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The Effects of Rumination on Psychological and Biological Recovery from Stress in Depression

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THE EFFECTS OF RUMINATION ON PSYCHOLOGICAL AND BIOLOGICAL RECOVERY FROM STRESS IN DEPRESSION

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Diathesis-stress models of depression highlight that stress triggers the onset of a depressive episode. Increasing evidence, however, suggest that increased risk comes not from the initial response to stress, but rather from difficulty regulating emotions in a way that facilitates recovery. Rumination is a maladaptive emotion regulation strategy shown to prolong negative affect in response to distress. The current study extends past research by comparing the effects of a rumination versus distraction induction on biological and psychological recovery from stress among individuals with major depressive disorder (MDD) and healthy controls (CTLs). Participants were exposed to a psychosocial stressor and then randomly assigned to either the rumination or distraction condition. Self-reported affect and markers of biological arousal (salivary cortisol and respiratory sinus arrhythmia; RSA) were assessed before, during, and after the stressor. Participants demonstrated significant reactivity to stress, evidenced by an increase in self-reported negative affect and decrease in RSA. In contrast, participants did not demonstrate the expected significant cortisol reactivity to stress. Also unexpected was a significant increase in RSA during the latter-half of the stressor, suggesting spontaneous parasympathetic recovery. Although no group differences were seen in stress reactivity, significant time by group by condition interactions were seen in stress recovery. Within the CTL group, participants in the rumination and distraction conditions did not differ in

their psychological or biological recovery from stress. Within the MDD group, however, participants in the rumination condition demonstrated higher negative affect and salivary cortisol during the recovery period than participants in the distraction condition. Evidence therefore suggests that rumination affects neuroendocrine and psychological recovery from stress in depression. Moreover, depressed participants in the distraction condition demonstrated significantly greater RSA withdrawal during the ER induction compared to control participants in the distraction condition, potentially suggesting that distraction was more effortful for the MDD versus CTL group. During the subsequent nature video, RSA recovery was greater in the MDD versus CTL group. Results from this study provide important insights into the effect of rumination on psychological and biological recovery from stress in MDD.

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Chapter 1: Introduction

Major depressive disorder (MDD) is among the most prevalent of all psychiatric disorders (Kessler & Zhao, 1999). It is estimated that 16% of the general population will experience clinically significant depression at some point in their lives (Kessler et al., 2003). The personal and societal costs of MDD are substantial: it is cited as the number one cause of disability worldwide (Lopez & Murray, 1998). Depressive disorders place a burden of over 40 billion dollars per year on the American economy and account for over 20% of costs for all mental illness (Stewart, Ricci, Chee, Hahn, & Morganstein, 2003). A diagnosis of depression also places individuals at increased risk for poor health outcomes, including increased risk of a cardiac event and faster progression of illnesses (Carney, Freedland, Rich, & Jaffe, 1995; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Leserman et al., 1999). The high prevalence and substantial costs of this disorder underscore the importance of identifying factors involved in the onset and maintenance of depressive episodes.

According to the DSM-IV-TR, a diagnosis of a major depressive episode is given when five out of nine depressive symptoms are present, at least one of which must include either loss of interest in pleasurable activities (anhedonia) or depressed mood (APA, 2000). Other symptoms can include sleep changes, appetite changes, psychomotor agitation or retardation, decreased energy, feelings of worthlessness or excessive guilt, difficulty making decisions or concentrating, and suicidal ideations. Symptoms must be present for at least two weeks and must cause marked impairment or distress. The diagnostic criteria show that sustained negative affect and reduced positive affect are the hallmark features of a depressive episode. Thus, depression, at its core, is a disorder of

emotion dysregulation. However, given that emotions are "whole-body phenomena" (Mauss et al., 2005), it is not surprising that depression is characterized not only by sustained subjective feelings of sad mood but also by physiological complaints such as muscle aches and pains, headaches, or indigestion (e.g., Mathew, Weinman, & Mirabi, 1981). Furthermore, people diagnosed with depression exhibit dysregulation in physiological and neuroendocrine responses to stress (e.g., Burke, Davis, Otte, & Mohr, 2005; Rottenberg, 2007). Given the physiological changes associated with a major depressive episode, it is important to pay closer attention to the relation between psychological and biological factors to better understand risk for the onset and maintenance of MDD (Fabes & Eisenberg, 1997).

Diathesis-stress models, which emphasize the role of stress in triggering the onset of a major depressive episode, are a particularly promising conceptualization of depression that allows for such an integration of biological and psychological factors (e.g., Flynn & Rudolph, 2007; Hammen, 2005; Monroe & Flynn, 1991). Indeed, numerous studies have demonstrated increased instances and recurrences of depression following a major life event (e.g., Monroe & Roberts, 1990; Monroe & Hidjiyannakis, 2002), chronic stress (e.g., Brown & Harris, 1986), and daily hassles (e.g., Lazarus & Folkman, 1984). Furthermore, prolonged biological responses to stress (measured via neuroendocrine and physiological processes) have been found amongst currently depressed individuals (e.g., Parker, Schatzberg, & Lyons, 2003) and those who are at risk for depression (e.g., Firk & Markus, 2009). Thus it seems clear that stressors play an important role in risk for depression and should be assessed when examining risk for the onset and maintenance of MDD.

It is also important to note, however, that not all people, even if exposed to severe stressors, experience a major depressive episode following a stressful event (e.g., Bonnanno, 2004). Thus experiencing a stressor is not sufficient for the onset of a depressive episode. Instead, diathesis-stress models propose a pre-existing vulnerability that affects people's responses to acute stressors and thereby increases their risk of developing a depressive episode (see Abramson et al., 2002, for a review). Cognitive theories of depression, for example, propose that negative cognitions and biased processing of emotional information play an important role in our understanding of the etiology and maintenance of MDD by serving as a pre-existing diathesis (e.g., Beck, 1976; Bower, 1981, 1987; Teasdale, 1988). Perhaps most prominently, Beck (1967, 1976) formulated a theory of depression that ascribes the onset and maintenance of this disorder to biases in how people process information following stress: dysfunctional schemas that were formed early in life are hypothesized to be activated by an acute stressor leading to mood-congruent biases in attention, memory and other cognitive processes. Biases in the way individuals attend to, interpret, and remember negative information contributes to depression risk. Studies examining Beck's theory, however, have yielded mixed support (see Mathews & MacLeod, 2005, for a review). Furthermore, given that mood-congruent biases are initially found not only in depressed people but also in non-depressed people after negative mood inductions or stressors, Beck's theory does not fully explain why only some individuals go on to experience sustained negative affect (i.e., a major depressive episode).

These gaps have led researchers to suggest that risk for depression comes not from individuals' initial response to stress, but rather from difficulties regulating the

subsequent emotional state (e.g., Flynn & Rudolph, 2007; Joormann, Yoon, & Siemer, in press; Nolen-Hoeksema, 1991, Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Thus, an important risk factor for depression is the tendency to regulate emotions in such a way as to preclude recovery from stress. Thus far, few studies have focused on differentiating stress reactivity from recovery. Those that have made that differentiation, tend to focus on the effect of emotion regulation strategies on mood or on psychological responses to stress, without incorporating biological measures. Important advances can come from integrating psychological and biological indices of stress, as doing so is likely to provide a more accurate and comprehensive understanding of the mechanisms that increase risk for depression following stress. I propose to extend past research by examining whether different emotion regulation strategies affect both psychological and biological responses to stress. Before discussing the biological stress response, I first will look at the concept of emotion regulation and the way different emotion regulation strategies affect one's mood.

Emotion Regulation (ER) in Depression

The construct of emotion regulation (ER), which evolved from the broader concept of coping, involves utilizing behavioral and cognitive strategies to modulate the intensity, duration, and expression of affect (Thompson, 1994). Emotions are regulated for a variety of reasons; ER may be used to enhance desirable feelings (Larson, 2000), maintain social norms, and foster social communication (Fischer, Manstead, Evers, Timmer, & Valk, 2004). The most prominent models of ER indicate that individuals can regulate their emotions at a variety of different points during the emotion-generation process (Gross, 1998, 2001). Some strategies are *antecedent-focused* because they are

used even before the onset of the emotion or physiological response (examples include situation selection, situation modification, and attentional deployment). In contrast, other strategies are *response-focused* because they are used after an emotion or physiological response has begun (examples include distraction and rumination).

Recent theories of depression emphasize that increased risk comes from the use of strategies that fail to down-regulate negative emotions after their initial onset, specifically response-focused strategies (e.g., Joormann et al., in press; Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008), which lead to prolonged negative affect (John & Gross, 2004). In response to stress, the use of effective ER strategies (e.g., distraction) is typically characteristic of healthy psychological functioning (Gross, 1998, 1999), whereas the use of ineffective ER strategies (e.g., rumination) has been more commonly associated with multiple psychological disorders including depression, schizophrenia, and borderline personality disorder (Kring & Werner, 2004). Understanding individual differences in emotion regulation is becoming increasingly important as a psychological vulnerability factor that affects individuals' ability to recover from stress. Although the majority of ER studies focus on reappraisal and suppression, within the depression literature the response-focused strategies of distraction and rumination have received most of the attention.

Distraction. Distraction involves engaging in positive or neutral activities in order to divert one's thoughts from symptoms of distress and depression (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; Lyubormirsky & Nolen-Hoeksema, 1995). For example, one may lower the level of subjective emotion experienced in a situation by focusing on thoughts unrelated to the situation (e.g., planning a supermarket shopping

list). It is important to differentiate attentional distraction, which is considered an antecedent-focused strategy, from cognitive distraction, which is categorized as a response-focused strategy. Attentional distraction involves shifting one's visual or auditory attention away from the emotion-producing event in order to avoid the experience of emotion. In contrast, cognitive distraction, which is the primary focus of the current study (from here on referred to as only *distraction*), involves shifting one's thoughts away from the emotion or emotion-producing event after the emotion has already been experienced. Distraction has been associated with adaptive outcomes such as faster physiological recovery from stress (Vickers, Vogeltanz-Holm, 2003), decreased depressed mood (Trask & Sigmon, 1999), and shorter durations of depressive symptoms (Nolen-Hoeksema, Morrow, & Fredrickson, 1993).

A critical factor in the effectiveness of distraction to alleviate negative mood is the availability of distracting thoughts or activities (e.g., see review by Kobe, 2009). Simply instructing individuals to "not think" about their emotional state has shown paradoxically to increase the experience of that emotion (Wegner , Erber,& Zanako, 1993; Wegner & Gold, 1995). In contrast, individuals asked to think about a specific yet emotionally unrelated item experienced diminished emotional state (e.g., Wegner, 1994). These findings underscore the importance of providing individuals with specific, distracting items if the effectiveness of distraction is to be ensured.

Given that any demanding task can distract individuals' thoughts away from their emotional state, even neutral tasks serve to help regulate negative emotions (e.g., Erber & Tesser, 1992). Indeed, studies have shown that distraction with neutral materials can reduce depressed (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow,

1993) or angry mood (Gerin, Davidson, Goyal, Christenfeld, & Schwartz, 2006; Rusting & Nolen-Hoeksema, 1998). For example, depressed participants who were asked to focus on descriptions of geographic objects and locations experienced reductions in negative mood compared to those who focused on their current emotion (Nolen-Hoeksema & Morrow, 1993). Together, these findings suggest that providing specific, neutral or positive, activities or thoughts can take one's mind off of the current negative emotions and alleviate negative mood (however, see Campbell-Sils & Barlow, 2007, and Kross, Ayduk, & Mischel, 2005, which point out that persistent distraction serves as an ineffective long-term ER strategy because it hinders effective problem solving).

Rumination. In contrast to distraction, rumination is a particularly maladaptive method of responding to a stressful event (Nolen-Hoeksema et al., 2008). Rumination is a means of responding to distress by repetitively and passively focusing on symptoms of distress and the potential causes or consequences of these symptoms (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008). This ER strategy is a particularly salient risk factor for depression because it exacerbates and prolongs depressed mood (see review by Nolen-Hoeksema et al., 2008; however, note that rumination designed to savor positive emotions has been associated with more adaptive outcomes; e.g., Bryant, 2003; Wood, Heimpel, & Michela, 2003). Empirical evidence has shown that people who tend to engage in rumination when distressed are more likely to develop depressive disorders and tend to experience more prolonged periods of depression (e.g., Just & Alloy, 1997; Nolan, Roberts, & Gotlib, 1998; Nolen-Hoeksema, 2000).

The most widely used measure of self-reported trait rumination is the Ruminative Responses Scale of the Response Styles Questionnaire (RSQ; Nolen-Hoeksema &

Morrow, 1991). Considerable evidence has linked higher trait rumination with the onset and maintenance of depression. Nolen-Hoeksema (2000), for example, examined a sample of approximately 1,300 adults randomly selected from the community. Among non-depressed individuals, rumination scores at first assessment predicted the onset of new major depressive episodes over the following year. In addition, longitudinal studies have shown that individuals who ruminate report higher levels of depressive symptoms, even after controlling for baseline depression levels (e.g., Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema , Parker, & Larson, 1994). For example, rumination scores measured two weeks before the 1989 Loma Prieta earthquake were found to predict depressive symptoms up to 10 weeks later, even after controlling for original levels of depression (Nolen-Hoeksema & Morrow, 1991). Similarly, Kuehner and Weber (1999) examined the effects of rumination on later depression among individuals who were recently discharged from an inpatient hospital. Post-discharge RSQ scores predicted levels of depression 4 months later, even after controlling for baseline depression. Self-reported trait rumination has thus been linked to both the onset and maintenance of depressive symptoms; however, questionnaire data provides only correlational evidence of a link between rumination and depression. In order to establish a causal link, rumination must be induced and manipulated; then one can examine the effects of this manipulation on other dependent variables.

Rumination and distraction inductions. Many studies have experimentally tested the effects of rumination using the emotion regulation (ER) induction procedure developed by Nolen-Hoeksema and Morrow (1993). In this widely used induction participants are instructed to focus on a series of phrases presented on cards (45 cards per

condition). Although participants are only allowed eight minutes to read and think about the cards, they are free to decide how long they focus on any one card during the allotted time. In the rumination condition, participants are asked to focus on their life and current feelings for eight minutes using emotionally neutral prompts (e.g., "Think about the kind of person you are"). This is typically contrasted with a distraction induction, in which participants are instructed to focus on external ideas for eight minutes (e.g., "Think about the layout of your local shopping center").

Numerous studies have shown that Nolen-Hoeksema's ER induction (Nolen-Hoeksema & Morrow, 1993) influences dysphoric individuals' thoughts, problem solving abilities, motivation, concentration, levels of stress, social relationships (e.g., see Lyubomirsky, & Tkach, 2003, for a review), and most relevant to the current study, their mood (Lyubomirsky, et al., 1998; Lyubomirsky, Tucker, Caldwell, & Berg, 1999; Nolen-Hoeksema & Morrow, 1993; Papageorgiou & Wells, 2000). For example, using the ER induction procedure with prompts presented on a slide projector, Vickers & Vogeltanz-Holm (2003) examined whether the rumination versus distraction induction differentially impacted dysphoric and non-dysphoric participants' negative mood as assessed by self-reported sadness and depression ratings (made on a scale of 1 = *not sad/depressed* to 10 = *very sad/depressed*). Results showed that dysphoric individuals assigned to the rumination condition reported significantly higher levels of post-rumination negative mood than did dysphoric distracters. In contrast, healthy controls, who remained in a neutral mood state, were not affected by the rumination/distraction induction (as is typically found; Lyubomirsky et al., 1998; 1999). Thus, rumination, in the context of dysphoria prolongs depressed mood.

Adding to the studies described above, many individuals have coupled the ER induction (Nolen-Hoeksema & Morrow, 1993) with a preceding negative mood induction. Interestingly, not only dysphoric individuals, but also healthy controls placed in a negative mood state experienced prolonged negative affect if assigned to ruminate compared to if assigned to distract (e.g., Broderick, 2005; Bushman et al., 2005; Kuehner, Huffziger, & Liebsch, 2009; Morrow & Nolen-Hoeksema, 1990). Negative mood states have been induced by instructing participants to watch sad film clips (e.g., Ciesla & Roberts, 2007) or engage in cognitive stress tasks (e.g., paced serial auditory addition task; Feldner, Leen-Feldner, Zvolensky, & Lejuez, 2006). For example, Morrow and Nolen-Hoeksema (1990) induced a negative mood by asking non-dysphoric participants to read a depressing story while listening to sad music and to imagine that the events in the story were happening to them. Following the mood induction, participants completed the ER induction. As expected, those in the rumination condition experienced prolonged self-reported sadness (as indicated via a 9-point Likert scale) compared to those in the distraction condition. When combined with a negative mood induction, the ER induction has also been shown to affect the cortisol production, physiological levels, cognitive processes, and autobiographical memory of non-dysphoric participants (e.g., Cui & Huang, 2007; Denson, Fabiansson, Creswell, & Pedersen, 2009). Together these studies provide strong evidence that rumination in the context of either a naturally occurring or experimentally induced depressed mood maintains dysphoria, enhances negative thinking, and impairs problem solving.

