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The ABCs of stress responding: Examining the time course of affective, biological, and cognitive responses to induced stress as prospective predictors of depressive symptoms

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The ABCs of Stress Responding: Examining the Time Course of Affective, Biological, and

Cognitive Responses to Induced Stress as Prospective Predictors of Depressive Symptoms

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Abstract

Vulnerability-stress models of depression posit risk for depression is characterized by the presence of underlying affective, biological, and cognitive vulnerabilities that become activated during life stress exposure. Extant research has shown heightened reactivity to stress across these vulnerability domains predicts depression; however, little is known whether the persistence of and failure to down-regulate these maladaptive stress responses conveys greater risk of depression than initial reactivity alone. The current study examined associations between the time course of responses to a laboratory stress induction and depressive symptoms. I hypothesized that prolonged maladaptive responses to the stressor across affective (state negative affect; NA), biological (respiratory sinus arrhythmia; RSA), and cognitive (rumination) domains would be most strongly associated with concurrent and prospective depressive symptoms, above and beyond trait vulnerabilities and initial reactivity to stress. I also expected these associations would be moderated by life stress exposure during the 8-week follow up period. The sample was comprised of 92 young adults ages 18-24 (M = 19.50; SD = 1.37), 72.9% of whom identified as Caucasian and 82.6% as female. Analyses indicated prolonged NA following the stressor was associated with concurrent (B = 8.11, p < .001), but not prospective (B = -0.74, p = .77) depressive symptoms. High NA during stress marginally interacted with life stress exposure to predict greater symptoms at follow up (B = 0.28, p = .15). RSA recovery from stress was not associated with symptoms concurrently (B = 2.2, p = .61) or prospectively (B = -1.08, p = .39) and did not interact with life stress exposure. Prolonged rumination about the stressor was also not associated with depressive symptoms concurrently (B = 0.70, p = .62) or at follow up (B = -1.42, p = .25) and was not moderated by life stress exposure. Although hypotheses were only

partially supported, the current study's findings provide important implications for understanding

the role of recovery from stress in the development, maintenance, and treatment of depressive

symptoms among young adults.

CHAPTER I

Introduction and Literature Review

Purpose

Depression is a significant mental health problem, particularly among adolescents and young adults. As early prevention and treatment are key to improved outcomes for depressive disorders (National Institute of Mental Health [NIMH], 2008), a clear understanding of the mechanisms that confer vulnerability for the development and maintenance of depressive symptoms is crucial. Vulnerability-stress models (Abramson, Metalsky, & Alloy, 1989; Nolen-Hoeksema, 1991) are among the most empirically supported models of depression that have attempted to identify salient etiological pathways to the disorder. Specifically, the ABC model of depression (Hyde, Mezulis, & Abramson, 2008) posits that certain affective, biological, and cognitive responses to stress represent proximal vulnerabilities for the development of adolescent depression. While extant experimental research has examined these domains of stress reactivity among depressed, nondepressed, and remitted individuals, few studies have examined their predictive relationships with depression, particularly in interaction with life stress exposure. Moreover, prior research using stress or mood inductions have typically only utilized measures of stress *reactivity*; thus, it is unclear what role stress *recovery* may have in contributing to depression. Depression is characterized by enduring negative cognitions and affect; thus, it is possible that the time course or persistence of maladaptive stress responses may function as proximal vulnerabilities to the disorder and may be more salient predictors of future depressive outcomes than initial reactivity alone.

The purpose of the current study was to better elucidate the relationship between proximal responses to induced stress and depressive symptoms over time. I hypothesized that patterns of affective (state emotional reactivity), biological (respiratory sinus arrhythmia [RSA]),

and cognitive (state rumination) reactivity to and recovery from a laboratory stress induction will predict depressive symptoms eight weeks later in a sample of young adults. I further hypothesized the relationship between stress response patterns and depressive symptoms would be moderated by exposure to life stress over the eight-week study period. In the sections that follow, I provide an overview of the epidemiology of depression among adolescents and young adults and discuss the utility of conceptualizing the etiology of depression from a vulnerability-stress perspective. Next, I provide a review of relevant theory and research examining affective, biological, and cognitive domains of both trait and state vulnerabilities to depression. I conclude the literature review by discussing how sustained abnormal responses to stressful events may indicate a unique vulnerability for depression.

Depression among Adolescents and Young Adults

While estimates vary, previous epidemiological research demonstrates that rates of depression increase considerably in the transition into adolescence and young adulthood. Among children, lifetime prevalence of major depressive disorder (MDD) is 1.1% (Kashani et al., 1983). However, by adolescence, rates of lifetime MDD diagnoses range from 4% (Whitaker et al., 1990) to as high as 24% by some estimates (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993).

Evidence suggests in addition to criterion-based diagnoses, subthreshold symptoms should also be considered when examining depression among youth. Among adolescents and young adults, depressive symptoms are associated with increased suicidality (Andrews & Lewinsohn, 1992), substance use (Lewinsohn, Solomon, Seeley, & Zeiss, 2000), and poor psychosocial functioning and academic performance (Gotlib, Lewinsohn, & Seeley, 1995; Rothon et al., 2008). Moreover, several studies have shown prospective associations between

adolescent depressive symptoms and adult-onset MDD (Bardone, Moffitt, Caspi, Dickson, & Silva, 1996; Fergusson, Horwood, Ridder, & Beautrais, 2005). These findings suggest depressive symptoms and diagnoses may be best viewed on a continuum of severity and that subthreshold symptoms may represent risk for later development of the disorder.

Studies utilizing self-report measures with community samples of adolescents estimate that 20-50% of youth experience subthreshold depressive symptoms (Kessler, Avenevoli, & Merikangas, 2001). College students exhibit similar prevalence rates. For example, Rosenthal and Schreiner (2000) found that in a diverse sample of nearly 600 undergraduate students at an urban university, 29% of respondents self-reported moderate levels of depressive symptoms, while 26.7% reported experiencing clinically significant symptom levels. In a nationally-representative survey of almost 16,000 undergraduates, 44.5% of respondents reported experiencing at least one time in the past year in which they felt so depressed that it was difficult to function (Kisch, Leino, & Silverman, 2005). Taken together, these data suggest adolescence and young adulthood are salient periods of risk for the development of depression and may be particularly important for understanding mechanisms that contribute to vulnerability for the disorder.

Vulnerability-Stress Models of Depression

Early etiological models of depression have posited two distinct primary pathways to the development of depression: *vulnerability* and *stress exposure*. Vulnerabilities (also termed *diatheses*) are defined as factors that predispose individuals to experiencing adverse states or outcomes (Ingram & Luxton, 2005). Depressogenic vulnerabilities can be broadly categorized into affective, biological, and cognitive domains (Hyde et al., 2008).

Early approaches to the conceptualization of depression also considered the contributing role of stress. Stress is conceptualized as strain or demands on an individual's ability to adaptively maintain both physiological and psychological homeostasis (Selye, 1963) and is most commonly examined in the context of eliciting environmental factors. These factors, termed *stressors*, are predominately comprised of both major and minor negative life events (Lazarus, 1990). The relationship between major and minor stressors and onset of depression is well-documented (see Monroe, Slavich, & Georgiades, 2009 for a review).

It is important to note the inherent quality of life events is neither positive nor negative. Lazarus and Folkman (1984) suggested a stressor is distinct from an individual's subjective experience of it. In other words, the extent to which a stressor is perceived as being stressful depends on the individual's appraisals, reactions, and subjective experiences of the event. According to this perspective, stress cannot be completely disentangled from cognitive appraisal processes that may be subject to influence by individual vulnerability factors. Indeed, data indicates only 20-50% of individuals develop depression following significant life stress (Monroe et al., 2009). This suggests individual difference factors may moderate the consequences of stress exposure. Therefore, it may be useful to consider the joint contributions of both stress and vulnerabilities.

Current etiological models of depression posit that vulnerabilities and stress exposure act in concert to confer heightened risk for the development and maintenance of depressive disorders. These models, commonly referred to as *vulnerability-stress* or *diathesis-stress* models, arose out of approaches that sought to explain the development of schizophrenia among individuals with genetic predispositions to the disorder. Specifically, Meehl (1962) posited that schizotypic personality (a phenotype of genetic risk for schizophrenia) is a necessary, but

insufficient etiological pathway for the disorder. Rather, stress (specifically aversive mother-child interactions) must be present to activate latent genetic risk. According to Meehl's perspective, even severe stress exposure will likely not result in schizophrenia onset unless a genetic predisposition is present.

Meehl's etiological model of schizophrenia (1962) laid the groundwork for the application of the vulnerability-stress framework to other psychopathologies, including depression. However, in contrast to Meehl's original conceptualization, current vulnerability-stress models emphasize a continuous, rather than dichotomous, approach. These models account for the presence of varying degrees of both vulnerability and stress and suggest that the likelihood one will develop depression is dependent upon the strength of the interaction (Monroe & Simons, 1991).

Vulnerability-stress perspectives hold great utility in that they help to identify which individuals may go on to develop depression and also aid in characterizing under what conditions onset will occur. These frameworks posit that stress activates latent endogenous vulnerabilities and brings them online to influence proximal responses to the stressor (Scher, Ingram, & Segal, 2005). In this way, an individual may exhibit a tendency towards high stress reactivity and poor recovery in the affective, biological, and/or cognitive domains; however, in the absence of stress exposure, these vulnerabilities are not likely to exert influence over mental health outcomes.

Much of the research applying vulnerability-stress models to depression conceptualize vulnerability factors as possessing traitlike qualities. Although evidence suggests many depressogenic vulnerabilities exhibit stability over time, the ways in which vulnerabilities are manifested during stress exposure may differ depending on the specific context. Indeed, prior research has shown some trait vulnerabilities to depression such as negative cognitive style share

limited variance with their state correlates (Hong, Gwee, & Karia, 2006). Moreover, many indices of trait rumination and trait affectivity rely on the use of hypothetical scenarios (e.g., Mezulis, Abramson, & Hyde, 2002) or contain items that reference specific contexts such as watching a sad movie (e.g., Larsen, Diener, & Emmons, 1986) that may not be salient enough to represent self-referential negative events that would theoretically contribute to depression onset. Such events are important to consider within the vulnerability-stress framework, as depression is hypothesized to occur after negative events that result in loss or failure in a valued domain (Pyszczynski & Greenberg, 1987). Moreover, depressogenic vulnerability is characterized by preferential processing of negative self-referential stimuli (Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997). Thus, the use of *in-vivo* stressors that elicit such latent processing biases as well as the use of event-specific or state measurement of these processes may allow for a more ecologically valid operationalization of depressogenic vulnerability. Thus, the current study draws upon the trait-state distinction by hypothesizing specific patterns of stress responding represent proximal state vulnerabilities that predict depressive symptoms above and beyond the contribution of trait vulnerabilities.

Affective Vulnerability to Depression

Sustained negative affect is one of the hallmark features of depressive disorders. In fact, depression is becoming increasingly recognized as a disorder of disrupted emotion regulation, suggesting that individuals who are depressed may have difficulty managing and coping with negative affect once elicited. From a vulnerability perspective, affective models of depression emphasize the role of individual differences in emotional reactivity and regulation capacity that may convey risk for the subsequent development of depressive disorders (Compas, Connor-Smith, & Jaser, 2004; Mezulis, Hyde, Simonson, & Charbonneau, 2011). Thus, affective models

assert that affectivity not only predicts the course of the disorder, but also precedes its onset. In the following sections, I discuss theories of affective vulnerability to depression and review relevant research examining both trait and reactive components of affectivity.

      Definition and theoretical foundations. Affective models of depression generally consider the trait-state distinction in discussing emotional vulnerability for the disorder. These models suggest that trait or dispositional levels of affectivity directly influence the propensity for experiencing certain types of emotions and as well as a heightened intensity of emotional experiences (Morris, Bylsma, & Rottenberg, 2009).

      Trait affectivity is typically indexed by measures of temperament. Temperament is conceptualized as multidimensional, constitutionally-based individual differences in basic emotionality and self-regulation that are present at infancy and that demonstrate stability across time and context (Rothbart & Bates, 1998). One broad temperamental construct that has been heavily implicated in the development of depressive disorders is trait negative affectivity (NA). This temperamental constellation is characterized by the presence of high levels of negative emotions such as fear, distress, and sadness, as well as well as heightened sensitivity to negative cues (Belsky, Hsieh, & Crnic, 1996). It is important to note trait NA does not include regulatory aspects of temperament and is therefore considered to only reflect the typical magnitude and frequency of negative emotional responses. Trait emotional reactivity falls under the umbrella of trait NA and is conceptualized as the high frequency, intensity, and greater duration of negative emotions in response to negative stimuli. The majority of prior literature examining affectivity at the trait level refers to the broader construct of NA, rather than emotional reactivity. Therefore, although the current study emphasizes the role of emotional reactivity as a vulnerability for depression, I refer more frequently to NA in the following literature review.

Trait negative affectivity as a vulnerability to depressive symptoms. Extant research has demonstrated a strong association between trait NA and depression (e.g., Anthony, Lonigan, Hooe, & Phillips, 2002; Lengua, West, & Sandler, 1998). In particular, Kendler, Gatz, Gardner, & Pedersen (2006) found that greater self-reported neuroticism (a construct often used interchangeably with trait NA) not only predicted greater lifetime risk for depression across a 25-year span, but also predicted first episodes of MDD. Moreover, mother-rated infant NA is associated with greater depressive symptoms in adolescence (Mezulis, Priess, & Hyde, 2011). Although trait NA may reflect common affective features of depression, these studies demonstrate the construct can be measured prior to depression onset; thus, trait NA functions as a salient vulnerability factor that can be distinguished from mood symptoms.

Recent investigations utilizing short-term designs have also demonstrated links between temperament and depressive symptoms. For example, Mezulis & Rudolph (2012) found that among a community sample of 110 adolescents, trait NA prospectively predicted greater weekly self-reported depressive symptoms across eight weeks. Other community adolescent studies using two-timepoint designs with longer 5-month (Verstraeten, Vasey, Raes, & Bijttebier, 2009) and 12-month (Wetter & Hankin, 2009) follow up periods similarly demonstrate greater trait NA at Time 1 predicts higher symptom levels at Time 2.

Affective reactivity to stress as a vulnerability to depressive symptoms. State affectivity is hypothesized to emerge largely from trait affectivity. That is, trait levels of affectivity are proposed to heavily influence the proximal experience of emotions, making it more likely one will experience transitory emotional states that are congruent with their affective disposition, particularly under conditions of stress exposure. State NA (or state emotional reactivity) is considered to be the manifestation of temperamental emotionality on an event-

specific basis (Morris et al., 2009; Simonson, Sánchez, Arger, & Mezulis, 2011). While it is hypothesized that trait NA or emotional reactivity will directly influence the potential to experience transitory negative affect, particularly in response to stress, these moment-by-moment affective reactions should be expected to vary to some degree, depending upon the context and salience of the stressor. Thus, it is possible affective reactivity to a given stressful event may predict future depression above and beyond trait levels of NA or trait emotional reactivity.

Consistent with the finding that trait NA prospectively predicts depressive symptoms and disorders, a number of studies have also demonstrated evidence for the role of state NA (O'Neill, Cohen, Tolpin, & Gunthert, 2004; Parrish, Cohen, & Laurenceau, 2011; Witchers et al., 2009; Witchers et al., 2010). For example, using daily diary designs with college students, Parrish and colleagues (2011) found daily state NA for stressful events assessed over the course of one week predicted depressive symptoms at a 2-month follow up, even after controlling for initial symptoms.

Affective recovery from stress may predict depressive symptoms. State negative affectivity may be indicated by both the intensity and duration of negative emotions following stress. Persistent negative affect is likely to exacerbate negative cognitions (Teasdale, 1988) and may indicate ineffective attempts to regulate emotions (Beevers, 2005). Only a handful of studies have examined poor affective recovery (or the persistence of negative affect in response to stress) as a vulnerability to depression. Across two experiments, Gilboa and Gotlib (1997) found that individuals with a history of clinically significant self-reported depressive symptoms were more likely to experience prolonged negative affective states following a mood induction than individuals without a history. Beevers and Carver (2003) extended these findings to the prospective prediction of depressive symptoms. They found that among college undergraduates,

persistent state NA following a similar paradigm interacted with negative life events to predict

depressive symptoms seven weeks later. However, while the authors controlled for pre-

induction levels of negative affect, they did not control for trait levels of affectivity and also

utilized a mood induction procedure (focusing on a best friends' possible death) that may not

have elicited negative self-referential thoughts. Thus, it is unclear whether poor affective

recovery from stress functions as a vulnerability factor for future depression above the effects of

trait NA.

Biological Vulnerability to Depression

Biological vulnerabilities to depression encompass a broad range of interrelated genetic

factors and neurobiological processes that can be measured in numerous ways. One biological

pathway that may contribute to the development of depression is the dysfunction of cardiac vagal

control (CVC). Often indexed by RSA, vagal control of the heart serves as a biomarker for self-

regulatory capacity (particularly under conditions of stress) and is associated with other

biological vulnerabilities to depression, such as the 5-HTTLPR polymorphism (Ellis, Beevers,

Hixon, & McGeary, 2011). Thus, CVC may be an important factor to consider within the

vulnerability-stress framework. Below, I discuss the theoretical background for CVC, define the

construct of RSA and review relevant literature examining the RSA-depression relationship,

particularly among adolescents.

Definition and theoretical foundations. The vagus nerve, also known as cranial nerve

X, originates in the brain stem and projects into the viscera. The vagus carries both afferent and

efferent motor and sensory signals and is considered to be part of a larger integrated autonomic

feedback system that regulates both affect and visceral state (Porges, 2001). According to

polyvagal theory (Porges, 1995), the mammalian vagus nerve contains two branches that

developed evolutionarily. Each branch is associated with the parasympathetic modulation of distinct adaptive physiological and behavioral responses to stress. The first branch, often termed the "vegetative" vagus, is an unmeyelinated pathway that is shared with primitive vertebrates. This branch originates in the dorsal motor nucleus and is responsible for immobilization behaviors, which include feigning death or freezing behaviors (Porges, 2007). These behaviors function to conserve metabolic energy in response to threat cues (Beauchaine, Gatzke-Kopp, & Mead, 2007).

