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Cognitive Vulnerability for Social Anxiety and Depression: A Transdiagnostic Investigation of Repetitive Negative Thinkers

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COGNITIVE VULNERABILITY FOR SOCIAL ANXIETY AND DEPRESSION: A TRANSDIAGNOSTIC INVESTIGATION OF REPETITIVE NEGATIVE THINKERS

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In the current study, emotional and cognitive vulnerability factors for social anxiety and depression were examined using an RDoC framework. The overarching goals of the study were to (1) to elucidate the symptom-specific and/or transdiagnostic nature of two cognitive vulnerability factors, interpretation biases and executive control, and (2) to examine the synergistic impact of these cognitive processes on emotional responding and clinical symptoms. To address these aims, the study recruited a sample of individuals at risk for current or future difficulties with social anxiety and depression symptoms (i.e., persons reporting elevated levels of repetitive negative thinking). The study then investigated how social anxiety- and depression-related interpretation biases and deficits in executive control were independently and interactively related to acute social-evaluative stress reactivity and recovery. In addition, the independent and interactive associations between interpretation biases and executive control were examined in relation to dimensional measures of social anxiety and depression symptoms. Findings supported the conceptualization of interpretation biases as transdiagnostic vulnerability factors associated with increased stress reactivity and symptoms of social anxiety and depression. In addition, interpretation biases interacted with executive control to predict elevated stress reactivity and more severe social anxiety symptoms. Future studies are

needed to more closely examine the directionality of these relationships and the possibility that dysregulated acute stress reactivity serves as a mediator between cognitive vulnerability factors and symptoms of social anxiety and depression

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Chapter 1: Introduction

Currently, clinical manifestations of social anxiety and depression are captured by the respective diagnoses of social anxiety disorder (SAD) and major depressive disorder (MDD; American Psychiatric Association [APA], 2013). These disorders are serious and disabling psychiatric conditions, which are highly prevalent, frequently comorbid, and linked with significant economic burden. Beyond the impairments in social role functioning that define SAD, individuals with clinical levels of social anxiety may experience low work productivity, increased financial dependency, and poorer quality of life (Kessler, 2003). Similarly, MDD is characterized by global dysfunction in occupational and psychosocial functioning, as well as increased risk of suicide and mortality from medical illness (Godard, Baruch, Grondin, & Lafleur, 2012; Hirschfeld & Davidson, 1988; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). Lifetime morbid risk for SAD is 18.4% and for MDD is 29.9%, making them two of the top three most commonly occurring anxiety and mood disorders in the United States (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Research also finds that these disorders frequently co-occur (Kessler, Chiu, Demler, & Walters, 2005; Ohayon & Schatzberg, 2010), and that comorbid SAD and MDD is associated with a more severe and chronic course of illness and greater social and occupational impairments (Wittchen, Fuetsch, Sonntag, Müller, & Liebowitz, 2000). Combined, SAD and MDD are estimated to place a burden of as much as $100 billion on the American economy each year (Kessler & Greenberg, 2002; Stewart, Ricci, Chee, Hahn, & Morganstein, 2003).

Given these numbers, it is clear that more research, dedicated to understanding and treating social anxiety and depression, is needed. Research identifying vulnerability

factors that contribute to the onset and maintenance of social anxiety and depression is particularly important, as it may inform etiological models and improve treatment and prevention efforts. From an etiological perspective, understanding individual differences in vulnerability may help to explain (1) why some individuals remain resilient in the face of acute stress, life adversity, or biological predisposition, whereas others go on to develop psychiatric illness, (2) why some individuals develop internalizing (e.g., SAD and MDD) disorders whereas other individuals develop externalizing disorders (e.g., substance use disorders), and (3) why some individuals are prone to develop particular internalizing symptoms over others (e.g., social anxiety versus depression).

Additionally, when considering current treatments for social anxiety and depression, it is well understood that even our most effective psychological and pharmacological interventions do not work all the time or for all individuals (Ehring & Emmelkamp, 2014; Rush et al., 2006). Studying vulnerability for social anxiety and depression may better our understanding of individual differences in treatment outcome, improve our ability to match individuals to particular treatments, and inform treatment augmentation and development efforts. Finally, investigations elucidating risk for the onset of social anxiety and depression symptoms may aid us in identifying individuals who stand to benefit from prevention efforts, and could also highlight novel targets for prevention programs.

Though there are many vulnerability factors that may contribute to the onset and maintenance of social anxiety and depression (e.g., genetics, early life adversity, gender), a large body of literature has documented emotional and cognitive vulnerability among persons with, or at risk for, these symptoms. For instance, individuals who report

elevated levels of negative affect, general distress, and anhedonia have been identified as at risk for the development of both social anxiety and depression (Clark & Watson, 1991; Hughes et al., 2006). Similarly, symptoms of social anxiety and depression are related to patterns of ineffective emotion regulation, characterized by greater reliance on emotional avoidance or suppression and less reliance on cognitive reappraisal (Aldao, Nolen-Hoeksema, & Schweizer, 2010). With regard to cognitive vulnerability, social anxiety and depression have been causally linked with a preference, or bias, toward emotionally congruent information (see Mathews & MacLeod, 2005). Finally, individuals with social anxiety and depression evidence difficulty inhibiting irrelevant cognitions and endorse frequent perseveration on negative thoughts, which may further exacerbate symptom severity (Aldao et al., 2010; Ehring & Watkins, 2008). Taken together, findings provide strong support for a variety of emotional and cognitive factors associated with the onset and maintenance of both social anxiety and depression. Despite this, it remains unclear whether these vulnerability factors confer symptom-specific or transdiagnostic risk for these clinical symptoms, which hampers our efforts to improve etiological models and refine intervention and prevention programs.

Disorder-Non-Specific Vulnerability for Social Anxiety and Depression

Whereas emotional and cognitive vulnerability has been implicated in the etiology and maintenance of social anxiety and depression, much of the extant work has been conducted in two parallel lines of research examining each disorder in isolation from the other. In comparison, relatively little research has examined how emotional or cognitive constructs cut across social anxiety and depression symptoms. This is problematic for two important reasons. First, it limits our ability to conceptualize vulnerability factors as

either symptom-specific or transdiagnostic. Second, there is growing concern that existing diagnoses are not accurately capturing the clinical phenomena for which they were created. As such, over the last eight years, the National Institute of Mental Health (NIMH) has made an explicit shift toward the prioritization of studies examining psycho-bio-behavioral vulnerability for psychopathology using a disorder-non-specific approach (NIMH, 2008; Insel et al., 2010).

The shift toward approaches that do not rely on traditional diagnostic categories, including the diagnoses specified by the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5; APA, 2013) and the International Classification of Diseases, Tenth Revision (ICD-10; World Health Organization [WHO], 1992), has been motivated by several concerns and limitations with these existing classification systems (for a more comprehensive discussion of these limitations, see Lilienfeld, 2014). First, extensive comorbidity across clinical disorders has been well documented within both research and clinical settings. Second, investigators have expressed concern regarding the inadequate construct validity of existing diagnostic categories. This concern is rooted in basic scientific findings from investigations of genetic and neurobiological pathways that often fail to differentiate clinical disorders from each other, as well as intervention studies documenting that many disorders respond to the same psychological and pharmacological interventions. Third, researchers have highlighted the considerable heterogeneity that exists within diagnostic categories. DSM-5 and ICD-10 diagnoses are often assigned when an individual presents with a select number of symptoms from a longer list (e.g., MDD only requires the presence of five out of nine symptoms; APA, 2013). As such, individuals receiving the same diagnosis may have vastly different phenotypic

presentations. These three concerns have called into question the extent to which existing diagnostic categories truly represent discrete entities and whether the boundaries between these discrete entities have been correctly identified. Finally, the current classification systems differentiate between illness and health using a categorical approach that relies on set (and arguably arbitrary) cut-points. In some instances, these cut-points have been so ineffective that they have left the majority of functionally impaired individuals with "not otherwise specified" (NOS) diagnoses (Fairburn & Bohn, 2005). Notably, the use of a cut-point assumes that it is possible to differentiate valid disease from normality. However, in many instance, research has not supported this assumption, finding instead that clinical symptoms and associated vulnerability factors are dimensionally distributed throughout the general population (Cuthbert & Kozack, 2013).

Each of these limitations to the existing classification system can be applied specifically to the extant research on social anxiety and depression. Approximately 20% of individuals with SAD also meet criteria for MDD (Kessler et al., 2005; Ohayon & Schatzberg, 2010). Considering this rate of comorbidity in conjunction with the documented heterogeneity that exists within these disorders (Chen, Eaton, Gallo, & Nestadt, 2000; Vriends, Becker, Meyer, Michael, & Margraf, 2007), the assumption that SAD and MDD are discrete diagnostic entities must be called into question. Indeed, rather than functioning as distinct diagnostic constructs, research on social anxiety and depression suggests that they are better conceptualized as two indicators of an underlying *internalizing* construct, which is dimensionally distributed throughout the general population (Haslam, Holland, & Kuppens, 2012; McGlinchey & Zimmerman, 2007). Further, results obtained from experimental psychopathology and clinical neuroscience

studies often fail to differentiate individuals with SAD from those with MDD. For instance, findings from magnetic resonance imaging (MRI) and functional MRI studies indicate that certain structural abnormalities, as well as patterns of blunted reactivity to positive stimuli, characterize patients with depression and patients with social anxiety (van Tol et al., 2010; van Tol et al., 2012). Lastly, social anxiety and depression respond to similar psychological (e.g., cognitive behavioral therapy; Barlow, Allen, & Choate, 2004) and pharmacological (e.g., selective serotonin reuptake inhibitors; Gorman & Kent, 1999) interventions. Taken together, these results support the conclusion that the diagnostic labels of SAD and MDD are not effectively capturing the underlying psycho-bio-behavioral dysfunction occurring among individuals who receive these diagnoses. Knowing this, it is essential that researchers adopt innovative approaches to studying individuals with symptoms of social anxiety and depression that do not rely on the existing categorical systems (Insel et al., 2010).

Despite the increasing value placed on investigations employing a disorder-non-specific and dimensional approach to the study of psychopathology, the ideal methods for conducting these types of investigations have remained elusive. Our traditional approach to experimental psychopathology has been to recruit a sample of individuals who meet diagnostic criteria for a particular disorder (e.g., SAD or MDD) and then to study our research domains of interest (e.g., emotional or cognitive vulnerability) within the sample in order to better understand the illness. For all the reasons stated above, this approach may be failing to advance our scientific understanding of social anxiety and depression and may inhibit the development of effective intervention and prevention programs.

The NIMH's Research Domain Criteria (RDoC) initiative offers an alternative methodological approach. The primary aim of the RDoC initiative is to provide new ways of classifying mental illness based on observable psycho-bio-behavioral measures that are dimensional in nature and agnostic to existing diagnostic categories. Subsumed within this aim are several specific goals (see Sanislow, Pine, Quinn, Kozack, & Garvey, 2010), including: (1) uniting basic and clinical scientists in the effort to identify behavioral mechanisms of dysfunction that cut across several clinical disorders and may be more amenable to neuroscientific approaches; (2) developing valid and reliable measures of behavioral mechanisms of dysfunction; (3) elucidating the full range in variation for identified psycho-bio-behavioral factors, thereby improving our ability to differentiate between wellness and illness; and (4) more fully integrating several units of analysis, including genetics, neurobiology, cognition, behavior, social processes, and subjective experience.

To provide a framework for this type of research, NIMH workgroups developed a matrix in which several research domains were broken down by units of analysis. Specifically, the research domains considered to be most appropriate for RDoC investigations included cognitive systems (e.g., cognitive control), negative valence systems (e.g., acute threat), positive valence systems (e.g., approach motivation), arousal and regulatory systems (e.g., reactivity and recovery), and social processes (e.g., perception and understanding of others; Badcock & Hugdahl, 2014). Units of analysis include genes, molecules, cells, circuits, physiology, behavior, self-report, and paradigms (Cuthbert & Kozack, 2013; Kozack & Cuthbert, 2016). The research domains and units of analysis identified by NIMH workgroups are not an exhaustive list, rather they were

intended to guide researchers in efforts to study biomarkers, behaviors, or symptoms that cut across many disorders. An example of an RDoC-consistent methodological approach is presented in Figure 1.1. In line with the primary aim and specific goals of the initiative, this approach involves identifying a *shared mechanism of dysfunction* that can be used as a selection criterion, then studying one's *research domains of interest*, including an investigation how research domains relate to each other, as well as how they relate to *dimensional measures of clinical symptoms*.

Identifying a Shared Mechanism of Dysfunction for Social Anxiety and Depression

The first step in applying this RDoC framework to research investigating emotional and cognitive vulnerability for social anxiety and depression was to identify a relevant *mechanism of dysfunction* to use as a selection criterion. Broadly speaking, this aspect of RDoC methodology has proved to be particularly challenging for the field. It has been made clear that sampling from multiple disorders (e.g., recruiting individuals with SAD, MDD, or both disorders) or from broad clinical contexts (e.g., any individual presenting for outpatient psychotherapy) is not sufficient, as the range of phenotypic presentations included in the sample may be too limited (Kozack & Cuthbert, 2016). Instead, it is recommended that sampling be based on a mechanism of dysfunction that is relevant to the research question at hand and which has been linked to both the research domains and clinical symptoms of interest. In addition, efforts to select control participants change markedly within RDoC investigations; sampling should not involve comparisons between the disordered and the well or between individuals with and without a given mechanism of dysfunction. Rather, recruitment should produce a sample with a broad range of scores across research domains and clinical symptoms of interest.

Doing so allows us to develop the strongest understanding of variation in a given construct and to more accurately discriminate individuals with and without pathological functioning (Kozack & Cuthbert, 2016; Sanislow et al., 2010).

Thus, in choosing a mechanism of dysfunction within the current study, it was critical that three conditions were met: the factor needed to be related to research domains and clinical symptoms of interest, the factor needed to be dimensionally distributed throughout the population, and the factor needed to be easy to employ as a screening tool. Repetitive negative thinking met each of these conditions.

Repetitive negative thinking has been conceptualized as frequent, perseverative, and uncontrolled cognitive activity that is focused on the negative aspects of past and future events, one's current situation, and/or one's emotional state and psychological symptoms (Ehring & Watkins, 2008; Mahoney, McEvoy, & Moulds, 2012). Though repetitive negative thinking is a core cognitive process and a common human experience, in extreme presentations it is also predictive of psychopathology (Mennin & Fresco, 2013).

Notably, repetitive negative thinking is a broad umbrella term, which encompasses several key constructs, including rumination, post-event processing, and worry (Ehring & Watkins, 2008). These constructs are associated with a wide range of psychological difficulties, including but not limited to social anxiety and depression symptoms (see Brozovich & Heimberg, 2008, and Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008, for reviews). Repetitive negative thinking has also been causally linked to comorbid anxiety and depression symptoms. Research has found, for instance, that rumination fully mediates the relation between anxiety and depression, as well as the

relation between depression and anxiety, in both cross-sectional and longitudinal models (McLaughlin & Nolen-Hoeksema, 2011).

Whereas much of the extant literature has sought to understand how specific aspects of repetitive negative thinking relate to specific disorders (e.g., rumination in individuals with MDD; post-event processing in individuals with SAD), research has begun to consider repetitive negative thinking as a transdiagnostic construct associated with the onset and maintenance of both types of clinical symptoms. As an example, findings indicate that individuals with SAD and MDD report similar levels of brooding and reflective pondering (two types of rumination), as well as similar levels of worry (McEvoy, Watson, Watkins, & Nathan, 2013). Additionally, using structural equation modeling in two discrete samples recruited through Amazon's Mechanical Turk, Arditte, Shaw, and Timpano (in press) found that several aspects of repetitive negative thinking, including rumination, post-event processing, and dampening of positive affect, loaded onto a single latent factor. Results of this latter study also revealed that the latent factor was positively correlated with symptoms of social anxiety and depression.

Beyond associations with clinical symptoms, repetitive negative thinking has been linked to several psycho-bio-behavioral vulnerability factors. In keeping with an RDoC perspective, repetitive negative thinking has been linked to both upstream (e.g., biological) and downstream (e.g., behavioral) processes (Woody & Gibb, 2015). For example, greater reliance on repetitive negative thinking following an acute social-evaluative laboratory stressor has been linked with prolonged subjective and physiological reactivity, including elevated reactivity of the sympathetic nervous system and blunted parasympathetic arousal (Brosschot & Thayer, 2006; Key, Campbell, Bacon,

& Gerin, 2008; LeMoult, Arditte, D'Avanzato, & Joormann, 2013; Woody & Gibb, 2015). Furthermore, there is a large body of literature linking elevated repetitive negative thinking with cognitive vulnerability factors, including difficulties with attention, concentration, and memory, deficits in executive control, and cognitive biases for negatively valenced stimuli (see Nolen-Hoeksema et al., 2008).

Finally, it should be noted that repetitive negative thinking is typically assessed via brief self-report measures, such as the Ruminative Responses Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003), Post-Event Processing Scale-Revised (PEPQ-R; McEvoy & Kingsep, 2006), or Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). Conducting assessments of repetitive negative thinking is, therefore, time-efficient and cost-effective, making this construct easy to use as a selection criterion in large samples of potential participants.

