66.04	12.75	ns
WM 10yr	165	10.56	1.91	131	10.48	1.84	ns
Inattention 10yr	178	6.41	6.08	148	7.41	5.27	ns
Hyperactivity/Impulsivity 10yr	178	4.41	4.89	148	5.09	5.03	ns

**$p<.01$. *t*-tests were conducted to estimate sex differences across all study variables. Sex was only significantly different for the covariate, 4-year Inattention. Males endorsed higher mother reported symptoms of inattention than females, $t(372) = 2.77$, $p = .006$

Figure 4.1. Nested Path Models

Nested path models assessing pathways of 5-year inhibition to 10-year WM to 10-year symptom expressions of ADHD. (a) Baseline Mediation Model, assesses sequential direct paths. (b) Parent Mediation Model, is the bigger more complex model assessing covariates and all hypothesized pathways. (c) Baseline Moderation Model, assesses all sequential direct paths with the interaction variable. (d) Parent Moderation Model, is the bigger more complex model assessing covariates and all hypothesized pathways.
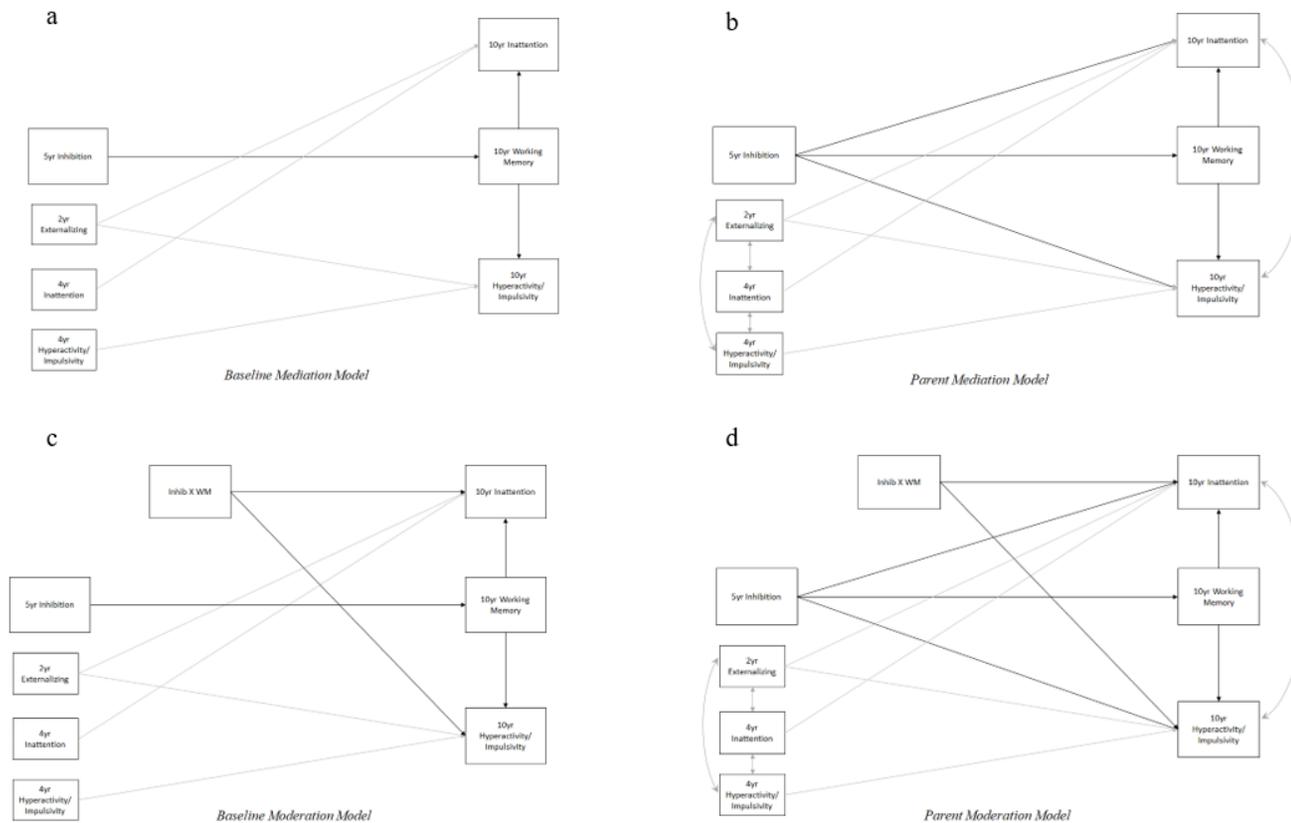
Figure 4.2. Final Indirect Model
Indirect model of 10-year WM mediating 5-year inhibition on later 10-year symptoms of ADHD using standardized path coefficients. Model includes covariates of 2-year externalizing, 4-year inattention, and 4-year hyperactivity/impulsivity variables.
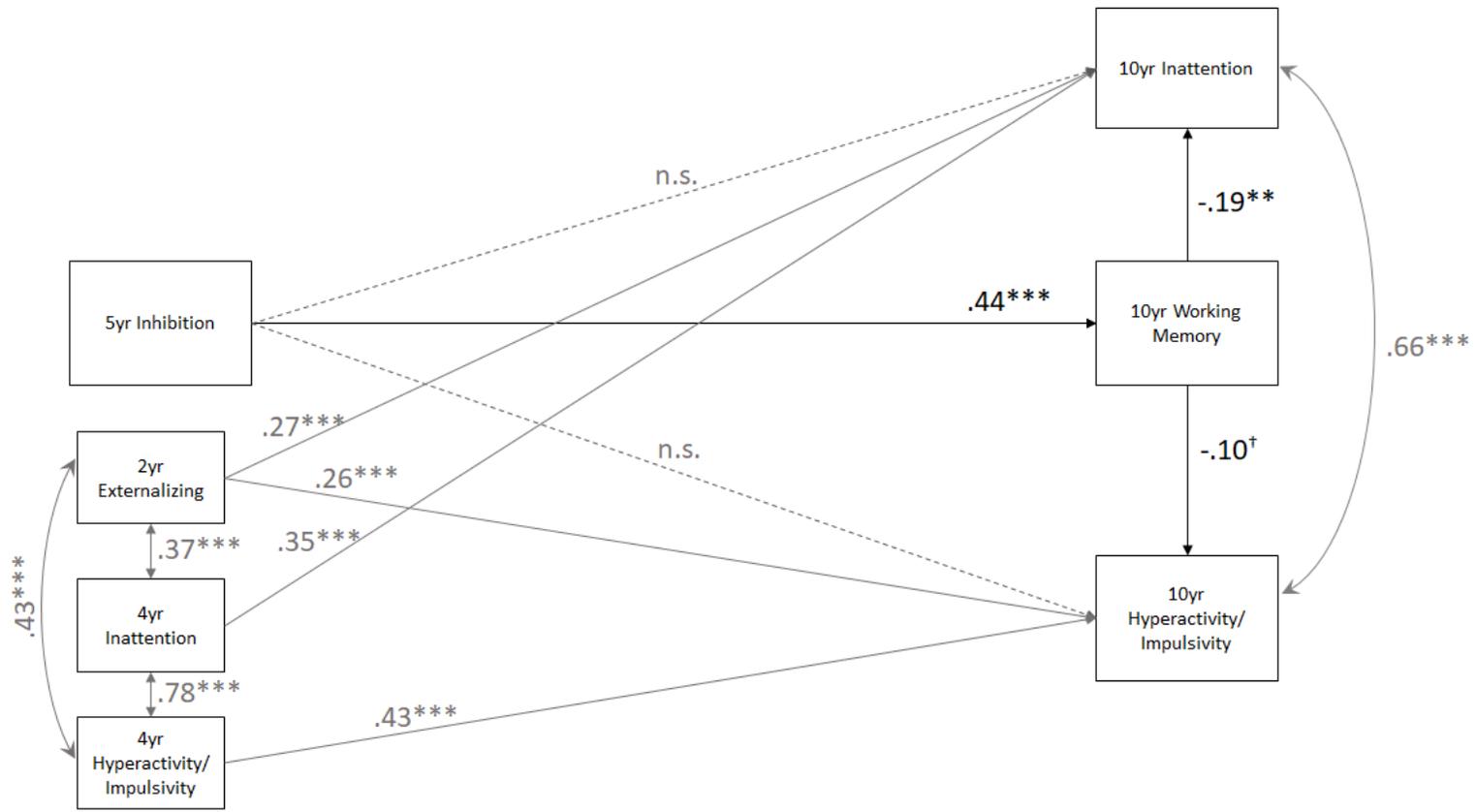
†p<. 10, *p<. 05, **p<. 01, ***p<. 001

