EXPLORING THE MODERATING EFFECTS OF EXECUTIVE FUNCTIONING ON THE RELATIONSHIP BETWEEN TRAIT ANXIETY AND RESPIRATORY SINUS ARRHYTHMIA DURING STRESS IN ADOLESCENTS WITH AND WITHOUT ATTENTION-DEFICIT/HYPERACTIVITY DISORDER (ADHD)

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ABSTRACT

Lauren R. Bangert: Exploring the Moderating Effects of Executive Functioning on the Relationship Between Trait Anxiety and Respiratory Sinus Arrhythmia During Stress in Adolescents with and without Attention-Deficit/Hyperactivity Disorder (ADHD)
(Under the direction of Aysenil Belger)

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood, characterized by persistent symptoms of inattention and/or hyperactivity and impulsivity that negatively impact social and academic functioning (APA, 2013; CDC, 2018b). Adolescents with ADHD commonly exhibit symptoms of trait anxiety, which can contribute to a decreased ability to effectively react to stress (Oh et al., 2018; Weems et al., 2005). Research has indicated that executive functioning also plays an important role in the regulation of stress (Ward et al., 2015), which suggests that adolescents with and without ADHD may respond differently to stressful situations given the variability in executive functioning profiles between these two populations (Kofler et al., 2018). The current study aimed to explore this further by examining how executive functioning moderates the relationship between trait anxiety and stress regulation in adolescents with and without ADHD.

This study used data from the Cognition and Neuroimaging in Teens (CogNiT) Study conducted at UNC Chapel Hill. Participants included 40 adolescents aged 9-16 years old who had a previous diagnosis of ADHD (N=18) or who were typical controls (N=22). Executive functioning processes, including cognitive flexibility, inhibitory control, and working memory, were measured using standardized neuropsychological assessments. Trait anxiety was measured using the Spielberger State-Trait Anxiety Inventory for Children (STAI-C; Spielberger, Edwards,

Montuori, & Lushene, 1973). Stress regulation was measured using average respiratory sinus arrhythmia (RSA) assessed at five timepoints before, during, and after participation in the Montreal Imaging Stress Task (MIST; Dedovic et al., 2005).

Results indicated between-group differences in RSA withdrawal, as well as differences in the relationship between trait anxiety and RSA withdrawal. Moderation effects of executive functioning were examined using multilevel modeling (MLM). Cognitive flexibility was found to have a significant moderating effect on the relationship between trait anxiety and stress regulation for both groups. No significant effects were found for inhibitory control or working memory. The results of this study contribute to our understanding of how higher order cognitive processes such as executive functioning influence physiological responses to stress in adolescents with and without ADHD. Implications for assessment and intervention are discussed, as are limitations of the current study and future directions.

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CHAPTER 1: INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood (Centers for Disease Control and Prevention; CDC, 2018a). This costly and often debilitating disorder is characterized by a persistent pattern of inattention and/or hyperactivity and impulsivity that impairs functioning and development (American Psychiatric Association; APA, 2013). The estimated prevalence of ADHD in the U.S. has changed over time, due in part to the variability in the measurements used across studies and the evolving definitions of ADHD symptoms; however, there has generally been an upward trend in estimates of parent-reported ADHD since the first national survey was conducted in 1997 (CDC, 2018b). According to a recent, large-scale study by the Centers for Disease Control and Prevention (CDC) using data from the 2016 National Survey of Children's Health (NSCH), an estimated 6.1 million children in the U.S. between the ages of 2-17 years have received a diagnosis of ADHD at some point in their lives, which accounts for 9.4% of the overall child population in the U.S. (Danielson et al., 2016).

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V; APA, 2013), a diagnosis of ADHD requires that symptoms of inattention and/or hyperactivity and impulsivity have persisted for at least six months to a degree that is inconsistent with developmental level and that negatively impacts or reduces the quality of social, academic, or occupational functioning. Symptoms must be present prior to age 12 years, are present in two or more settings, and symptoms cannot occur exclusively during the course of schizophrenia or another psychotic disorder. Symptoms cannot be better explained by another

mental disorder or solely the manifestation of oppositional behavior or a failure to understand tasks or instructions. Six or more symptoms are required for a diagnosis in children up to age 16 years and five or more symptoms are required for adults and adolescents older than 17 years. Symptoms of inattention often include failure to attend to details, difficulty sustaining attention, distractibility, failing to follow through on instructions or tasks, difficulty with organization, and forgetfulness (APA, 2013). See Table 1 for the complete list of diagnostic criteria for inattention.

Table 1

DSM-V Diagnostic Criteria for ADHD: Inattention (APA, 2013)

a) Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
b) Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
c) Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).
d) Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
e) Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).
f) Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
g) Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).
h) Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).
i) Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

Symptoms of hyperactivity and impulsivity include being fidgety, often "on the go," difficulty remaining seated when appropriate, talking excessively, difficulty waiting his or her turn, and interrupting others (APA, 2013). See Table 2 for the complete list of diagnostic criteria for hyperactivity/impulsivity.

Table 2

DSM-V Diagnostic Criteria for ADHD: Hyperactivity/Impulsivity (APA, 2013)

a) Often fidgets with or taps hands or feet or squirms in seat.
b) Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).
c) Often runs about or climbs in situations where it is inappropriate. (Note: In adolescents or adults, may be limited to feeling restless.)
d) Often unable to play or engage in leisure activities quietly.
e) Is often "on the go," acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).
f) Often talks excessively.
g) Often blurts out an answer before a question has been completed (e.g., completes people's sentences; cannot wait for turn in conversation).
h) Often has difficulty waiting his or her turn (e.g., while waiting in line).
i) Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

There are three subtypes of ADHD that can be diagnosed, depending on the clinical presentation of symptoms: (1) Predominantly inattentive presentation, (2) predominantly hyperactive/ impulsive presentation, or (3) combined presentation (APA, 2013). There are also severity specifiers depending on if the number of symptoms or level of impairment is mild, moderate, or severe. While ADHD is most commonly diagnosed in childhood, symptoms tend to persist well into adolescence and adulthood, making it a costly and chronically disabling condition. Studies have found that up to 70% of children diagnosed with ADHD continue to exhibit impairing symptoms into adolescence and 40 to 60% continue to have symptoms into adulthood (McAuley, Crosbie, Charach, & Schachar, 2017). The healthcare costs of ADHD in the U.S. are significant, with an estimated $31.6 billion spent annually on treatment and ADHD-related work absences among adults with ADHD and family members of individuals with ADHD (CDC, 2018d). According to the 2016 NSCH study by the CDC, of children with ADHD,

62% were taking medication and 46.7% had received behavioral treatment in the past year (Danielson et al., 2016). A large percentage (23%) had received neither treatment, despite the often deleterious effects of ADHD symptoms on social, academic, and occupational functioning. Children and adolescents with ADHD also commonly experience co-occurring psychiatric disorders and symptoms, including behavior or conduct problems, learning disorders, anxiety, and depression (CDC, 2018c). This often results in poor social and academic performance that has long-term consequences in regards to functionality (CDC, 2018c).

It is well known that children and adolescents with ADHD often exhibit symptoms of trait anxiety that can have compounding effects on overall functioning (Oh et al., 2018). Experiencing high levels of trait anxiety has been associated with decreased ability to effectively react to stress (Weems et al., 2005), which is a common experience during adolescence. Poor stress regulation during adolescence has been associated with negative psychological outcomes, including increased anxiety and depression in young adulthood (Romeo, 2010). Research has indicated that executive functioning processes play an important role in the regulation of stress (Ward et al., 2015). This has important implications when considering how children with and without ADHD may respond to stressful situations given the variability in executive functioning profiles in these two populations (Kofler et al., 2018). For children and adolescents who experience both ADHD and anxiety, understanding the effects of executive functioning on their ability to regulate stress can be significant in helping to alleviate the functional impairments of ADHD. The current study aims to examine the moderating effects that executive functioning processes have on the relationship between trait anxiety and stress regulation in adolescents with and without ADHD.

CHAPTER 2: LITERATURE REVIEW

Executive Functioning During Adolescence

Childhood and adolescence are significant periods of development that are indicative of substantial brain maturation as well as psychological and behavioral changes involving the maturation of self-regulatory processes, including executive functions. Multiple definitions and models of executive functioning have been proposed by researchers over the last few decades, with little consensus on a proper definition. Throughout the literature, executive functioning most commonly refers to the collection of neurocognitive processes implicated in the purposeful, goal-directed control and coordination of cognitions, behaviors, and emotions (Zelazo & Carlson, 2012; Blakemore & Choudhury, 2006; Anderson, 2002). Miyake et al.'s (2000) prominent model conceptualized executive functioning as consisting of three primary processes: switching (cognitive flexibility), inhibition (inhibitory control), and updating (working memory).

These higher order cognitive processes have been shown to be associated with frontal lobe function, and more specifically the prefrontal cortex (Keifer & Tranel, 2013). Longitudinal research has shown that EF undergoes the most rapid development during preschool years, however; reorganization of prefrontal systems during adolescence makes this a sensitive period for further development of executive functioning (Zelazo & Carlson, 2012). Changes in EF during adolescence occur simultaneously with substantial structural and functional changes in neural systems involving the prefrontal cortex (Miyake et al., 2000). For instance, gray matter volume in the prefrontal cortex reaches a peak and white matter continues to increase, neuronal axons in the frontal cortex become more myelinated, and synaptic density increases. All of these

are thought to contribute to better brain connectivity and improved executive functioning (Blakemore & Choudhury, 2006).

Interestingly, although adolescence is generally seen as a time of overall improvement in cognitive and executive functioning skills, behavioral studies have shown a non-linear and varied pattern of development across EF skills, suggesting that not all EF processes have the same developmental trajectory (Blakemore & Choudhury, 2006). Using Miyake et al.'s (2000) three-factor model as a basis, Lee, Bull, and Ho (2013) found age-related patterns of differences in EF processes, suggesting that the efficiency in the three executive control processes becomes more specialized and independent from each other with increased age. Specifically, they found that switching and inhibition correlated significantly enough to indicate that a two-factor model (inhibition-switch and updating) is more appropriate for describing EF patterns in younger children (ages 5-13 years), but that all three processes become less intertwined by age 15 years.

ADHD and Executive Functioning

Barkley's (1997) executive functioning model of ADHD proposed that ADHD is characteristic of a deficit in behavioral inhibition and that inhibition is dependent on four executive neuropsychological functions, including working memory, self-regulation of affect-motivation-arousal, internalization of speech, and reconstitution (or, behavioral analysis and synthesis). Inherent to this model is the assumption that if impairments in behavioral inhibition exist in ADHD, so must deficits in these other four executive functions. His study suggests that the evidence is strongest for impairments in behavioral inhibition, working memory, regulation of motivation, and motor control in those with ADHD.

Although ADHD is often described as being characteristic of deficits in executive functioning, not every child with ADHD exhibits impairments in all areas of EF, or even

impairments in EF at all. In fact, children and adolescents with ADHD tend to be quite heterogeneous in their executive functioning profiles (Roberts, Martel, & Nigg, 2017). Kofler and colleagues (2018) found that 89% of children with ADHD demonstrated impairment in at least one executive function, with 62% having impaired working memory, 27% having impaired inhibitory control, and 38% having impaired cognitive flexibility. According to their findings, 54% of children with ADHD showed impairment in one EF domain while only 34% showed impairment in two or all three executive functions. In a longitudinal study, Wåhlstedt, Thorell, and Bohlin (2008) found that early EF impairments in preschool were predictive of later problems with inattention and hyperactivity. Another longitudinal study by Murray, Robinson, and Tripp (2017) found that children with ADHD demonstrated improvements in executive functioning over time, even exceeding expected age-related gains. They also found that better baseline performance on measures of attentional control, information processing, cognitive flexibility, and goal setting was associated with improved ADHD symptoms over time.

ADHD and working memory. Working memory has been defined as the capacity to actively maintain and manipulate information in one's mind over a short period of time (Dick, 2014). Research has described two components of working memory: "storage capacity," or the ability to maintain information in memory after a delay, and "processing capacity," or the ability to engage in more complex tasks that involve updating or manipulating information held in working memory (Dick, 2014). In general, working memory is crucial to functioning in that it helps individuals to retrieve and maintain relevant information important to executing day-to-day tasks (Gibson et al., 2010).