Rather than relying on only dysphoric participants, recent studies have increasingly focused on individuals with MDD in order to determine whether rumination

has similar effects on those with clinical levels of depression. Such studies have shown that rumination does indeed lead to sustained negative mood, increased negative cognitions, increased overgeneral autobiographical memory, and decreased effective problem solving in depressed participants (e.g., Donaldson & Lam, 2004; Park, Goodyer, & Teasdale, 2004; Watkins & Moulds, 2005; Watkins, Teasdale, & Williams, 2000). For example, Donaldson and Lam (2004) examined the detrimental effects of rumination on negative mood and problem solving abilities in a group of depressed participants. Depressed and healthy control participants were randomly assigned to either the rumination or distraction condition of the ER induction and then the effects on mood and problem solving abilities were examined. Depressed participants who were assigned to ruminate experienced increased negative affect following the induction, and they gave poorer solutions to social problems. In contrast, depressed participants who were assigned to distract experienced decreased negative affect over time, and they generated more effective solutions to problems.

From the studies presented above, it is clear that those in a negative mood state, be it a current depressive episode (e.g., Donaldson & Lam, 2004; Park et al., 2004), subclinical dysphoria (e.g., Lyubomirsky et al., 1999; Nolen-Hoeksema & Morrow, 1993), or healthy controls who have been exposed to a negative mood induction (e.g., Morrow & Nolen-Hoeksema, 1900) are negatively impacted by rumination. Specifically, ruminating in response to negative affect perpetuates negative mood and leads to cognitive and social impairments (e.g., see review by Lyubomirsky & Tkach, 2003). Until recently, research has focused on the affective, behavioral, and cognitive consequences of rumination, and the biological consequences of rumination have been

largely unexamined. Important advances in our understanding of the link between stress and MDD onset can come from investigating how rumination influences biological responses to stress in depression. The current study proposes to fill this gap in the literature by extending our knowledge of biological reactivity and recovery from stress.

Biological Stress Response in Depression

Although the hallmark of MDD is sustained negative affect, research clearly indicates biological changes in response to stress associated with depression (see Carney, Freedland, & Veith, 2005, for a review). Before discussing the way ER strategies might influence these biological changes, I first present an overview of what is known about biological responses to stress in depression. MDD is associated with dysregulation in two biological systems that are critically involved in stress responding: the neuroendocrine system and the autonomic nervous system (e.g., see reviews conducted by Burke et al., 2005, and Carney et al., 2005). Activation of these two interconnected systems is central to humans' biological stress response; they prepare our body for action and help us recover once the threat has dissipated (Patchev & Patchev, 2006).

Neuroendocrine functioning. A central component of the neuroendocrine system is the hypothalamic-pituitary-adrenal axis (HPA axis). The interactions between the hypothalamus, pituitary gland, and adrenal gland play a central role in regulating the mammalian stress response (see Smith & Vale, 2006, for a review). Once the hypothalamus releases corticotrophin-releasing-hormone (CRH), it is transported through the blood vessels to the pituitary gland and triggers the release of adrenocorticotropic hormone (ACTH). ACTH is then carried by the blood to the adrenal gland, where it stimulates the biosynthesis of cortisol, a glucocorticoid hormone. The HPA axis is

critically involved in stress responses by activating physiological changes – increased heart rate, blood pressure, and respiration – that prepare people to cope with acute stress (Gunnar, Connors, & Isensee, 1989).

The primary stress hormone is cortisol. Cortisol affects many aspects of the body, such as metabolism and immune functioning. It is released spontaneously throughout the day and in response to stress (Kirschbaum & Hellhammer, 1994). Spontaneous cortisol secretion is affected by diurnal fluctuations. In healthy individuals, cortisol levels reach a peak within 30 to 45 minutes after waking and then gradually decline throughout the day until late afternoon, when the decline is interrupted by a brief spike. Stressful life events produce elevated levels of cortisol over and above the diurnal fluctuations (Dinan, 1996). A recent meta-analysis reported that peak cortisol response occurs 21 to 40 minutes following the onset of a stressor, and complete recovery to baseline values occurs within 41 to 60 minutes after stressor offset (Dickerson & Kemeny, 2004). Cortisol recovery is facilitated by negative feedback loops. Receptors on the hypothalamus and pituitary, as well as glucocorticoid and mineralcorticoid receptors in the hippocampus, identify elevated levels of cortisol and signal the HPA axis to stop cortisol production. Although an elevation in cortisol levels following stress is expected in all individuals, attenuated negative feedback can lead to poor inhibition of CRH and ACTH, resulting in chronically elevated levels of cortisol. Chronic cortisol elevation can disrupt individuals' ability to regulate emotions and cope effectively with stress (e.g., see McEwen, 2006 for a review).

Given that the functioning of the HPA system is so integrally related to the human stress response, it is not surprising that atypical patterns of both basal cortisol functioning and cortisol reactivity have been documented in disorders typified by dysfunctional

responses to stress such as MDD (e.g., Carroll et al., 2007; Gillespie & Nemeroff, 2005). In a meta-analysis examining cortisol patterns in depression, depressed individuals were shown to have lower cortisol levels upon wakening and 30 minutes thereafter (Burke et al., 2005). In contrast, depressed individuals exhibited elevated cortisol levels in the afternoon.

Even more applicable is the fact that studies examining cortisol levels in response to an acute stressor also reported differences between depressed and non-depressed participants (e.g., Young et al., 2000); however, the reported direction of these findings have been inconsistent. For example, whereas several studies reported a hyperactive cortisol response to stress in depressed compared to control participants (e.g., Heim et al., 2000), others reported a blunted cortisol stress response in depressed participants (e.g., Young et al., 2000). In their meta-analysis, depressed individuals exhibited a blunted response to stress (Burke et al., 2005); however, this was based on only seven studies, and these studies reported markedly different results. Importantly, several factors differentiated studies demonstrating hyperactive versus blunted stress response. For one, time of day: blunted stress reactivity in depressed participants was more likely in afternoon compared to the morning studies. Second, baseline cortisol levels: the higher the cortisol levels at baseline, the less cortisol was produced after the stressor. Third, demographic characteristics: blunted cortisol stress reactivity was most pronounced in more severely depressed participants. In contrast, several variables were found to have no influence on the association between depression and cortisol stress reactivity: diagnosis of comorbid post-traumatic stress disorder, sex, or the use of non-endocrine medication.

Interestingly, those at genetic risk for depression also show altered cortisol responses to stress (see Ising & Holsboer, 2006, for a review).

In contrast to the conflicting evidence regarding stress reactivity, studies consistently reported higher cortisol levels during the recovery period among depressed compared to control participants (see the meta-analysis by Burke et al., 2005). Thus, although it is unclear if depressed individuals' initial cortisol response is blunted or exaggerated, they clearly have trouble regulating post-stress arousal compared to non-depressed individuals. Prolonged post-stress recovery was most pronounced in older and more severely depressed participants.

Physiological functioning. Although less studied, preliminary evidence exists for a parallel dysregulation of the autonomic nervous system (ANS) in MDD (e.g., see Rottenberg, 2007, for a review). The ANS is the part of the peripheral nervous system, which involuntarily controls heart rate, respiration rate, digestion, salivation, and perspiration (Brownley, Hurwitz, & Schneiderman, 2000). It is comprised of the sympathetic and parasympathetic nervous systems. Both mental and emotional states, such as stress, lead to changes in the ANS. Whereas the sympathetic nervous system mediates the neuronal and hormonal stress response that primes the body for action, the parasympathetic nervous system returns the body to homeostasis after stressful events by inhibiting the sympathetic influences to the heart and dampening the HPA axis (Lovallo & Thomas, 2000; Rottenberg, 2007).

Of particular importance to examining the ability to recover from stress is the activation of the parasympathetic nervous system, which plays a large role in physiological recovery from stress, and thus is an important index of stress recovery

(Lovallo & Thomas, 2000). Historically, parasympathetic activity has been indexed through heart rate or general heart rate variability; however, these measures do not provide an accurate estimate of vagal control because they do not exist independent of the effects of the sympathetic nervous system (see Brownley et al., 2000, and Rottenberg, 2007, for a review). More recently, however, heart rate variation during paced respiration has been identified as an accurate yet noninvasive index of parasympathetic nervous system activity (e.g., Grossman, Stemmler, & Meinhardt, 1990; Stemmler, Grossman, Schmid, & Foerster, 1991). Using spectral analysis (a mathematical procedure to decompose autonomic mediators of heart rate variability at specific frequency components; see Berntson et al., 1997, for a review), heart rate variation occurring in different respiratory frequency bands can be identified (e.g., see Brownley et al., 2000 for an overview). If respiration frequency drops within the low- to mid-frequency band (< 0.12Hz), than the resulting output may include both sympathetic and parasympathetic input, and would provide a confounded measure of parasympathetic activity. In contrast, if respiration frequency remains within the high-frequency band (0.14 – .4 Hz), it is considered a sensitive index of pure parasympathetic nervous system activity (Berntson et al., 1997). This beat-to-beat variability in the timing of heart beats within the high-frequency band is referred to as respiratory sinus arrhythmia (RSA) and will be the focus of the current study.

Research on RSA in psychopathology has focused on (1) RSA level, often measured in a resting state, and (2) RSA fluctuations, often measured in response to stress. Both quantify the influence of vagal nerve activity on oscillations in heart rate and are involved in maintaining homeostasis in response to environmental demand (Brownley

et al., 2000). At rest the parasympathetic nervous system reduces energy expenditure (e.g., by inhibiting the sympathetic innervations of the heart and by dampening the HPA axis). In healthy individuals, RSA is therefore highest during unchallenged situations. However, when environmental demands become more challenging, such as in a time of stress, this inhibition needs to be actively and rapidly withdrawn in order to meet environmental demands.

Research has found that resting RSA level provides a baseline indicator of one's ability to control energy expenditure and thus provides an indirect indicator of one's tendency to react to the environment (see Beauchaine, 2001, for a review) or one's temperament (Izard et al., 1991). Individuals with high RSA levels are found to be more physiologically flexible, less hostile, and less conflict-prone, all of which that promotes socio-emotional competence (Beauchaine, 2001; Solomon, 2000). In contrast, low RSA levels have been associated with a range of psychopathologies such as hostility (Brosschot & Thayer, 1998; Demaree & Everhart, 2004; Sloan et al., 2001), anxiety (e.g., Cohen et al., 2000; Thayer, Friedman, & Borkovec, 1996; Watkins, Grossman, Krishnan, & Sherwood, 1998) impulse control (Beauchaine, 2001), and depression (Carney et al., 2000; Rechlin, Weis, Spitzer, & Kaschka, 1994; Rottenberg, 2007; Vaccarino et al., 2008).

The empirical literature on the parasympathetic nervous system and MDD has focused predominantly on RSA level, but has yielded mixed results (e.g., see Rottenberg, 2007). Although some studies report lower RSA levels in depressed compared to control participants (Dalack & Roose, 1990; Lehofer et al., 1999; Rottenberg, Clift, Bolden, & Salomon, 2007), others report no differences in RSA levels (e.g., Lehofer et al., 1997;

Moser et al., 1998). A recent meta-analysis conducted by Rottenberg (2007) focused on medically healthy individuals and found only a small-to-medium effect size (d = .33) indicating that depression was associated with lower levels of resting RSA. Reviews indicate several important mediators of the relation between RSA levels and depression. For one, medications, and in particular tricyclic antidepressants, have been found to suppress RSA in both depressed and other patient groups (e.g., Rechlin, 1994; McLeod, Hoehn-Saric, Porges, & Zimmerli, 1992). Depressed patients taking tricyclics had lower RSA levels compared to both unmedicated depressed patients and healthy controls (e.g., Lehofer et al., 1997). In addition, comorbid conditions such as anxiety disorders (Friedman & Thayer, 1997; Watkins et al., 1998) and physical illnesses such as cardiovascular disease (Dekker et al., 2000) have been identified as possible confounds associated with lower RSA levels.

The previous focus on RSA level as a proxy for RSA fluctuations is unfortunate given that the two constructs appear empirically distinct (correlations generally range from .4 to .6; Donzella, Gunnar, Krueger, & Alwin, 2000; Movius & Allen, 2005) and that RSA fluctuation has been identified as a more direct marker of biological stress reactivity and recovery (e.g., Brownley et al., 2000; Rottenberg, Wilhelm, Gross, & Gotlib, 2003). Decreases in RSA (or RSA withdrawal) are expected when we encounter stress and need to initiate a fight-or-flight response. Empirical evidence on RSA fluctuations in non-clinical individuals has shown that decreases in RSA are provoked by laboratory stressors such as cognitive challenges (Hansen, Johnsen, & Thayer, 2003; Houtveen, Rietveld, & De Geus, 2002) distress (Wilhelm & Roth, 1998), and arousal from film clips (regardless of valence; e.g., Frazier, Strauss, & Steinhauer, 2004).

Importantly, RSA is also affected by subsequent coping. Butler, Wilhelm and Gross (2006), for example, showed that non-clinical individuals who attempted to regulate their emotions following stress by suppressing or reappraising them showed larger increases in RSA than non-regulators.

In keeping with these findings, less RSA withdrawal has been associated with various forms of psychopathology, including depression (e.g., Rottenberg et al., 2007). Rottenberg et al. (2007), for example, reported that whereas non-depressed participants demonstrated the expected withdrawal of RSA in response to a stressful speech task, depressed participants' RSA *increased* during the speech task above baseline levels. Thus, depressed participants failed to show the expected decrease of the parasympathetic nervous system that would allow them to appropriately respond to stress. Similarly, a subset of depressed patients (those who cried) had less RSA reductions following a sad film clip than did control participants (Rottenberg et al., 2003). However, it is important to note inconsistent findings. For example, using a median split of Beck Depression Inventory (BDI) scores, dysphoric participants had greater RSA reductions in response to a stressful speech task than did non-dysphoric participants (Hughes & Stoney, 2000). In addition, null results have been reported in studies using medicated MDD patients (Straneva-Meuse et al., 2004) and patients with elevated cardiovascular risk (Taylor et al., 2006). Although these particular samples may confound measurement of RSA as discussed previously (e.g., Rottenberg, 2007), there is no convincing evidence to expect that depressed and control participants will differ in their RSA withdrawal to stress.

Although few studies have examined recovery (or reactivation) of RSA levels in MDD after stressor offset, Mezzacapa and colleagues emphasize that increases in RSA

are crucial for restoring biological homeostasis (Mezzacappa, Kelsey, Katkin, & Sloan, 2001). In a non-clinical sample, they found that prolonged RSA rebound during recovery from stress was associated with higher scores on standard risk factors for cardiovascular disease, which emphasizes the importance of looking specifically at this recovery period. In order to examine recovery following stress, Rottenberg and colleagues (Rottenberg et al., 2003) presented a sad film to depressed and control participants. As expected, control participants who cried exhibited increased RSA above baseline levels in the 90 seconds following crying, suggesting that an effective ER strategy such as crying, helps promote parasympathetic activation in healthy controls. In contrast, MDD participants (regardless of whether or not they cried) did not exhibit above-baseline increases in RSA, which was interpreted as evidence of a link between poor physiological recovery from stress and compromised ER in depression.

Taken together, these studies suggest important differences between depressed and non-depressed individuals' neuroendocrine and parasympathetic regulation in response to acute stress (see reviews by Burke et al., 2005, and Rottenberg et al., 2007). Specifically, in response to stress, MDD is occasionally associated with a blunted cortisol response and decreased RSA withdrawal, which suggests that depressed individuals exhibit less biological reactivity to acute stress than do controls. Although still tentative, initial evidence also exists for prolonged biological activation in response to stress as evidenced by prolonged cortisol secretion and decreased RSA re-engagement during the recovery period. It is important to note that this prolonged biological distress, which is associated with MDD, can be equated with the prolonged negative affect that is a hallmark of depression. Given this, researchers are beginning to question whether deficits

in ER, specifically rumination might underlie not only prolonged negative affect but also prolonged biological distress.

Rumination and Biological Response to Stress in Depression

Recent theories posit that rumination not only may play an important role in sustaining negative affect following exposure to stressors but also may be a critical mechanism underlying sustained biological reactivity to stress in depression (e.g., Brosschot, Gerin, & Thayer, 2006). Specifically, the continual processing or contemplation of stressful situations (i.e. rumination) is predicted to amplify or maintain biological arousal. Recent research testing this hypothesis has focused on the effects of rumination on the autonomic nervous system. These studies showed that ruminating in response to stress leads to prolonged physiological activity as measured by increased blood pressure, increased heart rate, and decreased RSA (e.g., Key, Campbell, Bacon, & Gerin, 2008; Ottaviani, Shapiro, Davydov, Goldstein, & Mills, 2009; Zoccola, Dickerson, & Zaldivar, 2008). Key et al. (2008) for example, exposed undergraduate women to a stress task and measured both trait and state rumination. Overall, high trait rumination (determined via scores on the Stress Reaction Rumination Scale; Robinson & Alloy, 2003) was associated with lower diastolic blood pressure and less RSA activity compared to low trait ruminators. State rumination also was associated with lower diastolic blood pressure and less RSA activity, but only among low trait ruminators. Surprisingly, this relation was not found among high trait ruminators. It is important to note, however, that Key and colleagues assessed spontaneous state rumination rather than choosing to induce rumination using Nolen-Hoeksema and Morrow's (1993) procedure. This choice may explain the surprising null effects of state rumination among high trait ruminators.