The "smart" vagus, on the other hand, is a myelinated branch unique to most mammals that originates in the nucleus ambiguus. This evolutionarily newer pathway is responsible for the transitory inhibition and disinhibition of cardiac output in response to environmental challenges (Porges, 2001). When an individual is at rest (i.e., not in contact with stressful stimuli), the application of this parasympathetic *vagal brake* slows heart rate, lowers blood pressure, and attenuates the effect of sympathetic nervous system (SNS) activity on the heart. Thus, high vagal control over the heart facilitates adaptive flexibility and responsiveness to one's environment through the promotion of social engagement and calming or self-soothing behaviors (Porges, 2001).

In contrast, when an individual encounters a stressor, the vagal brake is withdrawn in order to initiate fight-flight behaviors. This decreased CVC (also termed vagal *withdrawal*) facilitates increased SNS arousal and concomitant heart rate acceleration and greater attentional vigilance that efficiently enable the individual to better meet environmental demands (Beauchaine et al., 2007). It has also been hypothesized that vagal withdrawal supports adaptive active coping responses such as problem-solving or reappraisal (via increased engagement with the environment) that could buffer the effects of the stressor on future maladjustment. In

contrast, less vagal withdrawal within the context of stress exposure could indicate passive or disengaged coping (El-Sheikh, Keiley, Erath, & Dyer, 2012). After the acute stressor has abated, however, CVC should be reinstated, allowing attentional resources to be shifted away from the stressor and any related negative stimuli.

Vagal tone may serve as an objective measure of individual differences in emotion regulation. High baseline CVC indicates good autonomic flexibility and preparedness for responding to challenges in a context-appropriate manner. The ability to withdraw the brake as needed under conditions of stress and then recover to baseline levels also suggests the presence of good self-regulatory capacity (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). In contrast, an individual with low baseline vagal control may have a lower threshold for becoming sympathetically aroused and may thus possess a liability for directing their attention towards negative stimuli and affect more easily. Moreover, vagal withdrawal to stress with poor subsequent recovery to baseline may indicate poor attentional, emotional, and behavioral control and flexibility (Rottenberg, 2007; Thayer & Lane, 2000). Poor emotion regulation, prolonged difficulty disengaging attention from negative cues, and an inflexible style of responding to environmental challenges are all associated with depression (Joormann & D'Avanzato, 2010; Rottenberg, 2005). Thus, an individual who exhibits low baseline CVC and reactivity to stress accompanied by poorer recovery to baseline is hypothesized to possess greater vulnerability to depression than an individual who displays a more adaptive pattern of physiological responding to stress.

Vagal tone is most commonly indexed by RSA. RSA is the natural rhythmic variability in heart rate that occurs with each respiration cycle and is the product of vagal nerve modulation that occurs at the cardiac sinoatrial node (often termed the *pacemaker*), which is located in the

right atrium of the heart (Porges, 2007). During inhalation, efferent signals traveling from the vagus nerve to the heart decrease, thereby accelerating heart rate. Efferent signaling increases during exhalation, which decelerates heart rate. RSA cannot be assessed directly; rather, it is represented by analyzing the variability in an individual's interbeat intervals (IBIs) in an electrocardiogram. An IBI is defined as the time interval derived from the peak amplitude of an R wave in a given QRS complex to the next R peak. Only high frequency (HF) heart rate variability (HRV) data is used to estimate RSA, as signals between 0.15 and 0.5 Hz are considered to reflect the parasympathetic influence of vagal innervation of the sinoatrial node of the heart (Berntson et al., 1997). In contrast, frequencies below 0.15 Hz suggest both sympathetic and parasympathetic contributions to heart rate. Greater variability in HF HRV is indicative of greater CVC, while less variability suggests reduced CVC.

RSA may be measured in terms of its time course. Baseline RSA is described as a trait index of CVC when the individual is at rest. In contrast, RSA reactivity is a measure of CVC under conditions of stress and is most commonly calculated by subtracting baseline RSA from RSA recorded during the stressor or task period. Lastly, RSA recovery is operationalized as a measure of CVC immediately following removal of the acute stressor (Gentzler, Santucci, Kovacs, & Fox, 2009). Full recovery from the stressor may be indicated by a return to baseline from RSA reactivity observed during the stressor period; thus, RSA recovery is most often modeled as a recovery minus baseline change score. Such change scores may be constrained, however, by an individual's baseline RSA level and may be more appropriately considered as change during the reactivity to recovery periods.

Baseline RSA as a trait vulnerability to depressive symptoms. The majority of studies examining baseline RSA and depression have utilized concurrent designs or have compared

depressed and nondepressed samples. These studies have largely found small-to-moderate associations between depression and lower baseline RSA (for a review, see Rottenberg, 2007). Only a handful of studies have examined the association between baseline RSA and the development of depression prospectively; thus, it is unclear whether baseline RSA functions as a salient vulnerability.

High baseline RSA has demonstrated protective effects against children's internalizing symptoms associated with marital conflict (El-Sheikh, Harger, & Whitson, 2001). However, other studies have failed to find a relationship between baseline RSA and depression (Bosch, Riese, Ormel, Verhulst, & Oldehinkel, 2009; Gentzler et al., 2009; El-Sheikh et al., 2012). For example, in a longitudinal study of 1,653 early adolescents, baseline RSA did not prospectively predict depressive symptoms either alone or in interaction with stressful life events, though there was a surprising trend for high baseline RSA levels to be associated with future symptoms (Bosch et al., 2009). In a recent study, El-Sheikh and colleagues (2012) reported no association between RSA and trajectories of depressive symptoms among pre-adolescents ages 8-10. However, among girls with a high exposure to marital conflict, low baseline RSA in interaction with low baseline SNS activity (measured by electrodermal responding) was associated with high-stable symptom trajectories. This finding warrants clarification, as attenuated SNS responding has also shown strong associations with behavioral disinhibition and externalizing psychopathology (Beauchaine, 2001).

Few studies have examined prospective baseline RSA-depression associations among older adolescents or adults. Using a clinical adult sample, Rottenberg, Wilhelm, Gross, and Gotlib (2002) found that high baseline RSA predicted worsening depression at a six-month follow up, even after controlling for psychiatric medication use and initial symptom severity. In

another study, however, Rottenberg, Chambers, Allen, and Manber (2007) found no association between baseline RSA and symptom severity among a sample diagnosed with MDD at 8-week and 16-week follow ups. Given the mixed findings with regard to baseline RSA, it is possible that examining change in state indices such as RSA reactivity may better elucidate the relationship between CVC and depression. Prior research demonstrates that baseline RSA and RSA reactivity are only partially correlated ($r = .41$; Movius & Allen, 2005) and may thus represent distinct constructs. Indeed, CVC's inherent theoretical association with emotion regulation within the context of stress exposure would suggest that RSA reactivity may function as a better biomarker of stress responding than baseline RSA.

RSA reactivity as a state vulnerability to depressive symptoms. A growing number of recent studies have demonstrated prospective associations between RSA reactivity and depression. Attenuated RSA reactivity to a sad film has been shown to prospectively predict clinician-rated depressive symptoms among children and pre-adolescents, even after controlling for baseline internalizing symptoms (Gentzler, Santucci, Kovacs, & Fox, 2009). In the study described above by El-Sheikh and colleagues (2012), higher RSA and low skin conductance level during a problem solving task were jointly associated with the highest levels of depressive symptoms one year later, but again, this relationship only held for girls who were exposed to high levels of stress. In a separate study, Hinnant and El-Sheikh (2009) found that although RSA reactivity to a social stressor was not independently associated with future internalizing symptoms among children, it was a significant predictor when examined in interaction with baseline RSA.

Only a few studies have investigated the prospective relationship between RSA reactivity and depression among adults; however, these studies have only examined recovery from

depression and not onset. For example, Rottenberg and colleagues (2005) found individuals with depression who showed greater RSA withdrawal to a sad film were more likely to fully recover from their symptoms six months later, even after taking initial symptom severity into account. Importantly, the authors showed that a lack of RSA withdrawal among nonrecovered individuals during the mood induction was not accounted for by lower baseline RSA. Given the mixed findings with regard to RSA reactivity and depression, and particularly the limited data on predictive relationships, further research is warranted to clarify what patterns of physiological responding, if any, are associated with depressive symptoms.

Biological recovery from stress may predict depressive symptoms. One potential reason studies examining the relationship between RSA and depression have yielded inconsistent results may be the lack of consideration of RSA *recovery*. As RSA reactivity indexes flexible responding and engagement with environmental demands (Porges, 1995) as well as attentional deployment (Mulder & Mulder, 1981), it is reasonable to infer that some degree of RSA withdrawal in response to stress will be observed among most individuals. However, during the recovery period after the stressor has abated, we should expect that among non-depressed or non-vulnerable individuals, engagement with stressful stimuli will decrease and that attentional resources will be redirected elsewhere. During this period, one should be able to simultaneously observe increases in RSA that represent a reengagement of the vagal brake. In contrast, RSA should theoretically remain in a state of withdrawal among individuals who continue to allocate attentional resources toward the stressful stimulus or memory of the stimulus, Supporting this hypothesis, Santucci and colleagues (2008) found that young children ages 4-7 who exhibited poor RSA recovery following a delay-of gratification task were more likely to display sadness

and remain focused on the desired object. These children were also less likely to use adaptive regulation strategies and distraction following the task.

As sustained attention towards negative stimuli may indicate vulnerability to depression (Joormann & D'Avanzato, 2010), it is possible poor RSA recovery from stress may serve as a stronger biomarker for depressogenic risk than RSA reactivity. Despite this theoretical link, surprisingly few studies have examined RSA recovery in relation to depression. Rottenberg and colleagues (Rottenberg, Clift, Bolden, & Salomon, 2007; Rottenberg, Wilhelm, Gross, & Gotlib, 2003) found that compared with nondepressed groups, individuals diagnosed with MDD failed to exhibit increases in RSA following sad film and stress inductions. These studies support the suggestion that depression is characterized by deficits in emotion regulation that would facilitate recovery from stress and negative affect. However, it is difficult to know from these findings whether the RSA biomarker for such deficits is a concomitant of depression, or whether it can be detected prior to depression onset. Gentzler and colleagues (2009) found poor RSA recovery one minute following a sad film viewing was marginally correlated with future depressive symptoms. Thus, it is possible that RSA recovery is likely linked with depression; however, further examination of prospective relationships is clearly indicated.

Cognitive Vulnerability to Depression

One of the most empirically supported etiological theories of adolescent depression is the cognitive vulnerability-stress model, which suggests that certain cognitive responses to stress may confer risk for the development of depression. While original generic cognitive vulnerability-stress models emphasized the role of maladaptive cognitive *content* in the development of depression (e.g., dysfunctional attitudes [Beck, 1987] and negative inferences [Abramson et al., 1989]), more recent discussions have also stressed the importance of cognitive

processes, or the ways in which individuals think about events and stimuli. One such well-established cognitive process is rumination. In the sections that follow, I define rumination discuss relevant theory and research relating rumination to depression.

Definition and theoretical foundations. Rumination was originally described by Nolen-Hoeksema (1991) as a pattern of repetitive self-focus on one's depressive symptoms as well as the potential causes and consequences of those symptoms. Nolen-Hoeksema (1991) proposed that individuals who ruminate in response to transient negative affect will exacerbate and prolong negative mood states, which may in turn lead to the onset of a depressive episode or contribute to a longer, more severe symptom course.

While the basic premise of Nolen-Hoeksema's original theory still holds, the definition of rumination has been expanded to not only include focus on depressive symptoms and affect, but to also explicitly include perseverative attention to negative thoughts and negative life events (Mezulis et al., 2002; Robinson & Alloy, 2003). This type of rumination has frequently been termed *stress-reactive rumination*, while rumination about negative mood or affect is referred to as *depressive rumination*.

Increasingly, rumination has been characterized as a product of maladaptive information processing, specifically impaired control over the ability to disengage attention from negative stimuli. When individuals encounter stress or negative emotional states, some degree of self-referent information processing is normative. However, when negative self-focus is prolonged, it may interfere with attentional allocation towards behaviors that may facilitate mood repair, such as problem solving (Lyubomirsky & Nolen-Hoeksema, 1995) or cognitive reappraisal (Joormann & D'Avantzato, 2010). Indeed, rumination is associated with a number of information processing biases (see Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008 for a review)

and may thus reflect individual differences in the ability to disengage and switch attention away from negative self-referential stimuli (Koster, De Lissnyder, Derakshan, & De Raedt, 2010).

Trait rumination as a vulnerability to depressive symptoms. Rumination is most frequently measured at the trait or dispositional level. Trait (as opposed to state) rumination can be conceptualized as the overarching tendency or propensity to ruminate in response to negative internal or external stimuli. Trait rumination demonstrates strong associations with both the onset and course of depressive disorders and symptoms. Using a subsample from the Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project, Spasojević and Alloy (2001) followed 137 initially nondepressed college students for 2.5 years and found trait rumination was related to the number of prospective MDEs experienced over the course of the study. Further, trait rumination mediated the relationship between depressogenic vulnerabilities (past MDE history, negative cognitive style, self-criticism, and neediness) and the development of new MDEs, suggesting that trait rumination functions as a proximal mechanism through which other factors influence vulnerability to depression. A number of other studies have also shown a predictive relationship between trait rumination and the onset, symptom severity, and duration of clinically significant depression (Just & Alloy, 1997; Nolen-Hoeksema, 2000; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Moreover, trait rumination is prospectively associated with increases in depressive symptoms among both adolescents (Cox, Funasaki, Smith, & Mezulis, 2011; Mezulis, Priess, et al., 2011; Mezulis, Simonson, McCauley, & Vander Stoep, 2011; Skitch & Abela, 2008; Verstraeten et al., 2009) and college students (Sarin, Abela, & Auerbach, 2005).

State rumination as a vulnerability to depressive symptoms. An individual's tendency to exhibit ruminative responses to stress or depressed mood is generally considered to

be a stable characteristic (Nolen-Hoeksema, 1991; Smith & Alloy, 2009); thus the majority of

prior investigations have relied upon the use of trait measures of depressive or stress-reactive

rumination. Rumination, however, may be akin to other depressogenic vulnerabilities (e.g.,

affective vulnerability) in that despite an individual's trait tendency to ruminate, we should

expect to observe some degree of variability in responses over time or across contexts.

Consistent with this suggestion, a small but growing number of studies have utilized measures of

state rumination. These indices are intended to capture momentary, event-specific rumination

(e.g., ruminating about one's failing grade on an exam the previous day or ruminating about a

specific argument with a friend). Studies utilizing these measures have found that event-specific

or state rumination does indeed fluctuate, depending on the personal salience of the stressor

(Lavallee & Campbell, 1995) and the concurrent presence of state negative affect (Moberly &

Watkins, 2008).

To date, only one known study has examined state rumination's association with

depression. Mezulis and Rudolph (2012) found across an eight-week diary study that

adolescents' state rumination about their two most salient self-selected stressors in a given week

predicted fluctuations in depressive symptoms the following week, even after controlling for trait

rumination and concurrent depressive symptoms. These results suggest that variations in state

rumination may predict depressive symptoms above and beyond the trait tendency to ruminate.

Cognitive recovery from stress may predict depressive symptoms. Rumination is by

definition characterized by persistent negative self-focused attention as well as attention towards

negative events. However, laboratory paradigms assessing state rumination typically measure

the construct immediately following the stress or mood induction and leave little room for the

trajectory of ruminative thought processes to unfold. It is possible an individual may initially

direct their attention towards the negative event and its implications for the self, but may later use adaptive or maladaptive regulatory strategies to modulate their initial level of negative self-focus. Initial negative self-focus or attention towards the stressor may not be inherently maladaptive if the individual is able to later redirect their attention. It is possible initial ruminative responses may represent attempts to identify and resolve discrepancies between one's current and desired state (Pyszczynski & Greenberg, 1987). This self-regulatory process is shared across disordered and nondisordered individuals and is hypothesized to only become maladaptive when the individual is unable to escape from the self-focusing cycle.

The inability to disengage negative attention towards the self and related stimuli may indicate a failure of adaptive self-regulation. This persistent rumination in turn leads to the exacerbation and sustenance of the negative thoughts and affect that characterize depression. Thus, it should stand to reason that a delayed assessment of rumination following stress would be a more powerful predictor of depressogenic outcomes than more immediate assessments. Only a handful of studies have considered the time course or trajectory of ruminative thinking (e.g., Grant & Beck, 2010); however, no known prior published investigations have examined recovery from rumination in the prediction of depression. Our own research using prospective diary designs among adolescents suggests this may be a potential avenue for further exploration (Rudolph & Mezulis, in preparation). Utilizing multilevel modeling, we found that greater event-specific rumination about adolescents' self-identified stressors one week after the events occurred predicted greater depressive symptoms two weeks after the stressor. These effects held even after controlling for trait rumination, initial event-specific rumination about the stressor, and concurrent depressive symptoms (coefficient = .65, t = 3.17, p = .002).

Time Course of Stress Responding May Influence Risk for Depression

In examining state or proximal vulnerability to depression, most prior laboratory studies have only considered indices of initial reactivity, typically assessed during or immediately following a stressor task. However, such views of state vulnerability may be incomplete. An individual's ability to recover from a given stressor may be just as critical in predicting depression as their initial level of reactivity. This hypothesis is drawn from theories of emotion processing that differentiate between emotional reactivity[1] and emotion regulation (Koole, 2009). Emotional reactivity is considered the primary response to an emotion-eliciting situation and is influenced not only by the characteristics of the stressful stimulus, but also by individual differences such as trait depressogenic vulnerabilities (discussed in the preceding sections). An individual's secondary response constitutes their ability to recover or return to baseline. The quality of a secondary response varies as a function of online emotion regulation processes (Koole, 2009). These responses can involve up-regulation (in which the individual heightens their response to the stressor) or down-regulation (the dampening of the response).   Further, an individual may simply maintain the magnitude of the initial response by failing to down-regulate while at the same time abstaining from up-regulation.

