

2011

Relationships among the Behavioral Inhibition System, response inhibition, heart rate variability, and anxiety sensitivity between older adolescents with and without significant anxiety

Erin Tarcza Reuther

Louisiana State University and Agricultural and Mechanical College

Follow this and additional works at: https://digitalcommons.lsu.edu/gradschool_dissertations



Part of the [Psychology Commons](#)

Recommended Citation

Reuther, Erin Tarcza, "Relationships among the Behavioral Inhibition System, response inhibition, heart rate variability, and anxiety sensitivity between older adolescents with and without significant anxiety" (2011). *LSU Doctoral Dissertations*. 3746.

https://digitalcommons.lsu.edu/gradschool_dissertations/3746

This Dissertation is brought to you for free and open access by the Graduate School at LSU Digital Commons. It has been accepted for inclusion in LSU Doctoral Dissertations by an authorized graduate school editor of LSU Digital Commons. For more information, please contact gradetd@lsu.edu.

RELATIONSHIPS AMONG THE BEHAVIORAL INHIBITION SYSTEM, RESPONSE
INHIBITION, HEART RATE VARIABILITY, AND ANXIETY SENSITIVITY BETWEEN
OLDER ADOLESCENTS WITH AND WITHOUT SIGNIFICANT ANXIETY

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Psychology

by

Erin T. Reuther
B.S., University of Florida, 2004
M.A., Southeastern Louisiana University, 2006
August 2011

ACKNOWLEDGEMENTS

Many thanks are due to my advisor and committee chair, Dr. Thompson E. Davis III for his assistance in guiding me through this project and my graduate career. I would also like to extend thanks to my committee members, Dr. Johnny Matson, Dr. Emily Elliott, Dr. Julia Buckner, Dr. Russell Matthews, and Dr. Stacia Haynie, whose time and thoughtful suggestions about the project are greatly appreciated.

I would also like to thank my parents for their constant love and support; they are the root of and continue to serve as the bar for my accomplishments and ambition. And finally, I would not be where I am today without the encouragement and support of my husband, Stephen. He has been the foundation around which I have grown for the past decade, and I look forward to our continued growth in the future.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS	ii
LIST OF TABLES	iv
LIST OF FIGURES	v
ABSTRACT	vi
INTRODUCTION	1
METHOD	28
RESULTS	34
DISCUSSION	41
REFERENCES	57
APPENDIX: TABLE OF DESCRIPTIVES BY GENDER	66
VITA	67

LIST OF TABLES

Table 1. Descriptive Statistics for Primary Dependent Variables by Group.....	37
Table 2. Bivariate Correlations between Dependent Variables.....	39
Table 3. Regression Table of Indexes of Inhibition Predicting ASI Total Score.....	40
Table 4. Regression Table of ASI Subscales Predicting BIS Scale.....	40
Table A1. Descriptive Statistics for Dependent Variables by Gender.....	66

LIST OF FIGURES

Figure 1. Stimulus Presentation of the Day/Night Stroop Task.....31

ABSTRACT

Differences in the Behavioral Inhibition System (BIS; Gray, 1982), response inhibition, and cardiac vagal control are evident between individuals with anxiety and nonclinical control participants. In this study, the role of inhibition in anxiety was examined, as well as relationships between the primary indexes of inhibition including the behavioral inhibition system (BIS), response inhibition, and cardiac vagal tone (or heart rate variability) at rest and during tasks of response inhibition. Additionally, anxiety sensitivity, an established risk factor for developing anxiety disorders, was examined and how it relates to indexes of inhibition. As expected, inhibition and anxiety sensitivity were found to be related symptoms of anxiety in an older adolescent sample. Indexes of inhibition were found to be related to each other; however, several relationships were not statistically supported. Finally, inhibition was found to be a related and likely an important factor in anxiety sensitivity. These findings extend research in the area of anxiety risk factors and neurophysiology of anxiety and have implications for informing etiological models of anxiety.

INTRODUCTION

Anxiety disorders are the most common class of psychological dysfunction, with lifetime prevalence rates of up to 28.8% for anxiety disorders in adults and prevalence rates of 10% to 20% in children and adolescents (Albano, Chorpita, & Barlow, 2003; American Psychiatric Association, 2000; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Kashani, Orvaschel, Rosenberg, & Reid, 1989; Kessler et al., 2005). Much of the recent literature on anxiety has begun to focus on etiological and maintaining factors for anxiety disorders, and the Behavioral Inhibition System (BIS) of the brain has been repeatedly implicated in both adult and child anxiety problems (Gray 1982; Gray & McNaughton, 2000). Also implicated in anxiety, but less understood, are problems with response inhibition and differences in cardiac vagal control. Finally, it is possible that these variables are also implicated in anxiety sensitivity, a related construct to anxiety. In this dissertation, the impact of the BIS, response inhibition, and cardiac vagal control are examined regarding their respective roles in anxiety in an older adolescent population. Additionally, relationships among these variables are examined, as well as possible impacts on anxiety sensitivity.

Introduction to the Behavioral Inhibition and Activation Systems (BIS/BAS)

The BIS and Behavioral Activation System (BAS) were first introduced by Gray (1982, 1983). The BIS and BAS are separate neural networks that control different aspects of human behavior. The BIS aids in responses to three classes of stimuli—1) signals for punishment, 2) signals for non-reward, and 3) novel situations (Gray, 1982). Thus, BIS is thought to be involved in the processing of situations that are novel and have potential for harm. When BIS is processing, it is also thought to cause increased anxiety due to the nature of the information. The BAS is thought to mediate approach responses to appetitive stimuli. Also, Gray conceptualized

the fight/flight system as a third independent system which controls behavior during immediate fear and panic responses.

Gray and McNaughton (2000) have since revamped Gray's (1982) theory in light of advances in research since its inception. In this revision, the BIS was delineated as operating parallel to and separately from both the fight-flight-freeze system (FFFS), which controls fear reactions to present and immediate danger, and the BAS, which controls both conditioned behavior and inborn personality tendencies to approach potential rewards (Gray & McNaughton; Pickering & Corr, 2008). Thus, BIS would be more involved with anxiety, or worry, and the FFFS would be involved in fear or panic. Although the BIS was originally proposed to function in avoidance behaviors only, in Gray and McNaughton's revision, the primary function of the BIS was conceptualized to be conflict resolution. Conflict resolution in the BIS includes resolution of BAS-avoidance conflicts and FFFS-avoidance conflicts as well as conflicts within these systems (e.g., choosing between two options of approach in the BAS). The BIS is thought to resolve these conflicts by processing negative information about the conflict through "recursive" loops involving several higher and lower structures in the brain until the person arrives at a resolution (Pickering & Corr). When the BIS is active in a conflict resolution process, it also translates into a state of anxiety in the individual.

While both frontal and lower order brain structures are included in the BIS, the limbic structures of the septo-hippocampal system and the subiculum have the strongest evidence of support for operating as part of this system (Degroot & Treit, 2004; McNaughton, 2006). Some of the first evidence that the septo-hippocampal system was involved in the BIS included findings that anxiolytic medications seemed to have mechanisms that worked primarily on noradrenergic, serotonergic, and GABAergic neurons in these regions (Degroot & Treit; Gray,

1983; Gray, 1988; Gray & McNaughton, 2000; Leslie, Shaw, McCabe, Reynolds, & Dawson, 2004; McNaughton & Gray, 2000). Continued animal research in the field has shown that lesions of the hippocampus and administration of both older and newer anxiolytic medications have similar effects (see McNaughton & Gray for a review). The involvement of the hippocampus in the BIS is also congruent with memory literature in that, rather than storing long-term memories, the hippocampus aids in the processing of information which then may or may not become long-term memories (McNaughton & Gray). More recently, brain imaging studies have shown that adults who score highly on self-report measures of BIS also have larger hippocampal volume (Cherbuin et al., 2008).

The subiculum, another proposed part of the BIS, is thought to interact with the septo-hippocampal region to aid in conflict resolution. The hippocampus first receives information about the conflict and sorts it according to important and non-important information. The important aspects of the conflict are then processed by the subiculum where the negative aspects of the conflict are magnified. The subiculum further processes the information by comparing and integrating possible solutions, eventually leading to behavioral output to resolve the conflict (see McNaughton, 2006 for a review). The BIS is also said to be a “recursive” process because information may loop through several systems within the subiculum before behavioral output is reached (McNaughton). Additionally, the septo-hippocampal area and subiculum are thought to be involved in resolving current goal conflicts only. Higher processing of goal conflicts, such as ordering subgoals within a sequence and multi-step planning of conflict resolution, are thought to be mediated by the prefrontal cortex (McNaughton & Gray, 2000).

Chronic overactivation of the BIS, or BIS sensitivity as a personality trait, is typically measured by the self-report BIS/BAS Scales (Carver & White, 1994). Higher scores on this self-

report measure have been shown to predict more brain activity in areas of the brain associated with the BIS. Following this research, the BIS/BAS scales have generally been considered the gold standard for assessing BIS given their accuracy and their practicality. Physiologically, BIS has been indexed by several different measurements including theta waves in electroencephalography (EEG) and mismatch negativity in EEG (Hansenne et al., 2003; McNaughton & Gray). According to Fowles' (1980) three arousal model measurements of heart rate index BAS activity, whereas electrodermal activity indexes BIS activation. This theory has accumulated support, as anxiolytic medication reduced anxiety during an impromptu speech task as measured by skin conductance, but not by self-reported anxiety or heart rate in a sample of undergraduate male students (Landon, Sher, & Shah, 1993).

BIS and Anxiety

The BIS has been implicated in the etiology and maintenance of anxiety disorders and is thought to cause many other associated difficulties in individuals with anxiety. According to Chorpita and Barlow's (1998) behavioral inhibition model of anxiety, children who experience a low sense of control over their environment (e.g., overprotectiveness by the parents, household that has less warmth or sensitivity) will be more likely to develop overactivation of the BIS, thus predisposing them to developing a behaviorally inhibited temperament as well as anxiety disorders. Behaviorally inhibited temperament describes a behavioral pattern in early childhood of being withdrawn and less willing to seek out new experiences in the environment (Albano et al., 2003). This temperament has been related to Gray's (1982) theory of the BIS, and chronic overactivation of this system is thought to result in clinically significant anxiety disorders (Gray & McNaughton, 2000). Behaviorally inhibited temperament has been conceptualized as a heritable trait which predisposes an individual to developing anxiety disorders (Barlow, 2002).

Evidence of BIS in Anxious Adults

This conceptualization of BIS potentiating anxiety has been supported by a wealth of literature demonstrating more self-reported BIS sensitivity in individuals with anxious personality styles, as well as in adults and children with anxiety disorders. In a sample of undergraduate students, higher scores on the BIS scale of the self-report BIS/BAS scales (Carver & White, 1994) predicted both anxious and depressed symptoms (Kimbrel, Nelson-Gray, & Mitchell, 2007). Additionally, it was found that BIS mediated the relationship between maternal overprotectiveness predicting anxiety as an adult (Kimbrel et al.). In another study of undergraduate students, anxiety was differentiated from depression by individuals with anxiety showing more BIS sensitivity, and individuals with depression showing less BAS sensitivity in addition to more BIS sensitivity, particularly for symptoms of anhedonia (Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapil, 2007). In a confirmatory factor analytic approach with young adult participants, BIS factors were found to share a positive relationship with measures of neuroticism and a negative relationship with measures of extraversion (Gomez & Gomez, 2005).

BIS has also been found to show convergent validity with scales of harm avoidance and sensitivity to punishment in undergraduates (Caseras, Àvila, & Torrubia, 2003). Undergraduates' scores on the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) scales of neuroticism and negative affect have been found to have significant, positive relationships with BIS scores, while BAS scores have positive and significant relationships with impulsivity, positive affect, disinhibition, extraversion, and anger (Segarra et al., 2007). The MMPI-2 constraint scale was also a positive predictor of BIS sensitivity (Segarra et al.). BIS was found in conjunction with a factor of perfectionism to predict the amount of worry in college students (Chang et al., 2007).

Although most research has been done under the older model of BIS/BAS systems, the updated (Gray & McNaughton, 2003) model which more clearly delineates fear and anxiety has received support as well. When a sample of young adults completed self-report measures of BIS sensitivity, trait anxiety, and tissue damage fear, confirmatory factor analysis revealed that anxiety and BIS sensitivity were related and were both distinct from tissue damage fear (Cooper, Perkins, & Corr, 2007). This lends support to Gray and McNaughton's (2000) distinction of separate systems for anxiety and fear (Cooper et al.).

Relations between BIS and anxiety have also been measured through tasks which measure sensitivity to reward and punishment, processing of immediate and delayed reward and punishment, and processing of negatively valenced emotional stimuli. In a study examining the effects of reward and punishment on inhibition and mood, undergraduate students were divided into one of two groups—punishment or reward—to perform an instrumental learning task (Gomez & McLaren, 1997). In the punishment group, participants lost a small amount of money for each wrong response and were not rewarded for correct responses. In the reward group, participants earned a small amount of money for each correct response and were not punished for incorrect responses. Authors found that participants in the punishment group made fewer impulsive decisions and reported being more nervous and less happy than participants in the reward group. Additionally, participants in the punishment group had higher skin conductance, indexing BIS activity, but did not have higher heart rate, indexing a lack of BAS activity, than the reward group (Gomez & McLaren). This indicates that when individuals are responding to punishment signals rather than rewards, there is greater activation of the BIS. Similarly, another sample of female undergraduate students' performance was measured on a counter-conditioning task in which participants were attempting to earn the highest number of points. One response

caused an immediate small gain of points, while another response caused an immediate small loss of points but also caused a very large gain of points if the first response was given before it (Avila, Parcet, Ortet, & Ibáñez-Ribes, 1999). Thus the way to earn the maximum number of points was to alternate button pushes such that the individual would lose a small number of points with every other response but would gain a very large number of points every other response. Participants who scored lower on a self-report sensitivity to punishment scale, indexing lower BIS scores, learned the counter-conditions faster and attained higher scores on the task (Avila et al.). This indicates that because individuals high in BIS and with a high sensitivity to punishment avoid cues of punishment, they may also be slower to learn in situations in which aversive stimuli lead to later rewards.