Furthermore, rumination was not compared to any other emotion regulation strategy, making it difficult to conceptualize the thought processes of those low in state rumination and determine if they were engaged in a more adaptive emotion regulation strategy.

In the one study that induced rumination following stress, delayed cardiovascular recovery was seen (Glynn, Christenfeld, & Gerin, 2002). Emotional stressors that were followed by a rumination induction (mental re-creation of the stressor) were associated with delayed blood pressure recovery in a non-clinical sample. In contrast, those followed by a distraction induction showed better cardiovascular recovery. While this study sheds light on the impact of induced rumination on biological recovery in a healthy population, it still does not include a clinically depressed sample. It is also unclear why rumination was induced using mental re-creation of the stressor rather than the well-respected ER induction (Nolen-Hoeksema & Morrow, 1993). In addition, it may be possible to obtain a more direct measure of physiological recovery by indexing the parasympathetic nervous system with RSA (see overview by Brownley et al., 2000).

Recent studies have sought to examine the effects of rumination on neuroendocrine functioning as well (Kuehner et al., 2009; Zoccola et al., 2008). In one of the first investigations, Young and Nolen-Hoeksema (2001) exposed high and low-trait ruminators to a speech task. Although increased cortisol secretion was seen post-stressor, high and low-trait ruminators did not differ in their cortisol response. Further analyses, however, revealed that no group differences existed in spontaneous state rumination levels, which further underscores the importance of using a rumination induction. One group of researchers did randomly assign individuals to ruminate versus distract in response to sad mood (Kuehner et al., 2009). In this study, an interaction was found

between BDI scores and rumination. Undergraduate participants were exposed to a sad mood induction using negative autobiographical recall coupled with sad music. They then completed the ER induction. Participants assigned to the rumination condition who had high BDI scores showed blunted cortisol stress reactivity compared to those with low BDI scores. Although rumination in general was not associated with greater cortisol levels compared to distraction, all participants in this study were non-clinical undergraduates and BDI-II scores were used as a proxy for a clinical diagnosis of depression.

In sum, in an effort to better elucidate the factors contributing to a prolonged psychological and biological response to stress, researchers have begun to investigate the use of a maladaptive emotion regulation style, rumination. Considerable evidence links the use of rumination to sustained negative affect following stress amongst currently depressed individuals (e.g., Donaldson & Lam, 2004; Lyubomirsky et al., 1999; Watkins & Moulds, 2005). Additionally, several studies have demonstrated a tentative link between rumination and prolonged physiological arousal (e.g., Glynn et al., 2002). However, a critical question remains: What is the direct effect of experimentally induced rumination on the biological recovery from stress in MDD? The current study aims to answer this question by examining how a rumination versus distraction inductions effects biological and psychological stress recovery in a group of individuals diagnosed with major depressive disorder and never-disordered controls.

Current Study

Major Depressive Disorder (MDD) is a highly prevalent disorder that is associated with costly psychological and physical symptoms (Kessler & Zhao, 1999;

Kiecolt-Glaser et al., 2002; Lopez & Murray, 1998). Identifying factors involved in the onset and maintenance of depressive episodes is therefore of the utmost importance. Depression theories emphasize the role of stress in triggering the onset of a depressive episode (e.g., see Hammen, 2005). Empirical evidence also supports the importance of stress in depression through studies that have demonstrated prolonged negative affect and biological arousal following exposure to stress among participants with MDD (e.g., Burke et al., 2005; Parker et al., 2003; Monroe & Hidjiyannakis, 2002). The current study will thus focus on improving our understanding of mechanisms that underlie this atypical stress response in depression.

Nolen-Hoeksema's response styles theory posits that risk for depression comes not from abnormality in the initial response to stress, but rather from difficulties regulating individuals' subsequent emotions (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008). This underscores the importance of focusing on recovery from stress rather than only initial reactivity. Thus, in the current study, I will purposefully separate reactivity and recovery, and I will focus on the role two different emotion regulation (ER) strategies (rumination versus distraction) play in efficient recovery following stress.

Experimental work has used Nolen-Hoeksema and colleagues' ER induction (Nolen-Hoeksema & Morrow, 1993) to manipulate ER in the laboratory. Nolen-Hoeksema and colleagues found that depressed participants randomly assigned to the ruminate induction reported prolonged negative affect compared to depressed participants randomly assigned to the distraction induction and control participants in either ER condition. Adding to the literature, several studies have coupled the ER induction with a preceding negative mood induction. Interestingly, not only depressed individuals, but

also healthy controls placed in a negative mood state experienced prolonged negative affect if assigned to ruminate than if assigned to distract (e.g., Kuehner et al., 2009; Morrow & Nolen-Hoeksema, 1990). A great deal of research has been conducted to identify the behavioral and emotional consequences of rumination (see Lyubomirsky & Tkach, 2003, and Nolen-Hoeksema et al., 2008, for excellent reviews); however, very little is known about the biological consequences of rumination in MDD. The current study will attempt to fill this gap by including both psychological and biological measures.

Although little is known about the biological consequences of rumination following a stressor, a substantial body of research has demonstrated an abnormal stress response among individuals with MDD. The neuroendocrine and autonomic nervous systems play primary roles in our body's reactivity to and recovery from stress (Patchev & Patchev, 2006). A central component of the neuroendocrine system and a well-used index of stress is the hormone cortisol. Empirical evidence on cortisol responses to an acute stressor, however, has yielded mixed results. Although a recent meta-analysis of seven studies reported that depressed participants exhibited blunted cortisol reactivity compared to control participants (Burke et al., 2005), there are exceptions that report depressed participants demonstrated hyperactive cortisol reactivity (e.g., Heim et al., 2000). More consistent evidence regarding cortisol and stress in depression comes from studies focused on stress *recovery*: higher cortisol levels were consistently found during the recovery period among depressed compared to control participants (see meta-analysis by Burke et al., 2005). Similar dysregulation has been shown in the autonomic nervous system (e.g., see the review by Rottenberg, 2007). The parasympathetic branch of the

autonomic nervous system is critically important to the body's ability to regain homeostasis after a stressful event (e.g., Lovallo & Thomas, 2000). As arguably the best indicator of parasympathetic activity, respiratory sinus arrhythmia (RSA) has become increasingly studied in recent years (e.g., for an overview see Brownley et al., 2000). Like the data on cortisol reactivity, findings on RSA reactivity are inconsistent: although one study found that depressed individuals demonstrated a significant RSA increase during stress (Rottenberg et al., 2007) others have reported no differences between depressed and control participants (e.g., Straneva-Meuse et al., 2004; Taylor et al., 2006). In contrast, evidence suggests that individuals with depression fail to exhibit the typical reengagement of RSA activation following a stressor offset, thereby deferring *recovery* to homeostasis (e.g., Rottenberg et al., 2003).

Thus, although it is unclear if depressed and control participants differ in their biological reactivity to stress, mounting evidence indicates that depressed participants exhibit prolonged biological arousal compared to controls, indicating that MDD is associated with difficulty recovering from stress. Given the important role that the neuroendocrine and autonomic nervous systems play in reactivity and recovery from stress, I selected cortisol and RSA as indicators of the biological stress response. I also sought to extend past research by examining whether the use of a particular ER strategy (rumination) might contribute to the prolonged elevation of those indicators following stress.

Recent work has begun to examine whether rumination might be a mechanism underlying the prolonged biological recovery to stress (e.g., Zoccola et al., 2008). Engaging in rumination has been shown to lead to prolonged physiological activity (e.g.,

Key et al., 2008; Glynn et al., 2002) and cortisol secretion (Zoccola et al., 2008). Although these studies offer initial evidence of a link between rumination and prolonged recovery, they used non-clinical samples and examined predominantly self-report measures of rumination rather than experimental manipulations such as Nolen-Hoeksema and Morrow's (1993) ER induction, which makes results difficult to interpret.

The current study, thus, extends past research by examining how a rumination versus distraction induction effects psychological and biological stress recovery among individuals with major depressive disorder and healthy controls. I aim to test whether rumination is a mechanism underlying the prolonged psychological and biological response that is seen in MDD. To do so, participants will be exposed to an acute laboratory stressor and then will be randomly assigned to participate in the rumination or distraction condition of the ER induction. Self-reported affect and markers of biological arousal (cortisol and RSA) will be measured during baseline, stressor, and recovery in order to examine whether the randomly assigned ER condition affects recovery from stress.

Hypotheses

The hypotheses were as follows:

1. Groups differences in stress reactivity
    a. Psychological reactivity: Both the depressed and control groups will report increased distress from baseline to stressor. Psychological stress reactivity will be greater in the depressed than control group, evidenced by a significant group by time interaction.

b. Cortisol response: Both the depressed and control groups will demonstrate increased cortisol levels from baseline to stressor. Cortisol stress reactivity, however, will not differ in the depressed and control groups, evidenced by a significant main effect of time but not a significant group by time interaction.

c. RSA response: Both the depressed and control groups will demonstrate decreased RSA levels from baseline to stressor, representing a significant RSA withdraw in response to stress. RSA withdrawal, however, will not differ in the depressed and control groups, evidenced by a significant main effect of time but not a significant group by time interaction.

2. Group by condition differences in recovery from stress

   a. Psychological recovery: Compared to depressed participants assigned to the distraction condition and control participants (regardless of condition), depressed participants assigned to the rumination condition will demonstrate less psychological recovery, measured via less decrease in distress from peak stressor. I therefore anticipate a significant time by group by condition interaction.

   b. Cortisol recovery: Compared to depressed participants assigned to the distraction condition and control participants (regardless of condition), depressed participants assigned to the rumination condition will exhibit less cortisol recovery, measured via less decrease in cortisol from peak stressor. I therefore anticipate a significant time by group by condition interaction.

   c. RSA recovery: Compared to depressed participants assigned to the distraction condition and control participants (regardless of condition), depressed

participants assigned to the rumination condition will exhibit less RSA recovery, measured via less increase in RSA levels. I therefore anticipate a significant time by group by condition interaction.

Chapter 2: Method

Participants

Participants (51 MDDs and 55 CTLs) were recruited from the community through print media, online advertisements, and outpatient clinics. Individuals were required to be 18 to 60 years of age and fluent in English. Participants in the MDD group were included if they met diagnostic criteria for current MDD, whereas the control participants were included if they were free of current or past Axis-I disorders (as confirmed using the Structural Clinical Interview for DSM-IV, SCID-I; First, Spitzer, Gibbon, & Williams, 1995). Participants were excluded if they experienced severe head trauma, had learning disabilities, bipolar disorder, psychotic symptoms, or met DSM-IV criteria for alcohol or substance abuse within the past six months. Within each diagnostic category, participants were randomly assigned to the rumination or distraction condition. Within the CTL group, 27 participants were assigned to the rumination condition and 28 to the distraction condition. Within the MDD group, 25 participants were assigned to the rumination condition and 26 to the distraction condition. The number of participants selected was based on the estimated power of $d = .92$ needed to detect differences between MDD and CTL participants in the rumination and distraction conditions.

Procedure (see Table 1)

Prescreening. Following approval from the University of Miami's Institutional Review Board (IRB), participants were screened on the telephone for exclusion and inclusion criteria (see above). Those likely to be eligible will be invited into the laboratory for Session 1.

Session 1. After signing the consent form, diagnostic status was confirmed in the laboratory using the SCID-I (First et al., 1995), which lasted between 40 and 90 minutes.

Interviews took place at the University of Miami. Inclusion and exclusion criteria were confirmed at this time. If eligible, participants completed several questionnaires (see below). Eligible participants also were invited to return to the laboratory within two weeks to complete Session 2.

Session 2 (see Figure 2). Upon returning to the University of Miami, participants were seated in front of a computer monitor. A respiration belt, plethysmograph transducers, and electrodes were attached to in order to collect a continuous measure of RSA. In order to obtain a baseline measure of RSA, the experimenter instructed participants to relax while they watched a five-minute, calming nature video and then left the room. Upon the experimenter's return, participants were told that we were examining "the relationship between people's cardiovascular health and their emotional and academic intelligence" and that it was important that they do their best on the next three tasks to obtain accurate measure of their intellectual ability. In reality, this portion of the experiment was designed to elicit stress in the participants. Specifically, they completed a twenty-minute, three-part stressor: 1. an emotional intelligence task, 2. a verbal intelligence task, and 3. an arithmetic task. All three tasks were completed in the presence of the experimenter to enhance the stressfulness of the tasks. Immediately after these stress tasks, participants were told that the second part of the experiment was going to begin. They were randomly assigned to either the rumination or distraction induction. The experimenter left participants alone in the room during the 14-minute ER induction. Subsequently, all participants watched a calming nature video for 35 minutes, also without the experimenter present. Following the video, participants were disconnected

from the psychophysiology equipment and filled out several questionnaires. Participants provided salivary cortisol samples and self-reported affect ratings throughout session 2.

Given that the hormone cortisol follows a typical circadian rhythm (Kirschbaum & Hellhammer, 1994), all of the sessions were scheduled at between 12noon and 6pm. This was necessary to minimize the effects of diurnal variations on cortisol sampling, which is vital to the assessment and interpretation of cortisol profiles. Participants were instructed not to eat or drink (except water) two hours before their arrival at the laboratory as these variables can also affect cortisol levels.

Acute Laboratory Stressor

Participants were asked to complete three separate tasks, all in the presence of the experimenter, in order to experimentally induce a negative mood/stressful state. Order of tasks was the same for all participants, who were told that these are being administered to "evaluate different aspects of their intelligence." The first was an emotional intelligence task with false feedback indicating poor performance. The second was a verbal intelligence task containing solvable and unsolvable anagrams. The third was a challenging arithmetic task.

Several factors went into determining the duration, type and order of stressors. The duration was chosen so as to ensure that individuals' cortisol levels peaked before the onset of the ER induction. This enabled us to more accurately separate cortisol reactivity due to stress from the effects of the ER induction on cortisol recovery. The type of stressors was selected given that a recent meta-analysis found this combination of motivated performance tasks with both social evaluation and uncontrollability (such as those that manipulated task difficulty by presenting impossible tasks or providing false

feedback) most effective in eliciting a cortisol response to acute stress ($d = 0.92$; Dickerson & Kemeny, 2004). Finally, the order of tasks was chosen in order to ensure that all participants experienced an uncontrollable task first (the emotional intelligence task) because that is an important determinant in individuals biological response to stress (Maier et al., 2006).

Emotional intelligence task. Participants were told that people's ability to subliminally perceive emotions reflects their emotional intelligence and social perception skills (i.e., their ability to read others' emotions). In addition, the experimenter stressed that these skills are vital to functioning well in social situations and being successful in academic and occupational domains. Participants were asked to identify the emotional expression of faces presented subliminally on the computer screen (see Appendix A for instructions). Each face was flashed on the computer screen for a brief duration (10, 20, or 30 ms) and was immediately followed by a picture of the same actor portraying a neutral facial expression. Participants were instructed to indicate the emotion depicted in the first face by selecting "H" for happy, "A" for angry, or "S" for sad.

After each of the five blocks (consisting of 10 trials each), participants received feedback regarding their performance "relative to other participants who had already completed the task." In reality, all participants received the same pre-determined feedback in order to minimize variance. After the practice block, block 1 and block 3, participants were told that they are performing in the middle 1/3 of participants, which indicates that they made about the same number of errors as past participants. After block 2, block 4, and block 5, participants were told that they are performing in the bottom 1/3, which indicates that they performed worse than did most past participants. The

experimenter stood next to participants so that she could watch participants complete the task and could read the computer-generated performance feedback. This task lasted approximately 10 minutes.

Verbal intelligence task. The second stressor task was described to participants as a test of their verbal abilities using anagrams. This task was adapted from MacLeod et al. (2002), and has been shown to induce stress in participants (e.g., Bushman et al., 2005; MacLeod et al., 2002; Salemink, van den Hout, & Kindt, 2007). Participants were informed that they had five minutes to solve as many anagrams as possible but that they would be allowed only 30 seconds to solve each anagram. A backward counting clock in the upper right corner of the screen reminded participants of the time limit. If the correct solution was not provided within 30 seconds, the computer automatically advanced to the next anagram. Words were adapted from Bushman et al. (2005) and were of varying difficulty and commonality. Online anagram testers were used to add five unsolvable anagrams. Anagrams were presented in random order (see Appendix B for a complete list). The experimenter stood next to participants during the task in order to once increase pressure on their performance. Similar unsolvable anagram tasks have been used as stressors in previous studies and have shown promising results (e.g., Bushman et al., 2005; Pederson et al., 2000). This task lasted 5 minutes.

Arithmetic task. For the final stress task, participants were asked to count backwards aloud from 2,083 to zero in 13-step sequences as quickly and accurately as possible. Participants were not allowed to use pencil and paper or any automatic calculating device. When a mistake was made, the experimenter said “error” and asked the participant to start again at 2,083. This task is part of the well used Trier Social Stress

Task (Kirschbaum, Pirke, & Hellhammer, 1993), which has consistently generated psychological and biological stress responses in participants (e.g., Kirschbaum et al., 1995; Kirschbaum, Wüst, & Hellhammer, 1992). Participants were given 5 minutes to perform this task.