Extending this reactivity-recovery distinction across domains, persistently elevated state affective, biological, and cognitive responses following stress may indicate continued vulnerability to maladaptive outcomes. Stressful life events inherently evoke some degree of negative affect, prompt individuals to engage their physiological response systems, and promote some degree of self-referent processing. Thus, greater emotional reactivity, RSA withdrawal, and rumination during immediate stress exposure may to some degree be considered normative. In contrast, prolonged responding after the stressor has abated may suggest the individual has

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[1] Emotional reactivity is also commonly termed *emotional sensitivity* in the emotion processing literature.

failed to implement successful regulation strategies. The persistence of maladaptive responses may comprise the "final" proximal reaction to stress and may thus represent the most salient form of vulnerability to depression.

The Current Study

Extant research has clearly established prospective links among trait NA, trait rumination, and depressive symptoms. Although less consistent, theory and a small body of emerging evidence also suggests low resting RSA may function as vulnerability factor. Further, studies consistently demonstrate that stress exposure moderates the prospective relationship between trait vulnerabilities and depressive symptoms (e.g., Charbonneau, Mezulis, & Hyde, 2009; Morrison & O'Connor, 2008). Surprisingly little research has examined how trait vulnerabilities may manifest themselves on a state level to predict subsequent onset of symptoms. It is possible that the ways in which individuals respond to and recover from stress may better predict depressogenic outcomes than trait measures that assess an individual's perception of how they typically respond to hypothetical stressful situations. In line with the vulnerability-stress perspective, maladaptive trait patterns of stress responding are thought to only emerge under conditions of stress (Scher et al., 2005). Thus, it is necessary to not only examine how state vulnerabilities are activated within the context of a controlled laboratory stress paradigm, but to also establish how these event-specific factors interact with life stress to predict future depression. These questions represent an important and novel contribution toward understanding individual differences in proximal depressogenic vulnerability.

The current study aimed to provide a rigorous test of the vulnerability-stress model by examining whether patterns of affective, biological, and cognitive responses to induced stress prospectively predicted depressive symptoms eight weeks later in sample of young adults. I

specifically expected that participants' recovery from the laboratory stressor would be the strongest predictor of future depressive symptoms. My hypotheses were as follows:

1) Initial reactivity to the stress induction would prospectively predict depressive symptoms eight weeks later. I expected these effects would hold above and beyond the contribution of trait vulnerabilities (H_1). See Figure 1 below. Specifically, I hypothesized that:

   a. Controlling for trait emotional reactivity, higher state emotional reactivity immediately following the stressor would predict greater symptoms (H_{1a}).

   b. Controlling for baseline RSA, blunted RSA withdrawal during the stressor would predict greater symptoms (H_{1b}).

   c. Controlling for trait rumination, greater event-specific rumination immediately following the stressor would greater predict symptoms (H_{1c}).

2) The relationship between domains of initial stress reactivity and depressive symptoms would be moderated by life stress exposure over the eight-week study period ($H_{2a, b, c}$). I specifically hypothesized that as stress exposure increased, the magnitude of the positive relationship between stress reactivity and depressive symptoms would increase. See Figure 2 below.

3) Prolonged reactivity from the stress induction would predict subsequent depressive symptoms eight weeks later. I expected these effects would hold above and beyond the contribution of trait vulnerabilities and initial levels of reactivity (H_3). See Figure 3 below. Specifically, I hypothesized that:

a. Controlling for trait emotional reactivity and state emotional reactivity immediately following the stressor, higher state emotional reactivity during the recovery period would predict symptoms (H_{3a}).

b. Controlling for baseline RSA and RSA reactivity, poorer RSA recovery from the stressor would predict greater symptoms (H_{3b}).

c. Controlling for trait rumination and event-specific rumination immediately following the stressor, more rumination about the stressor during the recovery period would predict greater symptoms (H_{3c}).

4) The relationship between domains of stress recovery and depressive symptoms would be moderated by stress exposure over the eight-week study period ($H_{4a, b, c}$). I specifically hypothesized that as stress exposure increased, the magnitude of the positive relationship between stress recovery and depressive symptoms would increase. See Figure 4 below.
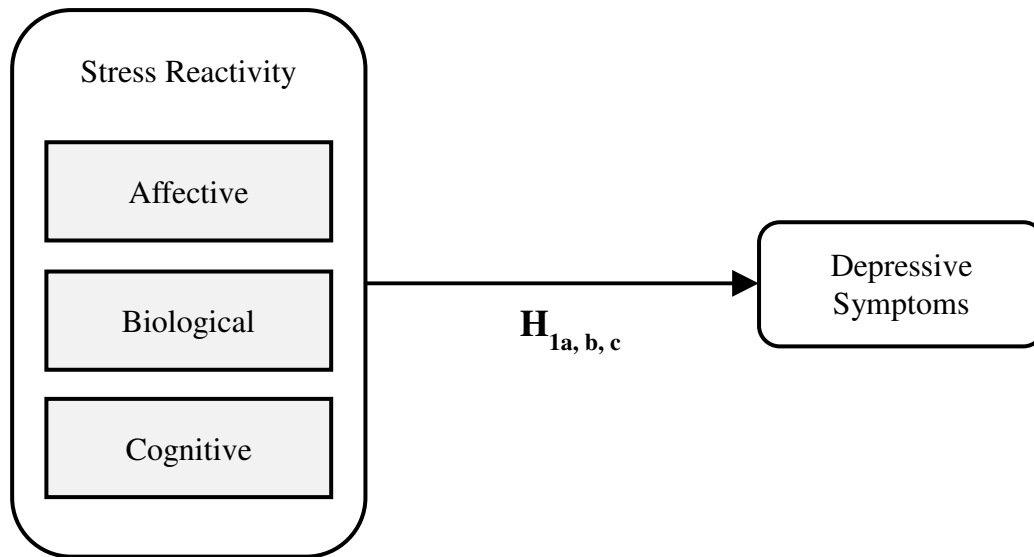
Figure 1. Conceptual model of hypothesis 1. Domains of stress reactivity will prospectively predict depressive symptoms.
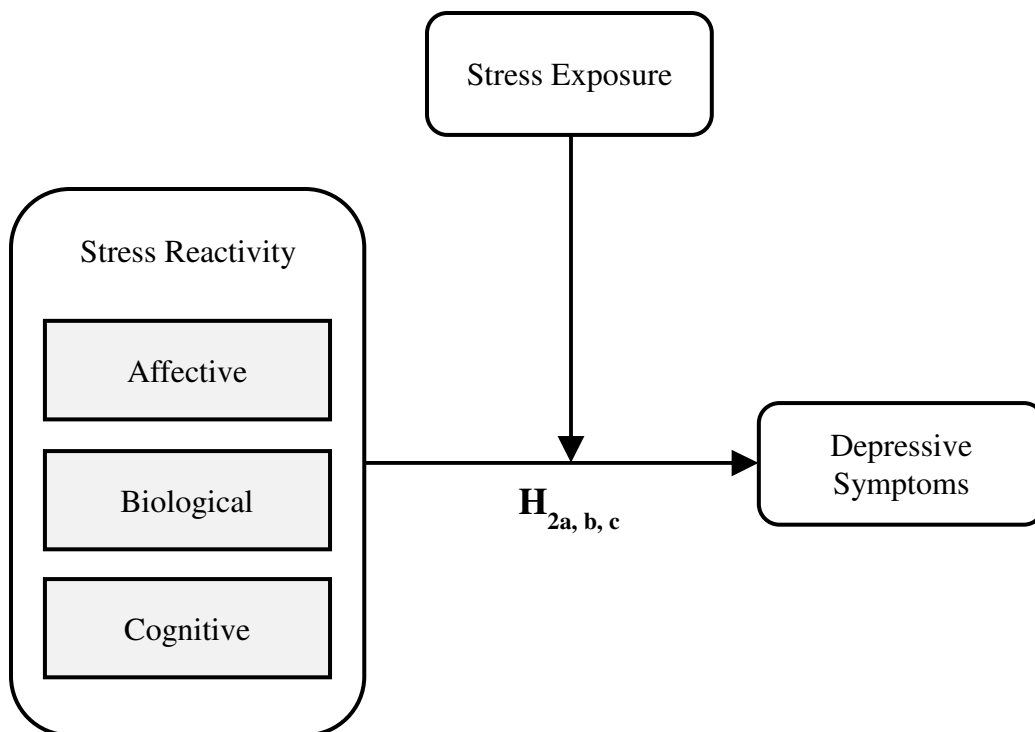
Figure 2. Conceptual model of hypothesis 2. Stress exposure will moderate the prospective relationship between domains of stress reactivity and depressive symptoms.
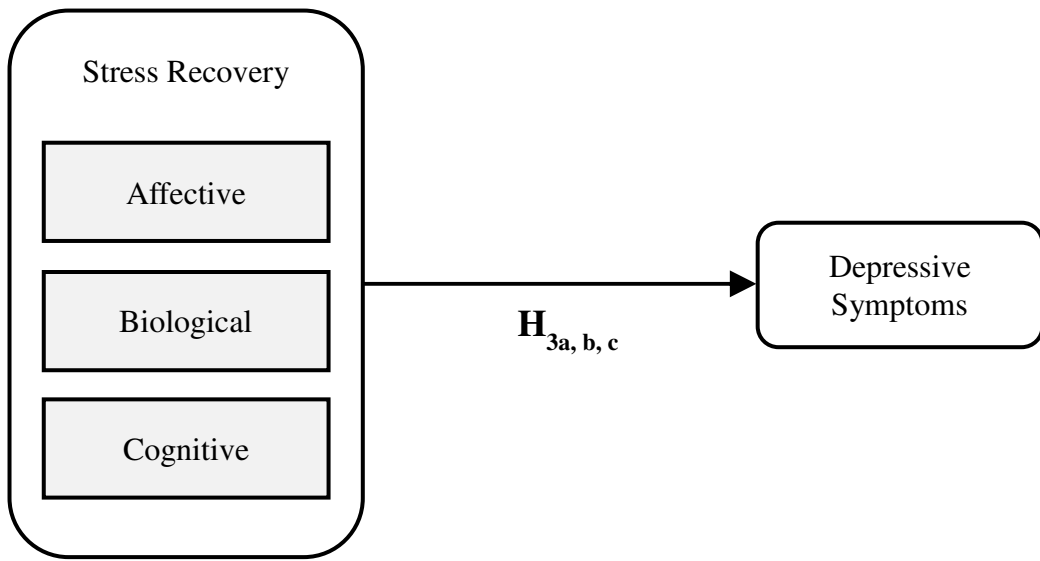
Figure 3. Conceptual model of hypothesis 3. Domains of stress recovery will prospectively predict depressive symptoms.
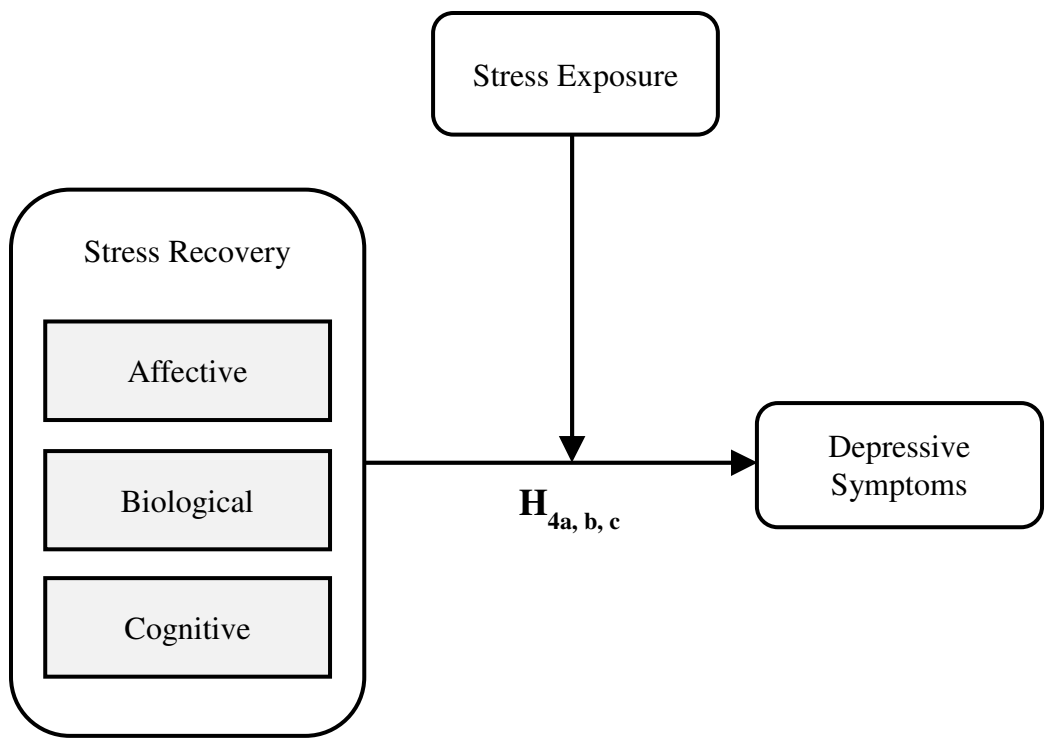
Figure 4. Conceptual model of hypothesis 4. Stress exposure will moderate the prospective relationship between domains of stress recovery and depressive symptoms.

CHAPTER II

Method

Recruitment

Participants ages 18-24 were recruited from Seattle Pacific University (SPU), an urban liberal arts university in the Pacific Northwest. The minimum age of 18 was selected so participants could provide informed consent for participation without the need for parental consent. The upper age cutoff of 24 was selected to ensure the sample was representative of an older adolescent/young adult population.

All study procedures and materials were approved by the SPU Institutional Review Board. Participants were recruited through oral presentations made to undergraduate psychology classes. Students who indicated interest in participating were asked to provide their email address at the conclusion of the presentation. The principle investigator then sent a recruitment email to interested students containing a link to the study's online survey platform. Potential participants were asked to read the online consent and indicate whether they met the study's age criteria and wished to participate. Students who consented were then directed to complete the Time 1 – Part A ($T1_a$) questionnaires online. Those who did not meet the age criteria or who decided not to participate were thanked and given the opportunity to exit the website. Participants who expressed initial interest by providing their email address, but who did not electronically consent or decline consent within five days were sent a reminder email.

Procedure

The current study was comprised of two timepoints (see Figure 5 below for a graphic representation of the study timeline and measures). $T1_a$ was completed online immediately after participants provided consent and contained a set of demographic questionnaires as well as self-

report measures assessing current depressive symptoms, trait emotional reactivity, and trait rumination. All online data was transferred to a secure database accessible only by the principal investigator and faculty sponsor/co-investigators.

After completing $T1_a$, the principal investigator contacted participants to schedule Time 1 – Part B ($T1_b$), which consisted of an on-campus laboratory visit. Every effort was made to schedule the visit within one month of $T1_a$ completion. All $T1_b$ stimuli and self-report measures were administered on a 17-inch Dell computer monitor using MediaLab v.2012 and DirectRT v.2012 software (Empirisoft Corporation, New York, NY). Figure 6 provides a visual outline and time course of $T1_b$ procedures.

After completing a hard copy consent form and additional assessments not included in the current analyses, participants completed a brief self-report questionnaire that assessed the use ("yes" or "no") of antidepressants, stimulants, and antihistamines over the previous 24 hours, as such agents are known to have effects on cardiac functioning (Salomon, Clift, Karlsdóttir, & Rottenberg, 2009). Participants were asked to apply two electrocardiographic (ECG) electrodes to their torso in a Lead II configuration with instruction from the experimenter. Next, the participant completed a 5-minute baseline physiological recording period in during which they were instructed to view a series of neutral nature scenes on the computer while breathing at their normal rate. The ECG response signals obtained during the final two minutes of the baseline recording period were used to quantify baseline RSA. Immediately following nature scene offset, participants were prompted to complete a rating of current affect.

Stress induction. Next, participants were presented with a 7-minute stressor task[2] during which they completed a modified computer version of the Paced Auditory Serial Addition Task (PASAT-C; Lejuez, Kahler, & Brown, 2003) and received false negative feedback about their performance on the test. The PASAT was originally developed as a neuropsychological test designed to assess information processing capacity in individuals with head injuries (Gronwall, 1977); however, it has been adapted in recent years for use in laboratory stress inductions. During the test, participants were presented with sets of pre-recorded aural numeric stimuli ranging from one to nine. Participants were instructed to sum each newly presented stimulus with the number they heard immediately prior to it (e.g., 7 +2 [correct response = 9] +3 [correct response = 5] + 7 [correct response = 12]). Therefore, the task required participants to actively inhibit encoding of their previous response to facilitate continuous summing with the next digit presented. Participants were informed that incorrect or skipped items would count against their total score.

To avoid vocalization confounds during psychophysiological recording, participants indicated their responses during the PASAT-C by clicking numbers on a keyboard analogue on the computer screen. Three sets of stimuli containing 60 numbers each were presented at varying latencies. In the first set, numbers were spaced three seconds apart. The speed increased

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[2] It should be noted that I originally proposed using an impossible anagram task as the stressor paradigm. However, during pilot testing, multiple participants reported during a fill-in-the-blank measure of negative inferences regarding performance that the paradigm was rigged. Additionally, during debriefing procedures, many participants articulated suspicion of the task, calling into question whether the paradigm lacked face validity. While prior studies have successfully utilized impossible anagrams as a means of inducing stress, I hypothesized that my use of filler tasks in the current study gave participants multiple opportunities to reappraise performance. As anagrams are likely familiar to most college students, participants may have begun the task believing they *should* be able to solve the items and were surprised when they were unable to. This discrepancy between their pre-task belief and performance may have become more pronounced during the recovery period, prompting some participants to conclude that their difficulty with the task must be due to experimenter deception. Therefore, I elected to change the stressor to the PASAT-C. All participant data reported in this manuscript were collected utilizing the PASAT-C paradigm.

in subsequent sets such that the second and third sets were presented at a latency of 1.5 seconds and one second, respectively. Two 30-second pause periods were used to separate the sets.