Taken together, the literature provides solid support for repetitive negative thinking as a mechanism of dysfunction that is dimensionally distributed throughout the general population, relevant to social anxiety and depression, and associated with several other emotional and cognitive vulnerability factors. In addition, because the construct is typically assessed via brief self-report measures, screening potential participants for elevated levels of repetitive negative thinking is fast, easy, and inexpensive.

Selecting the Research Domains of Interest

The second step in applying the RDoC framework to the investigation of vulnerability for social anxiety and depression was to select the *research domains of interest*. As discussed above, several emotional and cognitive processes have been theoretically and empirically linked with social anxiety and depression symptoms. These

vulnerability factors include dysregulated stress responding, cognitive biases, and deficits in executive control. Still, several key questions about the nature of these vulnerability factors in relation to symptoms of social anxiety and depression remain unanswered. Presented below is a review of the extant research on each of the three research domains included in the current investigation. A discussion of the limitations of the extant literature is presented in section entitled, "Limitations to the Extant Research."

Acute stress responding. Supported by an extensive body of literature, acute stress responding is well established as a vulnerability for psychopathology (Gutman, 2011; Claes, 2004; McEwen, Eiland, Hunter, & Miller, 2012; Kessler, Price, & Wortman, 1985). Diathesis-stress models posit that the manner in which individuals respond to stressful life events influences their risk for the development and maintenance of clinical symptoms (Flynn & Rudolph, 2007; Schmidt & Fox, 1999). More specifically, individuals who respond to stress with negative emotionality, elevated sympathetic arousal, and/or blunted parasympathetic activity may be particularly prone to long-term emotional difficulties. This is because individuals who experience dysregulated acute stress responding are also more likely to experience a myriad of other negative outcomes, including prolonged negative affect, maladaptive patterns of cognition (e.g., negative appraisals), and interfering behaviors (e.g., avoidance or hypervigilance) known to contribute to the development and maintenance of psychopathology, including symptoms of social anxiety and depression (Jamieson, Mendes, & Nock, 2013).

Though stress can occur in a variety of contexts, stress activated by social-evaluative situations may be especially relevant to individuals with, or at risk for, social anxiety and depression (Denson, Spanovic, & Miller, 2009). According to cognitive

theory, social-evaluative stress is most likely to arise in acute contexts in which one perceives his or her social status to be threatened (e.g., when giving a presentation to classmates or coworkers). Social-evaluative stress then triggers symptoms of social anxiety and/or depression by activating cognitive appraisals (*I am making a fool of myself*) and latent schemas (*others perceive me as incompetent*) that contribute to symptom onset (Beck, 1967; Rapee & Heimberg, 1997). Moreover, cognitive models posit that symptom exacerbation occurs when social-evaluative stress increases vigilance for threat-relevant cues. For example, someone who has appraised that he is making a fool of himself may be overly attentive to judgmental facial expressions, such as those communicating anger or disgust. He may also be more likely to interpret ambiguous facial expressions in a judgmental manner.

As with other forms of acute stress, dysfunctional responding to social-evaluative situations has been characterized by increased negative emotionality and dysregulated physiological responding (Burke, Davis, Otte, & Mohr, 2005; Kudielka, Hellhammer, & Kirschbaum, 2007; Sharpley, 2002). Whereas negative emotionality is most often captured through self-report of subjective experience, there are several indices used to measure psychophysiological responding. Importantly, heart rate variability, or the variation in time between heart beat intervals, may be particularly relevant to the study of social anxiety and depression. Heart rate variability is an index of parasympathetic arousal that has been theoretically and empirically linked to stress and emotion dysregulation (Rottenberg, Wilhelm, Gross, & Gotlib, 2003). More specifically, low heart rate variability is considered an index of parasympathetic withdrawal, which may lead to elevated reactivity and/or prolonged recovery from stress (Key et al., 2008).

Research indicates that individuals with social anxiety and depression are more likely to exhibit physiological profiles characterized by dominance of the sympathetic nervous system and withdrawal of the parasympathetic nervous system (Sharpley, 2002; Woody & Gibb, 2015). Individuals with social anxiety and depression exhibit lower levels of resting heart rate variability than their healthy counterparts (Hughes & Stoney, 2000; Pittig, Arch, Lam, & Craske, 2013). Similarly, low heart rate variability during acute social-evaluative stress has been correlated with dimensional measures of anxiety and depression symptoms (Shinba et al., 2008; Verkuil, Brosschot, & Thayer, 2014).

Of note, most of the extant experimental research on social-evaluative stress responding has focused on dysfunctional *reactivity* to a stressor. However, prolonged *recovery* from social-evaluative stress may also be implicated in risk for social anxiety and depression (Flynn & Rudolph, 2007; Key et al., 2008; LeMoult et al., 2013; Nolen-Hoeksema et al., 2008). Therefore, in the current study, dysfunctional acute stress responding was assessed using a multi-modal method, including measures of negative emotionality and heart rate variability during both reactivity to and recovery from an acute social-evaluative laboratory stressor.

Despite all that is known about dysfunctional patterns of acute stress responding, it remains unclear why some people are prone to aberrant stress responding while others are not. As discussed above, cognitive theory posits that individual differences in cognitive processes influence stress responding and set the stage for difficulties with social anxiety and depression (Beck, 1967; Rapee & Heimberg, 1997). Empirical research supports cognitive theory, finding that specific cognitive processes, including cognitive biases and deficits in executive control, predict dysregulated subjective and

physiological responding to acute social-evaluative stress (Joormann, Waugh, & Gotlib, 2015; Mathews & MacLeod, 2005; MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002; Williams, Suchy, & Rau, 2009; Wilson, MacLeod, Mathews, & Rutherford, 2006).

Cognitive biases. Cognitive biases, defined as the preferential processing of emotionally salient stimuli, have been well documented within multiple areas of cognition, particularly attention, memory, and the automatic interpretation of ambiguous stimuli. In addition, biased cognition is thought to play an important role in the onset and maintenance of social anxiety and depression (see Mathews & MacLeod, 2005). Imagine, for example, that it is Saturday afternoon and a woman with depression is walking down the street when she sees a friendly acquaintance walking towards her. The acquaintance approaches then passes the woman without making eye contact or saying hello. Research indicates that this woman is likely to interpret this ambiguous social interaction in a mood-congruent way, perhaps thinking to herself "nobody ever notices me" (*interpretation bias*; Cowden Hindash & Amir, 2012). Subsequently, she may selectively remember confirmatory past experiences, such as the time when she felt ignored at a recent social gathering (*memory bias*; Whalley, Rugg, & Brewin, 2012). As the woman continues walking down the street, she may attend to environmental stimuli consistent with her thoughts and emotions (*attention bias*); she may thus notice the two other people who do not smile as she passes, while the three people who do smile remain unobserved (Kellough, Beevers, Ellis, & Wells, 2008). Illustrated in this way, it is easy to see how cognitive biases exacerbate and maintain negative affect or clinical symptoms.

Research has linked cognitive biases to several units of analysis, ranging from neurobiology to subjective experience of social anxiety and depression. Neuroimaging studies find that negative cognitive biases are related to hyper-responsivity of the amygdala (Bishop, 2007), supporting the notion that these biases are bottom-up vulnerabilities that set the stage for elevated negative affectivity. In addition, studies find that cognitive biases are causally associated with greater stress reactivity (MacLeod et al., 2002), current symptom severity (Beard & Amir, 2008), and risk of symptom recurrence (Browning, Holmes, Charles, Cowen & Harmer, 2012). In sum, cognitive biases are easily linked with upstream and downstream processes, making them an appropriate research domain for RDoC investigations.

Within the context of the current study, particular emphasis was placed on the bias to interpret ambiguous situations in an emotionally congruent manner. From a theoretical perspective, interpretation biases are thought to trigger repetitive negative thinking among individuals with or at risk for social anxiety and depression. When presented with an ambiguous social-evaluative encounter, these individuals may engage in repetitive negative thinking as a form of self-evaluation that serves to identify upward counterfactual thoughts (i.e., "if only I…" statements that can be used to inform future behavior; Kocovski, Endler, Rector, & Flett, 2005). Despite the perceived utility of repetitive negative thinking in response in social-evaluative situations, use of this strategy over time can further exacerbate negative interpretation biases. Indeed, research has found that individuals high in trait rumination are more likely to make rumination-consistent interpretations of ambiguous situations (Mor, Hertel, Ngo, Shacher, & Redak,

2014). As the cycle between negative interpretation biases and repetitive negative thinking continues, symptoms of social anxiety and depression are likely to worsen.

Executive control. In order to break the cycle between negative interpretation biases and repetitive negative thinking, individuals must be able to effectively disengage from negative thoughts when they are no longer helpful or informative. This process requires top-down regulation in the form of executive control. Broadly speaking, executive control includes a large set of cognitive functions (e.g., the ability to update working memory, shift between tasks or mental sets, and inhibit prepotent impulses) that serve to flexibly regulate one's thoughts, behaviors and emotions (Miyake & Friedman, 2012). However, deficits in the ability to inhibit irrelevant information from one's working memory may be particularly relevant to individuals with high levels of repetitive negative thinking.

For example, in her attentional inhibition theory of rumination, Linville (1996) proposed that attentional inhibition is an automatic cognitive process that serves to limit irrelevant environmental stimuli and internal thoughts that interfere with goal-directed behavior. When deficits in attentional inhibition exist, irrelevant information, such as repetitive negative thoughts, may enter working memory and interfere with goal pursuit. Empirical research supports this theory, finding that individuals high in trait-level rumination are more likely to demonstrate impairments in their ability to inhibit task-irrelevant information (Joormann, 2006; Zetsche & Joormann, 2011).

Notably, impairments in behavioral measures of inhibitory functioning have been linked with reduced activity in areas of the prefrontal cortex associated with top-down efforts to regulate emotional reactivity (Bishop, 2007). Individuals with difficulties in this

area demonstrate heightened subjective and biological reactivity to social-evaluative stressors (Hendrawan, Yamakawa, Kimura, Murakami, & Ohira, 2012). They are also at increased risk for the onset and maintenance affective disorders (e.g., Joormann, 2010). Thus, as with cognitive biases, deficits in executive control can be connected with upstream and downstream processes making executive control an appropriate cognitive vulnerability to study using RDoC methodology.

Including Dimensional Measures of Clinical Symptoms

The final step involved in applying the RDoC framework to research on social anxiety and depression is to examine the ways in which emotional and cognitive vulnerability factors relate to *dimensional measures of clinical symptoms*. Though relatively straightforward from a methodological standpoint, it is a critical component of RDoC methodology. The vast majority of previous research in this area has utilized a categorical approach by examining individuals with and without SAD or MDD. Including dimensional assessments of clinical symptoms, the current investigation was able to examine emotional and cognitive vulnerability for social anxiety and depression without relying on the faulty assumptions underlying the existing classification systems.

Limitations to the Extant Research

Whereas there is a wealth of literature linking dysregulated stress responding, interpretation biases, and deficits in executive control to the development and maintenance of social anxiety and depression symptoms, two key questions remain unanswered. First, our understanding of the identified cognitive processes as either transdiagnostic or symptom-specific vulnerability factors remains unclear. Some research has found general deficits in executive control across individuals with social anxiety and

depression, whereas other research has found evidence for specific deficits associated with the processing of symptom-congruent material (Schmid, Kleiman, Amodio, 2015, but Najmi, Cowden Hindash, & Amir, 2010). In contrast, most research on cognitive biases has assumed that they are emotion-specific (Mathews & MacLeod, 2005). Specifically, studies have sought to document an association between anxiety symptoms and threat-related cues or between depression and dysphoric cues. Because little research has examined cognitive biases in relation to both social anxiety and depression symptoms, it remains unclear whether such biases are symptom-specific. Are individuals with social anxiety only biased toward resolutions of ambiguous information that convey social threat? Can we expect individuals with depression to only be biased toward self-referent, mood-congruent interpretations? Elucidating our understanding of these processes as either transdiagnostic or symptom-specific vulnerability factors will significantly contribute to our theoretical understanding of the link between cognition, emotion, and psychopathology and will inform neurobehavioral interventions for social anxiety and depression.

Second, it remains unknown whether interpretation biases and executive control interact with each other to predict acute stress responding or clinical symptoms of social anxiety and depression. As reviewed by Bishop (2007), a neural circuit inclusive of bottom-up hyper-responsivity of the amygdala and top-down hypo-responsivity of the prefrontal cortex underlies emotion and cognitive dysregulation in the internalizing disorders. The hypothesis that concurrent difficulties with bottom-up and top-down cognitive processes confer the greatest vulnerability for social anxiety and depression has been further supported by preliminary evidence from behavioral studies (De Lissnyder et

al., 2012; Quinn & Joormann, 2015a; Quinn & Joormann, 2015b). Despite this, previous research has almost invariably examined interpretation biases in isolation from executive control. As the field moves toward conceptualizing psychopathology based on difficulties across the psycho-bio-behavioral continuum (Insel et al., 2010), it is essential to gain an understanding of how these two areas of cognition interact to produce dysfunction and vulnerability for social anxiety and depression. More specifically, examining how these processes uniquely or interactively predict stress reactivity and symptoms of social anxiety and depression could extend existing etiological and maintenance models of these clinical conditions.

Given the abovementioned gaps in the extant literature, the proposed project examined the independent and interactive associations between cognitive processes in relation to acute emotional reactivity to and recovery from social-evaluative stress. In addition, the study examined the relations among cognitive processes and symptoms of social anxiety and depression.

The Current Study

As reviewed above, using a disorder-non-specific approach to investigate emotional and cognitive vulnerability for social anxiety and depression has the potential to improve our existing classification system by informing transdiagnostic and symptom-specific conceptualizations of these conditions and by laying an empirical foundation for future intervention and prevention efforts. Despite the value of such work, the optimal method for studying disorder-non-specific vulnerability for social anxiety and depression remains unclear. Notably, the NIMH's RDoC initiative (Cuthbert & Kozack, 2013; Insel et al., 2010; Kozack & Cuthbert, 2016; Sanislow et al., 2010) provides a novel

framework for this type of research. First, rather than recruiting individuals with a particular "disorder," investigations can recruit individuals based on a relevant mechanism of dysfunction. Investigators can then examine their research domains of interest, as they would have before, but including an assessment of how research domains relate to dimensional measures of clinical symptoms.

The current study employed these RDoC methods to investigate emotional and cognitive vulnerability for social anxiety and depression. Given the strong empirical evidence linking repetitive negative thinking to the onset and maintenance of social anxiety and depression symptoms (Arditte et al., in press; Brozovich & Heimberg, 2008; Nolen-Hoeksema et al., 2008), this construct was selected as the shared mechanism of dysfunction. Within a sample of individuals reporting high levels of repetitive negative thinking, the investigation then examined three research domains, including (1) acute stress responding, (2) interpretation biases, and (3) executive control, as they related to each other and to dimensional measures of social anxiety and depression symptoms.

The selection of these three research domains was based upon their relevance to the social anxiety and depression literatures. For example, stress response profiles, characterized by hyperreactivity to and prolonged recovery from social-evaluative stress, have been found to play an important role in the onset and maintenance of clinically impairing social anxiety and depression (Burke et al., 2005; Kudielka et al., 2007, Sharpley, 2002). Still, it remains unclear why certain people are prone to aberrant stress responding, whereas others are not. Individual differences in cognitive processes may help to explain this variability in stress responding. Indeed, cognitive processes (e.g., cognitive biases, executive control) have been theoretically and empirically linked to

heightened subjective and biological stress reactivity (Beck, 1967; Hendrawan et al., 2012; MacLeod et al., 2002). Of note, these same cognitive factors may also influence the presence, persistance, and severity of social anxiety and depressive symptoms (Beard & Amir, 2008; Browning et al., 2012; Joormann, 2010).

From the extant literature, it is also clear that several key questions remain unanswered. Are certain patterns of cognition differentially related to social anxiety versus depressive symptoms? Do cognitive processes interact with each other to influence acute stress responding? Addressing these questions will be instrumental to our theoretical understanding of vulnerability for dysregulated stress responding and specific symptom profiles. Moreover, given the recent emergence of interventions targeting cognitive processes (e.g., cognitive bias modification and other neurobehavioral interventions; Siegle, Ghinassi, & Thase, 2007), studying the ways in which these processes uniquely and interactively relate to acute stress responding and symptom severity may have novel implications for the treatment of social anxiety and depression.

Thus, the current study examined social anxiety- and depression-related interpretation biases and deficits in executive control, as they independently and interactively predicted responding to an acute social-evaluative stressor. The independent and interactive associations between cognitive processes were also examined in relation to symptoms of social anxiety and depression in order to determine whether they represented transdiagnostic or symptom-specific vulnerability factors.

Aims and Hypotheses

Aim 1. To examine the independent and interactive associations between cognitive processes in relation to acute *emotional reactivity* to stress.

Hypothesis 1.1. It was predicted that interpretation biases would be related to subjective (i.e., self-reported negative affect) and biological (i.e., heart rate variability) stress reactivity, such that social anxiety- and depression-related biases would predict heightened subjective and blunted biological responding. No differences between social anxiety- and depression-related biases and their relations with stress reactivity were expected to emerge.