Emotion Regulation (ER) Induction

Participants were randomly assigned to either the rumination or distraction condition of the ER induction, which was based on the standard induction procedure developed by Nolen-Hoeksema and Morrow (1993). This ER induction was designed to direct the content of participants' thoughts by requiring them to focus their attention and "think about" a series of prompts (adapted from Lyubomirsky & Nolen-Hoeksema, 1993; 1995; Nolen-Hoeksema & Morrow, 1993; Morrow & Nolen-Hoeksema, 1990). It has been used by numerous researchers, occasionally in a modified form, to investigate the effects of rumination versus distraction on participants' mood (e.g., Lyubomirsky et al., 1998; Donaldson & Lam, 2004; Lavender & Watkins, 2004; Lyubomirsky & Nolen-Hoeksema, 1993, 1995; Watkins & Baracaia, 2002; Watkins & Moulds, 2005; Watkins & Teasdale, 2001), retrieval of autobiographical memories (Lyubomirsky et al., 1998; McFarland & Buehler, 1998; Pyszczynski, Hamilton, Herring, & Greenberg, 1989), interpretation biases (Lyubomirsky & Nolen-Hoeksema, 1995; Greenberg, Pyszczynski, Burling, & Tibbs, 1992), predictions for the future (e.g., Lavender & Watkins, 2004; Pyszczyski, Holt, & Greenberg, 1987; Rimes & Watkins, 2005), problem solving (e.g., Donaldson & Lam, 2004; Watkins & Baracaia, 2002), and engagement in instrumental behavior (e.g., Wenzlaff, Wegner, & Roper, 1988).

In the original induction procedure (Nolen-Hoeksema & Morrow, 1993), participants are asked to think about a series of 45 prompts, each presented on their own card, for 8 minutes. Participants are able to control the amount of time they focus on any one card. All participants are given the same instructions, to read and think about the statements provided; however, the prompts differ between the rumination and distraction condition. Based on Nolen-Hoeksema's (1991) definition of rumination, the rumination condition contains prompts that focus participants' attention on thoughts that are emotion focused, symptom focused, and self-focused. Importantly, however, participants are not told to specifically to think about negative emotions or negative personal attributes. Examples of original prompts include "why you turned out this way," "trying to understand your feelings," and "your character and who you strive to be." In contrast, the distraction condition contains prompts that focus participants' attention on thoughts that are not related to symptoms, emotions, or the self. For example, they were asked to think about "clouds forming in the sky," or "the expression on the face of the *Mona Lisa."*

For the current study, the framework and instructions of Nolen-Hoeksema and Morrow's (1993) original task were maintained; however, slight adaptations were made to lengthen the induction procedure to 14 minutes so as to better ensure that the observed cortisol changes could be attributable to the induction rather than carry-over effects from the stressor. These adaptations were made in line with Bushman et al. (2005), who made similar changes during their 25-minute induction procedure. Changes were also in line with Denson and colleagues, who used a very similar procedure during their 20-minute induction (Denson et al., 2009). Given the longer induction time, there was concern that participants' attention might stray during the task if they were not provided more

structure. Thus a subset of seven rumination and distraction statements were chosen from Nolen-Hoeksema and Morrow's original list, and participants were asked to think and write about each statement for 2 minutes. Three of the rumination statements were slightly altered so as to be focused on the current situation; modifications were made so that statements remained in line with Nolen-Hoeksema's (1991) definition of rumination. For example, participants were asked to think about "why your performance on the tests earlier today made you feel the way it did." Similarly, alterations in the distraction statements were necessary in order to ensure that each statement was universally relatable so that it could be attended to for the entire 2 minutes. For example, participants were asked to think about, "the layout of a mall you have been to. Walk the entire length in your mind and describe the stores, things, or people you would see" or "how to make a peanut butter and jelly sandwich. Describe it in as much detail with as many steps as possible" (see Appendix C for the entire list). Each statement was presented for 2 minutes in black font on a white computer screen. Statements were presented one at a time in random order.

Although most ER induction procedures do not employ a manipulation check, Young and Nolen-Hoeksema's (2001) failure to find rumination following their stress-induction prompted several non-invasive checks to be used in the current study. For their manipulation check, Young and Nolen-Hoeksema used an audiotape recording of participants' thoughts, which was added to the study only to explain initial null findings. Given that speech can interfere with the measure of respiration, and therefore can confound accurate RSA readings, participants in the current study were instead provided a pen and paper so that they could write down their thoughts (see Lyubomirsky, Sousa, &

Dickerhoof, 2006, for evidence on the equivalent effects of talking and writing on mood, and see Bushman et al., 2005, for precedence using this procedure). Written statements were coded for the extent to which they reflected the definition of rumination put forth by Nolen-Hoeksema et al. (2008). Rumination score codes, which were based on Hilt and Pollak (2012), were made on a 5-point Likert scale that ranged from 1 (*Not at all ruminating*) to 5 (*Completely ruminating*). Statements were coded by two experimenters who were blind to condition and group, ICC = .84.

In addition, after each ER prompt, participants were asked to indicate how well they were able to concentrate on the previous phrase on a 7-point Likert scale ranging from 1 (*not at all*) to 7 (*very well*) as a check of participants' concentration. Thus, in the current study participants were presented with seven prompts one-at-a-time on the computer screen. They were asked to think and write about each prompt for two minutes. At the end of two minutes a brief tone sounded letting them know that the computer automatically progressed to the concentration ratings. The tone was necessary so that we could be sure all participants were aware that the two minutes have elapsed even if they were not looking at the computer screen. Once participants provided their concentration ratings, the computer progressed to the next prompt. After three prompts, the computer paused and participants were asked to complete a cortisol sample for two minutes. Following, they completed the remaining four prompts.

Measures of Stress Reactivity and Recovery

Negative Affect. Self-reported negative affect was assessed 10 times during Session 2 (S1-S11 but not S5; see Figure 2). Negative affect was not collected at S5 because the program could not be run concurrent with the ER induction program. At each

of these 10 time points, participants indicated the degree to which they felt nine different emotions (e.g., angry, sad, amused, anxious; see Appendix D for complete list) at the current moment on an 11-point Likert-scale ranging from 0 (not at all) to 10 (very much). Following previous recommendations (Kendall, Hollon, Beck, Hammen, & Ingrain, 1987) and in line with past studies (e.g., Lyubomirsky et al., 1998), negative emotions (e.g., angry, anxious, tense, sad) were averaged at each time point. Higher negative affect scores represent greater levels of distress.

Negative affect ratings at S1 and S2 represent baseline1 and baseline2 respectively. Ratings at S3 and S4 reflect negative affect during the stressor, with S4 (peak stress) expected to reflect peak stress. S6 reflects negative affect ratings during the ER induction. S7 through S11 reflect negative affect ratings during the recovery period (recovery1 through recovery5).

Salivary Cortisol. Salivary cortisol samples were collected as a measure of neuroendocrine functioning. Participants provided cortisol samples 9 times during the session (S3-S11; see Figure 2). Cortisol was measured using saliva samples collected with salivettes from Sarstedt (Rommelsdorf, Germany). All saliva samples were shipped to a laboratory at the University of Dresden in Germany (Director: Dr. Clemens Kirschbaum) for analysis. Dr. Kirschbaum's laboratory has established an international reputation for the quality of their analyses.

When interpreting cortisol values it is important to take into account the delay in salivary cortisol levels. A recent meta-analysis found that cortisol levels typically peaked 21 to 40 minutes after stressor onset and recovered to baseline 41 to 60 minutes after stressor offset (Dickerson & Kemeny, 2004). With this in mind, cortisol values at S3 and

S4 reflect baseline1 and baseline2 respectively, with S4 likely representing the true baseline given the possibility that cortisol levels at S3 will likely still be influenced by the stress of coming into the lab. S5 reflects peak cortisol, given that it is approximately 25 minutes after stressor onset, and S6 stress2. S7 and S8 represent cortisol levels during the emotion regulation induction, and S9 through S11 represent cortisol during the recovery period.

Respiratory Sinus Arrhythmia (RSA). Electrocardiograph (ECG) and Respiration Frequency (RF) were recorded continuously with a computer-based data acquisition system (MP150, Biopac Systems). Three standard electrodes were attached bilaterally to participants' left and right upper rib cage and right collarbone. To measure RF, a strain-gauge transducer belt was attached around the chest above the ribcage and below the bust. Data was collected using BIOPAC bioamplifiers. The ECG and RF signals were sampled at a rate of 1,000 Hz, digitized with a 16-bit analog-to-digital converter, and processed using AcqKnowledge and MindWare software. Vagal activation was indexed by measuring heart rate variability in the frequency band between .14 and .4 Hz (high frequency- respiratory sinus arrhythmia; HF-RSA). Beat-to-beat interval series were obtained from the ECG and converted into time series of instantaneous beat-to-beat intervals with a resolution of 4 Hz. Spectral analysis using the Welch method determined the power spectral density in the frequency band between .14 and .4 Hz. This value was then log-transformed to provide an index of HF-RSA. Calculations were made using MindWare HRV 2.16 software. R-wave markers in the ECG signal were evaluated for artifacts by visual inspection and the MAD/MED artifact detection algorithm implemented in MindWare software (Mindware Heart Rate Variability Application,

version 2.51; Mindware Technologies Ltd.). Identified artifacts were then manually corrected. This approach accords with current guidelines for frequency domain methods to determine RSA (Berntson et al., 1972).

After artifact correction, minute-by-minute estimates of RSA were determined. Baseline RSA was calculated by averaging RSA during the 5 minutes of nature video #1. Three RSA measures were calculated during the stressor: stressor1 is average RSA during the emotion intelligence task, stressor2 is average RSA during the anagram task, and stressor3 is average RSA during the arithmetic task. Two RSA measures were calculated during the ER induction: ER1 is average RSA during the first 6 minutes, and ER2 is average RSA during the last 8 minutes. Lastly, two measures were calculated during the recovery period: recovery1 includes the first 5 minutes of nature video #2, and recovery2 includes the next 5 minutes after the cortisol sample.

Questionnaires

Several questionnaires were administered at the end of Session 1 and Session 2. These were necessary to (a) ensure that individuals in the rumination and distraction conditions did not differ on demographic variables, depressive symptoms, or trait rumination, and (b) assess variables that may impact cortisol or RSA measurement (e.g., medications, food consumption, or sleep habits).

Demographic questionnaire. Participants were asked to report on variables such as their age, marital status, race, ethnicity, years of education, and household income. See Appendix E for the demographics questionnaire.

Beck Depression Inventory-Second Edition. The Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Garbin, 1988; Beck & Steer, 1993) is a 21-item

self-report questionnaire assessing the severity of current depressive symptoms. Items include sadness, loss of pleasure, guilt, suicidal thoughts or wishes, crying, indecisiveness, worthlessness, loss of energy, changes in sleeping pattern, concentration difficulty, tiredness/fatigue, and loss of interest in sex. Participants rated the frequency that each item was experienced in the past two weeks on a 4-point Likert-scale ranging from 0 to 3, with higher numbers reflecting increased frequency. The BDI-II is one of the most widely used self-report depression rating scales among adults. It has high test-retest reliability (r = 0.93; Beck, Steer, & Brown, 1996) and good internal consistency (α = .91; Beck et al., 1996; Beck, Steer, Ball, & Ranien, 1996; Dozois, Dobson, & Ahnberg, 1998). It is scored by summing responses for each item; overall scores range from 0 to 63; higher scores indicate higher levels of depressive symptoms.

Ruminative Responses Scale. The Ruminative Responses Scale of the Response Style Questionnaire (RRS; Nolen-Hoeksema & Morrow, 1991) is a 22-item self-report questionnaire assessing individual differences in the tendency to ruminate when sad, blue, or depressed (e.g., "think about how alone you feel"). Participants rated the frequency of each thought or action on a four-point Likert-scale ranging from 1 (almost never) to 4 (almost always). The RSQ-22 has demonstrated high internal consistency and acceptable convergent validity (Bagby, 2004; Butler & Nolen-Hoeksema, 1994; Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1991) as well as high internal and test-retest reliability (Luminet, 2004). A total score was calculated for each participant by summing scores for each item; higher scores indicate higher levels of trait rumination.

Health questionnaire. A health questionnaire was given to all participants in order to assess variables such as medical history, current medical conditions, medication

use, sleep patterns, physical exercise, recent food consumption, and intake of caffeine/alcohol (see Appendix F). This information has been shown to affect both cortisol and RSA, and it is thus necessary to include in data analysis procedures.

Chapter 3: Results

Preliminary Analysis

Participant characteristics. Table 2 displays the demographic and clinical characteristics of participants in the rumination and distraction conditions.[1] There were no significant differences in age across group, $F(1, 102) = 0.46$, or condition, $F(1, 102) = 0.08$, and the group by condition interaction was not significant, $F(1, 102) = 3.31$, all ps $> .05$. There was also no significant difference in proportion who were female across group, $\chi^2(1, N = 106) = 3.67$ or condition, $\chi^2(1, N = 106) = 3.15$, ps $> .05$, and the proportion female did not differ by condition within the CTL, $\chi^2(1, N = 55) = 2.50$, or MDD groups, $\chi^2(1, N = 51) = 0.94$, ps $> .05$. Years of education completed did not differ by group, $F(1, 102) = 0.40$, or condition, $F(1, 102) = 0.38$, and the group by condition interaction was not significant, $F(1, 102) = 0.28$, all ps $> .05$. Similarly, income bracket did not differ by group, $\chi^2(5, N = 102) = 8.91$, or condition, $\chi^2(5, N = 102) = 4.28$, ps $> .05$, and income did not differ by condition within the CTL, $\chi^2(5, N = 54) = 4.60$, or MDD groups, $\chi^2(5, N = 48) = 2.59$, ps $> .05$. Percent Caucasian also did not differ by group, $\chi^2(1, N = 105) = 0.35$, or condition, $\chi^2(1, N = 105) = 0.05$, ps $> .05$, and the percent Caucasian did not differ by condition within the CTL, $\chi^2(1, N = 54) = 0.01$, or MDD groups, $\chi^2(1, N = 51) = 0.16$, ps $> .05$.

Responses on the health questionnaire indicated that proportion of participants who used nicotine did not differ by group, $\chi^2(1, N = 96) = 2.95$, or condition, $\chi^2(1, N = 96) = 0.05$, ps $> .05$, and the proportion who used nicotine did not differ by condition within the CTL, $\chi^2(1, N = 49) = 0.61$, or MDD groups, $\chi^2(1, N = 47) = 1.05$, ps $> .05$.

Although there was a larger proportion of participants who regularly engaged in physical exercise in the control versus depressed group, $\chi^2(1, N = 96) = 11.96$, $p < .05$, proportion who exercised did not differ by condition, $\chi^2(1, N = 96) = 0.39$, $p > .05$, and the proportion who exercised did not differ by condition within the CTL, $\chi^2(1, N = 50) = 0.68$, or MDD groups, $\chi^2(1, N = 46) = 0.03$, ps $> .05$. Proportion who ate or drank within the last two hours did not differ by group, $\chi^2(1, N = 104) = 0.01$, or condition, $\chi^2(1, N = 104) = 1.96$, ps $> .05$, and the proportion who ate or drank within the last two hours did not differ by condition within the CTL, $\chi^2(1, N = 53) = 0.98$, or MDD groups, $\chi^2(1, N = 51) = 0.98$, ps $> .05$. Body mass index (BMI) did not significantly differ by group, $F(1, 92) = 0.62$, or condition, $F(1, 92) = 0.19$, and the group by condition interaction was not significant, $F(1, 92) = 0.69$, all ps $> .05$. Average caffeine consumption also did not significantly differ by group, $F(1, 93) = 0.35$, or condition, $F(1, 93) = 0.15$, and the group by condition interaction was not significant, by group, $F(1, 91) = 0.95$, or condition, $F(1, 92) = 1.42$, and the group by condition interaction was not significant, $F(1, 92) = 0.72$, all ps $> .05$. For females, phase of menstrual cycle did not differ by group, $\chi^2(4, N = 35) = 7.08$, or condition, $\chi^2(4, N = 35) = 3.63$, ps $> .05$, and phase of menstrual cycle did not differ by condition within the CTL, $\chi^2(4, N = 17) = 2.69$, and MDD groups, $\chi^2(4, N = 18) = 7.80$, ps $> .05$.