Prior to beginning the PASAT-C, participants were told the test was a measure of intelligence and cognitive processing ability that had previously been shown to predict success in college and in the workplace. They were also informed they would receive their score on the computer screen after the test was completed. To increase performance pressure, the experimenter also told participants she would remain in the room during the test and feedback period so she could record the score for data collection purposes. The experimenter positioned herself diagonally behind the participant for the duration of the test. The experimenter's presence was intended to add a social stress element to an otherwise achievement-oriented task, as researchers have suggested that laboratory stressors comprised of social challenges may provide greater semblance to genuine stressors than nonsocial tasks (Steptoe, 1985).

Following completion of the PASAT-C, participants were provided with standardized negative performance feedback on the computer monitor indicating that they got 64%, or 116 out of a possible 180 items correct. This feedback was presented for 30 seconds and was given regardless of actual performance on the test. During this time, the experimenter leaned forward and recorded the score on a clipboard. ECG was recorded continuously during the PASAT-C and feedback presentation and was used to quantify RSA reactivity.

The PASAT-C and negative performance feedback jointly comprised the stress induction procedure. Prior studies have used mood inductions (e.g., watching sad films) to elicit physiological arousal as well as emotional and cognitive reactivity; however, a mood induction design does not provide specific, self-referential information from which the participant can draw inferences about themselves. In contrast, an induced stress paradigm provides an in-vivo

failure experience that may more closely represent a stressor encountered in everyday life. While the stress induction method involves the use of participant deception, I elected to utilize this paradigm as a more ecologically valid means of eliciting stress responses, rather than a negative mood state. The PASAT has been shown to elicit changes in heart rate and electrodermal arousal (Holdwick & Wingenfeld, 1999; Lejunez et al., 2003) as well as distress in young adult samples (Feldner, Leen-Feldner, Zvolensky, & Lejuez, 2006; Simonson et al., 2011). This distress is typically mitigated by the use of debriefing procedures outlined below.

Physiological recovery and self-reported reactivity. Following feedback offset, the participant viewed a screen asking them to wait quietly for several minutes while the next experiment tasks loaded on the computer. During this period, ECG signals were recorded for 90 seconds, which served as the RSA recovery period. Next, participants completed self-report measures of current affect and event-specific rumination about their performance on the PASAT-C (termed hereafter referred to as *task* measurements).

Filler tasks. Following completion of the self-report measures at the task administration, participants completed two filler tasks lasting a total of 18 minutes. The purpose of the filler tasks was to provide a time buffer between the task and recovery periods for the self-report measures. The first filler task was comprised of a computerized trial-by-trial exogenous cueing task (Posner, 1990). This task consisted of an initial 500 ms presentation of a fixation cross positioned between two rectangles on the computer screen. A word cue then appeared in one of the two rectangles and remained onscreen for 1500 ms. Word cues were comprised of 30 negative and 30 neutral words matched for word length and selected from Donaldson, Lam, and Mathews (2007). Fifty ms following cue offset, a target asterisk probe was randomly presented in one of the rectangles. Participants were instructed to press a key indicating in which rectangle

the target was located. This paradigm consisted of 150 total trials that included two repetitions of each word type set (randomized across 120 trials) and 30 uncued trials. Although the exogenous cueing task is most commonly used as a measure of attentional bias, prior studies have also utilized similar attention tasks as time fillers in laboratory paradigms (Beevers & Carver, 2003; Jamieson & Harkins, 2011).

The second filler task consisted of a computerized guided thought sampling and free-write task. The thought sampling was comprised of 20 sentence stems randomly drawn from the Rotter Incomplete Sentence Blanks-Second Edition (RISB-2; Rotter, Lah, & Rafferty, 1992). Examples of stems included, "The best time is…" and "I regret…" Participants were instructed to use the stems to form complete sentences by typing the first words that came to mind after reading each item. Once participants completed the selected items from the RISB-2, they were presented with a blank text box on the computer screen and prompted to type anything that was on their mind. Participants were asked to write continuously until they received instructions on the screen to stop.   The entire guided and free-write filler task took 10 minutes. Variability in the amount of time participants used to complete the RISB-2 was accounted for by MediaLab software timing capabilities such that participants who took longer on the sentence stems had a relatively shorter free-write duration. Likewise, participants who finished the RISB-2 in a shorter amount of time spent longer on the free-write.

The exogenous cueing and thought sampling tasks were selected based on their neutral demands on participants' attention. The purpose of a filler task is to satisfy a desired time buffer; thus, I did not want to direct participants' attention to a particular type of stimulus. Instead, my intention was to employ a neutral set of tasks that would allow participants to direct their attention toward stimuli they would typically attend to following a stressor. In other words, I

expected that participants who perseverated on their performance during the stress induction would continue to direct their attention toward negative thoughts and affect during the filler tasks and that these patterns of sustained stress responding would be reflected in my recovery measures.

Self-reported recovery and debriefing.   Following the filler tasks (approximately 22 minutes after receiving the failure feedback), the participant completed a third assessment of current affect and a second assessment of event-specific rumination. These assessments served as measures of self-reported recovery. Following completion of the recovery measures and removal of the ECG electrodes, participants were fully debriefed. A standardized script was used to inform participants the feedback they received did not reflect their actual performance on the PASAT-C, that the test was not predictive of intelligence, cognitive processing ability, or success, and that the experimenter was present to increase pressure. The rationale for the use of deception was explained and participants were given the opportunity to ask questions about the procedure. Course credit was given for $T1_a$ and $T1_b$ participation.

Prospective follow up. Eight weeks after completing the $T1_b$ laboratory visit, the principal investigator sent participants an email inviting them to complete T2. This final timepoint was administered online via an online survey platform and contained measures of life stress exposure and depressive symptoms. Participants received a $10 Amazon.com gift card for completing T2.