Baddeley's (2000) model of working memory is commonly cited in the ADHD literature. In this theory, working memory is characterized as having two limited-capacity short-term

memory features, including the phonological loop, which is responsible for the temporary storage and rehearsal of verbal information (such as remembering a series of numbers), and the visuospatial sketchpad, which is responsible for the temporary storage and rehearsal of visuospatial information (such as remembering spatial directions) (Gomez, Gomez, Winther, & Vance, 2014). In this model, working memory also involves a "central executive" that coordinates higher levels of processing, such as integrating information from the phonological loop and the visuospatial sketchpad, as well as the conversion of information into long-term memory storage. While children with ADHD are heterogeneous in their working memory profiles, evidence has been found for increased difficulties with all three components, with more significant deficits in the visuospatial sketchpad and the spatially-involved central executive (Gomez et al., 2014). Relatedly, Tillman and colleagues (2011) found that visual working memory was associated with inattentive symptoms in children and adolescents aged 6-16 years.

Generally, studies have shown that working memory deficits are reported in 30-37% of ADHD cases (Coghill, Seth, & Matthews, 2014). Similarly, Fried and colleagues (2016) found that significantly more children with ADHD had working memory deficits than controls (31.9% vs. 13.7%, respectively). Their study also noted that impairments in working memory were associated with increased risk for academic and cognitive dysfunction in children with ADHD, beyond the risks attributable to ADHD alone. Kofler and colleagues (2018) found that working memory deficits were associated with higher parent- and teacher-report symptoms of inattention and hyperactivity/impulsivity.

ADHD and inhibitory control. Inhibitory control is perhaps the most complicated of the three primary executive functioning constructs to understand due to the many terms it is referred by in the literature (i.e. inhibition, behavioral inhibition, response inhibition, interference control,

self-regulation, effortful control, etc.; Miyake & Friedman, 2012), the different theoretical perspectives by which it is considered (i.e. neuropsychology, evolutionary development, temperament, etc.), and the fact that many measures used to assess inhibitory control are not "pure" in their assessment (i.e. they often concurrently measure other executive functions). In general, inhibitory control refers to a set of cognitive processes involved in the ability to withhold or stop an ongoing response, or resist interference from competing or prepotent responses (Kofler et al., 2018; Lee, Bull, & Ho, 2013).

Researchers have hypothesized that for children and adolescents with ADHD, there is a deficiency in their cognitive control of response inhibition, which impairs their ability to inhibit or delay a response (Wodka et al., 2007). Seemingly inherent in the definition of ADHD symptomology for those with the hyperactive/impulsive subtype is difficulty with inhibiting impulsive behavior. Recent estimates indicate that deficits in inhibitory control are detected in 21-46% of pediatric ADHD cases (Coghill, Seth, & Matthews, 2014). Kofler et al. (2018) found that only 27% of children with ADHD had impairments in inhibitory control.

ADHD and cognitive flexibility. Cognitive flexibility refers to the ability to efficiently switch between mental processes in order to adjust one's behavior according to changes in the environment (Dajani & Uddin, 2015). Cognitive flexibility has been shown to allow individuals to adapt more effectively in response to changes, and has been associated with better academic, social, and emotional outcomes throughout the lifespan. Cognitive flexibility has been found to start developing early in childhood, with an increase in skills between the ages of 7 and 9 years old and substantial maturation by the age of 10 years (Dick, 2014). However, cognitive flexibility skills continue to advance throughout adolescence and early adulthood, with peak functioning occurring during one's twenties (Dick, 2014).

In describing cognitive flexibility as an executive function, neuroscience literature commonly refers to two different cognitive processes involved in cognitive flexibility: set shifting and task switching (Dajani & Uddin, 2015). Set shifting is considered a lower-level cognitive flexibility task and is described as the ability to shift one's attention between different "sets," or rules, within a task. For example, common tasks used to measure this type of cognitive flexibility involve sorting cards based on one characteristic of the card and shifting to sort the cards based on a different characteristic of the card (e.g. Wisconsin Card Sorting Task). Task switching, on the other hand, is considered to be a more complex, higher-order cognitive flexibility ability that involves switching between two entirely different tasks (Dajani & Uddin, 2015). Research suggests that tasks requiring cognitive flexibility tend to result in slower response times and decreased accuracy due to the additional cognitive demands it takes to inhibit one's response set for the previous task and to adapt to the new task or goal (Dajani & Uddin, 2015). As such, although cognitive flexibility is considered to be its own domain of executive functioning, it employs the use of several other executive functioning abilities in order to produce a successful outcome. For instance, neuroimaging studies have shown that cognitive flexibility tasks also recruit neuronal networks implicated in attentional, working memory, and inhibitory processes (Dajani & Uddin, 2015).

Studies involving neuroimaging have shown that children with ADHD show reduced activation in regions of the prefrontal and parietal lobe and in the basal ganglia during cognitive flexibility tasks (Bálint, Bitter, & Czobor, 2015). On neurocognitive testing, cognitive flexibility has been shown to be impaired in children with ADHD (Marzocchi, Oosterlaan, & Zuddas, 2008). In one study, researchers found that children with ADHD showed larger switch costs, meaning that when required to switch tasks, their responses were slower and less accurate when

compared to typical controls (Cepeda, Cepeda, & Kramer, 2000). Interestingly, they also determined that children who were medicated with methylphenidate (a common stimulant medication used to treat ADHD) had better ability to inhibit responses in order to shift to a new task, regardless of the frequency and unpredictability of the task switch.

Trait Anxiety and Executive Functioning

The relationship between trait anxiety and executive functioning is a complex one that has garnered much attention in the developmental neuropsychology and neuroscience literature. Attentional control theory is a major theoretical perspective that provides a framework through which the relationship between anxiety and executive functioning is commonly examined. Attentional control theory (Eysenck, Derakshan, Santos, & Calvo, 2007) was developed as an extension of the processing efficiency theory proposed by Eysenck and Calvo (1992), which suggested that individuals high in trait anxiety experience diminished efficiency in task performance due to the cognitive interference caused by excessive worry. The processing efficiency theory emphasized that anxiety negatively impacts processing efficiency, or the effort and resources spent performing a task, significantly greater than it affects overall task effectiveness, or the accuracy with which the task is completed. While this theory offered an important foundational explanation for how anxiety may influence cognitive performance, a primary limitation noted by the authors of attentional control theory is that the processing efficiency theory does not specify which executive functions are most negatively affected by anxiety (Eysenck, Derakshan, Santos, & Calvo, 2007).

Addressing the limitations of the processing efficiency theory, attentional control theory describes how high levels of anxiety negatively affect attentional control through the disruption of basic executive functions necessary for directing and maintaining attention during cognitive

tasks (Eysenck et al., 2007). According to this theory, anxiety reduces attentional focus, unless threatening stimuli is present (in which case, individuals with high anxiety are likely to pay *more* attention). Essentially, attentional control is hindered when anxiety interrupts the balance between two attentional systems commonly discussed throughout the literature, the goal-directed (or top-down) attentional system and the stimulus-driven (or bottom-up) attentional system.

Waszczuk, Brown, Eley, and Lester (2015) found that children with higher levels of trait anxiety exhibited poorer attentional control. Ursache and Raver (2014) demonstrated that higher levels of trait anxiety were associated with lower levels of executive functioning, particularly inhibitory control, in a sample of typically developing children between the ages of 9 and 12 years. Sportel and colleagues (2011) found that high behavioral inhibition was associated with increased anxiety symptoms in a sample of non-clinical adolescents. Further, their study found that attentional control moderated this relationship, such that the association between behavioral inhibition and anxiety was weakened in those with higher attentional control.

ADHD and Trait Anxiety

The relationship between ADHD and anxiety is a complicated one given the overlap of symptoms and common comorbidity. For instance, children with either ADHD or anxiety may appear restless, irritable, or have difficulty with attention and concentration. However, research indicates that even when this overlap in symptoms is controlled for, between 15% and 35% of children with ADHD demonstrate significant anxiety (Pliszka, Carlson, & Swanson, 1999). Several studies have examined the relationship between ADHD and anxiety, both in terms of the co-morbidity of ADHD and anxiety disorders and in terms of the presence of non-clinical anxiety in individuals with ADHD. Researchers typically explore non-clinical anxiety symptoms by examining levels of state and trait anxiety, which are often measured concurrently using the

State-Trait Anxiety Inventory for Children (STAI-C; Spielberger, 1973). State anxiety refers to the intensity of anxious feelings experienced as an emotional state at a particular time (Spielberger & Reheiser, 2009). Trait anxiety has been defined as a relatively stable individual disposition to perceive stressful situations as more threatening, and a tendency to respond to such situations with increased frequency and intensity of anxiety-related feelings, cognitions, and physical symptoms, including hyperarousal (Spielberger & Reheiser, 2009). Having high trait anxiety, even at a non-clinical level (i.e. having several core symptoms of anxiety but not meeting full diagnostic criteria for an anxiety disorder), has been associated with increased likelihood of other co-occurring psychopathologies, functional impairment, and even suicidal behavior (Balázs et al., 2013). Therefore, trait anxiety is an important construct that captures those who are in between the extremes of low-anxiousness and pathological anxiety.

For children and adolescents with ADHD, there has been conflicting results in the literature regarding the potential pathways by which ADHD and anxiety co-occur. Some research has indicated that anxiety is associated with increased inhibition and attentional control in those with ADHD (Rodríguez et al., 2014). Other studies have shown that children with ADHD and anxiety are more likely to be inattentive rather than impulsive. High impulsivity has been found to be related to adjustment problems, while low impulsivity is associated with behavioral inhibition, social withdrawal, and internalizing behaviors such as anxiety. In examining differences in trait anxiety by ADHD subtype, González-Castro and colleagues (2015) found that children with the combined subtype had significantly higher levels of trait anxiety than the other subtypes and controls. Additionally, high trait anxiety in children with inattentive-type ADHD was associated with poorer concentration.

Several studies have indicated that increased anxiety in children with ADHD can influence performance on cognitive tasks, though there is a lack of consensus regarding findings (Ruf, Bessette, Pearlson, & Stevens, 2017). Results from Ruf and colleagues (2017) suggested that adolescents with ADHD and high trait anxiety performed better on measures of sustained attention and reaction time. They interpreted this as indicative of a "protective" effect of trait anxiety for those with ADHD, such that the brain's arousal mechanisms may be more balanced in this group which allows them to be more successful at certain cognitive and behavioral inhibition tasks. Rodríguez and colleagues (2014) examined the impact of trait anxiety on attentional functions in children with and without ADHD. Using a four-group design (ADHD only, ADHD + trait anxiety, trait anxiety only, and typical controls), they found that the children with ADHD performed worse on selective and sustained attention tasks than both the typical control group and the group with only trait anxiety. However, there were no statistically significant differences in performance between the ADHD only and the ADHD + trait anxiety groups, suggesting that anxiety did not impact attentional control in this study. Other studies have found that child-reported anxiety is associated with slower response speed and better behavioral inhibition (Bloemsma et al.,2013).

Stress Regulation during Adolescence

Stress is a common experience for most adolescents. Increased social pressures, academic and familial demands, and personal identity development all represent potential areas in which adolescents might experience stress. Along with the increased opportunities for stressful situations to arise, the neurobiological mechanisms by which individuals respond to stress go through significant changes during the adolescent period. Pre-adolescence and adolescence are marked by significant brain maturation in the limbic and cortical areas associated with stress

regulation. Specifically, studies have shown volumetric increases in the hippocampus and amygdala, especially during the early stages of puberty (Eiland & Romeo, 2013). Other studies have shown developmental changes in cortical gray and white matter volume, such that there are increases in frontal and temporal cortical volumes from childhood to the onset of puberty and then a period of cortical thinning during adolescence (Eiland & Romeo, 2013).

This developmental stage is also distinguishable by the many neuroendocrine shifts that occur during this period, including changes in the hormonal responses to stress. In animal studies, prepubescent animals show a more prolonged hormonal stress response. Specifically, in prepubescent and mid-adolescent animals, ACTH and corticosterone levels can take twice as long to return to baseline following a stressor in comparison to adults (Eiland & Romeo, 2013). Human studies have indicated that boys and girls in later stages of adolescence (ages 15-17 years old) show greater stress-induced levels of cortisol than those who are in late childhood or early adolescence (ages 9-13 years old) (Romeo, 2013). The important changes in stress regulation that occur during adolescence makes this period a critical time to study these processes.

Hypothalamic-Pituitary-Adrenal (HPA) Axis

When an individual experiences an acute physiological or psychological stressor, two hormonal systems are initiated in an attempt to help the individual respond effectively to the situation. The initial, more immediate response is mediated by the sympathetic nervous system (SNS), which activates the release of epinephrine and norepinephrine into the blood stream (Romeo, 2013). This is often referred to as the "fight-or-flight" response. The second, more prolonged reaction to stress is mediated by the hypothalamic-pituitary-adrenal (HPA) axis. This response is activated by neurons in the paraventricular nucleus of the hypothalamus (PVN), which secrete corticotropin-releasing hormone (CRH) to stimulate the pituitary to release

adrenocorticotropic hormone (ACTH) (Romeo, 2013). ACTH then signals the adrenal glands to synthesize and secrete glucocorticoids, or cortisol. After a stressor has ended, the glucocorticoids provide feedback to the pituitary gland and forebrain regions, including the hypothalamus, hippocampus, and prefrontal cortex, which ceases the stress response by diminishing the production and release of CRH and ACTH (Romeo, 2013). Cortisol impacts the activity of several physiological systems, including the function of the autonomic nervous system (ANS), which controls cardiac responses to stress (i.e. increased heart rate) (Rotenberg & McGrath, 2016).

