Working Memory Deficits and Emotion Dysregulation in Youth with Attention-Deficit/Hyperactivity Disorder: Understanding Relationships and Treatment Implications

Jodi Zehava Uderman
Graduate Center, City University of New York

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WORKING MEMORY DEFICITS AND EMOTION DYSREGULATION IN YOUTH WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER: UNDERSTANDING RELATIONSHIPS AND TREATMENT IMPLICATIONS.

by

JODI Z. UDERMAN

A dissertation submitted to the Graduate Faculty in Clinical Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York

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dissertation requirement for the degree of Doctor of Philosophy.

Anil Chacko, Ph.D.

Date Chair of Examining Committee

Joshua Brumberg, Ph.D.

Date Executive Officer

Anil Chacko, Ph.D.
Deborah Walder, Ph.D.
Anne-Claude Bedard, Ph.D.
Supervisory Committee

THE CITY UNIVERSITY OF NEW YORK
ABSTRACT

WORKING MEMORY DEFICITS AND EMOTION DYSREGULATION IN YOUTH WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER: UNDERSTANDING RELATIONSHIPS AND TREATMENT IMPLICATIONS

by

Jodi Z. Uderman

Advisor: Dr. Anil Chacko

Evidence suggests that working memory (WM) impairment is a primary deficit in attention-deficit/hyperactivity disorder (ADHD), underlying core symptoms of the disorder and associated impairments. However, the relationship between deficits in different WM components and emotional problems specifically in ADHD has not yet been studied. Knowledge of the cognitive substrates contributing to emotional deficits in ADHD could inform efforts toward refining cognitive remediation as a treatment for emotion dysregulation in this population.

The first aim of this study (Aim 1) investigated whether WM deficits, as a global construct, were related to and mediated the relationship between ADHD and emotion dysregulation. The first sub-aim (Aim 1A) examined whether different components of WM, as defined by the traditional Baddeley Model (i.e., phonological loop, visuospatial sketchpad, central executive), were related to and differentially mediated emotion regulation deficits in ADHD. The second sub-aim (Aim 1B) examined whether WM constructs mediated the relationship of ADHD symptoms severity with degree of emotion dysregulation, after examining mediating effects of, and adjusting for, other factors related to emotional functioning. Other factors included inattention (relative to total ADHD symptoms), hyperactivity/impulsivity
(relative to total ADHD symptoms), comorbid ODD symptoms, negative parenting behaviors, parental stress, and social skills deficits. Given a possible relationship between WM deficits and emotion dysregulation, Aim 2 investigated whether improvement in WM skills following cognitive remediation was related to improvement in emotion regulation ability.

Participants were youth with ADHD (ages 7 – 11 years) who were recruited for a larger treatment study investigating the augmentative and complementary effects of combined WM training and behavioral parent training. At a baseline visit, objective neuropsychological data measuring Baddeley components of WM ability and parent-ratings of emotion dysregulation, ADHD symptoms, comorbid ODD symptoms, parenting behaviors, and parental stress were collected (Aim 1). Teacher ratings of ADHD symptoms, ODD symptoms, and social skills were also collected at baseline. For Aim 2, participants were randomly assigned to an active or low-level, non-scaffolded (“placebo”) condition of WM training. Upon the completion of WM training, WM deficits and parent-rated emotion dysregulation were again measured.

Aim 1 and sub-aims results: Deficits in WM, as a composite measure, were not associated with emotion dysregulation in this sample and, therefore, not included in the mediation model. With regard to the Baddeley components of WM, a deficit in the phonological loop was associated with parent-rated emotion dysregulation in ADHD (but not visuospatial sketchpad and central executive deficits). As such, the phonological loop factor was included in the mediation model. Lastly, a phonological loop deficit, comorbid ODD symptom severity, negative parenting behaviors (e.g., inconsistent use of discipline), parental stress, and social skills deficits together mediated the relationship between ADHD and emotion dysregulation. However, no variables contributed to the mediation above and beyond comorbid ODD symptom severity. Aim 2 Results: Improvement in overall WM functioning was significantly associated
with better emotion regulation from pre-to-post WM training in the active group relative to the placebo group.

Results suggest that many factors, including a deficit in the phonological loop, contribute to emotional impairment in youth with ADHD, the strongest being severity of comorbid ODD symptoms. Results also suggest that, while comorbid ODD may contribute most strongly to emotion dysregulation in this study, improvements in total WM ability after cognitive remediation were nonetheless associated with improvement in emotion regulation skills. Taken together, cognitive remediation may have effects on emotional functioning in youth with ADHD; developing a more precise understanding of the neurocognitive substrates underlying emotional deficits in ADHD could thus inform treatment strategies.
ACKNOWLEDGEMENTS

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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abstract</td>
<td>iv</td>
</tr>
<tr>
<td>Acknowledgements</td>
<td>vii</td>
</tr>
<tr>
<td>List of Tables</td>
<td>x</td>
</tr>
<tr>
<td>List of Figures</td>
<td>xi</td>
</tr>
<tr>
<td>Specific Aims</td>
<td>1</td>
</tr>
<tr>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>Introduction to Attention-Deficit/Hyperactivity Disorder</td>
<td>4</td>
</tr>
<tr>
<td>Working Memory and Attention-Deficit/Hyperactivity Disorder</td>
<td>10</td>
</tr>
<tr>
<td>Emotion Regulation and Attention-Deficit/Hyperactivity Disorder</td>
<td>18</td>
</tr>
<tr>
<td>Working Memory, Emotion Regulation, and Attention-Deficit/Hyperactivity Disorder</td>
<td>29</td>
</tr>
<tr>
<td>Treatment Implications</td>
<td>33</td>
</tr>
<tr>
<td>The Current Study</td>
<td>36</td>
</tr>
<tr>
<td>Methods</td>
<td></td>
</tr>
<tr>
<td>Participants</td>
<td>41</td>
</tr>
<tr>
<td>Measures</td>
<td>42</td>
</tr>
<tr>
<td>Procedure</td>
<td>49</td>
</tr>
<tr>
<td>Statistical Analyses</td>
<td>52</td>
</tr>
<tr>
<td>Results</td>
<td>56</td>
</tr>
<tr>
<td>Discussion</td>
<td>76</td>
</tr>
<tr>
<td>References</td>
<td>88</td>
</tr>
</tbody>
</table>
### LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Demographic Information for Aim 1 Sample</td>
<td>57</td>
</tr>
<tr>
<td>2.</td>
<td>Means, Standard Deviations (SD), and Ranges for all variables used in the baseline sample</td>
<td>59</td>
</tr>
<tr>
<td>3.</td>
<td>Pearson Product Correlations of emotion dysregulation and all hypothesized independent variables and mediators</td>
<td>61</td>
</tr>
<tr>
<td>4.</td>
<td>Pearson Product Correlations of parent-rated ADHD symptoms and all hypothesized mediators</td>
<td>63</td>
</tr>
<tr>
<td>5.</td>
<td>Pearson Product Correlations of all Included Mediator</td>
<td>65</td>
</tr>
<tr>
<td>6.</td>
<td>Mediation of the effect of parent-rated ADHD symptom severity on emotion dysregulation through phonological loop, parent-rated inattention, parent-rated ODD, inconsistent discipline, parenting stress, and social skills</td>
<td>66</td>
</tr>
<tr>
<td>7.</td>
<td>Demographic Information for Aim 2 Sample, by Condition</td>
<td>69</td>
</tr>
<tr>
<td>8.</td>
<td>Means and Standard Deviations (SD) of Change Scores and Cohen’s d from Pre-to-Post Cogmed across Groups</td>
<td>72</td>
</tr>
<tr>
<td>9.</td>
<td>Pearson Product Moment Correlations and Fisher r-to-z transformation of Changes in Working Memory Variables and Emotion Dysregulation from Pre-to-Post CWMT</td>
<td>74</td>
</tr>
</tbody>
</table>
LIST OF FIGURES

Page

1. Multiple mediation model with j factors, adapted from Preacher & Hayes (2008)..........................54
Introduction

Specific Aims

The first aim of this research was to investigate whether working memory deficits mediated the relationship between emotion dysregulation and ADHD. A variable is thought to act as a mediator when it can account for the relation between a predictor variable and the outcome (Baron & Kenny, 1986). Because working memory deficits have been shown to underlie a host of symptoms and related impairment in ADHD (Rapport, 2001; Rapport et al., 2008) and because working memory deficits are related to emotional dyscontrol in other populations (e.g., Stout & Rokke, 2010; Schmeichel & Demaree, 2010; Schmeichel, Volokhov, & Demaree, 2008; Van Dillen & Koole, 2007; McRae, Jacobs, Ray, John, & Gross, 2012; Joormann & Gotlib, 2010), it was hypothesized that:

1. Working memory impairment would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.

Aim 1A

A sub-aim (Aim 1A) investigated this relationship more precisely by examining whether the separate Baddelely components of working memory differentially mediated the relationship between ADHD symptom severity and degree of emotion dysregulation, relative to using a more general composite measure. Working memory components included the visuospatial sketchpad, the phonological loop, and the central executive. In response to Baddeley’s (2013) theoretical model, it was hypothesized that:

1. Central executive deficits would most strongly mediate the relationship between ADHD symptom severity and degree of emotion dysregulation, as compared to phonological loop and visuospatial sketchpad deficits. Although visuospatial working memory deficits are
more common in ADHD relative to phonological deficits, the relative importance of visuospatial central executive functions versus phonological central executive functions in the mediation of ADHD symptom severity and degree of emotion dysregulation was exploratory.

**Aim 1B**

A second sub-aim (Aim 1B) examined whether other factors that are thought to impact emotional functioning in these youth, such as ODD symptom severity, inattention (uniquely), hyperactivity/impulsivity (uniquely), negative parenting behaviors (e.g., poor monitoring/supervision, low parental involvement, lack of positive parenting practices, inconsistent discipline, use of corporeal punishment), parenting stress, and social skills deficits mediate the relationship between ADHD symptom severity and degree of emotion dysregulation. Because “emotion dysregulation” is a multifaceted and complex construct, it is likely that several factors work together to contribute to emotional problems in youth with ADHD and that working memory deficits would mediate this relationship, along with several other variables. Utilization of a multiple mediation model allowed for the simultaneous investigation of each variable as a possible mediator individually, while controlling for the effect of the other proposed mediators. It also allowed for the direct comparison of the strength of each mediator (see below). Although there is a paucity of literature on this topic, there is some theoretical and/or empirical evidence that the factors listed above (e.g., ODD, inattention, hyperactivity/impulsivity, negative parenting behaviors and emotions, social skills deficits) may be associated with emotion dysregulation in ADHD and/or in other pediatric populations (e.g., Banaschewski et al., 2012; Melnick & Hinshaw, 2000; Sobanski et al., 2010). Taken together, Aim 1B further investigated how these factors were related to emotion regulation deficits in youth with ADHD, in addition to working memory deficits.
Based on past empirical literature (Banaschewski et al., 2012; Melnick & Hinshaw, 2000; Sobanski et al., 2010) it was hypothesized that:

1. ODD symptom severity would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.
2. Inattentive symptoms would not mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.
3. Hyperactive/impulsive symptoms would not mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.
4. Negative parenting behaviors, including lack of involvement, lack of positive parenting, poor monitoring/supervision, use of inconsistent discipline, and use of corporeal punishment would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation. Because each of these behaviors was analyzed individually, the relative strength of each separate negative parenting behavior in the mediation of ADHD symptom severity and degree of emotion dysregulation was exploratory.
5. Parental stress would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.
6. Social skills deficits would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.

An examination of the relative importance of each mediator was exploratory. Moreover, separate constructs were used for parent- and teacher-rated ADHD and ODD symptoms in order to utilize cross-informant data and to understand these relationships across settings. Hypotheses regarding the differences between parent- and teacher-rated constructs across aims were exploratory in
nature, as the use of teacher-rated data when studying emotion dysregulation in ADHD has been limited.

Aim 2

Given recent evidence that cognitive remediation (particularly CWMT) may increase aspects of working memory in youth with ADHD (e.g., Klingberg, Forssberg, & Westerberg, 2002) and that working memory training may improve emotion regulation ability in healthy adults (Schweizer, 2013), the second aim of this research was to investigate the effects of working memory remediation on emotion dysregulation among youth with ADHD. It was hypothesized that:

1. In the CWMT active group relative to control group, improvement in working memory (e.g., total working memory change score) would be associated with improvement in emotion regulation (e.g., emotion dysregulation change score) from pre-to-post working memory training. Moreover, with regard to the Baddeley components of working memory, in line with the hypothesis above, it was hypothesized that change in central executive working memory ability would be most strongly related to change in emotion regulation ability.

Introduction to Attention-Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common childhood psychiatric disorders (Merikangas et al., 2010) with prevalence rates from three to nine percent of all school-aged children (APA, 2000) and a worldwide prevalence estimate of five percent (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). Prevalence rates do not significantly differ across regions of the world (Willcutt, 2012). As its name suggests, the disorder is characterized by symptoms of inattention (e.g., difficulty sustaining attention, forgetfulness, distractibility), hyperactivity (e.g., fidgeting/squirming, leaving one’s seat), and impulsivity (e.g.,
often interrupting/intruding, blurring out answers) at levels inappropriate for one’s developmental stage (APA, 2013). According to The Diagnostic and Statistical Manual- Fourth Edition classification (DSM-IV; APA, 2000), there are three disorder subtypes: primarily inattentive, primarily hyperactive/impulsive, and combined (i.e., presenting with both inattention and hyperactivity/impulsivity). Among these, the inattentive subtype is the most prevalent, but patients with the combined subtype are referred most often for clinical services (Willcutt, 2012). Moreover, ADHD is often chronic. The disorder persists into adolescence in 50 to 80 percent of cases and into adulthood in 30 to 50 percent of cases (Barkley, Fischer, Edelbrock, & Smallish, 1991; Klein & Mannuzza, 1991; Klein et al., 2012; Weiss, 1993).

To receive a DSM-IV diagnosis of ADHD, one must also have functional impairment in academic, social, and/or occupational domains (APA, 2000). Indeed, youth with ADHD have a number of impairments, including negative academic outcomes (Langberg & Becker, 2012; Langberg, Becker, & Dvorsky, 2013), poor grades, low achievement test scores, high rates of grade retention, dropout, suspensions, and expulsions (DuPaul, 2003; Langberg et al., 2011). They also have social impairment (Bagwell, Molina, Pelham, & Hoza, 2001; Greene et al., 2001; Hoza, Gerdes, et al., 2005), including problematic peer relationships (Hoza, 2007; Landau, Milich, & Diener, 1998; Pelham & Fabiano, 2008), less popularity amongst peers (Elkins, Malone, Keyes, Iacono, & McGue, 2011), higher rates of peer rejection (Hoza, Mrug, et al., 2005), fewer friendships (Blachman & Hinshaw, 2002), and increased levels of social withdrawal and isolation (Hinshaw, 2002; Hodgens, Cole, & Boldizar, 2000). Moreover, individuals with ADHD have poorer family relations, early substance experimentation and abuse, and more driving accidents and speeding violations (Barkley, DuPaul, & McMurray, 1990; Barkley et al., 1991; Barkley, Guevremont, Anastopoulos, DuPaul, & Shelton, 1993; Biederman,
1992; Hinshaw, 1994). Many of these impairments, plus difficulties with relationships, marriage, employment, and finances are evident into adulthood (Barkley et al., 1990; Barkley et al., 1991; Barkley et al., 1993; Barkley, Murphy, & Kwasnik, 1996; Klein et al., 2012; Murphy & Barkley, 1996; Weiss, 1993).

Although ADHD is traditionally considered a disorder of “cognition,” it is also associated with emotional impairment, including notable emotion dysregulation and a wide range of psychiatric, behavioral, and other problems (Barkley, 2006). Approximately 45 to 84 percent of youth with ADHD meet criteria for comorbid oppositional defiant disorder (ODD) or conduct disorder (CD), and up to half of youth with ADHD have a comorbid anxiety or depressive disorder (Barkley, 2006). These comorbidities further complicate the clinical picture, associated impairment, treatment, and long-term outcome (Becker, Luebbe, & Langberg, 2012; Nigg, 2012). For instance, those with ADHD and aggression and/or conduct problems are more likely to have increased long-term risk of antisocial tendencies, substance use disorders, underemployment, divorce, and interpersonal conflict relative to those with ADHD alone (Nigg, 2012).

**ADHD Etiology.** To date, there are no definitive etiologies of ADHD, but there are a number of factors under investigation. Some have limited evidence, while others have stronger support in playing a role in underlying ADHD pathogenesis. Overall, however, the predominant etiological model of ADHD is that it is a pathophysiologically complex and heterogeneous disorder, resulting from several causal pathways. In particular, it likely emerges from multiple underlying developmental processes and is the product of the dynamic interplay between numerous individual risk factors (Sonuga-Barke & Halperin, 2010).

**Factors with mixed support.** There are many hypothesized etiological factors for ADHD
that have mixed evidence. Factors that may be associated with increased risk of developing ADHD but are not known to play causal roles include pre- and/or peri-natal factors, such as maternal smoking (Langley, Rice, van den Bree, & Thapar, 2005), maternal stress (Glover, 2011), maternal alcohol and substance use (Linnet et al., 2003), and low birth weight/prematurity (Bhutta, Cleves, Casey, Cradock, & Anand, 2002). Exposure to environmental toxins, such as organic pollutants, lead (Nigg, 2008), and polychlorinated biphenyls (Sagiv et al., 2010) have also been associated with increased risk of ADHD. Psychosocial adversity (Scahill et al., 1999) and some dietary factors (Nigg, Lewis, Edinger, & Falk, 2012) are similarly associated with ADHD, but there is not yet evidence that they play a causal role in the development of the disorder (Thapar, Cooper, Eyre, & Langley, 2013).

**Genetic Factors.** ADHD is an inherited disorder. First degree relatives of those with ADHD are two to eight times more likely than relatives of unaffected individuals to also have the disorder (Faraone et al., 2005). Twin studies lend support to the role of genetic factors in ADHD as well. Cross culturally, twin studies have shown heritability rates of ADHD to be as high as 71 to 90 percent (Faraone et al., 2005; Nikolas & Burt, 2010; Thapar, Holmes, Poulton, & Harrington, 1999), with adoption studies showing similar inheritance rates (Sprich, Biederman, Crawford, Mundy, & Faraone, 2000). It is also important to note, however, that genes do not act in isolation; risk for developing ADHD is produced by an interaction of both genetic and environmental processes (Nigg, Nikolas, & Burt, 2010).