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┌─────────────┐        ┌──────────────────┐        ┌─────────────┐
│    T1ₐ      │───────▶│      T1_b        │───────▶│     T2      │
│  (online)   │        │ (laboratory visit)│        │  (online)   │
└─────────────┘        └──────────────────┘        └─────────────┘
```

T1_a *(online)*

Measures:
- Depressive Symptoms
- Trait Rumination
- Trait Emotional Reactivity

T1_b *(laboratory visit)*

Baseline Measures
- State NA
- RSA

Task Measures
- State NA
- RSA
- State Rumination

Recovery Measures
- State NA
- RSA
- State Rumination

T2 *(online)*

Measures:
- Stress Exposure
- Depressive Symptoms
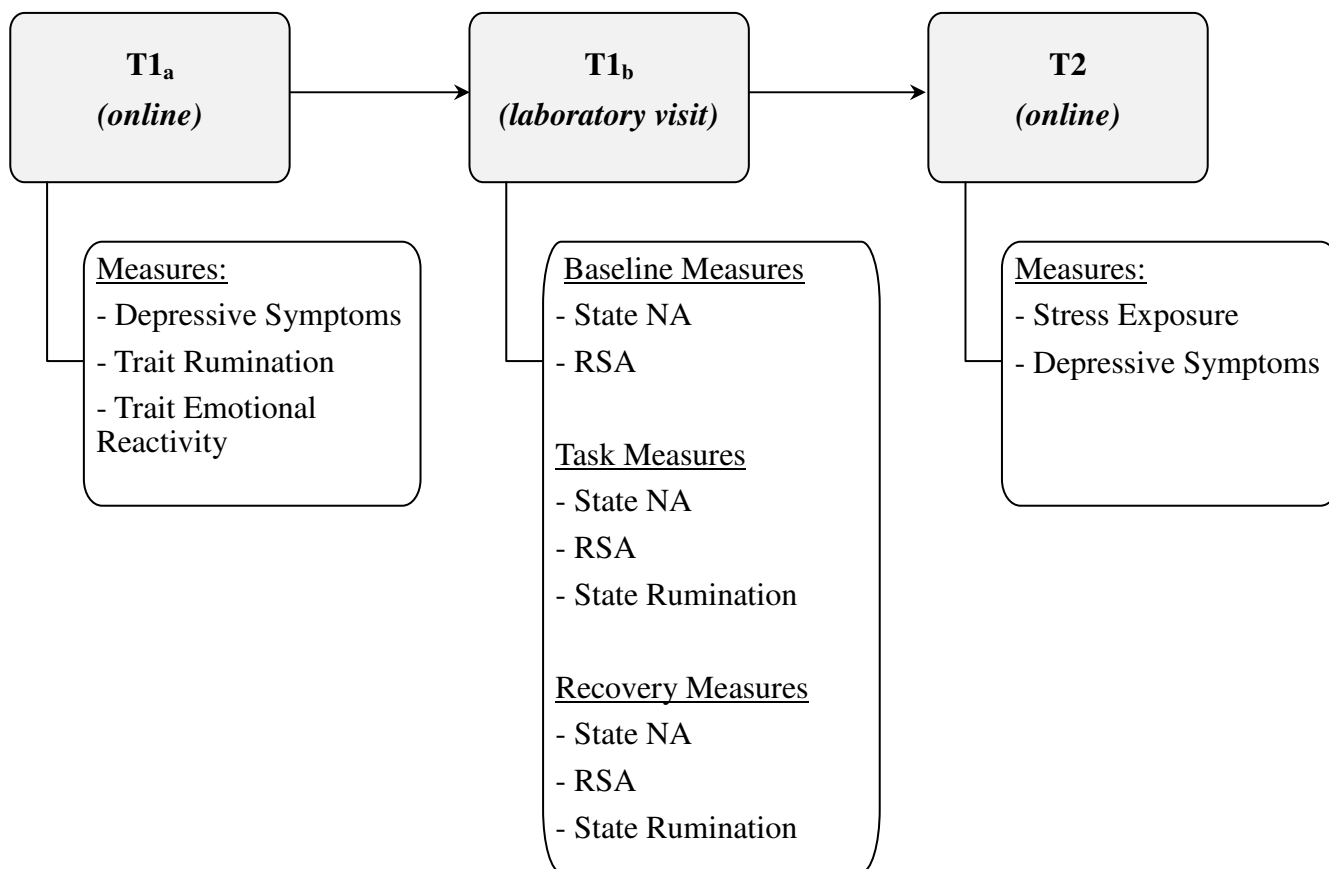
Figure 5. Study timeline and measures.
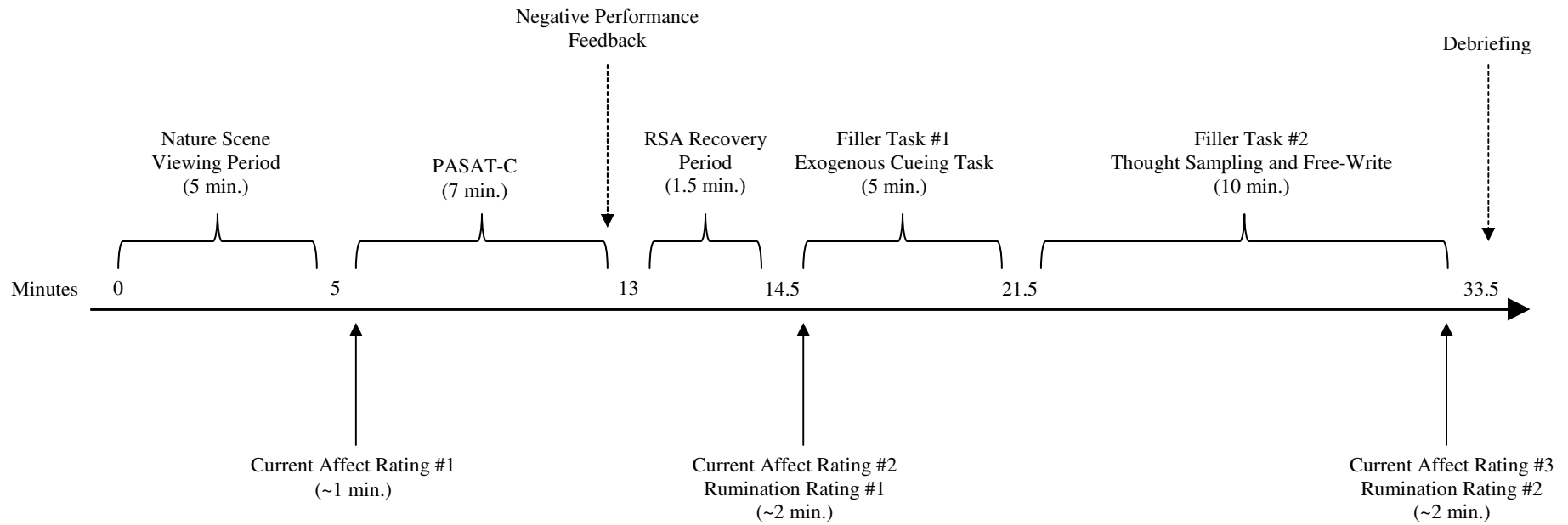
Figure 6. Time sequence of the T1ᵦ laboratory visit.

Measures

   Trait emotional reactivity. Trait emotional reactivity was measured at $T1_a$ with the

Emotion Reactivity Scale (ERS; Nock, Wedig, Holmberg, & Hooley, 2008). The ERS is a 21-

item self-report questionnaire intended to assess the characteristic frequency, intensity, and

duration of an individual's subjective experience of their negative emotions. Sample items

include: "When I am angry/upset, it takes me much longer than most people to calm down" and

"People tell me that my emotions are often too intense for the situation." Participants were asked

to rate the magnitude with which each item is true of their emotional experience using a 5-point

Likert scale ranging from 0 *(not at all like me)* to 4 *(completely like me).* Total scores range from

zero to 84, with higher scores indicating the presence of higher levels of emotional reactivity. In

the current study, the 21 items on the ERS were used to create a mean composite score.

   Exploratory factor analysis has indicated the presence of a three-factor structure on the

ERS: emotional sensitivity (10 items), emotional arousal/intensity (seven items), and emotional

persistence (four items). However, Nock and colleagues (2008) recommend the ERS total scale

should be used to index emotional reactivity, citing high intercorrelations and high factor

loadings (all greater than .44) in the single factor solution.

   The full ERS demonstrates good psychometric properties. Specifically, the ERS has

shown good convergent validity with measures of similar temperamental constructs, including

trait NA, as well as divergent validity with unrelated temperamental constructs such as

behavioral activation (Nock et al., 2008). Internal consistency for the full scale ranges from .94

(Nock et al., 2008) to .97 (Deckersbach et al., 2011). Coefficient alpha for the current sample

was .91.

Trait rumination. Trait rumination was measured during $T1_a$ using the Perseverative Attention to Negative Events scale (PANE; Mezulis et al., 2002). The PANE is a 45-item self-report assessment of trait rumination in response to negative events in the achievement, interpersonal, and body image/attractiveness domains. Only the achievement and interpersonal scenarios were used in the current study, resulting in a total of 30 items. Participants were given six negative event scenarios (three in each of the event domains) and were prompted to imagine how they would react if the events happened to them. Examples of scenarios include: "You fail an important exam at school" (achievement domain) and "A romantic partner ends an important relationship with you, although you want the relationship to continue" (interpersonal domain). Five items consisting of common ruminative responses to negative events accompanied each scenario. Participants were asked to rate how well each item would characterize their response to the given situation using a 1 (*Very unlike me*) to 5 (*Very like me*) Likert scale. Sample items included, "I'd keep thinking about how down I felt" and, "I'd keep thinking about what I could have done differently." Scores on the two-scale version of the PANE range from 30 to 150, with higher scores indicating greater rumination. In the current study, the 30 items drawn from the achievement and interpersonal domains were averaged to create mean composite scores for each participant. The PANE has demonstrated good internal consistency for the full scale ($\alpha = .96$) as well as the achievement ($\alpha = .92$) and interpersonal ($\alpha = .89$) domains (Mezulis et al., 2002). Coefficient alpha in the current sample was .94.

Depressive symptoms. Depressive symptoms were measured at $T1_a$ and T2 with the Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977). The CES-D is a 20-item measure developed for use with nonclinical adult populations to assess the presence of depressive symptoms over the past week. Sample items include: "I had crying spells" and "I felt

that everything I did was an effort." Participants rated the frequency with which they experienced each symptom during the past week using a 4-point Likert scale ranging from 0 (*rarely or none of the time [less than 1 day]*) to 3 (*most or all of the time [5-7 days]*). Scores were calculated by reverse-scoring positively worded items (e.g., "I felt hopeful about the future"), and then summing the items to produce a composite depressive symptom score. CES-D scores range from zero to 60, with higher scores indicating a greater degree of symptomology. Scores above 16 suggest a significant level of depression (Radloff, 1977).

The CES-D is considered a valid measure of depressive symptoms, with scores correlating moderately with clinician ratings of depression ($r = .53$; Radloff, 1977). Test-retest reliabilities range from $r = .51$ to .67 over 2-8 week periods (Radloff, 1977). These moderate reliabilities reflect the variability symptom measures are expected to capture over time. The CES-D has demonstrated adequate internal consistency in studies utilizing community samples (e.g., $\alpha = .85$; Radloff, 1977). Coefficient alpha for the CES-D among college student samples ranges from .78 to .87 (Radloff, 1991; Verhaeghen, Joormann, & Khan, 2005). For the current study, internal consistencies for $T1_a$ and T2 were .90 and .88, respectively.

State emotional reactivity. State emotional reactivity was measured during $T1_b$ using the Negative Affect scale of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item self-report questionnaire that purports to capture a range of positive and negative affective descriptors such "distressed" or "strong" (Watson et al., 1988). Participants were asked to rate how much their current affect aligned with each descriptor using a Likert scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*). The PANAS was designed for use as either a trait or state measure, which allows for modification of participant instructions to reference their emotions over various timeframes including "the

present moment", "today", "the past few days", "the past few weeks", "the past year", and

"generally or on average" (Watson et al., 1988). As the current study used the PANAS as a

measure of immediate affective distress, the instructions to participants were adapted to read,

"Indicate to what extent you feel this way right now (that is, at the present moment)."

The PANAS is comprised of two 10-item scales: Positive Affect (PA) and Negative

Affect (NA). All items from both scales were administered to participants to mask the function

of the paradigm, which was to induce stress and negative affect. However, only the NA scale

was utilized in the current study. A mean score was generated from these 10 items, with higher

scores indicating the presence of more negative affect at a specific administration (e.g., baseline

or pre-stress, task (immediately post-stress), or recovery (18.5 minutes post-stress).

Previous studies have demonstrated good convergent validity between the PANAS NA

scale and other measures of distress or unpleasant mood states (Watson et al., 1998). Test-retest

reliabilities vary depending on the timeframe referenced in the participants' prompt. As would

be expected, the eight-week test-retest correlation is low when the prompt references current

affect levels (Watson et al., 1988). The NA scale has shown good internal reliability, with

coefficient alphas ranging from .85 to .88 (Crawford & Henry, 2004; Merz & Roesch, 2011;

Watson et al., 1988). Internal reliabilities across all PANAS NA scale administrations in the

current study were as follows: .80 (baseline), .86 (task), and .87 (recovery).

State rumination. State rumination about PASAT-C performance was assessed twice

during $T1_a$ using an event-anchored version of the PANE. As referenced above, the original

PANE provides negative event scenarios and asks participants to self-report on their

endorsement of probable ruminative responses to those events. Rather than inquiring about

responses to hypothetical events, the event-anchored PANE (EA-PANE) references a real event,

such as performance on a test. In the current study, the participant prompt was modified to read:

"Regarding your performance on the PASAT…" As in the original PANE, the EA-PANE

contains five items accompanying the prompt that consist of common ruminative responses to

negative events (e.g., "I'm playing the event over and over in my mind"). Participants were

asked to rate on a 1 (*Very unlike me*) to 5 (*Very like me*) Likert scale how well each item

characterized their response to the PASAT. The EA-PANE was administered both post-stress

and 18.5 minutes post-stress. It has demonstrated good internal consistency among older

adolescents and college students ($\alpha = .85$ to $\alpha = 88$; Mezulis & Rudolph, 2012). In the current

sample, coefficient alpha for task and recovery administrations were .88 and .90, respectively.

RSA data acquisition and calculation. During $T1_b$, ECG signals were amplified and

sampled continuously at 1,000 Hz during pre-stress (baseline), task, and recovery using a Biopac

MP150 Data Acquisition System (Biopac Systems, Inc., Goleta, CA). Two pre-gelled Ag/AgCL

electrodes were placed in a Lead II configuration on the participant's torso. Signals were

acquired with Biopac's AcqKnowledge 4.1 software. HRV 2.51 (MindWare Technologies Ltd.,

Gahanna, OH) was used to identify artifacts and ectopic beats in the ECG R-wave time series

through a combination of both visual examination and the MAD/MED distribution-based

detection algorithm (Berntson, Quigley, Jang, & Boysen, 1990) applied by the software.

Abnormal data detected through this process was manually corrected according to guidelines

suggested by Berntson and colleagues (1997). Next, RSA was calculated in HRV 2.51 using

spectral analysis. This technique employs a Fast Fourier Transformation to reduce the R-wave

time series into HRV frequency bands. High frequency (HF) values in the power spectrum (0.15

to 0.50 Hz) are considered to primarily reflect vagal influence on heart rate. These values were

log transformed to account for skew and used as indicators of RSA, which was calculated across 30-second epochs.

Baseline RSA was represented by the mean of 30-second epochs across the last two-minutes of the 5-minute nature scene viewing period. I calculated RSA task (the period during PASAT-C administration) by first removing the initial epoch of each PASAT task period to allow for orientation to the increasing demands of each new period. Next, I obtained the mean for the remaining epochs of each task period. Lastly, I averaged the three task periods together. This grand mean served as the RSA task score. As each task period was a different length, I chose to calculate means for each period before obtaining a grand mean to ensure equal weighting.

Finally, RSA recovery was represented by an average of the 1.5-minute physiological recovery period following feedback offset. Prior research on the prototypical time course of vagal recovery from stress is limited; however, studies suggest CVC may be reinstated relatively rapidly (within 90 seconds) to facilitate a return to homeostasis (Rottenberg et al., 2003; Rottenberg, Clift, et al., 2007). Thus, in the current study, I elected to use a shorter interval between task and recovery than for self-reported state NA and event-specific rumination.

Importantly, I also assessed for current medication usage prior to the stressor paradigm during $T1_b$. Several classes of medication are known to affect CVC, including antidepressants, stimulants, and antihistamines (for a review, see Rottenberg, 2007). Participants were asked to indicate "yes" or "no" to questions regarding use of these medications in the 24 hours prior to the laboratory visit.

Stress exposure. Stress exposure was measured at T2 with two stressful events checklists: the Adolescent Perceived Events Scale (APES; Compas, Davis, Forsythe, & Wagner,

1987) and the Undergraduate Stress Questionnaire (USQ; Crandall, Preisler, & Aussprung, 1992). The USQ is an 83-item self-report questionnaire intended to assess the frequency and intensity of stressful life events typically encountered by undergraduates. Participants are asked to indicate which stressors they have experienced within a given time frame. Events range from minor hassles (e.g., "Got to class late") to major stressful events (e.g., "Was a victim of a crime" or "Experienced a death [family member or friend]"). Reliability calculations are often not suitable for stress checklists. Specifically, internal consistencies may be low due to measurement of distinct stressors in a checklist that may have little to no relation with one another. Four-week test-retest reliability data are available for the USQ and range from $r = .53$ to .86 in undergraduate samples (Crandall et al., 1992). Low correlations between administrations may be expected, as many nonchronic stressful events abate by retest.

Similar to the USQ, the APES is designed to assess the presence and intensity of stressful events. The measure was developed by pooling lists of recent stressful events identified by 658 13- to 20-year olds. The final full APES is comprised of 210 stressful life events that were deemed to be most representative of adolescents' experience (Compas et al., 1987), with a greater emphasis on interpersonal stress than the USQ. To reduce participant burden, various short forms of the APES have been utilized, including a 100-item version (Grant & Compas, 1995). The two-week test-retest reliability for the full APES ranges from $r = .77$ to .85 (Compas et al., 1987).

Given that interpersonal stress and its related sequelae have been identified as salient predictors of depression among adolescents and young adults (Hammen, 1991), I sought to include a stress checklist such as the APES that captures a range of interpersonal stressful events. However, the APES includes items that may not be developmentally appropriate for a primarily

residential undergraduate sample (e.g., "I wasn't accepted into the college I applied to" or "I lost privileges or got punished at home"). Thus, I chose to also utilize checklist items that were relevant to college students from the USQ. Prior studies (e.g., Pettit, Lewinsohn, Seeley, Roberts, & Yaroslavsky, 2010) have also elected to use combined stressor checklists drawn from multiple measures.

In order to reduce burden on participants and create a measure that would capture a wide range of stressors most relevant to the study's sample, I eliminated all items on the 100-item short form of the APES that were developmentally inappropriate or that were deemed repetitive with item content on the USQ. The resultant number of total stressor items on the combined APES and USQ checklist was 77. In the current study, participants were asked to indicate (1 = *yes* or 0 = *no*) which items on the checklist they had experienced over the past eight weeks. A total stress exposure score was calculated by summing participants' responses. Possible scores ranged between 0 and 77, with higher scores indicating a greater number of stressful events experienced in the eight-week duration between $T1_b$ and T2.

Participants

During the 2011-2012 academic year, SPU reported a total of 3,194 enrolled undergraduate students with a mean age of 21 years. Of those students, 26% identified as an ethnic minority. While undergraduate statistics on gender distribution are not publicly available, campus-wide data (undergraduate, post-baccalaureate, and graduate enrollment combined) show that 68% of SPU students are female (Seattle Pacific University, 2011). Based on these data, I expected Caucasian females to be overrepresented in the study sample.

An a priori power analysis was conducted using G*Power 3.1.2 software (Faul, Erdfelder, Lang, & Buchner, 2009) to determine the sample size required to test study

hypotheses. The power analysis was conducted for the stress recovery hypothesis (H_4), as this analysis utilized the largest number of variables. To examine H_4, I elected to control for baseline depressive symptoms, trait vulnerabilities, and indices of reactivity to the stressor. Thus, I entered six variables as predictors in the power analysis: stress recovery, stress exposure, and the recovery*stress exposure interaction term. The power analysis indicated that in order to detect a minimum Cohen's f^2 of .15 using power of .80, I would need a minimum of 77 participants.

In total, 294 students consented to participate in the study and 274 subsequently completed $T1_a$. Of the $T1_a$ completers, 79 participants were not invited to $T1_b$ due to 1) participant recruitment limits determined by the Director of Undergraduate Research, or 2) technical problems with the MediaLab software needed to run the $T1_b$ experimental paradigm. $T1_b$ email invitations were sent to 195 participants. A total of 59 participants declined further participation in the study, did not reply the email invitation, or did not attend their scheduled $T1_b$ visit. One-hundred and thirty-six participants completed $T1_b$ and were subsequently invited to complete the T2 follow up. A total of 44 participants did not respond to their T2 email invitations. Consequently, 92 participants completed T2. This number comprised the final sample. Independent samples *t*-tests indicated no significant differences between T2 completers and T2 noncompleters on $T1_a$ variables and $T1_b$ state emotional reactivity (see Table 1).

Table 1

Descriptive Statistics for Baseline Variables and T1ᵦ Emotional Reactivity by T2 Completion Status

Variable	T2 Completers		T2 Noncompleters		*M* Diff	*t*	*p*
	M	*SD*	*M*	*SD*			
ERS	0.54	0.47	0.62	0.53	0.08	0.71	.48
CES-D	15.44	9.74	17.00	9.77	1.56	0.67	.50
PANE	3.51	0.70	3.57	0.69	0.07	0.45	.68
PANAS Change	0.35	0.52	0.26	0.45	-0.09	-0.76	.45

Note. M Diff = Difference between means on study variables by T2 completion status.
PANAS Change was calculated by subtracting PANAS task scores from baseline PANAS scores.

Participants who completed T2 ranged in age from 18.02 to 24.15 years (*M* = 19.50; *SD* = 1.37). As expected, the majority of the final sample (82.6%) identified as female, while 17.4% identified as male. With regard to ethnicity, 6.6% of participants reported being of Hispanic or Latino descent. The sample's racial demographics were similar to SPU's overall student population, with 72.9% identifying as Caucasian. Smaller numbers identified as Asian (13.6%), "Mixed/Other" (8.6%), and African-American (4.9%). No participants in the final sample reported being Native American.

CHAPTER III

Results

Data Analytic Plan

All data analyses were performed in SPSS 21.0. Hierarchical linear regression was

used to examine the main effects of domains of stress reactivity and recovery on the

prediction of T2 depressive symptoms (H_1 and H_3). The PROCESS macro for SPSS (Hayes,

2013) was utilized to separately examine the hypotheses that domains of stress reactivity and

recovery would interact with stress exposure to predict T2 depressive symptoms (H_2 and H_4).

The PROCESS macro yields coefficient and standard error estimates for both the moderator

and interaction term and is intended for use in moderation analyses that can be represented

by a single regression coefficient. PROCESS estimates simple slopes at the sample mean of

the moderator, as well as one standard deviation above and below the mean. Additionally,

the macro is advantageous for its ability to probe interaction effects using the Johnson-

Neyman (J-N) technique. The J-N technique yields moderator values quantifying at which

point the focal predictor's effects transition between statistical significance and

nonsignificance, thereby avoiding the arbitrary process of choosing high, medium, and low

moderator values from which the predictor's effects can be estimated.

Data Preparation Prior to Analysis

Prior to analysis, all data were visually examined for out-of-range values that were