Polyvagal theory. The polyvagal theory, proposed by Steven Porges (1995), is one of the most prominent models of stress regulation. The theory emphasizes the role of the vagus, or the 10th cranial nerve, in modulating autonomic nervous system responses to stress by influencing the activity of both the sympathetic and parasympathetic nervous systems (PNS). The theory posits that the vagus, also referred to as the vagal nerve, contains myelinated pathways that originate in the nucleus ambiguus and function as an active "vagal break" which can rapidly inhibit or disinhibit the vagal tone of the heart to quickly mobilize or calm an individual (Porges, 2007). It does this by actively inhibiting the influence of the sympathetic nervous system on the heart and decreasing HPA axis activity. Porges (2007) suggested that the PNS remains consistently active under non-stressful conditions, inhibiting cardiac output by applying the vagal break on the heart. When a stressful situation occurs, the vagal break is quickly withdrawn to increase cardiac output. This vagal withdrawal allows for a more controlled adjustment of heart rate in response to stress and is considered a more adaptive stress response that limits the engagement of the SNS, which is more metabolically straining (Wolff, Wadsworth, Wilhelm, & Mauss, 2012).

Heart Rate Variability (HRV)

One way stress regulation is measured is through cardiac biomarkers such as heart rate variability (HRV). HRV is considered to be a standard and reliable measurement of the continuous interaction between sympathetic and parasympathetic influences on heart rate (HR), which provides information about autonomic regulatory ability (Appelhans & Luecken, 2006). Studies on HRV changes throughout development show that there is a progressive maturation of the autonomic nervous system after birth, with a gradual increase in parasympathetic relative to sympathetic mediation in the first six to ten years of life, followed by a gradual decrease (Silvetti, Drago, & Ragonese, 2001). During the first ten years of life, mean HR decreases and tends to be lower in males than females, while HRV indices increase and tend to be higher in males than females. It is thought that this decrease of HR and increase in HRV is related to an increase in parasympathetic modulation of the sinus node. After age ten, there continues to be a decrease in HR and an increase in HRV with age (Kazuma, Otsuka, Wakamatsu, Shirase, & Matsuoka, 2002).

Respiratory sinus arrhythmia (RSA). While HRV is influenced by both the sympathetic and parasympathetic nervous systems, one component of HRV called respiratory sinus arrhythmia (RSA) is entirely mediated by the PNS, making it a commonly used index of PNS activity (Appelhans & Luecken, 2006). RSA refers to the rhythmic oscillation in heart rate caused by respiration, or breathing air into the lungs. When air is breathed into the lungs, the PNS influence on heart rate is temporarily gated off and heart rate increases. PNS influence is reestablished when air is breathed out of the lungs, which results in a decrease in heart rate. By using RSA as a measure of HRV, one can ascertain a clearer indicator of PNS control during acute stress. Consistent with the polyvagal theory, measuring the amplitude of RSA provides a

sensitive index of the functional impact of the vagal break, or vagal tone (Porges, 2007). In general, RSA withdrawal refers to the withdrawal of the vagal break, which allows for a more controlled and adaptive response to stress. Therefore, higher RSA withdrawal is associated with better vagal tone and overall healthier stress response.

Trait Anxiety and Stress Regulation

The primary interest of the current study is the relationship between trait anxiety and stress regulation, and how executive functioning may moderate this relationship differently in adolescents with and without ADHD. Studies that examine anxiety and stress regulation have shown that youth with high anxiety demonstrate differences in their physiological responses to stressful experiences when compared to their non-anxious counterparts, including hormonal responses (i.e. cortisol release) and cardiac output (i.e. heart rate) (Weems et al., 2005). In general, reduced RSA and excessive RSA reactivity (i.e. withdrawal) to stress has been associated with poor emotion regulation and psychopathology, including anxiety (Beauchaine, 2015).

Most relevant studies to date have examined the relationship between anxiety and stress either in terms of how children with specific anxiety disorders (i.e. generalized anxiety, social anxiety disorder, or panic disorder) respond to stress or focus on adult populations with high trait anxiety. There are few studies that focus on the relationship between trait anxiety and stress regulation in children and adolescents, leaving a gap in our understanding of how non-clinical levels of anxiety might impact stress regulation in youth. However, potential connections between high trait anxiety and stress response in children and adolescents may be gleaned from the available research.

Theoretically, anxiety is thought to be characterized by a deviation from normal autonomic activity in response to stress, such that high anxiety is associated with more intense physiological responses to mildly threatening stimuli (i.e. hyperarousal) (Weems et al., 2005). In examining RSA levels in children with anxiety symptoms, Viana and colleagues (2017) found that the highest levels of anxiety disorder symptoms were present among children with lower baseline and lower stress-induced RSA levels (i.e. poor vagal control). In a study with adults, Watkins et al. (1998) also found that high trait anxiety was associated with significantly reduced vagal control of the heart, as indicated by a reduction in RSA.

Though there is a paucity of research on how trait anxiety impacts an adolescent's ability to regulate stress, several studies have shown that there is a link between stress exposure and negative psychological outcomes (Wolff et al., 2012). Heightened SNS reactivity, specifically, has been found to be associated with the development of stress-related psychopathology and medical illness (Wolff et al., 2012). In children, SNS reactivity has adverse effects on a number of outcomes, including increased anxiety (Wolff et al., 2012; Bakker, Tijssen, van der Meer, Koelman, & Boer, 2009). Experiencing increased stress during adolescence has also been associated with greater incidences of anxiety and depressive disorders in adulthood (Romeo, 2010).

Executive Functioning, ADHD, and Stress Regulation

Thayer and Lane (2000) proposed the neurovisceral integration model, which links executive functioning and the regulation of autonomic functions, including cardiac vagal tone. Specifically, they suggest that the subcortical prefrontal inhibitory circuits involved in self-regulation are connected to the heart by the vagus nerve (Park & Thayer, 2014). In this regard, higher resting HRV is associated with better executive functioning and more adaptable responses

to stress. Conversely, lower resting HRV has been found to be associated with underactivity in prefrontal subcortical structures implicated in self-regulation. Higher resting RSA, HRV, and vagal tone are all associated with better emotional self-regulation, as are changes in these biomarkers that reflect active regulation of emotional arousal (Bridgett, Burt, & Deater-Deckard, 2015). ADHD has been shown to be associated with autonomic nervous system dysfunction, including impaired cardiac control in response to physical or emotional stress (Tonhajzerová et al., 2014). Studies have specified that ADHD is correlated with low resting RSA, blunted RSA withdrawal, and generally decreased vagal tone (Rukmani et al., 2016).

Trait Anxiety, Executive Functioning, and Stress Regulation: Rationale for Current Study

Though research has explored the relationships between trait anxiety and EF, trait anxiety and stress regulation, and EF and stress regulation, no study could be found that examines the moderating effects of EF on the relationship between trait anxiety and stress regulation in children and adolescents. Further, no such study could be found in ADHD populations. Therefore, the current study will address these gaps in the literature by illuminating potential pathways by which these factors interact with each other in adolescents with ADHD. The findings of such a study may have important implications for assessment and intervention for youth with ADHD.

CHAPTER 3: METHODS

Statement of Purpose for the Current Study

The purpose of the present study was to explore the relationships between trait anxiety, executive functioning processes, and stress regulation in adolescents with ADHD. Specifically, this study aimed to build on the current literature by examining the three primary executive functioning processes (working memory, inhibitory control, and cognitive flexibility) and how they each may moderate the relationship between trait anxiety and stress regulation. This research can help inform interventions that target the development of executive functioning skills as a means of minimizing the negative effects of anxiety and stress for adolescents with ADHD.

Research Questions

This study examined the following research questions and hypotheses:

1. Is level of trait anxiety significantly associated with a change in RSA (RSA withdrawal)?

   Hypothesis 1: Trait anxiety will be negatively associated with stress regulation, such that higher trait anxiety will be associated with a decreased change in RSA (RSA withdrawal).

2. To what extent does executive functioning (working memory, inhibitory control, and cognitive flexibility) moderate the relationship between trait anxiety and change in RSA?

   Hypothesis 2: All three EF variables will moderate the relationship, with cognitive flexibility and inhibitory control each having a stronger effect than working memory.

3. How do these relationships differ in adolescents with ADHD compared to typical controls?

   Hypothesis 3: Group (ADHD diagnosis) will have a significant effect when added to the model, indicating group differences in the moderation relationships.

Methods

Participants

The current study used data from the Cognition and Neuroimaging in Teens (CogNiT) Study (UNC IRB #13-3857) currently being conducted by the Neurocognition and Imaging Research Lab at the University of North Carolina at Chapel Hill (UNC NIRL). Recruitment for the larger CogNiT study targeted two groups: (1) a control group consisting of males and females ages 9-16 years who have no previous psychiatric diagnoses and (2) a "medium to high risk" group consisting of males and females ages 9-16 years with ADHD, anxiety, and other behavioral/conduct disorders, as well as children and adolescents with first-degree relatives (a parent or sibling) with a psychotic disorder. Participants were excluded if they had a DSM-IV psychotic or mood disorder, autism spectrum disorder, substance abuse disorder, were pregnant, were taking any medication that directly alter cardiovascular function, had a medical illness, including colds, for one week prior to study evaluations, or has magnetic metal in their body (MRI safety risk). The current study analyzed data collected from approximately 40 participants, which included typical controls (N=22) and medium-to-high risk participants with a previous diagnosis of ADHD (N=18). Control participants were recruited from schools and the community via flyers, and the ADHD group was recruited from local pediatric health and mental health clinics. A phone screen was conducted with parents to determine whether their child may be eligible for this study. If the child met all of the phone screen criteria, a baseline clinical assessment appointment was scheduled to confirm eligibility in the study. All participants are to be evaluated at baseline, 12-months, and 24-months timepoints.

The control group consisted of twenty-two adolescents, 41% male (N=9) and 59% female (N=13), with an average age of 13.41 years (SD=2.34 years) and an average grade level of 7.73

(SD=2.45). Of the twenty-two adolescents in the control group, 68% identified as Caucasian (N=15), 18% were African American (N=4), and 14% were interracial (N=3). The ADHD group consisted of eighteen adolescents, 67% male (N=12) and 33% female (N=6), with an average age of 13.99 years (SD=2.05 years) and an average grade level of 8.53 (SD=2.11). The ADHD group was 89% Caucasian (N=16) and 11% Interracial (N=2). Of the ADHD group, 22% (N=4) were identified as receiving special education services, while 5% (N=1) of the control group received special education. None of the control participants were taking psychiatric medication at the time of the study, while 61% of the ADHD group (N=11) were taking medication to treat ADHD.

In terms of their performance on the neurocognitive assessments administered, both groups performed similarly across tasks. The control group had an average full-scale IQ of 112.73 (SD=13.99), while the ADHD group had an average full-scale IQ of 113.22 (SD=9.28), putting both groups slightly above the average national sample. Independent samples t-tests were conducted on demographic variables and determined that the two groups were significantly different in race ($F=4.614$, $df=38$, $p=0.001$), medication usage ($F=402.325$, $df=38$, $p=0.000$,), and special education status ($F=14.417$, $df=38$, $p=0.001$). Groups did not significantly differ in their executive functioning performance on inhibitory control, cognitive flexibility, and working memory tasks or in levels of trait anxiety. See Table 3 below for more details.