**Neuroanatomical.** ADHD is also associated with structural and functional neurological deficits. Alterations in cortical development (e.g., global thinning of the cortex, particularly in anterior attention networks) have been illustrated in longitudinal studies, whereby degree of cortical thinning is related to the extent of impairment (Shaw et al., 2006). Neuroimaging studies
have also consistently shown alterations in frontal-striatal-thalamic circuitry in ADHD (e.g., deficits in response suppression, distractibility, working memory, organization, and planning), as well as frontal-cerebellar circuits (e.g., motor coordination deficits, problems with the timing and timeliness of behavior), and frontal-limbic circuits (e.g., emotional dyscontrol, motivation deficits, hyperactivity/impulsivity, and proneness to aggression; (Bush, 2011; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg & Casey, 2005; Sagvolden, Johansen, Aase, & Russell, 2005). It is important to note, however, that ADHD symptoms often appear before the development of the prefrontal cortex, and so it is difficult to assign a direct causal role of prefrontal deficits to ADHD symptomatology (e.g., Halperin & Schulz, 2006). It is hypothesized that symptoms may, instead, be due to deficits in non-cortical aspects of these circuits, whereas recovery of symptoms with age is associated with compensation of the prefrontal cortex as it develops and exacts more top-down control (Halperin & Schulz, 2006). In one study evaluating this theory, it was found that the degree of functional integration of the thalamus and prefrontal cortex, in particular, may parallel symptom remission (Clerkin et al., 2013). Other structural and functional abnormalities in ADHD include the anterior cingulate and temporal and posterior parietal cortices (Bush, 2011; Cherkasova & Hechtman, 2009). The emergence of diffusor tensor imaging (DTI) technology has allowed researchers to examine white matter integrity in ADHD. DTI studies have shown widespread problems throughout the brain in ADHD, but most consistently in the right anterior corona radiata, right forceps minor, bilateral internal capsule, and left cerebellum (Castellanos & Hyde, 2010; Konrad et al., 2010; Konrad & Eickhoff, 2010; Nagel et al., 2011; van Ewijk, Heslenfeld, Zwiers, Buitelaar, & Oosterlaan, 2012).

**Neurochemical.** There are also neurochemical anomalies in ADHD, particularly involving dopamine and norepinephrine (Wu, Xiao, Sun, Zou, & Zhu, 2012). This makes sense
given that dopamine is integral for information processing in the striatum and prefrontal cortex (Arnsten, 1998; Goldman-Rakic, 1999; Lou, Andresen, Steinberg, McLaughlin, & Friberg, 1998). Moreover, the noradrenergic system is important for regulating higher-order cortical functions associated with ADHD, such as attention, alertness, vigilance, and executive functions (Biederman, 1999). Research using rat models links a decrease in inhibitory dopaminergic activity and an increase in norepinephrine activity with the prefrontal cortex to ADHD pathogenesis (Russell, Allie, & Wiggins, 2000). Studies of patients with ADHD similarly show that symptoms may be a result of deficient dopamine-mediated reinforcement mechanisms (Sagvolden & Sergeant, 1998). For instance, evidence points to an increase in dopamine transporter density in individuals with ADHD has been found; this suggests that dopamine may be cleared from the synapse too quickly in individuals with ADHD (Dougherty et al., 1999). Moreover, a positive correlation between the expression of DAT1 alleles and hyperactive/impulsive symptoms has been shown (Waldman et al., 1998). In addition, noradrenergic drugs, such as desipramine and a2 adrenoceptor agonists, have been used to treat ADHD symptoms (Arnsten, 1998; Solanto, 1998), further suggesting catecholamine involvement.

**Neurocognitive.** There are also well documented neurocognitive impairments in ADHD. It is hypothesized that neurocognitive mechanisms may have an underlying etiological role in ADHD (Sonuga-Barke, Bitsakou, & Thompson, 2010). For instance, executive dysfunction is often a prominent feature in an ADHD neuropsychological profile (Geurts, Verte, Oosterlaan, Roeyers, & Sergeant, 2005; Loo et al., 2007; Martel, Nikolas, & Nigg, 2007; Riccio, Homack, Jarratt, & Wolfe, 2006; Scheres et al., 2004; Solanto et al., 2007; Wodka et al., 2008), evident in 30 to 50 percent of youth with ADHD (Biederman et al., 2004; Lambek et al., 2010, 2011; Loo
et al., 2007; Nigg & Casey, 2005; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). “Executive functioning” is the conscious control of thought and action needed for future-oriented and purposeful behavior (Zelazo, 1997) and involves a diverse set of cognitive processes. Executive functions documented to be impaired in ADHD include working memory, planning and organization, set shifting, and response inhibition (Barkley, 1997; Mariani & Barkley, 1997; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Others propose an impairment in regulating allocation of energy and effort in ADHD (Sergeant, 2005). High reaction time variability is also consistently seen in ADHD (Andreou et al., 2007; Bidwell, Willcutt, Defries, & Pennington, 2007; Rommelse et al., 2008).

In summary, ADHD is a common and often chronic disorder, associated with short- and long-term impairments in both cognitive and emotional domains. As such, it is of utmost importance for researchers and clinicians to understand underlying mechanisms associated with impairing deficits in ADHD, in order to more appropriately target treatment strategies. Exact etiological mechanisms underlying ADHD are unclear, but there is strong evidence that the disorder is associated with genetic factors, as well as alterations in neuroanatomical and neurochemical systems. There are also notable neurocognitive deficits in ADHD, such as executive dysfunction. Given the complex, multi-faceted nature of ADHD, it is likely that these factors work in concert, rather than in isolation, to contribute to symptomology and associated deficits and impairment (Castellanos et al., 2006; Coghill, Nigg, Rothenberger, Sonuga-Barke, & Tannock, 2005; Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). In this context, the current study will focus on the contribution of working memory, a particular aspect of executive functioning, to ADHD symptomology and emotional functioning.

Working Memory and ADHD
Introduction to Working Memory. “Executive function” has typically been studied as an umbrella term in the ADHD literature, comprised of a number of different cognitive processes. However, working memory, in particular, has been gaining prominence as a significant executive function deficit in ADHD (e.g., Rapport 2008; Kofler 2014). Working memory is a limited capacity system that temporarily stores, maintains, and manipulates information for use in guiding behavior (Baddeley, 1998, 2003). Importantly, working memory allows for one to maintain information for immediate recall, while simultaneously processing or utilizing other information (Baddeley & Larsen, 2007). One of the leading models of working memory is described by Baddeley (1974). In the traditional model (1974), he posits three main and dissociable components of working memory, including the central executive and its two “slave systems:” the visuospatial sketchpad and the phonological loop. A fourth component, the episodic buffer, was added in a later model (Baddeley, 2000). The Baddeley model is widely used across both pediatric and adult populations (Alloway, 2004; Baddeley, 1996). It has also been used across the literature to study working memory deficits within the context of ADHD (Alderson, Rapport, Hudec, Sarver, & Kofler, 2010; Brocki, Randall, Bohlin, & Kerns, 2008; Kofler et al., 2014; Kofler, Rapport, Bolden, Sarver, & Raiker, 2010; Nyman et al., 2010).

The Baddeley Model. The visuospatial sketchpad is important for maintenance and manipulation of visuospatial information (Gathercole, 1994). There are two theorized components of the visuospatial sketchpad: a visual storage aspect and a more dynamic retrieval and rehearsal process. The phonological loop is the most widely studied aspect of the model and is posited to underlie the maintenance of auditory-verbal information. It is comprised of a phonological store, which holds memory traces for a few seconds before they fade, and an articulatory rehearsal process, paralleling subvocal speech (Logie, 1995). The central executive
is considered the “most important but least understood” (Baddeley, 2003) component of working memory and is described as a general, top-down “control system” (Baddeley & Hitch, 1974). In the original model proposed by Baddeley, the central executive was otherwise largely undefined and thought to have a general processing ability, which subserved complex issues that exceeded the capacity of the slave systems. The first real theory attempting to model the central executive paralleled the attentional control model of Norman and Shallice (1980). This model described two systems that guide behavior: one related to habits/patterns/schemas and is relatively automatic, and the second, the supervisory activating system, which intervenes when routine is deviated. Today, the central executive is still not well understood but described largely as a regulatory system that underlies a variety of executive functions, such as the coordination of the slave systems, filtering relevant information, suppression of distracting information, dual task coordination, and shifting (Baddeley, 1996; Fournier-Vicente, Largauderie, & Gaonac’h, 2008; Kofler et al., 2010).

The original model proposed by Baddeley, however, had a number of limitations. For instance, the model of the phonological loop could not explain the phenomenon whereby one can increase (beyond the span of the phonological loop) their immediate recall when extracting meaning into material. More specifically, it was unclear how information was integrated from one’s semantic store of knowledge. There is also evidence for integration of information from the phonological loop and visuospatial sketchpad, as, for instance, visual similarity has an impact on verbal recall. Thus a “binding problem” was created-- the question of how information from a range of independent sensory routes is bound together to generate the perception of a coherent picture. As such, a fourth component to the Baddeley model was conceptualized, the episodic
buffer, which is a mechanism that fuses information from the subsystems to form a temporary, intact representation (Baddeley, 2003).

Imaging and lesion studies have supported the Baddeley model and localized the original three domains. For instance, the phonological loop has been found to be associated with the left temporoparietal regions and Brodmann areas [BA] 40, 6, and 44. The visuospatial sketchpad is associated with right BAs 40, 6, 47, and 19, and the central executive is associated with the left frontopolar cortex/BA 10 to left middle frontal/BA 46 (Awh, 1996; De Renzi & Nichelli, 1975; Della Sala, 2002; Hanley, Young, & Pearson, 1991; Jonides et al., 1993; Jonides, 1996; Jonides, 1997; Kosslyn et al., 1993; Paulesu, Frith, & Frackowiak, 1993; Singer, 1999; Smith & Jonides, 1997; Smith, Jonides, & Koepppe, 1996; Vallar, Di Betta, & Silveri, 1997; Van der Linden et al., 1999; Warrington, Logue, & Pratt, 1971). This reinforces the idea that these functions are independent, dissociable, and contribute differentially to various abilities.

**Implications.** The appropriate development of working memory is critical, as intact working memory is essential for daily functioning and the ability to successfully engage in complex tasks, such as learning, comprehension, reasoning, and planning (Baddeley, 2003; Baddeley & Larsen, 2007). Moreover, poor working memory is associated with learning and language disabilities in children (de Jong, 1998; McLean & Hitch, 1999), impaired academic and scholastic achievement (Gathercole, 2004; Gathercole, Tiffany, Briscoe, Thorn, & team, 2005), internalizing and externalizing problems (Brunnekreef et al., 2007), and social deficits (Alloway et al., 2005).

In summary, working memory is a limited capacity system that temporarily stores, maintains, and manipulates information for use in guiding behavior. According to the traditional Baddeley Model (1974), there are three separate and dissociable components of working
memory, including the visuospatial sketchpad, phonological loop, and central executive. Intact working memory is essential for everyday functioning across a number of domains, and so the development of effective working memory ability is critical.

**Working Memory Deficits in ADHD.** Although working memory deficits are not unique to ADHD, it is well documented that, as a group, individuals with ADHD have significant working memory impairments. Deficits are found across all three of the traditional Baddeley components of working memory relative to typically developing youth (Bolden, Rapport, Raiker, Sarver, & Kofler, 2012; Dovis, Van der Oord, Wiers, & Prins, 2013; Karatekin, 2004; Kasper, Alderson, & Hudec, 2012; Kofler et al., 2010; Mariani & Barkley, 1997; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Rapport et al., 2008; Re, De Franchis, & Cornoldi, 2010; Rosenthal, Riccio, Gsanger, & Jarratt, 2006; Willcutt et al., 2005), with the largest differences found in central executive functioning (Rapport et al., 2008) and, among the two “slave systems,” in visuospatial sketchpad ability (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005). Working memory impairments are evident across ADHD presentations (Cockcroft, 2011) and cultures (Li-Jie, 2006). They are seen across developmental stages, from children as young as preschool and kindergarten (Re et al., 2010; Thorell & Wahlstedt, 2006) to adults with ADHD (Alderson, Hudec, Patros, & Kasper, 2013; Alderson, J.Kasper, Hudec, & Patros, 2013; Finke et al., 2011). Moreover, both girls and boys with ADHD show a similar working memory profile, which is not the case for other executive functions (O’Brien, Dowell, Mostofsky, Denckla, & Mahone, 2010).

**Neuropsychological Evidence.** There is an abundance of neuropsychological evidence supporting the idea that impaired working memory may be inherent to ADHD and underlie core symptoms of the disorder, due to shared underlying neural circuitry. In particular, prefrontal-
basal ganglia circuitry involved in ADHD has been noted to subserve attentional control of memory storage. The globus pallidus filters irrelevant information before storage, which serves to reduce load and enhance working memory capacity by making it more efficient (McNab & Klingberg, 2008). This circuit is also important in the actualization of a chosen plan/action when representations are in working memory. As such, malfunction in these areas in early childhood, which is seen in ADHD, may also result in the storage of irrelevant information, which, in turn, overloads working memory capacity. Event-related potential [ERP] (Myatchin, Lemiere, Danckaerts, & Lagae, 2012) and functional connectivity (Fassbender et al., 2011; Mills et al., 2012; Sheridan, Hinshaw, & D'Esposito, 2007) studies specifically show that these neural pathways subserving working memory are impaired in ADHD (Mills et al., 2012). Given the important role of dopamine in ADHD, it is also not surprising that dopamine is present in prefrontal-basal ganglia circuitry and is important for enhancing memory, strengthening inputs from the basal ganglia to the frontal cortex, and for disinhibition of motor responses (Frank, Loughry, & O'Reilly, 2001). This model is also consistent with the developmental course of ADHD. The frontal-basal ganglia circuits begin to function at a young age (typically before the age of 2), with some immature coordination of attention and working memory processes (Thurber, Sheehan, & Roberts, 2011). Neuroimaging studies have shown that, as the prefrontal cortex develops throughout childhood and adolescence, there is a corresponding emergence of executive processes, which become more refined with greater activation of basal ganglia and prefrontal regions subserving these abilities (Adleman et al., 2002; Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Tamm, Menon, & Reiss, 2002). In fact, as prefrontal functions become more advanced throughout development, they can ultimately compensate for deficient filtering via the globus pallidus, an idea alluded to above. Indeed, there appears to be a
parallel part in the frontal regions of this circuit, the left inferior frontal gyrus, which is important for filtering relevant information (Thompson-Schill et al., 2002). As discussed above, the extent of this frontal compensation may underlie individual differences in natural attenuation of ADHD with age (Halperin & Schulz, 2006; Thurber, Sheehan, & Roberts, 2011).

There is also genetic and neurochemical evidence of an association between working memory deficits and ADHD. Arias-Vasquez et al. (2011) found that CDH13, a gene linked to risk for developing ADHD, is also associated with working memory functioning. Although the evidence is currently mixed, there may be further genetic support for the role of working memory in ADHD. For instance, the functional polymorphism in the COMT gene leads to a fourfold difference in dopamine-catabolic efficiency and predicts working memory performance (Egan et al., 2001). The COMT polymorphism has also been associated with an ADHD diagnosis in one study (Eisenberg et al., 1999), although not in others (Barr et al., 1999; Hawi, Millar, Daly, Fitzgerald, & Gill, 2000; Tahir et al., 2000). As such, more work needs to be done on this topic. Lastly, it is not surprising that working memory is impaired in ADHD, given the prominent role of catecholamines in the pathophysiology of ADHD. As alluded to above, dopaminergic modulation of prefrontal neurons is important in working memory performance (Sawaguchi, 2001).

Implications: The Functional Working Memory Model of ADHD. Given a clear association between working memory deficits and ADHD, recent literature has gone a step further to propose a Functional Working Memory Model of ADHD, which posits that core deficits in the disorder truly stem from working memory impairment (Kofler et al., 2014; Rapport, 2001; Rapport et al., 2008). Working memory is essential for the organization and control of behavior and the ability to appropriately interact with the environment. One needs
effective working memory processes in order to generate and maintain representations of input stimuli, search memory for matches, and then access and maintain appropriate behavioral responses to the target stimulus (Thurber et al., 2011). As such, deficits in these functions would naturally lead to disorganized and unregulated behavior. Poor working memory also contributes to “stimulus seeking”—in other words, because representations in deficient working memory are not sufficiently maintained, behavioral output will work to compensate to increase the frequency at which stimuli are exposed to working memory. Taken together, a working memory deficit would theoretically lead to disorganized behavior and an “overly-active”/stimulation seeking behavioral profile, as seen in ADHD (Thurber et al., 2011).

There has been a recent upsurge of empirical literature supporting the idea that working memory impairment underlies core deficits in ADHD, including inattention (Alderson et al., 2010; Burgess et al., 2010; Kofler et al., 2010), distractibility, (Re et al., 2010), hyperactivity (Raiker, Rapport, Kofler, & Sarver, 2012; Rapport et al., 2009), and impulsivity/behavioral disinhibition (Alderson et al., 2010; Raiker et al., 2012). Recent research suggests that reaction time variability evident in individuals with ADHD stems from working memory deficits (Kofler et al., 2014). Moreover, working memory deficits in youth with ADHD are predictive of problems with goal setting skills (Nyman et al., 2010), social impairment (Kofler et al., 2011), and academic and school difficulties (Alloway, Gathercole, & Elliott, 2010; Rogers, Hwang, Toplak, Weiss, & Tannock, 2011). More specifically, inattention, hyperactivity, impulsivity/behavioral disinhibition, reaction time variability, and problematic goal setting skills in youth with ADHD are related specifically to central executive dysfunction, as measured by objective neuropsychological tasks tapping into Baddeley working memory functions (Alderson et al., 2010; Brocki et al., 2008; Kofler et al., 2014; Kofler et al., 2010; Nyman et al., 2010).
In summary, working memory deficits in ADHD are quite common. The relationship between ADHD and working memory deficits is not surprising, given overlapping neuroanatomical, genetic, and neurochemical substrates between ADHD and working memory functions. Furthermore, empirical research validates the Functional Working Memory Model of ADHD, which suggests that core symptoms of the disorder, along with areas of associated impairment, stem from working memory deficits, particularly in the central executive. This literature suggests that working memory impairment may be a primary deficit in ADHD.