CHAPTER V

DISCUSSION

Recent research highlights the complex associations between EF and ADHD symptomatology. The current study contributes to the literature by testing two developmental EF hybrid models incorporating Barkley's and Rapport's theories of ADHD which implicate the role of inhibition and WM. To my knowledge, this is the first study to merge these theories using a developmental EF framework thereby showing that both EFs have influential roles in the manifestation of ADHD symptom expression over time. Given that much ADHD research examining inhibition and WM are mostly cross-sectional and provide mixed support as to whether one or both EFs predict to ADHD outcomes (Martinussen et al., 2005; Mullins et al., 2005; Willcutt, Doyle, et al., 2005), the two hybrid developmental models serve to further elucidate how these two EFs underlie ADHD behaviors.

Moreover, research to date shows that EFs emerge and undergo significant development across childhood and therefore it is essential for ADHD research examining EF contributions to consider using a developmental approach to delineate EF pathways that support the manifestation of ADHD behaviors. This study, to my knowledge, is one of the first studies to use a longitudinal framework to account for developing EF roles on later ADHD outcomes, controlling for earlier expressions of ADHD. As such, the models tested in this study sought to establish whether earlier EFs of inhibition influence the later

more advanced EF of WM and whether WM plays a mediational as well as moderational role in ADHD symptomology. The models tested in this study lend evidence toward an EF model of ADHD as elaborated by both Barkley (2013) and Rapport et al (2008), as well as the integrative model of EF proposed by Garon et al (2008).

Research Findings and Implications

Summary of Study Hypotheses

The current study's hypotheses were mostly confirmed for the mediation model. Specifically, children's inhibition skills in early childhood positively and significantly predicted to later WM performance in preadolescence (Hypothesis 1). In turn preadolescent WM was found to significantly predict negatively to concurrent preadolescent inattention and marginally predict negatively to hyperactivity/ impulsivity (Hypotheses 4,5). Moreover, preadolescent WM was found to mediate effects of early childhood inhibition to preadolescent inattention (Hypothesis 6).

Contrary to expectations, early school age inhibition did not predict to preadolescent inattention or hyperactivity/ impulsivity (Hypotheses 2,3). Nor was it found that preadolescent WM mediated the relation between early childhood inhibition and preadolescent symptom expression of hyperactivity/impulsivity (Hypothesis 7). Moreover, due to the poorly fitted moderation model, (Hypotheses 8, 9) which proposed that the interaction term between early childhood inhibition and preadolescent WM would be significant, could not be analyzed; hence, moderating effects on preadolescent ADHD symptom expression could not be confirmed.