Table 3

Descriptive Statistics for Demographics and Study Variables

Demographics and Study Variables	Control (N=22)		ADHD (N=18)	
	M	SD	*M*	SD
Age (Years)	13.41	± 2.34	13.99	± 2.05
Grade	7.73	± 2.45	8.53	± 2.11
Sex				
Male	(N=9) 41%		(N=12) 67%	
Female	(N=13) 59%		(N=6) 33%	
Race*				
Caucasian	(N=15) 68%		(N=16) 89%	
African American	(N=4) 18%		(N=0) 0%	
Interracial	(N=3) 14%		(N=2) 11%	
Special Education*	(N=1) 5%		(N=4) 22%	
Medication*	(N=0) 0%		(N=11) 61%	
	M	SD	*M*	SD
Full Scale IQ	112.73	± 13.99	113.22	± 9.28
Cognitive Flexibility				
CWIT Inhibition/Switching Scaled Score	10.23	± 2.74	9.38	±2.97
CWIT Total Self-Corrected Errors	2.59	± 2.13	1.78	±1.52
CWIT Total Uncorrected Errors	1.27	± 1.39	0.94	±1.76
Inhibitory Control				
Tower Test Achievement Scaled Score	10.76	± 2.09	10.22	±1.63
Working Memory				
Digit Span Scaled Score	11.05	± 3.26	9.28	± 2.44
Trait Anxiety	30.82	± 5.16	31.78	± 6.49

*Significant at p≤0.05

Research Design

The purpose of the larger CoGNiT study was to evaluate the relationship between neural, physiological, and behavioral measures of working memory capacity and atypical arousal/stress regulation, and examine their contribution to cognitive dysfunction severity in adolescents. A

quantitative approach was used to conduct a cross-sectional secondary analysis of data from the larger multimodal imaging study (CogNiT) in order to explore the relationships between trait anxiety and stress regulation, as well as the moderating effects of executive functioning processes (working memory, inhibitory control, cognitive flexibility). The independent variable was trait anxiety, the dependent variable was stress regulation, and the moderator variables were each component of executive functioning, which were analyzed individually.

Procedure

As part of the CoGNiT study, participants first completed baseline clinical assessments to confirm eligibility in the study. Blood samples were obtained during the baseline visit in order to examine novel genetic markers of brain development and stress regulation. Urine toxicity screenings were also conducted prior to neuroimaging to rule out substance use. Neurocognitive tests occurred once eligibility was confirmed through clinical assessments. Participants then returned for baseline EEG and MRI sessions. Therefore, the baseline timepoint for this study consisted of up to a total of 4 separate appointments. The 12-month and 24-month follow-up timepoints will each include up to three separate visits, including neurocognitive testing, a clinical assessment, and an EEG session.

The data used for the purposes of the current study were collected over two consecutive visits to the UNC NIRL, generally occurring about one week apart during 2017-2018. During the first 3-hour visit, participants underwent a comprehensive clinical interview and were administered a neurocognitive assessment battery, including measures of executive functioning. During the second visit, which was around 90 minutes in duration, each participant completed several questionnaires and was then administered a stress task while undergoing an fMRI. The Montreal Imaging Stress Task (MIST; Dedovic et al., 2005) is a stress task that employs

challenging computerized mental arithmetic problems in the presence of negative social evaluative 'feedback.' The block design version of the MIST was administered to all participants in the scanner during fMRI data collection. Participants were read a script while in the scanner prior to beginning the task. This script sets up the paradigm by informing the participants they will be presented with experimental blocks of math problems that will be recorded, scored, and compared to other participants, as well as non-recorded, non-rated control blocks. In actuality, the math problems and responses are not recorded or evaluated. Arithmetic difficulty is titrated based on individual performance and computerized task feedback is adjusted so that a participant can never score above 50% or exceed the perceived performance of others. Negative feedback is given by the experimenter after the first and second run with increasingly negative content. The feedback following the first run informs the participant that the experimenter is watching and that their performance is below that of other participants during the experimental condition. Feedback after the second run reemphasizes their comparatively poor performance and stresses the importance of the participant performing at a minimum level for their data to count. This induces both performance and social evaluative stress. After the participants completed the entire MRI protocol and exited the scanner, they were debriefed about the MIST paradigm. During debriefing, participants were reassured that their performance was not judged or compared to others, and they were given the opportunity to ask questions and process their experience.

The specific timeline for the MRI was as follows: simulator screening and practice to acclimate participants to MRI scanner (20 minutes); instructions and set up (10 minutes); structural MRI (5 minutes); resting state baseline (5 minutes); baseline working memory task (16 minutes); MIST task (10 minutes); MIST feedback (2 minutes); MIST task (8 minutes); working memory task after stress exposure to MIST task (16 minutes); resting state after MIST stress

exposure (5 minutes); and diffusion tensor imaging (DTI; 6 minutes). Salivary cortisol and alpha amylase were collected prior to instruction/set up, between working memory baseline and MIST, after working memory task given post-MIST/stress exposure, and after DTI, however; this data was not examined in the current study. Electrocardiogram (EKG) for heart rate variability collection was measured from resting state baseline through the end of the MIST task and again from MIST task (post-MIST feedback) through resting state after stress exposure.

Measures

In addition to demographic data, the following measures were used to assess each variable:

Table 4

Variables and Measures

Variable	Measure	Numerical Score	Type of Data
Stress Regulation	Respiratory Sinus Arrhythmia (RSA)	Average RSA Change in RSA	Continuous
Trait Anxiety	Spielberger's State-Trait Anxiety Inventory for Children (STAI-C); Trait Anxiety Scale (A-Trait)	Total Score 20-80	Continuous
Inhibitory Control	Delis-Kaplan Executive Function System (D-KEFS)	Tower Test Total Achievement Scaled Score	Continuous
Cognitive Flexibility	Delis-Kaplan Executive Function System (D-KEFS)	Color-Word Interference Test Condition 4: Inhibition/Switching Scaled Score	Continuous
Working Memory	Wechsler Abbreviated Scale of Intelligence (WASI)	Digit Span Total Scaled Score	Continuous
Covariates			
Group	Demographic Data	Control (0) ADHD (1)	Dichotomous
Age	Demographic Data	Ages 9-16 years	Continuous
Sex	Demographic Data	Male (0) Female (1)	Dichotomous
Grade Level	Demographic Data	Grade Level 3-11	Continuous
Special Education	Demographic Data	No (0) Yes (1)	Dichotomous
Medication	Demographic Data	No (0) Yes (1)	Dichotomous
General Intellectual Ability	Wechsler Abbreviated Scale of Intelligence (WASI)	Full Scale IQ Standard Score	Continuous

Trait anxiety. Prior to the fMRI task during the second visit, participants completed the Spielberger's State-Trait Anxiety Inventory for Children, which consists of two 20-item scales that measure state and trait anxiety in children between the ages of 8 and 14 years (STAIC; Spielberger, Edwards, Montuori, & Lushene, 1973). The Trait Anxiety scale of the STAIC has been shown to have high internal consistency, with a reliability coefficient of α=.91, and as being highly correlated with other measures of childhood anxiety, such as the Revised Children's Manifest Anxiety Scale (RCMAS; r=0.88) (Morosini, Magliano, Brambilla, Ugolini, & Pioli, 2000). The Trait Anxiety scale was used to provide a measure of trait anxiety. This scale is a self-report measure that contains 20 items requiring the participant to rate how often they experience certain anxiety-related symptoms on a 3-point scale (1=hardly ever, 2=sometimes, 3=often). The ratings for all items are then summed to calculate a total Trait Anxiety score between 20 and 60, with a higher value indicating higher trait anxiety. Examples of items include: "I worry about making mistakes," "I worry about things that may happen," or "I have trouble making up my mind."

Working memory. As part of the neurocognitive test battery, participants completed the Digit Span test from the Wechsler Abbreviated Scale of Intelligence (WASI), which was used to measure working memory ability. Digit Span consists of two parts, Digit Span Forward and Digit Span Backward. The participant is required to repeat 3-9 digits forward and 2-9 digits backward, which are presented verbally. One point is given for each correct response, with eight items consisting of two trials each. The participant must answer correctly on at least one of the trials in each item in order to move on to the next item. The raw scores from Digit Span Forward and Digit Span Backward are added and then converted into a Scaled Score. The Digit Span Scaled Score was used to measure working memory.

Inhibitory control. Participants completed the Tower Test from the Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001) as part of a larger neurocognitive test battery administered for the CogNit study. The D-KEFS Tower Test is a modified version of other commonly used tower tests (Tower of London and Tower of Hanoi), and was designed to measure planning and problem solving, as well as rule learning, inhibitory control, and maintenance of instructional sets (Shunk, Davis, & Dean, 2006). Internal consistency coefficients for the D-KEFS Tower Test range from .43 to .84 by age group and test-retest reliability for the Total Achievement Score was moderate (r=.40) (Larochette, Benn, & Harrison, 2009). Adequate convergent and discriminant validity for the D-KEFS Tower Test have also been shown (Larochette, Benn, & Harrison, 2009).

The test includes five differently-sized disks and a board with three pegs. The examiner places the disks in a predetermined arrangement on the pegs and the participant is asked to move the disks to reproduce an "ending position" shown in a stimulus book. There are 9 items that increase in complexity, with the minimum number of moves ranging from 1 to 26. The participant is required to complete each item in the least amount of moves possible in the shortest amount of time while following two rules: (1) the participant cannot move more than one disk at a time and (2) the participant cannot place a larger disk on top of a smaller disk. The Total Achievement Scaled Score from the Tower Test was used to measure inhibitory control.

Cognitive flexibility. Participants also completed the Color-Word Interference Test (CWIT) from the D-KEFS (Delis, Kaplan, & Kramer, 2001). The D-KEFS is a widely-used standardized battery comprised of nine individual neuropsychological tests that measure different domains of executive functioning in individuals aged 8-89 years. Reliable and valid normative data are based on a nationally representative sample of 1750 children, adolescents, and adults

between the ages of 8 and 89 years old (Shunk, Davis, & Dean, 2006). The CWIT was modified from the classic Stroop task and was designed to measure inhibition (inhibitory control) and switching (cognitive flexibility). The CWIT consists of four conditions: Color Naming (Condition 1), Word Reading (Condition 2), Inhibition (Condition 3), and Inhibition/Switching Condition 4) (Lippa & Davis, 2010).

For the Color Naming condition, participants view a page containing several rows of red, green, and blue squares, which they are instructed to name as quickly as they can without making mistakes. For the Word Reading condition, participants are presented with a page with the words "red," "green," and "blue" printed in black ink, which they are asked to read aloud as quickly as they can without making mistakes. For the Inhibition condition, participants view a page with the words "red," "green," and "blue" printed incongruently in red, green, or blue ink. Participants are required to name the color of the ink in which the word is printed as quickly as they can without making mistakes. For the Inhibition/Switching condition, the participant is presented with a page that contains the words "red," "green," and "blue" printed in red, green, or blue ink, half of which are enclosed within boxes. For words that are not inside boxes, the participant is asked to name the color of the ink in which the word is printed. For words that appear inside a box, the participant is asked to read the word aloud (and not the name of the ink color). The participant is required to name the words or the ink colors as quickly as he/she can without making mistakes.

Performance on the CWIT is measured by the time it takes to complete each condition, as well as the number of uncorrected errors and self-corrected errors for each condition. A scaled score for each condition is also derived from the raw scores. For the purposes of this study, the Scaled Score derived from Condition 4: Inhibition/Switching was used to measure cognitive flexibility. Total corrected and uncorrected errors were also used for analysis.

Stress regulation. Autonomic nervous system (ANS) activity was measured before, during, and after the stress exposure condition (fMRI MIST task). A Biopac pulseometer was used to collect heart rate (HR) data during the fMRI. This data was processed using Acknowledge software during the scan, then converted into EKG data using the CardioEdit program and manually edited. Data was then analyzed using Cardiobatch software, which produces metrics including average HR and average RSA. Average RSA was age adjusted and calculated at each timepoint to plot average RSA values at baseline (Resting State 1; RS1), at three timepoints during the stress task (MIST1, MIST2, and MIST3), and at recovery (Resting State 2; RS2). Change in RSA from baseline was calculated to determine extent of vagal withdrawal by subtracting RS1 average RSA from MIST3 average RSA.

Analytic Procedures

Univariate descriptive statistics for each variable and demographics, as well as a bivariate correlation matrix are analyzed. The following analytic approaches were used:

Research Question 1: Is level of trait anxiety significantly associated with a change in stress regulation?

To examine RQ1, a bivariate correlation was presented to show if trait anxiety is significantly associated with average RSA at each timepoint as well as Change in RSA. See Figure 1, where *c* is the direct effect of trait anxiety on stress regulation.

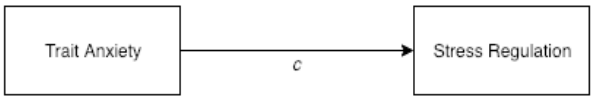

Figure 1. Direct Effect Model for Research Question 1

Research Question 2: To what extent does executive functioning (working memory, inhibitory control, and cognitive flexibility) moderate the relationship between trait anxiety and stress regulation?

Due to the relatively small sample size and the availability of longitudinal data (multiple RSA timepoints), a multilevel modeling (MLM) analytic approach was utilized to answer RQ2. Multilevel modeling (MLM), also referred to as hierarchical linear modeling (HLM), is indicated when observations at one level of analysis are "nested" within another level of analysis, such that the observations at one level of analysis are interdependent (Nezlek, 2012). Repeated measures from a longitudinal study where multiple observations are "nested" within individuals is considered to be a nested, hierarchical data structure appropriate for MLM (Peugh, 2010). In order to conduct a multi-level model analysis, several steps are involved.