Clinical characteristics. As expected, the MDD group obtained significantly higher scores on the BDI than the CTL group, $F(1, 101) = 215.60$, $p < .001$; however, there were no significant differences in BDI scores across condition, $F(1, 101) = 0.07$, and the diagnosis by condition interaction was not significant, $F(1, 101) = 0.01$, ps $> .05$. In addition, although the MDD group obtained significantly higher RRS scores than the

CTL group, $F(1, 100) = 363.15$, $p < .001$, there was no significant main effect of condition, $F(1, 100) = 0.03$, nor was there a diagnosis by condition interaction, $F(1, 100) = 0.01$, $ps > .05$.[1] Similarly, although a larger portion of the MDD versus CTL group reported taking medication, $\chi^2(1, N = 106) = 8.98$, $p < .01$, the proportion using medication did not differ by condition, $\chi^2(1, N = 106) = 0.13$, $p > .05$, and the proportion taking medication did not differ by condition within the CTL, $\chi^2(1, N = 55) = 0.01$, or MDD groups, $\chi^2(1, N = 51) = 0.23$, $ps > .05$. Two individuals reported taking an antihypertensive (1 CTL; 1 MDD), one reported taking an antibiotic (MDD), one reported taking an anti-inflammatory (MDD), one reported taking a muscle relaxant (MDD), three reported taking asthma medication (1 CTL; 2 MDD), six reported taking birth control (5 CTL; 1 MDD), one reported taking a narcotic (MDD), three reported taking a sedative or tranquilizer at bedtime (all MDD), and 15 reported taking psychotropic medication (all MDD). Within the MDD group, the number of comorbid diagnoses did not differ by condition, $t(49) = 0.07$, $p > .05$. Overall, 39 met criteria for one or more comorbid anxiety disorder (11 with panic disorder, 2 with agoraphobia without a history of panic disorder, 28 for social anxiety disorder, 15 for a specific phobia, 1 for obsessive compulsive disorder, 4 for posttraumatic stress disorder, 13 for generalized anxiety disorder, and 1 for anxiety disorder not otherwise specified).

Manipulation Check

To ensure that participants were concentrating during the ER induction, the 7 self-reported concentration ratings were averaged. Although there was not a main effect of condition, $F(1, 100) = 0.47$, $p > .05$, there was a significant main effect of group, $F(1, 100) = 5.63$, $p < .05$. The group by condition interaction was also significant, $F(1, 100) =$

5.03, $p < .05$. Within the distraction condition, concentration ratings did not differ between those in the CTL ($M = 5.42$, $SD = 1.18$) and MDD ($M = 5.39$, $SD = 1.39$) group, $t(50) = 0.10$, $p > .05$; however, within the rumination condition, CTLs ($M = 6.21$, $SD = 1.38$) reported significantly more concentration than MDDs ($M = 4.97$, $SD = 1.51$), $t(50) = 3.09$, $p < .01$, reflecting the possibility that rumination did not come as naturally to control participants. In addition, within the CTL group, individuals in the rumination condition reported significantly more concentration than those in the distraction condition, $t(52) = 2.25$, $p < .05$. Within the MDD group, people in the rumination and distraction condition did not differ, $t(48) = 1.02$, $p > .05$.

To check the amount participants were ruminating during the ER induction, their written responses were coded for the extent to which they reflected the definition of rumination put forth by Nolen-Hoeksema et al. (2008).[2] Based on Hilt and Pollak (2012), each statement was coded on a 5-point Likert scale that ranged from 1 (*Not at all ruminating*) to 5 (*Completely ruminating*), and scores were added across the 7 statements. Statements were coded by two experimenters who were blind to condition and group, ICC = .84. There was a significant main effect of group, $F(1, 101) = 33.18$, $p < .001$, and condition, $F(1, 101) = 72.21$, $p < .001$. Additionally, the group by condition interaction was significant, $F(1, 101) = 13.98$, $p < .001$. Between-group differences were not found within the distraction condition, $t(51) = 1.78$, $p > .05$; however, within the rumination condition, MDDs ruminated significantly more than CTLs, $t(50) = 5.77$, $p < .001$. Importantly, however, people randomly assigned to the rumination condition ruminated significantly more than those randomly assigned to the distraction condition in both the CTL, $t(52) = 5.01$, $p < .001$, and MDD groups, $t(49) = 6.81$, $p < .001$.

Baseline Differences in Functioning

Differences in participants' baseline negative affect ratings, cortisol levels, and RSA levels were examined via three separate analyses of variance (ANOVAs). All significant findings were followed up via *t*-test.

Negative affect baseline. To examine baseline differences in negative affect, a repeated-measures ANOVA was conducted with group (MDD, CTL) as the between-subject factor and time (baseline1, baseline2) as the within-subjects factor on negative affect ratings.[3] Following previous recommendations (Kendall, Hollon, Beck, Hammen, & Ingrain, 1987) and in line with past studies (e.g., Lyubomirsky et al., 1998), negative emotions – angry, tense, anxious, irritated, sad, upset, and nervous – were averaged at each time point ($\alpha \geq .93$ for affect ratings 1-10). Higher negative affect scores represent greater levels of distress.

See Figure 3 for timing of negative affect ratings. There was a significant main effect of time $F(1, 103) = 33.39$, $p < .001$, $\eta^2 = .25$, and group, $F(1, 103) = 53.08$, $p < .001$, $\eta^2 = .34$, which were qualified by a significant time by group interaction, $F(1, 103) = 11.19$, $p < .01$, $\eta^2 = .10$. To better understand the significant time by group interaction follow-up tests were conducted. There was a significant decrease from baseline1 to baseline2 in both the CTL, $t(53) = 2.08$, $p < .05$, and MDD groups, $t(50) = 5.55$, $p < .001$. See Figure 4. Although this change was significantly larger in the MDD than control group, $t(103) = 3.35$, $p < .01$, the MDD group reported higher negative affect than the CTL group at both baseline1, $t(103) = 7.46$, $p < .001$, and baseline2, $t(103) = 6.64$, $p < .001$.

Cortisol baseline. To examine baseline differences in cortisol, a repeated-measures ANOVA was conducted with group (MDD, CTL) as the between-subject factor and time (baseline1, baseline2) as the within-subjects factor on cortisol levels.[4] See Figure 5 for information about timing of cortisol samples; labels in Figure 5 reflect the typical 20 to 30 minute delay in salivary cortisol level. There was a significant main effect of time, $F(1, 94) = 6.28$, $p < .02$, $\eta^2 = .06$, indicating a significant decrease in cortisol ratings from baseline1 to baseline2. See Figure 6. There was not, however, a significant main effect of group, $F(1, 94) = 0.01$, $\eta^2 = .00$, and the time by group interaction was not significant, $F(1, 94) = 0.32$, $\eta^2 = .003$, ps $> .05$. Given that baseline1 was likely affected by the stress of coming into the lab, baseline2 represents a more accurate measure of baseline cortisol.

RSA baseline. To examine baseline differences in RSA, an independent samples *t*-test was conducted with group (MDD, CTL) as the between-subject factor on baseline RSA (average RSA across the 5 minutes during nature video #1).[5] See Figure 7 for information on timing of RSA measurement. MDD and CTL participants did not differ in baseline RSA, $t(90) = 0.56$, $p > .05$. See Figure 8.

Hypothesis 1: Group Differences in Stress Reactivity

Three separate repeated-measures ANOVAs were conducted to investigate hypothesis 1, which examined group differences in psychological, cortisol, and RSA reactivity to stress. Follow-up tests were conducted as needed. When multiple baseline measures were taken, the last measure was used to represent baseline in the following analyses.

Psychological reactivity. I expected participants in both the MDD and CTL group to exhibit increased distress from baseline to stressor, which I expected would be larger in the MDD versus CTL group. A repeated-measures ANOVA was conducted with Group (MDD, CTL) as the between-subject factor and Time (baseline2, stress1, stress2) as the within-subject factor on negative affect ratings (e.g., average angry, tense, anxious, irritated, sad, upset, and nervous ratings). There was a significant main effect of time, $F(2, 206) = 31.18$, $p < .001$, $\eta^2 = .23$, and group, $F(1, 103) = 58.20$, $p < .001$, $\eta^2 = .36$, which were qualified by a significant time by group interaction, $F(2, 206) = 3.67$, $p < .05$, $\eta^2 = .03$. Follow-up tests indicated a significant increase from baseline2 to stress1 in both the CTL, $t(53) = 2.36$, $p < .05$, and MDD groups, $t(50) = 5.00$, $p < .001$, which was significantly larger in the MDD than CTL group, $t(103) = 2.78$, $p < .01$. There was also a further increase in negative affect from stress1 to stress2 in both the CTL, $t(53) = 3.11$, $p < .01$, and MDD groups, $t(50) = 2.06$, $p < .05$, which did not significantly differ by group, $t(103) = 0.06$, $p > .05$. Regardless, the MDD group reported significantly greater negative affect than the CTL group at stress1, $t(103) = 7.39$, $p < .001$, and stress2, $t(103) = 6.54$, $p < .001$. See Figure 4.

Cortisol reactivity. I expected participants in both the MDD and CTL group to exhibit increased cortisol levels from baseline to stressor; however, I did not expect cortisol reactivity to differ by group. A second repeated-measures ANOVA was conducted with Group (CTL, MDD) as the between-subject factor and Time (baseline2, peak cort) on salivary cortisol levels. There was no significant main effect of time, $F(1, 95) = 0.01$ $\eta^2 = .00$. In addition, there was no main effect of group, $F(1, 95) = 0.40$, $\eta^2 = .00$, or time by group interaction, $F(1, 95) = 1.11$ $\eta^2 = .01$, all ps $> .05$. See Figure 6.

RSA reactivity. I expected participants in both the MDD and CTL group to exhibit a significant RSA withdrawal from baseline to stressor; however, I did not expect RSA withdrawal to differ by group. A repeated-measures ANOVA was conducted with Group (CTL, MDD) as the between-subject factor and Time (baseline, stressor1, stressor2, stressor3) as the within-subject factor on RSA level. There was a significant main effect of time, $F(2, 270) = 15.70$, $p < .001$, $\eta^2 = .15$. However, there was no significant main effect of group, $F(1, 90) = 0.30$, $\eta^2 = .003$, or time by group interaction, $F(2, 270) = 0.06$, $\eta^2 = .001$, both ps $> .05$. See Figure 8. As anticipated, paired samples t-tests indicated that there was a significant RSA withdrawal from baseline to stressor1, $t(91) = 3.14$, $p < .01$. Unexpectedly, however, there was a significant increase in RSA from stressor1 to stressor 2, $t(91) = 2.40$, $p < .02$, and a further increase from stressor2 to stressor3, $t(91) = 6.05$, $p < .001$, suggesting spontaneous RSA recovery. As would be expected there was also a significant difference from stressor 1 to stressor 3, $t(91) = 6.69$, $p < .001$.

Hypothesis 2: Group by Condition Differences in Recovery from Stress

Three separate repeated-measures ANOVAs were conducted to investigate hypothesis 2, which examined group by condition differences in psychological, cortisol, and RSA recovery from stress. Follow-up tests were conducted as needed. When multiple measures were taken during the stressor, the highest value was used to represent peak stress.

Psychological recovery. I expected depressed participants in the rumination condition to exhibit less psychological recovery from stress compared to depressed participants in the distraction condition and compared to control participants (regardless

of condition). A repeated-measures ANOVA was conducted with Group (CTL, MDD) and Condition (rumination, distraction) as the between-subject factors and Time (peak stress, ER, recovery1, recovery2, recovery 3, recovery4, recovery5) as the within-subject factor on negative affect ratings. There was a significant main effect of time, $F(6, 606) = 28.37$, $p < .001$, $\eta^2 = .22$, and group, $F(1, 101) = 37.96$, $p < .001$, $\eta^2 = .27$, and a significant time by group interaction, $F(6, 606) = 4.38$, $p < .001$, $\eta^2 = .04$. These were qualified by a significant time by group by condition interaction at the quadratic level, $F(1, 101) = 4.83$, $p < .05$, $\eta^2 = .05$.[6] However, there was no significant main effect of condition, $F(1, 101) = 2.01$, $\eta^2 = .02$, time by condition interaction, $F(6, 606) = 1.40$, $\eta^2 = .01$, or group by condition interaction, $F(1, 101) = 3.50$, $\eta^2 = .03$, all ps $> .05$.

To follow-up on the significant 3-way interaction and test my a priori hypothesis, change scores (Δ) were computed by subtracting negative affect ratings during peak stress from negative affect ratings during the emotion regulation (ER) and recovery periods (recovery1 – recovery5). The more negative the change score, the greater the recovery. See Figure 9. A repeated-measures ANOVA with time (Δpeak stress, ΔER, Δrecovery1, Δrecovery2, Δrecovery3, Δrecovery4, Δrecovery5) and condition (rumination, distraction) was conducted on the negative affect change scores separately for each group. Within the CTL group, there was a significant main effect of time, $F(6, 312) = 8.19$, $p < .001$, $\eta^2 = .14$, reflecting that negative affect decreased from peak stressor through the recovery period. However, there was no significant time by condition interaction, $F(6, 312) = 0.87$, $p > .05$, $\eta^2 = .02$, suggesting that psychological recovery did not differ between those in the rumination versus distraction conditions. In contrast, within the MDD group, the main effect of time, $F(6, 294) = 2.35$, $p < .001$, $\eta^2 = .29$, was

qualified by a significant time by condition interaction at the cubic order, $F(1, 49) = 3.03$, $p < .05$, $\eta^2 = .08$. MDD individuals experienced a significant decrease in negative affect during the recovery period regardless of whether they were in the rumination, $F(6, 144) = 5.08$, $p < .001$, $\eta^2 = .18$, or distraction condition, $F(6, 150) = 19.92$, $p < .001$, $\eta^2 = .44$. However, there was a significantly greater decrease in negative affect from peak stress to ER in the distraction versus rumination condition, $t(49) = 2.68$, $p < .02$, suggesting that for depressed individuals the initial psychological recovery was greater when assigned to distract versus ruminate. Within the MDD group, the decrease in negative affect from peak stressor did not differ by condition at any other time point (Δrecovery1, Δrecovery2, Δrecovery3, Δrecovery4, Δrecovery5), $t(49) = 1.78$, 1.35, 1.38, 1.51, and 1.21 respectively, all ps $> .05$.

Cortisol recovery. I expected depressed participants in the rumination condition to exhibit less cortisol recovery from stress compared to depressed participants in the distraction condition and compared to control participants (regardless of condition). A repeated-measures ANOVA was conducted with Group (CTL, MDD) and Condition (rumination, distraction) as the between-subject factors and Time (peak cort, stress2, ER1, ER2, recovery1, recovery2, recovery3) as the within-subject factor on salivary cortisol levels. There was a significant main effect of time, $F(6, 558) = 17.90$, $p < .001$, $\eta^2 = .16$, a time by group interaction, $F(6, 558) = 2.41$, $p < .05$, $\eta^2 = .03$, and a time by condition interaction, $F(6, 558) = 2.19$, $p < .05$, $\eta^2 = .02$, which were qualified by the expected time by group by condition interaction, $F(6, 558) = 3.40$, $p < .01$, $\eta^2 = .04$.[7] There was no significant main effect of group, $F(1, 93) = 1.60$, $\eta^2 = .02$, main effect of

condition, $F(1, 93) = 0.18$, $\eta^2 = .002$, or group by condition interaction, $F(1, 93) = 0.81$, $\eta^2 = .009$, $ps > .05$.

To follow-up on the significant 3-way interaction and test my a priori hypothesis, changes scores (Δ) were computed by subtracting Cortisol Peak Stress from cortisol levels during the recovery periods. The more negative the change score, the greater the decrease in cortisol from peak stress. See Figure 10. A repeated-measures ANOVA with time (Δpeak cort, Δstress2, ΔER1, ΔER2, Δrecovery1, Δrecovery2, Δrecovery3) and condition (rumination, distraction) was conducted on the cortisol change scores separately for each group. Within the CTL group, there was a significant main effect of time, $F(6, 294) = 38.82$, $p < .001$, $\eta^2 = .44$, reflecting that cortisol levels decreased from peak stressor through the recovery period. However, there was no significant time by condition interaction, $F(6, 294) = 0.24$, $p > .05$, $\eta^2 = .005$, suggesting that cortisol recovery did not differ between those in the rumination versus distraction conditions. In contrast, within the MDD group, the main effect of time, $F(6, 264) = 2.35$, $p < .05$, $\eta^2 = .05$, was qualified by a significant time by condition interaction, $F(6, 264) = 3.22$, $p < .01$, $\eta^2 = .07$. Individuals in the MDD group randomly assigned to the distraction condition experienced a significant decrease in cortisol levels during the recovery period, $F(6, 132) = 9.27$, $p < .001$, $\eta^2 = .30$; however, individuals in the MDD group randomly assigned to the rumination condition did not experience a significant decrease in their cortisol levels during the recovery period, $F(6, 132) = 0.41$, $p > .05$, $\eta^2 = .018$. One-sample *t*-tests indicated that the MDD + distraction group's decrease in cortisol significantly differed from zero at all time points, $t(22) = 2.60, 3.48, 3.33, 3.20, 3.37,$

3.53, all *p*s < .02. In contrast, the MDD + rumination group's decrease in cortisol did not significantly differ at any time point, all $t(22) < 1$, all *p*s > .05.

As planned, I also calculated area under the curve (AUC) analyses to ground using trapezoidal integration. A univariate ANOVA was conducted with Group (CTL, MDD) and Condition (rumination, distraction) as the between-subject factors on AUC. There was no significant main effect of group, $F(1, 93) = 0.90$, $\eta^2 = .01$, or condition, $F(1, 93) = 0.27$, $\eta^2 = .003$, and the group by condition interaction did not reach significance, $F(1, 93) = 1.63$, $\eta^2 = .02$, *p*s > .05. See Table 3.