BIS activity has also been examined in the processing of emotional stimuli. In one such study, adult participants completed self-report measures of BIS and BAS sensitivity, impulsivity, anxiety, and current positive or negative mood (Gomez & Gomez, 2002). They then completed tasks which required processing of negative, neutral, and positive information. Congruent with Gray's BIS/BAS theory, BIS sensitivity and anxiety were associated with better processing of negative information while BAS sensitivity and impulsivity were associated with better processing of positive information (Gomez & Gomez). Likewise, healthy adults who scored high on BIS sensitivity reported more negative affect during the aversive tasks, while individuals who scored high on BAS sensitivity reported more positive affect during an appetitive task (Heponiemi, Keltikangas-Järvinen, Puttonen, & Ravaja, 2003).

There has also been evidence that higher sensitivity to BIS is evident in adults with clinically significant anxiety. A gene has been identified which has been shown to affect both BIS activity and emotional processing (Montag et al., 2008). In a healthy sample of adult

women, a gene encoding for the dopamine catabolic enzyme catechol-O-methyltransferase (COMT Val158Met) was shown to significantly amplify acoustic affective startle reflex. Higher scores on a measure of BIS sensitivity were also found to be associated with a larger startle reflex (Montag et al.). Interestingly, although individuals with clinically significant social anxiety have high sensitivity to BIS, they respond in a similar way on an emotional Stroop task as individuals who are high in BAS (Putman, Hermans, & van Honk, 2004). In emotional Stroop tasks, individuals are asked to identify colors shown over neutral faces and colors shown over happy or angry faces. Vigilance to angry faces are typically found to index BAS sensitivity rather than BIS; however, because socially phobic individuals view angry faces as potentially threatening information, they respond similarly to individuals who are high in BAS sensitivity, showing greater vigilance to angry faces (Putman et al.). Further, although both BIS and BAS are found to be active during *in vivo* exposure to feared stimuli, differences have been found in virtual reality exposures (Willhelm et al., 2005). When young adults with and without a significant fear of heights were exposed to a virtual reality elevator simulation, individuals with extreme fears showed heightened electrodermal responses, indicating BIS activity, but few heart rate differences, which would indicate BAS activity. Individuals with little or no fear showed few responses in either electrodermal or heart rate recordings (Willhelm et al.). Authors concluded that because virtual reality simulation is not as realistic as *in vivo* exposure, it indexes BIS activity only, even in fearful individuals (Willhelm et al.).

Research on the BIS has yielded important information aiding in the understanding of how this system influences anxiety and behavior. First, the BIS includes both higher and lower structures in the brain, with higher processes being controlled by the prefrontal cortex, and lower processes being controlled by the septo-hippocampal area and the subiculum (Degroot & Treit,

2004; McNaughton, 2006). Next, higher BIS sensitivity has been associated with anxious, neurotic, and introverted personality styles in adults. Adults with higher BIS sensitivity have also been found to be more sensitive to punishment cues in tasks, evidenced by a more pronounced negative mood state during aversive tasks and being more avoidant of small punishments even if it causes one to gain greater rewards later (Avila et al., 1999).

Differences in BIS processing among children and adults have also been found, including differences in EEG recordings at rest in children and adults with high BIS sensitivity (Knyazev & Slobodskaya, 2003). Children high in BIS sensitivity also show differences in the processing of negative information, showing more attentional bias to potentially threatening information, which causes them to be more likely to develop fear and avoidance of objects and situations (Field, 2006). This suggests that BIS may be involved in anxiety throughout development and that it may be useful to examine its involvement in anxiety at different stages throughout development.

Response Inhibition and Anxiety

Response inhibition is another factor which has been proposed to maintain anxiety and avoidant behavior. Response inhibition is the ability to suppress a dominant or automatic response. The BIS is thought to be involved in this process because it requires an individual to suppress conflicting stimuli while attending vigilantly to the task at hand (Casey, Tottenham, & Fossella, 2002). Response inhibition is also considered an executive function because it requires proper ordering of events to achieve optimal performance on a novel task (Diamond & Taylor, 1996). Supporting this, research has shown that the frontal lobes are involved in tasks requiring response inhibition, and individuals high in BIS sensitivity have more prominent responses in areas of the brain in which the BIS is present (Amodio, Master, Yee, & Taylor, 2008; Mitchell,

Rhodes, Pine, & Blair, 2008). Literature on how or whether it is involved in the maintenance of anxiety has been mixed with both adults and children, with some studies showing individuals with anxiety have worse response inhibition, some showing no differences in response inhibition, and some studies suggesting that individuals with anxiety should have better response inhibition. Furthermore, like BIS, differences have been found between children and adults in response inhibition abilities, indicating that the relationship between response inhibition and anxiety may change throughout development.

Several studies have supported involvement of the BIS in response inhibition and examined areas of the brain required for this ability. Response inhibition is generally measured by having participants complete a task that requires response inhibition, such as a Stroop task or Go/No-go task, or a planning task that requires response inhibition, such as the Tower of Hanoi task or the Tower of London task. In response inhibition tasks, individuals are asked to inhibit a pre-potent response. See the method section for a more detailed description of two response inhibition tasks used in the current study. Participants' accuracy on tasks and response time on tasks are common dependent measures of response inhibition. In one such examination, a sample of undergraduate students completed self-report measures of BIS/BAS sensitivities as well as a Go/No-go task requiring response inhibition while EEG was recorded (Amodio et al., 2008). Individuals higher in BIS had N2 signals of higher amplitudes during the No-go trials indicating specific responding to conflict monitoring and inhibition cues. Additionally, brain activity during the No-go trials was found in areas of the brain connecting areas of the limbic system with areas of the prefrontal cortex, indicating that the prefrontal cortex plays an important role in response inhibition (Amodio et al.).

Evidence has also been found for differences in response inhibition among individuals with anxiety disorders. In a comparison of women with histories of trauma, groups were found to differ in performance on several executive functioning tasks (Stein, Kennedy, & Twamley, 2002). The groups comprised women who had suffered intimate partner violence and developed posttraumatic stress disorder (PTSD), women who had suffered intimate partner violence but had not developed PTSD, and women with no significant trauma history. Women who had suffered from intimate partner violence were found to perform worse on several measures of executive functioning, including a Stroop task, which requires response inhibition (Stein et al.). However, no significant differences were found in response inhibition among the groups of women who had suffered trauma (Stein et al.).

Similarly, a group of Vietnam veterans with PTSD demonstrated EEG differences during a response inhibition task compared to nonclinical civilians (Shucard, McCabe, & Szymanski, 2008). The group of veterans had longer P3 latency in the frontal region of the brain during No-go trials, and they had greater amplitude in the frontal P3 area to irrelevant stimuli during the task. These differences were also more pronounced in those veterans who had more hyperarousal symptoms of PTSD. Authors suggested that differences in response inhibition and slowed processing in the frontal lobes may be what contributed to attention and concentration problems in individuals with PTSD (Shucard et al.).

The BIS has been proposed to influence response inhibition in adults who are high in either BIS or impulsivity while completing a Go/No-go discrimination and response inhibition task (Knyazev, Levin, & Savostyanov, 2008). During the discrimination task, individuals high in BIS were found to have higher alpha power in their EEG recordings, indicating brain differences in how these individuals respond to a task requiring response inhibition (Knyazev et al.). Also,

Hagopian and Ollendick (1994) examined response inhibition in college students who scored either very high or very low on a measure of test anxiety while performing a Go/No-go discrimination task; however, participants were assigned to either the punishment condition, in which they lost points for incorrect responses, or the non-reward condition, in which their score remained the same for incorrect responses. Both groups were given additional points for correct responses. Participants who were highly test anxious were found to have more inhibited response tendencies, meaning that they responded less frequently to ambiguous stimuli, made more errors of omission, and made fewer errors of commission (Hagopian & Ollendick). Additionally, the highly test anxious group reported experiencing more anxiety before and during the test on a measure of state anxiety (Hagopian & Ollendick). This illustrates differences in response inhibition among adults with and without anxiety. In addition, these findings highlight the importance of examining response tendencies during task performance rather than simply examining the number of correct responses.

Response Inhibition and Obsessive-Compulsive Disorder

Much of the recent literature on response inhibition in anxiety has come from the area of obsessive-compulsive disorder (OCD). In a review of the literature, it was concluded that there was sufficient evidence to support that problems in response inhibition were evident in adults with OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). This was conceptualized as being due to an inherited dysfunction in the lateral orbitofrontal loop which causes problems with response inhibition and other executive functions in OCD. It was also suggested that response inhibition may be an endophenotype for OCD (Chamberlain et al.). Endophenotypes are heritable, observable traits that are found in individuals with a particular disorder. To meet the definition of an endophenotype, a trait must also be present in individuals

both during a current diagnosis and when they do not meet diagnostic criteria, and it must be present in non-clinical family members of an individual with the disorder (Gottesman & Gould, 2003). Following this, additional evidence has been found which supports response inhibition as an endophenotype. Deficits in a task requiring response inhibition were found for “recovered” individuals with OCD compared to healthy controls (Rao, Reddy, Kumar, Kandavel, & Chandrashekar, 2008). Also, when compared to nonrelated matched control participants, both individuals with OCD and their first-degree relatives demonstrated impaired performance and delayed responding on a Stop-Signal task requiring response inhibition (Menzies et al., 2007). Those individuals demonstrating impaired performance on the task were also found to have more gray matter in the orbitofrontal and right inferior frontal regions of the brain, and less gray matter in the cingulate, parietal, and striatal regions of the brain according to MRI scans (Menzies et al.). Because these impairments were found in individuals without anxiety but those who had a family history of anxiety, this further supports response inhibition as a potential endophenotype for OCD.

Additional evidence has also been found for impairments in response inhibition in OCD since Chamberlain’s (2005) review. Compared to a group of healthy control participants, individuals with OCD were found to have less activation in the frontal cortex during a Go/No-go task as examined in fMRI scans (Roth et al., 2007). Response inhibition impairments in individuals with OCD are also seen in EEG recordings, with the N2 being particularly sensitive to No-go trials of a Go/No-go task (Kim, Kim, Yoo, & Kwon, 2007). This has been replicated when comparing individuals with OCD to nonclinical controls, and this pattern is correlated with OCD symptom severity (Herrmann, Jacob, Unterecker, & Fallgatter, 2003). A sample of adult OCD patients were found to display weaknesses on three different response inhibition tasks

including the Go/No-go task, the Stop paradigm, and a motor Stroop paradigm compared to healthy controls (Penadés et al., 2007). Moreover, when groups of individuals with symptomatic OCD, remitted OCD, panic disorder, or no diagnosis completed an inhibition task which involved threat information, participants with symptomatic and remitted OCD both displayed impairments when compared to the panic group and the nonclinical control group (Bannon, Gonsalvez, & Croft, 2008). When threat information was added to the paradigm, individuals with either symptomatic OCD or panic disorder demonstrated impairments in inhibition (Bannon et al., 2008). Also, while comorbid conditions were found to account for some problems in executive functioning in adults with OCD, response inhibition was robust even when comorbid conditions were accounted for (Aycicegi, Dinn, Harris, & Erkmén, 2003). Individuals with OCD were also found to display a different response pattern in a Go/No-go response inhibition task compared to individuals with panic disorder, with OCD participants making more errors of commission, more errors overall, and having longer reaction times overall (Bannon, Gonsalvez, Croft, & Boyce, 2002).

Some studies have also examined how symptom categories of OCD might affect response inhibition. Individuals with OCD who had washing and checking compulsions were found to perform worse on a task of response inhibition than individuals with social phobia or nonclinical control participants (van der Linden, Ceschi, Zermatten, Dunker, & Perroud, 2005). Most of the clinical groups were receiving therapy or medication at the time of the study (van der Linden et al.). Additionally, in a non-treatment seeking sample, individuals with autogenous obsessions—those obsessions that are not generally triggered by external stimuli, are ego-dystonic with a person's values, and are perceived as extremely aversive (Lee & Kwon, 2003)—were found to have more problems with response inhibition compared to individuals with reactive obsessions

and individuals with subclinical OCD symptoms (Lee, Yost, & Telch, 2009). Also, when a sample of OCD individuals with hoarding symptoms was compared to a clinical control group consisting of individuals with other anxiety or depression and a nonclinical control group, the individuals who hoarded were found to have more problems with response inhibition as well as more problems with other executive functions, indicating that individuals with hoarding tendencies may have more problems with response inhibition (Grisham, Brown, Savage, Steketee, & Barlow, 2007).

Disparate Findings in Response Inhibition and Anxiety

Oddly, there have also been findings in the extant literature which have found no differences in response inhibition among individuals with anxiety or OCD, and some authors have even suggested that anxious individuals should perform better on tasks that require response inhibition (Price & Mohlman, 2007; Schmidtke, Schorb, Winkelmann, & Hohagen, 1998). When a sample of non-medicated patients with OCD were compared to a group of nonclinical control participants on a battery of neuropsychological tests, patients with OCD were not found to have more errors on executive functioning tests, including the Tower of Hanoi and a concept formation task (Schmidtke et al.). Furthermore, patients with OCD were not found to make more mistakes on a task requiring the executive functioning skills of set shifting or a Stroop task requiring response inhibition (Schmidtke et al.). However, certain patterns of responding in the OCD group may have masked potential differences. As found in Hagopian and Ollendick (1994), one would expect anxious individuals to have longer latencies, make more errors of omission, and make fewer errors of commission. In the study on OCD patients, the clinical group was found to take significantly longer to complete the Stroop task and the set shifting task (Schmidtke et al.). Additionally, there was no time constraint on the tasks of

executive functioning which may have masked differences between the groups (Schmidtke et al.). In a group of non-medicated older adults with generalized anxiety disorder (GAD), the clinical group was not found to significantly differ in performance on a color-word Stroop task from a group of age-matched nonclinical participants (Price & Mohlman). However, again, the score on the Stroop task was measured by subtracting the total errors from the total number of items completed only, without examining the pattern of responses. Additionally, within the clinical group with GAD, scores on measures of trait anxiety and worry were found to share a significant positive correlation with Stroop performance; however, this relationship was not found in the nonclinical control group (Price & Mohlman). These studies emphasize the importance of examining the pattern of responses in tasks that require response inhibition rather than the overall score. Additionally, some studies show that even when patients are taking medication, the effects of response inhibition are sometimes still robust, although according to Gray (1982) and Gray and McNaughton (2000), anxiolytic medications should reduce effects on the BIS in individuals with anxiety.