Explanation of Results

The study's findings from the first hypothesis help to confirm Garon's integrated developmental model of EF. Support was found for the contribution of early childhood inhibition to later preadolescent WM. Specifically, earlier inhibition in childhood was positively related to later WM in preadolescence thereby indicating lower levels of early childhood inhibition as predicting to lower WM performance in preadolescence. This finding is consistent with Garon's (2008) integrated EF framework which suggests an EF deficits model where weaker earlier EFs undermine the development of more advanced integrated EFs. It may be the case that earlier maturation of inhibition precedes and is necessary for the more protracted development of WM (Best & Miller, 2010; Garon et al., 2008); and that inhibitory functions such as behavioral suppression and interference control may become integrated as part of later WM (Baddeley, 2001; Best & Miller, 2010). Such inhibitory mechanisms would allow for the necessary delays and efficient processing of information characteristic of more advanced WM functioning (Alderson et al., 2010; Barkley, 1997).

Some support was found for Rapport's model of WM and ADHD. The results of the current study mostly supported Hypotheses 4 and 5 in which WM significantly and negatively predicted to behaviors of inattention while also marginally and negatively predicted to behaviors of hyperactivity/impulsivity. These findings are mostly consistent with Rapport's model of ADHD which posits that difficulties in WM reflect disrupted informational processing of the CE. These disruptions translate to difficulties with recall, rehearsal, planning, sense of timing, and problem solving which support inattentive

behaviors of ADHD such as distraction, forgetfulness, and trouble focusing or concentrating (Barkley, 1997; Rapport et al., 2008). Further, it may also be the case that when demands of the environment exceed WM capacities of storage, rehearsal, and manipulation of information, resulting behaviors of distraction, trouble focusing, and low sustained effort are likely to be endorsed by the individual. The current findings contribute to the growing body of evidence that WM difficulties contribute to inattentive symptoms of ADHD (Fair et al., 2012; Kofler et al., 2010; Nikolas & Nigg, 2013; Rapport et al., 2008).

The current results regarding the hypothesized relation of lower preadolescent WM performance at age 10 predicting to concurrent ADHD behaviors of impulsivity and hyperactivity (Hypothesis 5) did not reach statistical significance at p<.05 value. However, a trend toward statistical significance was observed in the expected direction at the *p*=.07 value. Future studies are needed to better delineate whether disruptions in WM contribute to behaviors that are impulsive and/or hyperactive. Confirmation would lend support to the idea that perhaps low interference control in WM limits informational processing used in the organization and generation of effortful and appropriate behavioral responses or rule-guided behavior. Hence disruptions in WM would encourage stimulus driven or escape-like behaviors that are positively or negatively reinforcing characterized as impulsive (reactive) or hyperactive (increased motor or verbal activity) behaviors.

Results supported Hypothesis 6 in which an indirect effect for WM was found for behaviors of inattention but not for behaviors of hyperactivity/impulsivity (Hypothesis 7). These findings somewhat support Rapport's and Barkley's models of WM and ADHD.

The indirect effect by WM transmitting earlier effects of inhibition to later outcomes of ADHD symptom expression of inattention provides the first finding, to our knowledge, of longitudinal support for developing EF effects on later ADHD behaviors. The model's mediational finding suggests that more mature inhibitory capacities in early childhood is foundational to the more protracted development of WM. Conversely, weaker inhibition in early childhood may undermine WM functioning later in childhood and preadolescence. This impaired functioning denotes lower CE functioning in WM characterized by frequent disruptions of irrelevant information which impede appropriate storage and processing of relevant information in mind. As such, these disruptions may result in difficulties with concentrations, temporal ordering, planning, reasoning, and problem solving (Barkley, 1997; 2013; Rapport et al., 2001; 2008) manifesting as inattentive behaviors of low concentration, low persistence, difficulty in resisting distractions, difficulty in organizing complex tasks, and forgetfulness. Of note, difficulties with inattention may be especially evident during the preadolescent period when greater academic, social, and emotional demands are being made on WM.