Hypothesis 1.2. Executive control would be related to subjective and biological stress responding, such that poorer executive control would be associated with greater subjective and lesser biological stress responding.

Hypothesis 1.3. Interpretation biases and executive control were expected to interact to predict subjective and biological stress reactivity. Specifically, individuals who displayed greater interpretation biases and poorer executive control were expected to evidence the strongest subjective and biological stress response.

Aim 2. To examine the independent and interactive associations between cognitive processes in relation to acute *emotional recovery* from stress.

Hypothesis 2.1. It was hypothesized that interpretation biases would be related to subjective and biological stress responding, such that social anxiety- and depression-related biases would be associate with a greater residual stress response following a recovery period. Again, no differences between social anxiety- and depression-related biases and their relations with stress recovery were expected to emerge from analyses.

Hypothesis 2.2. Executive control would be related to subjective and biological stress responding, such that poorer executive control would be associated with a greater residual subjective and biological stress response following the recovery period.

Hypothesis 2.3. It was expected that interactions between interpretation biases and executive control would be associated with recovery from the stressor. Specifically, individuals who displayed greater interpretation biases and poorer executive control were expected to experience the greatest residual subjective and biological stress response.

Aim 3. To examine the independent and interactive associations between cognitive processes in relation to symptoms of social anxiety and depression.

Hypothesis 3.1. It was expected that social anxiety-related interpretation biases would be uniquely associated with symptoms of social anxiety, but not depression. In contrast, depression-related interpretation biases were expected to be uniquely associated with symptoms of depression, but not social anxiety.

Hypothesis 3.2. It was predicted that executive control would be inversely associated with both social anxiety and depression, even after controlling for the comorbidity between social anxiety and depression symptoms.

Hypothesis 3.3. Interpretation biases and executive control were expected to interact to predict clinical symptoms. Specifically, individuals with a social anxiety-related interpretation bias and poorer executive control were expected to endorse the highest levels of social anxiety symptoms, whereas individuals with depression-related interpretation biases and poorer executive control would endorse the highest levels of depression symptoms.

Chapter 2: Method

Participants

Eligibility criteria. The study recruited a sample of participants between the ages of 18 and 65 who were fluent in English, had access to the Internet, and were willing to provide informed consent. There were no gender, race, or ethnicity restrictions. Individuals were excluded from participation if they self-reported 1) a current or lifetime diagnosis of a psychotic or bipolar disorder, 2) a current substance use disorder, 3) an organic mental or developmental disorder, or 4) current homicidality or suicidality.

Study eligibility also required that participants report elevated levels of repetitive negative thinking in response to stress. This was assessed using the Repetitive Negative Thinking (RNT) subscale of the Repetitive Thinking Questionnaire (RTQ; McEvoy, Mahoney, & Moulds, 2010). Previous research was used to select an appropriate cutoff for *elevated* repetitive negative thinking. In a study by Mahoney and colleagues (2012), a sample of 186 outpatients was recruited from a specialty clinic (42.9% of participants received a primary diagnosis of SAD, 19.4% received a secondary diagnosis of SAD, 5.4% received a primary diagnosis of a depressive disorder, and 40.3% received a secondary diagnosis of a depressive disorder). Across participants, the average RNT subscale score was an 86.96 (SD = 23.87), which the authors noted, was markedly higher than that found within the student sample (M = 71.97, SD = 22.02; McEvoy et al., 2010). Based on these findings, eligibility for the current study required participants to achieve a score at or above the previously identified clinical mean (i.e., ≥ 87).

Recruitment. Participants were recruited from the community via flyers, ads posted on online forums and in local newspapers, and referrals from University of Miami

clinics and/or research laboratories. After receiving a brief description of the study, its purpose, and procedures, interested persons were provided with a link to an online survey, which assessed study inclusion and exclusion criteria. Recruitment data were collected using Qualtrics Survey Software (Qualtrics, 2015). Upon receipt of survey responses, eligible participants were contacted and scheduled for an appointment.

In addition, some participants were recruited from an introductory psychology course. During a pre-screening session, students completed the RNT subscale. Those who scored at or above the cutoff of 87 were then eligible to sign up for a laboratory session. At the time of this appointment, participants were assessed for all other abovementioned selection criteria and included or excluded from the study as appropriate.

Participant characteristics. A sample of $N = 57$ individuals ($n = 31$ recruited from the community and $n = 23$ students) was recruited for participation. Demographic characteristics, including age, gender, race, and ethnicity, for the sample as a whole and within each of the two subsamples are presented in Table 2.1. On average, participants were approximately 30 years old and the majority of the sample was female. This is consistent with previous research on repetitive negative thinking, and particularly depressive rumination (e.g., Charles & Carstensen, 2008; Johnson & Whisman, 2013; Nolen-Hoeksema & Jackson, 2001).

As can be seen from the group-wise comparisons in Table 2.1, participants recruited from the community did not differ from student participants with regard to gender and race. However, community participants were more likely to be older and to identify as Hispanic as compared to student participants. To ensure that findings were not

influenced by these demographic differences, age and ethnicity were included as covariates in analyses.

Table 2.1 also presents the basic clinical characteristics of the sample, including their RNT subscale scores, the percentage of participants who endorsed a current or lifetime diagnosis of MDD or SAD, and the percentage of participants currently prescribed psychotropic medication, blood pressure medication, or any other medication (e.g., allergy medication, antibiotics, etc.). When examining psychological constructs across community and student subsamples, a consistent pattern emerged, indicating that community participants were more severely symptomatic than their student counterparts. Community participants had higher levels of repetitive negative thinking and were more likely to endorse a diagnosis of MDD, SAD, and/or the use of psychotropic medications. Despite these differences, RNT subscale scores were normally distributed in each of the two subsamples, when considered separately, and the range of RNT subscale scores (87 – 135) was identical across both the community and student participants. When the two subsamples were combined, RNT scores remained normally distributed (skew = .43, *SE* = .32; kurtosis = -1.08, *SE* = .62), and no outliers were identified. No differences between community and student participants were found with regard to the use of blood pressure or other miscellaneous medications.

Evaluations and Procedures

Screening for elevated repetitive negative thinking. As discussed above, repetitive negative thinking was assessed during an online screening questionnaire using the RNT subscale of the RTQ.

Repetitive Thinking Questionnaire (RTQ; McEvoy et al., 2010). The 27-item RNT subscale was developed as a transdiagnostic measure of repetitive negative thinking. It was created by selecting relevant items from existing measures of similar constructs, including the RRS (Treynor et al., 2003), PEPQ-R (McEvoy & Kingsep, 2006), and PSWQ (Meyer et al., 1990), and then modifying items to remove symptom-specific language. Instructions for the RNT subscale asked participants to anchor their responses to a recent distressing event. Participants are then asked to rate each item on a 5-point Likert scale with anchors 1 (*Not true at all*) to 5 (*Very true*), based on the degree to which the item applied to them during the period of time following their identified distressing event. Items are summed to create a total score, ranging from 27 to 135, with higher scores indicating greater repetitive negative thinking in response to stress. The RNT subscale has been previously validated within both student and clinical samples (Mahoney et al., 2012; McEvoy et al., 2010). Internal consistency in the current study was good ($\alpha = .87$ across all cases; $\alpha = .89$ for community participants; $\alpha = .78$ for student participants). A copy of the RNT subscale is included within the Appendix.

Online questionnaires. Once eligible participants had been scheduled for an appointment to complete the study's laboratory session, they were provided with access to a second online survey. Data were again collected using Qualtrics Survey Software (Qualtrics, 2015) and the survey took approximately 20 minutes to complete. Within the survey, participants were asked to report on their current symptoms of social anxiety and depression, to complete an assessment of crystallized intelligence, and to provide basic demographic and health information. Descriptions of the measures assessing clinical symptoms and crystallized intelligence are provided below.

***Brief Fear of Negative Evaluation Scale – Straightforward Items* (BFNE-S; Rodebaugh et al., 2004).** Fear of negative evaluation by others is a defining symptom of SAD (APA, 2013). One of the most widely used measures of this fear is the 12-item, Brief Fear of Negative Evaluation Scale (BFNE; Leary, 1983). However, several variations of the BFNE also exist (Taylor, 1993; Carleton, Collimore, & Asmundson, 2007). For instance, Rodebaugh and colleagues (2004) recommended the use of an 8-item variation of the measure, including only the straightforwardly worded items of the original BFNE (BFNE-S). This recommendation was based on a factor analysis showing that the four reverse coded items loaded onto a separate factor from the other eight items, as well as results indicating that the straightforwardly worded items demonstrated stronger convergent validity. More recent research also supported the use of the BFNE-S, finding it more parsimonious and psychometrically superior to a 12-item variant (Carleton, Collimore, McCabe, & Antony, 2011).

Instructions for the BFNE-S asked participants to rate the extent to which items were characteristic or true of themselves. Each item was rated on a 5-point Likert scale, with anchors 1 (*Very little*) to 5 (*Very much*). Items were then summed to create a total score ranging from 8 to 40, with higher scores indicating more severe social anxiety symptoms. Previous research indicates that the BFNE-S has strong psychometric properties, including internal consistency, factorial validity, and construct validity across both student and clinical samples (Rodebaugh et al., 2004; Carleton et al., 2007; Carleton et al., 2011). Internal consistency in the current study was excellent ($\alpha = .95$ across all cases; $\alpha = .95$ for community participants; $\alpha = .94$ for student participants).

***Beck Depression Inventory - II* (BDI-II; Beck, Steer, & Brown, 1996).** The BDI-II is a 21-item measure of depression symptoms experienced within the past two weeks. Each item maps on to a particular symptom (e.g., sad mood) of depression. Participant responses were rated on a 4-point Likert scale from 0 (e.g., "I do not feel sad") to 3 (e.g., "I am so sad or unhappy I can't stand it"). Item responses were then summed to create a total score ranging from 0 to 63, with higher scores indicating greater levels of depression. When administered to both clinical and non-clinical populations, the BDI-II has demonstrated high levels of internal consistency and appropriate convergent and divergent validity (Beck et al., 1996; Dozois, Dobson, & Ahnberg, 1998; Steer & Clark, 1997). In the current study, internal consistency was excellent ($\alpha = .94$ across all cases; $\alpha = .90$ for community participants; $\alpha = .94$ for student participants).

***Shipley Institute of Living Scale – Vocabulary Test* (Shipley; Shipley, 1967).** The Shipley is a self-administered 40-item measure of crystallized intelligence. For each item, participants must decide which of four response options are most similar in definition to a given word. Correct items are then summed to create a total score ranging from 0 to 40, with higher scores indicating greater crystallized intelligence. The Shipley has demonstrated strong convergent validity with other measures of intelligence (Matthews, Orzech, & Lassiter, 2011). Because intelligence has been both theoretically and empirically linked with indices of executive control (Dempster, 1991; Friedman et al., 2006), the Shipley was included as a covariate in the current study in order to reduce the likelihood that significant effects of executive control on outcomes of interest were not driven by individual differences in this construct.

Laboratory session. Laboratory sessions were scheduled to occur within two weeks of the online questionnaires. Upon arrival in the laboratory, individuals were asked to provide informed consent. They then completed two computerized tasks, one assessing interpretation biases and the other assessing executive control. To minimize order effects or the impact of fatigue on task performance, the order of these assessments was randomized across participants. Both tasks were administered using E-Prime 2.0 Professional software (Psychology Software Tools, Inc., 2012). Task stimuli were presented on a 40-inch Samsung 1080p high definition television screen, which was mounted on the opposite wall from where participants sat, within a sound isolation enclosure (Model 7296, WhisperRoom Inc., Morristown, TN).

After the completion of these tasks, participants remained in the sound isolation enclosure, where they underwent a social-evaluative stressor while their subjective and physiological reactivity to and recovery from the stressor was monitored. Finally, participants were debriefed by the experimenter about study aims and hypotheses, as well as the use of minor deception during stressor procedures. During debriefing, the experimenter also inquired about current levels of distress. Though no participants reported acute distress at the conclusion of the laboratory session, several individuals were provided with treatment referral information owing to the severity of their clinical symptoms and/or the acute distress they experienced during stressor procedures. Upon completion of the study, community participants received $20 and student participants received 5 research familiarization credits.

Assessment of interpretation biases. Interpretation biases were assessed using the Word Sentence Association Paradigm (WSAP; Beard & Amir, 2009; Beard, Weisberg, &

Amir, 2011). In this task, participants are presented with a series of ambiguous sentences (e.g., *everyone stops talking when you enter a room*). Each sentence is preceded by a word conveying a negative interpretation (e.g., *mocked*), and the individual must decide whether the word and sentence are related. Biases are assessed by examining the percentage of word-sentence pairs that a participant endorses as being related.

This relatively new paradigm improves upon previous methods for the assessment of interpretation biases in two important ways (Beard & Amir, 2009). First, by presenting a negatively valenced word prior to an ambiguous sentence, it is thought that the WSAP activates the latent beliefs that are proposed to influence the interpretation of contextual cues. Second, to assess biases, the WSAP examines self-reported interpretations, but does not require the participant to choose one interpretation over the other and does not directly ask about the participants' interpretations. Using the WSAP, interpretation biases have been documented within both the social anxiety and depression literatures (Beard & Amir, 2009; Beard et al., 2011; Cowden Hindash & Amir, 2012).

To examine the transdiagnostic or symptom-specific nature of interpretation biases, the WSAP employed in the current study included both social anxiety- and depression-related trials. Social anxiety-related trials included ambiguous sentences centered on social interactions (e.g., *your friend comments on your new haircut*), paired with negative (e.g., *pity*) words. In contrast, depression-related trials included self-referent and affectively ambiguous sentences (e.g., *people always tell you to smile*), paired with negative (e.g., *defective*) words. The task included 10 Non-Affective Practice trials, 40 Social Anxiety trials, and 40 Depression trials.

As depicted in Figure 2.1, each WSAP trial began with the presentation of a fixation cross (500ms). This was done to ensure that the participant's gaze was directed toward the center of the screen at the beginning of each trial. Next, a word appeared on the screen (500ms). As discussed above, this word was either threatening or benign in nature. After the word disappeared, a sentence appeared on the screen. In previous versions of the task (Beard & Amir, 2009; Beard et al., 2011), the sentence remained on the screen until the participant made a decision about its relatedness to the prior word. In the current study, sentence presentation time was set at 1500ms. This modification was made in consultation with one of the creators of the task, Dr. Courtney Beard (personal communication, October 2, 2013), and was meant to prevent participants from remaining on the screen indefinitely, thereby limiting the opportunity for participants to override an initial interpretation. Finally, a screen appeared asking if the word and the sentence were related. Participants were instructed to press the "1" key on their keyboard if they believed the word-sentence pair were related and the "3" key on their keyboard if they believed the pair were unrelated. Participants were then required to press the space bar to move on to the next trial.

Assessment of executive control. The Flanker Task (Ericksen & Ericksen, 1974) was used in the current study as a measure of executive control. The Flanker Task is well suited for assessing deficits in attentional inhibition, as it requires the individual to ignore distracting visual information in order to respond quickly and accurately to a target stimulus. Distracting information may be from either the same (congruent) or a different (incongruent) category of stimuli as the target. Because distracting information that is congruent with the target does not need to be inhibited in order to provide a response, we

can expect individuals to be faster and more accurate on congruent trials. Conversely, when distracting information is incongruent, the individual must inhibit this information in order to respond to the target. Understanding this, deficits in executive control are typically measured by assessing the magnitude of the difference in accuracy or response latencies across congruent and incongruent trials. This difference has been termed the *interference effect* (e.g., Najmi et al., 2010).

Several versions of the Flanker Task have been used to document an association between impaired inhibitory functioning and psychopathology (Leskin & White, 2007; Najmi et al., 2010; Ochsner, Hughes, Robertson, Cooper, & Gabrieli, 2008; Reinholdt-Dunne, Mogg, & Bradley, 2009; Schmid et al., 2015; Zetsche, D'Avanzato, & Joormann, 2012). Importantly, versions vary in whether they include affective (e.g., valenced words) or non-affective (e.g., arrows) stimuli, and it remains unclear whether psychopathology-related inhibitory deficits only emerge within the context of emotion-congruent stimuli (e.g., Schmid et al., 2015, but Najmi et al., 2010). Despite this, given the aims of the current study, and a desire to tease apart biases for emotional information from deficits in executive control as potential vulnerabilities for social anxiety and depression, the study included a non-affective version of the task. This non-affective version of the Flanker Task was recently included in the NIH Toolbox Cognition Battery version 1.0, as a standardized measure of executive control that is both valid and reliable (see Troller-Renfree, Barker, Pine, & Fox, 2015).

Prior to the start of the task, participants received instructions that they would be looking at a series of images in which five arrows appeared horizontally across the screen. Their task was to decide whether the center (target) arrow pointed to the left or to

the right. If the arrow pointed to the left, participants were instructed to press the "1" key on their keyboard, if it pointed to the right, they were instructed to press the "3" key. To do this as quickly and accurately as possible, participants were told to ignore the four flanking arrows, which could point in either the same (congruent trial) or the opposite (incongruent trial) direction from the target arrow.