Summary of Response Inhibition Effects in Anxiety

Response inhibition is an executive function which has been shown to tap the frontal cortex and many areas also implicated in the BIS. For this reason, it is suspected that response inhibition may be a behavioral measure that is a manifestation of problems in the BIS and brain areas affected in anxiety. Additionally, both adults and children with high trait anxiety and anxiety disorders, including PTSD, test anxiety, panic disorder, and OCD have been found to have impairments manifested as differential response patterns compared to nonclinical controls. However, measurement of response inhibition in anxiety has proved difficult thus far. Many different tasks have been used to index response inhibition, including Go/No-go tasks, Stop-

tasks, and aversive conditioning tasks, among others. The complexity of some these tasks has made it difficult to determine patterns of responding, which may cloud interpretations of response inhibition ability when performance is scored in a simple percent correct manner (Hagopian & Ollendick, 1994). Use of simple and straightforward tasks of response inhibition may aid in clarifying some of these potential confounds which interfere with appropriate interpretation.

Cardiac Vagal Control in Anxiety

A physiological measure important in anxiety is cardiac vagal control. Much of the connection between cardiac vagal control and anxiety comes from the polyvagal theory and dynamic systems models (Friedman, 2007; Porges, 2007). Polyvagal theory describes how the mammalian autonomic nervous system (ANS) controls behavioral responses to the environment (Porges). According to polyvagal theory, there are multiple inputs of the vagus nerve on the heart, which are phylogenetically ordered. These include the myelinated vagus, which controls behaviors of social communication, self-soothing, and inhibition of sympathetic arousal; the sympathetic adrenal system, which controls more basic fight/flight responses; and the unmyelinated vagus, which controls even more basic passive responding such as feigning death. The two vagi are held to have different source nuclei in the brainstem; the myelinated vagus originates in the *nucleus ambiguus*, and the unmyelinated vagus emerges from the *dorsal motor nucleus*. These influences are phylogenetically ordered responses, such that the higher ordered responses inhibit the lower. Through the process of *neuroception*, higher order brain functions control vagal influences and adapt behavior according to the situation in the present environment. For example, if a situation cannot be resolved by the higher responses mediated by the myelinated vagus, the brain would decrease the parasympathetic influence on the heart and

allow the sympathetic system to activate so that the organism can address the situation appropriately. Additionally, if a more evolved system fails for some reason, the organism will move to the next response, and so forth. This system is said to be dynamic in that it must be able to change according to environmental conditions.

The highest ordered response, mediated by the myelinated vagus in the parasympathetic nervous system, is termed the “vagal brake” and has been tied to human primary emotions including love, fear, and socialization (Porges, 2007). One reason it is termed the “vagal brake” is because in its functioning, the parasympathetic system inhibits the sympathetic fight/flight response and slows the heart rate. Furthermore, this parasympathetic influence is conceptualized as a flexible and dynamic system with the ANS adjusting the amount of influence of the myelinated vagus more or less depending on the situation (Friedman, 2007). Thus, healthy individuals would be able to easily increase or decrease the amount of vagal influence adaptively from situation to situation.

If this response system were compromised, however, one would expect increased activity in the fight/flight response mediated by the sympathetic nervous system and increased activity in the HPA axis, both of which have been observed in anxiety disorders (Friedman, 2007). Anxiety disorders are thought to be marked by “bad brakes,” meaning that these individuals are less able to inhibit the sympathetic influences on heart rate via the myelinated vagus (Friedman). Individuals with anxiety disorders are also thought to have less dynamic vagal modulation, and so are less adaptive to changing situations, an idea that has received support in the literature (see Friedman, 2007, for a review of the topic). Likewise, problems with the vagal brake have been found in emotion regulation and children with conduct disorders (Beauchaine, Gatzke-Kopp, & Mead, 2007; Porges, 2003, 2007).

Physiologically, the influence of the myelinated vagus on the heart can be observed through several indexes that measure high frequency heart rate variability (HRV), including spectral analysis, respiratory sinus arrhythmia (RSA), or mean of squared successive differences (MSSD). Very high correlations (order of magnitude: 0.8-0.9 or higher) have been reported among various vagal HRV measures indicating that they should be comparable across studies (Beauchaine, 2001; Friedman, Allen, Christie, & Santucci, 2002; Grossman, van Beek, & Wientjes, 1990; Hayano et al., 1991), although controversy exists over their respective validities as cardiac vagal tone indexes (Porges, 2007). The influence of the myelinated vagus in healthy individuals results in a pattern in which heart rate increases during inspiration and decreases during exhalation (Miyawaki & Salzman, 1991). When the influence of the parasympathetic system on autonomic functioning decreases, heart rate becomes more rhythmically uniform and HRV decreases as the sympathetic system takes over. RSA is calculated by a variety of methods that assess heart rate variability, or differences between inter-beat intervals, within the frequency band of normal respiration (Allen, Chambers, & Towers, 2007). Problems with the vagal brake in anxiety are typically observed as low RSA at rest, indicating less cardiac vagal control, and as smaller changes in RSA during tasks that require effort, indicating more static cardiac vagal control and a decreased ability to modulate the control according to the situation (Porges, 2007). RSA has also been found to be a reliable and stable measure at rest, during a variety of cognitively and emotionally challenging tasks, across time for individuals, and across different age ranges including infancy, preschool, school-age, and adolescence (Alkon et al., 2003; Bornstein & Suess, 2000; El-Sheikh, 2005; Fracasso, Porges, Lamb, & Rosenberg, 1994; Kuznetsova & Son'kin, 2008; Leitch & Allen, 2008; Salomon, 2005; Suess, Porges, & Plude, 1994). Additionally, movements, such as small hand gestures often required for cognitive tasks,

have not been found to significantly affect measurements of RSA (Porges et al., 2007).

Moreover, RSA has been widely employed in studies of both adult and child anxiety.

In a recent review of polyvagal and autonomic flexibility models in adult anxiety, authors found clear and robust supporting evidence across studies of both nonclinical adults and adults with anxiety that were congruent with these predominant theories (Friedman, 2007). Cardiac vagal tone was found to correlate with general anxiety symptoms and has an inverse relationship with negative mood and thought suppression. Evidence for differences in cardiac vagal control has also been found across several anxiety disorders including panic disorder, PTSD, GAD, and specific phobia. These typically include finding less cardiac vagal tone at rest and fewer changes in cardiac vagal tone during stressful tasks. The author also examined four studies on childhood anxiety disorders and similar results were found (Friedman). Additionally, trait anxiety was suggested as a moderator of the relationship between cardiac vagal control and depression in a review on the topic (Rottenberg, 2007).

In polyvagal theory, Porges (2003, 2007) has explained how problems with cardiac vagal control can affect the social engagement system in humans. Influences of the parasympathetic system determine the range of emotion and socialization available to utilize at any given point in time. In this way appropriate social engagement is dependent on the parasympathetic system suppressing influences of the sympathetic system. This relationship exists because the myelinated vagus projects to the heart and facial muscles, both of which are engaged in expressing emotion and producing social cues. These expressions can include relations of empathy or reciprocity for shared experiences, among others. Unless an individual has adequate vagal influence, he or she will not be able to access the cognitive and social resources required. The social engagement system has been fairly well-researched in regard to infant emotional

development and cardiac vagal control. This research generally shows elevations in RSA during social interactions, indicating that infants are utilizing more parasympathetic suppression during socialization. Field and Diego (2008) recently reviewed this topic.

Literature examining the implications of polyvagal and dynamic systems theories has generally supported a relationship between poor cardiac vagal control and a vulnerability to anxiety in both children and adults. Discrepancies in the literature suggest that cardiac vagal control is best conceptualized as a vulnerability factor for developing anxiety, in that it may be a trait that predisposes an individual to anxiety, although it is neither necessary nor sufficient to produce anxiety disorders on its own.

Anxiety Sensitivity and Inhibition

Anxiety sensitivity is typically described as a personality trait which functions as a predisposition to developing fears (Reiss, 1991; Reiss & McNally, 1985). More specifically, anxiety sensitivity represents individual differences in how likely an individual is to interpret physiological symptoms of anxiety in a catastrophic manner, with individuals who attribute more meaning to their symptoms being at higher risk for clinical anxiety (Reiss, 1991). This is similar to the concept of catastrophic cognitions increasing symptoms of panic Clark's (1986) model of panic disorder. The concept of anxiety sensitivity was originally proposed as part of the expectancy model of developing fears (Reiss & McNally, 1985). This model proposes fear (Fb) is a result of several factors including danger expectancy (Ed), anxiety expectancy (Ea), and anxiety sensitivity (Sa) represented in the formula $Fb = Ed + (Ea \times Sa)$. According to this model, danger expectancy is the amount of actual physical or social danger a person expects when encountering a feared stimulus or situation. Anxiety expectancy refers to the amount of anxiety (or symptoms of panic) one anticipates experiencing when encountering the feared stimulus or

situation. And anxiety sensitivity is a personality trait which functions as a predisposition to developing fears.

While Reiss, Peterson, Gursky, and McNally (1986) found that levels of anxiety sensitivity were the highest in individuals with agoraphobia, they also found that individuals with other anxiety disorders scored much higher than a sample of non-fearful college students. In more recent literature, anxiety sensitivity has been supported as a risk factor for developing anxiety disorders. In a meta-analysis, Olantunji and Wolitzky-Taylor (2009) found that anxiety sensitivity is higher in individuals with anxiety disorders than in nonclinical control participants or individuals with mood disorders. They also found that individuals with panic disorder and posttraumatic stress disorder have higher levels of anxiety sensitivity than those with other anxiety disorders (Olantunji and Wolitzky-Taylor). In a recent analysis of the nature of anxiety sensitivity, it was found that the construct is taxonic in nature and that it is best understood as having a 3-part multidimensional structure (Bernstein et al., 2010). These three parts are consistent with the three factors of the anxiety sensitivity index (Reiss, Peterson, Gursky, & McNally, 1986), which are labeled physical concerns, mental incapacitation or psychological concerns, and social concerns. The physical concerns and psychological concerns were found to be stronger predictors of clinical anxiety (Bernstein et al., 2010).

While anxiety sensitivity has been well-established as a risk factor for anxiety, research comparing it to inhibition problems such as response inhibition and cardiac vagal tone, and other risk factors for anxiety, has been scarce. In a study of the serotonin transporter gene, Schmidt et al. (2000) found that anxiety sensitivity interacted with the L form of the gene to predict decreased HRV during a CO₂ task in a community sample, suggesting that anxiety sensitivity may be involved in HRV responses to challenging tasks. Additionally, Melzig, Weike, Hamm,

and Thayer (2009) investigated startle reflex and HRV in sample of individuals high or low in anxiety sensitivity, and separately in groups of individuals with and without panic disorder. They found that individuals high and low in anxiety sensitivity did not differ in HRV during a startle paradigm. Individuals with and without panic disorder also did not differ in HRV during the startle paradigm. However, the authors did find that individuals with low resting HRV across all groups had exaggerated startle responses in response to a threat of shock (Melzig et al.). While the authors did not draw direct conclusions about anxiety sensitivity and HRV, they did suggest that low HRV at rest may be an endophenotype for some of the anxiety disorders (Melzig et al).

Summary

Differences in the BIS, response inhibition, and cardiac vagal control are all evident between individuals with anxiety and nonclinical control participants. Both adults and children with anxiety are found to have more activation of the BIS than individuals with no psychopathology. Additionally, individuals with anxiety perform differently on tasks requiring response inhibition, with some studies showing an overall deficit in response inhibition and some studies suggesting that there is a varying pattern of response in anxiety such that they are less likely to take blind guesses or respond impulsively. This is supported by findings of increased reaction times, more errors of omission, and fewer errors of commission in individuals with anxiety than either nonclinical controls or individuals with externalizing problems. However, research on response inhibition in individuals with anxiety is fairly mixed, and no consistent conclusions can be drawn from the extant literature. Furthermore, individuals with anxiety demonstrate reduced cardiac vagal control at rest and less change in cardiac vagal tone during a stressful or effortful task. Finally, anxiety sensitivity, a personality factor and risk factor for

anxiety, is likely to be related to inhibition; however, research has yet to fully examine this in a meaningful way.

These three indexes of inhibition—namely BIS, response inhibition, and cardiac vagal control—would be better understood if relationships and interactions between them were more clearly delineated. One theory is that BIS can be thought of as a higher system in the brain that serves to mediate response inhibition and help regulate cardiac vagal control. Problems with the BIS translate into differences in inhibiting behaviorally and approaching appropriate stimuli in one's environment, as well as problems with the ability of the myelinated vagus to inhibit the sympathetic nervous system, leading to both underactive and more static cardiac vagal control. Additionally, when examining etiological models from a developmental perspective, BIS has been proposed as a vulnerability factor that predisposes individuals to developing clinical anxiety. Response inhibition and vagal control may be applicable as observable behavioral and biological vulnerabilities that individuals inherit and that predispose them to developing anxiety disorders, and some research has suggested that they may serve as endophenotypes for certain types of anxiety. Conversely, response inhibition may not be related to anxiety at any level since the research is currently mixed.

Rationale and Purpose of Current Study

Anxiety has long been associated with problems with inhibition; however, there are many different types of inhibition affecting different areas of the brain and physiology that may or may not affect anxiety differently. Three types of inhibition thought to be mediated through different parts of the brain are the BIS, response inhibition, and cardiac vagal control. While all of these represent inhibition, they manifest in different ways. The BIS is thought to be mediated through lower structures of the brain to serve the primary function of conflict resolution (Gray &

McNaughton, 2000). Individuals with anxiety are generally found to have over-activation in the BIS in that they process negative information in conflicts more frequently and to a greater degree than individuals without anxiety (Gray & McNaughton). Response inhibition is an executive function and is thought to be mediated by the prefrontal cortex or frontal lobes of the brain (Casey et al., 2002), and unfortunately little is known about how this processing is affected in anxiety. Extensive research has been done with OCD, however, which has focused primarily on just this disorder or specific symptoms of the disorder. While there is sufficient evidence to conclude that individuals with OCD have poor response inhibition (Chamberlain et al., 2005), it is still unclear whether it is affected the same way across the other anxiety disorders. Since one of the functions of the prefrontal cortex is to order conflicts and relay messages to the BIS, it is likely that individuals with other anxiety disorders would have deficits in this which then causes over-activation of the BIS. Finally, cardiac vagal control represents the parasympathetic system inhibiting the sympathetic system (Friedman, 2007). Individuals with anxiety have less inhibition over the sympathetic system, evidenced by less HRV at rest; they also have problems with modulation of cardiac vagal tone across different environmental conditions evidenced by more static HRV across different situations (Friedman).