Step 1. Data Preparation. To prepare data for multilevel modeling, multivariate data needs to be transformed into univariate data, meaning that rather than each row representing one participant and each column representing a different variable, each row represents a specific observation such that each participant can have multiple rows of observations for the same variable (Kwok et al. 2008). For example, the data for the current study was restructured so that instead of having a different column for each RSA timepoint, a new variable called "Timepoint" was created such that each participant has four rows of data to represent the four different RSA timepoints used. Additionally, a new outcome variable "Change in RSA" was created to adhere to the same data structure, so that each timepoint also had a "Change in RSA" value. This was calculated by subtracting the average baseline RSA (RS1) from the average RSA at each subsequent timepoint according to Table 5.

Table 5

Calculation for Change in RSA

Timepoint after Baseline		**Change in RSA**
1	MIST1	*MIST1 – RS1*
2	MIST2	*MIST2 – RS1*
3	MIST3	*MIST3 – RS1*
4	RS2	*RS2 – RS1*

Step 2. Model Specification. In the current study, a two-level data structure was used, with Level-1 representing the multiple timepoints at which average RSA was obtained after baseline (MIST1, MIST2, MIST3, and RS2) and Level-2 representing individual characteristics (demographic variables, trait anxiety, and executive functioning performance). Level 1 has 160 observations (four RSA timepoints after baseline multiplied by 40 participants), while Level 2 has 40 observations (each variable has one observation per participant). The basic equations used for the two-level model are presented below:

Level-1 Model:

$$[\text{Change in RSA}]_{ti} = \beta_{0i} + e_{ti}$$

Where:

t = time index

i = participant index

β_{0i} = estimated average Change in RSA for each participant over the four timepoints

e_{ti} = within-individual random error (difference between observed Change in RSA at time t and the average score of the i'th participant

Level-2 Model:

$$\beta_{0i} = \gamma_{00} + \gamma_{01}\text{Sex}_i + \gamma_{02}\text{Age}_i + \gamma_{03}\text{IQ}_i + \gamma_{04}\text{Group}_i + \gamma_{05}\text{Race}_i + \gamma_{06}\text{TraitAnxiety}_i + U_{0i}$$

Where:

γ_{00} = the grand mean of the 40 average Change in RSA scores

$\gamma_{01}, \gamma_{02}, \gamma_{03}...$[Sex, Age, IQ, …]$_i$ = individual-level fixed effects

U_{0i} = difference between the *i*'th average Change in RSA score and the grand mean

Specific models are then fitted by adding in variables of interest. In the current study, executive functioning measures and the interaction between trait anxiety and the executive functioning measures were added to the basic model in a step-wise manner in order to assess for individual and moderation effects.

Step 3. Calculating the Intraclass Correlation Coefficient. In multilevel modeling, an intraclass correlation coefficient (ICC) is calculated prior to fitting specific models in order to assess the extent of within-individual variances of the outcome variable (Kwok et al., 2008). Using an unconditional means model, the ICC was calculated using the equation below:

$$ICC = \tau_{00} / (\tau_{00}+\sigma^2)$$

Where:

τ_{00} = between-individual variances

σ^2 = within-individual variances

The ICC for Change in RSA was 55.71 / (55.71 + 30.74) = 0.6444, indicating that 64.44% of the variance in Change in RSA in the sample is due to within-individual differences. A low ICC is an indicator that MLM is not the best statistical method because there is not enough between-individual variance (Nezlek, 2012). The ICC calculated for the current data is high enough to indicate adequate variance to continue with MLM analysis.

Step 4. Determining covariance structure. A diagonal covariance structure, which is the default variance components structure, was chosen for each model. The default structure was kept due to issues of non-convergence when other covariance structures were fitted to each model (e.g. autoregression [AR1], compound symmetry, unstructured, etc.), indicating that these covariance structures are not appropriate for the current analyses.

Step 5. Fixed vs. Random Effects. Models were run with both fixed and random intercepts and significance was not changed by including a random intercept. As such, for model simplicity and interpretability, patient-level random intercepts were omitted and fixed intercepts were used for each model. All independent variables were treated as fixed-effects due to their time-invariant nature, meaning they are relatively stable and do not change with time.

Step 6. Analysis. Several multilevel models were fitted by adding the variables (trait anxiety and executive functioning performance) separately in order to answer each component of the research question. Interaction terms (trait anxiety*working memory, trait anxiety*inhibitory control, and trait anxiety*cognitive flexibility) were also added to the models to analyze moderation effects. Models were analyzed individually for significant predictors of the outcome variable, Change in RSA. Models for each variable and interaction of interest were compared using the Bayesian Information Criterion (BIC) generated from each model. Generally, the model with the lowest BIC indicates a better fit, with differences less than 2 indicating no substantial difference and differences larger than 10 indicating a substantial difference between two models (Kwok et al., 2008).

The figures below represent the general moderation effect analyzed for each of the three executive functioning processes (working memory, inhibitory control, and cognitive flexibility) on the relationship between trait anxiety and stress regulation (see Figures 2, 3, and 4).

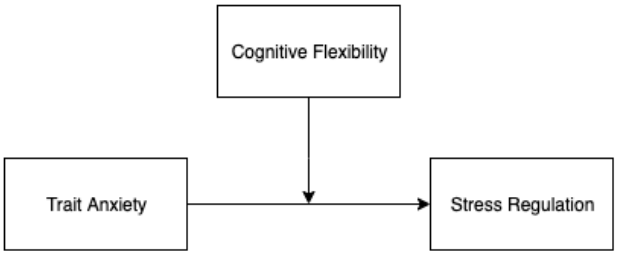

Figure 2. Moderator Model for RQ2 (Cognitive Flexibility)

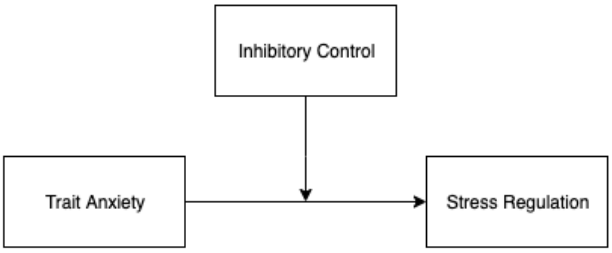

Figure 3. Moderator Model for RQ2 (Inhibitory Control)

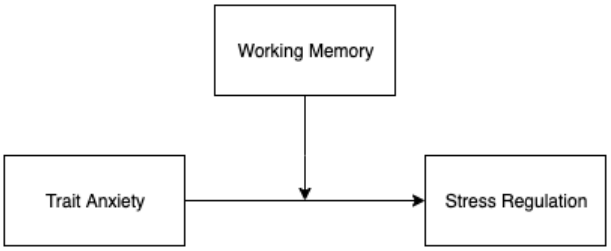

Figure 4. Moderator Model for RQ2 (Working Memory)

Research Question 3: How do these relationships differ in adolescents with ADHD compared to typical controls?

To answer RQ3, three multilevel models were generated that tested the hypothesis that there would be a significant main effect of having an ADHD diagnosis. This was done by adding the variable "group" to the final three moderator models for cognitive flexibility, inhibitory control, and working memory.

CHAPTER 4: RESULTS

The purpose of this study was to explore the relationships between trait anxiety, executive functioning, and stress regulation in adolescents with and without ADHD. To address the research questions, data was analyzed using IBM SPSS Statistics Version 25. Prior to running the models to examine each research question, the data set was prepared by screening the overall CoGNiT study sample to determine which participants: 1) had available data for each of the variables used, and 2) belonged to the control group or had a diagnosis of ADHD. Participants who did not have complete data for each variable at the time of this paper were excluded. Categorical variables were dummy coded into dichotomous variables for use in the analytic models. Descriptive statistics are presented in Table 3 and in Table 6 below. Preliminary analyses were conducted to determine if there were any violations of the assumptions of normality, linearity, multicollinearity, and homogeneity of variance, with no significant issues identified.

Average RSA

Table 6 indicates the average RSA at each timepoint (before, during, and after the stress task), as well as the average Change in RSA (RSA withdrawal), for each group. Here, change in RSA was calculated by subtracting the average RSA for RS1 from the average RSA for MIST3, which serves as the indicator of change in RSA from baseline (RS1) to the most stressful point of the MIST (MIST3). Independent samples t-tests and one-way between-groups analyses of variance (ANOVA) were conducted and results indicate that there are no significant differences between the control group and the ADHD group in the means or variances for any individual

timepoint. However, Levene's Test for the Equality of Variances revealed that there is a statistically significant difference in the variances of the control and ADHD groups for Change in RSA (F=12.901, df=38, p=0.001).

Table 6

Average RSA at Each Timepoint by Group

Stress Regulation	Control (N=22)		ADHD (N=18)	
Timepoint	*M*	SD	*M*	SD
Resting State 1 (Pre-Stress) RSA	6.99	± 1.41	6.68	± 1.25
MIST1 RSA	6.85	± 1.22	6.63	± 1.06
MIST2 RSA	6.59	± 1.43	6.52	± 1.09
MIST3 RSA	6.61	± 1.37	6.45	± 0.97
Resting State 2 (Post-Stress) RSA	6.80	± 1.34	6.67	± 1.32
*Change in RSA (MIST3-RS1)**	-0.38	± 1.45	-0.22	± 0.52

*Significant at p≤0.001

Change in RSA (RSA Withdrawal)

To further explore the group differences in the variance of Change in RSA, a boxplot was generated (Figure 5). This provides a visual representation of the distribution of RSA for Change in RSA for each group, which allows us to see that the variability of RSA levels is much greater in the control group (M= -0.38, SD=1.45) than in the ADHD group (M= -0.22, SD=0.52). The boxplot also revealed an outlier in the ADHD group, which upon further examination, was not determined to be extreme enough to exclude from analyses and therefore remained in the sample.

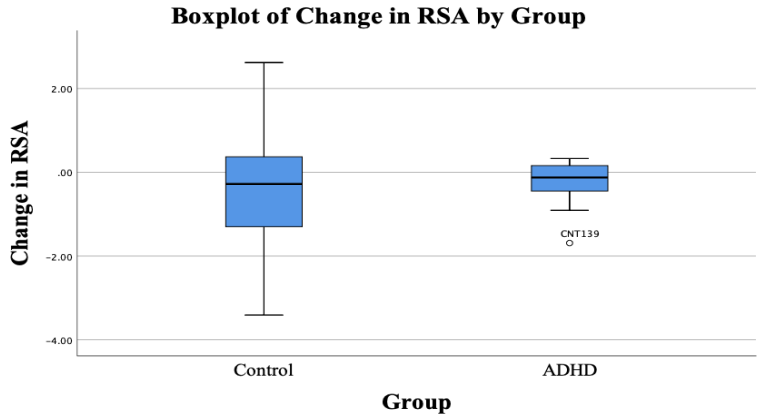

Figure 5. Boxplot of Change in RSA by Group

Stress regulation for both groups was also examined by plotting the average RSA for each timepoint before, during, and after the stress task, which is represented in Figure 6. This figure illustrates the average stress response for each group throughout a stressful experience. The control group had higher baseline RSA (M=6.99, SD=1.41) than the ADHD group (M=6.68, SD=1.25), which is consistent with previous literature. The ADHD group shows more blunted RSA withdrawal between timepoints than the control group, which is also consistent with results from previous studies (Tonhajzerová et al., 2014). Larger variability between baseline RSA and MIST3 RSA for the control group is expected, given that this is an indicator of healthier vagal tone and a more appropriate stress response (Bridgett et al., 2015). The less varied response from the ADHD group is also expected, which indicates that the vagal break is not being withdrawn as well and the stress response is not as well-regulated.

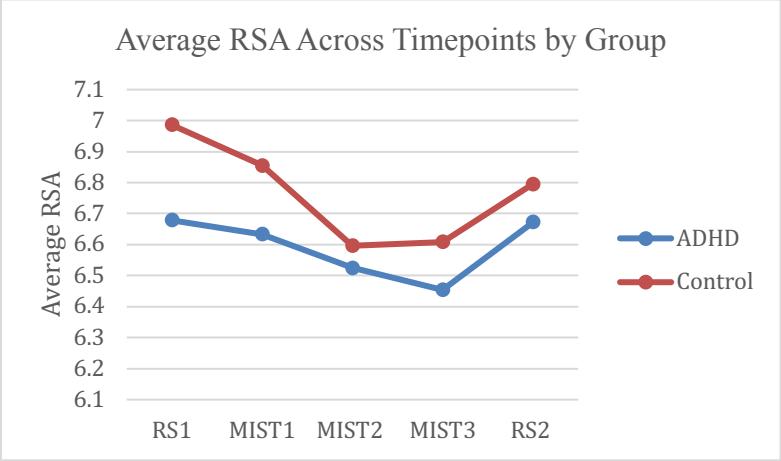

Figure 6. Plot of Average RSA Across Timepoints by Group

Research Question 1: Is level of trait anxiety significantly associated with stress regulation?