As can be seen in Figure 2.2, trials began with a fixation cross (500ms). Consistent with the WSAP procedures described above, the fixation cross was used to ensure participants began the trial with visual gaze in the center of the screen. Next, the stimuli were presented on the screen for 1500ms or until a response was detected. In addition to congruent and incongruent trials, the task included trials in which a single target arrow appeared in the center of the screen with dashes on either side (-- -- → -- --). Participants were instructed to simply determine whether the arrow was pointing to the left or to the right. Finally, some trials included a prolonged presentation of the fixation cross. In these cases, participants were instructed not to do anything. Per the methods used by Ochsner and colleagues (2008), these latter two trial types were used to avoid conflict adaptation effects, which can occur with the repetition of trial types. The task consisted of 139 trials (7 Practice, 40 Congruent, 40 Incongruent, 40 Arrow Filler, and 12 Fixation Crosses).

Laboratory stressor. To examine reactivity to and recovery from a social-evaluative stressor, participants completed a slightly modified version of the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993). Procedures for the TSST typically require that participants perform stress tasks before a panel of three evaluators. Within the current study, these tasks were performed before a single evaluator (i.e., the

experimenter). The decision to modify the TSST procedures in this way was based on concerns of feasibility. However, previous research has demonstrated that employing this modified TSST successfully elicits stress responding (e.g., LeMoult et al., 2013).

The procedures for the TSST are laid out in Figure 2.3. Participants began the task by resting quietly for 10 minutes while watching a nature film (*Baseline*). Next, they were given 5 minutes to prepare a speech in which they were required to promote their candidacy for a job (*Preparation*). Participants were told that their speeches would be videotaped by a camera mounted on the wall opposite from where they sat in the sound isolation enclosure, and that the film would later be viewed for voice and behavioral analysis. It was also explained that the experimenter would be taking notes during the speech to aid in the behavioral analysis. These instructions are part of the standardized TSST procedures and are meant to promote the participants' perception of social evaluation. In reality, however, participants were not videotaped and evaluations of speech performance were not used for subsequent analyses. The use of this minor deception was disclosed during participant debriefing at the end of the study session. After the 5-minute preparation period, participants gave a 5-minute speech, while the experimenter "evaluated" them by writing down notes on a clipboard. During the speech, the experimenter maintained a stoic appearance and provided no verbal or non-verbal feedback on the participants' performance. Upon completion of the speech task, participants were asked to complete a second, unexpected task in which they spent 5 minutes counting backwards in increments of 13, beginning at the number 2083. If participants incorrectly completed a calculation, the experimenter said the word "error" and participants were required to start over again (*Stressor*). After the stressor,

participants rested quietly for a period of 10 minutes while watching a second nature film (*Recovery*). Meta-analytic findings suggest that these TSST procedures are highly effective at eliciting a robust stress response (Dickerson & Kemeny, 2004).

Data on subjective and biological stress responding were collected throughout the entirety of the TSST procedures. Specifically, subjective emotional responding was collected at four time points, before (Baseline 1) and after (Baseline 2) the baseline period, following completion of the stressor (Stressor), and following completion of the recovery period (Recovery).

Subjective emotional responding. Subjective emotional responding was collected using the Negative Affect subscale of the Positive and Negative Affect Schedule (PANAS-N), a 10-item measure of current negative affect (Watson, Clark, & Tellegen, 1988). Participants rated each item (e.g., "Distressed") on a scale of 1 (*Not at all*) to 5 (*Extremely*) based on how much they were experiencing the emotion in that moment. Item responses were summed to create a total score ranging from 10 to 50, with higher scores indicating greater levels of negative affect. The psychometric properties of this scale have been assessed within both clinical and non-clinical samples. Results indicated that the measure is internally consistent and has strong convergent and discriminative validity (Crawford & Henry, 2004; Dyck, Jolly, & Kramer, 1994; Watson et al., 1988).

Biological responding. To assess biological responding, electrocardiographic and respiratory data were collected and respiratory sinus arrhythmia (RSA) data were examined. RSA examines heart rate variability by looking at the increase in heart rate as one inhales and the decrease in heart rate as one exhales (Sharpley, 2002). Previous research has found that individuals who demonstrate blunted RSA in response to stress

may have difficulties with acute emotion regulation, as well as symptoms of social anxiety and depression (e.g., Rottenberg et al., 2003; Woody & Gibb, 2015). Two Adult Multipurpose Silver EKG/ECG electrodes (Model 93-0100-00; Mindware Technologies, Ghana, OH) were attached to participants' right collarbones and lower left ribs. This modified Lead II placement places sensors on areas of the body that are relatively free of fatty tissue and muscle, reducing movement and associated artifacts during data collection (Stern, Ray, & Quigley, 2001). Respiratory rate was measured using the girth method, in which a strain gauge (i.e., Respiration Belt with Pulse Lock [BioNex pl500]; Model 50-4504-00; Mindware Technologies, Ghana, OH) was wrapped around participants' chests and positioned at the base of their sternum. The degree of strain placed on the belt clasp was measured as participants inhaled and exhaled. The girth method is one of the most commonly used and cost-effective methods for measuring respiration, as it is non-invasive and easy to collect (Stern et al., 2001).

Chapter 3: Data Preparation and Analytic Plan

Data Preparation

Calculation of WSAP indices. Interpretation biases were examined using the percentage of trials endorsed for a given trial type and separate indices were created for social anxiety-related and depression-related trials. For each of these indices, a score of .50 (50%) was interpreted to mean there was no bias present. A score greater than .50 was interpreted to mean there was a bias to endorse that trial type and a score less than .50 was interpreted to mean there was a bias to reject that trial type.

Calculation of Flanker Task indices. The study examined two Flanker Task indices, accuracy interference and reaction time interference. Accuracy interference was examined by creating a difference score, in which the percentage of accurate incongruent trials was subtracted from the percentage of accurate congruent trials. Here, a score of zero was interpreted to mean there was no deficit in executive control, whereas a positive value indicated an interference effect (i.e., the participant responded incorrectly to more incongruent than congruent trials). Further, the size of the positive value indicated the magnitude of the interference effect, with larger values indicating greater interference. Only two participants had a negative accuracy interference score. In both cases, these participants were 3% more accurate on incongruent versus congruent trials.

Reaction time interference was only examined for accurate trials. Interference was examined by creating a difference score, in which the mean reaction time for incongruent trials was subtracted from the mean reaction time for congruent trials. A score of zero was interpreted to mean there was no bias present, whereas a negative value indicated an interference effect (i.e., participants were slower to respond to incongruent trials than to

congruent trials), and the size of the negative value indicated the magnitude of the interference effect. Only two participants had a positive reaction time interference score, neither of whom was identified as an outlier within the accuracy interference data. Both of these individuals were slightly faster to respond to incongruent trials than to congruent trials (interference scores were 157.76ms and 154.52ms, respectively). As there was no evidence that either accuracy or reaction time interference outliers were the result of artifact, as opposed to true performance, these data points were retained in analyses.

Calculation of RSA. Physiological reactivity and recovery data were cleaned and analyzed using Heart Rate Variability Analysis Software, Version 3.0.15 (MindWare Technologies Ltd., 2010). Data were collected in three epochs: Baseline, Stressor, and Recovery. Prior to analysis, data files were visually examined to ensure valid data were captured for both heart rate and respiration; indeed, valid data were collected from all 57 participants. Data files were then cleaned and analyzed in 30-second segments. To create a Baseline RSA variable, RSA values were averaged across the 20 segments of the baseline period. Similarly, to create a Stressor RSA variable, RSA values were averaged across the 10 segments of the speech preparation period, the 10 segments of the speech task, and the 10 segments of the calculation task. Finally, Recovery RSA was calculated by averaging values across the 20 segments of the recovery period data file.

Data Analytic Plan

Power calculations. A priori power analyses were conducted using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007). Based on the extant literature, as well as preliminary unpublished data from our laboratory, effect sizes were estimated to be in the moderate range. Power analyses with two-tailed $\alpha = .05$, desired power = .80 and an f^2

effect size estimated as .20, revealed that linear multiple regression with three predictors would require a sample of 59 individuals to obtain a critical $F = 2.77$.

Analytic approach. Analyses were conducted using IBM SPSS Statistics for Macintosh, Version 22.0 (IBM Corp., 2013). More information on the approach to each of the three study aims is provided below.

Aim 1. To initially examine the independent associations between cognitive processes and acute subjective and biological reactivity to the social-evaluative stressor, a series of zero-order, Pearson's *r* correlations were conducted. More specifically, the two indices of interpretation bias and the two indices of executive control were correlated with PANAS-N scores and mean RSA during the stressor. When significant associations were detected, correlations were followed up with regression analyses, which controlled for baseline PANAS-N scores or RSA values, as appropriate, as well as for demographic (i.e., age and ethnicity) and conceptual (i.e., Shipley total scores for analyses examining executive control and blood pressure medication status for analyses examining RSA) covariates of interest. Reporting results of analyses with and without the inclusion of covariates was based on the recommendations for researchers laid forth by Simmons, Nelson, and Simonsohn (2011) to promote transparency in research and reduce the likelihood of false positive findings.

To assess the interactive effects of interpretation biases and executive control on subjective and biological reactivity, a series of linear multiple regression analyses were conducted in line with standard procedures laid forth by Holmbeck (2002). Prior to running regression analyses, indices of interpretation biases and executive control were centered and interaction terms were created. Regression models were then specified to

include relevant covariates, baseline subjective negative affect or RSA, centered interpretation bias and executive control indices, and the interpretation bias by executive control interaction term. Statistically significant and trend-level interaction terms were further examined by testing the simple slopes of the model. Specifically, the impact of interpretation biases on outcomes of interest was examined at high (+1 SD) and low (-1 SD) levels of executive control.

Aim 2. As with the data analytic plan for Aim 1, to initially assess the independent associations between cognitive processes and subjective and biological recovery from the social-evaluative stressor, a series of zero-order, Pearson's *r* correlations were conducted. These correlations examined the associations between interpretation biases or executive control and recovery-related subjective negative affect or RSA. When significant associations were detected, follow-up regression analyses were conducted, controlling for subjective negative affect or RSA during the stressor, as well as demographic and conceptual covariates of interest. Tests of interpretation bias by executive control interaction effects were identical to those laid out in Aim 1, with the exception of controlling for stressor-related subjective negative affect or RSA, rather than baseline values for these variables.

Aim 3. Again, to examine the independent associations between cognitive processes and symptoms of social anxiety and depression, a series of zero-order, Pearson's r correlations were used. When significant associations emerged, these were further examined using linear regression models, which controlled for demographic and conceptual covariates of interest, as well as for the comorbidity between symptoms. For example, when examining a significant association between interpretation biases and

social anxiety symptoms, analyses controlled for participants' depression symptom severity. Procedures for conducting interaction analyses were identical to those described under Aims 1 and 2, with the exception of controlling for comorbidity between symptoms rather than for baseline subjective or biological emotional responding.

Chapter 4: Results

Preliminary Analyses

Self-report measures. Descriptive statistics for measures of social anxiety and depression are presented in Table 4.1. In general, participants endorsed clinical levels of social anxiety and depression. The sample's mean BFNE-S score was greater than the previously established clinical cutoff of 25 (Carleton et al., 2011) and the mean BDI-II score fell within the "Moderate" range (Beck et al., 1996). However, examination of the variability in BFNE-S and BDI-II scores also revealed that symptom severity ranged from very mild to severe. Thus, the study's recruitment strategy seemed to successfully capture social anxiety and depression as dimensionally distributed constructs. As expected, BFNE-S and BDI-II scores were moderately correlated ($r = .45$, $p < .001$).

Participants' Shipley scores are also presented in Table 4.1. Consistent with previous research using this measure (e.g., Quinn & Joormann, 2015a), the average participant answered approximately 80% of the items correctly.

Interpretation biases. Descriptive statistics for the WSAP indices are presented in Table 4.2. Overall, participants exhibited a bias to reject negative interpretations. A one-sample *t*-test revealed that the mean bias score for social anxiety trials was significantly lower than a score of .50, $t\ (56) = 3.41$, $p = .001$, $d = .91$. This was also the case for depression trials, $t\ (56) = 2.73$, $p = .009$, $d = .73$. However, the variability in scores was large and ranged from a strong bias to reject negative interpretations to a strong bias to endorse them. The magnitude of interpretation biases for social anxiety and depression trials were not significantly different from each other, $t\ (56) = 1.38$, $p = .17$, $d = .36$.

Executive control. Examination of accuracy interference on the Flanker Task revealed that, overall, participants were less accurate on incongruent trials than they were on congruent trials (M = .06, SD = .16, Range: -.03, .95). The size of this interference effect was significantly different from zero, t (56) = 2.73, p = .008, d = .73. The mean reaction time interference score also indicated that participants were slower to respond to incongruent trials than they were to congruent trials (M = -51.67, SD = 58.86, Range: -273.83, 157.76). Again, the magnitude of mean reaction time interference was significantly different from zero, t (56) = 6.63, p < .001, d = 1.77.

Stress reactivity and recovery. To ensure that the stressor elicited the predicted patterns of stress reactivity and recovery, two repeated-measures ANOVAs were conducted. The first examined PANAS-N scores at all four time points and results revealed a significant effect of time, F (3, 168) = 26.60, p < .001, partial η^2 = .32 (see Figure 4.1). A series of follow-up, paired samples t-tests indicated that participants' subjective negative affect decreased from Baseline 1 to Baseline 2, t (56) = 4.66, p < .001, d = .40, and then increased as expected from Baseline 2 to the Stressor, t (56) = 6.56, p < .001, d = .92. Also as expected, subjective negative affect decreased from the stressor period to the recovery period, t (56) = 6.62, p < .001, d = .80. At the completion of the recovery period, participants' PANAS-N scores were slightly lower than their Baseline 1 scores, t (56) = 2.69, p = .009, d = .41, but no different from their Baseline 2 scores, t (56) = -.85, p = .40, d = -.11.

Changes in biological responding over the course of the TSST were examined using participants' mean RSA levels during the baseline, stressor, and recovery periods. Once again, results revealed a main effect time, F (2, 112) = 35.55, p < .001, partial η^2 =

.39 (see Figure 4.2). A series of follow-up, paired samples *t*-tests revealed no significant difference in RSA between the baseline and stressor periods, t (56) = 1.54, p = .13, d = .12. However, an increase in RSA was found between the stressor and recovery periods, t (56) = 6.14, $p < .001$, d = .47. RSA during the recovery period was also significantly higher than RSA during the baseline period, t (56) = 9.93, $p < .001$, d = .60.

Aim 1: Cognitive Processes and Emotional *Reactivity* to Stress

Interpretation biases. As hypothesized, participants with a bias to endorse social anxiety interpretations reported greater subjective negative affect following the stressor (r = .41, p = .002). The same was true of participants with a bias to endorse depression interpretations (r = .30, p = .02). Given that, as expected, the association between interpretation biases and subjective stress reactivity did not differ across social anxiety- or depression-related trials, trial types were collapsed together to examine how a general bias toward threat related to subjective stress reactivity. Unsurprisingly, results revealed that individuals with a general bias toward threat demonstrated greater subjective negative affect following the stressor (r = .38, p = .004).

To further examine these associations, three multiple regression models were tested, which controlled for participants' age and ethnicity, as well as their Baseline 2 PANAS-N scores. Controlling for these factors, social anxiety-related biases continued to predict subjective stress reactivity (B = 15.52, SE = 6.27, β = .30, t = 2.48, p = .02), whereas the association between depression-related biases and subjective stress reactivity was reduced to a trend-level effect (B = 11.67, SE = 6.74, β = .21, t = 1.73, p = .09). When all threat trials were considered together, individuals with a general bias toward

negative interpretations continued to report significantly greater subjective negative affect following the stressor (B = 15.24, SE = 6.83, β = .27, t = 2.23, p = .03).

Pearson's r correlations were also used to initially examine the independent associations between interpretation biases and RSA during exposure to the stressor. No significant associations emerged from analyses examining social anxiety-related (r = -.03, p = .84) or depression-related (r = .11, p = .41) interpretation biases. Because initial associations were not significant, follow up analyses controlling for covariates were not conducted.

Executive control. Contrary to predictions, no significant associations emerged from analyses examining the independent associations between executive control and stress reactivity. Accuracy interference was unrelated to subjective negative affect following the stressor (r = .08, p = .56) and only marginally related to participants' Stressor RSA values (r = -.22, p = .09). Similarly, reaction time interference was unrelated to both participants' subjective (r = -.11, p = .41) and biological (r = -.03, p = .85) reactivity to the stressor. Given that none of these associations were statistically significant, no follow up analyses controlling for covariates were conducted.

Interaction analyses. Because analyses examining the independent associations between interpretation biases and stress reactivity revealed few differences across social anxiety- and depression-related trials, interaction analyses were conducted using the composite index of negative interpretation biases, which included responses to both social anxiety- and depression-related trials. This reduced the number of analyses conducted and thus the likelihood of Type I error.