Additionally, while these three types of inhibition have been researched to some degree in adults and generally to a lesser degree in children, little is known about how the effect of inhibition changes across development, especially in adolescents. Children's brains and physiology continue to develop far beyond birth and through adolescence and young adulthood. Of particular interest is that myelination of the prefrontal cortex continues throughout childhood and adolescence until early adulthood (Marsh, Gerber, & Peterson, 2008). Given the changes in physiology across development, it may be useful to examine these indexes of inhibition in a

sample of older adolescents, who have presumably developed most of the basic networks, but may still be completing their development.

As such, the primary aim and purpose of this investigation is to replicate and extend research examining the role of BIS, response inhibition, and cardiac vagal control in anxiety in a sample of older adolescents. The aims of the study for replication include demonstrating that individuals with anxiety have more anxiety sensitivity, BIS sensitivity, higher resting RMSSD, and less modulation in RMSSD from resting to an effortful response inhibition task.

Additionally, the study seeks to examine how these indexes of inhibition interact with each other, which has rarely been examined in previous literature and would help to further understanding of the neurophysiology of anxiety in a critical part of development. The aims of the study meant to extend previous research are to better understand whether individuals with anxiety have deficits in response inhibition by improving on methodology past studies. This will be done by using straightforward response inhibition tasks including the Day-Night Stroop and Go/No-go, which are not influenced by motor or speech artifact. Another way this study will improve upon methodology is by examining patterns of response and reaction time as dependent variables rather than overall accuracy of the task. Furthermore, this is the first study of its kind to examine relationships between indexes of inhibition rather than examining them individually. Given that BIS, response inhibition, and cardiac vagal control are all mediated by different parts of the brain, understanding how these interact with each other and the degree to which they are affected in adolescence would help to inform how different parts of the brain are affected by anxiety. Finally, this study seeks to further explore the role of inhibition in anxiety sensitivity, an established risk factor for anxiety in both adults and children. This would contribute to the field's basic knowledge about how inhibition affects anxiety across several different domains of

inhibition. It may also inform treatment recommendations regarding important targets for treatment, both pharmacologically and behaviorally, and what would be expected to change in treatment.

Hypotheses for Current Study

Hypotheses for the current study are as follows:

- 1) It is expected that older adolescents with significant anxiety symptoms, as measured by an anxiety screening tool, will score higher on a self-report measure of BIS sensitivity, will have longer latencies and make fewer errors on a simple and straightforward task of response inhibition, and will have lower resting RSA and less change in RSA during an inhibition task than participants without significant anxiety symptoms.

- 2) It is expected that all of these measures of inhibition will be correlated with each other. Namely, that scores on a self-report measure of BIS sensitivity will positively predict anxiety and anxiety sensitivity, positively predict performance on a response inhibition task, and negatively predict resting RSA and change in RSA during a response inhibition task.

- 3) Finally, it is expected that more BIS sensitivity, longer latencies, more errors of omission, and fewer errors of commission on response inhibition tasks, more RSA at rest, and less change in RSA from rest to during effortful tasks will predict higher levels of anxiety sensitivity. Additionally, while no a priori predictions about subscales of the ASI can be made, inhibition will be explored across the three dimensions of anxiety sensitivity, including physical concerns, psychological concerns, and social concerns.

METHOD

Participants

Participants were 60 older adolescents ages 18- and 19-years old (mean age = 18.60, *SD* = 0.49) recruited from undergraduate psychology classes. Forty-seven females and 13 males participated, and they identified themselves ethnically as 83.3% Caucasian, 11.7% African American, 3.3% Asian, and 1.7% Hispanic. They received course credit for their participation. Participants were divided into groups of those with and without significant anxiety symptoms based on whether or not their responses were above or below clinical cutoff (T-score ≥ 65) on the Multidimensional Anxiety Scale for Children (MASC; March, 1997; see Measures for a description). Using these criteria, 23 participants met criteria for the anxious group, and 37 participants were identified as non-clinical controls. This relatively high percentage of participants with significant anxiety symptoms was due to recruitment strategies targeted toward recruiting equal numbers of anxious and control participants (Please see the procedure section for more detailed information). All participants completed informed consent, and the study was approved by the Louisiana State University Institutional Review Board.

Measures

Demographic Questionnaire. This is a questionnaire designed by the author, which assessed for basic demographic information such as age, gender, and race. Additionally, participants were asked about any prescription or nonprescription medications taken in the past 24 hours which may have affected heart rate data or performance on the response inhibition task as well as handedness.

Multidimensional Anxiety Scale for Children (MASC; March, 1997). This 39-item self-report questionnaire assesses for the presence of anxiety in individuals ages 8-19. It consists

of a list of symptoms related to anxiety, and individuals rate how severely they have experienced each symptom on a Likert-scale ranging from 0 (*never true about me*) to 3 (*often true about me*). The MASC yields scales for several dimensions of anxiety including physical symptoms, harm avoidance, social anxiety, and separation/panic. It also yields an anxiety disorders index and a total score. This measure has been shown to have good internal consistency, test-retest reliability, a small standard error of measurement, and to accurately identify individuals with anxiety (March, Parker, Sullivan, Stalings, & Connors, 1997). T-scores for the total MASC score were used to divide participants into two groups of those with and without significant anxiety symptoms. Chronbach's alpha for the MASC total score in the current sample was .958.

BIS/BAS Scales (BIS/BAS; Carver & White, 1994). This 20-item self-report measure assesses sensitivity to BIS and BAS in adults. It consists of a list of sentences thought to index either BIS or BAS, and individuals rate each statement on a scale ranging from 0 (*strongly disagree*) to 4 (*strongly agree*). It has demonstrated reliability and convergent and discriminant validity in both clinical and control populations, strong psychometric properties, and it is generally considered the gold standard for assessing BIS and BAS in adults (Campbell-Sills, Liverant, & Brown, 2004; Carver & White). Chronbach's alpha for the BIS scale in the current sample was .841.

Anxiety Sensitivity Index (ASI; Reiss et al., 1986). The ASI is a 16-item questionnaire designed to index anxiety sensitivity in adults. It consists of 16 symptoms of anxiety including both catastrophic cognitions and uncomfortable physical sensations commonly experienced during anxiety. Individuals rate how true each statement is for them on a 5-point Likert scale ranging from 0 (*very little*) to 4 (*very much*). The ASI is generally considered to be the gold standard in measuring anxiety sensitivity. It yields a total score ranging from 0 to 64, with

higher scores indicating more anxiety sensitivity. The ASI has also been shown to have a reliable multidimensional structure. Although several subscales for the ASI have been referenced in the literature (e.g., Cox, Parker, & Swinson, 1996; Peterson & Heilbronner, 1987) the one most generally accepted and supported by the literature is that of Zinbarg, Barlow, and Brown (1997). This structure consists of a total score and three subscales, including physical concerns, mental incapacitation concerns or psychological concerns, and social concerns. The ASI has been shown to be reliable and valid in both clinical and non-clinical populations (Cox et al., 1996; Zinbarg et al., 1997). Chronbach's alpha in the current sample was as follows: total score = .936, physical concerns = .905, psychological concerns = .811, social concerns = .725.

Day/Night Stroop Task (Diamond & Taylor, 1996). This task measures response inhibition and is a version of the original Stroop task that does not require participants to be proficient at reading. It also does not require participants to read out loud, which makes it optimal for use during heart rate recording when movement artifact is a concern. In this task, participants viewed a large picture in the middle of the screen (the varying card) and two small pictures on the bottom of the screen (the response buttons) with a bulls-eye between them (See Figure 1). Participants then performed the task under two conditions—matching (in which they are supposed to click on the matching card, requiring no inhibition) and non-matching (in which they are supposed to click the on the non-matching small picture, requiring inhibition of the prepotent response to match). All participants performed the matching condition before the non-matching condition to further ingrain automaticity of the matching response. Participants performed each condition of the task for three minutes, completing as many trials as possible within that time. Because adults typically provide correct responses to this task (a ceiling effect),

reaction time between when the initial stimulus was presented and participants' responses were used as the primary measure of performance.

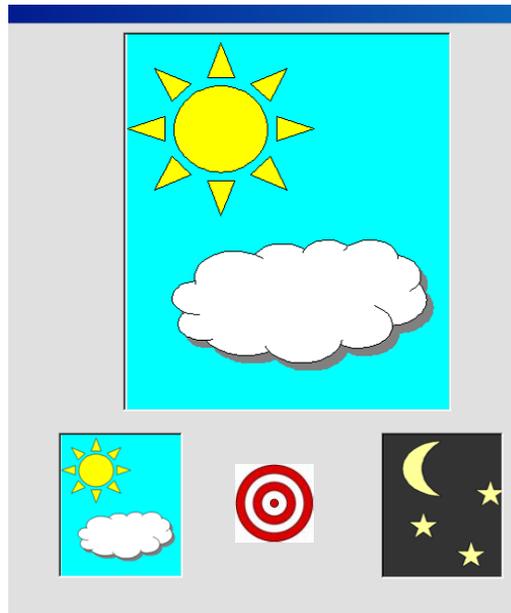


Figure 1. Stimulus Presentation of the Day/Night Stroop Task

Go/No-go Task (Boelhouwer, Teurlings, & Brunia, 1991). This task has been used throughout the literature to measure response inhibition. Participants were presented with single digits that appeared on a blank screen. They were instructed to press the space bar with their dominant hand when they saw any digit other than a 3 and to not press anything if a 3 appeared. They were also instructed to give equal attention to accuracy and time. Reaction time, number of false positives, and number of false negatives were used as the primary measures of performance.

Physiological Recording

Heart rate was recorded using a Polar S810 heart rate monitor, which records RR intervals, or the distance between peaks in the QRS complex, at rest and during the inhibition tasks. The Polar S810 system has the participant wear an elastic strap around their lower chest

which contains electrodes. The electrodes in the elastic strap record the heart rate and transmit the heart rate through a wireless signal to a wristwatch that the participant wears. The heart rate data is recorded and stored in the watch monitor until it is converted to a computer for further analysis. Once the heart rate data was converted to the computer, it was then edited using Polar Precision Performance Software. Following this, heart rate was further analyzed to root mean square successive difference (RMSSD) data using BioAnalysis Software (Niskanen, Tarvainen, Ranta-aho, & Karjalainen, 2004). The Polar system is completely noninvasive and does not require other unpleasant adhesives or electrode pastes frequently necessary with other systems.

Procedure

Participants were recruited from undergraduate psychology classes via an online experiment system, and they signed up to participate in the study online. During initial recruitment, participants signed up for the study and were consented when they presented for their individual testing session. To ensure appropriate power for comparisons between anxious and control groups, after all control-group participants were collected recruitment was targeted toward collecting participants who met criteria for the anxious group. This was done by screening in groups prior to individual testing, during which they were consented and completed the demographic questionnaire and the MASC. If they met criteria for the anxious group, they were invited to schedule an individual testing appointment. When participants presented for their individual testing session, they were tested individually in a comfortable testing room free of distractions. Participants completed the questionnaires either before or after the tasks in a counterbalanced order, with half of the participants completing questionnaire before the tasks and half completing them after. Participants that were screened prior to their individual testing session had already completed the demographic questionnaire and MASC and did not repeat

these. In the testing session, participants completed the demographic questionnaire, the MASC, BIS/BAS, and the ASI. The physiological equipment was then placed on the participant. To obtain a baseline measure of RMSSD, participants' resting heart rate was measured for 5 minutes while they watched a mildly stimulating cartoon on a computer in the sitting position. Following collection of baseline heart rate, participants completed the Day/Night Stroop task and the Go/No-go Task in counterbalanced order for 3 minutes each while heart rate was being recorded. To obtain a recovery measure of heart rate, the participant then sat for another 5 minutes watching a different video in the same position following completion of the tasks. Videos shown before and after baseline were also counterbalanced between participants. Participants were given extra credit in undergraduate psychology courses.

RESULTS

Preliminary Analyses

To test for differences due to gender and race, several preliminary analyses were performed. A *t*-test revealed no significant difference between anxious and control groups by age [$t(58) = .96, p = .338$]. Additionally, Chi Square analyses revealed no significant differences between the anxious and control groups due to gender [$\chi^2(1) = 1.63, p = .201$] or race [$\chi^2(3) = 2.43, p = .488$]. To examine effects of gender, race, and age on the dependent variables, a MANOVA analysis was run for each of the demographic variables. The MANOVA for gender revealed a significant omnibus effect [Wilks' Lambda = .662; $F(11,44) = 2.04, p = .047$]. Follow-up one-way ANOVAs revealed a significant difference in reaction time on the Day-Night Stroop task [$F(1,54) = 4.38, p = .041$], with men ($M = 773.94, SD = 81.69$) responding more quickly than women ($M = 844.41, SD = 112.42$). No other dependent variables differed by gender (See Appendix for a table of dependent variable descriptives by gender.). The results presented in the study do not account for differences in gender for reaction time in the Day-Night Stroop; however, all analyses were also run using the residual of reaction time in the Day-Night Stroop with the variance due to gender taken out. In these additional analyses, the pattern of significance in results did not change. Race [Wilks' Lambda = .613; $F(33,124) = 0.683, p = .898$] and age [Wilks' Lambda = .797; $F(11,44) = 1.02, p = .444$] were not found to affect the dependent variables.

Additionally, manipulation checks were run on the Day-Night Stroop and the Go/No-go tasks to ensure that participants responded in the anticipated manner. In the Day-Night Stroop task, participants' reaction times were significantly faster on the control trial ($M = 672.69\text{ms}, SD = 90.92$) than in the inhibition trial ($M = 832.85\text{ms}, SD = 14.59$) [$t(59) = 16.00, p < .001$]. This

held true for both the anxious and control groups [$t(59) < 9.02, p < .001$]. As expected, the percent correct on the Day-Night Stroop yielded a ceiling effect for both the control ($M = 100\%$, $SD = .005$) and inhibition ($M = 99\%$, $SD = .014$) trials, and reaction time on the inhibition condition was the primary dependent variable for this task. Surprisingly, the Go/No-go task also revealed a ceiling effect for percent correct ($M = 100\%$, $SD = .014$), including both control trials (non-3 trials $M = 100\%$, $SD = .005$) and inhibition trials (3 trials $M = 92\%$, $SD = .11$). For this reason, reaction time was used as the sole dependent variable for the Go/No-go task in the primary analyses, and patterns of responses were not analyzed. Distributions of reaction time for the Day-Night Stroop inhibition condition and the Go/No-go task were both positively skewed, indicating that most participants had short reaction times. Additionally, as a manipulation verification, a t -test revealed that the anxious group scored significantly higher on the MASC total T-score [$t(58) = 12.71, p < .001$].