The current study did not find any evidence to support Hypotheses 7 positing the mediating effect of 10-year WM between inhibition at age 5 and 10 year-ADHD behaviors of impulsivity/hyperactivity. This finding points to the possibility of the differentiating role of EF deficits as characterizing different pathway outcomes of ADHD. Given that WM incorporates inhibitory processes, as defined by CE functioning, deficits in WM may impact and support behaviors of inattention only. It may be the case that pathways leading to hyperactivity /impulsivity may be better accounted by either

more concurrent lapses in inhibition or via other mechanisms such as the brain's arousal system.

In fact, disconfirmation of the hypotheses supports observed evidence in the literature of children characterized with low WM as not exhibiting increased levels of motor activity and difficulties with impulse control (Lui & Tannock, 2007; Martinussen & Tannock, 2006). These findings suggest a more predominate and alternate pathway that supports behaviors of impulsivity and hyperactivity. The role of underlying tonic cortical hypoarousal may better explain children's difficulties with impulsivity and hyperactivity in ADHD (Nigg, 2013). The Optimal Stimulation Theory (Zentall & Zentall, 1983) and more recent theories of top-down executive control posit that ADHD symptoms of hyperactivity and impulsivity result from chronic states of underarousal or dysregulation of arousal and alertness (Loo et al., 2009; Sergeant, 2000; 2005) rather than failures in EF such as inhibition or WM. However, while there is evidence linking hypoarousal with ADHD status (James et al., 2016), there is little research highlighting this link exclusively with hyperactivity/impulsivity. As such, further research testing alternate ADHD models of arousal and EFs would further inform how these different mechanisms contribute to symptom expressions of ADHD; and whether these mechanisms differentiate among ADHD presentations of inattention and hyperactivity/impulsivity.

Another surprising set of findings of the current study was the lack of support for Hypotheses 2 and 3 which asserted that early childhood inhibition would directly predict

to ADHD symptom expression of hyperactivity/impulsivity and inattention, respectively.

Gains in inhibitory control have been observed during the preschool period in which

children begin to show decreases in behavioral impulsivity and greater verbal and motor

control (Garon et al., 2008). These gains are supported by greater attention to relevant

information that lead to rule guided behaviors (Best & Miller, 2010; Carlson, 2005;

Garon et al., 2008; Kochanska et al., 1996; Steinberg et al., 2008). It was hypothesized

that difficulties in suppressing dominant behavioral responses in early childhood would

set the stage for continued difficulty to enlist inhibitory responding across childhood.

Given developmental evidence showing the stability of inhibitory abilities across

childhood and adolescence (Garon et al., 2008; Rueda et al., 2011) the current study

asserted that uninhibited behaviors during the preschool period would positively predict

to later stimulus driven behaviors of impulsivity and hyperactivity such as increased

activity level, talkativeness, out of seat behavior, and difficulty delaying gratification

(Barkley, 1997; Kochanska et al., 1996) during the preadolescent period. Therefore, lack

of confirmation of this finding was unexpected especially when there is evidence to

support the observation of inhibition predicting to ADHD behaviors of hyperactivity and

impulsivity (Barkley, 1997; Miller et al., 2010; Rubia et al., 1998); as well as,

developmental evidence showing the stability of inhibitory abilities across childhood and

adolescence (Garon et al., 2008; Rueda et al., 2011).

Similarly, no support was found for the third hypothesis which asserted that lower

levels of inhibition during early childhood would predict to later preadolescent inattentive

behaviors. It was hypothesized disruptions in inhibitory interference control would allow

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for disruptions of irrelevant information resulting in inattentive behaviors such as distractibility, day dreaming, missing important details, forgetfulness, and difficulty listening (Barkley, 1997; Marchetta et al., 2008). The EF literature indicates that mastery of interference control comes online during the preschool period where it protects against disruptions of competing stimuli that support inattentive behaviors (Garon et al., 2008). Thus, children with low levels of interference control during the early school period may be on a pathway marked by lower interference control functioning expressed via inattentive behaviors. As such, the non-significant prediction of early inhibition on later outcomes of inattention is inconsistent with findings linking interference control with behaviors of distraction (Kofler et al., 2010; Palladino & Ferrari, 2013) as well as evidence indicating the stability of inattentive behaviors across the school-age years (Barkley, 1997; 2013).