Two regression models were conducted to analyze the interactions between interpretation biases and executive control as they related to subjective stress reactivity. The first examined interpretation biases and accuracy interference, controlling for participants' age and ethnicity, as well as their Shipley and Baseline 2 PANAS-N scores. Results are presented in Table 4.3. The overall model was significant, F (7, 48) = 3.57, p = .004, and explained 34% (R^2 = .34) of the variance in Stressor PANAS-N scores. Findings revealed a main effect of interpretation biases, but not executive control. Consistent with correlational analyses, individuals with a bias to endorse negative interpretations reported greater subjective negative affect following the stressor. This main effect was qualified by a marginally significant interpretation bias by accuracy interference interaction (see Figure 4.3). Probing of this interaction found that when accuracy interference was high (+1 SD), indicating poorer executive control, interpretation biases were associated with greater subjective negative affect following the stressor (B = 55.46, SE = 23.50, β = .99, t = 2.36, p = .02). In contrast, when accuracy interference was low (-1 SD), indicating stronger executive control, biases were unrelated to subjective stress reactivity (B = -10.65, SE = 16.48, β = -.19, t = .65, p = .52).

A similar pattern of results was found when examining threat biases and reaction time interference as they related to subjective stress reactivity. Again, the overall model was significant, F (7, 48) = 4.62, p = .001 and explained 40% (R^2 = .40) of the variance in Stressor PANAS-N scores. Additionally, as can be seen in Table 4.4, a main effect of interpretation biases, but not executive control, was qualified by a significant bias by executive control interaction. At -1 SD from the mean, representative of a more pronounced interference effect and poorer executive control, a bias to endorse threat trials

was associated with heightened subjective stress reactivity (B = 41.90, SE = 12.97, β = .75, t = 3.23, p = .002). In contrast, at +1 SD from the mean, representative of a less pronounced interference effect and greater executive control, there was no association between threat biases and subjective negative affect following the stressor (B = -11.62, SE = 13.64, β = -.21, t = .85, p = .40). This interaction is depicted in Figure 4.4.

Next, two regression models were used to examine the interactions between interpretation biases and executive control as they related to biological stress reactivity. The first model examined the interaction between interpretation biases and accuracy interference as a predictor of RSA during the stressor, controlling for age, ethnicity, Shipley total scores, blood pressure medication status, and baseline RSA. The overall model was significant, F (8, 47) = 18.74, p < .001, and accounted for 76% (R^2 = .76) of the variance in Stressor RSA. As can be seen in Table 4.5, a marginally significant main effect of interpretation biases was found, indicating that individuals with a tendency to endorse negative interpretations experienced greater, albeit not significantly, RSA in response to the stressor. In addition, a there was a significant main effect of accuracy interference; greater inference (indicating poorer executive control) was associated with greater RSA during the stressor. Finally, as can be seen in Figure 4.5, a significant bias by executive control interaction also emerged from analyses. Contrary to hypotheses, when accuracy interference was high (+1 SD), a bias to endorse threat trials predicted greater RSA during the stressor (B = 5.03, SE = 1.56, β = .84, t = 3.21, p = .002). When accuracy interference was low (-1 SD), a bias to endorse threat trials was inversely related to RSA during the stressor (B = -3.13, SE = 1.07, β = -.53, t = 2.92, p = .005).

The second model examined the interaction between threat biases and reaction time interference as they related to Stressor RSA, controlling for covariates. The overall model was significant, F (7, 48) = 16.59, $p < .001$, and accounted for 73% ($R^2 = .73$) of the variance in Stressor RSA. Though this model revealed no main effect of either negative interpretation biases or reaction time interference (see Table 4.6), a marginally significant interaction was found. Probing of this interaction revealed that at +1 SD from the mean, representative of a less pronounced interference effect and stronger executive control, there was no association between threat biases and Stressor RSA (B = -1.58, SE = .97, β = -.27, t = 1.64, p = .11). In contrast, at -1 SD from the mean, representative of a more pronounced interference effect and poorer executive control, a bias to endorse threat trials predicted greater Stressor RSA at a level that approached significance (B = 1.72, SE = .96, β = .29, t = 1.79, p = .08). This interaction is depicted in Figure 4.6.

Aim 2: Cognitive Processes and Emotional *Recovery* from Stress

Interpretation biases. Contrary to hypotheses, findings revealed no significant associations between interpretation biases and subjective negative affect following the recovery period, though associations were in the predicted direction. Social anxiety-related biases were marginally related to greater subjective negative affect (r = .23, p = .08). The association between depression-related biases and subjective negative affect was not significant (r = .13, p = .34). Given that neither association was significant, analyses controlling for covariates were not conducted. Social anxiety-related (r = -.05, p = .74) and depression-related (r = .04, p = .75) biases were also found to be unrelated to participants' RSA during the recovery period. Again, given the lack of significant associations follow-up analyses controlling for covariates were not conducted.

Executive control. Consistent with the pattern of results reported in Aim 1, examination of the independent associations between executive control and recovery from the stressor did not reveal any significant findings. Accuracy interference was unassociated with subjective negative affect (r = -.09, p = .49) and RSA (r = -.15, p = .28). Similarly, reaction time interference was unassociated with subjective negative affect (r = .06, p = .65) and RSA (r = -.06, p = .68).

Interaction analyses. Because analyses examining the independent associations between interpretation biases and stress recovery revealed few differences across social anxiety- and depression-related trials, interaction analyses were conducted using only the composite index of negative interpretation biases. As discussed in Aim 1, this reduced the number of analyses conducted and thus the likelihood of Type I error.

To examine the interactions between interpretation biases and executive control as they related to subjective negative affect following the recovery period, two regression models were tested. Each of these models included age, ethnicity, Shipley total scores, and subjective negative affect following the stressor as covariates. As seen in Table 4.7, the first model examined the interaction between interpretation biases and accuracy interference. The overall model was significant, F (7, 48) = 7.01, p < .001, and explained 51% (R^2 = .51) of the variance in subjective negative affect. Results revealed a marginally significant main effect of interpretation biases, as well as a significant main effect of accuracy interference. Main effects were qualified, however, by a significant interaction. Post-hoc probing of this interaction indicated that when Accuracy Interference was high, indicating poorer executive control, negative interpretation biases were associated with less subjective negative affect (B = -50.79, SE = 16.85, β = -1.16, t

= -3.02, p = .004). When accuracy interference was low, indicating stronger executive control, interpretation biases were associated with greater subjective negative affect (B = 29.12, SE = 11.08, β = .66, t = -2.63, p = .01). This interaction is depicted in Figure 4.7.

The second model examined the interaction between interpretation biases and reaction time interference as it related to subjective negative affect (see Table 4.8). Again, the overall model was significant, F (7, 48) = 5.92, p < .001, and explained 46% of the variance in subjective negative affect following the recovery period. Despite the fact that no main effects emerged from analyses, a significant interpretation bias by executive control interaction was found. At +1 *SD* from the mean, indicative of a less pronounced interference effect and stronger executive control, interpretation biases were associated with greater subjective negative affect (B = 22.99, SE = 9.85, β = .52, t = 2.34, p = .02). On the other hand, at -1 *SD* from the mean, indicative of a more pronounced interference effect and poorer executive control, threat biases were associated with less subjective negative affect (B = -26.79, SE = 10.54, β = -.61, t = -2.54, p = .01). This interaction is depicted in Figure 4.8.

Next, two regression models were conducted to examine the interactions between interpretation biases and executive control as they related to RSA during the recovery period. Each model controlled for age, ethnicity, Shipley total scores, blood pressure medication status, and Stressor RSA. The first model revealed no main effect of biases (B = -.69, SE = .47, β = -.12, t = -1.48, p = .15), no main effect of accuracy interference (B = -.46, SE = .80, β = -.07, t = -.58, p = .57), and no bias by executive control interaction (B = -9.61, SE = 7.72, β = -.16, t = -1.24, p = .22). Similarly, the second model, which examined the interaction between interpretation biases and reaction time interference,

revealed no main effect of biases (B = -.37, SE = .39, β = -.07, t = -.96, p = .34), no main effect of executive control (B = .001, SE = .001, β = .08, t = 1.06, p = .30), and no benign bias by executive control interaction (B = .02, SE = .01, β = .11, t = 1.46, p = .15).

Aim 3: Cognitive Processes and Symptoms of Social Anxiety and Depression

Interpretation biases. To examine the associations between interpretation biases and symptoms of social anxiety and depression, a series of Pearson's *r* correlations were first conducted. As can be seen in Table 4.9, a bias to endorse either social anxiety-related or depression-related trials was positively associated with symptoms of social anxiety. The same pattern was found for symptoms of depression; individuals with a bias to endorse either social anxiety-related or depression-related trials reported greater depression symptom severity. Because, contrary to hypotheses, social anxiety- and depression-related interpretation biases were not associated with symptom specificity, these trials were then collapsed together to examine a general bias toward negative interpretations. Overall, individuals who exhibited this bias were more likely to endorse symptoms of both social anxiety and depression.

Next, significant associations between interpretation biases and clinical symptoms were examined after controlling for demographic covariates, as well as for the comorbidity between social anxiety and depression symptoms within the sample. Both social anxiety-related (B = 25.43, SE = 5.37, β = .54, t = 4.73, p < .001) and depression-related (B = 16.48, SE = 6.31, β = .33, t = 2.61, p = .01) biases continued to predict symptoms of social anxiety even after controlling for age, ethnicity, and BDI-II scores. Likewise, social anxiety-related (B = 19.35, SE = 9.21, β = .29, t = 2.10, p = .04) and depression-related (B = 19.84, SE = 8.33, β = .29, t = 2.38, p = .02) biases continued to

predict symptoms of depression even after controlling for age, ethnicity, and BFNE-S scores. Finally, results revealed that a general negative interpretation bias, inclusive of social anxiety- and depression-related threat trials, continued to predict more severe symptoms of social anxiety (B = 24.21, SE = 6.04, β = .47, t = 4.01, p < .001) and depression (B = 21.89, SE = 9.36, β = .30, t = 2.34, p = .02), even after controlling for relevant demographic covariates and BDI-II or BFNE-S scores, respectively.

Executive control. Examination of the independent associations between executive control and clinical symptoms revealed deficits in executive control to be unrelated to symptoms of social anxiety and depression. No associations were found between participants' accuracy interference and their BFNE-S scores (r = -.02, p = .89) or their BDI-II scores (r = -.004, p = .97). Similarly, no associations were found between participants' reaction time interference and their BFNE-S (r = -.07, p = .60) or BDI-II (r = -.16, p = .23) scores. As there were no significant findings, follow up analyses controlling for covariates were not conducted.

Interaction analyses. Because findings supported a transdiagnostic conceptualization of interpretation biases, interaction analyses utilized only the composite indices of general negative interpretation biases. All interaction analyses controlled for age, ethnicity, Shipley total scores, and the comorbidity between social anxiety and depression symptoms within the sample.

The first model examined the association between threat biases and accuracy interference as they related to social anxiety. Results are presented in Table 4.10. The overall model was significant, F (7, 48) = 6.53, p < .001, and accounted for 49% (R^2 = .49) of the variance in participants' BFNE-S scores. Findings revealed a main effect of

negative biases, as well as a main effect of executive control that approached significance. Participants with greater accuracy interference endorsed marginally more severe symptoms of social anxiety. In addition, a trend-level interaction was found. Probing of this interaction revealed that when accuracy interference was high (+1 SD), indicating poorer executive control, a bias to endorse threat interpretations was related to more severe social anxiety symptoms (B = 59.36, SE = 19.72, β = 1.17, t = 3.01, p = .004). On the other hand, when accuracy interference was low (-1 SD), indicating stronger executive control, threat biases were unassociated with BFNE-S scores (B = 3.88, SE = 13.18, β = .08, t = .29, p = .77). This interaction is depicted in Figure 4.9.

No other significant interaction effects between interpretation biases and executive control were found. The regression model examining interpretation biases and reaction time interference as they related to social anxiety revealed a main effect of threat biases (B = 25.47, SE = 6.56, β = .50, t = 3.88, p < .001), but no main effect of executive control (B = -.01, SE = .02, β = -.04, t = -.37, p = .71), or a bias by executive control interaction (B = .13, SE = .17, β = .09, t = .77, p = .45). Similarly, the regression model examining interpretation biases and accuracy interference as they related to depression revealed a main effect of biases (B = 29.16, SE = 10.98, β = .41, t = 2.66, p = .01), but no main effect of executive control (B = -1.18, SE = 14.89, β = -.01, t = -.08, p = .94), nor a bias by executive control interaction (B = 73.05, SE = 136.81, β = .10, t = -.53, p = .60). Finally, the regression model examining interpretation biases and reaction time interference as they related to depression again revealed a main effect of biases (B = 26.93, SE = 9.14, β = .38, t = 2.95, p = .005), but no main effect of executive control (B =

-.03, SE = .02, β = -.13, t = 1.21, p = .23) and no bias by executive control interaction (B = -.02, SE = .22, β = -.008, t = -.073, p = .94).

Chapter 5: Discussion

In the current study, emotional and cognitive vulnerability factors for social anxiety and depression were examined using an RDoC framework. The overarching goals of the study were to (1) to elucidate the symptom-specific or transdiagnostic nature of two cognitive vulnerability factors - interpretation biases and executive control - and (2) to examine the synergistic impact of interpretation biases and executive control on stress responding and clinical symptoms. To address these aims, the study recruited a disorder-non-specific at-risk sample of individuals reporting elevated levels of repetitive negative thinking. The study then investigated how social anxiety- and depression-related interpretation biases and deficits in executive control were independently and interactively related to acute social-evaluative stress reactivity and recovery, as well as to dimensional measures of social anxiety and depression symptoms.

To assess the success of the study's recruitment strategy, the clinical characteristics of the sample were examined. Results provided support for repetitive negative thinking as a dimensionally distributed mechanism of dysfunction relevant to both social anxiety and depression symptoms. On average, symptoms of social anxiety and depression fell within the clinical range; however, symptoms ranged from non-clinical to severe and were only moderately correlated with each other. Thus, recruiting individuals with elevated levels of repetitive negative thinking allowed for the oversampling of individuals with or at risk for SAD and MDD, without relying on existing diagnostic criteria and using a dimensional, rather than a categorical approach.

Summary of Findings for Stress Reactivity

The first aim of the study was to examine how interpretation biases and executive control were independently and interactively related to reactivity to an acute social-evaluative stressor. Within this aim, the first hypothesis was that interpretation biases would be associated with elevated subjective negative affect and blunted RSA. No differences between social anxiety- and depression-related interpretation biases and their relations with stress reactivity were expected to emerge from analyses.

Findings partially supported Aim 1 hypotheses. Whereas it was expected that interpretation biases would be associated with blunted stressor RSA, no significant relations between these variables was found. Despite this, positive associations of moderate magnitude were found between interpretation biases and subjective negative affect following the stressor. As expected, these associations were found across social anxiety- and depression-related biases. The association between social anxiety-related biases and subjective negative affect was maintained even after controlling for relevant covariates and baseline subjective negative affect. The association between depression-related biases and subjective negative affect was somewhat less robust, as evidenced by the fact that it was reduced to a trend-level effect after controlling for baseline subjective negative affect and relevant covariates.

Though it is unclear why the magnitude of effects differed between social anxiety- and depression-related biases, it is logical that individuals with a tendency to interpret ambiguous social situations in a threatening manner (i.e., social anxiety-related interpretation biases) would also be at particular risk for elevated subjective negative affect in response to the TSST. In contrast, it is possible that individuals with a tendency

to interpret ambiguous self-referent material in a threatening manner (i.e., depression-related biases) are at greater risk for elevated subjective negative affect under other conditions of stress, such as in response to performance feedback or social rejection paradigms (e.g., Yale Interpersonal Stressor; Stroud, Tanofsky, Wilfley, & Salovey, 2000). This hypothesis is also supported by some previous research, which has found that the TSST elicits a relatively stronger stress response from individuals with social anxiety than individuals with major depression (see Kudielka et al., 2007, for a review).

The second Aim 1 hypothesis was that deficits in executive control would be associated with increased subjective negative affect and decreased RSA following the stressor. Results revealed no effect of executive control on subjective or biological stress reactivity. This is inconsistent with some previous research (Schmid et al., 2015), but similar to findings from other studies (Najmi et al., 2010). The current findings suggest that difficulties with non-affective components of executive control in the absence of other emotionally-laden stimuli (e.g., cognitive biases) may not be sufficient to dysregulate stress reactivity.

Finally, it was hypothesized that interpretation biases and executive control would interact to predict subjective and biological stress reactivity. Specifically, individuals who displayed greater interpretation biases and poorer executive control were expected to evidence the strongest stress response. Results supported the hypothesized interpretation bias by executive control interactions. More specifically, findings the association between interpretation biases and greater stress reactivity was significant only when executive control was poorer.

It has been previously documented that a neural circuit inclusive of bottom-up hyper-responsivity of the amygdala and top-down hypo-responsivity of the prefrontal cortex underlies emotion dysregulation in the internalizing disorders (Bishop, 2007). However, this is the first known study to examine this circuit using behavioral measures of bottom-up (i.e., interpretation biases) and top-down (i.e., executive control) cognitive processes. This finding helps to refine our understanding of stress and emotion dysregulation among individuals with or at risk for social anxiety and depression by integrating additional units of analysis into existing vulnerability models (Sanislow et al., 2010). Further, it suggests that bottom-up and top-down processes may need to be targeted in tandem in order to reduce vulnerability using neurobehavioral interventions.