**Emotion Regulation and ADHD**

**Introduction to Emotion Regulation.** Although literature on the Functional Working Memory Model of ADHD has been increasing rapidly within recent years, it has not yet extended to include the contribution of working memory impairment to emotional deficits in ADHD, such as emotion dysregulation (Barkley, 1997). Although there is not one widely accepted definition of the term “emotion regulation,” the construct typically includes the initiation, maintenance, and modulation of internal feeling states, physiological responses, and behavioral concomitants of emotions in service of accomplishing social or personal goals (Eisenberg, 1992; Eisenberg & Spinrad, 2004; Thompson, 1994). Thus, emotion regulation is not merely a passive suppression and/or controlling of emotion, but it is a dynamic and active process, essential for adaptive functioning in a constantly changing environment (Morelen, Zeman, Perry-Parrish, & Anderson, 2012). Shields and Cicchetti (1997) further conceptualize emotion regulation as the maintenance of appropriate arousal such that one can optimally interact with his or her environment. Others define emotion regulation via the processes and/or mechanisms used to regulate the experience and expression of emotion, including those that occur without conscious awareness or those that involve intrinsic and extrinsic properties (Gross, 1998; Thompson, 1994). In turn, “emotion
dysregulation” typically refers to 1) deficits in self-regulating the physiological responses caused by emotions, 2) problems inhibiting inappropriate behaviors that are made in response to positive or negative emotions, 3) difficulty refocusing attention away from strong emotions, and 4) disorganization of coordinated behavior in response to emotional arousal (Surman et al., 2011). These difficulties can be a result of both increased bottom-up emotional reactivity and/or reduced top-down emotional regulation skills (e.g., Banaschewski et al., 2012).

Like executive functions in general, including working memory, emotion regulation ability improves with age; this commonality is likely due to the development of shared, frontal-mediated regulatory processes involved in the top-down control of emotions alluded to above (Wolfe & Bell, 2007). The ability to regulate one’s emotions is a developed skill. Infants and toddlers rely on adults to regulate their emotional experience and expression (de Veld, Riksen-Walraven, & de Weerth, 2012). As early as the age of three or four years, children begin to show rudimentary control over their expression of negative emotion (Cole, 1986). Moreover, at this age, an understanding develops that displayed emotion does not have to match internal emotional experience (Zeman & Garber, 1996). Between six and ten years of age, the shift occurs from the use of more behavioral strategies (e.g., gaze aversion, suppression) to more cognitive ones (e.g., mental distraction, reappraisal; Meerum Terwogt, 1995). Emotion regulation ability continues to improve with age, and older children report less expression of anger and sadness relative to their younger peers, especially in social settings (Zeman & Garber, 1996). By the age of ten, emotion regulation strategies are fairly developed and effective in typically developing youth (Saarni, 1999; Zeman, Cassano, Perry-Parrish, & Stegall, 2006). As children become adolescents, the use of emotion regulation behaviors increase even more, likely due to the growing complexity of
their social environment (Zeman & Shipman, 1997) and to neuromaturation (e.g., for a review see Steinberg, 2005).

**Implications.** Intact development of emotion regulation ability is essential for optimal functioning, as effective skills are critical for prosocial behavior, positive relationships, and school readiness in youth (Denham, 2002; Underwood, 1992). Emotion dysregulation in youth is associated with increased levels of overall psychological maladjustment and internalizing and externalizing problems (Bowie, 2010; Eisenberg, 2007; Olson, 2009). Emotion dysregulation is also associated with a host of social dysfunction (Eisenberg et al., 1993; Maszk P, 1999), including poor social status (Maszk et al., 1999), social skills problems, less prosocial behavior (Eisenberg, Fabes, Guthrie, & Reiser, 2000), and increased levels of peer victimization and rejection (Hanish et al., 2004; Maszk P, 1999; Rosen, Milich, & Harris, 2012).

In summary, emotion regulation typically refers to the initiation, maintenance, and modulation of internal feelings states and their physiological and behavioral concomitants. This regulation is accomplished via a number of bottom-up and top-down strategies that become increasingly sophisticated and effective throughout development in typically developing youth. Top-down emotion regulation, in particular, improves with age due to the maturation of frontal mediated executive processes—overlapping with those involved in working memory. Like working memory ability, the appropriate development of intact emotion regulation skills is essential for positive outcomes in youth, given the important role it has across a number of domains.

**Emotion Regulation and ADHD**

Just as there are notable working memory deficits in ADHD, there is increasing evidence that many youth with ADHD have prominent emotion regulation deficits and are “emotionally
explosive” (Landau et al., 1998). This concept is not new; one of the first theorists of ADHD (Still, 1902, quoted in Barkley, 1997) described the disorder as being associated with increased emotional symptoms such as irritability, hostility, excitability, and an overall emotional hyper-responsiveness toward others. In fact, many argue for the inclusion of emotion dysregulation as a core feature of ADHD (Barkley, 1997; Mitchell, Robertson, Anastopulous, Nelson-Gray, & Kollins, 2012). Emotion dysregulation has been reported across studies utilizing a diverse range of methods, including observational paradigms, parental-, self-, and teacher-completed questionnaires, and physiological measures (Anastopoulos et al., 2011; Biederman et al., 2012; Carroll et al., 2006; Jensen & Rosen, 2004; Scime & Norvilitis, 2006). Observational studies using frustration-inducing paradigms indicate that youth with ADHD have heightened emotional reactions and greater levels of frustration compared with typically developing peers (Maedgen & Carlson, 2000; Melnick & Hinshaw, 2000; Walcott & Landau, 2004). Parental reports find that youth with ADHD are more emotionally reactive than their peers (Jensen & Rosen, 2004) and have greater levels of negative emotions, coupled with a reduced ability to self-regulate these emotions (Berlin, Bohlin, Nyberg, & Janols, 2004; Braaten & Rosén, 2000). Moreover, in a longitudinal study, parent-rated emotion dysregulation in youth with ADHD tended to persist over time (Biederman et al., 2012). Using self-report measures, both children and adults with ADHD rate themselves as having more difficulty in regulating their own emotions compared with their peers (Braaten, 1997; Scime & Norvilitis, 2006). Although there are few studies that utilized teacher reports of emotion dysregulation in youth with ADHD, evidence indicates that students with ADHD exhibit increased levels of emotion dysregulation in the classroom relative to their peers (e.g., Sobanski, et al. 2010). Physiological studies assessing biomarkers for emotion dysregulation via respiratory sinus arrhythmia (RSA) are scarce using pure ADHD
samples, and the results of these studies are variable, and at times, null (Beauchaine 2001; Crowell 2006). However, one study found blunted autonomic nervous system response (e.g., poor regulatory control) at baseline and across induction of positive and negative emotion in youth with ADHD and low prosocial behavior (but without a comorbid disruptive behavior disorder). This study also found increased parasympathetic activation (e.g., increased regulatory demand) during induction of positive emotion in those with ADHD and typical social behavior (Musser 2013). As such, both groups showed altered autonomic functioning suggestive of emotion dysregulation, but in distinct forms.

**Correlates of emotion dysregulation in ADHD.** The mechanisms driving emotion dysregulation in ADHD are currently unclear, as few studies have rigorously investigated predictive factors for emotion regulation/dysregulation in ADHD. Given the complex nature of emotion regulation and its dyscontrol, however, it is likely that one factor alone cannot thoroughly account for emotion regulation deficits in clinical populations, and multiple factors likely contribute to emotional problems in ADHD. That being said, there are currently several potential underlying factors influencing the relationship between emotion dysregulation and ADHD including: 1. Core symptoms of ADHD (Martel, 2009); 2. Comorbid externalizing disorders (Sobanski et al., 2010); 3: Familial/social environmental risk factors (Sobanski et al., 2010; Melnick & Hinshaw, 2000); 4. Temperament/personality risk factors (e.g., see Martel, 2009); and 5. Neurobiological risk factors (e.g., Barkley, 1997).

**Core symptoms of ADHD.** It is argued that emotion regulation deficits may stem directly from the two core symptom domains of ADHD (Martel, 2009). Theoretically, symptoms of inattention are associated with a lack of cognitive self-regulatory skills, which can directly contribute to decreased top-down control of emotions (Martel, 2009; Martel & Nigg, 2006).
Moreover, symptoms of hyperactivity/impulsivity in ADHD are related to excessive positive and negative emotionality (Martel, 2009). Taken together, the combination of poor top-down regulatory skills and the expression of excessive emotionality will result in emotion dysregulation. When this theory was tested empirically, Sobanski et al. (2010) found that hyperactive/impulsive symptoms explained eight percent of the variance of emotion dysregulation in ADHD, and inattentive symptoms explained less than one percent of the variance. However, hyperactive/impulsive symptoms did not predict emotion dysregulation after controlling for ODD and general “emotional problems” as rated by the Strengths and Difficulties Questionnaire (Goodman, 1997). According to these results, there is little evidence for the predictive value of ADHD core symptoms as an underlying factor driving the relationship between emotion dysregulation and ADHD above and beyond comorbid ODD and other emotional difficulties. Using the same data-set, Banaschewski et al. (2012) reported that severity of ADHD symptoms, as a unified construct, was associated with emotion dysregulation. However, the authors did not control for ODD symptoms in this particular study.

**Comorbid externalizing disorders.** In line with the results above, it has also been hypothesized that emotion regulation deficits in ADHD are merely artifacts of comorbid externalizing disorders, such that emotion dysregulation is non-specific to ADHD itself. Sobanski et al. (2010) reported that comorbid ODD explained 25 percent of the variance of emotion regulation in ADHD, while CD uniquely explained 15 percent. As such, these findings suggest that ODD and CD, in particular, may each play an important role in the underlying mechanisms of emotion dysregulation in ADHD; however, together they explained only a fraction of the variance and were neither necessary nor sufficient for the existence of emotional problems in ADHD. Consistent with this, while Sobanski et al. (2010) found that siblings of
youth with ADHD and high emotion dysregulation were at increased risk for emotion regulation problems as well, emotion dysregulation in probands was not associated with an increased risk for ADHD or ODD in their siblings. As such, this data suggests that comorbid externalizing disorders may contribute to emotion dysregulation seen in ADHD but are clearly just a piece of the larger picture. It is also important to note the inherent challenge in researching this topic, given the overlap of the construct of “emotion dysregulation” with diagnostic criteria for ODD (e.g., “often loses temper,” and “touchy or easily annoyed” are typically features of both emotion dysregulation and ODD). Thus, these two constructs are often difficult to disentangle.

**Familial/Social environmental risk factors.** There are also familial risk factors that may contribute to emotion dysregulation in youth with ADHD. Children and adolescents with ADHD and high emotion dysregulation tend to have siblings with increased levels of emotional dyscontrol as well, as mentioned above (Sobanski et al. 2010; Surman et al., 2011). However, it is unclear whether this association is a result of a shared familial environment or from genetic factors (see below). Nonetheless, it is likely that these factors work in concert. Moreover, there is evidence from healthy populations that parents contribute to their child’s acquisition of emotion-regulation skills via coaching, instructing, and modeling how to respond in an emotional situation (Miller & Sperry, 1987) and that other parenting (particularly maternal) styles are related to the child’s emotion regulation skills (Chang, Schwartz, Dodge, & McBride-Chang, 2003; Stack, Serbin, Enns, Ruttle, & Barrieau, 2010). There is some evidence of a relationship between parental factors and emotion regulation in the context of ADHD as well (Melnick & Hinshaw, 2000). In a mixed sample of boys with ADHD and healthy controls, maternal negativity was significantly associated with the child’s ability to regulate his emotions. Paternal advice giving and overall self-regulation were associated with positive coping strategies for the
child. Maternal and paternal anxiety and nervousness were associated with less emotion regulation behaviors in the children. The way in which familial factors influence the child’s ability to regulate his/her emotions has also been studied from a different perspective. Maternal pre-pregnancy weight predicted both inattention and emotional lability such that higher maternal body mass index (BMI) was associated with increased inattention and emotion dysregulation in offspring (Rodriguez, 2010). The exact underlying mechanism between this relationship (and subsequent causal role) remains unclear, though there are several hypotheses regarding hormonal factors in overweight woman that may play key roles in neural development and later cognitive and emotional functioning (e.g., see Rodriguez, 2010 for a review). Indeed, the connection between adverse maternal characteristics during pregnancy and negative neuropsychological outcome has been associated with other conditions, including gestational diabetes (Nomura et al., 2012; Rizzo TA et al., 1995; Sells, Robinson, Brown, & Knopp, 1994). Lastly, as mentioned previously, youth with ADHD have well documented social problems (Pelham, 1982). Sobanski et al. (2010) found that social problems explained seven percent of the variance of emotion dysregulation in ADHD.

**Temperament/Personality risk factors.** There is also evidence that emotion dysregulation may stem from temperament/personality traits inherent to individuals with ADHD. For instance, youth with ADHD tend to have higher negative and positive emotionality and neuroticism relative to their typically developing peers, with some documented extraversion and low agreeableness (Parker, Majeski, & Collin, 2004; White, 1999). Coupled with this, youth with ADHD tend to have poor regulation, conceptualized in this literature as effortful control or conscientiousness (Eisenberg et al., 2001; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005; Parker et al., 2004). Again, this theory has a similar theme (discussed above), regarding
excessive emotionality coupled with inferior control mechanisms, though the underlying nature of these deficits are hypothesized to stem from temperament/personality factors (For review see Martel, 2009).

**Neurobiological risk factors**

*Neurological.* There appears to be shared biological vulnerability between ADHD and emotional symptoms. The three primary neural circuits involved in ADHD (frontal-striatal, frontal-cerebellar, frontal-limbic) are thought to contribute to the regulation of emotions, including the control of emotionally surged behavior and the initiation and maintenance of internal emotional processes (Barkley & Fischer, 2010). In particular, Posner et al. (2011) found increased medial prefrontal cortex (mPFC) reactivity in response to emotional stimuli in youth with ADHD compared to their peers, and this activation was associated with severity of inattentive and hyperactive symptoms. The anterior cingulate cortex, another important area in ADHD and in general regulatory processes, is also related to emotional control (Bush, Valera, & Seidman, 2005; Ochsner & Gross, 2005; Paloyelli, Mehta, Kuntsi, & Asherson, 2007; Valera, Faraone, Murray, & Seidman, 2007). Lastly, it is notable that emotion dysregulation responds well to pharmacologic treatment of ADHD with stimulant and non-stimulant medication, suggesting shared neuroanatomical and/or neurochemical processes (Kratochvil et al., 2007; Skirrow, 2009). Consistent with this, Posner (2011) found normalized mPFC activation in response to emotional stimuli with stimulant medication. Given notable monoamine involvement in mood regulation, it is not surprising if there are overlapping neurochemical systems in both ADHD and emotion regulation.

*Genetic.* There is also a role for genetics when considering emotion regulation problems in ADHD. As mentioned above, youth with ADHD and severe emotion dysregulation are more
likely to have a sibling with high emotion dysregulation relative to those with ADHD and no emotional issues, suggesting a possible genetic component (Sobanski et al., 2010; Surman et al., 2011). Similarly, youth with ADHD and mild emotion regulation problems more often have siblings with mild emotion dysregulation versus those with more severe deficits. However, ADHD and severe emotion dysregulation did not cosegregate in ADHD probands (Sobanski et al., 2010). Velders et al. (2012) identified that the fat and obesity transcript gene (FTO) minor allele at rs9939609, a gene important in BMI, food intake, and eating behavior in older children, is a possible protective factor against both ADHD and emotional lability in children; this further suggests a genetic relationship between these constructs. It is important to reiterate, however, that genes do not act in isolation, and it is therefore difficult to disentangle the impact of shared environmental factors relative to genetics alone.

**Neuropsychological.** Barkley’s Executive Function Theory of ADHD (1997) posits that ADHD, as a disorder of executive dysfunction, is inextricably a disorder of emotion dysregulation as well. Barkley argues that prominent executive dysfunction in ADHD includes deficient self-regulatory processes (e.g., inattention) and inhibitory control processes (e.g., hyperactivity). In addition to inattention and hyperactivity, emotion regulation deficits would stem naturally from deficient self-regulatory processes (e.g., problems regulating emotion) and inhibitory control processes (e.g., problems inhibiting inappropriate emotional behavior) as well. As such, according to this theory, emotion dysregulation is tied to deficient executive control processes and is thus inherent to ADHD. When studied empirically, modest associations between behavioral disinhibition and emotion dysregulation in ADHD have been reported (Melnick & Hinshaw, 2000; Walcott & Landau, 2004).
Banaschewski et al. (2012) further investigated the association between executive functions and other neuropsychological variables (e.g., processing speed, response variability, choice impulsivity) and emotion dysregulation in ADHD, as well as the influence of energetic and/or motivational factors. Results suggested that neuropsychological variables as a whole were associated with emotion dysregulation in ADHD. However, further analyses revealed that this relationship was mediated by ADHD symptoms, and so the influence of neuropsychological variables on emotion dysregulation was indirect (Banaschewski et al., 2012). However, the authors discuss limitations to their findings, including the use of unaffected siblings and participants with subthreshold ADHD symptoms in their sample.