To answer RQ1, bivariate correlations were used to assess the direction and strength of the relationship between trait anxiety and stress regulation (RSA). First, Pearson product-moment correlation coefficients (r) were generated with the entire sample in order to explore the general relationship. Results indicate that there is a statistically significant positive relationship

between trait anxiety and average RSA during RS2 (r=.328, N=40, p=.039) for the combined sample, but not for other timepoints or overall change in RSA. This indicates that, for the combined sample, trait anxiety is associated with recovery from stress, but not withdrawal of the vagal brake during the stress task.

Table 7

Bivariate Correlation Matrix for Trait Anxiety and RSA for Combined Sample

	Trait Anxiety	RS1 Average RSA	MIST1 Average RSA	MIST2 Average RSA	MIST3 Average RSA	RS2 Average RSA	Change in RSA
Trait Anxiety	1	.267	.287	.247	.258	**.328***	-.043
RS1 Average RSA		1	**.785****	**.653****	**.615****	**.910****	**-.535****
MIST1 Average RSA			1	**.869****	**.782****	**.841****	-.100
MIST2 Average RSA				1	**.941****	**.723****	.228
MIST3 Average RSA					1	**.687****	**.337***
RS2 Average RSA						1	**-.351***
Change in RSA							1

*Correlation is significant at the 0.05 level (2-tailed)

**Correlation is significant at the 0.01 level (2-tailed)

Next, the relationship between trait anxiety and stress regulation was explored by group (control and ADHD). For the control group, there was a statistically significant positive correlation between trait anxiety and average RSA for MIST1 (r=.517, N=22, p=.014), MIST2 (r=.451, N=22, p=.035), MIST3 (r=.445, N=22, p=.038), and RS2 (r=.481, N=22, p=.023), with higher trait anxiety associated with higher average RSA. Trait anxiety was not associated with baseline RSA or change in RSA for the control group (see Table 8).

Table 8

Bivariate Correlation Matrix for Trait Anxiety and RSA for Control Group

	Trait Anxiety	RS1 Average RSA	MIST1 Average RSA	MIST2 Average RSA	MIST3 Average RSA	RS2 Average RSA	Change in RSA
Trait Anxiety	1	.418	**.517***	**.451***	**.445***	**.481***	.014
RS1 Average RSA		1	**.741****	**.529***	**.462***	**.905****	**-.539****
MIST1 Average RSA			1	**.842****	**.752****	**.798****	-.011
MIST2 Average RSA				1	**.942****	**.614****	.377
MIST3 Average RSA					1	**.571****	**.498***
RS2 Average RSA						1	-.343
Change in RSA							1

*Correlation is significant at the 0.05 level (2-tailed)

**Correlation is significant at the 0.01 level (2-tailed)

For the ADHD group, trait anxiety was not significantly associated with average RSA at any timepoint or with overall change in RSA (see Table 9).

Table 9

Bivariate Correlation Matrix for Trait Anxiety and RSA for ADHD Group

	Trait Anxiety	RS1 Average RSA	MIST1 Average RSA	MIST2 Average RSA	MIST3 Average RSA	RS2 Average RSA	Change in RSA
Trait Anxiety	1	.136	.060	.010	.040	.191	-.253
RS1 Average RSA		1	**.850****	**.890****	**.922****	**.923****	**-.684****
MIST1 Average RSA			1	**.928****	**.851****	**.908****	-.454
MIST2 Average RSA				1	**.943****	**.917****	-.377
MIST3 Average RSA					1	**.913****	-.348
RS2 Average RSA						1	**-.514***
Change in RSA							1

*Correlation is significant at the 0.05 level (2-tailed)

**Correlation is significant at the 0.01 level (2-tailed)

Research Question 2: To what extent does executive functioning (working memory, inhibitory control, and cognitive flexibility) moderate the relationship between trait anxiety and stress regulation?

To answer RQ2, several multilevel models were fitted in a step-wise manner in order to examine the individual effects of each executive functioning variable (working memory, inhibitory control, and cognitive flexibility) on change in RSA. Interaction terms for each executive functioning variable and trait anxiety were also modeled in order to assess for significant moderation effects.

Model 1: Demographic Variables

The first multilevel model served as a baseline model to examine the relationship of each covariate of interest and the outcome measure to determine potentially significant demographic predictors. The covariates included in the baseline model were age, sex, race, IQ, and trait anxiety (Level 2). The outcome measure used was longitudinal change in RSA for each timepoint (Level 1). This model showed non-significant fixed effects for all variables except for sex, which was significant at the $p<0.05$ level. This indicates that there is no main effect of age, group status (ADHD or control), medication, race, or trait anxiety on change in RSA (RSA withdrawal). There is a main predictive value of sex. The interaction between sex and group was added to the model to explore a potentially significant interaction effect, but the relationship was found to be non-significant.

Table 10

Fixed Effects Model for Demographic Variables

Model 1. Estimated Fixed Effects for Demographic Predictors of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-1.984386	1.090188	103.318	-1.820	.072	-4.146436	.177665
Sex	**.304624***	**.142522**	**103.318**	**2.137**	**.035**	**.021976**	**.587273**
Age	.005208	.002871	103.318	1.814	.073	-.000486	.010902
IQ	.004558	.006669	103.318	.684	.496	-.008667	.017783
Group	-.151710	.179018	103.318	-.847	.399	-.506738	.203317
Medication	.213108	.198428	103.318	1.074	.285	-.180412	.606628
Race (AA)	-.037710	.296253	103.318	-.127	.899	-.625236	.549817
Race (Caucasian)	.071775	.202624	103.318	.354	.724	-.330067	.473617
Trait Anxiety	.005662	.011712	103.318	.483	.630	-.017564	.028889

Note: Akaike's Information Criterion (AIC) = 443.319; Schwarz's Bayesian Criterion (BIC) = 455.388

Model 2: Cognitive Flexibility

Next, several models were fitted to explore the predictive value of each measure of cognitive flexibility on change in RSA, which included the Color-Word Interference Test (CWIT) Scaled Score, the number of Corrected Errors from the CWIT, and the number of Uncorrected Errors from the CWIT. Since the primary aim of RQ2 is to examine the moderation effect of each executive functioning measure on the relationship between trait anxiety and stress regulation, trait anxiety was also included as a variable in each of these models. Each measure of cognitive flexibility was added to the model separately to assess for individual contributions to the variance. Next, the interaction between trait anxiety and each measure of cognitive flexibility was added to the model to assess potential moderation effects.

Table 11

Fixed Effects Model for Cognitive Flexibility (CWIT SS)

Model 2A. Estimated Fixed Effects for Color-Word Interference Test SS as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.466148	.661201	107.810	-.705	.482	-1.776789	.844492
Sex	**.258261***	**.126173**	**107.810**	**2.047**	**.043**	**.008160**	**.508361**
IQ	.004150	.005508	107.810	.753	.453	-.006769	.015069
Trait Anxiety (TA)	.002480	.011251	107.810	.220	.826	-.019821	.024780
CogFlex CWIT SS	-.037530	.022857	107.810	-1.642	.104	-.082838	.007778

Note: CWIT SS= Color-Word Interference Test Scaled Score

Akaike's Information Criterion (AIC)=435.602; Schwarz's Bayesian Criterion (BIC)=447.776

Model 2A. Color-word interference test scaled score. Table 11 shows the fixed-effect model for CWIT Scaled Score, which produced a non-significant fixed effect for this measure of cognitive flexibility on change in RSA. A significant fixed effect for sex remained in this model.

Model 2B. Moderation effect of cognitive flexibility (CWIT SS). Table 12 shows the moderator model with the interaction term added for trait anxiety*CWIT SS. The interaction term was created by converting the scores for trait anxiety and the CWIT SS into z-scores and then multiplying the z-scores together. This model produced a significant fixed effect for sex. While CWIT SS did not show significance on its own, adding the interaction between trait anxiety and CWIT SS was significant in this model, indicating that cognitive flexibility (as measured by the CWIT SS) moderates the relationship between trait anxiety and change in RSA. Further, when comparing the BIC from Model 2A (BIC=447.776) and 2B (BIC=441.713), there is an improvement in model fit when the interaction between trait anxiety and CWIT SS is added. This finding is consistent with the hypothesis that cognitive flexibility would moderate the relationship between trait anxiety and stress regulation.

Table 12

Moderator Model for Cognitive Flexibility (CWIT SS)

Model 2B. Estimated Fixed Effects for Interaction of TA and CWIT SS as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.867238	.644803	100.392	-1.345	.182	-2.146449	.411972
Sex	**.291773***	**.121017**	**100.392**	**2.411**	**.018**	**.051691**	**.531856**
IQ	.007337	.005383	100.392	1.363	.176	-.003343	.018017
Trait Anxiety (TA)	.007035	.010832	100.392	.649	.517	-.014454	.028524
CogFlex CWIT SS	-.048408	.022214	100.392	-2.179	.032	-.092477	-.004339
TA x CogFlex CWIT SS	**.186501***	**.058044**	**100.392**	**3.213**	**.002**	**.071349**	**.301652**

Note: Akaike's Information Criterion (AIC)=429.565; Schwarz's Bayesian Criterion (BIC)=441.713

Model 2C. Color-word interference test (corrected errors). Table 13 shows the fixed-effect model for CWIT corrected errors, which produced a non-significant fixed effect for this measure of cognitive flexibility on change in RSA. Once again, a significant effect for sex was present in this model.

Table 13

Fixed Effects Model for Cognitive Flexibility (CWIT Corrected Errors)

Model 2C. Estimated Fixed Effects for CWIT Corrected Errors as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.639371	.654667	106.166	-.977	.331	-1.937288	.658546
Sex	**.251252***	**.127021**	**106.166**	**1.978**	**.051**	**-.000576**	**.503079**
IQ	.002077	.005407	106.166	.384	.702	-.008642	.012797
Trait Anxiety (TA)	.004583	.011282	106.166	.406	.685	-.017783	.026950
CogFlex CWIT Corrected Errors	-.010315	.033716	106.166	-.306	.760	-.077160	.056529

Note: Akaike's Information Criterion (AIC)=437.369; Schwarz's Bayesian Criterion (BIC)=449.542

Model 2D. Moderation effect of cognitive flexibility (CWIT corrected errors). Table 14 shows the moderator model with the interaction term added for trait anxiety*CWIT corrected errors. The interaction term was created by converting the scores for trait anxiety and the CWIT

corrected errors into z-scores and then multiplying the z-scores together. This model produced a significant fixed effect for sex, but the interaction between trait anxiety and CWIT corrected errors did not significantly add to the model. When comparing the BIC from Model 2C (BIC=449.542) and 2D (BIC=451.854), the addition of the interaction term actually decreases the fit of the model. This indicates that this measure of cognitive flexibility does not moderate the relationship between trait anxiety and change in RSA.

Table 14

Moderator Model for Cognitive Flexibility (CWIT Corrected Errors)

Model 2D. Estimated Fixed Effects for Interaction of TA and CWIT CE as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.756656	.672811	104.217	-1.125	.263	-2.090832	.577520
Sex	**.264007***	**.127909**	**104.217**	**2.064**	**.042**	**.010364**	**.517650**
IQ	.002643	.005465	104.217	.484	.630	-.008194	.013480
Trait Anxiety	.005834	.011386	104.217	.512	.609	-.016745	.028413
CogFlex CWIT Corrected Errors	-.004498	.034582	104.217	-.130	.897	-.073074	.064078
TA x CogFlex Corrected Errors	-.070832	.093437	104.217	-.758	.450	-.256118	.114453

Note: Akaike's Information Criterion (AIC)=439.706; Schwarz's Bayesian Criterion (BIC)=451.854

Model 2E. Color-word interference test (uncorrected errors). Table 15 shows the fixed-effect model for CWIT uncorrected errors, which produced a non-significant fixed effect for this measure of cognitive flexibility on change in RSA. There were also no other significant effects from this model.