One major explanation for these non-significant findings concerns whether independent effects of early school inhibition on later childhood ADHD behaviors should be expected. Barkley's theoretical viewpoint of inhibition being foundational and underlying the development of other EFs suggests that the independent effect of earlier childhood inhibition may be 'absorbed' by other EF processes that are needed for the regulation of behavior later in development. It may be the case that later symptoms of ADHD in preadolescence would be more greatly accounted by advanced and more developed EFs, such as WM. As noted by Miyake et al's theory of EF (2000; Miyake & Friedman, 2012), advanced EFs such as WM, are defined by integrated functioning of

simpler EFs. Specifically, WM is noted to have integrated inhibitory functions such as interference control as denoted by the CE (McLeod, 2008).

Given this, the effects of early childhood inhibition on later preadolescent ADHD behaviors may more likely bear an indirect influence, rather than a direct one, on the development of ADHD over time. Notably, the current study shows this. Moreover, the study's findings may be compatible with literature findings showing that inhibitory deficits are associated with ADHD symptoms during preschool and early school-age periods (Brocki & Bohlin, 2004; Brocki, Nyberg, Thorell, & Bohlin, 2007; Kerns et al., 2001; Sonuga-Barke et al., 2002) as opposed to older school age children--where WM deficits are more likely to be associated with ADHD symptoms (Brocki et al., 2007; Willcutt, Doyle et al., 2005). This may also be consistent with observations from several studies that show WM to be a more prominent neurocognitive marker of later ADHD behaviors when compared to other EFs like inhibition (Castellanos and Tannock, 2002; Martinussen et al., 2005; van Lieshout et al., 2017). As such, it may be more likely that early school inhibition would predict to concurrent early ADHD behaviors but not later preadolescent behavioral expressions.

Another explanation of the non-significant findings concerns the notion posited by Barkley who suggests that poor EF functioning in children with ADHD are due to delays in EF maturation (Barkley, 1997). This indicates that all children continue to make maturational EF gains. As a result, any improvements in inhibitory functioning over time may reduce observed direct effects from early inhibition on later ADHD behaviors. It

should be noted that within the study, a notable decrease in mode response for ADHD symptom/severity endorsement was observed from 4- to 10-year observations.

It may also be the case that children who showed lower levels of inhibitory responding and interference control may very well have been exhibiting ADHD behaviors across childhood. If so, then it is also possible that such children may have received pharmacological treatment, especially for those children showing greater symptom/severity endorsement of ADHD. Given that the current study did not take into consideration the effects of pharmacological interventions, such treatment may have affected observations of later ADHD behaviors, thereby reducing direct effects originating from early school age inhibitory functioning. However, given that the study's sample was not clinical in nature, it is not expected that a major portion of participants to have been on medication.

Lastly, findings from the study did not support a moderational model. This is surprising given that deficits in WM and inhibition are likely to exacerbate behavioral expressions of ADHD in a non-linear pattern where greater deficits predict to more extreme observations of ADHD impairment reaching the clinical range. As such, the current study may not have had enough participants within the sample endorsing clinical levels of ADHD. Post hoc review of ADHD symptom endorsement at 10 years of age show less than 5% of the sample obtaining scores at the very high range of ADHD symptom severity for either the inattentive, hyperactive/impulsive, or combined types. This low frequency would make it difficult to statistically establish a non-linear

relationship among the predictor variables, hence explaining why the moderation model was a poor fitting to start.