**Implications.** Compared with children with ADHD and low emotion dysregulation, children with ADHD and high emotion dysregulation have poorer social outcome and increased problem behaviors (Biederman et al., 2012; Maedgen & Carlson, 2000; Melnick & Hinshaw, 2000). In addition, ADHD and emotional dysregulation is associated with more severe core ADHD symptoms (particularly hyperactive/impulsive symptoms) and greater likelihood of comorbid psychopathology, including oppositional defiant disorder, conduct disorder, substance use, affective disorders, somatization, and perfectionism (Biederman et al., 2012; Sobanski et al., 2010). Youth with ADHD and emotional problems also miss more school days and utilize health-care resources more frequently relative to youth with ADHD and no emotional symptoms (Classi, Milton, Ward, Sarsour, & Johnston, 2012). Moreover, emotion dysregulation in ADHD has been found to fully mediate depressive symptoms found in this population (Seymour et al., 2012). Emotion dysregulation also mediated functional impairment, psychiatric comorbidity, and treatment services in ADHD youth relative to peers without ADHD (Anastopoulou et al., 2011). Not surprisingly, emotion dysregulation impairs quality of life in children and adolescents with
ADHD (Wehmeier, Schacht, & Barkley, 2010). Emotion dysregulation in ADHD also persists and may increase into adolescence (Biederman et al., 2012). Similarly, it can persist into adulthood and predict (relative to adults with ADHD but without emotion dysregulation) more severe impairment in occupational, educational, and marriage domains, as well as a more extensive criminal history (Barkley, 2010a, 2010b; Mitchell et al., 2012). Interestingly, emotion dysregulation has predictive power for these outcomes above and beyond the core symptoms of ADHD (Barkley & Murphy, 2010).

In conclusion, there are notable emotion regulation deficits in ADHD, which, albeit historical documentation, remain relatively understudied. Although many theories exist as to the underlying nature of these emotional deficits in ADHD, little empirical work has been done to rigorously test and compare these models and/or their utilities. Clearly, more research needs to be done on this topic in order to investigate and compare competing theories.

**Working Memory, Emotion Regulation, and ADHD**

As discussed above, clear links exist between working memory and ADHD, as well as between emotion dysregulation and ADHD. In addition to these relationships, there is also evidence that working memory and emotion regulation are related as well, spanning theoretical, basic science, and clinical research.

For instance, Baddeley (2013) recently discussed emotion regulation within the context of his working memory model. He theorizes that working memory, the central executive in particular, is integral for appropriate emotional functioning (Baddeley, 2013). As stated in his theory, executive processes act on emotions that are kept on-line via amplification (e.g., rumination) or attenuation (e.g., reappraisal). Thus, from a theoretical perspective, working memory appears to be integral in the regulation of emotions.
In addition, there are shared neuroanatomical and neurophysiological substrates between emotional control processes and working memory, including reliance on frontoparietal (e.g., DLPFC, inferior parietal, ACC) circuits (Schweizer, Hampshire, & Dalgleish, 2011). In fact, this shared neurological circuitry has led some to believe that working memory training in healthy populations could have transfer effects and improve emotion regulation, particularly when emotional stimuli are used during the training (Schweizer, Grahn, Hampshire, Mobbs, & Dalgleish, 2013; Schweizer et al., 2011). After 20 days of working memory training, Schweizer et al. (2013) found increased efficiency of this frontoparietal network in healthy adults, as well as improvement in ratings of emotion regulation ability. Improvement in emotion regulation was associated with increased activity in the frontoparietal network and other regions (e.g., subgenial anterior cingulate cortex) related to emotional control (Schweizer et al., 2013).

Empirical evidence from clinical investigations further support a connection between working memory and emotion regulation in healthy and patient populations spanning the lifespan. For instance, Wolfe and Bell (2007) found a positive association between working memory and emotion regulation in typically developing youth. In healthy adult populations, Stout and Rokke (2010) reported that working memory deficits predicted higher levels of emotion dysregulation, emotional distress, anxiety, and rumination. Greater working memory capacity in healthy individuals is also associated with utilization of spontaneous emotion regulation strategies in response to negative feedback, including increased self-enhancement (Schmeichel & Demaree, 2010), suppression of emotion (Schmeichel, Volokhov, & Demaree, 2008), and decreased negative affect following an undesirable event (Schmeichel & Demaree, 2010). Reappraisal ability has also been linked to working memory capacity in healthy populations (McRae, Jacobs, Ray, John, & Gross, 2012). In contradistinction, those low in
working memory ability appraise stimuli more emotionally and experience and express more emotion after negative feedback, relative to those with strong working memory ability (Schmeichel, Volokhov, & Demaree, 2008; Schmeichel & Demaree, 2010). Moreover, it was found that poor working memory storage and filtering ability interact to predict higher levels of emotional distress (e.g., depressive symptoms, rumination, state anxiety). However, there was little evidence for the direct impact of storage capacity alone on emotional experience (Stout & Rokke 2010).

Research in patient populations largely supports an association between working memory and emotion regulation as well. The inability to regulate the content of one’s working memory can lead individuals to engage in a cycle of negative thinking, as alluded to above. Individuals with depression have difficulty inhibiting negative thoughts from entering and remaining in working memory (Joormann & Gotlib, 2010). This leads to increased rehearsal and rumination of negative affect—factors important in the development and maintenance of depression (Nolen-Hoeksema, 1991). The inability to prevent negative thoughts from entering working memory also disrupts one’s ability to engage in appropriate emotion regulation strategies in this population, such as reappraisal and recall of mood incongruent information (Joormann & Gotlib, 2010). However, Gyurak et al. (2009) found no association between working memory deficits and emotion dysregulation in a range of populations with executive dysfunction, including patients with frontotemporal lobar degeneration and Alzheimer’s disease, as well as in healthy controls. The authors, however, comment that null findings were surprising and may be due to their small sample size. Given these mixed results, additional research on this topic is clearly necessary.

With regard to the relationship between working memory deficits and emotion
dysregulation in the context of ADHD, Barkley’s Executive Function model of ADHD, as reviewed above, posits that emotion dysregulation is an integral part of ADHD, due to deficient executive control processes shared with other core symptoms of the disorder. In particular, Barkley (1997) stresses that behavioral disinhibition in ADHD contributes to this emotional dyscontrol. However, Rapport’s Functional Working Memory Model of ADHD (Rapport, 2001; Rapport et al., 2008) states that working memory deficits underlie the behavioral disinhibition evident in ADHD. As such, it would be critical to extend these lines of research and examine whether emotion regulation deficits in ADHD are actually related to working memory deficits, and not behavioral disinhibition, per se. While Banaschewski (2012) started to investigate this question and included working memory (as measured by digit span backwards) as part of an aggregated index of “executive functions,” sample limitations were noted above. Moreover, only one aspect of working memory (e.g., phonological loop) was measured, whereas deficits in visuospatial elements of working memory appear to be more impacted in ADHD. To the author’s knowledge, there are no existing studies on the contribution of deficits in individual components of working memory on emotion dysregulation in ADHD. Because each component is dissociable and contributes uniquely to working memory ability and, subsequently, to working memory deficits in ADHD, measuring working memory as a unified construct and/or with a single measure may obscure true findings.

In conclusion, there is growing literature on the positive relationship between working memory and emotion regulation. Substantial literature suggests that working memory deficits are central to ADHD and that children with ADHD often have difficulty regulating their emotions. However, to the author’s knowledge, no studies have assessed whether deficits in the various components of working memory may be related to emotion dysregulation in children with
ADHD. Although traditionally deemed separate, there is increasing evidence for extensive connections between cognitive and emotional processes, and the development of both cognition and emotion are intricately tied (Bell & Wolfe, 2004). As such, to understand how these crucial cognitive and emotional skills are related in a neurodevelopmental disorder represents an exciting area of research and one that would greatly expand our understanding of this heterogeneous disorder. From a clinical neuropsychological perspective, it is important to understand the connections between cognitive and emotional aspects of a patient’s profile when formulating a case and making diagnostic conclusions, having the understanding that seemingly disparate symptoms may indeed stem from similar sources. Moreover, gaining a clear understanding of the factors underlying emotion dysregulation in ADHD could shed light on potential mechanisms of therapeutic action. In particular, elucidating the cognitive substrates of emotional problems in ADHD could add to the literature on the evidence-base for cognitive remediation in the treatment of ADHD.

**Treatment Implications**

Emotion dysregulation in ADHD has been treated in a variety of ways. As stated above, stimulant and non-stimulant pharmacotherapy improves emotion regulation ability in ADHD (Posner et al., 2011; Rosler et al., 2010; Sobanski et al., 2012), which is likely due to the alteration of shared underlying neural circuitry (Posner et al., 2011; Williams et al., 2008). Psychosocial treatments targeting parent-child relationship factors, social skills, and/or comorbid externalizing symptoms have also been shown to improve emotion regulation skills in this population, including parent training (Herbert, Harvey, Roberts, Wichowski, & Lugo-Candelas, 2013; Webster-Stratton, Reid, & Beauchaine, 2011) and child-centered play therapy (Ray, Schottelkorb, & Tsai, 2007).
Indeed, given that there are many different factors contributing to emotion dysregulation in ADHD (e.g., biological, psychosocial), there may be distinct, preferential treatment approaches amongst individual patients. Given a potential relationship between working memory, emotion regulation, and ADHD, working memory training may play an important role in the treatment of emotional problems in ADHD as well, particularly for individuals with significant working memory deficits. As stated above, Schweizer (2013) found that emotion regulation ability was improved in healthy adults after working memory training. Given evidence for this treatment effect in healthy adults, it would be essential to study this within an ADHD population as well. The degree to which findings are expected to generalize to ADHD and youth, in particular, remains unclear though theoretical (e.g., Baddeley, 2013) evidence are encouraging.

Specifically, Cogmed Working Memory Training (CWMT; www.cogmed.com) has gained interest as a cognitive remediation tool with a potentially important role in the treatment of ADHD (Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010; Gray et al., 2012; Green et al., 2012; Holmes et al., 2010; Klingberg et al., 2005; Klingberg, Forssberg, & Westerberg, 2002). CWMT is a computerized training program designed to improve working memory by increasing working memory capacity over a five week training period. This is accomplished via targeting the storage and storage plus processing/manipulation of phonological loop, visuospatial sketchpad, and central executive aspects of working memory. The specific therapeutic component of CWMT focuses on improving working memory through the use of a game-like interface where trials are titrated to the capacity of the individual using an adaptive, staircase design that adjusts the difficulty of the program on a trial-by-trial basis. That is, correct trials are followed by successive trials with heightened working memory demands, whereas incorrect trials
result in subsequent trials with diminished working memory load. In addition, several components of CWMT focus on supporting the user’s engagement to the CWMT intervention. Specifically, contingent reinforcement is integrated within the program (e.g., earning small rewards for successful completion of a training-week). Moreover, each individual’s training is supervised by a training aide (typically a parent or guardian when CWMT is implemented at home) and a certified CWMT coach, who is able to track closely (via online access) each individual’s performance. During CWMT, the training aide is responsible for supporting the user through reinforcing on-task behavior, effort, and completion of CWMT by providing praise and encouragement. The CWMT coach supports the training aide and the user by providing them with a detailed training review and efforts to problem-solve obstacles (e.g., motivational, logistical) to adherence.

Several randomized clinical trials of CWMT have been conducted in children and adolescents with ADHD over the past several years (Beck et al., 2010; Chacko, Bedard, et al., 2013; Gray et al., 2012; Green et al., 2012; Klingberg et al., 2005). Results of randomized clinical trials suggest that CWMT may increase some aspects of working memory in youth with ADHD. However, when rigorously studied, it appears that CWMT does not have generalizable effects for improvement in ADHD symptoms and academic achievement (Chacko et al., 2013). The role of working memory training in this population on emotional domains, however, has not yet been studied and would expand our understanding of the role of cognitive remediation, particularly working memory training, in the treatment of ADHD.

In conclusion, there is increasing evidence that working memory deficits may underlie core symptoms of ADHD and associated impairments, lending support to the Functional Working Memory Model of ADHD. In particular, this body of literature has frequently
investigated these deficits using the traditional Baddeley Model and has found impairments across all three Baddeley components in ADHD. However, despite growing literature on this topic, there remains limited research on the role of working memory deficits in emotion dysregulation in this population, even though youth with ADHD and emotion dysregulation have significantly worse outcomes relative to peers with ADHD alone. Indeed, emotion regulation deficits in ADHD are relatively understudied; while there are many factors that have some empirical and/or theoretical bases for underlying emotional deficits in ADHD (e.g., comorbid ODD, familial/social factors), the literature on the Functional Working Memory Model of ADHD has not yet expanded to include the question as to whether deficits in the Baddeley components of working memory can account for emotional problems in this population as well. It is important to note, however, that given the complex nature of emotional functioning in youth, it is unlikely that a single factor alone underlies emotional problems in ADHD youth. Rather, it is likely that working memory deficits, in addition to other psychosocial factors, contribute to emotion dysregulation in this population. Having a better understanding of the contributory factors of this dysregulation could help inform treatment strategies. For instance, pharmacological treatment of ADHD helps improve emotional functioning, likely as a result of neurochemical contributory factors. There is also evidence that psychosocial treatments help to improve emotional functioning in ADHD, likely due to changes in familial/social contributors. However, if emotional problems also stem from neurocognitive deficits, then cognitive remediation may also play an important role in treating emotional symptoms in this population.

**The Current Study**

The first aim of this research was to investigate whether working memory deficits mediated the relationship between emotion dysregulation and ADHD. A variable is thought to
act as a mediator when it can account for the relation between a predictor variable and the outcome (Baron & Kenny, 1986). Because working memory deficits have been shown to underlie a host of symptoms and related impairment in ADHD (Rapport, 2001; Rapport et al., 2008) and because working memory deficits are related to emotional dyscontrol in other populations (e.g., Stout & Rokke, 2010; Schmeichel & Demaree, 2010; Schmeichel, Volokhov, & Demaree, 2008; Van Dillen & Koole, 2007; McRae, Jacobs, Ray, John, & Gross, 2012; Joormann & Gotlib, 2010), it was hypothesized that:

1. Working memory impairment would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.

**Aim 1A**

A sub-aim (Aim 1A) investigated this relationship more precisely by examining whether the separate Baddeley components of working memory differentially mediated the relationship between ADHD symptom severity and degree of emotion dysregulation, relative to using a more general composite measure. Working memory components included the visuospatial sketchpad, the phonological loop, and the central executive. In response to Baddeley’s (2013) theoretical model, it was hypothesized that:

1. Central executive deficits would most strongly mediate the relationship between ADHD symptom severity and degree of emotion dysregulation, as compared to phonological loop and visuospatial sketchpad deficits. Although visuospatial working memory deficits are more common in ADHD relative to phonological deficits, the relative importance of visuospatial central executive functions versus phonological central executive functions in the mediation of ADHD symptom severity and degree of emotion dysregulation was exploratory.

**Aim 1B**
A second sub-aim (Aim 1B) examined whether other factors that are thought to impact emotional functioning in these youth, such as ODD symptom severity, inattention (uniquely), hyperactivity/impulsivity (uniquely), negative parenting behaviors (e.g., poor monitoring/supervision, low parental involvement, lack of positive parenting practices, inconsistent discipline, use of corporeal punishment), parenting stress, and social skills deficits mediate the relationship between ADHD symptom severity and degree of emotion dysregulation. Because “emotion dysregulation” is a multifaceted and complex construct, it is likely that several factors work together to contribute to emotional problems in youth with ADHD and that working memory deficits would mediate this relationship, along with several other variables. Utilization of a multiple mediation model allowed for the simultaneous investigation of each variable as a possible mediator individually, while controlling for the effect of the other proposed mediators. It also allowed for the direct comparison of the strength of each mediator (see below). Although there is a paucity of literature on this topic, there is some theoretical and/or empirical evidence that the factors listed above (e.g., ODD, inattention, hyperactivity/impulsivity, negative parenting behaviors and emotions, social skills deficits) may be associated with emotion dysregulation in ADHD and/or in other pediatric populations (e.g., Banaschewski et al., 2012; Melnick & Hinshaw, 2000; Sobanski et al., 2010). Taken together, Aim 1B further investigated how these factors were related to emotion regulation deficits in youth with ADHD, in addition to working memory deficits.

Based on past empirical literature (Banaschewski et al., 2012; Melnick & Hinshaw, 2000; Sobanski et al., 2010) it was hypothesized that:

7. ODD symptom severity would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.
8. Inattentive symptoms would not mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.

9. Hyperactive/impulsive symptoms would not mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.

10. Negative parenting behaviors, including lack of involvement, lack of positive parenting, poor monitoring/supervision, use of inconsistent discipline, and use of corporeal punishment would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation. Because each of these behaviors was analyzed individually, the relative strength of each separate negative parenting behavior in the mediation of ADHD symptom severity and degree of emotion dysregulation was exploratory.

11. Parental stress would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.

12. Social skills deficits would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation.

An examination of the relative importance of each mediator was exploratory. Moreover, separate constructs were used for parent- and teacher-rated ADHD and ODD symptoms in order to utilize cross-informant data and to understand these relationships across settings. Hypotheses regarding the differences between parent- and teacher-rated constructs across aims were exploratory in nature, as the use of teacher-rated data when studying emotion dysregulation in ADHD has been limited.

**Aim 2**

Given recent evidence that cognitive remediation (particularly CWMT) may increase
aspects of working memory in youth with ADHD (e.g., Klingberg, Forssberg, & Westerberg, 2002) and that working memory training may improve emotion regulation ability in healthy adults (Schweizer, 2013), the second aim of this research was to investigate the effects of working memory remediation on emotion dysregulation among youth with ADHD. It was hypothesized that:

1. In the CWMT active group relative to control group, improvement in working memory (e.g., total working memory change score) would be associated with improvement in emotion regulation (e.g., emotion dysregulation change score) from pre-to-post working memory training. Moreover, with regard to the Baddeley components of working memory, in line with the hypothesis above, it was hypothesized that change in central executive working memory ability would be most strongly related to change in emotion regulation ability.
Method

Participants

A total of 83 participants were included in Aim 1. 13 participants from Aim 1 were not used in Aim 2 because they did not start or complete the intervention or were lost to follow-up. There were no significant differences between the drop-out group and the group that remained in the intervention in baseline working memory ability and severity of emotion dysregulation, ADHD and ODD symptoms, as well as between basic demographic factors (e.g., age, sex, race, ethnicity, ADHD subtype, medication status). For Aim 2, a total of 77 participants were included (n = 42 for the active group, and n = 35 for the placebo group). Six individuals were included in Aim 2, but not Aim 1, due to missing data. Youth and their families were recruited through community advertisements for a larger clinical trial assessing the benefit of combined and sequenced effects of CWMT and behavioral parent training for youth with ADHD (Title: Combined cognitive remediation and behavioral intervention for ADHD; [http://clinicaltrials.gov/ct2/show/NCT01137318](http://clinicaltrials.gov/ct2/show/NCT01137318)). Inclusion criteria included: 1) youth between the ages of 7-11 years; 2) a consensus-based DSM-IV diagnosis of ADHD based on parent and teacher ratings on the Disruptive Behavior Disorder Rating Scale (DBD; Pelham, Gnangy, Greenslade, & Milich, 1992), Impairment Rating Scale (Fabiano et al., 2006), and a semi-structured interview with the parent using the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version [K-SADS-PL] (Kaufman, Birmaher, Brent, Rao, & Ryan, 1996); 3) fluency in English (parent and child), and 4) internet access at home. Youth were excluded if 1) they met criteria for severe mental illness (i.e., psychosis, bipolar, major depressive disorder) or autism spectrum disorder; 2) the youth or parent presented with emergency psychiatric needs that required immediate services (e.g., suicidal or
homicidal intent), and 3) the child had an estimated Full Scale Intelligence Quotient (FSIQ) below 80 on two subtests of the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999). Tables 1 and 7, below, include more detailed demographic information regarding participants included in aim 1 and 2, respectively.