the result of incorrectly entered data. Next, a missing value analysis was performed across

all variables for participants who completed T2. In total, 0.2% of item-level variables were

missing. Little's chi-square statistic, which is used to determine whether data is missing

completely at random (MCAR), was nonsignificant ($p = .10$). *P*-values above .05 in this

analysis indicate MCAR (i.e., that missingness is attributed to a factor unrelated to the data, such as a participant inadvertently skipping a questionnaire item). Therefore, I elected to follow the recommendation of Hayes, Slater, & Snyder (2008), who suggest that case deletion or Expectation Maximization (EM) are the most appropriate methods for handling missing values that are MCAR and comprise less than 2.0% of the total data. To preserve statistical power, I chose to use EM, which iteratively calculates expected values for missing data based on observed values and parameter estimates.

Finally, data were examined for normality. Analyses demonstrated that several variables were significantly skewed and/or kurtotic (see Table 2), as indicated by values greater than 1.00 (Field, 2009). Visual inspection of individual composite scores using boxplots showed the presence of several extreme outliers (i.e., scores over four standard deviations from the mean). Skew and kurtosis values were calculated after extreme outliers were removed (see Table 2). The ERS and all three PANAS administrations remained skewed and/or kurtotic. I elected to log transform ERS scores, which normalized the distribution. I chose not to perform transformations on $T1_b$ state data, as these values were expected to deviate from a normal distribution. For example, I expected that pre-stress PANAS scores would be skewed toward low levels of NA (negative affect), particularly because a habituation period was provided.

Table 2
Normality Results for Study Data

Variable	Pre-Cleaning		Post-Cleaning		
	Skew	Kurtosis	Outliers Removed	Skew	Kurtosis
ERS	1.72	4.29	1	0.57	-0.26
PANE	-0.10	-0.77	0	--	--
T1$_a$ CES-D	0.99	0.53	0	--	--
Baseline PANAS	2.10	5.08	1	1.86	3.80
PANAS Task	1.18	0.71	0	--	--
PANAS Recovery	2.16	5.74	1	1.64	2.18
PANE Task	0.32	-0.53	0	--	--
PANE Recovery	-0.54	-0.21	0	--	--
Baseline RSA	0.17	0.10	0	--	--
RSA Task	-0.10	-0.49	0	--	--
RSA Recovery	0.07	-0.14	0	--	--
T2 CES-D	0.43	-0.55	0	--	--
APES/USQ	1.11	2.75	3	0.05	-0.72

Descriptive Analyses

Means, standard deviations, and ranges for study variables are presented in Table 3.

Bivariate correlations among the variables are presented in Table 4. Correlations with

participant sex were also examined, as prior research has shown significant gender

differences in vulnerabilities to depression, including emotional reactivity and rumination

(see Hyde et al., 2008 for a review). As expected, trait emotional reactivity demonstrated

moderate positive associations with state emotional reactivity (task $r = .36$) and persistent

NA following the PASAT (recovery $r = .29$). Trait rumination was moderately correlated with initial rumination immediately following the task ($r = .39$) and at recovery ($r = .31$). Similarly, baseline RSA was highly correlated with task ($r = .64$) and recovery ($r = .68$). Therefore, I elected to control for trait vulnerabilities in all analyses in order to isolate the unique predictive effects of state vulnerabilities. Results also showed large correlations between task and recovery periods among all domains, indicating the need to control for task variables across recovery analyses. As expected, I also chose to enter $T1_a$ depressive symptoms as a covariate in all analyses due to moderate positive correlations with most independent variables ($r = .28$ to $r = .46$) and a strong correlation with T2 symptoms ($r = .52$). However, contrary to expectations, baseline RSA, task RSA, and recovery RSA was not associated with $T1_a$ or T2 depressive symptoms. Sex was not correlated with any predictor variables, likely due to the disproportionate number of females in the sample. Therefore, I elected to exclude sex as a covariate in all models.

A stressor manipulation check evaluated whether baseline levels of NA differed significantly from task NA. Results from a paired-samples t-test indicated a significant increase in NA from baseline to task ($t = -6.64$, $p < .001$). Thus, I concluded the PASAT-C was an effective means of inducing distress among participants.

Table 3
Means, Standard Deviations, and Ranges for Study Variables

Variable	*M*	*SD*	Min	Max
1. ERS	0.17	0.11	0.00	0.43
2. PANE	3.52	0.70	2.13	5.00
3. T1$_a$ CES-D	15.54	9.65	2.00	45.00
4. PANAS Baseline	1.23	0.30	1.00	2.50
5. PANAS Task	1.62	0.55	1.00	3.10
6. PANAS Recovery	1.32	0.41	1.00	2.80
7. PANAS BT Residual	0.00	0.51	-0.54	1.62
8. PANAS TR Residual	0.00	0.35	-0.92	1.20
9. EA-PANE Task	2.74	1.00	1.00	5.00
10. EA-PANE Recovery	1.75	0.90	1.00	4.00
11. EA-PANE TR Residual	0.00	0.67	-1.52	1.45
12. RSA Baseline	6.65	1.12	4.01	9.36
13. RSA Task	5.79	1.02	3.49	7.98
14. RSA Recovery	6.84	1.16	4.08	9.83
15. RSA BT Residual	0.00	0.85	-1.75	1.58
16. RSA BT Residual	0.00	0.85	-2.40	2.34
17. T2 CES-D	14.41	8.92	0.00	38.00
18. APES/USQ	20.37	8.87	0.00	47.00

Note. ERS mean, standard deviation, and range reflect log-transformed values. PANAS BT Residual = Standardized residual for PANAS baseline to task score. PANAS TR Residual = Standardized residual for PANAS task to recovery score. EA-PANE TR Residual = Standardized residual for EA-PANE task to recovery score. RSA BT Residual = Standardized residual for RSA baseline to task score. RSA TR Residual = Standardized residual for RSA task to recovery score.

Table 4

Correlations among Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. ERS	__	.36**	.45**	.15	.36**	.29**	.31**	.15	.27*	.21	.03	.14	-.08	.17	-.29*	.32**	.40**	.06	.02
2. PANE		__	.36**	.22*	.36**	.43**	.28**	.29**	.39**	.31**	.06	-.07	-.06	-.03	-.12	.03	.27**	.21	.14
3. T1$_a$ CES-D			__	.34*	.36**	.46**	.26*	.36**	.34**	.28**	.06	-.06	.00	.06	-.80	.05	.52**	.09	.16
4. PANAS Baseline				__	.30**	.34**	.00	.22**	.18	.07	-.07	.35**	.07	.28**	.07	.07	-.04	-.21*	.08
5. PANAS Task					__	.52**	.95**	.00	.55**	.34**	-.05	.07	-.08	.01	-.22	.35**	.32**	.15	-.02
6. PANAS Recovery						__	.38**	.85**	.36**	.24*	-.01	.13	.02	.17	-.16	.04	.30**	.33**	.07
7. PANAS BT Residual							__	.13	.51**	.33**	-.01	.35**	.02	.23	-.23	.31**	.33**	.22*	.01
8. PANAS TR Residual								__	.08	.06	.01	-.10	-.11	-.85	-.07	-.15	.17	.29**	.10
9. EA-PANE Task									__	.68**	.00	.13	.02	.17	-.09	.21	.21*	.20	.09
10. EA-PANE Recovery										__	.78**	-.06	-.13	.04	-.14	.15	.09	.19	.03
11. EA-PANE TR Residual											__	-.09	-.14	-.08	.31*	-.15	-.07	.07	-.04
12. RSA Baseline												__	.64**	.68**	.00	.51*	.07	-.13	-.07
13. RSA Task													__	.78**	.81**	.00	-.08	-.08	.15
14. RSA Recovery														__	.27*	.73**	-.06	-.06	-.03
15. RSA BT Residual															__	-.13	-.17	-.02	-.04
16. RSA TR Residual																__	.06	-.04	-.17
17. T2 CES-D																	__	.29**	.24*
18. APES/USQ																		__	.19
19. Participant Sex																			__

Note. Values presented for participant sex are point-biserial correlations.
PANAS BT Residual = Residual for PANAS baseline to task score. PANAS TR Residual = Residual for PANAS task to recovery score. EA-PANE TR Residual = Residual for EA-PANE task to recovery score. RSA BT Residual = Residual for RSA baseline to task score. RSA TR Residual = Residual for RSA task to recovery score.
$^*p < .05.$ $^{**}p < .01.$

Concurrent Analyses

Prior to conducting prospective analyses, main effect hypotheses were examined concurrently using the $T1_a$ CES-D as the dependent variable in each model. Multiple methods for modeling task and recovery were considered, including controlling for raw pre-stress or baseline levels of my independent variables, calculating difference scores, and using percent change scores or residual change scores. Although utilizing difference scores may be advantageous in this type of research (see Nelson, Shankman, Olino, & Klein, 2011), I ultimately elected to use raw scores to maintain consistency across the affective, biological, and cognitive analyses. Specifically, state rumination about the stressor does not have a proper baseline comparison condition, thus precluding the use of difference scores in this domain.

Affective responding. Hypothesis 1_a stated that NA at task would be associated with depressive symptoms above and beyond the contribution of trait emotional reactivity. As seen in Table 4, greater task NA was concurrently associated with depressive symptoms and accounted for 4% of the variance in the model above and beyond trait emotional reactivity and baseline levels of NA.

I also hypothesized that persistent NA during the recovery period would be associated with depressive symptoms (H_{3a}). Results can be seen in Model 3 in Table 4. Consistent with my hypothesis, higher NA at recovery was associated with concurrent depressive symptoms, accounting for 9% of the variance in the entire model. Of note, the relationship between task NA and depressive symptoms was reduced to nonsignificance when recovery NA was entered into the model.

Table 5

Concurrent Model of the Relationship between State Emotionality and Depressive Symptoms

Variable	*B*	*SE*	β	*t*	*p*	R^2	ΔR^2
Model 1						.26	--
Trait Emotional Reactivity	34.39	7.79	.41	4.41	.00		
Baseline NA	8.47	2.86	.27	2.97	.00		
Model 2						.28	.04*
Trait Emotional Reactivity	28.73	8.24	.34	3.49	.00		
Baseline NA	6.84	2.94	.22	2.32	.02		
Task NA	3.69	1.76	.21	2.10	.04		
Model 3						.35	.09***
Trait Emotional Reactivity	21.49	7.82	.26	2.75	.01		
Baseline NA	4.52	2.80	.15	1.62	.11		
Task NA	0.74	1.78	.04	0.41	.68		
Recovery NA	8.11	2.36	.36	3.44	.00		

Note. *$p < .05$. **$p < .01$. ***$p < .01$. $p < .001$.

Biological responding. Prior to evaluating concurrent biological hypotheses, I first examined correlations between RSA and medication use. Six participants endorsed taking antidepressants in the 24 hours prior to $T1_b$, while one participant reported taking stimulants and 15 endorsed taking antihistamines. The medication use and RSA correlation matrix is presented below in Table 5. As seen, medication usage was generally correlated with lower RSA; however, only antihistamine usage and RSA reactivity were significantly negatively correlated. Given the mixed results, and because I did not assess for medication dosage, I followed the methodology of Salomon, Clift, Karlsdóttir, and Rottenberg (2009) by running parallel analyses using the entire sample as well as only those who did not endorse medication usage ($n = 69$).

Table 6

Correlations among RSA Variables and 24-Hour Medication Usage

Variable	1	2	3	4	5	6
1. RSA Baseline	__	.64[**]	.78[**]	-.06	-.11	-.08
2. RSA Task		__	.68[**]	.05	.01	-.23[*]
3. RSA Recovery			__	-.02	-.05	-.14
4. Antidepressants				__	.40[**]	-.01
5. Stimulants					__	-.05
6. Antihistamines						__

Note. [*] $p < .05$. [**] $p < .01$.

Hypothesis H_{1b} predicted that higher RSA during the PASAT-C would be associated with greater depressive symptoms. Results for the entire sample are displayed in Table 6. Analyses showed that when controlling for baseline RSA, Task RSA did not demonstrate a relationship with concurrent depressive symptoms. Similarly, as seen in Model 3, low RSA recovery was not associated with symptoms (H_{3b}). While not significant, results trended in the opposite direction of what was expected, with higher RSA at recovery being associated with greater symptoms.

Table 7

Concurrent Model of the Relationship between Respiratory Sinus Arrhythmia and Depressive Symptoms – Full Sample

Variable	B	SE	β	t	p	R^2	ΔR^2
Model 1						.01	--
RSA Baseline	-0.59	0.87	-.07	-0.68	.50		
Model 2						.01	.00
RSA Baseline	-0.50	1.14	-.06	-0.44	.66		
RSA Task	-0.15	1.25	-.02	-0.12	.90		
Model 3						.01	.00
RSA Baseline	-0.96	1.46	-.12	-0.66	.51		
RSA Task	-0.42	1.36	-.05	-0.31	.76		
RSA Recovery	0.76	1.47	.10	0.52	.61		

Results for the non-medicated sample are presented in Table 7. Baseline RSA was not associated with concurrent depressive symptoms among those participants not reporting medication usage. In support of my first hypothesis, lower RSA during the laboratory stressor was marginally associated with less depressive symptoms, adding an additional 7% of variance to the overall model. RSA recovery demonstrated nonsignificant associations with symptoms; however, results were opposite of what was expected, with participants exhibiting better recovery back to baseline also reporting greater symptoms. Of note, Task RSA became more significant in the expected direction in the recovery model.

Table 8
Concurrent Model of the Relationship between Respiratory Sinus Arrhythmia and Depressive Symptoms – Non-Medicated Sample

Variable	*B*	*SE*	β	*t*	*p*	R^2	ΔR^2
Model 1						.03	--
RSA Baseline	-1.15	0.89	-.16	-1.28	.20		
Model 2						.07	.04
RSA Baseline	0.18	1.18	.02	0.15	.88		
RSA Task	-2.43	1.42	-.28	-1.71	.09		
Model 3						.10	.03
RSA Baseline	-1.09	1.48	-.15	-0.74	.46		
RSA Task	-3.44	1.58	-.39	-2.17	.03		
RSA Recovery	2.20	1.57	.31	1.40	.17		

Cognitive responding. Regarding cognitive stress responding, I hypothesized that greater rumination about PASAT-C performance immediately following completion of the task would be associated with depressive symptoms above and beyond the contribution of trait rumination (H_{1c}). As shown in Model 2 of Table 8, greater rumination at task was associated with concurrent symptoms and significantly explained an additional 5% of the variance in the model above and beyond trait levels. I also hypothesized that persistent rumination approximately 20 minutes after stressor offset would be associated with depressive symptoms

after controlling for both trait rumination and reactivity (H_{3c}). Results did not support this hypothesis. As seen in Model 3, when all variables were entered, only trait rumination emerged as significant. Rumination at the recovery assessment did not contribute any additional variance to the model.

Table 9
Concurrent Model of the Relationship between State Rumination and Depressive Symptoms

Variable	B	SE	β	t	p	R^2	ΔR^2
Model 1						.13	--
Trait Rumination	4.98	1.35	.36	3.70	.00		
Model 2						.17	.05[*]
Trait Rumination	3.75	1.48	.27	2.54	.01		
Rumination Task	2.28	1.03	.23	2.22	.03		
Model 3						.17	.00
Trait Rumination	3.70	1.49	.26	2.90	.02		
Rumination Task	1.86	1.33	.19	1.40	.17		
Rumination Recovery	0.70	1.42	.07	0.49	.62		

Note. [*]$p < .05$.

Prospective Analyses

Next, I examined main effects prospectively. T2 depressive symptoms were entered as the dependent variable. In order to model change in symptoms, $T1_a$ depressive symptoms were controlled for in all analyses. Finally, I conducted moderation analyses to evaluate potential interactive effects of state stress responses and life stress exposure on depressive symptoms (H2 and H4). Results are presented below by domain.

Affective responding. As seen in Model 1 of Table 9, $T1_a$ depressive symptoms, trait emotional reactivity, and baseline NA accounted for 35% of the total variance in T2 depressive symptoms. Contrary to expectations, lower levels of baseline NA prospectively predicted greater depressive symptoms. Task NA was examined in Model 2 and emerged as a marginal predictor of T2 depressive symptoms ($H1_a$), contributing 2% additional variance. $T1_a$ symptoms and

baseline NA remained significant, while trait emotional reactivity was reduced to a marginal predictor. I next tested Hypothesis 3_a, which stated that greater NA at recovery would be the strongest prospective predictor of symptoms. As seen in Model 3, results indicated that recovery NA was not associated with T2 symptoms and contributed 0% additional variance to the model.

Table 10

Prospective Model of the Relationship between State Emotionality and Depressive Symptoms

Variable	B	SE	β	t	p	R^2	ΔR^2
Model 1						.35	--
$T1_a$ Depressive Symptoms	0.50	0.10	.52	5.21	.00		
Trait Emotional Reactivity	16.19	7.76	.20	2.09	.04		
NA Baseline	-7.48	2.70	-.25	-2.78	.01		
Model 2						.38	.02[†]
$T1_a$ Depressive Symptoms	0.47	0.10	.49	4.75	.00		
Trait Emotional Reactivity	13.30	8.02	.16	1.66	.10		
NA Baseline	-8.24	2.76	-.28	-2.98	.00		
NA Task	2.46	1.64	.15	1.52	.13		
Model 3						.37	.00
$T1_a$ Depressive Symptoms	0.48	0.11	.48	4.41	.00		
Trait Emotional Reactivity	14.00	8.11	.17	1.73	.09		
NA Baseline	-8.52	2.82	-.29	-3.02	.00		
NA Task	2.53	1.77	.15	1.43	.16		
NA Recovery	0.74	2.50	.03	0.30	.77		

Note. [†] marginal.

As my prospective hypotheses did not hold, I sought to examine whether mean levels of task and recovery NA differed among individuals endorsing high vs. low symptoms. Specifically, I conducted post hoc ANCOVAs utilizing participants scoring in the upper 25th and lower 25th percentiles of T2 depressive symptoms to capture possible affective differences at the extremes. Results indicated no significant mean differences among the two groups for task, $F(4, 53) = 0.81$, $p = .37$, partial $\eta^2 = .02$, or for recovery, $F(5, 52) = 0.23$, $p = .63$, partial $\eta^2 = .01$. I also ran ANCOVAs comparing affective responding of individuals who showed an increase in symptoms from $T1_a$ to T2 versus those whose symptoms decreased. Findings indicated that

participants who had an increase in symptoms reported marginally higher levels of task NA, $F(3,$ 85) = 2.90, p =.09, partial η^2 = .03 and recovery NA, $F(4, 83)$ = 8.09, p =.01, partial η^2 = .09, as compared with those who exhibited a decrease in symptoms.

I next examined the affective moderation hypotheses, which hypothesized that state affective responses to induced stress would interact with life stress exposure during the 8-week study period to predict T2 depressive symptoms. Prior to conducting moderation analyses, I examined whether there was a main effect of life stress exposure. Results showed that after controlling for $T1_a$ symptoms, greater self-reported life stress exposure predicted higher depressive symptoms at T2, β = .25, t = 2.80, p = .006. The model accounted for 58% of the variance in symptoms at T2, with life stress exposure contributing an additional 33% above $T1_a$ symptoms alone (F = 21.23, $p < .001$).

I utilized the PROCESS macro for SPSS to conduct moderation analyses. Following the recommendation of Aguinis (2004), I tested each interaction model using a p-value of .10 to account for the reduced power for finding significant effects that occurs when conducting moderation analyses. I began by testing task NA (H_{2a}). T2 depressive symptoms were entered as the dependent variable. Task NA was entered as the independent variable and life stress was included as the moderator. $T1_a$ depressive symptoms, trait emotional reactivity, and baseline NA were entered as covariates in the model. The PROCESS macro mean-centered state emotional reactivity and life stress exposure prior to analysis. Results indicated the task NA*life stress interaction term was marginally significant, $B(SE)$ = 0.28(0.19), t = 1.44, p = .15 and contributed 2% additional variance over the main effects model. As the interaction was marginally significant, I elected to probe moderator values with the J-N technique. When mean-centered values of life stress exposure were between 1.60 and 24.08, greater task NA predicted higher

depressive symptoms. In other words, the magnitude of the positive relationship between task

NA and T2 depressive symptoms increased at higher levels of life stress exposure. Results are
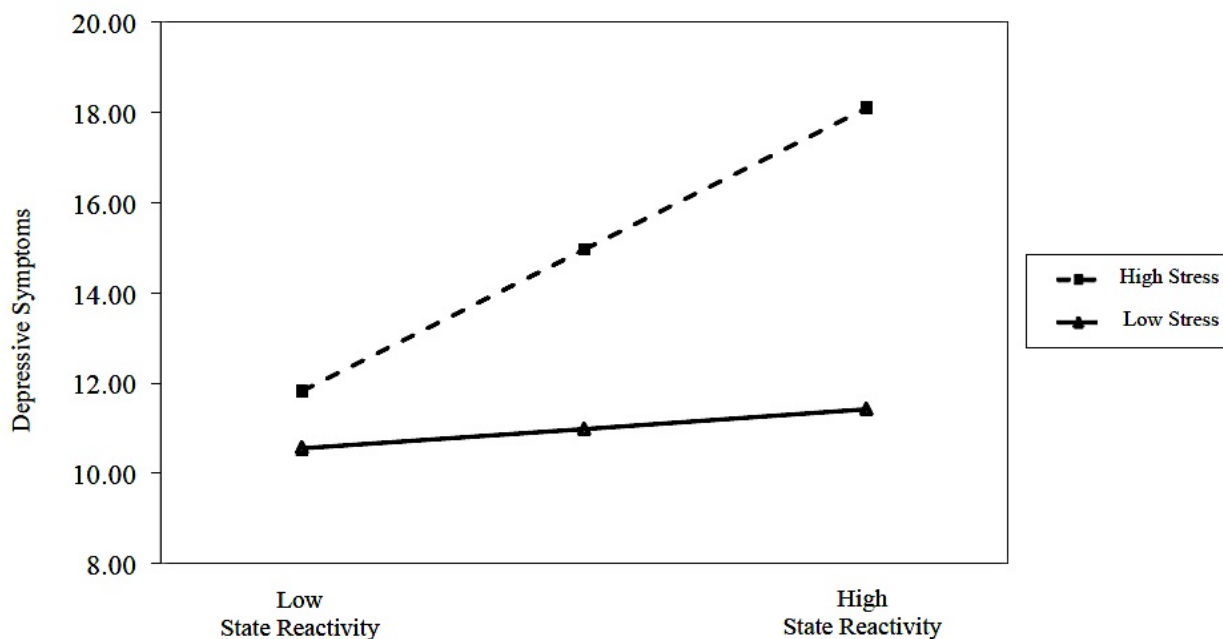
presented visually in Figure 7.

Figure 7. Interaction between state emotional reactivity and life stress exposure predicting
depressive symptoms.

I next examined life stress exposure as a moderator of the prospective relationship

between recovery NA and depressive symptoms (H_{4a}). Although results indicated there was not

a main effect of recovery NA on symptoms, I predicted a relationship would emerge under

higher levels of life stress during the 8-week study period. $T1_a$ depressive symptoms, trait

emotional reactivity, baseline NA, and task NA were entered into the model as covariates. Prior

to analysis, the PROCESS macro mean-centered recovery NA and life stress exposure. Results

showed no interactive effects of the predictors on T2 depressive symptoms, *B*(*SE*) = -0.01(.18), *t*

= -0.07, *p* = .94. Several variables retained significance or marginal significance in the

moderation model, including $T1_a$ depressive symptoms ($p < .001$), baseline NA ($p = .05$), and life stress exposure ($p = .06$). Therefore, H_{4a} was not supported.

Biological responding. H_{1b} stated that task RSA would prospectively predict depressive symptoms above and beyond baseline RSA. I first examined this hypothesis in the full sample. As seen in Table 10, after controlling for $T1_a$ depressive symptoms, there was a nonsignificant trend for higher baseline RSA to predict greater T2 depressive symptoms. This finding was contrary to theory and was maintained throughout all models. As shown in Model 2, task RSA was not associated with T2 symptoms; however results trended in the expected direction, with higher RSA during the PASAT-C prospectively predicting greater symptoms.