An alternative explanation is that lower levels of inhibition and WM may not necessarily scale to greater expressions of ADHD symptoms at a non-linear rate. This may be the case if participants have recruited compensatory skills or resources to help address cognitive and behavioral weaknesses, dampening or reducing overall behavioral expressions of ADHD. For example, a participant with poor WM at age 10 may have had the benefit of being placed in supportive class environments over time with minimal distractions and teachers helping them with planning and organizing. This in turn would allow them to develop compensatory skills that mask neurocognitive deficits via environmental aids while dampening ADHD symptom expression observed by caregivers.

Limitations and Future Directions

Limitations of the study are of both a theoretical and methodological nature. Theoretically, the EF constructs of both inhibition and WM may be considered impossible to truly measure. Although WM and inhibition are widely accepted, it is arguable that these constructs cannot be truly verified. There is much overlap in the neural networks and behaviors which distinguish these different EFs. As such, both inhibition and WM cannot be directly measured in a pure way making these constructs ultimately unfalsifiable (Parkin, 1998). Given this, caution is warranted in the interpretation of results and it is necessary to acknowledge the role of other explanatory variables as accounting for observed outcomes of ADHD behaviors.

Methodological limitations with the current study include the lack of a clinical sample, concurrent, as opposed to longitudinal, observations between WM and ADHD symptom expression, and parent report of preadolescent ADHD behaviors. Examination of this model on a clinical sample may help further confirm observed mediational effects, as well as reveal possible moderation effects by EFs. Use of earlier measures of WM before age 10 but after age 5 would have been a better longitudinal test of how developing EFs of inhibition and WM impact behavioral expressions of ADHD. Furthermore, use of better measures of EF that are developmentally appropriate and reduce task impurity would strengthen the study's findings. And lastly, use of teacher report of ADHD behaviors, relative to parent observations, may be a more reliable report of child functioning outcomes. Arguably, the school setting is likely to present demands that would tax inhibitory and WM deficits not found in the home setting. In addition, it may be the case that in a school setting, children are unable to rely on aids easily found in other environments, such as their home, that would mask EF deficits (i.e., parents helping with planning and problem solving) thereby making teacher report of ADHD behaviors more realistic.

Ultimately, future research would benefit from testing a crossed-lag model assessing stability of inhibition and WM across childhood into adolescence and its effects on ADHD outcomes. Testing more time points across childhood would help establish the role of earlier EFs on later more advanced EFs as well as confirm the stability of EFs over time. A cross-lag model would also help delineate the nature of how EFs of WM

and inhibition dynamically contribute to ADHD outcomes via indirect effects over time, lending more evidence toward the mechanistic role of EFs on ADHD.

The results of the current study suggest that EF processes of early inhibition and later WM play a role in the symptom expression of ADHD. There was strong evidence, of a longitudinal nature, for the indirect effect of inhibition by WM on ADHD behaviors of inattention but not for hyperactivity /impulsivity. Benefits of path analyses used in the current study allowed for the more accurate estimation of the influence of other variables (Sameroff, 2009) as well as the optimal assessment of direct and indirect effects among variables. These analyses therefore reinforce the study's findings supporting the plausibility of the causal model hypothesized (Jeon, 2015). As such, given the mixed support for the role of inhibition and WM in ADHD, further research that accounts for the transactional nature of these EFs across childhood within a cross-lag path model would further increase our understanding of these dynamic influences on child development of ADHD behaviors over time.

Relatedly, given that a major limitation of the study was the provision of two developmental snap shots (early school age and preadolescence) in the assessment of EF functioning; a huge area of needed research is in the understanding of EF development of inhibition and WM from ages 5 to 10. Unpacking how inhibition and WM unfold during this critical school period would significantly increase understanding how specific developments in inhibition and WM occur and predict to later functioning, especially regarding behavioral expressions of ADHD. Identification of individual differences in inhibition and WM would allow the field to better delineate how variation among

developmental trajectories lead to certain cognitive, emotional, and behavioral consequences that present as ADHD symptoms.