RSA recovery. I expected depressed participants in the rumination condition to exhibit less RSA recovery from stress compared to depressed participants in the distraction condition and compared to control participants (regardless of condition). A repeated-measures ANOVA was conducted with Group (CTL, MDD) and Condition (rumination, distraction) as the between-subject factors and Time (stressor3, ER1, ER2, recovery1, recovery2) as the within-subject factor on RSA. There was a significant main effect of time, $F(4, 352) = 24.10$, $p < .001$, $\eta^2 = .22$, and a significant interaction between time and diagnosis, $F(4, 352) = 2.65$, $p < .05$, $\eta^2 = .03$, which were qualified by a significant interaction between time, group, and condition at the cubic order, $F(1, 88) = 4.03$, $p < .05$, $\eta^2 = .04$.[8] There was no significant main effect of group, $F(1, 88) = 0.17$, $\eta^2 = .002$, main effect of condition, $F(1, 88) = 0.32$, $\eta^2 = .004$, time by condition interaction, $F(4, 352) = 0.34$, $\eta^2 = .004$, or group by condition interaction, $F(1, 88) = 0.70$, $\eta^2 = .008$, all *p*s > .05. See Figure 11.

To follow-up on the significant 3-way interaction and test my a priori hypothesis, changes scores (Δ) were computed. To assess RSA withdrawal during the ER induction,

RSA during ER1 and ER2 was subtracted from stressor3. Higher values indicate greater impact of the ER induction. To assess RSA recovery that occurred after the ER induction, RSA during ER2 was subtracted from recovery1 and recovery2. Higher values indicate greater recovery. Results indicated that ΔER1 and ΔER2 significantly differed from zero, $t(91) = 6.12$ and 5.65 respectively, ps $< .001$, indicating a significant RSA withdrawal during the ER period. In addition, Δrecovery1 and Δrecovery2 also significantly differed from zero, $t(91) = 5.84$ and 6.79 respectively, ps $< .001$, indicating significant RSA recovery during the recovery period. Four univariate ANOVAs were conducted with group (CTL, MDD) and condition (rumination, distraction) on each of the four change scores (ΔER1, ΔER2, Δrecovery1, Δrecovery2). At ΔER1, there was no significant main effect of group, $F(1, 88) = 0.68$, $\eta^2 = .008$, or condition, $F(1, 88) = 0.04$, $\eta^2 = .00$, both ps $> .05$; however, there was a significant group by condition interaction, $F(1, 88) = 3.87$, $p = .05$, $\eta^2 = .04$. Within the rumination condition, there was no significant difference in RSA withdrawal between the CTL and MDD group, $t(44) = 0.79$, $p > .05$. Within the distraction condition, however, MDD participants displayed a significantly greater RSA withdrawal than the CTL group, $t(44) = 2.02$, $p = .05$. At ΔER2, there was no significant main effect of group, $F(1, 88) = 0.14$, $\eta^2 = .002$, or condition, $F(1, 88) = 0.03$, $\eta^2 = .00$, and no significant group by condition interaction, $F(1, 88) = 2.31$, $\eta^2 = .03$, all ps $> .05$. At Δrecovery1, there was a significant main effect of group, $F(1, 88) = 4.39$, $\eta^2 = .05$, indicating that the MDD group displayed significantly greater RSA recovery than the CTL group. However, there was no significant main effect of condition, $F(1, 88) = 0.90$, $\eta^2 = .01$, and no significant group by condition interaction, $F(1, 88) = 1.49$, $\eta^2 = .02$, ps $> .05$. At Δrecovery2, there was a main effect of group, $F(1, 88) = 4.28$, $p < .05$, $\eta^2 = .05$,

indicating that the MDD group displayed significantly greater RSA recovery than the CTL group. However, there was no significant main effect of condition, $F(1, 88) = 0.80$, $\eta^2 = .01$, and no significant group by condition interaction, $F(1, 88) = 0.25$, $\eta^2 = .003$, both ps $> .05$.

Chapter 4: Discussion

Recent diathesis-stress models of depression emphasize that stress frequently triggering the onset of a depressive episode. However, increasing evidence indicates that risk for depression stems not from the initial response to stress, but rather subsequent difficulty recovering from the stressor. Rumination is a particularly maladaptive emotion regulation (ER) strategy known to prolong psychological distress in depression. However, little is known about the way rumination effects individuals' biological recovery from stress. The current study therefore focused on understanding how rumination versus distraction influenced participants' psychological and biological recovery from stress.

Participants were exposed to a psychosocial stressor and then randomly assigned to either the rumination or distraction induction. Psychological and biological stress reactivity and recovery were examined via three primary dependent variables: self-reported negative affect, salivary cortisol, and respiratory sinus arrhythmia (RSA). I first examined participants' response to stress. The primary goal of the study, however, was to test whether the rumination versus distraction induction influenced participants' recovery from stress.

Participants reported significant psychological and biological response to stress, evidenced by a significant increase in self-reported negative affect and a significant RSA withdrawal. Unexpectedly, however, participants demonstrated spontaneous RSA recovery during the second half of the stressor. Also contrary to expectations, I did not observe a significant increase in salivary cortisol in response to the stressor. During the recovery period, significant time by condition by group interactions were observed for

negative affect, salivary cortisol, and RSA. Within the CTL group, rumination versus distraction did not impact psychological or biological recovery from stress. Within the MDD group, however, rumination was associated with higher negative affect and salivary cortisol. Additionally, depressed participants assigned to the distraction condition demonstrated significantly greater RSA withdrawal during the ER induction compared to control participants in the distraction condition. During the subsequent nature video, the MDD group demonstrated significantly greater RSA recovery compared to the CTL group. Each of these findings will be discussed in turn.

Hypothesis 1: Group Differences in Stress Reactivity

Psychological reactivity. I expected both the depressed and control groups to report increased distress from baseline to stressor, and I expected psychological stress reactivity to be greater in the depressed than control group. Negative affect was measured at two time points during the 20-minute stress phase: in the middle and at the end. As expected, there was a significant increase in negative affect from baseline to mid-stress. There was also an additional increase from mid-stress to post-stress, suggesting a possible cumulative effect of the stress tasks (e.g., Margolin & Gordis, 2003). The increase in negative affect from baseline to mid-stress was greater for the depressed versus control group, which is in line with current conceptualizations of depression (e.g., Rottenberg, Gross, & Gotlib, 2005) as well as results indicating depressed individuals are more reactive to even minor negative events (e.g., Monroe & Harkness, 2005). Although the subsequent increase from mid-stress to post-stress did not differ by group, the MDD group continued to report higher negative affect at post-stress.

Cortisol Reactivity. I anticipated both the depressed and control groups would show increased cortisol levels from baseline to stressor, and I anticipated that this increase would not differ by group. Contrary to my hypothesis, the increase in salivary cortisol from baseline to peak stress was not significant. The nonsignificant increase in cortisol does not correspond with the significant increase in psychological distress. However, studies have shown a discrepancy between biological and psychological accounts of stress reactivity (e.g., Yoon & Joormann, 2012). For example, a recent meta-analysis did not find a significant correlation between cortisol reactivity to stress and changes in self-reported distress (Dickerson & Kemeny, 2004).

There are several possible reasons why a significant increase in cortisol was not observed during the stress period. It is possible that I did not accurately capture the peak cortisol response. Peak cortisol in the current study was found at cortisol sample 3, approximately 30 minutes after stressor onset. The timing of our peak cortisol is in line with meta-analysis findings showing that peak cortisol typically occurs 21 to 40 minutes after stressor onset (Dickerson & Kemeny, 2004). Moreover, cortisol levels sampled approximately 8 minutes before and after sample 3 were on average slightly lower, which makes it likely that sample 3 is an accurate reflection of cortisol levels at peak stress. A second possible reason that I did not observe a significant increase in cortisol from baseline to peak stress is that I did not accurately capture baseline cortisol. This possibility is supported by the fact that participants' cortisol levels at baseline were higher than cortisol levels at recovery. The current study used a 5 minute baseline period, and this may not have allowed sufficient time for participants' neuroendocrine system to recover from the stress of coming into the lab or being connected to the

psychophysiological equipment. Future work might therefore consider utilizing a longer baseline period in order to allow a more accurate measure of baseline cortisol before the stressor onset. For example, a 10-minute baseline has been used in other studies collecting salivary cortisol (e.g., Kirschbaum et al., 1993). If cortisol levels in the current study were elevated at baseline, nonsignificant cortisol reactivity should be interpreted with caution.

RSA reactivity. Historically, the parasympathetic nervous system is predominantly an index of stress *recovery*; more recent evidence, however, has identified activity of the parasympathetic nervous system as an important marker of adaptive stress *reactivity* as well (e.g., Cacioppo, Berntson, Binkley, Quigley, Uchino, & Fieldstone , 1994; Sack, Hopper, & Lamprecht, 2004). Research suggests efficient reactivity to stress is determined not only by activation of the sympathetic branch of the nervous system but also by efficient withdrawal of parasympathetic branch, often measured via RSA. I therefore expected both the depressed and control groups to show a significant RSA withdrawal in response to stress. This hypothesis was partially supported. Initially, participants displayed the expected RSA decrease from the baseline to the first 10 minutes of the stressor period. The initial, rapid RSA decrease reflects an adaptive withdrawal of the parasympathetic nervous system that allows sympathetic activation on the heart, as would typically be expected when faced with stress (e.g., Brownley et al., 2000).

Unexpectedly, there was a spontaneous and significant RSA increase in the second half of the stress phase. This is an interesting and potentially important finding because the majority of studies that examine autonomic nervous system reactivity in

depression utilize shorter stress tasks (e.g., 2 to 10 minutes; Hughes & Stoney, 2000; Rottenberg et al., 2003; Rottenberg et al., 2007), leaving less known about RSA functioning during stressors beyond 10 minutes. It is possible that the RSA increase during the second half of the stressor is due to the latter two stress tasks generating less distress. However, participants' self-reported distress does not support this conclusion: participants' report of distress increased during the latter two stress tasks. Another possible explanation is that during longer stressors the parasympathetic nervous system is activated even before the offset of the stress. The autonomic nervous system operates much faster than the neuroendocrine system (Brownley et al., 2000), and it is possible that the parasympathetic branch of the autonomic nervous system works to return the body to homeostasis even mid-stress. Additional research is needed in order to fully explore the time course of parasympathetic activation when faced with longer stress.

As expected, the depressed and control groups did not differ in their RSA withdrawal to stress. Although some studies suggest that greater parasympathetic withdrawal is associated with dysphoria or low self esteem (Hughes & Stoney, 2000; O'Donnell, Brydon, Wright, & Steptoe, 2008), this literature is both sparse and mixed. For example, whereas some studies report RSA increases in response to stress amongst those with MDD (Rottenberg et al., 2007), others report no differences between depressed and controls in RSA withdrawal (e.g., Straneva-Meuse et al., 2004; Taylor et al., 2006). Thus, overall, there does not seem to be convincing evidence for differences between control and depressed participants' parasympathetic withdrawal to stress.

Summary of Hypothesis 1. Taken together, results suggest that participants experienced increased distress in response to the stressor, evidenced by increased

negative affect and RSA withdrawal. In contrast, I did not find a significant increase in cortisol response to stress. Null findings, however, could be attributed to baseline cortisol being confounded by the stress of coming into the lab. Whereas our 5-minute baseline period may have been sufficient for psychological and physiological recovery, the slower-acting neuroendocrine system may not have had time to return to baseline prior to the onset of the stress tasks.

Interestingly, whereas group differences were found in psychological response to stress, I did not observe group differences in participants' biological response to stress. The literature mirrors this discrepancy in findings (e.g., see meta-analyses by Burke et al., 2005, and Rottenberg, 2007). Whereas depressed individuals are consistently found to report greater psychological reactivity to stress (e.g., Rottenberg et al., 2005), the literature is inconsistent with regards to depressed individuals demonstrating greater biological stress reactivity (e.g., see recent meta-analyses by Burke et al., 2005, and Rottenberg, 2007). Two important conclusions can be drawn from findings on stress reactivity. For one, given discrepant findings between psychological and biological dependent variables, it is important to examine multiple outcome measures and to include both psychological and biological indices of distress to fully understand the impact of stress in depression. Moreover, results do not suggest that there are consistent differences between the CTL and MDD group in the initial biological reactivity to stress. It is therefore possible that to better understand biological dysregulation in MDD one must also examine factors affecting the subsequent recovery from stress. Findings regarding recovery from stress will be examined below.

Hypothesis 2: Group by Condition Differences in Recovery from Stress

Psychological recovery. I expected depressed participants in the rumination condition to demonstrate less psychological recovery compared to all other groups. As expected, depressed participants randomly assigned to ruminate reported higher negative affect immediately after the emotion regulation induction compared to depressed participants assigned to distract and controls in either the rumination or distraction condition. This finding is in line with research demonstrating maladaptive effects of rumination on negative mood (e.g., Lyubomirsky, et al., 1998; Lyubomirsky et al., 1999; Nolen-Hoeksema & Morrow, 1993; Papageorgiou & Wells, 2000). Our findings, therefore, add to the substantial body of literature indicating that rumination is a key factor that contributes to the hallmark symptom of MDD, sustained negative affect.

Following the rumination induction, participants reported the expected decrease in negative affect. Contrary to our hypothesis, this decrease did not differ between depressed participants in the rumination versus distraction condition. Said another way, the effects of the ER induction did not sustain during the recovery period. At first this might appear discrepant from the existing literature on rumination. However, few studies have examined mood over a longer recovery period. The majority of studies focus on mood ratings made immediately after the ER induction (e.g., Lyubomirsky & Nolen-Hoeksema, 1995; Nolen-Hoeksema & Morrow, 1993; Park et al., 2004). More sadness is typically reported by people in the rumination versus distraction condition at this time point, providing evidence of the effects of rumination on mood. Our findings are consistent with this literature given that differences between depressed participants in the rumination and distraction condition were observed immediately following the ER

induction. The long-term effects of rumination on mood are less clear. Although some studies have found a return to baseline at time points after this first post-induction mood check (e.g., Wisco & Nolen-Hoeksema, 2009), others have found sustained negative mood hours later (e.g., Watkins, 2004). Watkins, for example, examined prolonged negative mood after writing in a ruminative versus factual way about a recent failure. Although lower mood was found in the rumination versus factual condition 12 hours after writing, this was only true in the group of individuals with high trait rumination. Watkins' findings suggest it is not the rumination induction alone that perpetuated negative mood, but rather the combination of the rumination induction and ongoing rumination that contributes to sustained negative mood. Thus, it is possible rumination only effects mood while people are ruminating. In the current study, the calming nature video began immediately after the ER induction, potentially serving as a distraction for individuals in both the rumination and distraction group (Lyubomirsky et al., 1998; Lyubormirsky & Nolen-Hoeksema, 1995). The video therefore could have prevented ongoing rumination for those in the rumination condition, which might have facilitated negative affect recovery.

Within the control group, psychological recovery did not differ between those in the rumination versus distraction condition. This was unexpected. In line with past research, rumination was expected to be associated with more negative affect than distraction given that controls were placed in a negative mood state (e.g., Morrow & Nolen-Hoeksema, 1990; Rusting & Nolen-Hoeksema, 1998). For example, Morrow and Nolen-Hoeksema exposed an unselected college sample to a sadness induction prior to the ER induction. As Morrow and Nolen-Hoeksema expected, individuals assigned to

ruminate reported more negative mood than those assigned to distract. Similarly, Rusting and Nolen-Hoeksema exposed an unselected sample to an anger induction prior to the ER induction. Once again, those in the rumination condition experienced heightened anger compared to those in the distraction induction. Methodological differences between the current study and past study designs might explain the lack of condition effects within our healthy control group. One important difference is the sample itself. An unselected college sample might not be equivalent to a carefully screened group of controls, who have not experienced an Axis-I condition in their lifetime. One might argue that base rates of psychopathology would mean that a certain portion of unselected college students would meet criteria for a DSM disorder (e.g., Blazer, Kessler, McGonagle, & Swartz, 1994). Compared to this unselected samples, our CTL group might have been less affected by the mood and/or ER induction.

Supporting the possibility that CTLs might have been less affected by the rumination induction, I observed differences in the amount CTLs and MDDs were ruminating during the rumination induction. Based on written statements provided during the ER induction, controls in the rumination condition obtained lower rumination scores than depressed participants in the rumination condition. This suggests that the rumination condition was less effective for controls compared to participants with depression. With this in mind, it might be hasty to conclude that this study provides evidence that rumination does not impact healthy controls in a negative mood state. Instead, it highlights the difficulty of inducing rumination in a non-depressed sample. In fact, past research has been unsuccessful in inducing rumination in non-depressed samples (e.g., Young & Nolen-Hoeksema, 2001). In contrast, the current study was mildly successful in

inducing rumination in the control group: controls in the rumination condition ruminated more than controls in the distraction condition. The current study therefore moved the literature closer toward the goal of identifying an effective ER induction for healthy controls. Nevertheless, additional work is needed to develop an ER induction that induces rumination in healthy controls to the same extent as in participants with depression.

Cortisol recovery. I expected depressed participants in the rumination condition to demonstrate less cortisol recovery compared to all other groups. Within the CTL group, results mirror those found with psychological recovery. Not only is this in line with data examining rumination in healthy controls, it is also in line with Young and Nolen-Hoeksema (2001), which showed that a rumination induction did not impact cortisol recovery in a non-depressed sample. As mentioned when discussing psychological recovery in the control group, these null findings should be interpreted with caution given that the rumination induction was less effective for controls than for participants with depression.