H_{3b} was examined in Model 3 of Table 11. Although results were not significant, the slope was in the expected direction, with low recovery RSA predicting greater depressive symptoms. The addition of the task and recovery variables did not add significant variance to the model. $T1_a$ symptoms remained the strongest predictor across both task and recovery analyses. Of note, baseline RSA became marginally significant when recovery was included in the model. Similar to Model 2, baseline RSA retained an unexpected positive relationship with T2 depressive symptoms.

Table 11

Prospective Model of the Relationship between Respiratory Sinus Arrhythmia and Depressive Symptoms – Full Sample

Variable	B	SE	β	t	p	R^2	ΔR^2
Model 1						.24	--
T1$_a$ Depressive Symptoms	0.48	0.94	.49	5.14	.00		
Baseline RSA	0.85	0.75	.11	1.13	.26		
Model 2						.25	.01
T1$_a$ Depressive Symptoms	0.48	0.94	.49	5.11	.00		
Baseline RSA	1.36	0.98	.17	1.38	.17		
Task RSA	-0.87	1.08	-.10	-0.81	.42		
Model 3						.26	.01
T1$_a$ Depressive Symptoms	0.48	0.09	.49	5.16	.00		
Baseline RSA	2.02	1.25	.26	1.61	.11		
Task RSA	-0.49	1.16	-.06	-0.42	.67		
Recovery RSA	-1.08	1.26	-.14	-0.86	.39		

Next, I conducted moderation analyses with the full sample to examine whether the relationships among T2 depressive symptoms, task RSA (H$_{2b}$), and recovery RSA (H$_{4b}$) were dependent upon levels of life stress exposure. I first examined the biological reactivity moderation hypothesis. Using the PROCESS macro, I entered T2 depressive symptoms as the dependent variable, task RSA as the independent variable, life stress exposure as the moderator, and lastly, T1$_a$ depressive symptoms and baseline RSA as covariates. Task RSA and life stress exposure were automatically mean-centered by the macro prior to running the analysis. Results indicated the Task RSA*life stress exposure interaction term was not significant, $B(SE)$ = -0.02(0.10), t = -0.25, p = .80. The moderation model contributed 0% additional variance to the main effects model, which accounted for 32% of the predictive power in T2 depressive symptoms. Similar to the main effect findings shown in Table 8, T1$_a$ symptoms retained a significant positive association with T2 symptoms ($p < .001$). Higher baseline RSA also continued to marginally predict greater T2 symptoms ($p = .11$).

The biological recovery moderation hypothesis was examined next with the full sample. Recovery RSA was entered as the independent variable. Task RSA was entered as a covariate, along with T1$_a$ depressive symptoms and baseline RSA. Life stress exposure was once again entered as the moderator and was mean-centered, along with recovery RSA. Results showed that the interaction between recovery RSA and life stress exposure did not prospectively predict T2 depressive symptoms, $B(SE) = -0.00(0.10)$, $t = -0.01$, $p = .99$ and contributed 0% variance to prediction model. T1$_a$ symptoms once again emerged as the strongest predictor of T2 symptoms ($p < .001$). Of note, as in previous models, baseline RSA showed a significant positive slope ($p = .05$). In summary, task RSA and recovery RSA were not prospectively associated with depressive symptoms, regardless of participants' levels of life stress exposure. Contrary to expectations, baseline RSA consistently demonstrated a positive predictive relationship with T2 symptoms.

Lastly, I examined all prospective biological analyses using only participants who did not endorse medication usage. As shown in Table 12, baseline RSA was not associated with T2 symptoms when controlling for T1$_a$ symptoms. Task RSA did not add additional variance to the model and was not associated with symptoms. In Model 3, recovery RSA only added an additional 1% of variance and was not significant; however, results were in the expected direction, with lower recovery RSA being associated with greater T2 symptoms. Similar to the model run with the full sample, T1$_a$ symptoms remained significant in across baseline, task, and recovery analyses.

Table 12

Prospective Model of the Relationship between Respiratory Sinus Arrhythmia and Depressive Symptoms – Non-Medicated Sample

Variable	B	SE	β	t	p	R^2	ΔR^2
Model 1						.32	--
T1$_a$ Depressive Symptoms	0.57	0.11	.57	5.45	.00		
Baseline RSA	0.49	0.78	.07	0.64	.52		
Model 2						.32	.00
T1$_a$ Depressive Symptoms	0.58	0.11	.58	5.36	.00		
Baseline RSA	0.29	1.01	.04	0.29	.77		
Task RSA	0.39	1.25	.04	0.31	.76		
Model 3						.33	.01
T1$_a$ Depressive Symptoms	0.60	0.11	.60	5.45	.00		
Baseline RSA	1.07	1.29	.15	0.83	.41		
Task RSA	1.06	1.42	.12	0.75	.46		
Recovery RSA	-1.36	1.38	-.20	-0.99	.33		

While the main effect hypotheses were not supported prospectively with the non-medicated sample, I sought to examine whether life stress exposure interacted to predict symptoms among this sample subset. Variables were entered in the same manner as in the moderation analyses using the full sample. Results indicated that task RSA did not interact with life stress exposure in the prediction of T2 depressive symptoms, $B(SE) = 0.05(0.02)$, $t = 0.50$, $p = .61$. Again, T1$_a$ symptoms remained the only significant predictor in the model ($p < .001$), accounting for 40% of the variance. The interaction contributed 0% additional variance.

The recovery moderation analysis yielded similar results. The recovery RSA*life stress interaction was not significant, $B(SE) = 0.07(0.10)$, $t = 0.69$, $p = .49$. As in previous prospective analyses, T1$_a$ symptoms were significant ($p < .001$). Total variance accounted for was 42%, with the interaction contributing 0% to the model.

Cognitive responding. The final set of analyses addressed hypotheses regarding state rumination. H$_{1c}$ stated that rumination about PASAT-C performance measured immediately after stressor offset (task rumination) would prospectively predict T2 depressive symptoms

above and beyond the contribution of trait rumination. Results are presented below in Table 12.

In contrast to theory and prior studies, trait rumination was not prospectively associated with T2

depressive symptoms after controlling for symptoms at T1$_a$ (see Model 1). Contrary to my

hypothesis, task rumination did not predict T2 symptoms and did not contribute any additional

variance to the prospective model (see Model 2).

H$_{3c}$ predicted that recovery rumination would emerge as the strongest predictor of T2

symptoms after controlling for trait rumination and task rumination. As seen in Model 3 of

Table 12, recovery rumination was not significantly associated with T2 symptoms; however, it

unexpectedly trended a negative slope such that less rumination 20 minutes after PASAT-C

completion predicted higher depressive symptoms eight weeks later. Model 3 accounted for

28% of the prospective variance in symptoms, but results indicated that T1$_a$ was the strongest

predictor.

Table 13
Prospective Model of the Relationship between State Rumination and Depressive Symptoms

Variable	B	SE	β	t	p	R^2	ΔR^2
Model 1						.28	--
T1$_a$ Depressive Symptoms	0.45	0.09	.49	5.01	.00		
Trait Rumination	1.17	1.22	.09	0.95	.34		
Model 2						.27	.00
T1$_a$ Depressive Symptoms	0.45	0.09	.49	4.82	.00		
Trait Rumination	0.91	1.33	.07	0.68	.50		
Task Rumination	0.15	0.92	.02	0.16	.87		
Model 3						.28	.01
T1$_a$ Depressive Symptoms	0.45	0.09	.49	4.88	.00		
Trait Rumination	0.98	1.33	.08	0.74	.46		
Task Rumination	0.98	1.16	.11	0.84	.40		
Task Rumination	-1.42	1.23	-.14	-0.16	.25		

I next conducted moderation analyses with the PROCESS macro to test whether state

rumination would predict depressive symptoms under specific levels of life stress exposure. I

examined rumination reactivity first (H$_{4c}$). T2 depressive symptoms were entered as the

dependent variable. Task rumination was entered as the independent variable and life stress

exposure as the moderator. Both variables were automatically mean-centered by the macro prior

to analysis. $T1_a$ depressive symptoms and trait rumination were entered as covariates. Results

indicated that the task rumination*life stress exposure interaction term marginally predicted T2

depressive symptoms, $B(SE) = 0.13(0.09)$, $t = 1.47$, $p = .14$. The model accounted for 35% of

the variance in depressive symptoms, with the interaction term contributing 2% above main

effects alone. The PROCESS macro did not probe the interaction using the J-N technique, as

there were no points at which task rumination became statistically significant within the

observed range of the moderator. However, I elected to graph the results to determine whether

the data trended in the expected direction (see Figure 8). As hypothesized, participants with

higher levels of stress and more task rumination tended to report the greatest number of

depressive symptoms at T2. However, there was an unexpected trend for participants with low

stress and low task rumination to report more symptoms than those with low stress and high
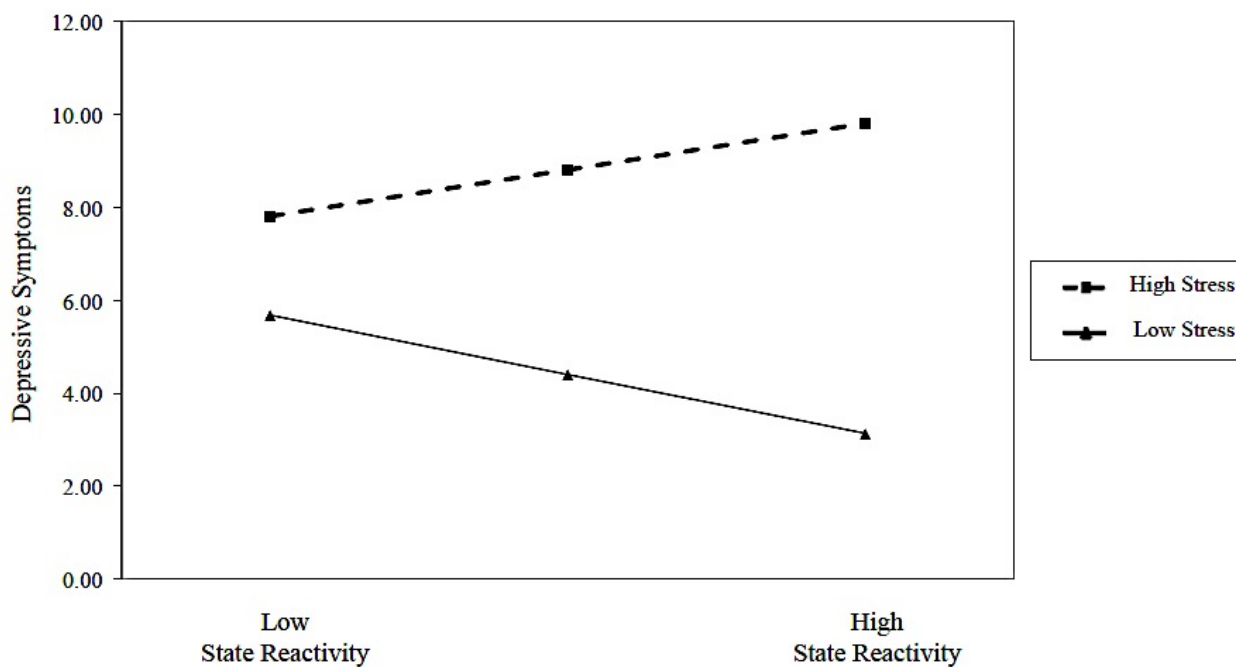
cognitive reactivity to the task.

Figure 8. Interaction between rumination reactivity and life stress exposure predicting depressive symptoms.

Lastly, I examined the recovery rumination hypothesis (H_{4c}). Recovery rumination was entered as the independent variable and life stress exposure as the moderator. Prior to analysis, the PROCESS macro mean-centered both predictors. $T1_a$ depressive symptoms, trait rumination, and task rumination were entered as covariates. Overall, the model accounted for 34% of the variance in symptoms; however, the recovery rumination*life stress exposure interaction term contributed 0% variance and was not significant, $B(SE) = 0.05(0.10)$, $t = 0.46$, $p = .65$. $T1_a$ symptoms were retained as the strongest predictor in the model. In summary, life stress exposure marginally interacted with task rumination, but not recovery, to predict depressive symptoms over time.

CHAPTER IV

Discussion

Vulnerability-stress models of depression posit that maladaptive responses to stressful events confer risk for the onset of depression. While most empirical studies of stress responding and depression focus on initial reactivity to stress, evidence from cognitive processing and emotional processing theories are increasingly pointing to the role of recovery from stress as being implicated in the development and maintenance of depression. The current study provides an initial prospective test of the stress recovery-depression link within a vulnerability-stress framework. I examined this model across affective, biological, and cognitive domains with particular attention to the time course of responding during a laboratory stress induction. I examined four primary hypotheses: 1) domains of reactivity to the stress induction would be associated with depressive symptoms above and beyond the contribution of trait vulnerabilities; 2) the relationship between domains of reactivity and symptoms would be moderated by life stress exposure; 3) domains recovery from the stress induction would be associated with symptoms and would emerge as the strongest predictor beyond trait vulnerabilities and reactivity; and 4) the relationship between domains of recovery and symptoms would be moderated by life stress exposure. I examined main effects both concurrently and prospectively. I also examined moderation models prospectively. In the following chapter, I summarize my findings and discuss their contribution to understanding vulnerability to depression. For clarity, the discussion of results is organized by stress response domain.

Affective Responding: Time Course Matters

Extant research has demonstrated a consistent relationship between trait NA and depressive symptoms, both concurrently and prospectively (e.g., Anthony et al., 2002; Mezulis &

Rudolph, 2012; Wetter & Hankin, 2009). Although there is less direct evidence to support a state NA-stress interaction, theory suggests exposure to negative life events activates state NA, particularly in temperamentally vulnerable individuals, which in turn predicts depression (Ingram, Miranda, & Segal, 1998). In line with this theory, state affective reactivity has been shown to interact with stress, particularly of an interpersonal nature, to predict depressive symptoms (e.g., O'Neill et al., 2004).

Results indicated the affective hypotheses were partially supported. Concurrently, task NA (state affective reactivity) and recovery NA (state affective recovery) were both associated with depressive symptoms. The full model showed that although trait NA continued to make a significant contribution, recovery NA following the stress induction emerged as the strongest predictor of current symptoms. Importantly, the relationship between task NA and symptoms was reduced to nonsignificance when recovery was entered into the model.

Prospective findings showed that task NA marginally predicted symptoms, particularly in the context of greater life stress exposure. In other words, participants who reported greater state NA immediately following the PASAT-C and reported more life stressors during the 8-week study period were increasingly more likely to also report higher symptoms. In contrast, participants who reported greater state NA immediately after laboratory stressor offset but who did not endorse high levels of life stress over the study period reported fewer depressive symptoms at follow up and tended to endorse similar levels of symptoms as participants who exhibited low state reactivity and low life stress exposure. In contrast to expectations, greater recovery NA did not prospectively predict depressive symptoms at follow up. Further, recovery NA did not interact with life stress exposure in predicting symptoms. However, post hoc

analyses showed a trend towards greater reactivity and poorer recovery among participants who had an increase in symptoms from $T1_a$ to T2.

Results lend support to both trait and proximal affective vulnerability-stress models of depression. Further, this study is the first to demonstrate the importance of considering the time course of proximal affective vulnerability during stress. A handful of prior laboratory studies have examined affective recovery (Beevers, 2003; Clasen, Wells, Ellis, & Beevers, 2013; Gilboa & Gottlib, 1997); however, these studies utilized mood inductions rather than stressor tasks and did not consider the contribution of trait NA. Induced stress paradigms may be more effective in examining vulnerability, as they allow for self-referential affective and cognitive processing. The persistence of negative affect following stress may exacerbate negative depressogenic cognitions (Teasdale, 1998), which in turn is likely to fuel additional negative affect and further impair an individual's ability to effectively recover from the event. Over time and in the absence of adaptive emotion regulation strategies, this repeated process may consolidate into risk for depression. Of note, poor affective recovery following the laboratory stressor was only associated with concurrent symptoms and did not hold prospective predictive power. Following the widely held characterization of persistent negative affect as a hallmark feature of depression, it is possible that difficulty with affective recovery may not serve as a pronounced proximal vulnerability, but rather as an associated feature of current depression or of more severe symptoms.

It is also plausible that individual differences in the time course of affective responding in the prospective model were masked by the broad hypothesis that the most vulnerable individuals would exhibit high reactivity *and* poor recovery to baseline. Clasen and colleagues (2013) found support for two distinct affective response patterns among individuals with MDD: 1) high

reactivity and poor recovery; and 2) high reactivity and pronounced rapid recovery back to baseline. In contrast to these negative potentiation patterns, a third is also likely in which some individuals with MDD experience attenuated affective reactivity to stress (Bylsma, Morris, & Rottenberg, 2008; Rottenberg, 2005). Here, one should expect little change from task or initial reactivity to recovery, as there would be little response to the stressor to begin with. While these three response patterns were drawn from research with individuals who were diagnosed with MDD, they are relevant to the current study's findings, particularly because the sample means for $T1_a$ and T2 symptoms neared the clinical significance cutoff score of 16 on the CES-D ($M = 15.54$ and $M = 14.41$, respectively). Therefore, it is possible I may not have been entirely capturing proximal *vulnerability*, but rather features of potentially significant symptoms.

In support of the attenuation hypothesis, Peeters, Berkhof, Rottenberg, & Nicolson (2010) found that higher levels of negative affective reactivity predicted better recovery from MDD at 18-month follow up. Individuals with attenuated reactivity showed the poorest outcomes. The authors speculated that reactivity may actually serve an adaptive role in that its aversiveness may signal the need for active coping such as engagement in goal-oriented and rewarding activities. Therefore, if the high levels of NA observed during the task and recovery periods of the current study were representative of genuine reactivity to life events, it is possible this reactivity actually triggered adaptive coping during the study period, which would theoretically result in lower reported depressive symptoms at T2. While post hoc results indicated a trend for high levels of NA at task and recovery to be associated with T2 symptoms, these analyses did not assess the trajectory of individual symptom courses.

If high NA actually serves as an antecedent cue to alter behavior, it stands to reason that individuals who are able to develop effective coping in the face of intense reactivity may have

intact reward sensitivity. In line with the tripartite theory (Watson et al., 1986) and emotion context sensitivity theory (Rottenberg, 2005), individuals who are at greatest risk for depression or who are currently exhibiting symptoms may show blunted reactivity to neutral or positively valenced stimuli. Assessing the joint contribution of the time course of both positive affect and negative affect within the context of stressful and rewarding laboratory paradigms may help clarify the mixed findings in the current study as well as conflicting results in the literature.

Clarifying the Role of Respiratory Sinus Arrhythmia

CVC has been conceptualized as a psychophysiological index of attentional engagement, psychological flexibility, and self-regulatory capacity (Thayer et al., 2012; Thayer & Lane, 2000). Given that poor emotion regulation, prolonged difficulty disengaging from negative stimuli, and inflexibility in responding to environmental challenges are features associated with depression (e.g., (Joormann & D'Avanzato, 2010; Rottenberg, 2005), it stands to reason that RSA may demonstrate associations with current depressive symptoms and may also serve as a vulnerability factor that interacts with life stress to convey future risk for disorder. There is a dearth of research on the relationship between RSA and depression; current studies have generally yielded inconsistent findings. I sought to gain clarification by examining RSA recovery from stress as a critical factor in disentangling these associations. I analyzed data from both the full sample and a subset that did not endorse current medication usage, given the potential effects of specific substances on cardiovascular functioning.

Surprisingly, baseline RSA did not demonstrate associations with depressive symptoms at baseline or follow up, suggesting a tonic measure alone may not be a sufficient assessment of risk. However, a handful of other studies (e.g., Licht et al., 2008) have found a baseline RSA-depression link. It is possible my use of a "vanilla baseline" (Diamond & Otter-Henderson,

2007) may have obscured natural associations. Vanilla baselines are assessments in which physiological measures are gathered while participants are instructed to engage quietly in a task with low attentional demand. In contrast, true physiological baselines are obtained while participants simply sit quietly without experimental stimuli for a given amount of time.   In the current study, participants were instructed to sit for a 5-minute period and watch a series of nature scenes on the computer screen. Baseline RSA was computed from the final two minutes. It is possible that the changing scenes on the screen may have facilitated some degree of attentional engagement, thereby unintentionally lowering baseline RSA across all participants and masking the effects of vulnerability or current symptoms.