**Measures**

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

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

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

**Wechsler Abbreviated Scale of Intelligence** (WASI: Wechsler, 1999). The WASI is a brief and reliable screening measure of intellectual abilities for individuals 6-89 years. The WASI has excellent psychometric properties with an average reliability coefficient of 0.98 (for FSIQ) and test-retest reliability of 0.92 (for FSIQ). For participants who have had an intellectual assessment within the past six months, the summary scores were requested. Those who had not completed an intelligence test were administered the WASI under the supervision of a clinical neuropsychologist.

**Outcome Measure (used in Aim 1 and Aim 2)**

*Parent report of emotion regulation.* Emotion dysregulation was measured using the Behavior Rating Inventory of Executive Functioning [BRIEF]: Parent Form (Gioia, Isquith, Guy, & Kenworthy, 2000). The BRIEF is an 86 item measure that asks parents to rate a variety of behavioral functions on a four-point likert scale (i.e., Never, Sometimes, Often, Always). In particular, the emotional control clinical scale was used to detect the child’s ability to modulate emotional responses appropriately. This scale includes 10 items, such as “overreacts to small problems, has explosive, angry outbursts, becomes tearful easily, mood changes frequently, and reacts more strongly to situations than other children.” The sum of individual items of this subscale was used to create a composite score, with higher scores indicating more severe
emotion dysregulation. This measure is widely used in this population in both research and clinical settings and was chosen due to its excellent psychometric properties (e.g., test re-test reliability= .79, alpha=.89, high inter-rater agreement). Individual items also had little overlap with DSM-IV criteria for ODD, thereby limiting possible overlap between these constructs. The BRIEF is also particularly useful due to its ecological validity and “real world” anchors.

**Predictors.**

**Working memory (Aim 1 and Aim 2).** The Automated Working Memory Assessment (Alloway, 2007) was used to objectively measure working memory ability. The AWMA is a computer-based assessment of working memory skills, with a user-friendly interface and fully automated administration and scoring. It consists of phonological loop, visuospatial sketchpad, and central executive memory tasks which tap the temporary storage and manipulation of information. The AWMA is suitable for use with individuals aged between four and 22 years. Test reliability of the AWMA has been measured on 128 individuals (mean = 10.4 years, SD = five years) with four weeks separating the two successive testing administrations. There was a close relationship between the individual’s performance at the first and the second time of testing, which indicates that there was very little change in the scores at the two testing points (r’s ranged from .69 to .90). Validity of the AWMA was established by comparing it with performance on the WISC-IV Working Memory Index (Alloway, Gathercole, Kirkwood, & Elliot, 2008). Results indicate that the AWMA has good diagnostic validity, as evidenced by the high classification accuracy (91%) of the digit span subtest of the WISC-IV. The short form measure of the AWMA (four subtests) was completed to assess phonological loop (Digit Recall) and phonological aspects of central executive (Listening Recall), as well as visuospatial sketchpad (Dot Matrix) and visuospatial aspects of the central executive (Spatial Recall). In
particular, phonological loop ability was measured using an automated digit span task, which asked the respondent to repeat a random sequence of numbers, exactly as they were presented. The span of digits increases with successive trials (Digit Recall). Phonological aspects of the central executive was measured with a task manipulating auditory information (Listening Recall). In this task, participants heard a sentence and then said whether it was “true” or “false.” They were then asked to recall the last word of the sentence. An increasing number of sentences were presented in which participants had to say “true” or “false” for each and then recall the last word for each sentence, in the order presented. Visuospatial sketchpad ability was measured using an automated task in which a stimulus (“O”) appeared on a grid in different locations. Participants were instructed to recall the location at which the stimulus appeared on the grid. The span of stimuli used increased, and participants were asked to recall the location the stimulus appeared in, in the order that it appeared (Dot Matrix). Visuospatial aspects of the central executive were measured in a task that manipulated visual information. Participants were shown two images that were either identical or mirror images of one another and were asked to judge whether they were the “same” or “opposite.” One of the images had a target stimulus (“O”) on one of its corners and participants were further asked to recall the location of the stimulus after judging if it was the “same” or “opposite.” An increasing span was used such that participants were asked to judge “same or opposite” for a number of shapes, and then recall where the target stimulus was for each set, in the same ordered that it appeared (Spatial Recall; Alloway, 2007). Standard Scores for each of the subtests were generated by the AWMA and used as outcome measures. With regard to measuring the separate components of the Baddeley Model, the visuospatial sketchpad was measured by Dot Matrix, and the phonological loop was assessed via
Digit Recall. The central executive was measured by Spatial Recall (visuospatial component) and Listening Recall (phonological component), similar to past studies (Alloway, 2008).

**Parent and teacher report of ADHD and ODD symptom severity (Aim 1).** Symptoms were measured using the Disruptive Behavior Disorders Rating Scale (DBD): Parent and Teacher Forms (Pelham, Gnagy, Greenslade, & Milch, 1992). The DBD is a 45-item measure that asks parents and teachers to rate symptoms of ADHD, ODD, and CD on a four-point scale (i.e., Not at all, Just a little, Pretty Much, or Very Much), with higher scores indicating a greater frequency of problems. The DBD evidenced internal consistency (Cronbach’s alpha = .82-.85 for parent ratings and .91-.96 for teacher ratings). Test-retest reliability from pre-to-post treatment ranged from .49-.61 for parent ratings. Inter-rater reliability ranged from .14-.26 in parent and teacher reports, respectively. Concurrent reliability with the Diagnostic Interview Schedule for Children (DISC) ranged from .38-.62 (Pelham, Fabiano, & Massetti, 2005). The average score of individual items for the inattentive symptoms and for the hyperactive-impulsive symptoms were calculated separately and used as dimensional outcomes measure for both parent and teacher ratings, respectively. The average score was also taken across all ADHD symptoms for both parent and teacher ratings, respectively, to make an average total ADHD symptom severity construct.

**Parent ratings of parental behaviors (Aim 1).** The Alabama Parenting Questionnaire (APQ) was used to assess parental ratings of specific parenting behaviors (Shelton, Frick, & J, 1996). It is a 42-item measure of parenting that asks parents to rate different domains of parenting on a five-point scale ranging from one (Never) to five (Always). The APQ can be divided into six scales: Involvement (e.g., “you help your child with his/her homework”), Positive Parenting (e.g., “you praise your child if he/she behaves well”), Poor
Monitoring/Supervision (e.g., “your child fails to leave a note or let you know where he/she is going), Inconsistent Discipline (e.g., “you threaten to punish your child and then do not actually punish him/her”), and Corporal Punishment (e.g., “you spank your child with your hand when he/she has done something wrong). The parent APQ scales evinced internal consistency (Cronbach’s alpha=.45-.80 in a clinic sample) and good test-retest reliability (r=.66-.89) in a sample of combined clinic and volunteer participants (Shelton, Frick, & J, 1996) Raw scores for each individual domain were used in the analyses.

**Parent ratings of parental stress (Aim 1).** Parenting Stress Index–Short Form (PSI-SF) was used to measure parental stress (Abidin, 1995). The PSI-SF is a direct derivative of the full-length test and consists of a 36-item self-scoring questionnaire/profile. It yields a Total Stress score from 3 scales: Parental Distress (e.g., I don’t enjoy things as I used to; I feel trapped in my responsibilities as a parent), Parent-Child Dysfunctional Interaction (e.g., I expected to have closer and warmer feelings for my child than I do and this bothers me; sometimes my child does things that bother me just to be mean; sometimes I feel like my child doesn’t like me or want to be close to me), and Difficult Child (e.g., my child appears to cry or fuss more often than other children; my child’s eating or sleeping schedule was much harder to establish than expected; my child makes more demands on me than most children). The alpha coefficients are 0.91 for Total Stress, 0.87 for Parental Distress, 0.80 for Parent-Child Dysfunctional Interaction, and 0.85 for Difficult Child (Abidin, 1995). The raw score for Total Stress was used in the analyses.

**Social skills (Aim 1).** Social skills were assessed via the Behavioral Assessment System for Children – 2: Parent and Teacher forms (Reynolds & Kamphaus, 2004). The BASC-2 is a well-standardized, multidimensional approach to evaluating the behavior of children. In particular, the average T-score of social skills by both parents and teachers from the social skills
subscale was used to assess social functioning. Select items from this subscale include, “compliments others, volunteers to help with things, shows interest in others’ ideas, politely asks for help, says, ‘please’ and ‘thank you.’” The alpha coefficients for the social skills subscale for teacher and parent forms are .92 and .87, respectively, based on a normative non-clinical sample. Alpha coefficients in an ADHD sample are .91 (teacher) and .85 (parent). Test re-test reliability for the subscale is .86 (teacher) and .84 (parent).

**Intervention Conditions:** In Aim 2, working memory (as measured by the AWMA) and emotion dysregulation (as measured by the parent-rated BRIEF) were measured before and after the intervention for both CWMT active and control conditions.

**Cogmed Working Memory Training Active (CWMT; Klingberg, Forssberg, & Westerberg, 2002).** CWMT Active is a computerized training program that targets the maintenance and manipulation of verbal and nonverbal working memory information. This is accomplished via training which takes place in approximately 30-45 minute increments over five days per week (25 training-days total). CWMT Active trials are titrated to the capacity of the individual using an adaptive staircase design that adjusts the difficulty of the program on a trial by-trial basis. Each individual’s training is supervised by a training aide (typically a parent or guardian) and a Cogmed Qualified Coach (i.e., an individual who successfully completed the Cogmed Professional Training and whose qualification is current and in good standing). The coach is able to closely track (via online access) each individual’s performance and provide support to the family through weekly coaching interactions (by phone). CWMT is currently being disseminated in routine clinical practice (Chacko, Feirsen, et al., 2013) and has been studied in ADHD populations (Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010; Gray et al., 2012; Green et al., 2012; Holmes et al., 2010; Klingberg et al., 2005; Klingberg et al.,
CWMT has also been studied and used in other pediatric populations, including children with Down Syndrome (Bennett et al., 2013), “special needs” (Dahlin, 2011), intellectual disability (Söderqvist et al., 2012), very low birth weight (Grunewaldt et al., 2013; Løhaugen), cancer (Hardy et al., 2013), and cochlear implants (Kronenberger et al., 2011). CWMT has also been studied in “at-risk” (Roughan & Hadwin, 2011) and healthy pediatric populations (Bergman-Nutley et al., 2011; Thorell et al., 2009). The evidence-base for CWMT as a treatment for ADHD is currently under investigation (Chacko et al., 2013).

**CWMT Placebo.** The CWMT Placebo condition includes a low-level, non-scaffolded (“placebo”) working memory training program that was identical to CWMT Active with respect to the types of training games utilized and the number of training trials per session (i.e., 90 trials). Unlike the active condition, difficulty level is not titrated according to each user’s performance. As with CWMT Active, parents in the CWMT Placebo served as training aides, and each family was supported by a coach who utilized comparable support procedures (e.g., as used in Klingberg et al., 2005).

**Procedure**

All study procedures were approved by the University’s Institutional Review Board.

**Baseline Assessment**

All assessments took place in a university based research lab, most frequently after school hours in the early evening (e.g., 4 PM) or throughout the day on the weekends. The baseline assessment occurred approximately two-to-four weeks prior to the start of treatment.

**Parent.** At the study intake, parents and youth were debriefed on the study’s procedures and general aims. Following parental consent and youth assent, a semi-structured interview (K-SADS-PL) was completed by a graduate student with the parents to ascertain psychiatric
diagnoses, including ADHD. All graduate students who conducted the K-SADS-PL received training and supervision on ADHD diagnostic assessments and administration of the measures. After completion of the interview, parents completed the measures described above, including the BRIEF (emotion regulation), DBD, (ADHD and ODD symptoms) APQ (negative parenting behaviors), PSI (parenting stress), and the BASC-2 (social skills). At the time of intake, the parents were also informed that their child would be randomly assigned to one of two computerized working memory training programs, and they were educated on the theoretical differences between the Active and Placebo versions. No information was provided regarding the relative benefits of the two programs. Of note, after all study procedures were completed, all children who were randomized to the Placebo condition were afforded the opportunity to access and complete the Active version as well.

**Child.** While the parent completed the procedures described above, the child worked simultaneously with a second trained and supervised graduate student. The child’s assessment occurred within the university lab setting, in a separate room from his or her parents. At this time, two subtests of the WASI (i.e., to ascertain FSIQ for inclusion criteria) and the AWMA (i.e., to measure working memory ability) were administered to the child. Because this study occurred within the context of a larger study (described above), the child also completed several academic measures (WRAT-4) and the AX-CPT, while wearing an actigraph, which were used in the larger study. The assessments were administered in a standardized order across participants, which included: the two subtest WASI, two subtests of the AWMA, WRAT-4, two subtests of the AWMA, and the AX-CPT. The four subtests of the AWMA were divided so as to minimize fatigue and potential frustration occurring as a result of the relatively challenging working memory tasks.
**Teacher.** A packet including the DBD and the BASC-2 were given to the parent at the baseline session in a self-addressed envelope to deliver to the child’s teacher. Teacher ratings of ADHD symptoms, ODD symptoms, and social skills were utilized for the current research.

**Treatment.** Participants who met study criteria were randomly assigned to treatment condition by a senior research staff (blind to youth profile) based on a random permutation calculator ([http://www.webcalculator.co.uk/statistics/permute3.htm](http://www.webcalculator.co.uk/statistics/permute3.htm)). Following randomization, research staff, who were all certified by Pearson as CWMT training coaches, were assigned cases and received an equal number of CWMT Active and CWMT Placebo cases. All families participated in a start-up session which introduced the basic features of CWMT, established a schedule for implementing the intervention, and scheduled the weekly coaching phone calls. In addition, all families and their coaches developed an individualized incentive system that focused on rewarding on-task behavior during training. This reward system, implemented for both conditions, augmented the standard CWMT incentive system (i.e., earning stickers) with contingent daily, weekly, and end-of treatment rewards that were selected by the child and agreed upon by the parent (e.g., picking a snack for lunch, dessert for dinner, extra television time, etc.). This simple enhancement was made to maximize compliance given the expected high rates of comorbid oppositional problems in the sample. All coaches completed a weekly fidelity and integrity questionnaire, developed specifically for this study to: (i) identify potential challenges to treatment compliance, (ii) establish an algorithm to titrate total training time, (iii) operationalize feedback during weekly coaching calls for all participants, and (iv) problem-solve challenges in performance and compliance. Treatment was supervised by senior staff who verified data and monitored coaching calls so that participants in both conditions received equal
support from their coaches throughout the training period. All training was conducted at home and was scheduled to be completed over a five-week period based on the family’s schedule.

**Post-Treatment Assessment**

As conducted in previous trials of CWMT (Klingberg et al., 2005), post-treatment assessments (i.e., the child was administered the AWMA) and rating scales (i.e., parent completed the DBD, BASC-2, BRIEF, PSI, and APQ; a packet was delivered to the teacher containing the BASC-2 and the DBD) were completed approximately three weeks after the final training day for each participant. All assessments were conducted by research staff who were blind to participant treatment randomization.

**Statistical Analyses**

**Aim 1 (1, 1A, 1B)**

Pearson product moment correlations were used to initially examine the relationships among the dependent variable (emotion dysregulation), proposed independent variables (parent-rated ADHD symptom severity and teacher-rated ADHD symptom severity), and all proposed mediators: working memory outcomes (total working memory composite score, phonological loop, visuospatial sketchpad, central executive), parent-rated ODD symptom severity, teacher-rated ODD symptom severity, parent-rated inattention, parent-rated hyperactivity/impulsivity, teacher-rated inattention, teacher-rated hyperactivity/impulsivity, negative parenting behaviors (e.g., poor involvement, lack of monitoring/supervision, decreased positive parenting practices, inconsistent discipline, use of corporeal punishment), parenting stress, and social skills deficits. For potential mediators, only those that were significantly correlated with emotion dysregulation at the p<.05 level were included in the analyses. Similarly, for potential independent variables
(parent-rated ADHD symptom severity, teacher-rated ADHD symptom severity), only those that were correlated at the p<.05 level with emotion dysregulation were used in the analyses.

In order to assess all of the factors included in Aim 1 and its sub-aims, it was proposed that separate multiple mediation models would be utilized in order to investigate the extent to which working memory (either as a composite measure or in its components), ODD symptom severity, inattention, hyperactivity/impulsivity, negative parenting behaviors, parental stress, and social skills deficits mediate the relationship between ADHD symptom severity and degree of emotion dysregulation, as well as their relative importance. Theoretically, one model would include the working memory composite measure as a mediator (Aim 1), while investigating and controlling for the other proposed mediators (Aim 1B). A separate multiple mediation model would include the individual components of working memory (Aim 1A), rather than the composite measure, while controlling for and investigating the other proposed mediators (Aim 1B).