Five participants in the anxious group reported taking anxiolytic medication within 24 hours of their individual testing appointment. To examine whether anxiolytic medication affected heart rate measurements, a MANOVA was run comparing participants in the anxious group who had taken medication with those who had not taken medication on the dependent variables of interest, including MASC T-score, the ASI and its subscales, reaction time on the Day-Night Stroop and Go/No-go tasks, resting RMSSD, change in RMSSD during the Day-Night Stroop task, and change in RMSSD during the Go/No-go task. There was no significant omnibus effect, suggesting no differences between these groups [Wilks' lambda = 0.374, $F(11,9) = 1.37, p = .325$]. Therefore, use of medication was not used as a covariate in analyses.

Primary Analyses

Differences between Anxious and Control Groups in Indexes of Inhibition. Due to

the number of statistical analyses proposed, alpha was set to .01, and a criterion of $p \leq .01$ was used to determine statistical significance. To examine the first two hypotheses, (1) that older adolescents with and without significant anxiety symptoms would respond differently on the BIS, perform differently on the Day/Night Stroop and Go/No-go tasks, and display differences in cardiac vagal control (both at rest and have trouble modulating appropriately during a task), a MANOVA was performed with the dependent variables being BIS score on the BIS/BAS scales, reaction time on the inhibition trials of the Day/Night Stroop, reaction time on the control trials of the Go/No-go task, resting vagal tone, and a difference score of vagal tone from mean during baseline to mean during the Day/Night Stroop task and between baseline and the Go/No-go task. The omnibus effect of the MANOVA was significant [Wilks' Lambda = .516; $F(6,49) = 7.66$, $p < .001$]. Follow-up one-way ANOVAs revealed significant differences between the anxious and control groups for the BIS scale [$F(1,54) = 39.44$, $p < .001$]. Additionally, the reaction time for the inhibition trials of the Day-Night Stroop was in the predicted direction, but was not significant [$F(1,54) = 2.94$, $p = .092$]. Both of these effects were in the expected direction. See Table 1 for descriptives of all dependent variables by group.

Relationships among Anxiety, Indexes of Inhibition, and Anxiety Sensitivity. To test the second hypothesis, that relationships between the types of inhibition, anxiety, and anxiety sensitivity will exist in the predicted directions, a Pearson's bivariate correlation table was calculated. Variables included in the correlation table were the MASC total T-score, the ASI total score as well as its three subscales, reaction time on the inhibition condition of the Day-Night Stroop, reaction time on the Go/No-go task, cardiac vagal tone (RMSSD) at rest, a change score from RMSSD at rest to the inhibition condition of the Day-Night Stroop, and a change score from RMSSD at rest to the Go/No-go task. Many correlations were in the predicted

directions (See Table 2 for correlation coefficients and significance values). Of note, the BIS scale was significantly correlated with the MASC ($r = .75, p < .001$) and all scales of the ASI ($r_s < .52-.63, p_s < .001$) but not with any other measures of inhibition (i.e., response inhibition or cardiac vagal tone). The two measures of response inhibition were correlated with each other ($r = .52, p < .001$). Regarding cardiac vagal tone, resting RMSSD was significantly correlated with change in RMSSD during the Go/No-go task ($r = .47, p < .001$) and was in the predicted direction with change in RMSSD during the inhibition condition of the Day-Night Stroop task ($r = .24, p = .079$).

Table 1. Descriptive Statistics for Primary Dependent Variables by Group

Dependent Variable	Anxious <i>M(SD)</i>	Control <i>M(SD)</i>	Total <i>M(SD)</i>
MASC total T-score*	74.00 (6.43)	46.92 (8.86)	57.30 (15.48)
Anxiety Sensitivity (AS) total*	39.35 (10.16)	16.92 (9.56)	25.52 (14.67)
AS physical concerns*	19.61 (5.02)	7.81 (6.07)	12.33 (8.08)
AS psychological concerns*	6.17 (3.38)	1.73 (1.88)	3.43 (3.34)
AS social concerns*	11.04 (2.96)	6.76 (2.80)	8.40 (3.53)
BIS scale*	1.17 (2.27)	-3.92 (3.37)	-1.97 (3.88)
Day-Night Stroop RT inhibition condition	869.68 (143.93)	809.95 (82.89)	832.85 (113.02)
Go/No-go RT	448.94 (81.78)	434.74 (90.54)	440.18 (86.86)
Resting RMSSD	31.78 (22.91)	35.96 (28.83)	34.42 (26.67)
Day-Night Stroop Δ RMSSD inhibition	2.87 (10.02)	-1.79 (38.28)	-0.04 (30.78)
Go/No-go Δ RMSSD	3.14 (8.62)	-1.83 (24.00)	0.01 (19.81)

Note. * $p < .05$ for difference between anxious and control group in MANOVA analyses. RT = reaction time. Δ = change from resting.

Anxiety Sensitivity Analyses. To replicate previous literature of individuals with anxiety endorsing more anxiety sensitivity on the ASI than individuals without anxiety, a MANOVA was performed to examine differences between the anxious and control groups on the ASI and its subscales. The omnibus effect of the MANOVA was significant [Wilks' Lambda = .426; $F(4,55) = 18.53, p < .001$]. Furthermore, the anxious and control groups differed on all scales of the ASI including the total score [$F(1,58) = 74.46, p < .001$], physical concerns scale

[$F(1,58) = 60.87, p < .001$], psychological concerns scale [$F(1,58) = 42.92, p < .001$], and social concerns scale [$F(1,58) = 31.77, p < .001$]. Again, please see Table 1 for descriptives of the ASI by group. To test the third hypothesis, that the three indexes of inhibition would account for a significant portion of anxiety sensitivity, a regression was performed using the ASI total score as the dependent variable and the BIS scale, reaction times on the Day-Night Stroop and Go/No-go, RMSSD at rest, and change scores in RMSSD from rest to the Day-Night Stroop and Go/No-go as predictors entered in a single step. The overall regression model was significant [$F(6) = 7.57, p < .001, \text{Adjusted } R^2 = .418$]. See Table 3 for the Regression Table. The only predictor that accounted for a unique and significant portion of the variance was the BIS scale ($\beta = .655, p < .001$). RMSSD at rest ($\beta = -.271, p = .025$) was also in the predicted direction but did not meet the criterion of $p \leq .01$. Additionally, because the BIS scale was significantly correlated with all scales of the ASI in the Pearson's correlation analyses, a regression was run with the subscales of the ASI as predictors and BIS as the dependent variable. This overall regression model was significant as well [$F(3) = 12.85, p < .001, \text{Adjusted } R^2 = .376$]. None of the predictors accounted for a unique portion of the variance in BIS, suggested that the subscales of the ASI have a significant amount of shared variance between them. See Table 4 for this Regression table.

Table 2. Bivariate Correlations between Dependent Variables

	MASC total T-score	AS total	AS physical concerns	AS psychological concerns	AS social concerns	BIS scale	Day-Night Stroop RT inhibition condition	Go/No-go RT	Resting RMSSD	Day-Night Stroop Δ RMSSD inhibition
Anxiety Sensitivity (AS) total	.782*									
AS physical concerns	.725*	.952*								
AS psychological concerns	.709*	.883*	.762*							
AS social concerns	.638*	.819*	.659*	.686*						
BIS scale	.754*	.632*	.573*	.606*	.524*					
Day-Night Stroop RT inhibition condition	.186	.208	.250	.105	.185	.180				
Go/No-go RT	.059	.123	.109	.028	.227	.055	.521*			
Resting RMSSD	-.025	-.137	-.220	-.024	-.048	.163	-.007	-.096		
Day-Night Stroop Δ RMSSD inhibition	.137	.078	.061	.163	.009	.079	-.161	-.117	.237	
Go/No-go Δ RMSSD	.228	.118	.078	.201	.092	.250	-.059	-.090	.471*	.619*

Table 3. Regression Table of Indexes of Inhibition Predicting ASI Total Score

Independent Variable	B	SE B	β	<i>p</i>
Step 1				
BIS scale	2.44	.40	.655	< .001*
Day-Night Stroop RT inhibition condition	0.01	.02	.045	.717
Go/No-go RT	0.01	.02	.065	.596
Resting RMSSD	-0.15	.07	-.271	.025*
Day-Night Stroop Δ RMSSD inhibition	0.05	.06	.105	.435
Go/No-go Δ RMSSD	0.001	.13	.001	.992

Note. $R^2 = .481$, Adjusted $R^2 = .418$. RT = reaction time. Δ = change from resting. * $p < .05$

Table 4. Regression Table of ASI Subscales Predicting BIS Scale

Independent Variable	B	SE B	β	<i>p</i>
Step 1				
Physical Concerns Scale	0.10	.18	.217	.197
Psychological Concerns Scale	0.39	.20	.339	.053
Social Concerns Scale	0.16	.16	.149	.318

Note. $R^2 = .408$, Adjusted $R^2 = .376$. * $p < .05$

DISCUSSION

In this study, the role of inhibition in anxiety was examined, as well as relationships between the primary indexes of inhibition including the BIS, response inhibition, and cardiac vagal tone (or HRV) at rest and during tasks of response inhibition. The study aimed to replicate previous research showing that individuals with anxiety have more anxiety sensitivity, more BIS sensitivity, higher resting RMSSD, and less change in RMSSD from rest to an effortful task than individuals without anxiety. The study sought to extend previous research by examining effects of anxiety on response inhibition by using improved methodology, as well as to examine relationships between indexes of inhibition. Additionally, anxiety sensitivity, an established risk factor for anxiety that has been shown to be related to the development of clinical anxiety in both children and adults, was examined in how it relates to indexes of inhibition, which has not been examined in extant research. As expected, inhibition and anxiety sensitivity were found to be related to symptoms of anxiety in an older adolescent sample. Indexes of inhibition were found to be related to anxiety and some relationships between indexes of inhibition were related as well. Finally, inhibition was found to be related to and likely an important factor in anxiety sensitivity. These findings are discussed in more detail, as well as their implications for future research in the field of anxiety and its etiology and risk factors.

Differences in Indexes of Inhibition by Group

Several analyses were run comparing an anxious group of older adolescents to a nonclinical control group. Significant differences on a self-report measure of BIS were found between the anxious and control groups, and, as expected, individuals in the anxious group reported more behavioral inhibition. This supports previous literature showing that individuals with significant anxiety typically have more BIS sensitivity than individuals with less anxiety

(e.g., Gomez & Gomez, 2005; Hundt et al., 2007; Kimbrel et al., 2007). This finding also reinforces BIS as an important risk factor and feature of clinical anxiety in older adolescents and implicates the parts of the brain that are part of the BIS in this population.

Response inhibition was not significantly different between the anxious and control groups, but reaction time in the Day-Night Stroop, one of the response inhibition tasks was in the predicted direction, and individuals with anxiety took longer to respond during the inhibition trial of this task than their non-anxious counterparts. Reaction time on the Go/No-go task was also in the expected direction but was not significantly different between the groups. Given that the research on response inhibition in individuals with anxiety is mixed, it was not surprising the differences in this study did not reach statistical significance. This suggests that response inhibition is not impacted by anxiety in an older adolescent population and that the prefrontal cortex that mediates response inhibition is not as severely impacted by anxiety in this age group. These effects being in the predicted direction, however, support the possibility that individuals with anxiety may have slower response times during an inhibition task, indicating that they are taking longer to process information than people without anxiety. This additional processing time could be accounted for by them being more careful, and being more willing to sacrifice time for accuracy across tasks. If this is the case, it may help to explain why individuals with anxiety have more difficulty on executive functioning tasks and on tests of intellectual functioning (Davis, Ollendick, & Nebel-Schwalm, 2008). Unfortunately, differences in patterns of responses on the response inhibition tasks could not be tested since both tasks yielded ceiling effects.

Regarding cardiac vagal tone differences, the anxious group had a lower resting vagal tone than the control group, as expected, but again this was not significantly different than the control group. The difference scores in how vagal tone changed from rest to during a response

inhibition task were relatively small in magnitude for both the anxious and non-anxious groups, and therefore, should not be interpreted other than showing that there were not significant differences between the groups. Neither group significantly modulated their vagal tone from baseline to during the task, as supported by failing to find significant differences in vagal tone from resting to during the task in both groups.

Anxiety sensitivity was also found to significantly differ between the two groups, with the anxious group reporting more anxiety sensitivity on both the total score and all subscales of the ASI. This follows a long line of research supporting increased anxiety sensitivity in individuals with more significant anxiety in both adults (e.g., Bernstein et al., 2010) and children (Bernstein, Zvolensky, Stewart, & Comeau, 2007; Muris, Merckelbach, & Meesters, 2001), as individuals with more anxiety consistently report higher levels of anxiety sensitivity.

Relationships between Indexes of Inhibition

Regarding findings of relationships between different indexes of inhibition (i.e., BIS, response inhibition, and cardiac vagal tone), some proposed relationships were supported, but many relationships between indexes of inhibition were not. BIS was found to be positively related to both anxiety, as measured by the MASC, and anxiety sensitivity, as measured by the ASI. Furthermore, BIS was positively related to all subscales of the ASI, including physical concerns, psychological concerns, and social concerns. However, BIS was not found to be significantly related to either response inhibition or cardiac vagal tone. It appears that BIS is strongly related to both anxiety and anxiety sensitivity. It was unexpected to find that it was not significantly or strongly related to either response inhibition or vagal tone.

Regarding response inhibition, reaction time on the two tasks was found to be strongly correlated. This was expected, as the tasks were both indexing response inhibition and

performance should be fairly consistent across the two tasks. Oddly, however, reaction times on the tasks were not related to either anxiety or anxiety sensitivity. Previous research has shown that poorer performance on response inhibition tasks was related to the development of OCD (Chamberlain et al., 2005). However, research on other anxiety disorders and anxiety in general has been very mixed, with some research showing that individuals with anxiety perform better on tasks of response inhibition and some showing that they perform worse (e.g., Knyazev et al., 2008; Schmidtke et al., 1998). This may be a result of measurement error, in which the variables present in assessing performance were not fully accounted for, and that rather than having “poorer performance” in general, individuals with increased anxiety showed a different pattern of response. Thus, depending on the types of errors examined, individuals with anxiety would have more errors of omission and slower reaction times, and they would have fewer errors of commission, which typically indicate impulsive responding. Again, this unfortunately could not be examined with this study, as the Go/No-go task had an unexpected ceiling effect, thus making it unfruitful to examine differences in response patterns. If this was possible, it may have been more likely to find performance on the response inhibition tasks, particularly the Go/No-go task, to be related to anxiety and anxiety sensitivity.