Analyses examining the associations between interpretation biases and executive control as they related to RSA during the stressor revealed significant interaction effects, but in the direction opposite to what were predicted. When executive control was weaker, a bias to endorse negative interpretations was associated with greater stressor RSA. Conversely, when executive control was stronger, a bias to endorse negative interpretations was associated with attenuated stressor RSA. These findings are inconsistent with those of several previously published investigations (Hughes & Stoney, 2000; Pittig et al., 2013; Shinba et al., 2008; Verkuil et al., 2014), which have linked clinical symptoms and/or vulnerability for clinical symptoms to decreased heart rate variability in response to acute stress.

Because RSA examines changes in heart rate during respiration, there is some research to indicate that vocalization during stressor procedures may influence RSA activity (Sharpley, 2002). Within the current investigation, participants were asked to

speak aloud during both the speech and calculation tasks. This is a methodological limitation of the current study and it is unclear how the study procedures may have influenced RSA results. Future research should examine these associations in the context of stress without vocalization to determine whether the patterns change.

We must also consider the differences between the current sample (i.e., individuals high in repetitive negative thinking) and those used in previous investigations (e.g., individuals with diagnoses of SAD or MDD and non-disordered controls). Whereas the latter samples were homogeneous groups of clinically disordered individuals, the current sample was intended to be heterogeneous and to include individuals with clinical, subclinical, and non-clinical phenotypic presentations. Though the characteristics of the current sample are a unique strength of the study in several ways, one issue is that there is little previous research against which to compare our findings. As such, more research will be needed to better understand and interpret these findings, especially in conjunction with the investigation's findings on subjective stress reactivity.

Summary of Findings for Stress Recovery

The second aim of the study was to examine how interpretation biases and executive control were related to recovery from the social-evaluative stressor. The first hypothesis within this aim was that interpretation biases would be related to subjective and biological stress recovery, such that individuals who displayed a more pronounced negative interpretation bias would evidence more prolonged subjective negative affect and attenuated RSA. As with the Aim 1, no differences were expected to emerge across analyses examining social anxiety- and depression-related biases.

Contrary to hypotheses, no significant findings emerged from analyses examining the associations between interpretation biases and subjective negative affect or from analyses examining interpretation biases and RSA. The overall lack of significant associations between interpretation biases and recovery from stress may mean that interpretation biases are simply unrelated to recovery from acute social-evaluative stress. This conclusion seems implausible, however, when we consider the robust associations found between interpretation biases and stress reactivity in the current study. As such, these results may also be influenced by methodological limitations.

It is possible that the methodological design in the current study was not sensitive enough to detect individual differences in recovery trajectories. Examination of subjective negative affect over the course of the TSST procedures revealed that most participants had fully recovered by 10 minutes post-stressor. Indeed, the mean subjective negative affect score at following the recovery period was even lower than participants' mean Baseline 1 scores and no different than their Baseline 2 scores. Given this, future research should look at recovery from acute social-evaluative stress with repeated assessments over the course of a 10-minute period to gain more detailed data on individual differences in recovery trajectories.

The second hypothesis was that executive control would be inversely associated with recovery from stress, such that individuals who demonstrated poorer executive control would experience greater residual subjective negative affect and dampened RSA during the recovery period. Results did not support this hypothesis; there was no evidence linking executive control to subjective or biological recovery from stress. These findings were consistent with those from Aim 1, examining the associations between executive

control and stress reactivity. Again, this suggests that in the absence of other factors (e.g., cognitive biases) lack of executive control may not be sufficient to dysregulate stress responding.

Finally, it was predicted that interpretation biases and executive control would interact to predict recovery from the stressor. More specifically, it was expected that individuals who displayed greater interpretation biases and poorer executive control would experience the greatest residual stress response (i.e., poorer recovery). Unexpectedly, results of analyses examining the interaction between interpretation biases and executive control revealed that individuals who demonstrated the greatest subjective stress reactivity (i.e., individuals with a strong bias to endorse threat interpretations and poorer executive control) showed the greatest recovery in their subjective negative affect. This was likely due to the fact that the majority of participants had fully recovered by the end of the 10-minute recovery period. As such, we can interpret these findings to mean that, under the conditions of this study, there was no evidence of impaired stress recovery among cognitively vulnerable individuals.

No significant interactions emerged from analyses examining the interactions between interpretation biases and executive control as predictors of RSA during the recovery period. Overall, these findings indicate that the cognitive processes examined in the current study play less of a role in recovery from, rather than reactivity to, acute stress. However, future studies may want to examine recovery from stress under other conditions (e.g., using different stress paradigms, including shorter or longer delays in assessment, or assessing other outcome variables) to determine the generalizability of this conclusion.

Summary of Findings for Symptoms of Social Anxiety and Depression

The third and final aim of the current investigation was to examine how interpretation biases and executive control were related to dimensional measures of social anxiety and depression symptoms. The first hypothesis under this aim was that threat interpretation biases would be significantly associated with greater clinical symptoms. In contrast to the first two aims of the study, in which we did not expect to see differences between social anxiety- and depression-related interpretation biases and stress responding, we did hypothesize specificity between interpretation biases and clinical symptoms.

Results supported the hypothesized association between interpretation biases and elevated clinical symptoms. Findings revealed that social anxiety- and depression-related interpretation biases were each significantly correlated with symptoms of social anxiety. Likewise, social anxiety- and depression-related interpretation biases were each correlated with symptoms of depression. These associations were maintained even after controlling for relevant covariates, as well as for the comorbidity between social anxiety and depression symptoms. Contrary to predictions, no evidence of specificity was found. This finding lends support to the conceptualization of biased interpretation as a transdiagnostic vulnerability factor.

It was also predicted that executive control would be inversely associated with both social anxiety and depression symptoms, even after controlling for the comorbidity between the symptoms. Unfortunately, no association between executive control and either social anxiety or depression symptoms was found. The lack of significant associations supporting this hypothesis is not surprising given the lack of significant

findings within Aims 1 and 2. Taken together, results from the current investigation do not support a simple association between non-affective executive control and social anxiety, depression, or emotional vulnerability for these symptoms. It is therefore recommended that future research only consider executive control in the context of emotional states and/or in conjunction with other emotionally valenced cognitive processes.

Lastly, it was hypothesized that interpretation biases and executive control would interact to predict clinical symptoms. Specifically, individuals with a bias to endorse negative interpretations and deficits in executive control were expected to endorse the most severe clinical symptoms. Among the four interaction models tested, only one interaction was found to be significant. Persons with a bias toward negative interpretations and deficits in executive control reported more severe social anxiety symptoms. This is consistent with previous theory and research on cognitive vulnerability for social anxiety (e.g., Rapee & Heimberg, 1997; Bishop, 2007; Beard & Amir, 2008).

It is unclear why none of the other regression analyses produced significant interaction effects. One hypothesis is that cognitive vulnerability, and particularly executive control, plays a bigger role in acute stress reactivity than it does in variations in clinical symptoms. If this is the case, perhaps cognitive processes influence clinical symptoms via stress dysregulation (i.e., an indirect rather than a direct path). For instance, Quinn and Joormann (2015b) did not find a direct association between trait-level executive control and symptoms of depression, but found a significant association between deficits in executive control under conditions of stress and greater depression symptom severity. Future studies should look to tease apart the directionality of the

association between executive control and stress reactivity and may consider implementing methodological designs that would allow for tests of mediation effects.

Limitations and Future Directions

It is important to consider the current investigation in light of its limitations. First, there appear to have been issues with the timing of the recovery period assessment. More specifically, the 10-minute recovery period may have been too long to see individual differences in recovery trajectories. Conversely, there is some evidence that individuals who engage in repetitive negative thinking may experience negative consequences of stress in a delayed fashion. A study by Glynn, Christenfeld, and Gerin (2007) found that individuals asked to ruminate about a stressor occurring one week prior experienced elevated autonomic arousal. In addition, they found that biological response to the stressor was not correlated with biological response to delayed rumination. From these results, the authors concluded that the effects of stress may exist long after termination of the stressor and that those who react intensely to an initial stressor may not be the same as those who experience intense delayed responses. Given this, researchers should consider adding assessments of subjective and biological responding on a smaller (e.g., two minutes) and larger (e.g., two days) time scale to future study designs.

A second limitation of the current investigation was the decision to examine only non-affective executive control. This cognitive process only predicted stress responding or clinical symptoms in a few specific instances and only when it was considered in conjunction with interpretation biases. Some previous research has found that executive control only confers vulnerability for clinical symptoms in the context of emotionally laden information (e.g., Najmi et al., 2010). Without administering an affective version of

the Eriksen Flanker task to participants, the extent to which the lack of significant effects were related to executive control as a broad construct or executive control in the absence of emotionally evocative stimuli cannot be determined. The decision to exclude an affective version of the task was based on theoretical (i.e., a desire to tease apart bottom-up reactivity to emotional stimuli assessed via interpretation biases and top-down regulation of emotional stimuli assessed via non-affective executive control) and practical considerations (i.e., time burden and fatigue effects for participants). However, future investigations interested in further clarifying the nature of executive control as a vulnerability for social anxiety and depression may consider including both versions of this task.

In addition, there were some limitations to the sample used in the current study. Individuals were recruited from the University of Miami, as well as from the Greater Miami Area. Though the study assessed for demographic and clinical differences between these two groups (and statistically controlled for differences as appropriate), it is possible that the two subsamples may have differed in other ways. In addition, the study examined dimensional measures of only social anxiety and depression symptoms. However, research has implicated repetitive negative thinking, dysregulated stress responding, and cognitive vulnerability in other clinical conditions as well (e.g., generalized anxiety, obsessive compulsive disorder, and posttraumatic stress; Arditte et al., in press; Ehring & Watkins, 2008; McEvoy et al., 2013). Whereas this limitation does not detract from the findings of the current study, future investigations should consider including a broader range of clinical constructs to test the limits of the assertion that biased interpretation truly represents a transdiagnostic cognitive vulnerability factor.

More importantly, because the sample included individuals with clinical and non-clinical symptoms of social anxiety and depression, it is possible that there were individual differences in the function or utility of the cognitive factors examined in the current study. The idea that cognitive processes may function differently across different individuals is supported by previous literature. For example, Joormann, Siemer, & Gotlib (2007) found that positive memory recall, a process that has been theoretically linked effective mood repair, improved mood among never depressed persons, left mood unchanged in formerly depressed persons, and actually worsened mood in currently depressed persons. Likewise, when an unselected sample of undergraduates were trained to attend to positive stimuli, results revealed subsequent changes in positive affect that were only present among individuals who reported low levels of rumination (Arditte & Joormann, 2014). It is therefore difficult to draw firm conclusions about the clinical implications of findings from the current investigation.

Despite this, the study's recruitment strategy was consistent with an RDoC framework. Thus, while we may not be able to directly compare the findings of the current investigation with previous research that recruited individuals using a categorical approach, this study can be considered a starting point for future endeavors. Investigators interested in continuing this line of work may consider linking research domains not only to dimensional measures of clinical symptoms, but to more functional measures (e.g., coping, quality of life, impairments in social or occupational functioning, or diagnostic data) to determine clinical relevance of findings.

Finally, because of the correlational nature of analyses in the current study, it is impossible to determine the causal associations between emotional and cognitive

vulnerability factors or between vulnerability factors and clinical symptoms. This is an issue that will need to be addressed in future studies. However, if cognitive and emotional vulnerability factors are determined to causally influence clinical symptoms, they may represent an important target of intervention and prevention efforts.

Conclusion

Findings from the current study support the conceptualization of interpretation biases, particularly biases to endorse threat-related interpretations, as transdiagnostic vulnerability factors associated with increased stress reactivity and symptoms of social anxiety and depression. In addition, interpretation biases may interact with executive control to disrupt stress reactivity and increase risk for social anxiety symptoms. Future studies are needed to more closely examine the directionality of these relationships and the possibility that dysregulated acute stress reactivity serves as a mediator between cognitive vulnerability factors and symptoms of social anxiety and depression.

However, results supplement extant etiological models, as well as prevention efforts. Specifically, they may inform who we think of as "high risk" and for what. In addition, findings lay a critical foundation for clinical research looking to target interpretation biases in social anxiety and depression. For one thing, we may not need to target social anxiety- and depression-related interpretation biases as separate constructs. In addition, we may want to consider targeting interpretation biases among individuals with impaired executive control or to simultaneously target interpretation biases and executive control. Finally, more research should be done to develop disorder-non-specific conceptualizations of social anxiety and depression with the ultimate goal of improving our classification and treatment of these debilitating conditions.

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Table 2.1. *Participant characteristics across community and student samples*.

	Entire Sample ($N = 57$)	Community Sample ($n = 31$)	Student Sample ($n = 26$)	t/χ^2
Age	$M = 30.16$ $SD = 14.56$	$M = 39.00$ $SD = 14.38$	$M = 19.20$ $SD = 1.16$	7.64**
Gender	72% Female 28% Male	81% Female 19% Male	62% Female 38% Male	2.56^{ns}
Race	58% White/Caucasian 16% Black/African-American 17% Asian 7% Multi-racial 2% "Other"	52% White/Caucasian 48% Racial Minority	65% White/Caucasian 35% Racial Minority	1.10^{ns}
Ethnicity	72% Non-Hispanic 28% Hispanic	58% Non-Hispanic 42% Hispanic	88% Non-Hispanic 12% Hispanic	6.47*
RNT	105.14 (14.79)	110.52 (14.79)	98.73 (12.20)	3.24**
MDD Dx	17% Yes 67% No 16% Unsure	29% Yes 45% No 26% Unsure	4% Yes 92% No 4% Unsure	14.15**
SAD Dx	25% Yes 61% No 14% Unsure	39% Yes 39% No 22% Unsure	8% Yes 88% No 4% Unsure	14.78**
Psych Meds	19% Yes 81% No	29% Yes 71% No	8% Yes 92% No	4.14*
BP Meds	4% Yes 96% No	6% Yes 94% No	0% Yes 100% No	1.74^{ns}
Other Meds	16% Yes 84% No	19% Yes 81% No	12% Yes 88% No	$.65^{ns}$

Note. ^{ns} $p > .10$; * $p < .05$, ** $p < .001$; RNT = Repetitive Negative Thinking subscale of the Repetitive Thinking Questionnaire; MDD Dx = % of participants endorsing major depressive disorder; SAD Dx = % of participants endorsing social anxiety disorder; Psych meds = Currently prescribed psychotropic medications; BP meds = Currently prescribed blood pressure medication; Other meds = Currently prescribed some other medication.

Table 4.1. *Descriptive statistics for self-report measures, including social anxiety (BFNE-S) and depression (BDI-II) symptoms, and crystallized intelligence (Shipley).*

	M	*SD*	Range
BFNE-S	25.60	9.18	8, 40
BDI-II	20.61	13.05	0, 48
Shipley	31.89	4.41	19, 40

Note. BFNE-S = Brief Fear of Negative Evaluation – Straightforward Items; BDI-II = Beck Depression Inventory-II; Shipley = Shipley Institute of Living Scale – Vocabulary Test.

Table 4.2. *Descriptive statistics for indices of interpretation biases obtained from the Word Sentence Association Paradigm (WSAP).*

	M	*SD*	Range
Social Anxiety Bias	.41	.20	.08, .88
Depression Bias	.43	.18	.00, .90
General Negative Bias	.42	.18	.05, .84

Table 4.3 *Multiple regression model examining the associations between negative interpretation biases and accuracy (Acc) interference as they relate to subjective stress reactivity (Stressor PANAS-N scores).*

	B	SE	β	*t*	*p*
Constant	9.21	10.08		.91	.37
Age	.01	.10	.02	.12	.91
Ethnicity	.31	2.65	.01	.12	.91
Shipley	5.45	11.96	.06	.46	.65
Baseline 2 PANAS-N	.57	.20	.38	2.84	.007
Negative Bias	22.40	7.87	.40	2.84	.006
Accuracy Interference	20.61	13.14	.32	1.57	.12
Bias x Acc Interference	212.38	120.21	.36	1.77	.08

Note. Negative Bias and Accuracy Interference represent centered scores. Shipley = Shipley Institute of Living Scale – Vocabulary Test; PANAS-N = Negative Affect subscale of the Positive and Negative Affect Schedule (PANAS-N) score.

Table 4.4 *Multiple regression model examining the associations between negative interpretation biases and reaction time (RT) interference as they relate to subjective stress reactivity (Stressor PANAS-N scores).*

	B	SE	β	*t*	*p*
Constant	6.00	9.65		.62	.54
Age	.05	.09	.07	.54	.60
Ethnicity	.59	2.58	.03	.23	.82
Shipley	5.85	11.48	.06	.51	.61
Baseline 2 PANAS-N	.64	.19	.42	3.30	.002
Negative Bias	15.14	6.60	.27	2.29	.03
RT Interference	.02	.02	.12	.99	.33
Bias x RT Interference	-.46	.20	-.27	-2.32	.03

Note. Negative Bias and RT Interference represent centered scores. Shipley = Shipley Institute of Living Scale – Vocabulary Test; PANAS-N = Negative Affect subscale of the Positive and Negative Affect Schedule (PANAS-N) score.