Although, assessing EFs such as inhibition and WM across the childhood years pose significant measurement challenges due to issues of task impurity and establishing measurement equivalence to compare changes across ages; future research may begin to address these challenges by employing similar EF tasks, robust to age, that only increase in difficulty as a function of age. For example, researchers could use simple manual motor and oculomotor inhibition tasks that rely on the principle of suppressing a prepotent response via eye or bodily movement (Munakata, Michaelson, Barker, & Chevalier, 2013; Roberts, Fillmore, & Milich, 2011). Inhibiting eye movements may require having a child resist the urge to look toward something compelling and distracting. As such, the number and quality of distracting events may increase and be modified with increasing age (Munakata et al, 2013; Roberts et al, 2011). Similarly, a Go-No-Go hand tapping task could also index inhibition because it requires the suppression of a motor response. This individual would tap his/her fingers after receiving one tap on his/her hand (by the experimenter) and would withhold from tapping when receiving two taps. To address changing abilities due to age, one can increase the task difficulty by presenting more complex sequences and making simple rule changes, such as reversing task instructions (Kipss & Hodges, 2005). Use of such measures may provide sensitivity in tracking developmental EF changes, such as inhibition and WM, across a period in childhood not often researched.

Another important area where future research is needed and that bears significant implications in the development of ADHD are sex differences. Notably, the current study did not find sex differences to outcomes of preadolescent ADHD behaviors. Moreover, post-hoc analyses show that the only study variable that revealed a significant effect for sex was the covariate, 4-year inattention (see Table 4.4). The lack of sex differences possibly suggest that these differences may occur at clinical levels of ADHD behaviors and not within normative ranges exhibited by a community sample. Replication of such findings would help the field understand perhaps when and how these differences emerge.

Conclusion

In sum, the purpose of this study was to examine longitudinal EF pathways in predicting preadolescent ADHD behaviors. In general, the results of the study supported the hypotheses of the longitudinal contributory effects of earlier EFs, specifically inhibition and WM, on later ADHD symptom expression. Specifically, regarding Barkley's and Rapport's models of ADHD, both models implicate a direct effects and causal role for inhibition (Barkley 1997; 2013) and WM (Rapport et al., 2008), in the emergence of ADHD behaviors. Some support for these models were found in the current study. In general, lower executive performance for both inhibition and WM contributed mostly to ADHD behaviors of inattention. In addition, findings also supported the mediational role of WM as transmitting the indirect effect of earlier inhibition to later behavioral expressions of ADHD inattention. However, little to no support was not found for the role of early childhood inhibition and preadolescent WM on behavioral

expressions of hyperactivity/impulsivity suggesting the possibility that deficits in WM may differentiate to behavioral presentations of inattention.

Results also lend support toward Garon et al's (2008) developmental EF framework where earlier development of EFs facilitate or contribute to the development of more complex EF's. This study showed that early childhood inhibition longitudinally predicted to preadolescent WM. Taken together, these findings expand the literature on child EF development and behavioral pathways that may lead to ADHD. Future studies should focus on cross-lagged longitudinal designs to understand the stability and transactional effects of EFs and ADHD behaviors using a symptom dimensional approach. Further, it should also be noted that the current study only focused on inhibition and WM and therefore other factors such as set-shifting or cortical arousal should be examined to further understanding in the area of ADHD.

Lastly, clinical implications of understanding childhood development of inhibition, WM, and ADHD behaviors on a continuum may shed light on prevention and intervention efforts that lessen social, emotional, cognitive, and behavioral impairments. For example, early detection of children's limits in their inhibitory and WM skills may support academic achievement, address early psychosocial difficulties, and foster self-regulation skills that may ameliorate other comorbidities associated with ADHD behaviors. Moreover, understanding sex differences and different EF profiles may help to identify children most at risk for adverse outcomes of ADHD. This in turn, would facilitate the provision of early intervention and supports that promote optimal social, emotional, and cognitive development across childhood.

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