Concurrently, results from the full sample analysis did not indicate any associations between task RSA and recovery RSA and depressive symptoms. However, in the non-medicated subsample, task RSA was marginally significant, such that individuals reporting greater current symptoms evidenced greater withdrawal to the PASAT-C, over and above the effect of baseline RSA, which remained non-significant. Prospectively, I did not find any associations between task RSA and T2 symptoms, either in the full sample or in the non-medicated subsample. In light of the general lack of research on RSA reactivity and depression, as well as the relatively small size of the overall sample and the non-medicated sub-group ($n = 69$), these data may be difficult to interpret. Theory suggests that attentional engagement is adaptive when one attempts to meet the demands of a given task (Mulder & Mulder, 1981; Porges, 1995); therefore, I expected to see some degree of withdrawal (e.g., lower task RSA) across all participants during the PASAT-C. However, I hypothesized that vulnerable individuals would show a blunted response due to decreased psychological flexibility. Rottenberg and colleagues (2007) reported that individuals with MDD actually showed increases in RSA during stress, while Taylor and

colleagues (2006) found no differences in RSA reactivity between MDD and non-MDD groups among older adults. Hughes and Stoney (2000) found greater RSA withdrawal among individuals with greater depressive symptoms. Therefore, at present, it appears there are no clear theoretically consistent patterns of RSA reactivity to stress that are associated with risk for depressive symptoms or with the disorder itself.

Given the discrepant findings on RSA reactivity, I hypothesized that differences would emerge at RSA recovery, such that participants reporting greater depressive symptoms at baseline and 8-week follow up would exhibit a protracted withdrawal period and have difficulty returning to baseline. Results of analyses using the full sample and non-medicated subsample were not significant; however, both findings trended in the expected direction, with lower recovery RSA associated with greater symptoms. While this trend should be interpreted cautiously, it is consistent with prior findings (Genzler et al., 2009; Rottenberg et al., 2007; 2003).

One potential reason for the inconsistent findings in the literature may be that most studies of RSA have examined indices independent of one another. Yaroslavsky, Rottenberg, and Kovacs (2013) noted null findings for main effects of RSA, but reported that high RSA and greater withdrawal during stress exerts protective effects against depression. Therefore, it is possible that various indices may either exacerbate or amplify one another and that examining joint contributions may be fruitful in lending clarity to the emergent literature on RSA and depression.

Rumination: Does Context Matter?

Rumination is one of the most empirically supported constructs in the field of mood disorders, with extant studies demonstrating associations between trait rumination and

depression across the lifespan (e.g., Nolen-Hoeksema, 2000; Sarin et al., 2005; Spasojević & Alloy, 2001). In particular, rumination is considered both a vulnerability and maintenance factor in depression. By definition, this cognitive process is characterized by a failure to disengage from heightened negative self-focused attention, which in turn impairs effective mood repair.

Although rumination is widely considered to be a persistent maladaptive cognitive emotion regulation strategy, this study is first known investigation to empirically examine the time course of rumination and its associations with depression. As expected, trait rumination was strongly associated with concurrent symptoms; however, the relationship did not hold prospectively. As hypothesized, findings indicated that rumination about the PASAT-C immediately following negative performance feedback (task rumination) was related to concurrent depressive symptoms above and beyond the contribution of trait rumination. Surprisingly, persistent rumination (recovery rumination) was not associated with symptoms; in fact, trait rumination emerged as the strongest predictor in the concurrent recovery model.

Contrary to theory, trait rumination did not prospectively predict depressive symptoms at the 8-week follow up. Additionally, neither task rumination nor recovery rumination prospectively predicted depressive symptoms eight weeks later. $T1_a$ symptoms remained the only significant predictor across both the reactivity and recovery models. When examined in conjunction with life stress exposure, task rumination showed a marginal trend as a predictor of T2 symptoms. As expected, participants who reported greater task rumination and more stressful life events were more likely to report greater symptoms at follow up. Individuals reporting high reactivity but low stress tended to report lower symptoms. Finally, the recovery moderation analyses did not hold prospectively.

The results summarized above lend partial support to the response styles theory and add to the emerging body of research examining state rather than trait rumination. Although the hypothesis that reactivity would be associated with symptoms was generally supported, it requires consideration in conjunction with the recovery findings. My cognitive hypotheses emerged from recent literature examining attention biases in depression (see Joormann & D'Avanzato, 2010 for a review). These studies suggest that depressed individuals, as well as those who are vulnerable to becoming depressed, do not necessarily initially engage more with negative stimuli; rather, they have difficulty disengaging their attention once it is captured. In line with this research, I proposed that initial self-focused attention is not necessarily maladaptive and may represent a normative self-regulatory attempt to resolve discrepancies between reality and expectation (Pyszczynski & Greenberg, 1987). For example, I expected most participants to examine their performance and low false score on the PASAT-C, as it may have deviated from what they anticipated. I expected vulnerable individuals to initially ruminate more, but to also have difficulty disengaging their attention from their experience with the PASAT-C; however, this recovery hypothesis was not supported.

There are several possibilities for the mixed findings. First, the results may have been influenced by my chosen assessments. I utilized the PANE (Mezulis et al., 2002) as a measure of trait rumination and modified it to a 5-item event-specific state version (EA-PANE; Mezulis & Rudolph, 2012) for the laboratory visit. Although the PANE is advantageous for its use of specific hypothetical scenarios across several domains and is easily adaptable for state use, it presents the same five items of potential ruminative responses for each scenario; therefore, it may not capture the full range of potential ruminative responses individuals may engage in following stress.

Additionally, it is possible the EA-PANE may actually measure multiple components of rumination that have demonstrated some differential associations with depression. The first component is termed *brooding*, which is characterized by focusing one's attention passively and judgmentally on negative, self-blaming, or gloomy thoughts. The second component, *reflection,* is defined as contemplative pondering with an intentional focus on problem solving (Treynor et al., 2003). While research on the maladaptive effects of pondering is somewhat mixed, brooding is consistently identified as being associated with depression (see Nolen-Hoeksema et al., 2008 for a review). With regard to the EA-PANE, the measure was intentionally worded in a neutral manner to mask the purpose of the questionnaire. However, it is possible that participants who were engaged in pondering could have endorsed items that were originally intended to assess brooding. For example, one could endorse "I keep thinking about what I could have done differently" in an attempt to characterize active engagement in adaptive problem solving about their performance on the PASAT-C. No prior research has examined whether the PANE is associated with other measures of brooding and reflection; however, its limited face validity may help explain the mixed pattern of findings in the current study.

Prior studies have assessed subjective distress and peripheral physiological responding following the PASAT-C (Feldner et al., 2006; Holdwick & Wingenfeld, 1999; Lejunez et al., 2003). This investigation is the first to examine ruminative responses elicited from the task. It is possible that contextual factors influenced participants' responses. Cognitive appraisal theory (Lazarus & Folkman, 1984) posits that our interpretation or appraisal of an event influences responding, particularly the use of cognitive emotion regulation strategies such as rumination or reappraisal (Joormann & Siemer, 2011). Prominent dimensions of appraisals include importance, expectedness, degree of control, and responsibility (Siemer, Mauss, & Gross, 2007).

As in many laboratory-based paradigms, participants may perceive the situation as artificial and make appraisals accordingly. For example, participants were compensated with course credit for participating in the $T1_b$ laboratory visit and were informed at the beginning of the visit they could stop any time without penalty. Therefore, it is possible a participant may have appraised the PASAT-C as having little personal importance, which turn could lead to less rumination about performance. However, the same participant may actually tend to habitually ruminate in response to genuine stressors such as exams or difficult interpersonal situations. Further, rumination is strongly influenced by current mood (Nolen-Hoeksema, 2000). Individuals endorsing greater levels of depressive symptoms may be at an increased likelihood of reporting rumination; this may account for the concurrent relationship between ruminative responding and symptoms.

Rumination has also been conceptualized as a mechanism through which other vulnerabilities to depression (e.g., negative cognitive style) may emerge (Spasojević & Alloy, 2001). It has also been suggested that rumination exacerbates the effects of maladaptive cognitive appraisals and inferences (Robinson & Alloy, 2003). While it is not clear whether rumination functions as a mediator or moderator of the relationship between other vulnerabilities and depression, it is becoming increasingly clear that in the absence of cognitive "fuel", rumination may simply take the form of reflective pondering, which is less likely to contribute to maintenance of symptoms. In line with Hyde and colleagues' integrative ABC model of depression (2008), it may be most useful to investigate the effects of rumination and negative cognitions jointly. Examining the time course of both maladaptive content and process may better clarify the nature of their associations with one another and further contribute to understanding of their proximal relationships with depression.

Clinical Implications

The current study's findings hold several implications for the treatment of young adults with depression. Cognitive-behavioral therapy (CBT; Beck, 1987) has shown to be particularly effective for reducing depressive symptoms (Butler, Chapman, Forman, & Beck, 2006). However, several studies have reported poorer post-treatment and follow up outcomes for some groups, particularly high ruminators (Jones, Siegle, & Thase, 2008). Additionally, research on other pre-treatment predictors of CBT effectiveness has been inconsistent (Hamilton & Dobson, 2002). Assessing the time course of individuals' stress responses may aid clinicians in identifying those who may be at highest risk for poor treatment outcomes by drawing attention to specific areas in which clients may become "stuck." For example, in the context of affective responding, it may not be immediately apparent to a clinician that a given client may struggle more with down-regulating persistent negative affect than with initial reactivity alone. This client may have difficulty fully engaging in challenging unhelpful thoughts and beliefs, which is at the heart of cognitive-behavioral therapy, without first addressing concerns around self-regulation.

Third-wave behavioral treatments such as acceptance and commitment therapy (ACT; Hayes, Strosahl, & Wilson, 1999) and dialectical behavioral therapy (DBT; Linehan, 1993) may be particularly effective for individuals experiencing difficulty with stress reactivity and recovery. Both treatments utilize mindfulness-based interventions, which facilitates down-regulation of affective, biological, and cognitive responses to stress though the practice of intentional and nonjudgemental awareness. This contrasts with the automaticity of persistent NA, rumination, and increased heart rate and also paves the way for the use of emotion regulation, distress tolerance, or cognitive diffusion skills that further promote adaptive stress

recovery. Indeed, prior research has shown mindfulness-based interventions to be effective in

reducing emotional reactivity (Hill & Updegraff, 2012), cardiac reactivity (Cambell, Labelle,

Bacon, Faris, & Carlson, 2012), and rumination (Hawley et al., 2014).

Limitations

Several limitations of the current study should be acknowledged. First, the sample size of

92 participants was relatively small. Although the final sample exceeded the size originally

proposed based on an a priori power analysis ($N = 77$), the complex analyses performed may

have lacked sufficient power to determine significant effects. For example, the prospective main

effect and moderation analyses each required up to six variables to be entered into the model.

Further, the homogenous nature of the sample precludes generalizability of results. Participants

were predominately Caucasian and female and were college students at a small Christian

university. Importantly, extant research has identified robust gender differences in depressive

vulnerabilities, particularly emotional reactivity and rumination (Hyde et al., 2008); the relatively

small sample size and underrepresentation of male participants prevented examination of this

important potential moderator.

Methodological considerations may have also contributed to the lack of support for

several of the hypotheses. It is possible that the PASAT-C did not serve as a sufficiently

stressful laboratory analogue to genuine life stress. While participants self-reported significant

changes in subjective NA from pre-to-post task, it is plausible the task did not elicit the same

degree of stress responding as actual negative life events that may precipitate the development of

depressive symptoms. Additionally, while I strove to include a social stress component in the

stress induction by keeping the researcher in the room while the participant completed the

PASAT-C, the primary element of stress was academic in nature. Interpersonal stress has been

shown to predict depression over non-interpersonal stress (Hammen, 1991); therefore, the generally non-social nature of the PASAT-C may have potentially precluded the emergence of greater stress responses.

Further, it is possible that demand characteristics may have influenced participant responding during the experiment. Repeated administrations of the state NA and rumination measures may have served as cues triggering suspicion about the intent of the experiment. I employed several measures to mask the purpose of the questions by including positive affect items on the PANAS and prompting participants to think about their PASAT-C performance, "regardless of whether you thought you did well or did poorly." However, repeated questioning of state affective and cognitive responses regarding performance may have exposed the intent of ascertaining whether participants had negative responses to the stressor. Additionally, it is possible participants detected the deceptive nature of the experiment during the task itself. The RSA baseline period may have primed participants to expect the PASAT-C was intended to induce stress. Further, participants may have detected deception upon being presented with negative performance feedback. I aimed to make the PASAT-C difficult enough that the feedback would be believable; however, during debriefing, several participants noted being surprised their score was either higher or lower than expected. While I considered using a post-experiment question assessing participant suspicion of deception, I elected not to include it prior to debriefing. Several prior studies have indicated that participants are often unwilling to disclose awareness in response to such inquiries and that they may be ineffective measures of suspicion (e.g., Blackhart, Brown, Clark, Pierce, & Shell, 2012; Nichols & Maner, 2008; Sagarin, Rhoads, & Cialdini, 1998).

Lastly, the 8-week follow up period may not have been sufficient to model clinically significant symptom changes. $T1_a$ and T2 symptoms were strongly correlated ($r = .52$); $T1_a$ symptoms remained significant in nearly all prospective models and accounted for a large proportion of variance in T2 symptoms. Longer follow up periods (e.g., six months or one year) may be better suited to capture symptom trajectories.

Future Directions for Research

The current study focused on three domains of stress and was intended to be an initial test of a recovery model of depressive vulnerability. However, it is important to acknowledge that extant theories (e.g., Hyde et al., 2008; Joormann & D'Avanzato, 2010) propose integrative models of vulnerability to depression. In other words, affective, biological, and cognitive responses to stress are not independent factors, but likely exhibit interactive and causal effects. For example, as noted above, rumination is influenced by current affective states and is also likely to maintain negative mood. Further, as RSA is thought to index emotional responding and regulation via attentional deployment (Thayer & Lane, 2000), it may serve as a psychophysiological correlate of both negative affect and rumination.

The current study method is novel for capturing the time course of state depressogenic responses to stress, particularly in the affective and cognitive domains. Time course studies may be well suited for examining integrative models of psychopathology and for understanding emotion regulation processes across disorders. Such studies could capture whether NA during a laboratory stressor predicts RSA and use of emotion regulation strategies, and whether these factors influence later affective and cognitive recovery from the stressor. In addition to laboratory stress induction paradigms, diary and ecological momentary assessment (EMA; Bolger, Davis, & Rafaeli, 2003) studies are well suited to further elucidate questions regarding

the time course of affective and cognitive responses to genuine life stressors. In particular, EMA allows for the in-vivo assessment of affect and cognitive processes associated with a given context. Although valid assessment of parasympathetic contributions to emotional processing may be difficult to capture outside the laboratory, pairing both experimental and diary methods within the same study is likely to extend understanding of how proximal vulnerabilities convey risk for symptoms and how correlates of MDD function from moment-to-moment to either promote or impair recovery from stress.

The current study examined depressive vulnerability from a vulnerability-stress framework, examining the contribution of life stress exposure as an amplifier of vulnerability. In addition to life stress, extant research supports relationships between the current study's variables of interest and other moderator variables. For example, attentional biases and cognitive control deficits may hinder the selection of adaptive emotion regulation strategies that facilitate mood repair (Joormann, 2009) and should be considered as potential moderators when examining recovery from stress. In addition to solely examining life stress exposure as a contextual amplifier of underlying vulnerability, future research may benefit from examining the role of potential vulnerability buffers, such as social support (Coyne & Downey, 1991). Establishing and testing an integrative model of factors that prolong or dampen momentary distress may contribute to our understanding of how depression develops and is maintained. This in turn may facilitate increasing use of interventions that specifically target maladaptive patterns of stress recovery.

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