There are several advantages to the utilization of a single multiple mediation model (with j variables) in place of separate simple mediation models, as outlined by Hayes (2013). First, testing the total indirect effect of X on Y (see Figure 1) is equivalent to conducting a regression analysis with several predictors, with the aim of investigating whether an overall effect exists. If a mediation effect is found, it suggests that the group of j variables mediates the effect of X on Y. Second, when using a multiple mediation model, one can determine the extent to which specific M variables mediate the X—Y effect, conditional on the presence of other mediators in the model. Third, one can determine the relative magnitude of the specific indirect effects associated with all mediators when using multiple mediators in one model. This would allow for the direct comparison of each competing theory regarding the underlying mechanism of emotion.
dysregulation in ADHD within one model. As Preacher and Hayes (2008) state, “theory comparison is good scientific practice.” Clearly, the factors involved in the emotional functioning of youth with ADHD are complex and numerous, and so a simple mediation model would likely not suffice. Moreover, by using one multiple mediation model, rather than many separate simple mediation models, it minimizes the number of tests necessary, thus reducing the risk of decision errors (Hayes, 2009). Lastly, the chance of parameter bias due to omitted variables is reduced when using multiple mediators (Preacher & Hayes, 2008). By contrast, when several simple mediation hypotheses are each tested with a simple mediator model, these separate models may have biased parameter estimates from omitted variables (Judd & Kenny, 1981).

![Multiple mediation model with j factors, adapted from Preacher & Hayes (2008).](image)

Figure 1. Multiple mediation model with j factors, adapted from Preacher & Hayes (2008).

Multiple mediation was tested using bootstrapping, a technique popularized recently (Preacher & Hayes, 2008), in order to assess the mediating effects of all of the variables together.
Bootstrapping does not assume multivariate normality, like other strategies (e.g., product-of-coefficients/Sobel test) and is thus the method of choice in small to moderate samples (Preacher & Hayes, 2008). It has been shown to be a more powerful alternative to using Baron and Kenny’s (1986) approach (Preacher & Hayes, 2004). This is due, at least in part, to its emphasis on the indirect effect, $ab$, instead of on individual pathways. Thus, whereas the causal-steps approach requires that $a$, $b$, and $c$ all be significant for mediation to be considered (see Figure 1 above), bootstrapping requires only that the indirect effect be significant (Preacher & Hayes, 2004; Preacher & Hayes, 2008). With regard to extending the Bootstrapping technique to a multiple mediation model, it has also been shown to be more powerful, with less type I error rates, than the multivariate product of coefficient strategy (Preacher & Hayes, 2008). Bootstrapping procedures can also identify the extent to which each of the factors individually contributed to the mediation.

**Aim 2**

Pearson product moment correlations were used to assess whether rehabilitation of working memory (i.e., change of working memory pre-to-post training) was associated with a change in emotion dysregulation (i.e. change of emotion dysregulation pre-to-post training) in the active and placebo groups separately. Fisher r-to-z transformations were conducted in order to assess significant differences among the associations between the intervention groups.
Results

Aim 1

Demographic information for participants in Aim 1 is presented in Table 1. Baseline descriptive statistics (e.g., means, SDs, and ranges) for all variables used in Aim 1 are presented in Table 2. Pearson product correlations of the independent variable (IV), dependent variable(s), and proposed mediators are presented in Tables 3, 4, and 5 respectively. Results from the multiple mediation analysis are presented in Table 6.

Aim 2

Demographic information for participants in Aim 2 is presented in Table 7. Descriptive statistics and effect sizes regarding change scores from pre-to-post CWMT are presented in Table 8. Pearson product correlations of working memory and emotion regulation change scores are presented in Table 9, as well as results of Fisher r-to-z transformations.
Table 1: Demographic Information for Aim 1 Sample (N=83)

<table>
<thead>
<tr>
<th>Age, Mean (SD) in years</th>
<th>8.40 (1.35)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, % Male</td>
<td>76</td>
</tr>
<tr>
<td>Full Scale IQ, Mean (SD)</td>
<td>105 (14.55)</td>
</tr>
<tr>
<td>Medicated for ADHD, %</td>
<td>27.7</td>
</tr>
<tr>
<td>Medicated with other pharmacological treatments influencing the central nervous system(^1), %</td>
<td>1.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Methylphenidate (MPH) Derivatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concerta</td>
</tr>
<tr>
<td>Focalin</td>
</tr>
<tr>
<td>Ritalin</td>
</tr>
<tr>
<td>Methylin</td>
</tr>
<tr>
<td>MPH unspecified</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Amphetamine Derivatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vyvanse</td>
</tr>
<tr>
<td>Adderall</td>
</tr>
<tr>
<td>Drexedrine Spansule (d-amphetamine sulfate)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Non-Stimulants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intuniv/Tenex (guanfacine)</td>
</tr>
<tr>
<td>Clonidine</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Polytherapy(^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N=3</td>
</tr>
</tbody>
</table>
| DSM-IV ADHD Subtype, % | Combined: 57.3  
Inattentive: 42.7  
Hyperactive/Impulsive: 0 |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Comorbid Conditions</td>
<td></td>
</tr>
<tr>
<td>Comorbid ODD(^2), %</td>
<td>41</td>
</tr>
<tr>
<td>Comorbid CD(^2), %</td>
<td>7.2</td>
</tr>
<tr>
<td>OCD, %</td>
<td>1.2</td>
</tr>
<tr>
<td>Panic Disorder with</td>
<td></td>
</tr>
<tr>
<td>Agoraphobia, %</td>
<td>1.2</td>
</tr>
<tr>
<td>Specific Phobia, %</td>
<td>1.2</td>
</tr>
<tr>
<td>Tourette Syndrome, %</td>
<td>1.2</td>
</tr>
</tbody>
</table>
| Race\(^3\), %          | Asian: 13.9  
Caucasian 45.8  
African American/Black 18.1  
“Multi-Cultural:” 19.4  
Other: 2.8 |
| Ethnicity, %           | Hispanic or Latino: 35.6  
Not Hispanic or Latino: 64.4 |
| Parent Marital Status, %| Married: 62.5  
Married but Separated: 5.6  
Divorced: 11.1  
Never Married/Single: 20.8 |
| Socioeconomic Index\(^4\), Mean (SD) | 57.26 (17.7) |
| Range                  | 22-97            |

Note: demographic data was not available for six participants

\(^1\) 1 participant was taking Ritalin and Methylin; 1 participant was taking MPH (unspecified) and Tenex; 1 participant was taking Ritalin and clonidine for ADHD and was also being treated with Lexapro and amantadine.

\(^2\) 2 participants met criteria for comorbid ODD and CD.

\(^3\) “Multi-cultural” was operationalized as identification with more than one racial background.

\(^4\) Socioeconomic Status (SES) was measured using the Nakao and Treas Socioeconomic Prestige Index (1994). Higher scores indicate higher SES. The higher value of mother or father was taken to represent the family SES at baseline.
Table 2: Means, Standard Deviations (SD), and Ranges for all variables used in the baseline sample (n=83)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Emotion Dysregulation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parent-rated BRIEF Emotional Control T-Score</td>
<td>60.39(^1) (11.99)</td>
<td>35-88</td>
</tr>
<tr>
<td><strong>Working Memory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AWMA Digit Recall Standard Score (phonological loop)</td>
<td>100.63 (12.91)</td>
<td>71-137</td>
</tr>
<tr>
<td>AWMA Listening Recall Standard Score (phonological aspect of the central executive)</td>
<td>97.94 (14.36)</td>
<td>66-132</td>
</tr>
<tr>
<td>AWMA Dot Matrix Standard Score (visuospatial sketchpad)</td>
<td>93.08 (13.92)</td>
<td>65-122</td>
</tr>
<tr>
<td>AWMA Spatial Recall Standard Score (visuospatial aspect of the central executive)</td>
<td>97.89 (13.87)</td>
<td>70-143</td>
</tr>
<tr>
<td>Total WM Average Standard Score</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Parent-Rated ADHD Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBD average raw score total symptoms</td>
<td>1.88(^2) (.572)</td>
<td>1-3</td>
</tr>
<tr>
<td>DBD Inattention average raw score</td>
<td>2.11(^2) (.54)</td>
<td>1-3</td>
</tr>
<tr>
<td>DBD Hyperactivity/Impulsivity average raw score</td>
<td>1.71(^2) (.6)</td>
<td>0-3</td>
</tr>
<tr>
<td><strong>Teacher-Rated ADHD Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBD average raw score total symptoms</td>
<td>1.53(^2) (.687)</td>
<td>0-3</td>
</tr>
<tr>
<td>DBD Inattention average raw score</td>
<td>1.7(^2) (.67)</td>
<td>0-3</td>
</tr>
<tr>
<td>DBD Hyperactivity/Impulsivity average raw score</td>
<td>1.28(^2) (.78)</td>
<td>0-3</td>
</tr>
<tr>
<td><strong>Parent-Rated ODD Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBD average raw score total symptoms</td>
<td>1.2(^2) (.793)</td>
<td>0-3</td>
</tr>
<tr>
<td><strong>Teacher-Rated ODD Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBD average raw score total symptoms</td>
<td>.73(^2) (.898)</td>
<td>0-3</td>
</tr>
<tr>
<td>Negative Parenting Behaviors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>APQ Use of Corporeal Punishment raw score</td>
<td>6.84&lt;sup&gt;3&lt;/sup&gt; (1.98)</td>
<td>3-12</td>
</tr>
<tr>
<td>APQ Poor Monitoring raw score</td>
<td>12.58&lt;sup&gt;4&lt;/sup&gt; (2.77)</td>
<td>10-22</td>
</tr>
<tr>
<td>APQ Parental Involvement raw score</td>
<td>38.7&lt;sup&gt;4&lt;/sup&gt; (4.710)</td>
<td>29-50</td>
</tr>
<tr>
<td>APQ Positive Parenting raw score</td>
<td>25.61&lt;sup&gt;5&lt;/sup&gt; (3.076)</td>
<td>19-30</td>
</tr>
<tr>
<td>APQ Inconsistent Discipline raw score</td>
<td>15.06&lt;sup&gt;5&lt;/sup&gt; (4.21)</td>
<td>6-26</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Parenting Stress</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>PSI Parenting Stress Composite raw score</td>
<td>89.89&lt;sup&gt;6&lt;/sup&gt; (19.94)</td>
<td>41-142</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Social Skills</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>BASC-2 Social Skills index average parent and teacher rated T-Score</td>
<td>42.08&lt;sup&gt;7&lt;/sup&gt; (6.48)</td>
<td>25-56</td>
</tr>
</tbody>
</table>

Note:
1= Higher scores indicate more dysregulation
2= Measured on a four-point likert scale ranging from 0 (not at all)-3 (very much)
3= Scores range from 3 (never)-15 (always)
4= Scores range from 10 (never)-50 (always)
5= Scores range from 6 (never)-30 (always)
6= Scores range from 36-180; clinical cut-off for this index is 86.
7= Lower scores indicate more deficits
Table 3: Pearson Product Correlations of emotion dysregulation and all hypothesized independent variables (IV’s) and mediators. (N=83)

<table>
<thead>
<tr>
<th>Parent BRIEF Emotional Control (Emotion Dysregulation)</th>
<th>AWMA Digit Recall (phonological loop)</th>
<th>AWMA Listening Matrix (phonological aspect of central executive)</th>
<th>AWMA Dot Matrix (visuospatial sketchpad)</th>
<th>AWMA Spatial Recall (visuospatial aspect of central executive)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Correlation</td>
<td>-.226*</td>
<td>.009</td>
<td>.103</td>
<td>-.097</td>
</tr>
<tr>
<td>Sig.</td>
<td>.040</td>
<td>.939</td>
<td>.352</td>
<td>.382</td>
</tr>
<tr>
<td>AWMA Total WM</td>
<td>DBD Parent-Rated ADHD</td>
<td>DBD Teacher-Rated Inat</td>
<td>DBD Parent-Rated Hyp/Imp</td>
<td></td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>-.079</td>
<td>.233*</td>
<td>.250*</td>
<td>.21</td>
</tr>
<tr>
<td>Sig.</td>
<td>.478</td>
<td>.034</td>
<td>.02</td>
<td>.056</td>
</tr>
<tr>
<td>DBD Teacher-Rated ADHD</td>
<td>DBD Teacher-Rated Inat</td>
<td>DBD Teacher-Rated Hyp/Imp</td>
<td>DBD Parent-Rated ODD</td>
<td></td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>-.158</td>
<td>-1.08</td>
<td>-.072</td>
<td>.640**</td>
</tr>
<tr>
<td>Sig.</td>
<td>.153</td>
<td>.33</td>
<td>.52</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>DBD Teacher-Rated ODD</td>
<td>APQ Corp Punish (negative parenting behavior)</td>
<td>APQ Poor Monitor (negative parenting behavior)</td>
<td>APQ Positive Parenting (negative parenting behavior)</td>
<td></td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>.049</td>
<td>.131</td>
<td>-.013</td>
<td>.114</td>
</tr>
<tr>
<td>Sig.</td>
<td>.659</td>
<td>.237</td>
<td>.909</td>
<td>.304</td>
</tr>
<tr>
<td>APQ Parental Involv (negative parenting behavior)</td>
<td>APQ Inconsis Discipl (negative parenting behavior)</td>
<td>PSI Parenting Stress (negative parenting behavior)</td>
<td>BASC-2 Social Skills (negative parenting behavior)</td>
<td></td>
</tr>
</tbody>
</table>
As seen in Table 3, among the hypothesized mediators, emotion dysregulation was significantly correlated at the p<.05 level with phonological loop ability, parent-rated inattention, parent-rated ODD, inconsistent discipline, parenting stress, and social skills. Other variables did not meet minimum criterion for model entry and were therefore excluded as mediators in the model. Specifically, excluded mediators included the total working memory composite construct, visuospatial sketchpad and central executive components of working memory, as well as parent-rated hyperactivity/impulsivity, teacher-rated inattention, teacher-rated hyperactivity/impulsivity, teacher-rated ODD, use of corporeal punishment, poor monitoring/supervision, lack of positive parenting strategies, and lack of parental involvement. Among the hypothesized independent variables, parent-rated ADHD symptom severity was significantly correlated with emotion dysregulation. Teacher-rated ADHD symptom severity did not meet minimum criterion for model entry and was therefore excluded as an independent variable in the model.
Table 4: Pearson Product Correlations of parent-rated ADHD symptoms and all hypothesized mediators. (N=83)

<table>
<thead>
<tr>
<th></th>
<th>AWMA Digit Recall (phonological loop)</th>
<th>AWMA Listening Matrix (phonological aspect of central executive)</th>
<th>AWMA Dot Matrix (visuospatial sketchpad)</th>
<th>AWMA Spatial Recall (visuospatial aspect of central executive)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DBD Parent-Rated ADHD Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>-.112</td>
<td>-.058</td>
<td>.225*</td>
<td>-.011</td>
</tr>
<tr>
<td>Sig.</td>
<td>.314</td>
<td>.602</td>
<td>.041</td>
<td>.922</td>
</tr>
<tr>
<td>DBD Parent-rated Inat</td>
<td>AWMA Total WM</td>
<td>DBD Parent-rated Inat</td>
<td></td>
<td>DBD Teacher-Rated ADHD</td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>.025</td>
<td>.744**</td>
<td>.721**</td>
<td>.258*</td>
</tr>
<tr>
<td>Sig.</td>
<td>.824</td>
<td>&lt;.0001</td>
<td>&lt;.0001</td>
<td>.019</td>
</tr>
<tr>
<td>DBD Teacher- Rated Hyp/Imp</td>
<td>DBD Teacher- Rated Inat</td>
<td>DBD Parent- Rated Hyp/Imp</td>
<td></td>
<td>DBD Teacher Rated ODD</td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>.143</td>
<td>.191</td>
<td>.190</td>
<td>.080</td>
</tr>
<tr>
<td>Sig.</td>
<td>.199</td>
<td>.083</td>
<td>.086</td>
<td>.475</td>
</tr>
<tr>
<td>APQ Corp Punish (negative parenting behavior)</td>
<td>APQ Poor Monitor (negative parenting behavior)</td>
<td>APQ Positive Parenting (negative parenting behavior)</td>
<td>APQ Parental Involv (negative parenting behavior)</td>
<td></td>
</tr>
<tr>
<td>Pearson Correlation</td>
<td>.048</td>
<td>-.056</td>
<td>.029</td>
<td>-.045</td>
</tr>
<tr>
<td>Sig.</td>
<td>.669</td>
<td>.617</td>
<td>.796</td>
<td>.684</td>
</tr>
<tr>
<td>APQ Inconsist Discipl (negative parenting behavior)</td>
<td>PSI Parenting Stress</td>
<td>BASC-2 Social Skills</td>
<td>Parent-BRIEF emotional control (Emotion Dysregulation)</td>
<td></td>
</tr>
</tbody>
</table>
As seen in table 4, of the included mediators, parent-rated ADHD was significantly correlated with parent-rated inattention and social skills.