Table 15

Fixed Effects Model for Cognitive Flexibility (CWIT Uncorrected Errors)

Model 2E. Estimated Fixed Effects for CWIT Uncorrected Errors as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.764839	.664758	105.548	-1.151	.253	-2.082851	.553174
Sex	.242906	.125818	105.548	1.931	.056	-.006552	.492365
IQ	.002613	.005419	105.548	.482	.631	-.008132	.013358
Trait Anxiety	.004987	.011232	105.548	.444	.658	-.017283	.027258
CogFlex CWIT Uncorrected Errors	.030106	.041132	105.548	.732	.466	-.051446	.111658

Note: Akaike's Information Criterion (AIC)=436.527; Schwarz's Bayesian Criterion (BIC)=448.701

Model 2F. Moderation effect of cognitive flexibility (CWIT uncorrected errors).

While CWIT uncorrected errors did not show significance on its own, adding the interaction between trait anxiety and CWIT uncorrected errors was significant in this model. When comparing the BIC from Model 2E (BIC=448.701) and 2F (BIC=446.998), there is an improvement in model fit when the interaction between trait anxiety and CWIT uncorrected errors is added. This indicates that cognitive flexibility (as measured by the CWIT uncorrected errors) moderates the relationship between trait anxiety and change in RSA.

Table 16

Moderator Model for Cognitive Flexibility (CWIT Uncorrected Errors)

Model 2F. Estimated Fixed Effects for Interaction of TA and CWIT UE as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-1.108118	.671459	101.478	-1.650	.102	-2.440035	.223800
Sex	**.300695***	**.125921**	**101.478**	**2.388**	**.019**	**.050916**	**.550473**
IQ	.004165	.005372	101.478	.775	.440	-.006492	.014822
Trait Anxiety	.010933	.011321	101.478	.966	.336	-.011524	.033391
CogFlex CWIT Uncorrected Errors	.006490	.041818	101.478	.155	.877	-.076460	.089440
TA x CogFlex Uncorrected Errors	**.200999***	**.092966**	**101.478**	**2.162**	**.033**	**.016591**	**.385407**

Note: Akaike's Information Criterion (AIC)=434.851; Schwarz's Bayesian Criterion (BIC)=446.998

Model 2G. All cognitive flexibility measures. To provide a more robust check of the contribution of the cognitive flexibility measures, all of the measures and their interactions with trait anxiety were added to a model together as predictors of change in RSA. According to this model, the demographic variables of sex and IQ were significant. The cognitive flexibility measure of CWIT SS was also significant. Additionally, the interactions of trait anxiety*CWIT SS and trait anxiety*CWIT uncorrected errors produced significant effects, consistent with the previous models examining these variables. This indicates that uncorrected errors are a better predictor of change in RSA than corrected errors.

Table 17

Fixed Effects Model for all Cognitive Flexibility Measures

Model 2G. Estimated Fixed Effects for Interaction of TA and CogFlex as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-1.209620	.670256	94.541	-1.805	.074	-2.540331	.121090
Sex	**.401823***	**.124909**	**94.541**	**3.217**	**.002**	**.153833**	**.649813**
IQ	**.011229***	**.005480**	**94.541**	**2.049**	**.043**	**.000350**	**.022108**
Trait Anxiety (TA)	.015261	.011004	94.541	1.387	.169	-.006585	.037108
CogFlex CWIT SS	**-.078975***	**.024509**	**94.541**	**-3.222**	**.002**	**-.127636**	**-.030315**
CogFlex CWIT Corrected Errors	-.020139	.033715	94.541	-.597	.552	-.087076	.046798
CogFlex CWIT Uncorrected Errors	-.030414	.041444	94.541	-.734	.465	-.112696	.051869
TA x CogFlex CWIT SS	**.167804***	**.058304**	**94.541**	**2.878**	**.005**	**.052050**	**.283559**
TA x CogFlex Corrected Errors	-.082628	.089429	94.541	-.924	.358	-.260178	.094923
TA x CogFlex Uncorrected Errors	**.268688***	**.096519**	**94.541**	**2.784**	**.006**	**.077062**	**.460314**

Note: Akaike's Information Criterion (AIC)=435.866; Schwarz's Bayesian Criterion (BIC)=447.909

Model 3: Inhibitory Control

Model 3A. Inhibitory control (tower test scaled score). Next, a model was fitted to explore the predictive value of inhibitory control on change in RSA. Once again, since the primary aim of RQ2 is to examine the moderation effect of each executive functioning measure

on the relationship between trait anxiety and stress regulation, trait anxiety was also included as a variable in this model. This model produced no significant results.

Table 18

Fixed Effects Model for Inhibitory Control

Model 3A. Estimated Fixed Effects for Inhibitory Control as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.623862	.668388	106.137	-.933	.353	-1.948987	.701263
Sex	.240360	.128382	106.137	1.872	.064	-.014166	.494887
IQ	.002705	.005816	106.137	.465	.643	-.008826	.014235
Trait Anxiety (TA)	.004273	.011232	106.137	.380	.704	-.017996	.026541
Inhibitory Control (IC)	-.008942	.036793	106.137	-.243	.808	-.081886	.064002

Note: Akaike's Information Criterion (AIC)=437.229; Schwarz's Bayesian Criterion (BIC)=449.403

Model 3B. Moderation effect of inhibitory control. Next, the interaction between trait anxiety and inhibitory control was added to the model to assess potential moderation effects. This model did not produce any significant results, indicating that inhibitory control does not moderate the relationship between trait anxiety and stress regulation.

Table 19

Moderator Model for Inhibitory Control

Model 3B. Estimated Fixed Effects for Interaction of TA and IC as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.231472	.697275	107.760	-.332	.741	-1.613626	1.150682
Sex	.195223	.129810	107.760	1.504	.136	-.062091	.452536
IQ	.001532	.005848	107.760	.262	.794	-.010059	.013124
Trait Anxiety (TA)	.000419	.011357	107.760	.037	.971	-.022093	.022931
Inhibitory Control (IC)	-.018972	.037053	107.760	-.512	.610	-.092420	.054476
TA x Inhibitory Control	-.135904	.070002	107.760	-1.941	.055	-.274664	.002857

Note: Akaike's Information Criterion (AIC)=437.229; Schwarz's Bayesian Criterion (BIC)=449.403

Model 4: Working Memory

Model 4A. Working memory (digit span scaled score). Next, a model was fitted to explore the predictive value of working memory on change in RSA. As with the previous models, since the primary aim of RQ2 is to examine the moderation effect of each executive functioning measure on the relationship between trait anxiety and stress regulation, trait anxiety was also included as a variable in this model. This model produced no significant results, although the effect of sex approached significance (p=.051).

Table 20

Fixed Effects Model for Working Memory

Model 4A. Estimated Fixed Effects for Working Memory as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.611527	.669934	105.692	-.913	.363	-1.939781	.716727
Sex	.256786	.130158	105.692	1.973	.051	-.001273	.514845
IQ	.001057	.006527	105.692	.162	.872	-.011883	.013998
Trait Anxiety (TA)	.004001	.011259	105.692	.355	.723	-.018321	.026323
Working Memory (WM)	.007804	.026145	105.692	.298	.766	-.044033	.059640

Note: Akaike's Information Criterion (AIC)=437.879; Schwarz's Bayesian Criterion (BIC)=450.053

Model 4B. Moderation effect of working memory. Next, the interaction between trait anxiety and working memory was added to the model to assess potential moderation effects. This model did not produce significant results for the interaction, indicating that working memory does not moderate the relationship between trait anxiety and stress regulation. There was a significant effect of sex.

Table 21

Moderator Model for Working Memory

Model 4B. Estimated Fixed Effects for Interaction of TA and WM as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.647817	.679692	104.500	-.953	.343	-1.995595	.699962
Sex	**.260369***	**.130937**	**104.500**	**1.989**	**.049**	**.000731**	**.520006**
IQ	.001094	.006547	104.500	.167	.868	-.011889	.014077
Trait Anxiety (TA)	.004883	.011567	104.500	.422	.674	-.018053	.027820
Working Memory (WM)	.008424	.026286	104.500	.320	.749	-.043699	.060547
TA x Working Memory	-.021222	.060203	104.500	-.353	.725	-.140599	.098155

Note: Akaike's Information Criterion (AIC)=441.540; Schwarz's Bayesian Criterion (BIC)=453.688

Research Question 3: How do these relationships differ in adolescents with ADHD compared to typical controls?

To answer RQ3, the final three multilevel models tested the hypothesis that there would be a significant main effect of having an ADHD diagnosis, such that adding "group" to the models would change the predictive value of the executive functioning variables as well as the moderating effects of each variable. However, the fixed effects for group were non-significant in each of the three models examining cognitive flexibility (see Table 22; Model 5), inhibitory control (see Table 23; Model 6), and working memory (see Table 24; Model 7). This indicates that the control group and ADHD group do not differ significantly in terms of how executive functioning, or the interaction of trait anxiety and executive functioning, predicts change in RSA.

Table 22

Moderator Model for Cognitive Flexibility and ADHD

Model 5. Estimated Fixed Effects for Interaction of TA x CF x ADHD as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-1.188314	.671787	93.329	-1.769	.080	-2.522288	.145661
Sex	**.372864***	**.131576**	**93.329**	**2.834**	**.006**	**.111594**	**.634135**
IQ	**.011296***	**.005487**	**93.329**	**2.059**	**.042**	**.000400**	**.022192**
Group	-.095788	.134018	93.329	-.715	.477	-.361908	.170333
Trait Anxiety (TA)	.014575	.011062	93.329	1.318	.191	-.007391	.036540
CogFlex CWIT SS	**-.074934***	**.025174**	**93.329**	**-2.977**	**.004**	**-.124923**	**-.024946**
CogFlex CWIT Corrected Errors	-.011772	.035712	93.329	-.330	.742	-.082686	.059141
CogFlex CWIT Uncorrected Errors	-.027873	.041647	93.329	-.669	.505	-.110571	.054825
TA x CogFlex CWIT SS	**.168089***	**.058378**	**93.329**	**2.879**	**.005**	**.052168**	**.284010**
TA x CogFlex Corrected Errors	-.085530	.089625	93.329	-.954	.342	-.263500	.092440
TA x CogFlex Uncorrected Errors	**.274056***	**.096930**	**93.329**	**2.827**	**.006**	**.081580**	**.466532**

Note: Akaike's Information Criterion (AIC)=437.538; Schwarz's Bayesian Criterion (BIC)=449.554

Table 23

Moderator Model for Inhibitory Control and ADHD

Model 6. Estimated Fixed Effects for Interaction of TA x IC x ADHD as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.221334	.701537	107.202	-.315	.753	-1.612019	1.169351
Sex	.188514	.133175	107.202	1.416	.160	-.075485	.452513
IQ	.001567	.005873	107.202	.267	.790	-.010076	.013209
Trait Anxiety (TA)	.000204	.011428	107.202	.018	.986	-.022451	.022859
Group	-.031142	.138000	107.202	-.226	.822	-.304704	.242420
Inhibitory Control	-.017765	.037665	107.202	-.472	.638	-.092429	.056899
TA x Inhibitory Control	-.131335	.073503	107.202	-1.787	.077	-.277042	.014372

Note: Akaike's Information Criterion (AIC)=439.099; Schwarz's Bayesian Criterion (BIC)=451.221

Table 24

Moderator Model for Working Memory and ADHD

Model 7. Estimated Fixed Effects for Interaction of TA x WM x ADHD as a Predictor of Change in RSA

						95% Confidence Interval	
Parameter	Estimate	*SE*	*df*	*t*	*P*	Lower	Upper
Intercept	-.473361	.697063	103.140	-.679	.499	-1.855799	.909078
Sex	.235778	.132750	103.140	1.776	.079	-.027497	.499052
IQ	-.000271	.006652	103.140	-.041	.968	-.013465	.012922
Trait Anxiety (TA)	.003862	.011592	103.140	.333	.740	-.019127	.026851
Group	-.158871	.144585	103.140	-1.099	.274	-.445616	.127874
Working Memory	.019680	.028171	103.140	.699	.486	-.036188	.075549
TA x Working Memory	-.040518	.062594	103.140	-.647	.519	-.164656	.083621

Note: Akaike's Information Criterion (AIC)=442.364; Schwarz's Bayesian Criterion (BIC)=454.485

CHAPTER 5: DISCUSSION

The current study explored the relationships between trait anxiety, executive functioning, and stress regulation in adolescents with and without ADHD. Specifically, this study aimed to show the moderating effects of three different executive functioning processes on the relationship between trait anxiety and stress regulation. While prior research has linked trait anxiety and stress regulation, trait anxiety and executive functioning, and executive functioning and stress regulation, no existing study was found that provided evidence for a relationship between all three of these factors. Further, previous research has not examined how this relationship may differ in adolescents with and without ADHD. Therefore, results from the present study fill a gap in the literature by contributing to the understanding of how the interaction of executive functioning and trait anxiety influence stress regulation processes in adolescents.