Table 4.5 *Multiple regression model examining the associations between negative interpretation biases and accuracy (Acc) interference as they relate to biological stress reactivity (Stressor RSA).*

	B	SE	β	*t*	*p*
Constant	1.22	.82		1.48	.15
Age	-.02	.007	-.26	-2.74	.008
Ethnicity	-.38	.17	-.16	-2.18	.04
Shipley	1.98	.78	.20	2.54	.01
BP Meds	.16	.46	.03	.36	.72
Baseline RSA	.72	.09	.69	8.35	< .001
Negative Bias	.95	.51	.16	1.87	.07
Accuracy Interference	2.11	.87	.30	2.43	.02
Bias x Acc Interference	26.23	7.98	.41	3.29	.002

Note. Negative Bias and Accuracy Interference represent centered scores. Shipley = Shipley Institute of Living Scale – Vocabulary Test; BP Meds = Currently prescribed blood pressure medication; RSA = respiratory sinus arrhythmia.

Table 4.6 *Multiple regression examining the associations between negative interpretation biases and reaction time (RT) interference as they relate to biological stress reactivity (Stressor RSA).*

	B	SE	β	*t*	*p*
Constant	.90	.87		1.04	.30
Age	-.02	.007	-.20	-2.07	.04
Ethnicity	-.26	.19	-.11	-1.36	.18
Shipley	1.70	.84	.17	2.04	.05
BP Meds	.16	.48	.03	.33	.75
Baseline RSA	.75	.09	.73	8.31	< .001
Negative Bias	.07	.46	.01	.15	.86
RT Interference	-.002	.002	-.09	-1.03	.31
Bias x RT Interference	-.03	.01	-.16	-1.96	.06

Note. Negative Bias and RT Interference represent centered scores. Shipley = Shipley Institute of Living Scale – Vocabulary Test; RSA = respiratory sinus arrhythmia.

Table 4.7 *Multiple regression model examining the associations between negative interpretation biases and accuracy (Acc) interference as they relate to subjective stress recovery (Recovery PANAS-N scores).*

	B	SE	β	*t*	*p*
Constant	-3.24	6.77		-.48	.63
Age	.12	.07	.22	1.84	.07
Ethnicity	.42	1.81	.02	.23	.82
Shipley	.06	.20	.04	.31	.76
Stressor PANAS-N	.52	.09	.66	5.66	< .001
Negative Bias	-10.83	5.79	-.25	-1.87	.07
Accuracy Interference	-32.22	9.09	-.63	-3.55	.001
Bias x Acc Interference	-256.71	83.71	-.55	-3.07	.004

Note. Negative Bias and Accuracy Interference represent centered scores. Shipley = Shipley Institute of Living Scale – Vocabulary Test; PANAS-N = Negative Affect subscale of the Positive and Negative Affect Schedule (PANAS-N) score.

Table 4.8 *Multiple regression examining the associations between negative interpretation biases and reaction time (RT) interference as they relate to subjective stress recovery (Recovery PANAS-N scores).*

	B	SE	β	*t*	*p*
Constant	-1.89	7.02		-.27	.79
Age	.04	.07	.07	.56	.58
Ethnicity	-.42	1.92	-.02	-.22	.83
Shipley	.15	.21	.08	.70	.49
Stressor PANAS-N	.52	.10	.66	5.35	< .001
Negative Bias	-1.90	5.14	-.04	-.37	.71
RT Interference	.01	.02	-.09	.74	.46
Bias x RT Interference	.42	.15	-.32	2.83	.007

Note. Negative Bias and RT Interference represent centered scores. Shipley = Shipley Institute of Living Scale – Vocabulary Test; PANAS-N = Negative Affect subscale of the Positive and Negative Affect Schedule (PANAS-N) score.

Table 4.9. Pearson's *r* correlations between interpretation biases and symptoms of social anxiety (BFNE-S) and depression (BDI-II).

	BFNE-S	BDI-II
Social Anxiety Bias	.63***	.43**
Depression Bias	.48***	.41**
General Negative Bias	.58***	.44**

Note. ** $p < .01$; *** $p < .001$; BFNE-S = Brief Fear of Negative Evaluation Scale – Straightforward Items; BDI-II = Beck Depression Inventory, Second Edition.

Table 4.10 *Multiple regression model examining the associations between negative interpretation biases and accuracy (Acc) interference as they relate to social anxiety symptoms (BFNE-S scores).*

	B	SE	β	*t*	*p*
Constant	14.54	8.11		1.79	.08
Age	-.11	.08	-.18	-1.37	.18
Ethnicity	4.52	2.23	.22	2.02	.05
Shipley	7.81	10.17	.09	.77	.45
BDI-II	.13	.10	.18	1.22	.23
Negative Bias	31.62	7.29	.62	4.34	<.001
Accuracy Interference	19.63	10.52	.33	1.87	.07
Bias x Acc Interference	178.24	97.04	.33	1.84	.07

Note. Negative Bias and Accuracy Interference represent centered scores. BFNE-S = Brief Fear of Negative Evaluation Scale – Straightforward Items; Shipley = Shipley Institute of Living Scale – Vocabulary Test; BDI-II = Beck Depression Inventory, Second Edition.

Figure 1.1. *Example of RDoC-consistent methodology*.

Figure 2.1. *Sample trial from the Word Sentence Association Paradigm (WSAP).*

Figure 2.2. *Sample trial from the Flanker task.*

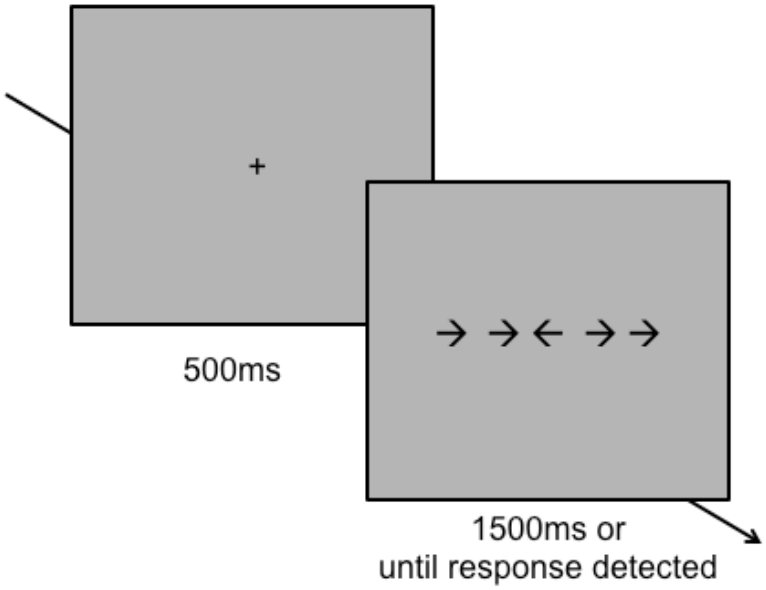


Figure 2.3. *Social-evaluative stress task procedures*.

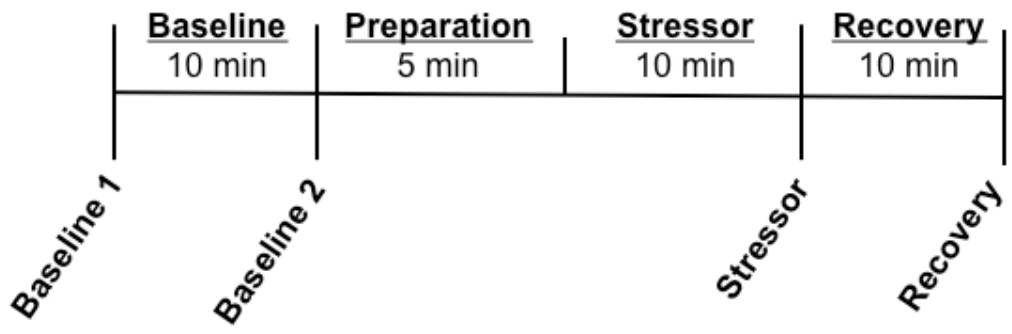
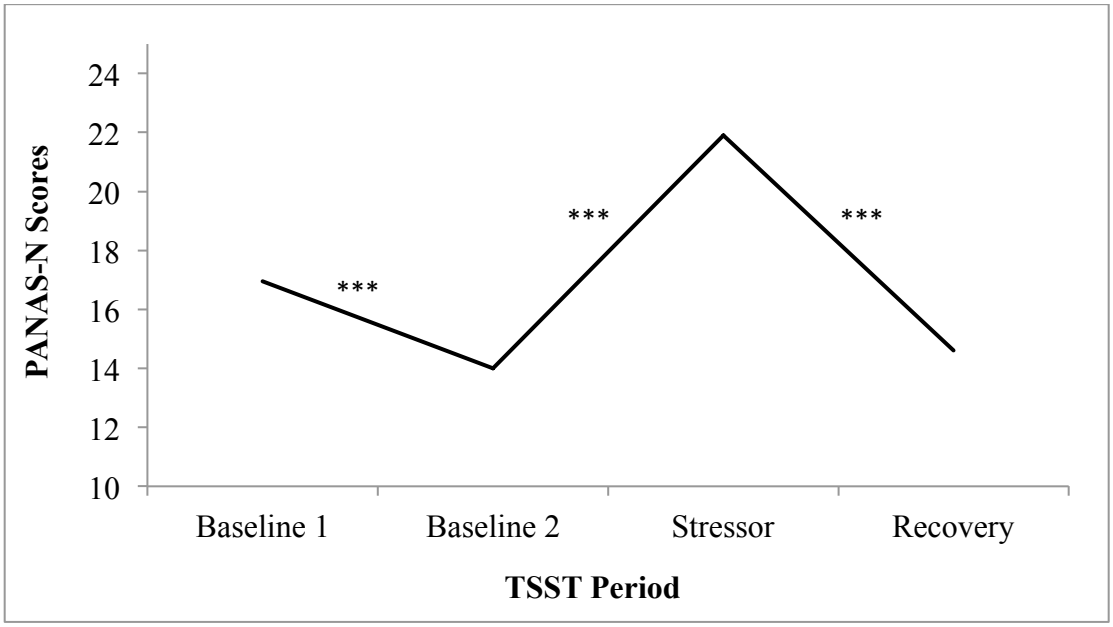
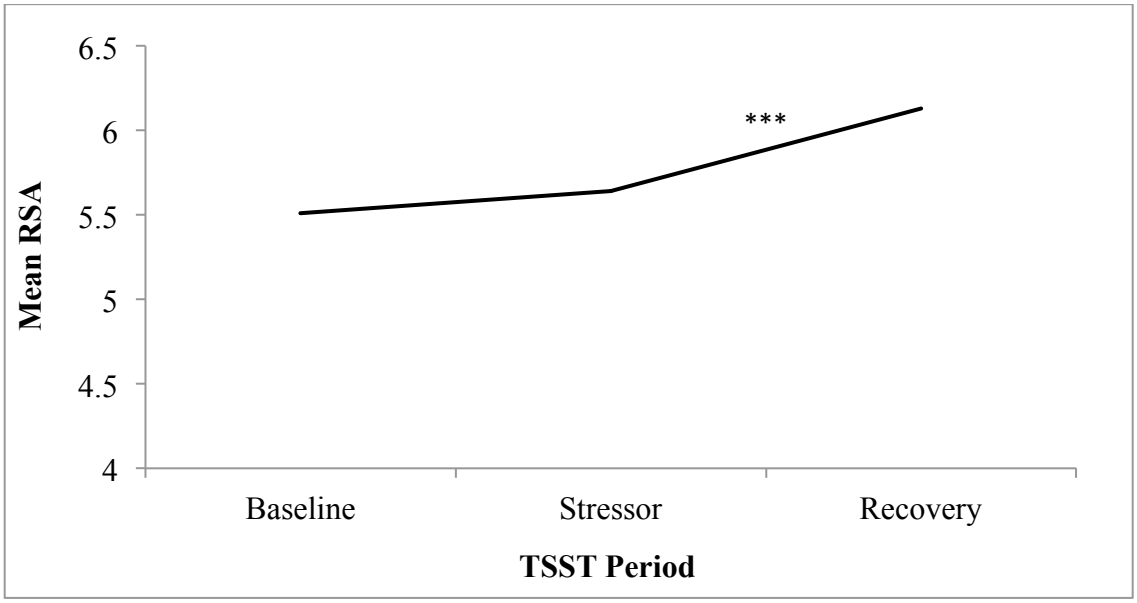


Figure 4.1. *Participants' subjective negative affect (PANAS-N) over the course of the Trier Social Stress Test (TSST).*



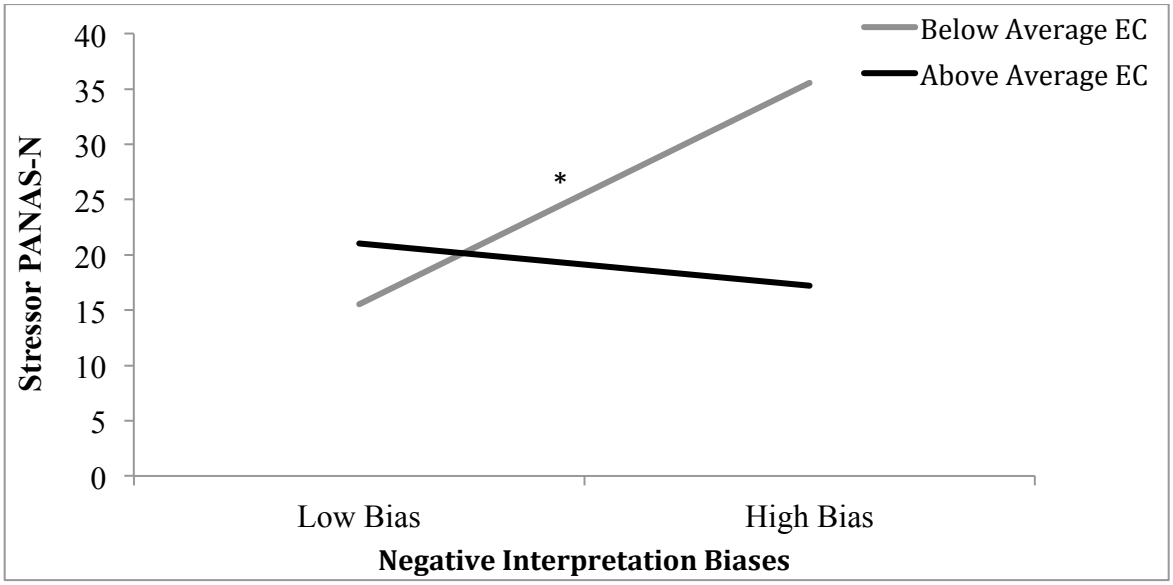
Note. *** $p < .001$; PANAS-N = Negative Affect subscale of the Positive and Negative Affect Schedule.

Figure 4.2. *Participants' physiological responsivity (RSA) over the course of the social-evaluative stressor.*



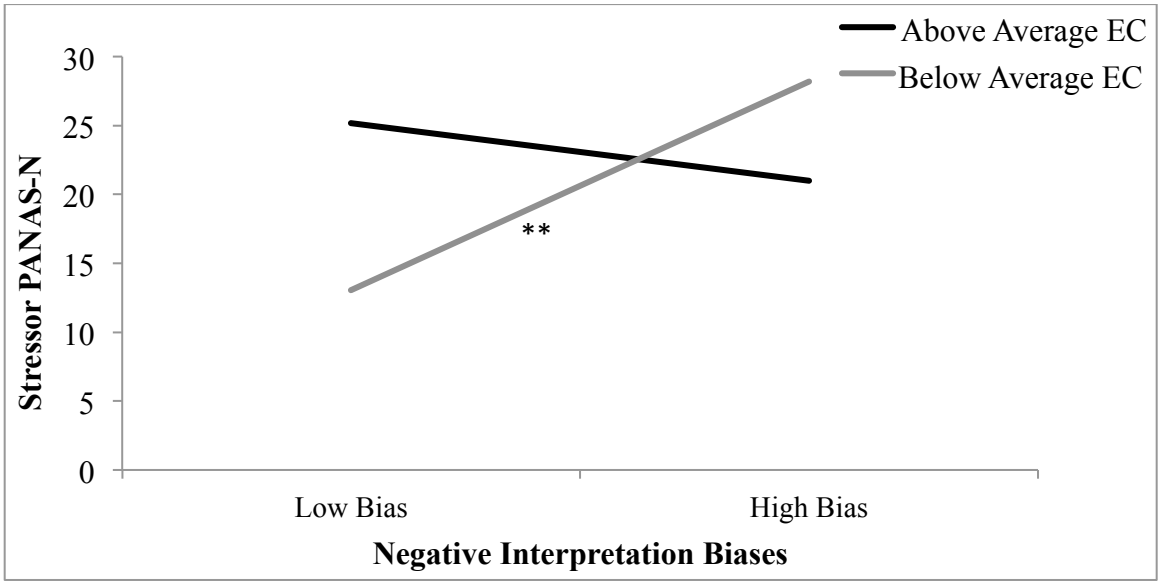
Note. *** $p < .001$; RSA = respiratory sinus arrhythmia.