Cardiac vagal tone also yielded interesting and some unexpected findings. Resting vagal tone, as measured by RMSSD, was not significantly correlated with any of the other variables. This was unexpected given that past research has shown lower resting RMSSD to be predictive of increased anxiety and higher resting RMSSD to be predictive of better task performance. However, most previous research was done with adults, and no differences were found in the analyses comparing anxious to control groups. Additionally, it was surprising to find no significant correlations between anxiety or anxiety sensitivity and change in vagal tone from

resting to task performance on either the Day-Night Stroop or the Go/No-go tasks. It was expected that individuals with higher anxiety would have lower resting RMSSD and would also have less change in RMSSD from resting to during an effortful or anxiety-provoking inhibition task as they would not be to modulate their vagal tone as effectively. In line with expectations, however, vagal tone at rest was positively correlated with more positive change in RMSSD from baseline to task. Therefore, individuals who had higher resting vagal tone also had more change in RMSSD from resting to task. These, however, were relatively weak correlations, and need further research and replication to merit any further interpretation.

Explanations for Unexpected Findings

Indexes of Inhibition Are Unrelated. The first and most parsimonious theory as to why the results of response inhibition and cardiac vagal tone resulted in unexpected findings is that they are unrelated to each other and that they are unrelated to anxiety in an older adolescent population. Given findings from previous literature, it is possible that this could be the case for response inhibition; however, it is unlikely that this is the case for cardiac vagal tone.

Additionally, previous literature has shown that high BIS sensitivity affects EEG during and performance on the Go/No-go task, which was not found in the current study. Knyazev et al. (2008) found that individuals high or low in BIS and BAS had differences in their EEG recordings during a Go/No-go task. And Hagopian and Ollendick (1994) showed that individuals with more anxiety responded differently on a Go/No-go task. However, reaction time was not the primary measure in either of these studies. The amount of research on response inhibition also differs greatly depending on the type of anxiety. For symptoms of OCD, research has consistently found that OCD symptoms are predictive of poor performance on response inhibition tasks (Chamberlain et al., 2005). It has also been found that individuals with OCD

have poorer performance on response inhibition tasks than individuals with other types of anxiety, including panic disorder and social phobia (Bannon et al., 2008; van der Linden et al., 2005). Thus, there are no clear findings that have been replicated for individuals with types of anxiety other than OCD.

It is unlikely, however, that cardiac vagal control is unrelated to anxiety, BIS, or response inhibition, even in an older adolescent population. There has been a wealth of literature showing cardiac vagal tone both at rest and its modulation from resting to an effortful task is strongly predictive of anxiety in both adults and children (Beauchaine, 2001; Friedman, 2007). Cardiac vagal tone has also been suggested as a risk factor for developing anxiety disorders in childhood and some studies have shown higher resting vagal tone is heritable and that children of parents with anxiety disorders have higher vagal tone (see Reuther, Davis, and Friedman, 2011 for a review). While this is unlikely, there has not been extensive research on how cardiac vagal tone relates to BIS, response inhibition, or anxiety sensitivity.

The Current Study Was Not Able to Detect Differences or Relationships. An alternative explanation of why the current study did not find differences between anxious and control participants in response inhibition or cardiac vagal tone is that the design of the study was not strong enough to detect them. Additionally, it may be the case that the three indexes of inhibition are related to each other and anxiety sensitivity, but that the study design did not allow for this to be found. Regarding differences between anxious and control groups, this is more likely the case than to assume that response inhibition and vagal tone are not related to anxiety given past research which has found these differences. One reason why differences in response inhibition were not detected may have been a problem with the Go/No-go task. There was an unexpected ceiling effect in performance on the task, which meant that it was not possible to

examine patterns of errors in responding between participants with and without anxiety. This suggests that the Go/No-go task may not have been challenging enough to produce a reasonable amount of variability in responses among older adolescents who are enrolled in a university. Although the inter-stimulus interval was similar to that of other studies in the past, those studies did not focus on patterns of response as a dependent variable. There was also a ceiling effect on the Day-Night Stroop, which was expected. Previous research has found that the Day-Night Stroop is a valid task to use with older adolescents and adults (Byrd, Reuther, McNamara, & Berg, 2010); however, the primary variable of interest was reaction time on the inhibition trial rather than response time. Additionally, anecdotally participants typically perceived this task to be easy, and generally did not notice that their response times were slower during the mismatch condition. The reaction times for these tasks were also positively skewed, meaning that most participants had very short reaction times, which would be expected if the task was not difficult. If participants perceived both of the tasks to be relatively easy and not challenging, this may have effected how their cardiac vagal tone was modulated from resting to during the task since vagal tone would only be expected to change from baseline is a person engages in an effortful or anxiety-provoking task. Both adults and children have been shown to modulate their vagal tone more during more difficult tasks, with the amount that RMSSD decreases being proportional to the difficulty level of the task (Byrd et al., 2010). Neither the anxious or control groups had significant changes in RMSSD from baseline to task, which suggests that participants in the study may not have needed to greatly modulate their RMSSD from baseline to task because the tasks were not perceived to be effortful tasks. It is unlikely that the study did not have enough power to detect differences between the groups as studies in the past have found differences in HRV with sample sizes of 20 participants in each group (Melzig et al., 2009).

Adolescents with Anxiety Have Poor Introspective Skills. Another possible reason for why differences were found in the self-report measures of the MASC, ASI, and BIS, but not in objective measures of response inhibition and HRV is because adolescents with anxiety have poor introspective skills. Past literature suggests that individuals with anxiety have a tendency to overestimate their physiological and behavioral symptoms of anxiety and report more symptoms than objective measures reveal. For instance, individuals with a fear of heights reported more fear and dizziness than non-fearful control participants during an exposure to heights, but they did not have more body sway or higher heart rate when measured objectively (Alpers & Adolph, 2008). Likewise, individuals with social phobia report more anxiety when others can perceive their symptoms of anxiety (e.g., blushing, increased heart rate); however, they also overestimate the degree to which others can perceive this and overestimate their physiological reactions (Gerlach, Murlane, & Rist, 2004). When comparing a group of young adults diagnosed with social phobia to a non-clinical control group, Gerlach et al. found that while individuals with social phobia had higher heart rate than control participants while being evaluated, their estimates of increased heart rate were above and beyond the physiological differences.

It is also likely that introspection of anxiety symptoms may improve with age along the trajectory of ability to take others' perspectives, with younger children having relatively poor introspective skills and adults having more accurate perspective-taking. The afore mentioned studies both used young adult samples slightly older than the sample used in the current study. The anxious group in the current study may have been overestimating their symptoms of anxiety, which would partially explain why differences were found between the self-report measures but not the objective measures of response inhibition and HRV. Additionally, it would be expected

that measures of the same method would be correlated; thus, the current results are likely a combination of a method effect and poor introspective skills in the anxious group.

Response Inhibition and Cardiac Vagal Tone Represent Endophenotypes or Risk Factors Rather Than Symptoms of Anxiety. A final possibility to be considered is that rather than being symptoms of anxiety that correlate with the severity of anxiety, response inhibition and cardiac vagal tone represent endophenotypes or potential risk factors for developing anxiety rather than symptoms. As previously mentioned, an endophenotype is a trait found in individuals before, during, and after meeting diagnosis according to clinical criteria. It is also found in first degree relatives of individuals with a disorder who do not show outward symptoms. If response inhibition is an endophenotype of anxiety, it is possible that the nonclinical control participants had first degree relatives with anxiety and that they showed similar deficits in response inhibition, such that differences between the two groups could not be found. Deficits in response inhibition have been found in individuals who have successfully recovered from OCD (Rao et al., 2008) and in first degree relatives of individuals with OCD who have never shown clinical symptoms (Menzies et al., 2007). Impairment in response inhibition has been suggested as an endophenotype for OCD (Chamberlain et al., 2005). Cardiac vagal tone has been suggested as a risk factor for anxiety, but is an unlikely candidate for an endophenotype since it has been shown to respond to treatment for anxiety (Reuther et al., 2011). One way of examining this possibility further and controlling for this in future research would be to ask participants if they have a family history of clinical anxiety or to have parents and siblings of participants complete self-report questionnaires about their own symptoms of anxiety. It would also be helpful to give a more thorough diagnostic interview to ensure that participants with anxiety have a clinical disorder rather than relying on self-report.

Findings for Anxiety Sensitivity

Anxiety sensitivity is a major component of clinical anxiety, which has been supported by a large-scale meta-analysis (Olantunji & Wolitzky-Taylor, 2009). Anxiety sensitivity is generally understood as a personality factor of attributing meaning to symptoms of anxiety; it can be inherited and serves as a risk factor for developing clinical anxiety later in life. While anxiety sensitivity has been established as a risk factor for anxiety, its relationships with other risk factors for anxiety, including indexes of inhibition, has only recently been a topic in the literature. As expected, anxiety sensitivity was found to differ between the anxious and control groups, with the anxious group reporting more anxiety sensitivity. This replicates previous literature demonstrating the same effect (e.g., Olantunji & Wolitzky-Taylor, 2009). Because there is sparse previous literature regarding relationships between anxiety sensitivity and indexes of inhibition, much of the research in this study was exploratory in nature.

In correlation analyses, the total score and all subscales of anxiety sensitivity were found to be strongly correlated with anxiety as measured by the MASC total T-score. Again, this replicates previous literature showing anxiety sensitivity is strongly related to clinical levels of anxiety. The results also revealed strong and significant correlations between anxiety sensitivity and self-reported BIS sensitivity, which is another personality trait related to anxiety. Furthermore, strong correlations were found for all subscales of the ASI, with more anxiety sensitivity being related to more BIS sensitivity, with the total score and the psychological concerns subscale sharing the strongest correlations. This is consistent with the findings of Bernstein et al. (2010), who found that psychological concerns and physical concerns were more predictive of clinical anxiety than social concerns. Surprisingly, anxiety sensitivity was not found to share correlations with either response inhibition or cardiac vagal control; however, this

may have been due to problems with the response inhibition tasks rather than the effects not being present, as previously discussed.

In the proposed regression analyses, the overall model of BIS, response inhibition, and cardiac vagal tone at rest and change in vagal tone from rest to during the tasks yielded a model that significantly predicted 48% of the variance in the ASI total score. This suggests that indexes of inhibition are important and predictive of anxiety sensitivity and suggests that the risk factors of inhibition and anxiety sensitivity are related, although some inhibition indexes may be more strongly linked than others. Upon examining individual predictors, only the BIS scale and RMSSD at rest were found to account for unique and significant portions of variance in anxiety sensitivity. This finding serves as further evidence that higher BIS sensitivity is related to higher anxiety sensitivity, and it calls for further research examining how these two risk factors of clinical anxiety might be related and interact to increase the likelihood of developing an anxiety disorder. Cardiac vagal tone at rest also accounted for a significant portion of the variance in anxiety sensitivity, with less RMSSD at rest being predictive of more anxiety sensitivity. This replicates several studies in the past that have explored whether anxiety sensitivity is related to vagal tone. Schmidt et al. (2000) found that anxiety sensitivity interacted with the L form of a gene related to anxiety to predict less vagal tone during a CO₂ challenge task, and Melzig et al. (2009) suggested that low cardiac vagal tone at rest may be an endophenotype for anxiety disorders in a study of the startle reflex and anxiety sensitivity.

To further explore the correlations between the subscales of anxiety sensitivity and BIS sensitivity, an exploratory regression was performed examining whether the three subscales of the ASI predicted BIS sensitivity. The overall regression model was significant and accounted for 41% of the variance in BIS sensitivity, indicating that anxiety sensitivity was related to and

predictive of BIS sensitivity. Additionally, while none of the ASI subscales accounted for a unique portion of the variance, the psychological or mental incapacitation concerns subscale was in the predicted direction and accounted for more variance than the other two subscales. While further research would need to be done before results can be interpreted, this suggests that psychological concerns in anxiety sensitivity may be a variable that should be given strong consideration in its involvement and interactions with BIS sensitivity and other indexes of inhibition associated with anxiety in future research. In general, both regression analyses suggest that the indexes of inhibition are related to anxiety sensitivity, particularly psychological concerns, and suggest that exploring how these risk factors interact with each other to increase the likelihood of developing anxiety is an area of research that is worth pursuing in a more detailed fashion.

Limitations and Future Directions

While the current study has closed several gaps in the previous literature and explored future areas of research, there are several ways the study could have been improved to more strongly identify how risk factors of anxiety including indexes of inhibition and anxiety sensitivity are related. First, the sample used was comprised of undergraduate students aged 18 and 19 years. There are several potential problems with this sample of convenience. First, to be enrolled in a university and take responsibility for arriving at an individual testing session requires a certain level of functioning. Thus, although the participants in the anxious group scored above the clinical cutoff on an anxiety questionnaire, they may not be representative of the most severe cases of anxiety. It is likely the case that the most anxious participants were taking medication, and thus their scores on all measures may have been decreased. It is also likely that the more anxious undergraduates were self-selected out of the study by choosing not

to participate in a study focused on anxiety. Additionally, the sample was grouped into anxious and nonclinical groups based on whether they were above or below a clinical cutoff on a single questionnaire, and it was not known whether they met criteria for an anxiety disorder. Because the sensitivity and specificity of any self-report measure such as the MASC is never 100%, the results of the current study cannot necessarily be generalized to individuals with anxiety disorders. The study may have been stronger if participants were given a clinical interview such as the Anxiety Disorders Interview Schedule (ADIS; Brown, DiNardo, & Barlow, 1994) and grouped according to whether they met clinical criteria for a disorder or even a particular anxiety disorder. This also would have made it possible to examine differences between different types of anxiety, such as whether differences existed between individuals with OCD and individuals with other types of anxiety disorders. Additionally, the demographic questionnaire did not inquire about family history of anxiety, which would be important in determining if an endophenotype interpretation should be given consideration.