Stress Regulation

Preliminary analyses conducted in the current study provide interesting information regarding differences in stress regulation between adolescents with ADHD and controls. In this study, stress regulation was measured using average RSA assessed at five timepoints, before (RS1), during (MIST1, MIST2, MIST3), and after (RS2) participation in the Montreal Imaging Stress Task (MIST; Dedovic et al., 2005). In these analyses, RSA withdrawal, or change in RSA, was calculated by taking the difference between RSA at the most stressful point of the stress task (MIST3) and baseline RSA.

Descriptive statistics and diagnostic plots generated for average RSA across timepoints indicated that the control group had a higher baseline RSA than the ADHD group, which is consistent with prior studies. However, this was not a statistically significant difference and therefore, it is not expected that this in particular would have a substantial impact on the overall results. In a large meta-analysis, Graziano and Derefinko (2013) pointed out that studies vary in terms of whether or not they control for baseline RSA when examining RSA withdrawal during stress. Baseline RSA was not controlled for in the current analyses, though it was examined in terms of its relationship with RSA withdrawal. Results demonstrated that baseline RSA was significantly associated with RSA withdrawal for both the control group and the ADHD group, with a stronger relationship present in the ADHD group. This finding is to be expected given that RSA withdrawal (change in RSA) is calculated using baseline RSA, however; the greater correlation found in the ADHD group is interesting to discuss. Graziano and Derefinko (2013) noted similar findings indicating a higher association between baseline levels of RSA and RSA withdrawal among clinical samples of children when compared to typical controls. Consistent with their study, results from the current study suggest that RSA withdrawal in the ADHD group is more dependent on their resting-state RSA than it is for healthy controls.

The trajectory of average RSA across timepoints also suggested a more blunted RSA withdrawal in the ADHD group, which indicates that the vagal break is not being withdrawn as well and the stress response system is less regulated. Despite the fact that group did not significantly predict stress regulation in this study, the general RSA trends described above support previous findings that individuals with ADHD demonstrate a more blunted response to stress (Rukmani et al., 2016).

Trait Anxiety and Stress Regulation

After examining descriptive findings regarding the stress response patterns in the sample, the first aim of this study was to establish the direct relationship between trait anxiety and stress regulation. Specifically, it was hypothesized that there would be a significant association between trait anxiety and RSA withdrawal (change in RSA). Results from correlational analyses using the combined sample indicated that trait anxiety was significantly associated with average RSA only during the recovery phase, but not baseline RSA or RSA during the stress task. This means that trait anxiety was positively associated only with recovery from stress, such that as trait anxiety increases, average RSA during the recovery phase also increases. When the groups were examined separately, some unexpected findings were revealed. For the control group, trait anxiety was significantly positively associated with average RSA at all timepoints during and after the stress task, but, surprisingly, not with baseline RSA or RSA withdrawal (change in RSA). For the ADHD group, trait anxiety was not found to be significantly associated with baseline RSA or RSA withdrawal at all. In general, these findings are contrary to previous literature, which has found that higher anxiety is significantly associated with lower baseline RSA and lower stress-induced RSA levels (Beauchaine, 2015; Viana et al., 2017). One explanation for these findings is that neither the control group nor the ADHD group reported elevated levels of trait anxiety to begin with, which likely impacted the ability to detect significant relationships. Additionally, the levels of trait anxiety were similar between the groups.

There were significant differences in the variation of levels of RSA withdrawal between groups, with the control group demonstrating more variability in their RSA withdrawal than the ADHD group. Therefore, the current analyses may not be accurately capturing the relationship

between trait anxiety and stress regulation due to less variation in trait anxiety and more variation in RSA levels between groups. Findings also suggest that since the levels of trait anxiety were so similar between groups, the diagnosis of ADHD may be the driving factor in these analyses rather than trait anxiety.

Moderating Effects of Executive Functioning

The second aim of the study was to examine the moderating effects of cognitive flexibility, inhibitory control, and working memory on the relationship between trait anxiety and stress regulation. Findings from moderation analyses partially supported the hypothesis that executive functioning would moderate the relationship, with results indicating that cognitive flexibility is a significant moderator. However, moderator models analyzing inhibitory control and working memory produced non-significant results, indicating that neither of these executive functions moderate the relationship between trait anxiety and stress regulation. The finding that cognitive flexibility is a significant moderator but not inhibitory control or working memory is important to explore. Since inhibitory and attentional processes are implicated in tasks involving cognitive flexibility (Dajani & Uddin, 2015), it may be that cognitive flexibility served as a more comprehensive measure of executive functioning in this study that also accounted for effects of inhibitory control and working memory. Further, it could be hypothesized that the inhibitory control one would expect to be involved in stress regulation occurs at a more physiological level, rather than a behavioral or cognitive level as measured in this study. Overall, this particular finding contributes to the literature by providing evidence that higher order cognitive processes influence the relationship between trait anxiety and physiological responses to stress.

Group Differences

The third and final aim of the study was to examine potential between-group differences in the relationship between trait anxiety, executive functioning, and stress regulation. Results did not support the hypothesis that group differences would exist, indicating that the control group and the ADHD group did not differ significantly in terms of how executive functioning, or the interaction of trait anxiety and executive functioning, predict RSA withdrawal.

It was hypothesized that all three executive functioning variables would moderate the relationship between trait anxiety and stress regulation, however; only cognitive flexibility demonstrated significant moderating effects. It was also hypothesized that having ADHD would further predict change in RSA, though no group differences were found in this regard. One explanation for the lack of group differences is simple: the groups generally did not differ on any primary variable except for change in RSA and ADHD diagnosis, making it difficult to tease apart significant group effects.

Although previous literature suggests there should be differences in EF performance between groups (Coghill, Seth, & Matthews, 2014; Cepeda, Cepeda, & Kramer, 2000), the ADHD group and the control group did not significantly differ on measures of their executive functioning performance. Both groups performed in the average range on all measures, with the ADHD group performing only slightly lower on measures of cognitive flexibility and working memory. One explanation for this may be that the overall sample used in the current study had higher than average general intelligence, which likely contributed to better overall EF and more heterogenous EF performance between groups. Mahone and colleagues (2002) found that IQ is a significant moderator of the relationship between ADHD and executive functioning, indicating that group differences (ADHD vs. control) in executive functioning performance are less

apparent in groups with higher IQ. Further, in their study, higher IQ was associated with better EF performance in both ADHD and control groups. Their findings, and the current results, suggest that adolescents with ADHD and higher IQ are likely able to compensate for potential executive functioning deficits and perform similarly to their non-ADHD peers on these tasks. Another explanation for this finding is that more than half of the participants in the ADHD group were taking medication for ADHD, which may have also improved their performance and impacted findings.

Practical Implications

Although further analyses are needed to determine the specific influence that cognitive flexibility has on the relationship between trait anxiety and stress regulation, these results suggest that cognitive flexibility may serve as a protective factor for adolescents with high trait anxiety, and provide preliminary evidence that better cognitive flexibility allows for a more adaptive physiological response to stress. Despite that fact that there were no significant differences between adolescents with and without ADHD found in this study in terms of the moderating effect of cognitive flexibility, these findings can contribute to informing assessment and intervention for adolescents in general. Specifically, this study demonstrates the importance of considering cognitive factors such as executive functioning when assessing and treating adolescents who have anxiety symptoms or poor coping skills when dealing with stress.

In regards to assessment, this study provides important information that can assist in the development of testing batteries for children and adolescents who present with a variety of presentations that involve anxiety, ADHD, executive functioning deficits, or increased stress. Results support the consideration of including an evaluation of an adolescent's executive functioning in these cases, since we know that these factors can contribute to stress regulation.

Findings from EF measures administered during a neuropsychological evaluation can undoubtedly yield valuable information that can help inform treatment. For instance, the results of the current study support the notion that adolescents with strong cognitive flexibility may be better able to cognitively reframe stressful situations and better regulate their physiological response to stress. This is consistent with a cognitive behavioral therapeutic framework commonly used in the treatment of anxiety and other psychopathologies. One relevant therapeutic approach in particular is the idea of helping individuals recognize physiological symptoms of anxiety and stress in order to facilitate control over automatic responses. The current study provides support that cognitive flexibility likely contributes to an individual's ability to utilize such therapeutic techniques more effectively.

Limitations and Future Directions

When reviewing the results from the current study, there are several limitations that should be considered. First, this study utilized data from a relatively small sample, which may have resulted in non-significant results that would have approached significance with a larger sample. Though this study used data from a larger study that consisted of a much bigger sample size, several participants were excluded due to missing physiological data collected during the fMRI session. This is important to consider given the potential participant self-selection that may have occurred during recruitment and data collection for the larger study. First, highly anxious or restless children may not agree to participant in such a study, or be unable to tolerate certain parts of the study (i.e. the fMRI stress task). Therefore, although one would expect the clinical ADHD group to demonstrate some differences in their trait anxiety levels or executive functioning performance, the two groups in the current study did not differ significantly in terms of their anxiety or executive functioning, and in fact, were generally within the average range on

all of these measures. Interestingly, six of the 18 participants in the ADHD group also had a previous diagnosis of generalized anxiety disorder, which would be expected to increase the average level of trait anxiety for the ADHD group. However, information regarding the severity of their diagnosed anxiety was not gathered. Therefore, it is likely that only those with less severe symptoms enrolled in the study or agreed to participate in all parts of the study. Having more variability in level of trait anxiety (those reporting both low and high levels of trait anxiety) and executive functioning performance may produce different results. Future studies should examine these relationships in a larger, more diverse sample, which would improve generalizability of the findings.

Additionally, ADHD subtype and symptom severity were not measured, though there is evidence to suggest that levels of trait anxiety, executive functioning performance, and average RSA differ depending on level of symptom severity and whether symptoms are predominantly inattentive or predominantly hyperactive/impulsive. For instance, children with ADHD combined subtype and inattentive subtype are more likely to have higher trait anxiety (González-Castro et al., 2015; Rodríguez et al., 2014), severity of hyperactive-impulsive symptoms of ADHD has been associated with poorer cognitive flexibility (Roberts, Martel, & Nigg, 2017), and overall ADHD severity has been associated with poorer vagal tone (Rukmani et al., 2016). Therefore, obtaining more comprehensive information regarding participants' ADHD symptomatology would be a valuable addition to the data that would allow for further analysis.

Another challenge present in the current study is the difficulty of measuring executive functioning processes by relying on performance on cognitive tasks given in a laboratory setting and measuring stress regulation from data collected through neurophysiological means. First, there may be measurement error due to a variety of tester, participant, and environmental factors

that are difficult to control for, such as inconsistency in test administration across participants, participants' ability or willingness to complete tasks, and technical difficulties with the neuroimaging and physiological monitoring equipment used. Additionally, though change in RSA is a widely accepted indicator of stress regulation, there are limitations to the generalizability of results due to variability in measures of stress used across stress studies (e.g. neuroimaging, assessing hormonal responses, HRV, RSA, etc.). While the current study did not examine the imaging data collected during the fMRI or the saliva cortisol samples gathered, an exploration of neuronal activation and cortisol levels during stress would undoubtedly contribute important findings to the current research questions.

Second, the current study was a secondary analysis that relied on the availability of executive functioning data collected as part of a larger study, meaning that the assessments used were not chosen for the explicit purposes of measuring the constructs examined in the current study. While the assessments used in this study are commonly utilized, they are not the only means by which to measure cognitive flexibility, inhibitory control, and working memory. In general, much like stress regulation, executive functioning studies vary widely in terms of the measures used, which makes it challenging to compare results across studies with dissimilar measurements. For example, executive functioning is also commonly measured in both research and clinical settings using the Behavior Rating Inventory of Executive Function (BRIEF), which is a rating scale of everyday functioning completed by informants such as parents and teachers (Roth, Isquith, & Gioia, 2014). The current study may be expanded by supplementing performance-based measures with rating scales such as the BRIEF, which would allow for interesting comparisons to be made.

Conclusions

While the current study contributes interesting findings to the literature, the relationships between trait anxiety, executive functioning, and stress regulation remain complex and in need of further exploration. This study corroborated results of previous studies in terms of finding differences in baseline average RSA and RSA withdrawal in adolescents with and without ADHD, but also revealed some confounding relationships between trait anxiety and stress response that warrant further study. This study is also the first, to the researcher's knowledge, to find evidence for the role of cognitive flexibility in the relationship between trait anxiety and stress regulation. These results expand on the neurovisceral integration model (Thayer & Lane, 2000) and the attentional control theory (Eysenck et al., 2007) by linking all three constructs in one model. This can have important implications for not only further understanding brain-heart-behavior interactions, but also contributing to assessment and intervention strategies for children and adolescents.

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