Figure 4.3. *The interaction between negative interpretation biases and Flanker Task accuracy interference, an index of executive control (EC), as it relates to subjective stress reactivity (PANAS-N).*



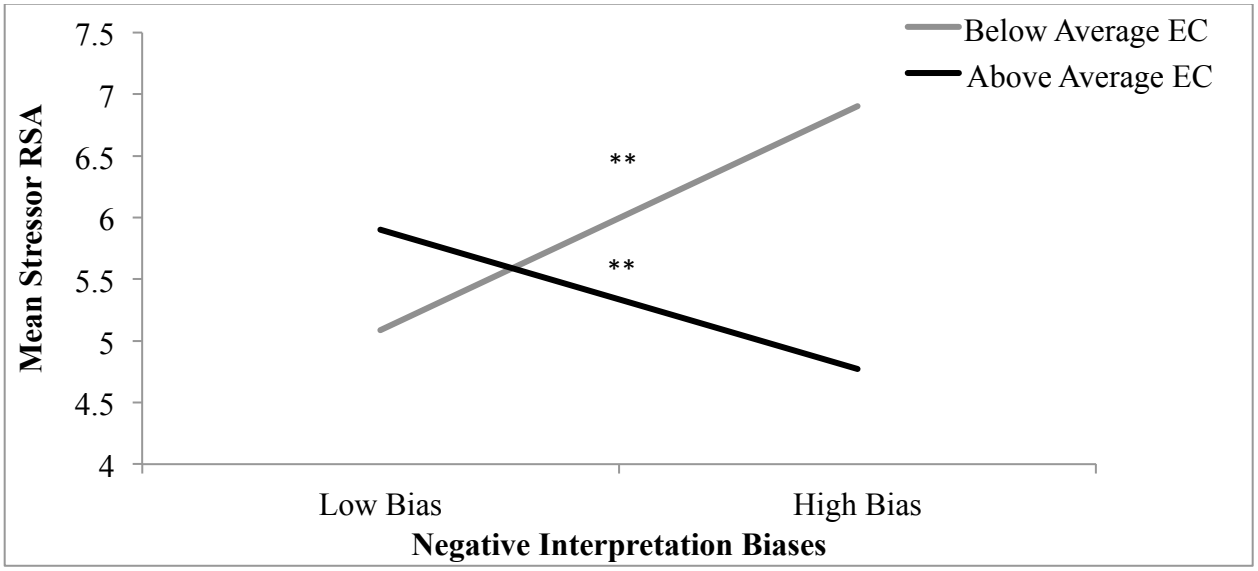
Note. * $p < .05$; Below Average EC = +1 *SD* from centered accuracy interference mean, whereas Above Average EC = -1 *SD* from centered accuracy interference mean; Covariates include age, ethnicity, Shipley total scores, and Baseline PANAS-N scores.

Figure 4.4. *The interaction between negative interpretation biases and Flanker Task accuracy interference, an index of executive control (EC), as it relates to subjective stress reactivity (PANAS-N).*



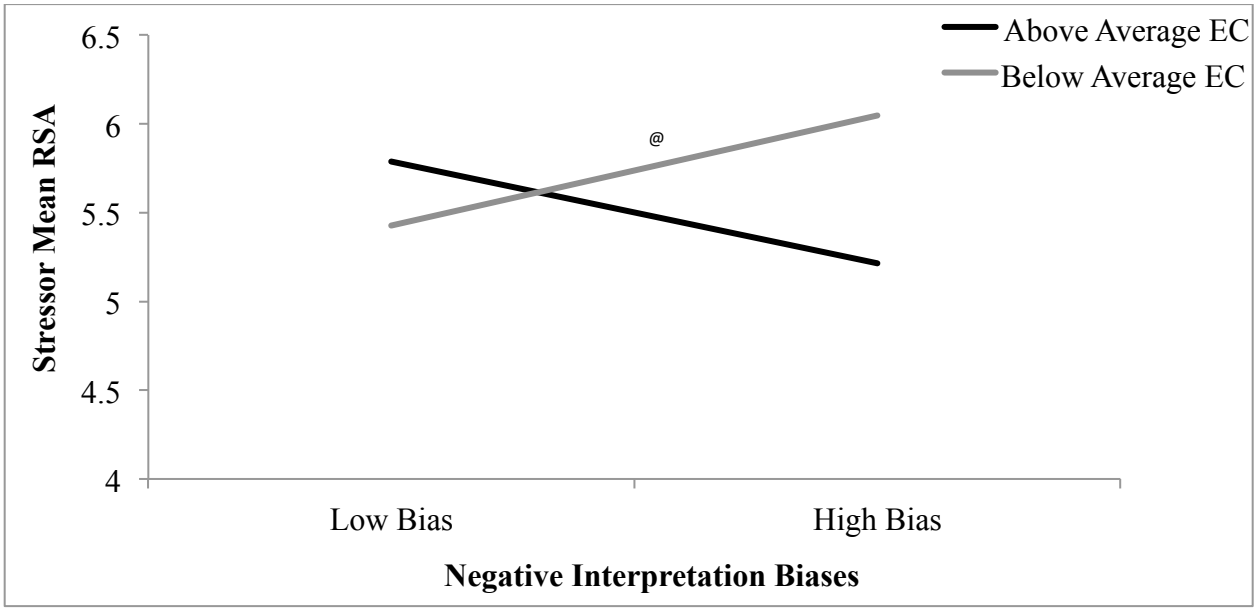
Note. ** $p < .01$; Below Average EC = -1 SD from centered reaction time interference mean, whereas Above Average EC = +1 SD from centered reaction time interference mean; Covariates include age, ethnicity, Shipley total scores, and Baseline PANAS-N scores.

Figure 4.5. *The interaction between negative interpretation biases and Flanker Task accuracy interference, an index of executive control (EC), as it relates to biological stress reactivity (RSA).*



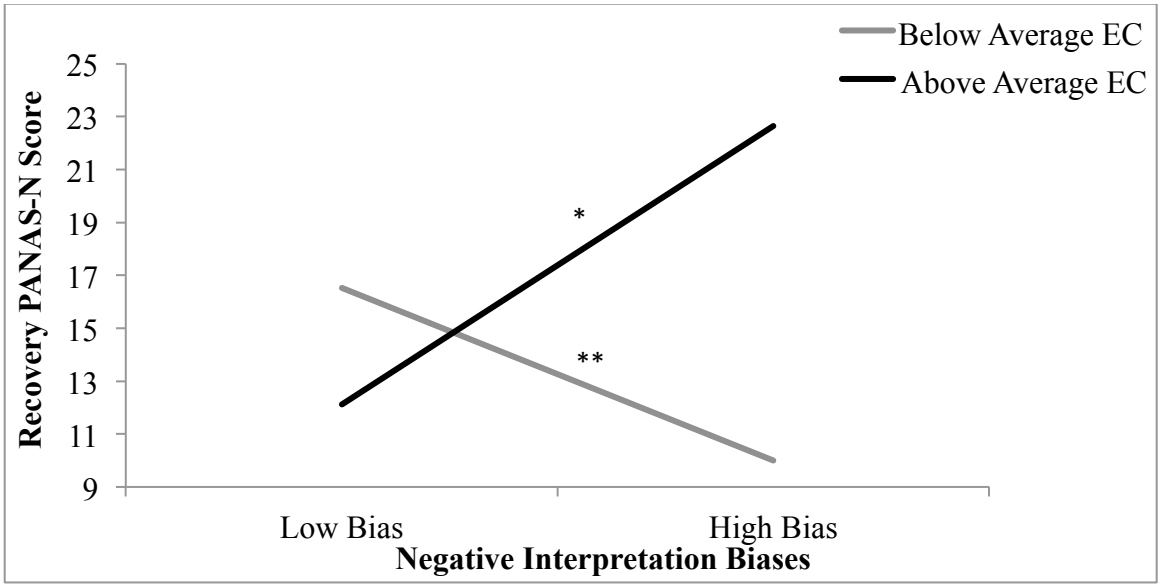
Note. ** $p < .01$; Below Average EC = +1 *SD* from centered accuracy interference mean, whereas Above Average EC = -1 *SD* from centered accuracy interference mean; Covariates include age, ethnicity, Shipley total scores, blood pressure medication status, and Baseline RSA.

Figure 4.6. *The interaction between negative interpretation biases and Flanker Task reaction time interference, an index of executive control (EC), as it relates to biological stress reactivity (RSA).*



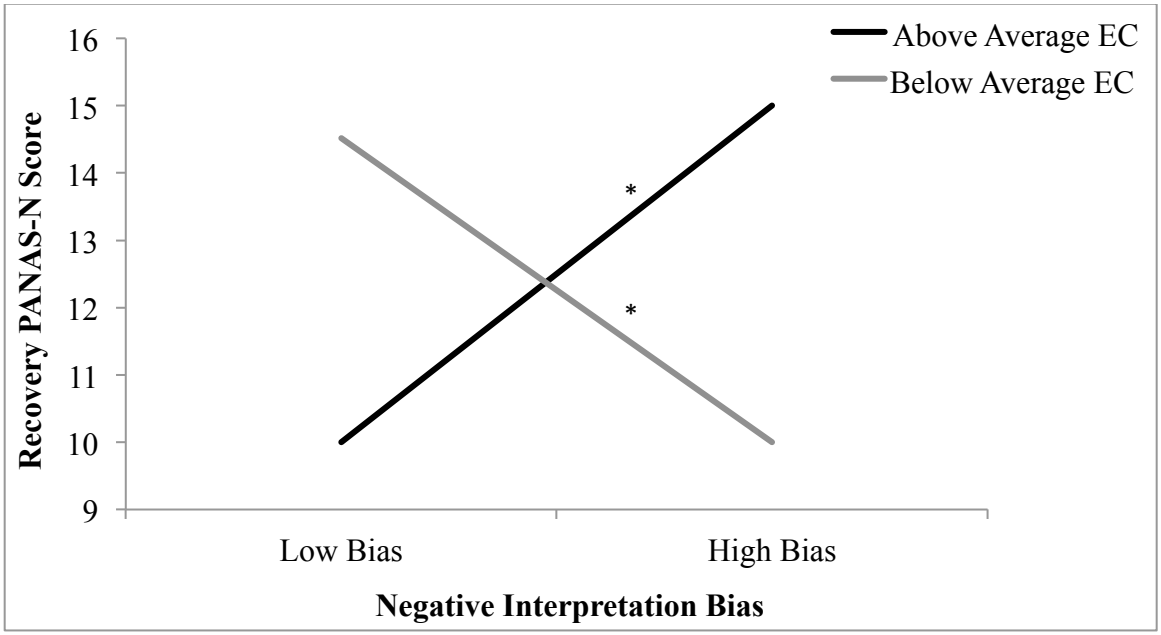
Note. @ $p < .10$; Below Average EC = -1 SD from centered reaction time interference mean, whereas Above Average EC = +1 SD from centered reaction time interference mean; Covariates include age, ethnicity, Shipley total scores, blood pressure medication status, and Baseline RSA.

Figure 4.7. *The interaction between negative interpretation biases and Flanker Task accuracy interference, an index of executive control (EC), as it relates to subjective stress recovery (PANAS-N).*



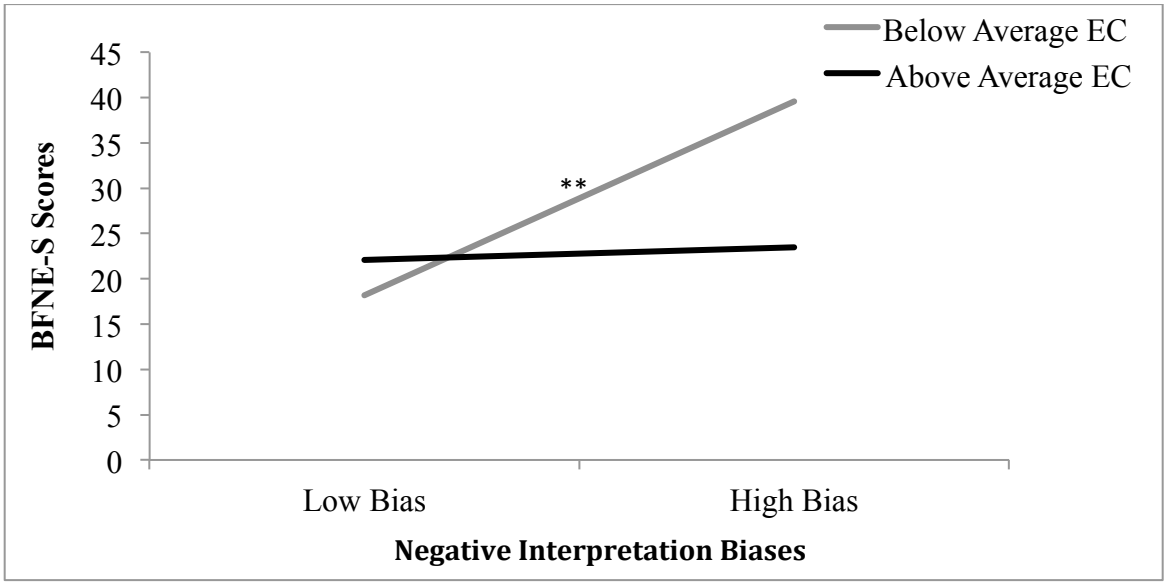
Note. * $p < .05$; ** $p < .01$; Below Average EC = +1 *SD* from centered accuracy interference mean, whereas Above Average EC = -1 *SD* from centered accuracy interference mean; Covariates include age, ethnicity, Shipley total scores, and Stressor PANAS-N scores.

Figure 4.8. *The interaction between negative interpretation biases and Flanker Task reaction time interference, an index of executive control (EC), as it relates to subjective stress recovery (PANAS-N).*



Note. * $p < .05$; Below Average EC = -1 SD from centered reaction time interference mean, whereas Above Average EC = +1 SD from centered reaction time interference mean; Covariates include age, ethnicity, Shipley total scores, and Stressor PANAS-N scores.

Figure 4.9. *The interaction between negative interpretation biases and Flanker Task accuracy interference, an index of executive control (EC), as it relates to social anxiety symptoms (BFNE-S scores).*



Note. ** $p < .01$; Below Average EC = +1 *SD* from centered accuracy interference mean, whereas Above Average EC = -1 *SD* from centered accuracy interference mean; Covariates include age, ethnicity, Shipley total scores, and BDI-II scores.

Appendix

A.1. Repetitive Negative Thinking subscale of the Repetitive Thinking Questionnaire

Please think of the last time you felt particularly distressed. Briefly describe what caused you to feel distressed here:

_____

_____

Now, please rate how accurately each of the following statements describes your experience after the distressing situation, using a scale from 1 – 5, where 1 means the statement is *not true at all*, 3 means it is *somewhat true*, and 5 means it is *very true* of your experience.

	Not at all true		Somewhat True		Very true
	1	2	3	4	5
1. I knew I shouldn't have thought about the situation, but I couldn't help it.	1	2	3	4	5
2. I noticed that I had been thinking about the situation.	1	2	3	4	5
3. I had thoughts or images of the situation that were difficult to forget.	1	2	3	4	5
4. Once I started thinking about the situation, I couldn't stop.	1	2	3	4	5
5. I had thoughts or images of the situation that I tried to resist thinking about.	1	2	3	4	5
6. I had thoughts or images that "*I won't be able to do my job/work because I feel so badly.*"	1	2	3	4	5
7. I thought about the situation all the time.	1	2	3	4	5
8. I had thoughts or images about all my shortcomings, failings, faults, mistakes.	1	2	3	4	5
9. I had thoughts or images about a past event that came into my head even when I did not wish to think about it again.	1	2	3	4	5
10. I had thoughts or images about the situation and wishing it had gone better.	1	2	3	4	5
11. I had a lot of thoughts or images of the situation after it was over.	1	2	3	4	5
12. My thoughts overwhelmed me.	1	2	3	4	5
13. I had thoughts or images about how alone I felt.	1	2	3	4	5
14. I went some place alone to think about my	1	2	3	4	5

feelings.

15. I had thoughts or images about how angry I was with myself.	1	2	3	4	5
16. I had thoughts or images asking "*Why do I always react this way*?"	1	2	3	4	5
17. The situation really made me think.	1	2	3	4	5
18. I had thoughts or images about the situation that occurred over and over again, that resulted in my feelings getting worse and worse.	1	2	3	4	5
19. I had thoughts or images about the situation that resulted in me avoiding similar situations and that reinforced a decision to avoid similar situations.	1	2	3	4	5
20. I went away by myself and thought about why I felt this way.	1	2	3	4	5
21. I had thoughts or images like "*Why can't I get going*?"	1	2	3	4	5
22. I was always thinking about something.	1	2	3	4	5
23. I had thoughts or images about turning the clock back to do something again, but do it better.	1	2	3	4	5
24. I had thoughts or images like "*Why do I have problems that other people don't have?*"	1	2	3	4	5
25. I listened to sad music.	1	2	3	4	5
26. When I was under pressure, I thought a lot about the situation.	1	2	3	4	5
27. I thought about the situation until it was all done.	1	2	3	4	5