Within the MDD group, participants in the rumination condition showed significantly less cortisol recovery compared to those in the distraction condition. This finding extends past work that showed a correlation between rumination and cortisol recovery (e.g., Key et al., 2008; Zoccola et al., 2008). By experimentally manipulating the ER condition, I am able to draw stronger conclusions about the impact of rumination on cortisol. Importantly, our results suggest a potentially detrimental effect of rumination on people's ability to return their neuroendocrine system to homeostasis following stress. Given the consequences of prolonged cortisol levels on cardiovascular health, rumination may be a key factor that places depressed individuals at increased risk for poor health

outcomes, such as increased risk of a cardiac event and faster progression of illnesses (Carney et al., 1995; Kiecolt-Glaser et al., 2002; Leserman et al., 1999). Interesting, whereas the impact of rumination on negative affect lasted only during the ER induction itself, the impact on participants' neuroendocrine system lasted throughout the recovery period. This highlights the importance of including biological markers of distress, such as cortisol, when examining the impact of rumination on stress reactivity in depression. We may otherwise underestimate the impact of rumination.

RSA recovery. I expected depressed participants in the rumination condition to demonstrate less RSA recovery compared to all other groups. Contrary to my hypothesis, I observed a significant RSA withdrawal – rather than recovery – during the ER induction. Although very few studies have examined physiological activity during an ER induction, evidence of a similar RSA withdrawal in response to rumination has been found (e.g., Ottaviani, Shapiro, Davydov, & Goldstein, 2008). In line with interpretations made by Ottaviani and colleagues, it is possible that RSA withdrawal during our ER induction reflects increased effort. In fact, increased effort is associated with activation of the autonomic nervous system, as indexed via significant parasympathetic withdrawal (e.g., Buchanan, al'Asi, & Lovallo, 1999; Lundberg & Frankenhaeuser, 1980).

I also observed a significant group by condition interaction in RSA recovery, which reflected a significantly greater RSA withdrawal in depressed participants assigned to distract compared to control participants assigned to distract. Group differences in RSA withdrawal during distraction could also be interpreted in terms of effort during the ER induction. No group differences were observed in the amount of effort needed to ruminate. In contrast, depressed participants required more effort to engage in distraction

compared to healthy controls. It is possible that the increased effort of distraction is one reason depressed participants are less likely to utilize this ER strategy (e.g., Nolen-Hoeksema et al., 2008). Reciprocally, the increased effort involved with distraction could stem from the fact that depressed individuals are less familiar with using distraction in response to stress. Additional research is needed in order to understand whether these or other mechanisms contribute to greater RSA withdrawal in depressed participants when they engage in distraction.

Following the ER induction, depressed participants demonstrated a significantly greater RSA recovery compared to controls; however, RSA recovery did not differ by ER condition. This finding was unexpected. I expected the ER induction to effect autonomic activity during the recovery period. Based on the rapid response of the autonomic nervous system, however, autonomic effects of the ER condition may not have continued beyond the ER condition and into the recovery period. This would explain the lack of condition differences during the recovery period. Using the same logic, group differences in RSA recovery during the nature video might also be explained via group differences in response to the video rather than carry over effects of the ER induction. As mentioned previously, the video may have served as an inadvertent distractor that captivated participants' attention enough to prevent rumination but did not require the same amount of effort that was required by the distraction condition of the ER induction. In fact, similar passive distraction inductions have been effectively used in other studies (e.g., Weiss, Dahlquist, & Wohlheiter, 2011). With this possibility in mind, it would be as if both depressed and control participants were exposed to a passive distraction induction – watching a calming nature video. During the nature video, depressed participants showed

greater recovery compared to controls. It is thus possible that passive distraction conditions might serve as a particularly effective method of recovering from stress for people with depression. Future research is needed in order to follow-up on the possibility that passive versus active distraction may yield a different physiological response and that passive distraction might be an especially adaptive method for depressed participants to recover from stress.

Summary of Hypothesis 2. Several important conclusions come from examining the impact of rumination versus distraction on stress recovery in depressed and control participants. For one, we found the rumination induction to have unexpectedly little effect on healthy controls. Results from a manipulation check suggest that the rumination induction was less effective for control versus depressed participants, which may explain null effects within the control group. It is thus possible that a stronger rumination induction is needed in order to obtain effects within the control group.

Second, within the MDD group, the rumination versus distraction induction yielded a significant effect on psychological and biological recovery from stress. We presented experimental evidence that rumination plays an important role sustaining not only negative affect but also may be a critical mechanism underlying biological dysregulation in response to stress. Our research, therefore, supports and extends our knowledge of the role rumination plays in risk for depression (e.g., Brosschot et al., 2006).

Third, we observed different effects of ER condition on the three different dependent variables. The effects of the ER condition on negative affect and RSA recovery were short compared to the effects on cortisol. It is not entirely clear why

rumination would have had more prolonged effects on the neuroendocrine system. One possible reason is in the nature of the neuroendocrine system, which is a slower-acting system compared to the more rapid responses of the autonomic nervous system (e.g., Brownley et al., 2000; Dickerson & Kemeny, 2004). Another possibility is that prolonged distress, such as that caused by a longstanding history of rumination, could have led to more permanent alterations in cortisol recovery mechanisms. Cortisol recovery is facilitated by negative feedback loops: receptors on the hypothalamus, pituitary, and hippocampus identify elevated levels of cortisol and signal the HPA axis to stop producing cortisol (see McEwen, 2006, for a review). Chronic cortisol elevation can damage the sensitivity of receptors, and thus may be responsible for difficulty down regulating cortisol once production begins. It is possible that a history of rumination contributed to frequent chronic cortisol secretion, which minimized the effectiveness of the negative feedback system.

Lastly, the current study also extends our understanding of stress reactivity in depression. Although we did not observe consistent group differences in the initial reactivity to stress, rumination was consistently associated with differential recovery from stress in the MDD group. Evidence supports more recent research on depression suggesting risk for depressive episodes stems not from the initial response to stress, but rather from difficulties regulating the subsequent emotional state (Flynn & Rudolph, 2007; Nolen-Hoeksema et al., 2008). Moreover, the current study suggests that rumination is a key mechanism that hinders both psychological and biological regulation in MDD.

Limitations and Future Directions

This study used an experimental design to extend our understanding of how rumination impacts stress recovery in major depressive disorder. It is important to acknowledge several limitations and identify areas for future work. For one, the baseline period in the current study was only 5 minutes. Although this was likely sufficient to allow psychological and physiological recovery from the stress of coming into the lab and psychophysiological hookup, it may have not been sufficient to allow an accurate cortisol baseline measure. Although a 5-minute baseline is in line with other studies examining stress reactivity, more recent research encourages the use of longer baseline periods (e.g., Kirschbaum et al., 2003). Future work might therefore consider using at least a 10-minute baseline period.

Along these lines, a second limitation was the possible interference of providing multiple cortisol samples. In an effort to achieve the appropriate balance between maximizing precision and minimizing burden, I reduced the number of cortisol collections from my original proposal. Despite this reduction, each participant still provided ten cortisol samples. The frequency of cortisol samples might have caused some mild distress, and fewer samples may decrease burden in future work.

It is also important to acknowledge the fact that the ER induction was less effective in the control versus depressed group. Although control participants in the rumination condition ruminated significantly more than those in the distraction condition, within the rumination condition, group differences were identified. Specifically, controls in the rumination condition ruminated less than depressed in the rumination condition. The ER induction was adapted from the well-used and well-respected Response Styles

Manipulation (e.g., Nolen-Hoeksema & Morrow, 1993). It could be argued that the minor adaptations made in the current study improved the effectiveness of the Response Styles Manipulation as some past work has failed to find evidence of rumination in the rumination condition (e.g., Young & Nolen-Hoeksema, 2001). The current study also improved upon past methodology by being among the first studies to include a manipulation check during the ER induction, thereby allowing rumination to be quantified across group. Nevertheless, the fact that group differences were found within the rumination condition calls into question whether the rumination induction was strong enough to impact recovery from stress in controls. Null results in the control group should therefore be interpreted with caution.

An additional limitation is that several participants had medical conditions, were in the post-menopausal phase, or were taking medication that could have possibly impacted cortisol levels at the time of testing. Although the sample size in the current study prevented us from examining the effects of specific medication classes on cortisol recovery, the pattern of results remained consistent when these participants were removed from the analyses. That the pattern of findings remains the same despite reduced power speaks to the strength of our findings.

The current sample only included individuals with current MDD or those without a history of an Axis-I disorder (CTLs). Recent evidence also demonstrates altered cortisol response to stress among those at genetic risk for depression (e.g., see Ising & Holsboer, 2006, for a review). Future work might consider examining individuals at genetic risk for depression who have not yet experienced a major depressive episode. Focusing on an at-

risk sample would help elucidate whether the observed consequences of rumination are causal factors in MDD or are correlates of a depressive episode.

In the current study, we focused on the maladaptive components of rumination. However, it is important to acknowledge that rumination is multifaceted. In fact, studies have identified more adaptive outcomes of rumination that is focused on positive emotions (e.g., Bryant, 2003; Wood et al., 2003). Conversely, distraction is also not one sided. Although generally considered an adaptive ER strategy, persistent distraction is believed to be an ineffective long-term ER strategy because it hinders effective problem solving (e.g., Campbell-Sills & Barlow, 2007, and Kross et al., 2005). Along these lines, evidence from the current study suggests that it may be important to differentiate active versus passive forms of distraction (e.g., Weiss et al., 2011). Future work, therefore, might do more to examine times and circumstances under which rumination and distraction might be more or less harmful to participants' recovery from stress.

Lastly, the current study focused on RSA and cortisol as our biological indices. This decision was made because RSA and cortisol have been identified as primary markers of the human stress response. Additional biological indices, however, might also provide unique information. Future research might therefore consider including additional measures, such as alpha-amylase or catecholamines, which have been show to be influenced by psychosocial stress (e.g., Rohleder, Nater, Wolf, Ehlert, & Kirschbaum, 2004).

Conclusions

The current study provides important information about how rumination may increase risk for depression. Evidence suggests that it is not simply exposure to stressful

life events that increases depression vulnerability, but rather the way emotions are regulated in response to stress (e.g., Flynn & Rudolph, 2007). Rumination has been identified as a particularly maladaptive emotion regulation strategy, yet we do not fully understand why rumination increases risk for MDD. Results from the current study suggest one underlying mechanism may be that rumination hinders depressed people's ability to psychologically and biologically recover from stress. By prolonging negative mood states, rumination contributes to the hallmark symptom of depression, sustained negative affect. In addition, chronic cortisol elevations can disrupt functioning in brain regions directly responsible for responding to and regulating emotion, including the prefrontal cortex, amygdala, and hippocampus (Gold, Drevets, & Charney, 2002). Complementary chronic low RSA is thought to mediate the increased risk for cardiac mortality found in depression (e.g., for a review see Musselman, Evans, & Nemeroff, 1998). Understanding factors, such as rumination, that prolong psychological and biological dysregulation, may therefore provide critical insights into risk for and severity of depressive episodes.

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NOTES

[1]Inconsistent degrees of freedom reflect that some participants elected not to provide some demographic or health information. Overall, the number of questions participants elected to skip was low.

[2] Concentration data was not provided for two participants (1 CTL, 1 MDD). One participant in the CTL group provided illegible written responses during the ER induction and could not be included in these analyses. One participant in the CTL group did not provide valid negative affect data and had to be excluded from all analyses.

[3]Eight participants (5 MDDs and 3 CTLs) were not included in the cortisol analyses because of errors in extracting cortisol from their saliva samples.

[4]One additional participant in the CTL group was excluded because baseline cortisol value was more than 10 standard deviations greater than the mean.

[5]Fourteen participants (7 MDDs and 7 CLTs) were not included in the RSA analyses because of errors when collecting the psychophysiological readings (e.g., sensors becoming detached, excessive movement by the participant, or power outage).

[6]The time by group by condition interaction remained significant at the quadratic level when we excluded participants with medical conditions ($n = 2$), post-menopausal ($n = 4$), or on medication ($n = 2$) that could impact RSA or cortisol, $F(1, 93) = 5.39$, $p < .05$, $\eta^2 = .05$.

[7]The time by group by condition interaction remained significant when we excluded participants with medical conditions, post-menopausal, or on medication that could impact RSA or cortisol, $F(6, 510) = 11.86$, $p < .01$, $\eta^2 = .03$.

[8]The time by group by condition interaction remained at the cubic level when we excluded participants with medical conditions, post-menopausal, or on medication that could impact RSA or cortisol, $F(1, 81) = 3.28$, $p = .07$, $\eta^2 = .04$.

Table 1

Overview of the study design

Time Point	Measures
Prescreening	Phone screen for initial inclusion/ exclusion criteria
Session 1	1. SCID-I 2. If eligible, questionnaires
Session 2 (see Figure 2)	1. Exposure to psychosocial stress, ER induction, recovery period - RSA, cortisol, and affect taken throughout. 2.Questionnaires

Table 2

Participant Characteristics

	CTL (N = 55)		MDD (N = 51)	
Variable	Rum	Dist	Rum	Dist
Age (*SD*)	34.41 (11.39)	39.18 (11.51)	40.08 (12.05)	36.58 (11.86)
Sex (female: male)	7:20	13:15	12:13	16:10
Yrs Ed (*SD*)	13.91 (2.19)	14.42 (2.84)	13.52 (2.60)	15.33 (2.31)
Income (%)				
< $10,000	19.23	32.14	47.83	40.00
$10,000-$25,000	34.62	17.86	26.09	32.00
$25,000-$50,000	38.46	39.29	17.39	16.00
$50,000-$75,000	0.00	3.57	4.35	8.00
$75,000-$100,000	7.69	3.57	4.35	0.00
> $100,000	0.00	3.57	0.00	4.00
% Caucasian	38.46	39.29	36.00	30.77
% Smokers	16.00	25.00	43.48	29.17
% Engage in physical exercise	80.77	70.83	40.91	41.67
% Ate in last 2 hrs	0.00	3.70	0.00	3.85
BMI (*SD*)	27.10 (4.88)	28.73 (4.89)	27.15 (7.49)	26.64 (7.63)

# Cups Caffeine	0.92 (1.23)	1.32 (2.25)	1.14 (1.46)	1.79 (2.04)
% Drank Caffeine				
Today	28.00	28.00	40.91	45.83
Hrs Awake Before				
Cort1	7.77 (2.28)	6.89 (1.76)	7.32 (2.01)	7.22 (2.44)
Phase Menstrual				
Cycle (%)				
Not regular	14.29	40.00	16.67	0.00
Menstruation	14.29	0.00	16.67	16.67
Proliferative	28.57	20.00	33.33	0.00
Ovulation	14.29	20.00	0.00	16.67
Luteal	28.57	20.00	33.33	66.67
BDI (*SD*)	3.70 (6.52)	4.14 (5.21)	29.83 (11.74)	30.31 (11.61)
RSQ (*SD*)	31.08 (10.03)	31.36 (8.40)	66.38 (8.57)	33.73 (10.59)
% on Medication	11.11	10.71	32.00	38.46
# Comorbid (SD)	NA	NA	2.68 (0.99)	2.65 (1.57)

Note. CTL = control group; MDD = Major Depressive Disorder; Rum = Rumination Condition; Dist = Distraction Condition; BMI = Body Mass Index; BDI = Beck Depression Inventory; SRQ = State Rumination Questionnaire; RSQ = Response Style Questionnaire

Table 3

Area Under the Curve (AUC) to ground by group (CTL, MDD) and condition (rumination, distraction)

	CTL (N = 51)		MDD (N = 46)	
AUC	Rum	Dist	Rum	Dist
Mean	506.01	439.08	476.95	636.61
SD	246.97	247.92	618.72	536.83

Note. CTL = control group; MDD = Major Depressive Disorder; Rum = Rumination Condition; Dist = Distraction Condition; AUC = Area Under the Curve

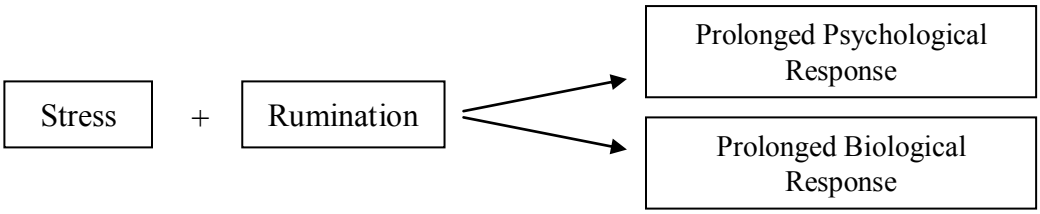

Figure 1. Model for prolonged response to stress in MDD

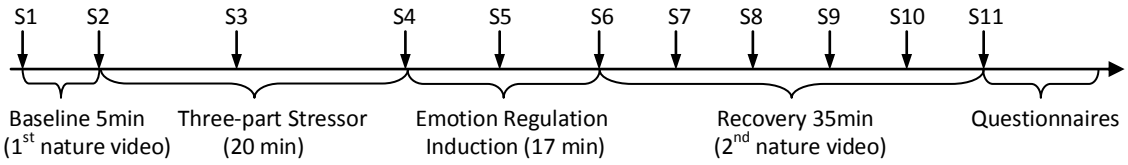

Figure 2. Procedure of Session 2. Negative affect ratings were provided at S1-S11, except not at S5. Cortisol samples were provided at S3-S11. RSA was measured during baseline, stressor, emotion regulation induction, and first 10 minutes of recovery.