<table>
<thead>
<tr>
<th>Pearson Correlation</th>
<th></th>
<th></th>
<th>-.28**</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sig.</td>
<td>.102</td>
<td>.489</td>
<td>.01</td>
<td>.034</td>
</tr>
</tbody>
</table>

Note: *significant at the .05 level; ** significant at the .01 level
Table 5: Pearson Product Correlations of all Included Mediators. (N=83)

<table>
<thead>
<tr>
<th></th>
<th>AWMA Digit Recall (phonological loop)</th>
<th>DBD Parent-Rated ODD</th>
<th>BASC-2 Social Skills</th>
<th>DBD Parent-Rated Inattention</th>
<th>PSI Parenting Stress</th>
<th>APQ Inconsist. Parenting</th>
</tr>
</thead>
<tbody>
<tr>
<td>AWMA Digit Recall (phonological loop)</td>
<td>Pearson Correlation</td>
<td>1</td>
<td>.031</td>
<td>.378**</td>
<td>.179</td>
<td>.250*</td>
</tr>
<tr>
<td></td>
<td>Sig.</td>
<td></td>
<td>.773</td>
<td>&lt;.0001</td>
<td>.097</td>
<td>.019</td>
</tr>
<tr>
<td>DBD Parent-Rated ODD</td>
<td>Pearson Correlation</td>
<td>.031</td>
<td>1</td>
<td>-.176</td>
<td>.300**</td>
<td>.499**</td>
</tr>
<tr>
<td></td>
<td>Sig.</td>
<td></td>
<td>.773</td>
<td>.103</td>
<td>.005</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>BASC-2 Social Skills</td>
<td>Pearson Correlation</td>
<td>.378**</td>
<td>-.176</td>
<td>1</td>
<td>.077</td>
<td>.043</td>
</tr>
<tr>
<td></td>
<td>Sig.</td>
<td></td>
<td>&lt;.0001</td>
<td>.103</td>
<td>.479</td>
<td>.691</td>
</tr>
<tr>
<td>DBD Parent-Rated Inattention</td>
<td>Pearson Correlation</td>
<td>.179</td>
<td>.300**</td>
<td>.077</td>
<td>1</td>
<td>.297**</td>
</tr>
<tr>
<td></td>
<td>Sig.</td>
<td></td>
<td>.097</td>
<td>.005</td>
<td>.479</td>
<td>.005</td>
</tr>
<tr>
<td>PSI Parenting Stress</td>
<td>Pearson Correlation</td>
<td>.250*</td>
<td>.499**</td>
<td>.043</td>
<td>.297**</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Sig.</td>
<td></td>
<td>.019</td>
<td>&lt;.0001</td>
<td>.691</td>
<td>.005</td>
</tr>
<tr>
<td>APQ Inconsistent Parenting</td>
<td>Pearson Correlation</td>
<td>.250*</td>
<td>.415**</td>
<td>.186</td>
<td>.235*</td>
<td>.550**</td>
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<tr>
<td></td>
<td>Sig.</td>
<td></td>
<td>.020</td>
<td>&lt;.0001</td>
<td>.084</td>
<td>.028</td>
</tr>
</tbody>
</table>

Note: *significant at the .05 level; ** significant at the .01 level

As seen in table 5, phonological loop ability was significantly and positively correlated with social skills, parenting stress, and inconsistent parenting. Parent-rated ODD was significantly and positively correlated with parent-rated inattention, parenting stress, and inconsistent parenting. Parenting stress was also significantly and positively correlated with parent-rated inattention and inconsistent parenting. Inconsistent parenting was also significantly and positively correlated with parent-rated inattention.
Table 6: Mediation of the effect of parent-rated ADHD symptom severity on emotion dysregulation through phonological loop, parent-rated inattention, parent-rated ODD, inconsistent discipline, parenting stress, and social skills. (N=83)

<table>
<thead>
<tr>
<th>IV</th>
<th>Mediating Variable (M)</th>
<th>DV</th>
<th>Effect of IV on M (a path)</th>
<th>Effect of M on DV (b path)</th>
<th>Total Effect (c path)</th>
<th>Direct Effect (c’ path)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DBD Parent-Rated ADHD</td>
<td>AWMW Digit recall (phonological loop)</td>
<td>Parent BRIEF emotional control (Emotion dysregulation)</td>
<td>-.073</td>
<td>-.12</td>
<td>6.67*</td>
<td>-.72</td>
</tr>
<tr>
<td></td>
<td>DBD Parent-rated ODD</td>
<td></td>
<td>.45**</td>
<td>9.17**</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>BASC-2 Social skills</td>
<td></td>
<td>-3.30*</td>
<td>-.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>DBD Parent-rated inattention</td>
<td></td>
<td>.93**</td>
<td>2.30</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PSI Parenting Stress</td>
<td></td>
<td>6.10</td>
<td>.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>APQ Inconsistent Discipline</td>
<td></td>
<td>1.46</td>
<td>.23</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: *significant at the .05 level; ** significant at the .01 level

Normal Theory Tests (Products of Coefficients Approach) for Indirect Effects and Bootstrapping Results

<table>
<thead>
<tr>
<th></th>
<th>Products of coefficients</th>
<th>Bootstrapping BC 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Point estimate</td>
<td>SE</td>
</tr>
<tr>
<td>AWMA Digit Recall (phonological loop)</td>
<td>.37</td>
<td>.41</td>
</tr>
<tr>
<td>DBD Parent-Rated ODD</td>
<td>2.0</td>
<td>1.21</td>
</tr>
<tr>
<td></td>
<td>BASC-2 Social Skills</td>
<td>DBD Parent-Rated Inattention</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>----------------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td></td>
<td>.45</td>
<td>3.01</td>
</tr>
<tr>
<td></td>
<td>.56</td>
<td>1.89</td>
</tr>
<tr>
<td></td>
<td>.80</td>
<td>1.59</td>
</tr>
<tr>
<td></td>
<td>-.49</td>
<td>-.55</td>
</tr>
<tr>
<td></td>
<td>1.94</td>
<td>6.44</td>
</tr>
</tbody>
</table>

### Contrasts

<table>
<thead>
<tr>
<th></th>
<th>Digit Recall (phonological loop) x Parent-Rated ODD</th>
<th>Digit Recall (phonological loop) x Social Skills</th>
<th>Digit Recall (phonological loop) x Parent-Rated Inattention</th>
<th>Digit Recall (phonological loop) x Parenting Stress</th>
<th>Digit Recall (phonological loop) x Inconsistent Discipline</th>
<th>Parent-Rated ODD x Social Skills</th>
<th>Parent-Rated ODD x Parent-Rated Inattention</th>
<th>Parent-Rated ODD x Parenting Stress</th>
<th>Parent-Rated ODD x Inconsistent Discipline</th>
<th>Social Skills x Parent-Rated Inattention</th>
<th>Social Skills x Parenting Stress</th>
<th>Social Skills x Inconsistent Discipline</th>
<th>Parent-Rated Inattention x Parenting Stress</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-4.14</td>
<td>-3.5</td>
<td>-2.12</td>
<td>-.40</td>
<td>-.33</td>
<td>3.79</td>
<td>2.02</td>
<td>3.74</td>
<td>3.81</td>
<td>-1.78</td>
<td>-.05</td>
<td>.02</td>
<td>1.72</td>
</tr>
<tr>
<td></td>
<td>1.58</td>
<td>.66</td>
<td>2.95</td>
<td>.57</td>
<td>.56</td>
<td>1.71</td>
<td>3.29</td>
<td>1.59</td>
<td>1.62</td>
<td>2.99</td>
<td>.77</td>
<td>.65</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>-2.62</td>
<td>-.53</td>
<td>-.72</td>
<td>-.71</td>
<td>-.59</td>
<td>.22</td>
<td>.61</td>
<td>2.36</td>
<td>2.35</td>
<td>-.59</td>
<td>-.07</td>
<td>.03</td>
<td>.57</td>
</tr>
<tr>
<td></td>
<td>-7.17</td>
<td>-1.94</td>
<td>-8.79</td>
<td>-2.11</td>
<td>-2.01</td>
<td>1.03</td>
<td>-4.98</td>
<td>1.50</td>
<td>1.20</td>
<td>-8.64</td>
<td>-1.94</td>
<td>-1.21</td>
<td>-4.80</td>
</tr>
<tr>
<td></td>
<td>-1.64</td>
<td>.83</td>
<td>4.41</td>
<td>.66</td>
<td>.76</td>
<td>7.05</td>
<td>8.69</td>
<td>7.17</td>
<td>7.15</td>
<td>4.92</td>
<td>1.34</td>
<td>1.59</td>
<td>8.33</td>
</tr>
</tbody>
</table>
As presented in Table 6, parent-rated ADHD symptom severity indirectly influenced degree of emotion dysregulation through the six mediators. The total effect (c path) of parent-rated ADHD symptom severity and the six mediators on emotion dysregulation was significant (p=.01). Together, parent-rated ADHD symptom severity and the six mediators accounted for over 50 percent of the variance of emotion dysregulation in these youth ($R^2=.51$). However, there was no evidence that parent-rated ADHD symptom severity influenced degree of emotion dysregulation independent of its effect through the six mediators, as the direct effect (c’ path) of parent-rated ADHD symptom severity on emotion dysregulation was not significant (p=.85). The difference between the total and direct effect is the total indirect effect (a*b paths) through the six mediators. A bias-corrected bootstrap confidence interval for the indirect effect based on 5,000 bootstrap samples was entirely above zero. Thus, we can claim that the difference between the total and the direct effect of parent-rated ADHD symptom severity on emotion dysregulation is different from zero. An examination of the specific indirect effects indicated that parent-rated ODD was a mediator even when controlling for all other mediators, since its 95% confidence interval did not contain zero. No other variables (e.g., phonological loop, parent-rated inattention, inconsistent discipline, parenting stress, and social skills) contributed to the indirect effect above and beyond parent-rated ODD. Examination of the pairwise contrasts of the indirect effects shows that the specific indirect effect through parent-rated ODD was significantly larger than the specific indirect effects of phonological loop, social skills, parenting stress, and inconsistent parenting.
### Aim 2

Table 7: Demographic Information for Aim 2 Sample, by Condition

<table>
<thead>
<tr>
<th></th>
<th>Active (n=42)</th>
<th>Placebo (n=35)</th>
<th>Sig. level between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, Mean (SD) in years</td>
<td>8.53 (1.4)</td>
<td>8.28 (1.28)</td>
<td>P = .729</td>
</tr>
<tr>
<td>Sex, % Male</td>
<td>73.3</td>
<td>72.4</td>
<td>P = .123</td>
</tr>
<tr>
<td>Full Scale IQ, Mean (SD)</td>
<td>103.67 (16.13)</td>
<td>102.72 (14.44)</td>
<td>P = .25</td>
</tr>
<tr>
<td>Medicated for ADHD, %</td>
<td>26</td>
<td>25.7</td>
<td>P = .989</td>
</tr>
<tr>
<td>Methylphenidate (MPH) Derivatives</td>
<td>N=5</td>
<td>N= 2</td>
<td></td>
</tr>
<tr>
<td>Concerta</td>
<td>N=1</td>
<td>N=1</td>
<td></td>
</tr>
<tr>
<td>Focalin</td>
<td>N=0</td>
<td>N=3</td>
<td></td>
</tr>
<tr>
<td>Ritalin</td>
<td>N=0</td>
<td>N=1</td>
<td></td>
</tr>
<tr>
<td>Methylin</td>
<td>N=1</td>
<td>N=0</td>
<td></td>
</tr>
<tr>
<td>MPH unspecified</td>
<td>N=2</td>
<td>N=0</td>
<td></td>
</tr>
<tr>
<td>Amphetamine Derivatives</td>
<td>N=0</td>
<td>N=1</td>
<td></td>
</tr>
<tr>
<td>Vyvanse</td>
<td>N=0</td>
<td>N=1</td>
<td></td>
</tr>
<tr>
<td>Adderall</td>
<td>N=0</td>
<td>N=1</td>
<td></td>
</tr>
<tr>
<td>Dexedrine</td>
<td>N=2</td>
<td>N=1</td>
<td></td>
</tr>
<tr>
<td>Spansule (d-amphetamine sulfate)</td>
<td>N=1</td>
<td>N=0</td>
<td></td>
</tr>
<tr>
<td>Non-Stimulants</td>
<td>N=1</td>
<td>N=1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>DSM-IV ADHD Subtype, %</td>
<td>Comorbid ODD, %</td>
<td>Comorbid CD, %</td>
</tr>
<tr>
<td>--------------------------</td>
<td>------------------------</td>
<td>----------------</td>
<td>---------------</td>
</tr>
<tr>
<td></td>
<td>Inattentive: 36.7</td>
<td>40</td>
<td>5.4</td>
</tr>
<tr>
<td></td>
<td>Hyperactive/Impulsive: 0</td>
<td>44.8</td>
<td>44.8</td>
</tr>
<tr>
<td></td>
<td>Combined: 63.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inattentive: 44.8</td>
<td></td>
<td>8.6</td>
</tr>
<tr>
<td></td>
<td>Hyperactive/Impulsive: 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Combined: 55.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P = .629</td>
<td></td>
<td>P = .524</td>
</tr>
</tbody>
</table>

Table: Intuniv/Tenex (guanfacine) Clonidine Polytherapy

1 Polytherapy

DSM-IV ADHD Subtype: Inattentive: %, Hyperactive/Impulsive: %, Combined: %

Comorbid ODD, %: 40%

Comorbid CD, %: 5.4%

Race, %: Asian: 10.3, Caucasian: 44.8, African American/Black: 24.1, "Multi-Cultural:” 20.7, Other: 0

Ethnicity, %: Hispanic or Latino: 35.7, Not Hispanic or Latino: 64.3

Parent Marital Status, %: Married: 57.1, Never Married/Single: 25, Divorced: 10.7, Separated: 7.1

Socioeconomic Index Mean (SD) Range: 57.5 (20.45); 22-97

Baseline Emotion Dysregulation: 60.17
### Table

<table>
<thead>
<tr>
<th>Task</th>
<th>Mean Active</th>
<th>Mean Placebo</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline Digit Recall (phonological loop; Standard Score)</td>
<td>102.79</td>
<td>97.53</td>
<td>P = .069</td>
</tr>
<tr>
<td>Baseline Listening Recall (phonological aspect of central executive; Standard Score)</td>
<td>98.29787</td>
<td>97.55882</td>
<td>P = .962</td>
</tr>
<tr>
<td>Baseline Dot Matrix (visuospatial sketchpad; Standard Score)</td>
<td>93.31915</td>
<td>93.55882</td>
<td>P = .981</td>
</tr>
<tr>
<td>Spatial Recall (visuospatial aspect of central executive; Standard Score)</td>
<td>99.14894</td>
<td>96.08824</td>
<td>P = .350</td>
</tr>
<tr>
<td>Baseline Total Working Memory (Average Standard Score)</td>
<td>98.53191</td>
<td>96.29412</td>
<td>P = .299</td>
</tr>
</tbody>
</table>

Note: Data was missing from 12 participants

1 participant in the active condition was taking both MPH (unspecified) and Tenex. 1 participant in the placebo condition was taking Ritalin, clonidine, Lexapro, and amantadine.

There were no significant differences between groups on all demographic variables and on emotion dysregulation and working memory at baseline.
Table 8: Means and Standard Deviations (SD) of Change Scores and Cohen’s d from Pre-to-Post Cogmed across Groups.

<table>
<thead>
<tr>
<th></th>
<th>Active (n=42)</th>
<th>Placebo (n=35)</th>
<th>Sig. level between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean change (SD)</td>
<td>Cohen’s d Pre/post</td>
<td>Mean change (SD)</td>
</tr>
<tr>
<td>Emotion Dysregulation (T-score)</td>
<td>-.46 (10.65)</td>
<td>.05</td>
<td>-5.46 (10.328)</td>
</tr>
<tr>
<td>Digit Recall</td>
<td>6.89 (12.5)</td>
<td>.5</td>
<td>2.77 (11.27)</td>
</tr>
<tr>
<td>(phonological loop; Standard Score)</td>
<td>4.25 (14.93)</td>
<td>.3</td>
<td>3.29 (14.26)</td>
</tr>
<tr>
<td>Listening Recall</td>
<td>23.61 (18.135)</td>
<td>1.36</td>
<td>5.93 (16.39)</td>
</tr>
<tr>
<td>(phonological aspect of central executive; Standard Score)</td>
<td>6.11 (16.3)</td>
<td>.33</td>
<td>1.20 (16.23)</td>
</tr>
<tr>
<td>Spatial Recall</td>
<td>10.21 (7.7)</td>
<td>.96</td>
<td>3.30 (8.54)</td>
</tr>
<tr>
<td>(visuospatial aspect of central executive; Standard Score)</td>
<td>.05 (.4)</td>
<td>.07</td>
<td>.26 (.39)</td>
</tr>
<tr>
<td>Parent-Rated ODD (^1)</td>
<td>.05 (.4)</td>
<td>.07</td>
<td>.26 (.39)</td>
</tr>
</tbody>
</table>

Note: \(^1\) Measured on a four-point likert scale ranging from 0 (not at all)-3 (very much)

As presented in Table 8, there was a significant improvement in visuospatial sketchpad ability in the active group relative to the placebo group from pre-to-post CWMT, with a large
effect size; t(2,2)=4.57, p<.001. There was a significant improvement in total working memory performance in the active group relative to the placebo group from pre-to-post CWMT, with a large effect size; t(2,2)=3.92, p<.001. There were no significant differences among changes in all other variables between groups from pre-to-post CWMT. Based on results from Aim 1, exploratory analyses were conducted outside of the original aims regarding change in parent-rated ODD from pre-to-post CWMT. Reduction in parent-rated ODD symptom severity was significantly greater in the placebo relative to the active group, with a medium/large effect size; t(2,2)=2.18, p=.03.
Table 9: Pearson Product Moment Correlations and Fisher r-to-z transformation of Changes in Working Memory Variables and Emotion Dysregulation from Pre-to-Post CWMT

<table>
<thead>
<tr>
<th></th>
<th>Active</th>
<th>Placebo</th>
<th>Fisher r-to-z</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in Digit Recall</td>
<td>-0.437**</td>
<td>-0.358*</td>
<td>z -0.39</td>
</tr>
<tr>
<td>(phonological loop;</td>
<td>Sig. 0.004</td>
<td>0.035</td>
<td>Sig. 0.35</td>
</tr>
<tr>
<td>Standard Score)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in Listening</td>
<td>0.007</td>
<td>0.18</td>
<td>z -0.73</td>
</tr>
<tr>
<td>Recall</td>
<td>Sig. 0.966</td>
<td>0.301</td>
<td>Sig. 0.23</td>
</tr>
<tr>
<td>(phonological aspect of</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>central executive;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Standard Score)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in Dot Matrix</td>
<td>-0.160</td>
<td>-0.074</td>
<td>z -0.32</td>
</tr>
<tr>
<td>(visuospatial sketchpad;</td>
<td>Sig. 0.313</td>
<td>0.672</td>
<td>Sig. 0.36</td>
</tr>
<tr>
<td>Standard Score)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in Spatial Recall</td>
<td>-0.332*</td>
<td>0.04</td>
<td>z -1.61</td>
</tr>
<tr>
<td>(visuospatial aspect of</td>
<td>Sig. 0.032</td>
<td>0.819</td>
<td>Sig. 0.06</td>
</tr>
<tr>
<td>central executive;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Standard Score)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in Total</td>
<td>-0.438**</td>
<td>-0.059</td>
<td>z -1.72*</td>
</tr>
<tr>
<td>Working Memory</td>
<td>Sig. 0.004</td>
<td>0.735</td>
<td>Sig. 0.04</td>
</tr>
<tr>
<td>(Standard Score)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: *= significant at the .05 level. **=significant at the .01 level