Another limitation is that the Go/No-go task may not have been difficult enough to index inhibition. Differences in response patterns and types of errors made between the anxious and nonclinical groups could not be examined due to ceiling effects on task performance—all participants had an average of 92% accuracy. Additionally, if the task was perceived as very easy, which the accuracy rate suggests, it may not have been operating as a test of response inhibition. If this were the case, it would not be surprising to fail to find differences in reaction time between the anxious and nonclinical control groups. Also, cardiac vagal tone was measured at rest and during two response inhibition tasks that were perceived as easy judging from ceiling effects on both tasks. It may have been useful to examine modulation of vagal tone from rest to a more difficult task, such as the N-back task, in which the difficulty can be adjusted to ensure

the task is effortful enough to require modulation of vagal tone so that it can be more accurately measured.

Results from the current study suggest several areas that can inform future research. It would be interesting to examine indexes of inhibition across different types of anxiety. Additionally, rather than examining individual disorders, it may be useful to examine different types of disorders. Recent research has begun to differentiate between anxiety disorders, obsessive-compulsive and related disorders, and trauma and stressor-related disorders, which also reflect potential changes to criteria for diagnosing disorders in the *DSM-V* (American Psychiatric Association, 2010). It may be useful to examine differences in indexes of inhibition and anxiety risk factors between these types of anxiety. Additionally, anxiety sensitivity was found to be strongly related to BIS sensitivity. This relationship should be replicated and studied in more detail to be expanded upon. It would be particularly interesting to examine how anxiety sensitivity and BIS sensitivity may interact to make development of clinical anxiety more likely. It also appeared in several analyses that psychological concerns, a subtype of anxiety sensitivity, may be uniquely related to BIS sensitivity. This may be a relationship worth examining in more detail in the future.

Although the current study examined a group of older adolescents, it would be interesting to examine how BIS, response inhibition, and cardiac vagal tone are related in individuals of different ages. Because myelination of the frontal lobes, where response inhibition and the higher orders of inhibition, is not complete until older adolescence or early adulthood (Diamond & Taylor, 1991; Rosso, Young, Femia, & Yurgelun-Todd, 2004), it is possible that the relationships between BIS, response inhibition, vagal tone, and anxiety may differ between different age groups that are at different stages of brain development. It would be interesting to

see how the relationships between types of inhibition may differ between a group of children and older adolescents and between a group of older adolescents and a group of middle-aged adults. Additionally, if research uses child samples, it would be interesting to pursue the possibility of response inhibition as an endophenotype and cardiac as a risk factor for anxiety by involving their parents in completing family histories of anxiety problems and by having parents or other first degree relatives of individuals with anxiety participate in research and comparing them to asymptomatic individuals without a family history of anxiety.

Implications of the Current Research

In this dissertation, three indexes of inhibition were examined for their contribution to anxiety and how they interact with each other to increase chances of developing anxiety. Indexes of inhibition were also examined in how they may be related to anxiety sensitivity, a risk factor for developing clinical anxiety. The results of this study clearly suggest that inhibition is important in both anxiety and anxiety sensitivity. Of interest, BIS sensitivity appears to be clearly related to anxiety sensitivity in older adolescents. Other indexes of inhibition, including response inhibition and cardiac vagal tone, were also found to be related but require further research to establish clear and replicated patterns. This enhances the current literature by increasing the understanding of risk factors for anxiety disorders and how they may relate to each other. For instance, anxiety sensitivity, BIS sensitivity, and cardiac vagal tone have been proposed as risk factors for clinical anxiety. This study takes a step in further understanding how these risk factors interact with each other and in understanding the neurophysiology of anxiety in an older adolescent population, which may aid in elaborating etiological models of anxiety, such as Barlow's (2002) triple vulnerability model, in which anxiety disorders develop as a result of generalized biological vulnerabilities, generalized psychological vulnerabilities, and specific

stressor vulnerabilities. The results of this study suggest that in this age group, distinct differences are found between individuals with and without anxiety in BIS sensitivity, which is controlled by lower or mid-brain structures. However, results were less clear with response inhibition, which is mediated by the frontal lobes.

Further, if these risk factors for anxiety are understood to a degree of being reliably predictive of anxiety disorders, it may be possible to create screening tools for children and adolescents who are at higher risk for developing clinical anxiety, and intervening early in the anxiety process to prevent anxiety disorders from developing into impairing and distressing problems. For instance, if a reliable and predictive questionnaire of anxiety disorder risk factors such as BIS and anxiety sensitivity could be administered in primary care settings, it would be possible for physicians to refer children who are at high risk for developing anxiety disorders, such as separation anxiety disorder or OCD, to prevention-based psychology services before they develop clinical levels of anxiety. The family could then be given psychoeducation about how anxiety develops, initial signs of anxiety developing, and what they can do to help the child fight anxiety symptoms before they become an impairing problem. Older children and adolescents could also be involved in this process and learn about their own risk factors and how to manage them in daily life to avoid developing clinically significant anxiety.

REFERENCES

- Albano, A. M., Chorpita, B. F., & Barlow, D. H. (2003). Childhood anxiety disorders. In E. J. Mash & R. A. Barkley (Eds.) *Child Psychopathology (2nd Edition)*. New York, NY: The Guilford Press. (pp. 279-329).
- Alkon, A., Goldstein, L. H., Smider, N., Essex, M. J., Kupfer, D. J., Boyce, W. T. et al. (2003). Developmental and contextual influences on automatic reactivity in young children. *Developmental Psychobiology, 42*, 64-78.
- Allen, J. J. B., Chambers, A. S., & Towers, D. N. (2007). The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. *Biological Psychology, 74*, 243-262.
- Alpers, G. W., & Adolph, D. (2008). Exposure to heights in a theme park: Fear, dizziness, and body sway. *Journal of Anxiety Disorders, 22*, 591-601.
- American Psychiatric Association. (2010). Proposed revisions for DSM-5. Downloaded from <http://www.dsm5.org/proposedrevision/Pages/Default.aspx> May 5, 2010.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders – Text Revision (4th ed.)*. Washington, DC: Author.
- Amodio, D. M., Master, S. L., Yee, C. M., & Taylor, S. E. (2008). Neurocognitive components of the behavioral inhibition and activation systems: Implications for theories of self-regulation. *Psychophysiology, 45*, 11-19.
- Aycicegi, A., Dinn, W.M., Harris, C.L., & Erkmen, H. (2003). Neuropsychological function in obsessive-compulsive disorder: Effects of comorbid conditions on task performance. *European Psychiatry, 18*, 241-248.
- Bannon, S., Gonsalvez, C.J., & Croft, R.J. (2008). Processing impairments in OCD: It is more than inhibition! *Behaviour Research and Therapy, 46*, 689-700.
- Bannon, S., Gonsalvez, C.J., Croft, R.J., & Boyce, P.M. (2002). Response inhibition deficits in obsessive-compulsive disorder. *Psychiatry Research, 110*, 165-174.
- Barlow, D. H. (2002). *Anxiety and Its Disorders (2nd Edition)*. New York, NY: The Guilford Press.
- Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13*, 182-214.

- Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology, 74*, 174-184.
- Bernstein, A., Zvolensky, M. J., Stewart, S., & Comeau, N. (2007). Taxometric and factor analytic models of anxiety sensitivity among youth: Exploring the latent structure of anxiety psychopathology vulnerability. *Behavior Therapy, 38*, 269-283.
- Bernstein, A., Stickle, T. R., Zvolensky, M. J., Taylor, S., Abramowitz, J., & Stewart, S. (2010). Dimensional, categorical, or dimensional-categories: Testing the latent structure of anxiety sensitivity among adults using factor-mixture modeling. *Behavior Therapy, 41*, 515-529.
- Bornstein, M. H., & Suess, P. E. (2000). Child and mother cardiac vagal tone: Continuity, stability, and concordance across the first 5 years. *Developmental Psychology, 36*, 54-65.
- Boelhouwer, A. J., Teurlings, R. J., & Brunia, C. H. (1991). The effect of an acoustic warning stimulus upon the electrically elicited blink reflex in humans. *Psychophysiology, 28*, 133-139.
- Brown, T. A., DiNardo, P. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV*, Oxford University Press.
- Byrd, D.L., Reuther, E.T., McNamara, J.P., & Berg, W.K. (2010). Age Differences in Respiratory Sinus Arrhythmia and Performance Reactivity to Increased Executive Function Load in Three Tasks. Manuscript in preparation.
- Campbell-Sills, L., Liverant, G. I., & Brown, T. A. (2004). Psychometric evaluation of the behavioral inhibition/behavioral activation scales in a large sample of outpatients with anxiety and mood disorders. *Psychological Assessment, 16*, 244-254.
- Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment. *Journal of Personality and Social Psychology, 67*, 319-841.
- Caseras, X., Àvila, C., & Torrubia, R. (2003). The measurement of individual differences in behavioural inhibition and behavioural activation systems: A comparison of personality scales. *Personality and Individual Differences, 34*, 999-1013.
- Casey, B. J., Tottenham, N., & Fossella, J. (2002). Clinical, imaging, lesion, and genetic approaches toward a model of cognitive control. *Developmental Psychobiology, 40*, 237-254.

- Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive-compulsive disorder. *Neuroscience and Biobehavioral Reviews*, *29*, 399-419.
- Chang, E. C., Zumberg, K. M., Sanna, L. J., Girz, L. P., Kade, A. M., Shair, S. R., et al. (2007). Relationship between perfectionism and domains of worry in a college student population: Considering the role of the BIS/BAS motives. *Personality and Individual Differences*, *43*, 925-936.
- Cherbuin, N., Windsor, T. D., Anstey, K. J., Maller, J. J., Meslin, C., & Sachdev, P. S. (2008). Hippocampal volume is positively associated with behavioural inhibition (BIS) in a large community-based sample of mid-life adults: the PATH through life study. *Social Cognitive and Affective Neuroscience*, *3*, 262-269.
- Cooper, A.J., Perkins, A.M., & Corr, P.J. (2007). A confirmatory factor analysis study of anxiety, fear, and behavioral inhibition system measures. *Journal of Individual Differences*, *28*, 179-187.
- Costello, E. J., Mustillo, S., Erkanli, A., Keeler, A., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry*, *60*, 837-844.
- Cox, B. J., Parker, J. D. A., & Swinson, R. P. (1996). Anxiety sensitivity: confirmatory evidence for a multidimensional construct. *Behaviour Research and Therapy*, *34*, 591-598.
- Davis III, T. E., Ollendick, T. H., & Nebel-Schwalm, M. (2008). Intellectual ability and achievement in anxiety-disordered children: A clarification and extension of the literature. *Journal of Psychopathology and Behavioral Assessment*, *30*, 43-51.
- Degroot, A., & Treit, D. (2004). Anxiety is functionally segregated within the septo-hippocampal system. *Brain Research*, *1001*, 60-71.
- Diamond, A., & Taylor, C. (1996). Development of an aspect of executive control: Development of the abilities to remember what I said and to “do as I say, not as I do.” *Developmental Psychobiology*, *29*, 315-334.
- El-Sheikh, M. (2005). Stability of respiratory sinus arrhythmia in children and young adolescents: A longitudinal examination. *Developmental Psychobiology*, *46*, 66-74.
- Field, A. P. (2006). Watch out for the beast: fear information and attentional bias in children. *Journal of Clinical Child and Adolescent Psychology*, *35*, 431-439.
- Field, T., & Diego, M. (2008). Vagal activity, early growth and emotional development. *Infant Behavior & Development*, *31*, 361-373.
- Fowles, D. C. (1980). The three arousal model: Implications of Gray’s two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology*, *17*, 87-104.

- Fracasso, M. P., Porges, S. W., Lamb, M. E., & Rosenberg, A. A. (1994). Cardiac activity in infancy: Reliability and stability of individual differences. *Infant Behavior and Development, 17*, 277-284.
- Friedman, B. H. (2007). An autonomic flexibility-neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology, 74*, 185-199.
- Friedman, B. H., Allen, M. T., Christie, I. C., & Santucci, A. K. (2002). Validity concerns of common heart-rate variability indices: Addressing quantification issues in time- and frequency-domain measures of HRV. *IEEE Engineering in Medicine and Biology, 21*, 35-40.
- Gerlach, A. L., Murlane, D., & Rist, F. (2004). Public and private heart rate feedback in social phobia: A manipulation of anxiety visibility. *Cognitive Behaviour Therapy, 33*, 36-45.
- Gomez, A., & Gomez, R. (2002). Personality traits of the behavioural approach and inhibition systems: Associations with processing of emotional stimuli. *Personality and Individual Differences, 32*, 1299-1316.
- Gomez, R., & Gomez, A. (2005). Convergent, discriminant, and concurrent validities of measures of the behavioural approach and behavioural inhibition systems: Confirmatory factor analytic approach. *Personality and Individual Differences, 38*, 87-102.
- Gomez, R., & McLaren, S. (1997). The effects of reward and punishment on response disinhibition, moods, heart rate, and skin conductance level during instrumental learning. *Personality and Individual Differences, 23*, 305-316.
- Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *The American Journal of Psychiatry, 160*, 636-645.
- Gray, J. A. (1982). *The neuropsychology of anxiety*. New York, NY: Oxford University Press.
- Gray, J. A. (1983). Anxiety, personality, and the brain. In A. Gale and J. A. Edwards (Eds.) *Physiological Correlates of Human Behavior, Vol. 3: Individual Differences and Psychopathology*. New York, NY: Academic Press. (pp. 31-43).
- Gray, J. A. (1988). The neuropsychological basis of anxiety. In C. G. Last and M. Hersen (Eds.) *Handbook of Anxiety Disorders*. New York, NY: Pergamon Press. (pp. 10-37).
- Gray, J. A., & McNaughton. (2000). *The Neuropsychology of Anxiety: An Enquiry into the Functions of the Septo-Hippocampal System*. Oxford: Oxford University Press.
- Grisham, J.R., Brown, T.A., Savage, C.R., Steketee, G., & Barlow, D.H. (2007). Neuropsychological impairment associated with compulsive hoarding. *Behaviour Research and Therapy, 45*, 1471-1483.