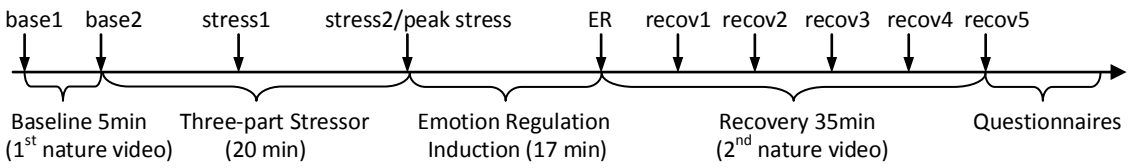

Figure 3. Timing of negative affect ratings.

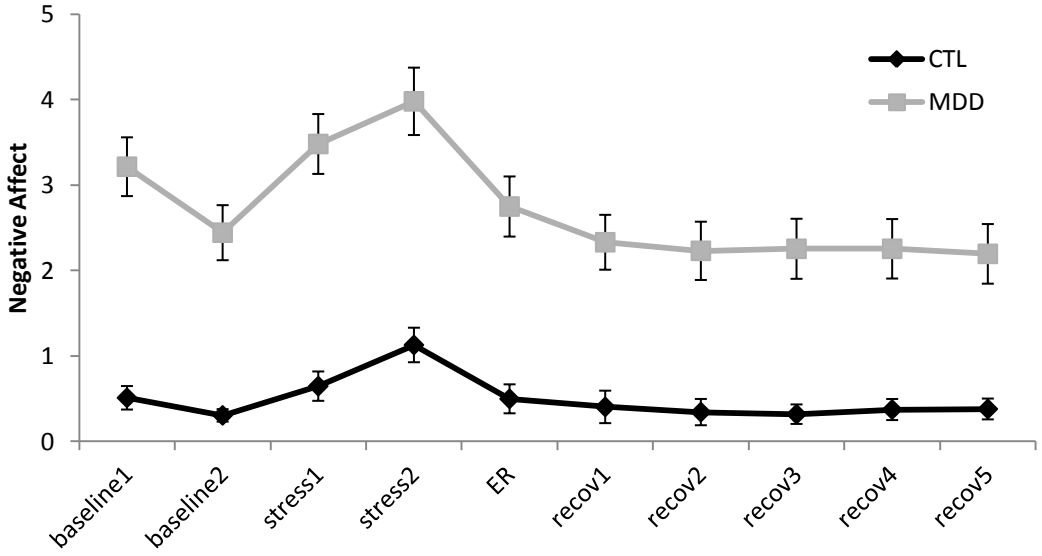

Figure 4: Negative affect ratings in the control (CTL) and depressed (MDD) groups. Error bars = +/- 1 SE.

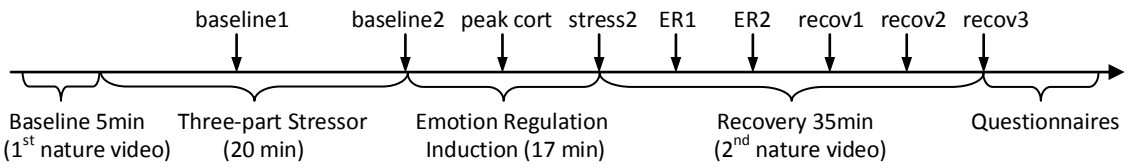

Figure 5. Timing of cortisol samples.

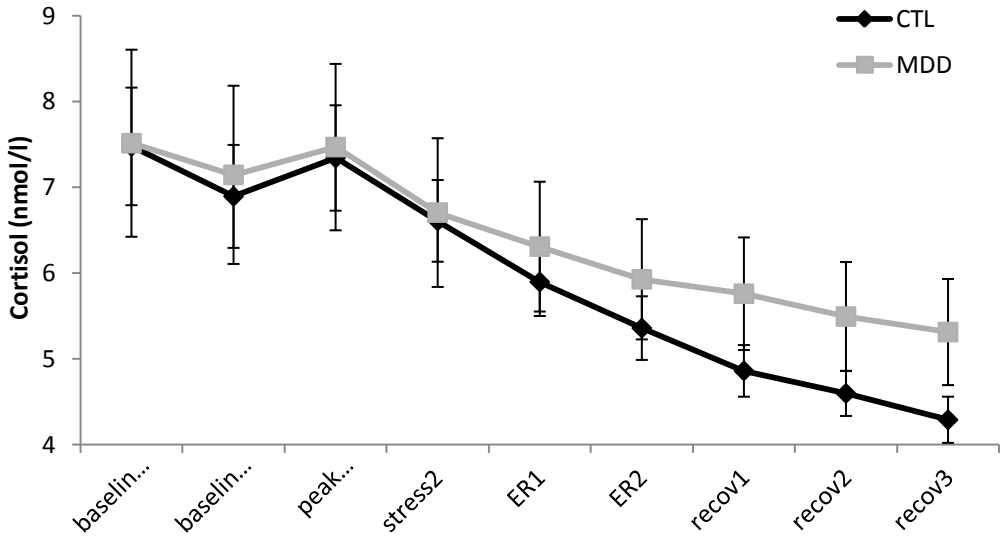

Figure 6: Cortisol levels in the control (CTL) and depressed (MDD) groups. Error bars = +/- 1 SE.

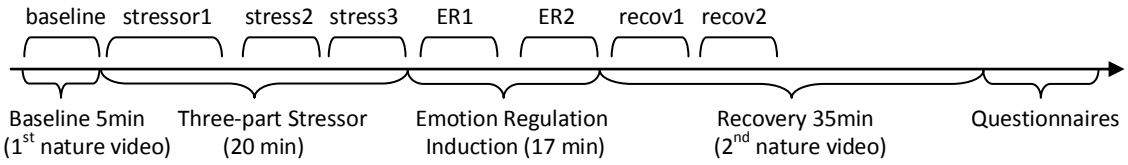

Figure 7. Timing of RSA measurement.

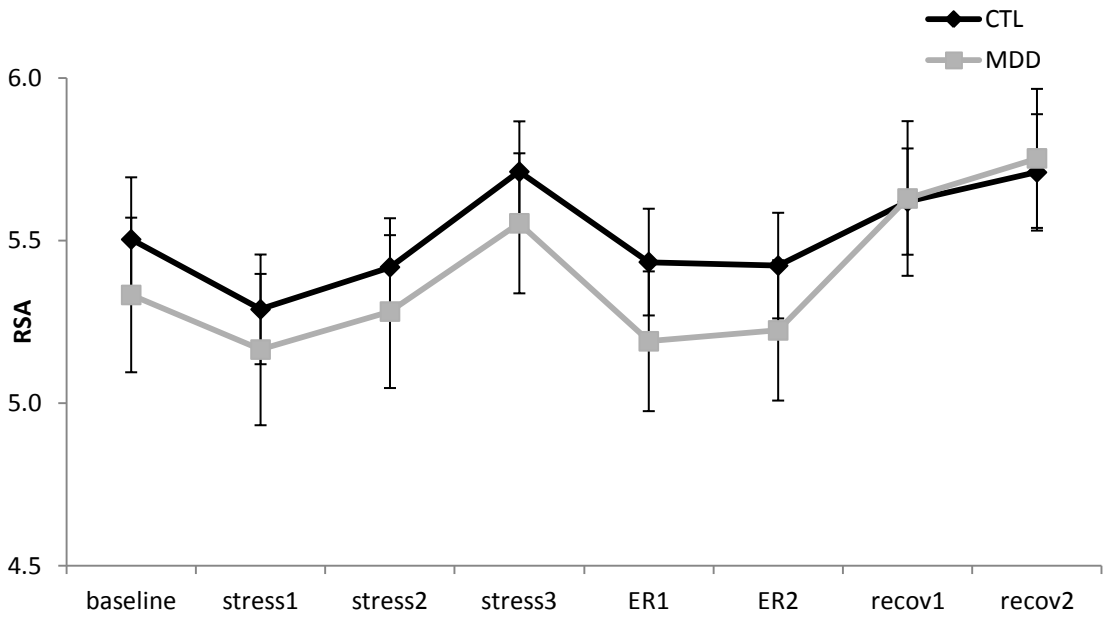

Figure 8: RSA levels in the control (CTL) and depressed (MDD) groups. Error bars = +/- 1 SE.

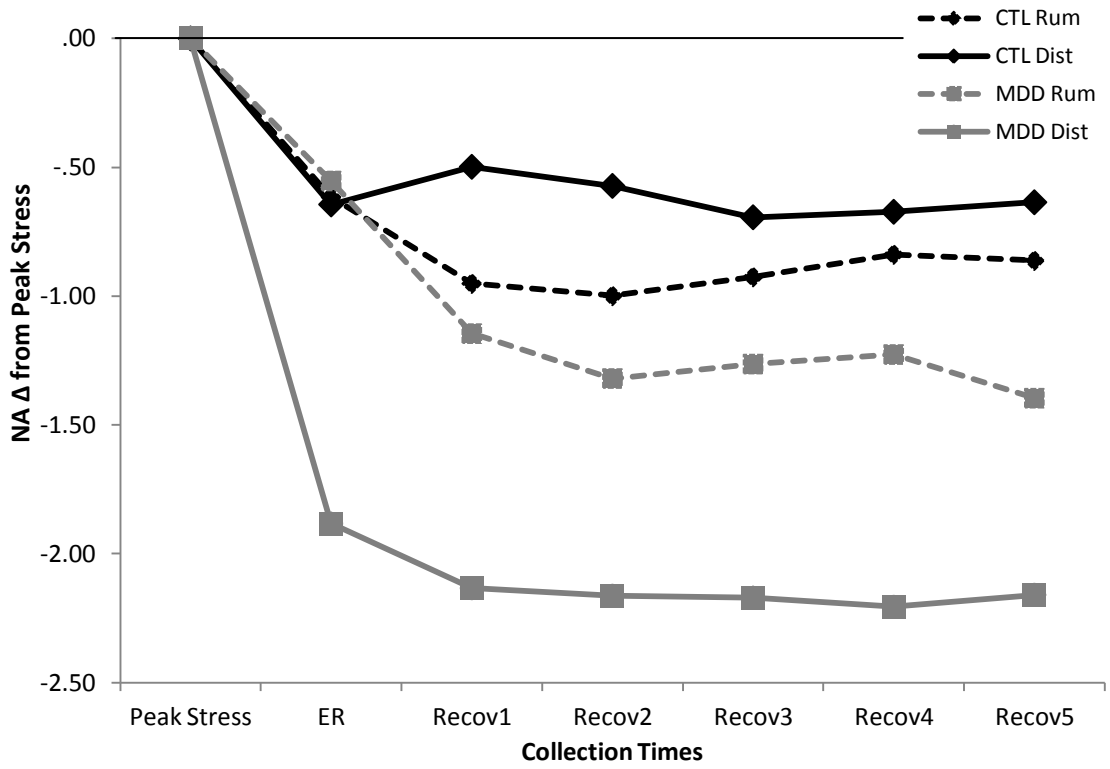

Figure 9: Change in Negative Affect (NA) from peak stress

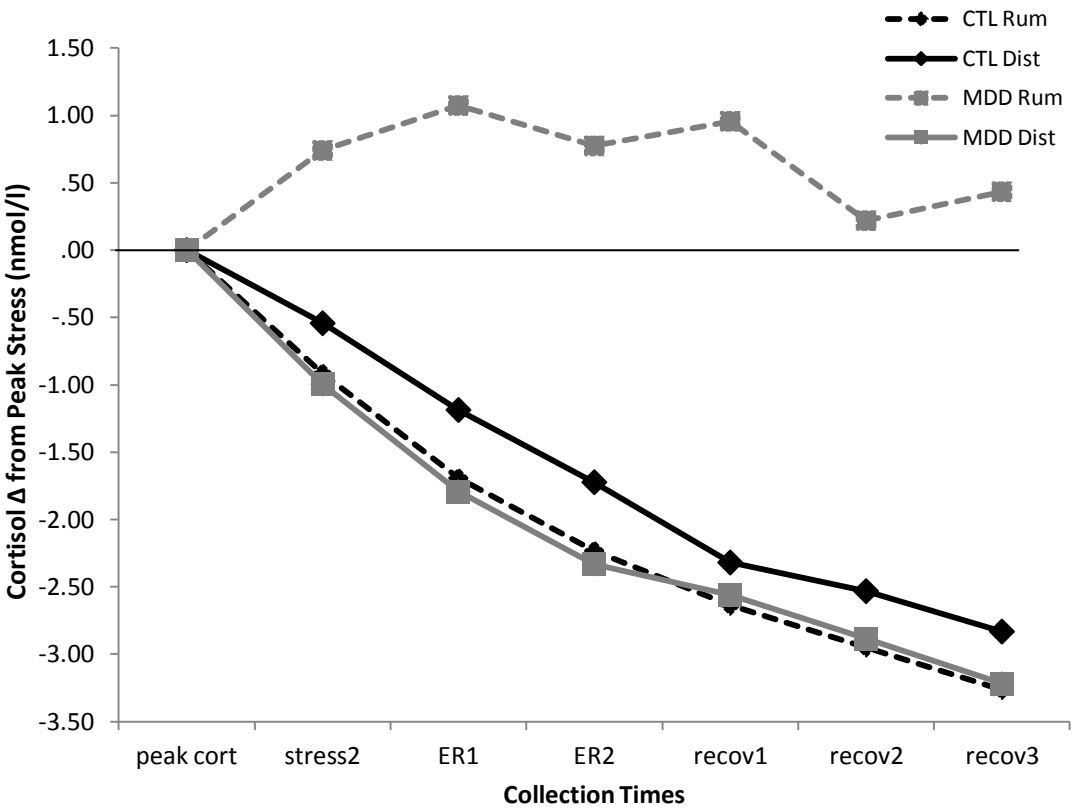

Figure 10: Change in cortisol from peak stress

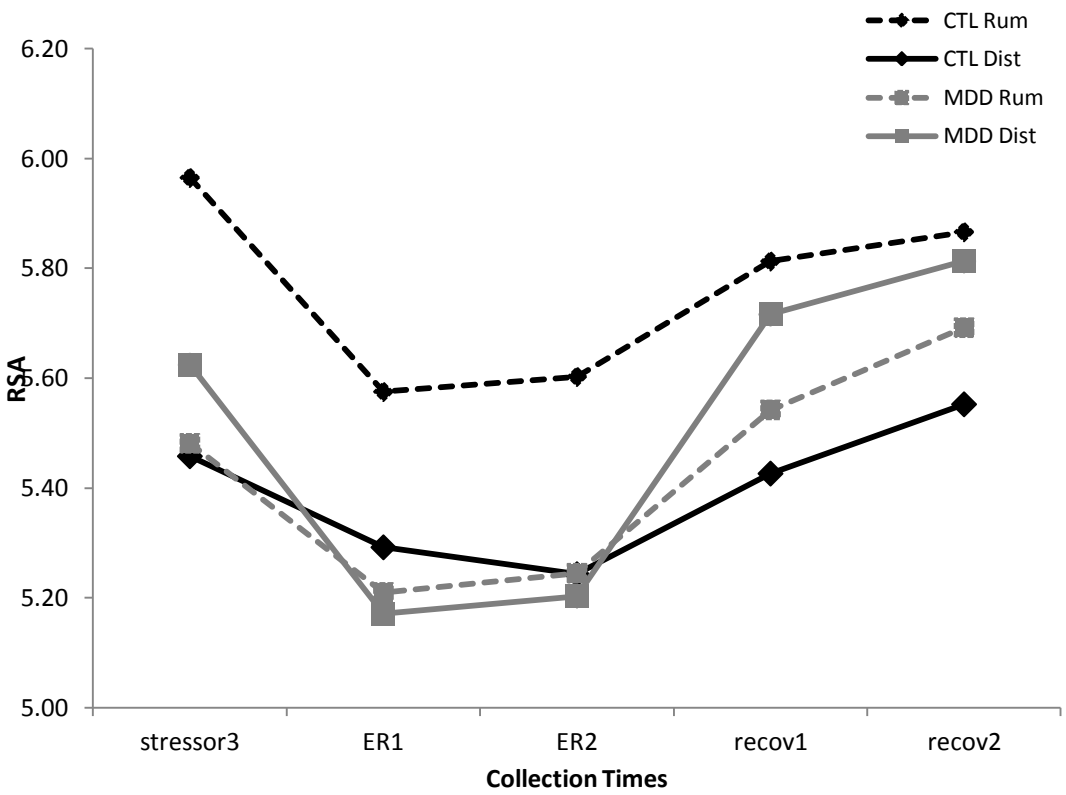

Figure 11: RSA levels during emotion regulation induction and recovery periods.