As presented in Table 9, changes in phonological loop, the central executive (visuospatial aspect), and total working memory, respectively, were significantly negatively correlated with change in emotion dysregulation from pre-to-post CWMT in the active group. In other words, improvement in phonological loop, central executive, and total working memory was related to improvement in emotion regulation ability from pre-to-post CWMT in the active group. There were no significant correlations amongst change in visuospatial sketchpad and phonological aspects of the central executive and change in emotion dysregulation from pre-to-post CWMT in the active group. In the placebo group, only change in phonological loop ability was
significantly negatively correlated with change in emotion dysregulation from pre-to-post CWMT such that improvement in phonological loop was related to improvement in emotion regulation ability. There were no significant correlations amongst change in all other aspects of working memory, including the total working memory composite, and change in emotion dysregulation from pre-to-post CWMT in the placebo group. Direct comparison revealed that the correlation between change in total working memory and change in emotion dysregulation was significantly stronger in the active group versus the placebo group.
Discussion

Aim 1

The first aim of this research was to investigate a novel hypothesis regarding the relationship between working memory deficits and emotion dysregulation in the context of ADHD. This question was investigated in two different manners. For a more general analysis, a composite working memory measure was utilized to investigate whether working memory, as a unitary construct, was related to emotion dysregulation in ADHD (Aim 1). Moreover, for a more precise analysis, the construct of working memory was studied in its different components, as defined by the traditional Baddeley Model (e.g., phonological loop, visuospatial sketchpad, central executive); this was done in order to investigate whether different aspects of working memory were differentially related to emotion dysregulation in ADHD, rather than using only a composite measure (Aim 1A). It was hypothesized that working memory deficits, overall, would mediate the relationship between ADHD symptom severity and degree of emotion dysregulation (Aim 1) and that, specifically, the central executive would most strongly mediate this relationship (Aim 1A). In addition, given the complexity of emotional functioning in these youth, the current research also investigated whether a variety of other cognitive (e.g., severity of inattention), behavioral (e.g., severity of hyperactivity/impulsivity and oppositionality/defiance), parental (e.g., utilization of negative parenting behaviors, degree of parental stress), and social (e.g., social skills deficits) factors mediate the relationship between ADHD symptom severity and degree of emotion dysregulation as well (Aim 1B). To the author’s knowledge, this is the first study that directly compared the contribution of multiple variables, spanning domains of functioning, on emotional troubles in children with ADHD. In line with past theoretical and empirical research on populations with and without ADHD (e.g., Sobanski et al. 2010), it was
hypothesized that inattention and hyperactivity would not mediate the relationship between ADHD symptom severity and degree of emotion dysregulation but that the other behavioral, parental, and social variables would (Aim 1B). Hypotheses regarding the relative strength of each mediator were exploratory. Moreover, in order to have cross-informant analyses, separate parent and teacher ratings were used to measure severity of ADHD (as the independent variable), as well as for ratings of inattention, hyperactivity/impulsivity, and comorbid ODD symptoms (as mediators).

**Preliminary Analyses**

Preliminary analyses revealed that phonological loop deficits, severity of parent-rated inattention, severity of parent-rated ODD symptoms, parental use of inconsistent discipline, degree of parenting stress, and social skills deficits were significantly related to emotion dysregulation in children with ADHD. Thus, these six factors were included in the mediation model. These results confirm that there is a wide range of factors associated with emotional functioning in youth with ADHD—including those related to the child’s cognitive, behavioral, and social functioning, as well as to parental behaviors and stress.

Of the proposed independent variables, only parent-rated ADHD symptom severity was significantly related to emotion dysregulation. Teacher-rated ADHD symptom severity was not associated with emotion dysregulation. Similarly, no teacher-rated variables (e.g., ODD symptoms, inattention, hyperactivity/impulsivity) were associated with emotion dysregulation. This was discrepant with past research in that Sobanski et al. (2010) found that the degree of parent- and teacher-rated ADHD and ODD symptom severity was significantly associated with degree of emotion dysregulation in ADHD. However, Sobanski et al. utilized a composite
measure of parent- and teacher-rated emotion dysregulation, whereas the current research used only a parent-rated emotion dysregulation measure. As such, the degree to which results are impacted by having the same informant for both the independent and dependent variables is unclear and represents a limitation to the current study. In addition, the samples used in the current study and in Sobanski’s differed. The average rating of emotion dysregulation in the current sample was clinically and statistically less severe (mean T-Score= 60.39, standard deviation=11.99) than that of Sobanski’s sample (mean T-Score=68.2, standard deviation=16.6). This significant discrepancy may be accounted for the fact that the sample utilized in this study was mostly male and younger than that of Sobanski’s. Results from Sobanski indicated that females and older individuals were more likely to experience higher degrees of emotion dysregulation. As such, this younger, male-dominated sample may be at a lower-risk of experiencing significant levels of emotion dysregulation, and so this may have limited our ability to detect significant associations. However, it would be important to stress to clinicians that the emotional status of youngsters with ADHD must nonetheless be closely monitored, given evidence that this dysregulation may develop with adolescence, when environmental demands are likely to increase. It would also be important to replicate this study with a larger female sample to investigate the extent to which gender may impact results.

**Mediation Analyses**

Mediation analyses revealed that, together, the six mediators used in the model (phonological loop, parent-rated inattention, parent-rated ODD, inconsistent discipline, parenting stress, and social skills) significantly mediated the relationship between parent-rated ADHD symptom severity and emotion dysregulation. However, of the six mediators, none of the variables contributed to the mediation above and beyond parent-rated ODD symptom severity.
As such, our hypotheses were not supported in that working memory (total or in components) did *not* mediate the relationship between ADHD and emotion dysregulation while controlling for the other factors, notably severity of comorbid ODD symptoms. In fact, of the working memory variables, only phonological loop deficits were significantly associated with emotion dysregulation in the context of ADHD. It may be that higher level executive functions are not as important in explaining emotion dysregulation in ADHD. Perhaps lower level functions, such as motivational factors, may instead be an important area of focus such that alternative models of ADHD (e.g., see Sergeant, 2000) may better explain these results. Moreover, it is worth noting that the direction in which factors were included in the model may have impacted results. While hypotheses were formed based on The Functional Working Memory Model (Rapport 2001; Rapport et al., 2008), which would suggest that working memory deficits may underlie emotion dysregulation in ADHD, there is also literature that illustrates a reciprocal relationship between these factors such that emotional state can also influence cognitive functioning, including working memory ability (Gray, 2004; Storbeck, 2012).

Robust results that parent-rated ODD symptom severity is an important factor in contributing to emotion dysregulation in ADHD were consistent with past literature in this area. As reviewed above, Sobanski et al. (2010) also found that comorbid ODD symptom severity accounted for the most variance in emotion dysregulation in ADHD (25%), relative to other factors studied. Of note, because of the inherent difficulty in disentangling the construct of “ODD” and “emotion dysregulation,” given that many symptoms of ODD are characterized by emotional lability, the current study used a measure of emotion dysregulation that did not have overlapping items with symptom ratings of ODD, and so these results are not simply an artifact of the inherent similarity between the constructs of ODD and emotion dysregulation.
Nonetheless, it appears that ODD is not the only factor that contributes to emotion dysregulation in ADHD, given that comorbid ODD only explains a fraction of the variance of emotion dysregulation in ADHD, and ODD is neither necessary or sufficient for the development of emotion dysregulation in ADHD. Clearly, there are factors inherent to ADHD that are contributing to these difficulties as well. As such, future investigation is warranted in this area using “pure” ADHD samples. Given the high levels of comorbidity between ADHD and ODD, the majority of research in this area includes a mixed sample of youth with ADHD and youth with comorbid ADHD and ODD. To the author’s knowledge, only Scime & Norvilitis (2006) excluded participants with comorbid ODD. In the “pure” ADHD sample, it was found that ADHD youth reported experiencing significantly more frustration after a frustration-inducing task and that they engaged in less mood-repair. Moreover, it may be that different factors are related to emotion regulation difficulties in “pure” ADHD youth relative to their peers with comorbid ODD, as past research illustrates distinct differences between these two populations, including their respective neurocognitive profiles (Luman et al., 2009). Lastly, it would also be important to investigate the potential role of other highly comorbid conditions of ADHD, including CD, anxiety, and depressive disorders, in contributing to emotion dysregulation in ADHD. Although these disorders are often seen as comorbid conditions in ADHD, the current sample did not have enough participants with these conditions to adequately study this question. Taken together, a follow up study investigating emotion dysregulation and its mediators in ADHD by comparing “pure” ADHD samples with ADHD comorbid samples is recommended to fully clarify these questions.

Of note, although not part of the original aims, exploratory analyses were conducted to explore possible covariates. It was found that gender was significantly related to social skills
ability, such that boys in the sample were rated as having significantly more impaired social
skills relative to girls. However, re-running the analyses with gender as a covariate did not yield
any significant changes. Moreover, the only demographic factor found to be associated with
degree of emotion dysregulation in the sample was race in that those who identified as Caucasian
or “Multi-cultural” (i.e., endorsing more than one race) had significantly higher ratings of
emotion dysregulation relative to their peers who identified as Asian or African American/Black.
Interestingly, race was not associated with comorbid ODD, and so it will be important to
investigate this variable looking forward. This is especially true considering that many of the
samples used in the literature on this topic were predominately of Caucasian descent (e.g.,
Anastopoulos et al., 2011; Banaschewski et al., 2012; Jensen & Rosen, 2004; Maedgen &
Carlson, 2000; Mitchell et al., 2012; Scime & Norvilitis, 2006; Sobanski et al., 2010; Walcott &
Landau, 2004). It is also important to note that Sobanski (2010), whose results were often used
for comparison purposes and to support the study’s hypotheses, utilized a sample spanning the
United Kingdom, Belgium, Germany, the Netherlands, Ireland, Israel, Spain, and Switzerland.
As such, the extent to which results were impacted by cultural differences influencing the
perception, expression, and measurement of emotion remains unclear. The assessment of
emotion dysregulation in more diverse ADHD samples is clearly warranted, as the prevalence
and/or measurability of emotion dysregulation across various racial groups may differ.

Additional exploratory analyses, outside of the original aims, were also done in order to
investigate the degree to which medication usage in the sample influenced results.
Pharmacological treatment for ADHD has well documented effects on both emotion
dysregulation (Posner et al., 2011; Rosler et al., 2010; Sobanski et al., 2012) and working
memory (Bedard, Jain, Johnson, & Tannock, 2007; Bedard, 2004; Bedard & Tannock, 2008),
and so Aim 1 was repeated using only participants from the sample that were medication naïve. When using this sample, however, there was no longer a significant mediation effect. This may be a reflection of reduced power, due a decrease in sample size, and so it is encouraged that this investigation be replicated with larger samples.

Lastly, a limitation of using a multiple mediation model is that specific indirect effects are generally, but not always, attenuated to the extent that the mediators are correlated (Preacher & Hayes, 2008). This is often a manifestation of the phenomenon of collinearity, or redundancy amongst predictors. When this occurs, results can be impacted as the paths from each mediator to the outcome are estimated controlling for all other mediators. Multicollinearity between predictors increases sampling variance in estimates of their partial relationships with an outcome (Cohen, 2003). This sampling variance will propagate throughout the estimates of indirect effects and increase the width of confidence intervals or increase p-values from normal theory tests for specific indirect effects (Hayes, 2013). Because many of the mediators were correlated in this sample, this may have limited our ability to detect successful mediation. Despite these limitations, however, it is important to remember that, according to Hayes (2013), when mediators are correlated, this is precisely the situation in which a multiple mediator model is most useful. Including correlated mediators in a model allows one to disentangle spurious and epiphenomenal variables from potential causal variables, even though this, in turn, comes at the cost of greater sampling variance and reduced power.

Aim 2

The goal of Aim 2 was to explore whether cognitive remediation, specifically CWMT, would impact emotional functioning in youth with ADHD. More specifically, the goal was to investigate whether improvement in working memory ability would be associated with a
decrease in emotion dysregulation. CWMT, in particular, has been studied as a non-pharmacological treatment option for youth with ADHD and has been reported to impact some aspects of working memory. Although the treatment has not been shown to consistently improve ADHD symptoms and/or other untrained outcomes (Chacko et al. 2013), the influence of CWMT on emotional functioning in children with ADHD has not yet been studied. As such, a major goal of this research, overall, was to understand the cognitive mechanisms contributing to emotional functioning in ADHD and to determine whether, in turn, cognitive remediation could ameliorate some of these difficulties.

Consistent with results from Aim 1, improvement in phonological loop was significantly associated with a decrease in emotion dysregulation from pre-to-post CWMT across both the active and placebo groups. Moreover, in the active group, improvement in central executive (visuospatial component) and in the total working memory composite measure was significantly associated with a decrease in emotion dysregulation from pre-to-post CWMT as well. Direct comparisons revealed that the relationship between change in total working memory and emotion dysregulation was significantly greater in the active group relative to the placebo group. Moreover, it is important to note that further, exploratory analyses were conducted using an analysis of variance (ANOVA) to examine whether CWMT condition (e.g., active vs. placebo) moderated the relationship between change in working memory and change in emotion dysregulation from pre-to-post CWMT. Across all working memory measures, there were no significant moderation effects. However, there was a trend toward significance for CWMT condition to moderate the effect of change in total working memory from pre-to-post CWMT and change in emotion dysregulation from pre-to-post CWMT (p=.052). In other words, results using an analysis of variance were largely consistent with results of Aim 2, noted above, in that
improvement in working memory was associated with improvement in emotion regulation in the active group relative to the placebo group.

Given results of Aim 1, additional exploratory analyses were conducted outside of the original aims to explore whether improvement in emotion regulation after CWMT was associated with a change in ODD symptom severity. Results suggested that there was virtually no change in ODD symptom severity in the active group from pre-to-post CWMT. In fact, while the reduction in ODD symptom severity in the placebo group was not statistically significant, it was significantly greater than that of the active group. In other words, participants in the placebo condition had a significantly greater reduction in ODD symptoms from pre-to-post CWMT relative to the active group. This result was surprising and may be because the children who had to complete the active (e.g., more difficult) version of CWMT expressed more defiance relative to those who had the non-scaffolded (e.g., easier) version, and so these parents had higher subjective ratings of ODD.

Nonetheless, while correlational analyses are limited in that one cannot infer a causative or directional relationship, it appears that improvement in working memory is indeed associated with increased emotion regulation ability as a function of treatment (i.e., in the active condition relative to the placebo condition). While literature with adults have shown that this relationship was due to the strengthening of shared neural circuitry, including the DLPFC and other frontoparietal areas such as the ACC (e.g., Schweizer et al., 2013), it would be interesting to further explore neurobiological mechanisms of change in children and adolescents, as frontal areas are not yet developed in these populations. Rather, as mentioned above, evidence has shown that non-cortical aspects of frontal-striatal circuits are important in contributing to working memory deficits and emotion dysregulation in youth with ADHD (Shaw, 2014). As
such, mechanisms of change for improvement in both working memory and emotion regulation in children and adolescents may differ from adults and require further investigation. Indeed, while results of this and past studies continue to indicate that comorbid ODD symptom severity is a significant factor contributing to emotional problems in these youth, these results further substantiate the idea that there are additional mechanisms (e.g., neurocognitive) underlying these difficulties—mechanisms that may be significant treatment targets.

However, it is important to note that the exact clinical implication of these findings remains unclear. There were no significant differences in change in emotional functioning across active and placebo groups; indeed, the average improvement in emotion dysregulation in the active group overall was minimal (e.g., average T-Score change = -.46). However, because the standard deviation was quite large (e.g., T-score change SD =10.65), the average findings may not be applicable to the individual patient, and so further study on this topic is clearly warranted. Because females with ADHD may be more vulnerable to developing emotional troubles, as stated previously, it is recommended that this question be studied in samples that include more female participants with ADHD, as females may differentially benefit.

In addition to the limitations stated above, it is noteworthy that the sample utilized in the current study may be limited in that this was a treatment-seeking sample. This sample tends to have less variability and more severe symptoms, and so the sample used in the current study may differ from others used in this area of research. To the author’s knowledge, no other study on the correlates of emotion dysregulation in ADHD youth included a treatment seeking sample, and so this may limit the generalizability of the current results. Moreover, because a large proportion of the sample was medicated to treat ADHD symptoms, emotion dysregulation symptoms may have already been addressed with pharmacotherapy, leaving lesser room for improvement.
There were also certain methodological limitations with the implementation of CWMT. While the active and placebo versions were identical in terms of the types of tasks comprising the training and the degree of support received, there were many other factors that were likely not equivalent between the active and placebo versions, with time on task across groups being the most notable. Although this factor was addressed in that additional trials were added to the placebo version if the completion time fell under a certain criteria (e.g., 30 minutes), the placebo group, overall, spent less time on the training relative to the active group. Moreover, parents were used as training aids (see above) for CWMT. As parents also completed ratings of emotion dysregulation, potential bias may have confounded results.

Lastly, a significant limitation to the body of literature on emotional functioning in youth with ADHD is the fact that there is no “gold standard” method of measuring emotion dysregulation in pediatric populations and in ADHD populations, in particular. This limits the generalizability of the research being conducted on this topic and obfuscates one’s ability to compare findings. The establishment of such a gold-standard method of measuring this construct could certainly help to clarify remaining questions and discrepancies within this literature. In particular, a multi-modal approach to measuring emotion dysregulation in this population is also recommended (e.g., observational, physiological, multi-informant, etc.) for a more precise analysis. Moreover, other key constructs that are now considered important in understanding emotion regulation and dysregulation in children were not included in this study, including the child’s utilization of adaptive and/or maladaptive coping strategies (Braet et al., 2014), parental (specifically maternal) emotion regulation ability and emotional awareness, parenting practices specifically related to emotion, and family emotional climate (Morelen, Shaffer, & Suveg, 2014).
As such, future research including these important variables may further aid in our understanding of emotional difficulties in youth with ADHD.
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