- Grossman, P., van Beek, J., & Wientjes, C. (1990). A comparison of three quantification methods for estimation of respiratory sinus arrhythmia. *Psychophysiology*, *27*, 702-714.
- Hagopian, L. P., & Ollendick, T. H. (1994). Behavioral inhibition and test anxiety: An empirical investigation of Gray's theory. *Personality and Individual Differences*, *16*, 597-604.
- Hansenne, M., Pinto, E., Scantamburlo, G., Renard, B., Reggers, J., Fuchs, S., et al. (2003). Harm avoidance is related to mismatch negativity (MMN) amplitude in healthy subjects. *Personality and Individual Differences*, *34*, 1039-1048.
- Hayano, J., Sakakibara, Y., Yamada, A., Yamada, M., Mukai, T., Fujinami, T., et al. (1991). Accuracy of assessment of cardiac vagal tone by heart rate variability in normal subjects. *American Journal of Cardiology*, *67*, 199-204.
- Heponiemi, T., Keltikangas-Järvinen, L., Puttonen, S., & Ravaja, N. (2003). BIS/BAS sensitivity and self-rated affects during experimentally induced stress. *Personality and Individual Differences*, *34*, 943-957.
- Herrmann, M.J., Jacob, C., Unterecker, S., & Fallgatter, A.J. (2003). Reduced response-inhibition in obsessive-compulsive disorder measured with topographic evoked potential mapping. *Psychiatry Research*, *120*, 265-271.
- Hundt, N. E., Nelson-Gray, R. O., Kimbrel, N. A., Mitchell, J. T., & Kwapil, T. R. (2007). The interaction of reinforcement sensitivity and life events in the predication of anhedonic depression and mixed anxiety-depression symptoms. *Personality and Individual Differences*, *43*, 1001-1012.
- Kashani, J. H., Orvaschel, H., Rosenberg, T. K., & Reid, J. C. (1989). Psychopathology in a community sample of children and adolescents: A developmental perspective. *Journal of the American Academy of Child and Adolescent Psychiatry*, *28*, 701-706.
- Kessler, R. C., Berglund, B., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of *DSM-IV* disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, *62*, 593-602.
- Kim, M., Kim, Y. Y., Yoo, S. Y., & Kwon, J. S. (2007). Electrophysiological correlates of behavioral response inhibition in patients with obsessive-compulsive disorder. *Depression and Anxiety*, *24*, 22-31.
- Kimbrel, N. A., Nelson-Gray, R. O., & Mitchell, J. T. (2007). Reinforcement sensitivity and maternal style as predictors of psychopathology. *Personality and Individual Differences*, *42*, 1139-1149.

- Knyazev, G. G., Levin, E. A., & Savostyanov, A. N. (2008). Impulsivity, anxiety, and individual differences in evoked and induced brain oscillations. *International Journal of Psychophysiology*, *68*, 242-254.
- Knyazev, G. G., & Slobodskaya, H. R. (2003). Personality trait of behavioral inhibition is associated with oscillatory system reciprocal relationships. *International Journal of Psychophysiology*, *48*, 247-261.
- Landon, J. F., Sher, K. J., & Shah, J. H. (1993). Effects of oxazepam on anxiety: Implications for Fowles' psychophysiological interpretation of Gray's model. *Psychopharmacology*, *113*, 137-143.
- Leslie, J. C., Shaw, D., McCabe, C., Reynolds, D. S., & Dawson, G. R. (2004). Effects of drugs that potentiate GABA on extinction of positively-reinforced operant behavior. *Neuroscience and Biobehavioral Reviews*, *28*, 229-238.
- March, J. S. (1997). Manual for the Multidimensional Anxiety Scale for Children (MASC). San Antonio, TX: Pearson Publication.
- March, J. S., Parker, J. D. A., Sullivan, K. Stallings, P., & Connors, C. K. (1997). The multidimensional anxiety scale for children (MASC): Factor structure, reliability, and validity. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 554-565.
- Marsh, R., Gerber, A. J., & Peterson, B. S. (2008). Neuroimaging studies of normal brain development and their relevance for understanding childhood neuropsychiatric disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*, 1233-1251.
- McNaughton, N. (2006). The role of the subiculum within the behavioural inhibition system. *Behavioural Brain Research*, *174*, 232-250.
- McNaughton, N., & Gray, J. A. (2000). Anxiolytic action on the behavioural inhibition system implies multiple types of arousal contribute to anxiety. *Journal of Affective Disorders*, *61*, 161-176.
- Melzig, C. A., Weike, A. I., Hamm, A. O., & Thayer, J. F. (2009). Individual differences in fear-potentiated startle as a function of resting heart rate variability: Implications for panic disorder. *International Journal of Psychophysiology*, *71*, 109-117.
- Menzies, L., Achard, S., Chamberlain, S.R., Fineberg, N., Chen, C., del Campo, N., et al. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain*, *130*, 3223-3236.

- Mitchell, D. G. V., Rhodes, R. A., Pine, D. S., & Blair, R. J. R. (2008). The contribution of ventrolateral and dorsolateral prefrontal cortex to response reversal. *Behavioural Brain Research, 187*, 80-87.
- Miyawaki, E., & Salzman, C. (1991). Autonomic nervous system tests in psychiatry: Implications and potential uses of heart rate variability. *Integrative Psychiatry, 7*, 21-28.
- Montag, C., Buckholtz, J. W., Hartmann, P., Merz, M., Burk, C., Hennig, J., et al. (2008). COMT genetic variance affects fear processing: Psychophysiological evidence. *Behavioral Neuroscience, 122*, 901-909.
- Muris, P., Merckelbach, H., & Meesters, C. (2001). Learning experiences and anxiety sensitivity in normal adolescents. *Journal of Psychopathology and Behavioral Assessment, 23*, 279-283.
- Niskanen, J., Tarvainen, M. P., Ranta-aho, P. O., Karjalainen, P. A. (2004). Software for advanced HRV analysis. *Computer Methods and Programs in Biomedicine, 76*, 73-81.
- Olantunji, B., & Wolitzky-Taylor, K. B. (2009). Anxiety sensitivity and the anxiety disorders: A meta-analytic review and synthesis. *Psychological Bulletin, 135*, 974-999.
- Penadés, R., Catalán, R., Rubia, K., Andrés, S., Salamero, M., & Gastó, C. (2007). Impaired response inhibition in obsessive compulsive disorder. *European Psychiatry, 22*, 404-410.
- Peterson, R. A., & Heilbronner, R. L. (1987). The anxiety sensitivity index: construct validity and factor analytic structure. *Journal of Anxiety Disorders, 1*, 117-121.
- Pickering, A. D., & Corr, P. J. (2008). J. A. Gray's reinforcement sensitivity theory (RST) of personality. In G. J. Boyle, G. Matthews, & D. H. Saklofske (Eds.) *The SAGE Handbook of Personality Theory and Assessment*. Thousand Oaks, CA: SAGE Publications, Inc. (pp. 239-256).
- Porges, S. W. (2003). Social engagement and attachment: A phylogenetic perspective. In J. A. King, C. F. Ferris, & I. Isreal (Eds.) *Roots of Mental Illness in Children*. New York, NY: New York Academy of Sciences. (pp. 31-47).
- Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology, 74*, 116-143.
- Porges, S. W., Heilman, K. J., Bazhenova, O. V., Bal, E., Doussard-Roosevelt, J. A., & Koledin, M. (2007). Does motor activity during psychophysiological paradigms confound the quantification and interpretation of heart rate and heart rate variability measures in young children? *Developmental Psychobiology, 49*, 485-494.
- Price, R. B., & Mohlman, J. (2007). Inhibitory control and symptom severity in late life generalized anxiety disorder. *Behaviour Research and Therapy, 45*, 2628-2639.

- Putman, P., Hermans, E., & van Honk, J. (2004). Emotional Stroop performance for masked angry faces: It's BAS not BIS. *Emotion, 4*, 305-311.
- Rao, N. P., Reddy, J., Kumar, K. J., Kandavel, T., & Chandrashekar, C. R. (2008). Are neuropsychological deficits trait markers of OCD? *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 32*, 1574-1579.
- Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review, 11*, 141-153.
- Reiss, S., & McNally, R. J. (1985). Expectancy model of fear. In S. Reiss and R. R. Bootzin (Eds.), *Theoretical Issues in Behavior Therapy* (pp. 107-121). Orlando, FL: Academic Press, Inc.
- Reiss, S., Peterson, R.A., Gursky, D.M., & McNally, R.J. (1986). Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. *Behaviour Research and Therapy, 24*, 1-8.
- Reuther, E. T., Davis III, T. E., & Friedman, B. H. (2011). Polyvagal theory and childhood anxiety disorders: Implications for etiological models. Manuscript in preparation.
- Rosso, I. M., Young, A. D., Femia, L. A., & Yurgelun-Todd, D. A. (2004). Cognitive and emotional components of frontal lobe functioning in childhood and adolescence. *Annals of the New York Academy of Sciences, 1021*, 355-362.
- Roth, R.M., Saykin, A.J., Flashman, L.A., Pixley, H.S., West, J.D., & Mamourian, A.C. (2007). Event-related functional magnetic resonance imaging of response inhibition in obsessive-compulsive disorder. *Biological Psychiatry, 62*, 901-909.
- Rottenberg, J. (2007). Cardiac vagal control in depression: A critical analysis. *Biological Psychology, 74*, 200-211.
- Salomon, K. (2005). Respiratory sinus arrhythmia during stress predicts resting respiratory sinus arrhythmia 3 years later in a pediatric sample. *Health Psychology, 24*, 68-76.
- Segarra, P., Ross, S. R., Pastor, M. C., Montañés, S., Poy, R., & Moltó, J. (2007). MMPI-2 predictors of Gray's two-factor reinforcement sensitivity theory. *Personality and Individual Differences, 43*, 437-448.
- Schmidt, N. B., Storey, J., Greenberg, B. D., Santiago, H. T., Li, Q., & Murphy, D. L. (2000). Evaluating gene x psychological risk factor effects in the pathogenesis of anxiety: A new model approach. *Journal of Abnormal Psychology, 109*, 308-320.
- Schmidtke, K., Schorb, A., Winkelmann, G., & Hohagen, F. (1998). Cognitive frontal lobe dysfunction in obsessive-compulsive disorder. *Biological Psychiatry, 43*, 666-673.

- Shucard, J.L., McCabe, D.C., & Szymanski, H. (2008). An event-related potential study of attention deficits in posttraumatic stress disorder during auditory and visual go/nogo continuous performance tasks. *Biological Psychology, 79*, 223-233.
- Stein, M.B., Kennedy, C.M., & Twamley, E.W. (2002). Neuropsychological function in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry, 52*, 1079-1088.
- Suess, P. E., Porges, S. W., & Plude, D. J. (1994). Cardiac vagal tone and sustained attention in school-aged children. *Psychophysiology, 31*, 17-22.
- van der Linden, M., Ceschi, G., Zermatten, A., Dunker, D., & Perroud, A. (2005). Investigation of response inhibition in obsessive-compulsive disorder using the Hayling task. *Journal of the International Neuropsychological Society, 11*, 776-783.
- Vanoli, E., Adamson, P., Ba-Lin, Pinna, G., Lazzara, R., & Orr, W. (1995). Heart rate variability during specific sleep stages. *Circulation, 91*, 1918-1922.
- Willhelm, F. H., Pfaltz, M. C., Gross, J. J., Mauss, I. B., Kim, S. I., & Wiederhold, B. K. (2005). Mechanisms of virtual reality exposure therapy: The role of the behavioral activation and behavioral inhibition systems. *Applied Psychophysiology and Biofeedback, 30*, 271-284.
- Zinbarg, R. E., Barlow, D. H., & Brown, T. A. (1997). Hierarchical structure and general factor saturation of the anxiety sensitivity index: Evidence and implications. *Psychological Assessment, 9*, 277-284.

APPENDIX: TABLE OF DESCRIPTIVES BY GENDER

Table A1. Descriptive Statistics for Dependent Variables by Gender

Dependent Variable	Female <i>M(SD)</i>	Male <i>M(SD)</i>	Total <i>M(SD)</i>
MASC total T-score	57.93 (16.24)	55.62 (11.90)	57.39 (15.28)
Anxiety Sensitivity (AS) total	26.40 (15.22)	21.23 (12.86)	25.20 (14.76)
AS physical concerns	13.07 (8.15)	8.85 (7.22)	12.09 (8.08)
AS psychological concerns	3.42 (3.38)	3.23 (3.44)	3.38 (3.37)
AS social concerns	8.51 (3.81)	8.00 (2.58)	8.39 (3.55)
BIS scale	-1.35 (3.91)	-3.77 (3.77)	-1.91 (3.96)
Day-Night Stroop RT inhibition condition*	844.41 (112.42)	773.94 (81.69)	828.05 (109.58)
Go/No-go RT	440.60 (84.54)	436.78 (102.93)	439.71 (88.16)
Resting RMSSD	31.89 (20.75)	45.09 (39.87)	34.96 (26.59)
Day-Night Stroop Δ RMSSD inhibition	-2.17 (31.60)	6.98 (27.90)	-0.04 (30.78)
Go/No-go Δ RMSSD	0.79 (12.53)	3.44 (27.51)	1.40 (16.92)

Note. * $p < .05$ for difference between anxious and control group in MANOVA analyses. RT = reaction time. Δ = change from resting.

VITA

Erin Tarcza Reuther earned her Bachelor of Science degree in psychology in May 2004 from the University of Florida in Gainesville, Florida. She later earned a Master of Arts degree in general psychology from Southeastern Louisiana University in Hammond, Louisiana, in May 2006. Mrs. Reuther is currently a candidate for the degree of Doctor of Philosophy at Louisiana State University and Agricultural and Mechanical College in Baton Rouge, Louisiana. Her area of specialization is child clinical psychology under the direction of Thompson E. Davis III, Ph.D. Mrs. Reuther has pursued a number of research interests, including brief cognitive behavioral interventions for specific phobias, the relationship between biological markers such as heart rate and anxiety, and the phenomenology of anxiety disorders such as specific phobia, obsessive-compulsive disorder, and posttraumatic stress disorder. Mrs. Reuther is currently a pre-doctoral intern in clinical and health psychology at the Shands Medical Center at the University of Florida in Gainesville, Florida. There she provides clinical services to youth with a variety of psychological and medical conditions through the Psychology Clinic and Behavioral Health Unit in the Psychiatry Department. In July 2011, Mrs. Reuther will begin a post-doctoral fellowship in child psychology in the Psychology Division of the Psychiatry Department at the Louisiana State University Health Sciences Center School of Medicine in her hometown of New Orleans, Louisiana, where she can continue her involvement in both clinical and research activities with child anxiety.